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ÍNDICE

Harold Davis, BA, RVT, VTS

1.1.1. CPR an Update

Harold Davis, BA, RVT, VTS (ECC) (Anesth & Analgesia)

INTRODUCTION

Cardiopulmonary arrest (CPA) can be considered the ultimate emergency. CPA is defined as the sudden cessation of functional ventilation and effective circulation. The veterinary clinic health care team may be involved with the management of a CPA. It is the responsibility of the staff to be prepared to respond to such an emergency. In 2012 RECOVER (Reassessment Campaign on Veterinary Resuscitation) guidelines were established for the management of cardiopulmonary resuscitation. This discussion will review the principles of CPR along with the recommendations from the RECOVER guidelines.

Preparation

Preparation is key to the management of a cardiac arrest. Clinic staff must be trained in the principles of CPR. Initial training can take place online. For more information go to https://recoverinitiative.org/first-responders-other-pet-professionals/cpr-bls-als/

The ideal number of participants in a resuscitation attempt is three to five. Practice drills should be held at least monthly. A stuffed animal can be used as the patient during these drills. Each person should understand what his or her responsibilities would be during an arrest. After each practice session or actual resuscitation, a team self-evaluation should be performed.

Where will the resuscitation attempt take place? Some people prefer to perform CPR wherever the patient is located and bring the resuscitation equipment and supplies to the patient. Others prefer to designate an area in the clinic. When selecting an area take into consideration the space available; is there enough room for a CPR team (3 plus people) and equipment? An oxygen source should be readily available. Good lighting is a must. If CPR is to be performed on a table, then the height of the table should be adjustable. If the height of the table is not adjustable then a footstool should be made available, or CPR should be performed on the floor

Crash carts/kits help to make the resuscitation endeavor more efficient by having all the supplies readily available. If a cart is used then in addition to the endotracheal tubes, drugs, catheters, syringes etc. equipment may be stored on the cart such as suction machine, ECG, and defibrillator. The crash cart or kit should be checked at the beginning of each shift and restocked immediately after each use.

CPR PROTOCOL

The RECOVER guidelines include an algorithm (flowchart) to guide the CPR endeavor as well as a drug dosage chart. Both can be obtained from the Veterinary Emergency and Critical Care Society (www.VECCS.org).

RECOGNITION OF CPA

The existence of cardiac arrest must be recognized early if we are to effectively resuscitate the patient. The ABCs should be rapidly checked in the apneic unresponsive patient. The absence of a palpable pulse, audible heart sound, or effective ventilation (agonal breaths should not be considered effective breaths) all supports the assessment of cardiopulmonary arrest. Even under the best of circumstances it may be difficult to palpate a pulse therefore, not much time should be spent trying to assess pulses. If there is any question that CPA has taken place the patient should be treated as such until proven otherwise.

BASIC LIFE SUPPORT (BLS)

Circulation

Once it has been recognized that a CPA has taken place chest compression can be instituted without waiting to intubate the patient, in other words intubation and chest compression can be carried out simultaneously. Chest compressions are performed with the patient in lateral recumbency, alternatively if the patient has a barrel chest confirmation it may be placed in dorsal recumbency and the sternum is compressed. With the arms extended and locked, the hands are placed one on top of the other and the fingers interlaced, then the hands are placed over the appropriate area on the chest. The shoulders are squarely over the patient's chest and the compressive force is applied by bending at the waist. The person delivering the chest compressions should not compress the chest by bending the elbows; it will be difficult to generate an appropriate force to affect perfusion. To

take advantage of the cardiac pump mechanism in deep or keel chested dogs we should apply pressure laterally, directly over the heart and compress the chest 1/3 – ½ its width, at a rate of 100 to 120 compressions per minute. In the case of medium to giant patients the hands are placed over the widest portion of the chest and compressions are performed; this will take advantage of the thoracic pump mechanism. Alternately in cats and small dogs, a one-handed circumferential chest compression with the hand wrapped around the sternum directly over the heart or the thumb and first two index fingers can be used to compress the chest (taking advantage of the cardiac pump mechanism). Allow the chest to recoil completely between compressions, approximately equal compression and relaxation modes. It is not necessary to try and synchronize breaths between compressions. The person delegated to compress the chest should change every 2 minutes to prevent fatigue; this is considered one BLS cycle. Minimize interruptions to chest compressions; make interruptions no longer than 10 seconds

Airway / Breathing

Following confirmation of a patent airway, an endotracheal tube is inserted. If there is absence of effective ventilation, then positive pressure ventilation should be begun with 100% oxygen. The ventilatory rate is one breath every six seconds (10 breaths / min) with a tidal volume of 10 ml/kg and an inspiratory time of 1 second. Hyperventilation should be avoided. Higher respiratory rates, longer inspiratory times and higher tidal volumes lead to impaired venous return due to increased intrathoracic pressure as well as decreased cerebral and coronary perfusion due to vasoconstriction, all of which has led to poor outcomes in people.

Assessing Effectiveness

The effectiveness of the team's efforts must be monitored frequently. Improvement in mucous membrane color and the presence of a palpable pulse during CPR has been used for assessing effectiveness. However, even in the best of circumstances palpation of a pulse can be difficult. The placement of a Doppler flat probe on the cornea has been used as a tool to assess effectiveness but it should be used with caution. There is potential for motion artifact or retrograde venous blood flow and not arterial flow. If a direct arterial line is in place, arterial pressure waveforms and pressures can be used to assess effectiveness of therapy. In essence you will have a compression-to-compression assessment of your technique. The goal is to achieve a diastolic pressure of 40 mm Hg or greater. There is strong evidence supporting the use of end-tidal CO2 (ETCO2) to non-invasively assess resuscitation efforts. Studies have shown that ETCO2 varies directly with cardiac output during cardiac arrest. Dramatic decreases in ETCO2 occur during cardiac arrest; with effective CPR increase in ETCO2 is seen. Data suggest that ETCO2 values of >15 mmHg in

the dog and >20 mmHg in cats may be associated with a higher rate of return of spontaneous circulation.

Internal Cardiac Massage

If external chest compression is not effective, then the doctor may elect to perform internal cardiac massage. Internal cardiac massage is associated with better cardiac outputs, cerebral and coronary perfusion. If external cardiac compressions are not effective (generation of a palpable pulse, improved mucous membrane color, detection of blood flow with Doppler or increase in ETCO2) within 5 minutes or if the heart has not started beating spontaneously in 10 minutes, then internal cardiac massage should be initiated. In some cases, it might be indicated to go with internal massage from the outset.

ADVANCED LIFE SUPPORT

Categories of Cardiac Arrest

The type of cardiac arrest dictates which therapy will be utilized in CPA. There are three primary categories of cardiac rhythms seen during a CPA event: perfusing, non-shockable (asystole and pulseless electrical activity [PEA] and shockable (ventricular fibrillation [VF] and pulseless ventricular tachycardia [PVT]).

Asystole (Figure1) is characterized by no electrical or mechanical activity. On the ECG, a flatline represents no electrical activity. There will also be no detectable pulse or heartbeat. Epinephrine (Adrenaline) / vasopressin and atropine are the primary drugs used to treat this rhythm.

Pulseless rhythms (Figure2) may be near normal in appearance or wide and bizarre QRS complexes. There will also be no detectable pulse or heartbeat. RECOVER guidelines state that if the QRS is repeatable and the heart rate is less than 200 and there are no pulses then consider the rhythm to be PEA. Epinephrine or vasopressin is indicated, and fluid bolus and atropine should be considered.

Ventricular fibrillation (Figure 3) is characterized by chaotic electrical activity and no coordinated mechanical activity. The ECG display will show no definable pattern, marked irregularity in rhythm, P waves and QRS complexes are unidentifiable. There will also be no detectable pulse or heartbeat. Defibrillation is the treatment of choice.

RECOVER guidelines characterize pulseless ventricular tachycardia (Figure 4) as a repeatable QRS complexes with a rate greater than 200 beats per minute and absent pulses. This is considered a shockable rhythm.

Table 1 Therapies used during CPR. All drugs are given IV.

Therapy	Action	Indication	Comment
Epinephrine (Adrenaline)	Arterial vasoconstrictor; increases diastolic pressure resulting in augmented coronary and cerebral blood flow	Asystole PEA Prolonged VF/VT	Dose every other 2 min BLS cycle
Vasopressin	Direct smooth muscle vasoconstrictor	Asystole PEA Prolonged VF/VT	Alternative to epinephrine
Atropine	Parasympatholytic	CPA associated with intense vagal stimulation	
Crystalloid Fluids	Increase perfusion	hypovolemia	If known to have prearrest hypovolemia
Reversal Agents	Naloxone → opioid Flumazenil → benzodiazepines Atipamezole → Alpha - 2- agonist		Administered if drugs given in peri-CPA period.
Sodium Bicarbonate	Increases pH to correct metabolic acidosis	CPA > 10 – 15 minutes	

Defibrillation

Defibrillation is the treatment of choice for ventricular fibrillation (VF) and pulseless ventricular tachycardia (PVT). If the patient has VF or PVT and the duration is less than 4 minutes or if VF is diagnosed during a rhythm check

between BLS cycle the heart is defibrillated immediately. However, if the duration of VF or PVT has been suspected to be greater than 4 minutes then a 2-minute cycle of BLS must be performed prior to defibrillation.

The precordial thump is a method of mechanical defibrillation where the patient is struck with the heal of the hand directly over the heart in the hopes of converting the patient. While there is minimal efficacy for this technique it should be considered only if a defibrillator is not available.

1.1.2. FLUID THERAPY AN OVERVIEW

Harold Davis BA, RVT, VTS, (ECC)

Fluid therapy can be a major component to the management of the emergent or critically ill patient.

Determining Fluid Requirements

A patient history along with a complete physical examination is the foundation for the development of a fluid therapy plan. It is important to determine the degree of dehydration and the perfusion status of the patient prior to beginning fluid therapy. There are several clinical and laboratory methods which may be used to determine the hydration status of the patient.

History

The owner should be asked questions about food and water intake. Is the animal eating? If not, when did it last eat? Is the animal drinking water, if so, increased, or decreased amounts? Is the animal suffering any abnormal losses such as vomiting, diarrhea, or polyuria? What is the duration of theses abnormal losses?

Physical Findings

Skin turgor or skin elasticity is a crude way of determining the interstitial compartment volume (hydration) status. When assessing skin turgor its best to use the same location for consistency in technique. The lateral thorax or between the shoulder blades are good locations to assess skin turgor. With 5% dehydration the skin, when lifted, will return to its normal position quickly but slightly slower than normal. With 8% dehydration the skin returns to its normal position slower than 5% dehydration but faster than 10% dehydration. When the patient is 10% dehydrated the skin will remain tented and not return to its normal position. Elasticity of the skin is affected by cachexia and obesity. It is possible to have a normally hydrated patient that has reduced skin elasticity due to cachexia; or a dehydrated patient that has normal skin elasticity because of being fat. Other signs consistent with dehydration include dry skin and mucus membranes, oliguria, and signs of compensatory peripheral vasoconstriction. Perfusion is assessed by evaluating 6 parameters known as the perfusion parameters. They include mentation, mucous membrane color, capillary refill time, heart rate, pulse quality, and extremity temperature. In poor perfusion states it is not uncommon to see decreased mention, pale mm color, prolonged capillary refill, increased heart rate (in the dog), cool extremities, and poor pulse quality.

Laboratory Analysis:

Packed cell volume (PCV) and total protein (TP) are simple tests that can be used to evaluate hydration. PCV and TP are often elevated with dehydration. In an animal with both anemia and dehydration, the PCV may appear to be normal, but this is due only to hemoconcentration. A urine specific gravity greater than 1.030 usually indicates that the kidneys are responding to the dehydration in an appropriate manner. Electrolyte and acid base status can be a valuable addition to evaluation of the emergency patient. Depending on the disease process, it is not uncommon to find electrolyte abnormalities with sodium and / or potassium. Lactate levels > 2 mmol/L suggest poor perfusion and inadequate tissue oxygenation. Monitoring lactate can help identify patients that may benefit from fluid therapy as well as a monitoring tool to determine if fluid therapy provided was adequate.

Dehydration vs Hypovolemia

It is important to recognize the difference between dehydration and hypovolemia. Dehydration is a decrease in interstitial fluid volume as evidenced by dry mucous membranes, and decreased skin elasticity. Hypovolemia is a decrease in circulating blood volume and evidenced by poor perfusion parameters. Severe dehydration can lead to hypovolemia; however, a hypovolemic patient does not

have to be dehydrated (e.g., a previously healthy dog that has suffered trauma). The fluid deficit occurring with dehydration is corrected over hours (commonly 6 – 12 hours) while hypovolemia should be corrected in less than one hour.

Fluid Type	Total Shock Dose*	
	Dogs (mL/kg)	Cats (mL/kg)
Isotonic crystalloids	80 – 90	50 – 55
7.5% Hypertonic saline	4 – 6	3 – 4
Synthetic colloids*	10 – 20	5 – 10
7.5% Hypertonic saline & synthetic colloid	1.5 – 3 & 3 - 6	1.5 & 3
Poor Perfusion/Hypovolemic Patient		

Table 1 Fluid doses for various types of fluids and combination. *Note Vetstarch and Voluven may be dosed up to 40 mL/kg

If the initial patient assessment revels that the patient is poorly perfused or hypovolemic then a fluid plan will need to be instituted to address the deficits. The approach to fluid therapy in the hypovolemic patient is approached differently than the simple dehydrated patient. A commonly cited fluid dosage goal of isotonic crystalloids for hypovolemia is 80 - 90 mL/kg/hr for the dog and 50 - 55 mL/kg/hr for the cat (equivalent to one blood volume). Individual animal requirements are variable it may be necessary to administer more or less of this volume to effectively resuscitate the patient. To administer the fluids, a commonly used approach is to administer ¼ dose increments rapidly and then reassess the perfusion parameters looking for improvement or resolution in the signs of poor perfusion. For example, a dog or cat would be given approximately 20 and 10 mL/kg incremental doses of fluids respectively. It is necessary to reassess the patient's condition frequently (i.e., about every 10 - 15 minutes) during large or rapid volume fluid administration. Resuscitation is not limited to crystalloid fluids. Hypertonic saline, synthetic colloids and blood products may be indicated.

Simple Dehydrated Patient

The basic components of a fluid therapy plan (for the simple dehydrated patient) include the determination, calculation, and replacement of the volume deficit (percent dehydration); abnormal on-going losses; and maintenance needs.

To determine the volume deficit replacement, multiply the percent dehydration by the patient's body weight (kg), this will equal the volume of fluids in liters estimated to correct dehydration. Losses through vomiting, diarrhea, excessive urination, burns and transudation into body cavities are abnormal ongoing losses. This volume of these losses should be estimated and calculated into the fluid therapy plan. Typically, abnormal losses should be replaced mL for mL. Normal losses occur through breathing, salivation, urination, and defecation and these are accounted for with the maintenance fluid rate. A rough rule of thumb for the maintenance fluid rate is to give 50 75 mL/kg per day or 2 – 3 mL/kg/hr. Once the volume deficit replacement needs, abnormal ongoing losses and maintenance needs are calculated, the three are totaled. The fluid infusion rate may be determined by totaling up the volume of fluids to be given and dividing that by the total number of hours over which the patient is to be rehydrated. If there aren't enough hours available to safely administer the fluids the patient may be sent to an emergency clinic for continued care or give some of the required fluids subcutaneously. Generally, we like to correct the fluid deficit (volume needed to correct dehydration) over four to eight hours.

Potassium chloride might be added to fluids when serum potassium levels are known to be low (hypokalemia). If potassium levels are not known, hypokalemia may be expected in cases of fluid loss due to gastrointestinal loss, diuresis, and anorexia. Based on the magnitude of these losses the body potassium depletion is thought to be mild, moderate, or severe, the fluids should be supplemented with potassium to 20, 30, or 40 mEq/L respectively. Fluids are not routinely supplemented with potassium if given at faster rates for hypovolemia or rehydration. Potassium should not be administered at a rate faster than 0.5 mmol/kg/hr.

Because the calculation of fluid volume is based on subjective data, potential inaccuracies occur. Therefore, it is necessary to reassess (physical exams [including perfusion parameters], body weight, ins and outs, and pertinent lab test) the patient often. Your patient may require more or less of the original calculated fluid volume; being careful not to cause fluid overload. You are looking for a resolution in the signs that indicated that the patient needed fluids.

1.1.3. INITIAL ASSESSMENT OF THE EMERGENCY ROOM PATIENT

Harold Davis, BA, RVT, VTS (ECC)

INITIAL OBSERVATION

An initial ‘eye-ball’ of the patient provides important information, before any hands-on contact is made. In this time two major issues are addressed. First, how serious does the situation look? Obviously, the animal that can maintain sternal recumbency and is aware of its surroundings is far less concerning than the animal in lateral recumbency with no apparent response to external stimuli.

Second, are there any obvious life-threatening problems that will require attention at or before the time you can evaluate the A-B-Cs? Problems such as arterial hemorrhage or an open chest wound may warrant immediate intervention.

The following patient assessment is then performed within 1-2 minutes. Flow by oxygen should be provided until the assessment is complete and the requirement for ongoing oxygen therapy has been adequately evaluated.

PATIENT HISTORY

The animal's signalment (age, sex, and breed) can provide direction for the diagnostic workup. For example, pediatric or juvenile patients are more likely to contract infectious conditions. Respiratory distress due to heart failure is noted more frequently in male than female cats. Feline asthma is noted more frequently in Siamese cats compared with other breeds. Signalment provides clues to the underlying disorder but never provides a definitive diagnosis.

Does the patient have any pre-existing medical conditions or on medications? Historically, the owner may be able to provide information that supports a reason for hypovolemia such as trauma, excessive urination, diarrhea, or vomiting. What is the travel history of the pet? Pet's environment gives the veterinarian a sense of potential exposure to toxins or infectious diseases.

PHYSICAL ASSESSMENT

A = Airway:

All emergent and critically ill animals should have the patency of their airway evaluated. This involves assessment of the gag reflex and ensuring no airway obstruction is evident. For many patients this is a cursory examination as they show obvious swallowing and resistance to opening of their mouth and there is an absence of signs of obstruction. Recumbent animals that have reduced jaw tone should be closely evaluated for the presence of a gag reflex.

B = Breathing:

Following evaluation of the airway it is necessary to confirm that the animal is making breathing efforts. Is the effort normal, decreased or increased? Are breath sounds normal, diminished, or increased? Can the breath sounds be characterized (crackles, wheezes)? By observing the patient breathing and listening to breath sounds one may be able to localize the breathing difficulty to the pulmonary parenchyma, pleural space or thoracic wall.

C = Circulation:

Assessment

There are 6 physical examination parameters that allow a rapid evaluation of the circulatory status. Keep in mind that circulatory shock is a clinical diagnosis.

1. Mentation
2. Mucous membrane colour
3. Capillary refill time
4. Heart rate
5. Pulse quality
6. Extremity temperature

These clinical findings should be evaluated in combination; an abnormality in a single parameter does not have the clinical significance of multiple abnormalities.

Common clinical findings of circulatory shock:

- Reduced mentation
- Pale or white mucous membranes
- Slow capillary refill time
- Tachycardia
- Reduced pulse quality
- Cool extremities in comparison with core body temperature

Patients with vasodilatory shock can have red mucous membranes, rapid CRT, bounding pulses and warm extremities. As these animals are likely to present

with concurrent hypovolemia these signs of vasodilation may not be evident until after adequate fluid resuscitation.

Tachycardia is the appropriate and expected response to circulatory shock. The presence of normocardia or bradycardia in canine shock patients (ie. Patients with abnormalities of the other 5 parameters) is of concern as it suggests decompensated shock and is associated with greater severity of illness and a poorer prognosis. Feline shock patients will often present without a tachycardia and this is not considered to have prognostic relevance as it does in dogs.

D = Dysfunction (neurologic):

Assessment:

1. Mentation
2. Pupils
3. Posture

Descending from a higher level of consciousness to a lower level, suggest worsening neurologic involvement. ocular trauma. Pupils that are poor to non-responsive with unilateral or bilateral mydriasis, bilateral miosis, or pupils that are miotic and become mydriatic suggest neurologic involvement. Three types of abnormal postures are: Schiff-Sherrington, decerebellate and decerebrate rigidity; these postures suggest spinal cord, cerebellar, or brain stem injury respectively.

E = Exposure & Environmental Control:

In human medicine exposure is an essential aspect of patient evaluation as clothing can hide serious injuries or abnormalities. Once an animal is considered safe for movement the removal of blankets, turning the patient etc is important for the same reasons. Environmental control in the veterinary context is the removal of any source of ongoing harm. For example, washing off caustic liquids or inducing emesis of a recently ingested toxin.

Body Temperature:

Body temperature is not addressed in the standard ABCs and may not be required for the assessment of every patient. But extremes of body temperature

are an indication for urgent medical attention and as a result the temperature of at-risk patients should be measured during assessment. Any recumbent, poorly responsive patient should have their temperature measured, especially in small patients who are extremely prone to hypothermia. Animals that are rapidly panting or have obvious signs of exertion such as tremors or seizures should also have their temperature evaluated.

Summary

Following initial stabilization and management a definitive care plan is developed for further diagnostics, therapy, monitoring, and supportive / nursing care.

1.1.4. Gaining and Maintaining Venous Access

Harold Davis BA, RVT, VTS (Emergency & Critical Care) (Anesthesia/Analgesia)

Gaining venous access is an important life saving measure in the management of the emergent or critically ill patient. Once venous access has been obtained it may be used to administer fluids and/or medications; provide nutritional support facilitate cardiovascular monitoring; or collect blood samples.

Catheter Insertion Site

Peripheral insertion sites include the cephalic, lateral, and medial saphenous, and the auricular vein.

Central vein insertion sites include the jugular, lateral and medial saphenous veins. To achieve central vein catheterization via the saphenous vein, long catheters must be used; they are threaded so that they lie in the caudal vena cava.

Vein Selection

Selection of a vein depends on several factors such as the skill of the person placing the catheter, available veins, therapeutic goals, and the animal's problem or disease. Any vessel that is visible should be considered a candidate for percutaneous catheterization.

Catheter Insertion Methods

Percutaneous

This percutaneous insertion technique is simple. With this technique, the needle (butterfly or the through-the-needle catheter or the over-the-needle catheter is inserted through the skin, subcutaneous tissues, and ultimately into the vein. A potential problem with this technique is the possibility of the catheter tip flaring or tearing because of the drag on the catheter as it goes through the skin.

Seldinger (Guide wire Technique)

The Seldinger or guide wire technique is used most to place multi-lumen central catheters or long single lumen catheters. A long flexible guidewire is inserted into the vein and ultimately a catheter is threaded over the guidewire and into the vessel. This is described in more detail under multi-lumen catheter.

Central Venous Access via a Peripheral Vein

At times it is desirable to place a peripheral catheter for the purpose of serial blood sample collections. Short peripheral catheters often do not aspirate well and so a long catheter, advanced to a large central vein is used. Long catheters are through-the-needle, and such needles are commonly too large for small patients. In this situation, a common approach is to first place a short over-the-needle catheter into the medial or lateral saphenous vein. A long through-the-needle catheter (the needle has been removed) is advanced into the pre-placed short catheter. The two catheter hubs are joined tightly together, and the catheter is secured to the patient.

Jugular Catheterization

There are times when using a central line is preferred to a peripheral vein. Most veterinary patients better tolerate the jugular catheter. It allows for uninterrupted flow of the IV infusion. Jugular catheters also facilitate the collection of blood samples and make possible the administration of hyperosmotic fluids and CVP measurements. Single or multi-lumen catheters may be placed.

Procedure

A wide area of hair over the catheterization site should be clipped. The insertion site is surgically prepped, and aseptic techniques is employed (gloving and draping).

Patient positioning is key to a successful outcome. Placement of the jugular catheter is best done with the patient in lateral recumbency. It is important that the patient be positioned properly, and the vein immobilized. If the vein is not immobilized properly, it may roll laterally or wrinkle longitudinally.

Through-the-Needle Catheter Insertion

The catheter needle should be introduced subcutaneously. The needle tip is positioned over the vein and aligned as close as possible to the longitudinal axis of the vein. Insert the needle tip into the vein; it may require that you apply a little angle to the needle to pick up the vein wall. Once it is estimated that the entire needle tip is within the lumen of the vein, the catheter is threaded into the vein. Once the catheter is threaded apply pressure over the catheter puncture site and back the needle out. A needle guard is placed, the catheter is aspirated and flushed confirming the placement. The catheter is secured and a lite bandaged is placed.

Multi-Lumen Catheter

Multi-lumen catheters are available in both the over-the-needle and through-the-needle style. Multi-lumen catheters have two to three separate lumens in one catheter. Multi-lumen catheters allow simultaneous infusions at one catheter site. Though one catheter is placed, the multi-lumen catheter provides the same functions as two to three separately introduced single-lumen catheters. The catheter site is prepared as previously described. Catheter placement is usually completed percutaneously with a guide wire technique. Once inserted the catheter is sutured in place and an occlusive bandage is applied.

Catheter Maintenance

Catheter maintenance entails catheter site inspection (looking for signs of phlebitis, thrombosis, infection) and / or fluid infiltration), assessing patency,

cleansing of the site and re-bandaging. Peripheral venous catheters should only be replaced when clinically indicated and routine replacement every 72 to 96 hours is not necessary. If routine catheter care is performed, and the catheter removed when problems are first noticed, one can often exceed the 72- 96-hour rule. A study looking at peripheral and jugular venous catheter contamination in dogs and cats supports this.

Human literature suggests that normal saline (without heparin) may be as effective as heparinized saline in the maintenance of catheter patency. , . Heparinized flushes may be warranted in peripheral catheters placed with the intention of performing serial blood draws.

Complications

Catheter related complications include phlebitis, infection, thrombosis, extravasation of fluids, and catheter emboli.

Vascular Access Options in Difficult Placements

At time vascular access can be difficult for several reasons. It may be due to peripheral edema, tough skin, non-visible, palpable, or “flat” veins which may be secondary to hypovolemia. There are options to consider which may facilitate vascular access.

Tough Skin - Facilitative Incision or Relief Hole

A facilitative incision or relief hole reduces the skin tension and friction against the catheter. It is indicated in severely dehydrated patients or patients with tough skin. A facilitative incision may be made with a number eleven blade or a 20-gauge needle. A 0.5 – 1 millimeter incision is made directly over the vessel extending through the dermis. Care should be taken to avoid the vessel when making the relief incision. Local anesthetic blocks are rarely needed.

Inability to “Raise a Vein” - Application of a Warm Towel

Application of lukewarm towel around the limb for two – three minutes may make the vessel prominent. While the towel is in place the vessel is held off or a

tourniquet is applied, and the paw is repeatedly squeezed. Placement of the warm towel should increase local blood flow resulting in venous distension making the vein prominent.

Inability to See or Feel a Vein – Ultrasound Guided Insertion

Ultrasound guided catheter insertion has been utilized in human medicine. Given the fact that ultrasound units are more prevalent in veterinary practice, ultrasound guidance for difficult catheter placement is becoming more common. There have been a few papers written about the use of Ultrasound Guided catheter insertions; one paper concluded that the ultrasound guided technique was feasible and comparable to the landmark-based technique for placement of central venous catheters in dogs.

Venous Cutdown

A full cutdown is indicated when the patent is severely hypovolemic and hypotensive or if the patient is obese or has subcutaneous edema. This procedure should be done under full aseptic conditions. The risks of cutdown, procedures include bleeding and infection.

Intraosseous (IO) Catheter Placement

In the event vascular access cannot be obtained the establishment of an intraosseous (IO) line is a reasonable alternative. Fluid or drugs administered by this route are rapidly taken up into the circulatory system.

Access Sites

The most common sites for access include the trochanteric fossa of the femur, the greater tubercle of the humerus, the anterior aspect of the proximal humerus and tibial crest.

Insertion

The necessary supplies are obtained (IO catheter / needle, lidocaine, scalpel blade, and syringe). Clip and aseptically prepare the insertion site. Inject lidocaine into the skin and periosteum. A stab incision may be made with the scalpel blade over the site of penetration. The bone marrow needle is placed on the site of penetration; pressure is applied to the needle while making firm 30◦ rotations. Apply steady pressure as the rotation maneuver is made and the needle passes through the cortex.

As an alternative, the EZ IO drill and specially made bone marrow needles (EZ IO, Mila International, Erlanger KY) can be used. The EZ IO drill is a battery powered drill made for the rapid insertion of bone marrow needles. Once the needle is firmly seated placement is confirmed. Once the placement of the needle is confirmed, attach the IV set, and begin the fluid infusion. Fluids may be administered with gravity flow or under pressure.

In human's osteomyelitis is the complication of greatest concern. The complication rate reported in one paper was approximately 0.6% in 4000 cases. Osteomyelitis is possible related to the use of hypertonic solutions, faulty technique or prolonged infusion.

1.1.5. OXYGEN THERAPY – CHASING THE BLUES AWAY

Harold Davis, BA, RVT, VTS (ECC)

OVERVIEW

Hypoxemia is defined as inadequate oxygenation of arterial blood (PaO_2 < 80 mm Hg or SPO_2 <~ 95%). Hypoxemia is a result of either low fractional inspired oxygen (FIO_2), hypoventilation, or venous admixture (ventilation/ perfusion

mismatch (most types of lung disease), right to left anatomic shunts, and diffusion defects). Oxygen therapy may be beneficial to those patients with impaired gas exchange. The goal of oxygen therapy is to provide adequate oxygen to the tissues, using the lowest possible inspired oxygen concentrations. In the absences of arterial blood gases or pulse oximetry one will need to rely on clinical signs. Clinical signs of hypoxemia include cyanosis, increased respiratory rate and effort, tachycardia, orthopnea, use of accessory respiratory muscles, and anxiety. Individually, the clinical signs do not prove hypoxemia, but together they are suggestive of hypoxemia and indicate the need for more definitive testing.

METHODS OF OXYGEN ADMINISTRATION

There are a variety of methods of oxygen delivery. The method selected depends on the expected duration of therapy, patient size, demeanor, and equipment availability. Available methods include flow-by, face mask, oxygen bag (hood), oxygen collar / tent, oxygen cage, transtracheal, nasal insufflation and high flow nasal canula.

Face Mask

Masks are readily available and easy to use. Masks are only good for short-term use. High-inspired oxygen concentrations can be obtained if a properly fitted facemask is used. Unfortunately, patients often fight the face mask (unless obtunded) thereby increasing oxygen consumption and canceling the effects of the oxygen therapy. The patients face and nose should fill the mask as much as possible to reduce the amount of dead space in the mask. Increased dead space will increase the work of breathing. Sometimes it is helpful to remove the rubber dam from the face mask to achieve a better fit.

Oxygen bag (hood)

An alternative to the face mask is the oxygen bag. A clear plastic bag is placed over the head of a patient and a hose from an oxygen source is placed near the animal's nose. The bag remains open along the animal's neck to allow the gas to escape. A flow rate of five to eight liters per minute is used. It has been reported that animals tolerate this bag/hood method when they resist the oxygen mask . Care should be taken to ensure that the bag does not collapse around the nose and mouth. It is very easy for animals (especially large dogs) to overheat with this technique and should be avoided if dogs become hot.

Oxygen Collar (tent)

Another alternative to the mask and hood is the oxygen tent. Take an Elizabethan collar (e-collar) placing plastic wrap over the front, leaving a one – two-inch (5.1 Cm) opening at the top. The opening allows expired carbon dioxide and heat to escape. An oxygen hose is inserted into the e-collar.

Oxygen Cage

A good oxygen cage should have the following features: it must have a system for eliminating carbon dioxide; deliver a known amount of oxygen in a concentration beneficial to the patient (40 - 50%); and a mechanism for controlling temperature (70ºF) and humidity (50%). The disadvantages to this system are, it's expensive to operate, the nursing staff have minimal access to the patient, and it is difficult to accommodate large patients.

Transtracheal

The placement of a "through-the-needle" catheter into the trachea can facilitate the administration of oxygen. The transtracheal route may be indicated when the nasal route is contraindicated. The procedure is carried out much like a transtracheal wash. An area over the trachea is clipped and prepared. Using aseptic technique, the catheter is inserted two - three tracheal rings below the cricoid cartilage or through the cricothyroid membrane. The tip of the catheter should lie in the region of the carina. A light bandage is placed around the patient's neck. The catheter is attached to a humidified oxygen source. Humidified oxygen is used to prevent irritation of the mucosal lining of the airway. Excessive oxygen flow rates are avoided, to prevent trauma from catheter “whip” to the tracheal wall. In one study it was determined that transtracheal oxygen administration permitted lower oxygen flow rates than nasal oxygen administration. The flow rates used for transtracheal O2 administration produced significantly higher inspired O2 concentrations and PaO2 than corresponding nasal O2 flow rates . The flow rates evaluated in this study ranged from 10 ml/kg/min - 250 ml/kg/min.

NASAL INSUFFLATION

Nasal insufflation is an excellent method for administering oxygen. The technique is inexpensive; minimal restraint is required, the nursing staff has good access to the patient, and most of the supplies needed may be found in most practices. A nasal oxygen catheter is easily placed and well tolerated by most patients. Nasal insufflation is contraindicated in patients with significant nasal masses, rhinitis, nasal fractures and nasal hemorrhage.

After treatment with a topical anesthetic and the premeasurement of the nasal catheter (3.5 – 10 Fr patient size dependent) from the tip of the nose to the medial canthus of the eye, the catheter is inserted. A lubricated catheter can be placed in the ventral nasal meatus (angling ventromedially) to the predetermined distance. Multiple catheter fenestrations minimize the risk of jet lesions of the mucous membranes. The catheter is sutured or stapled as it exits the alar notch and along side the face or brought up and over the forehead between the eyes and is secured again. The catheter is attached to a humidified oxygen source. A flow rate of 50 - 200 ml/kg/min should be effective in increasing tracheal O2 concentration in most patients to 40% or greater2, . Human nasal prongs are an alternative that can be effective and less invasive but may deliver a lower fraction of inspired oxygen than a nasal catheter. The ability to use nasal prongs will depend on patient anatomy and behavior.

Several complications may be observed: gastric distension, epistaxis, sneezing and serous nasal discharge. High flow rates can cause catheter "whip" trauma to the nasopharyngeal mucosa. To prevent mucosal drying humidify the oxygen through an in-line bubble humidifier.

High Flow Nasal Oxygen (HFNO)

HFNO has more recently been utilized in dogs. This method is advantageous because it can achieve flow rates up to 40-60 L/min and can more reliably deliver high FiO2. HFNO systems can deliver an FiO2 of 21-100%. The air is heated and humidified, then delivered to the patient through a heated breathing circuit and specialized nasal prongs sized to occlude about 50% of the nares. The system allows 100% humidification and control of temperature, thereby enhancing patient comfort and tolerance of these high flow rates

SUMMARY

A variety of options for oxygen therapy have been discussed. The lowest flow rate that improves the patient’s condition is the desired flow rate. The patient's

response to oxygen therapy should be evaluated at periodic intervals. The goal is to see an improvement in mucous membrane color, decreased anxiety, decreased breathing and or heart rate, decrease in the magnitude of respiratory distress, and an improvement in PaO2 or SPO2 to an acceptable level.

1.1.6. POST-OPERATIVE / ANESTHETIC COMPLICATIONS

Harold Davis, RVT, VTS (Emergency & Critical Care)

As veterinary personnel responsible for post-operative / anesthesia nursing care, we must be prepared to respond to rapid changes in the patient's condition. Knowledge of anesthetic protocols and operative procedures will help us to anticipate potential complications associated with them. We must be able to recognize life-threatening situations and respond to them immediately. Post-operative/ anesthetic or recovery nursing care requires knowledge in monitoring techniques, potential complications, therapeutic interventions, nursing considerations for specific types of surgical cases, and pain management. In addition, veterinary personnel should be skilled and knowledgeable in initiating cardiopulmonary resuscitation. The goal of this discussion is to provide the veterinary personnel with the tools necessary to function effectively in the recovery room / ICU.

ADMISSION TO THE RECOVERY ROOM / ICU

The post-operative / anesthetic recovery period begins at the cessation of the operative procedure(s) and / or anesthetic. The recovery period will continue until the patient's physiologic parameters have normalized. When the patient arrives in the recovery area recovery personnel must attend to the ABCs (airway, breathing and circulation), obtain a baseline physical examination, and communicate with the surgeon and anesthetist.

ABCs

The priority is to assess the ABCs, airway, breathing, and circulation. Typically, the patient arrives in the recovery area with an endotracheal tube in place. The endotracheal tube should not be kinked or crimped. A tube must be kept clear of secretions such as blood, mucous, and saliva. If the patient is breathing spontaneously, several questions are asked when assessing the patient's ability to breath. Is the rate and tidal volume adequate; is the breathing effort smooth

and easy or labored; is the breathing pattern regular? Are you able to auscultate normal breath sounds? Intubation is usually continued until the patient has regained its swallowing reflex and begins to cough or “buck” the tube. In brachycephalic breeds, extubation is delayed as long as possible and maintenance of airway patency is continuously observed during and immediately following extubation.

Perfusion parameters (Pulse rate, pulse quality, mucous membrane color, capillary refill time (CRT), mentation and extremity temperature) are all used to assess the circulatory status. Pulse rate is a non-specific parameter and may be increased (Hypovolemia, hypotension, hypoxemia, pain, fever, and drugs,) or decreased (Drugs, hyperkalemia, severe hypothermia, increased vagal tone, and heart block) for several reasons. Mucus membrane color and CRT are used to assess vasoconstriction and vasodilation. Vasoconstriction decreases peripheral perfusion and can easily be recognized by pale mucous membranes (when not due to anemia) and prolonged CRT. Many of the same reasons that cause tachycardia can also cause vasoconstriction. "Brick Red mucous membranes with a rapid CRT suggest vasodilation. Cyanotic (blue) membranes are a late indicator of hypoxemia. In the anesthetic recovery period mentation will be altered by the sedatives/anesthetics. Cool extremities are indicative of vasoconstriction which would be consistent with poor perfusion or significant hypothermia.

Physical Examination

Once the ABCs have been addressed a baseline physical examination (PE) should be performed. The initial PE establishes the baseline for further comparisons during the recovery period. In addition to physical examination the temperature is obtained, surgical wounds, dressings, tubes, and catheter sites are inspected.

1.1.7. ADVANCED MONITORING TECHNIQUES

The level of recovery care may be as simple as obtaining and recording a temperature, pulse, and respiration or it may be complex as invasive physiologic monitoring. Many of the advanced post-operative monitoring techniques are dictated by the intraoperative / anesthetic period, and the current condition of the patient. Some of these monitoring techniques include, electrocardiogram, blood pressure (non-invasive or direct), pulse oximetry, end-tidal CO2, and arterial pH and blood gases.

1.1.8. GENERAL POST-OPERATIVE / ANESTHETIC COMPLICATIONS AND TREATMENTS

Agitation / Rough Recovery

Upon recovery, some patients experience an excitatory phase, which is like the excitement phase of induction. These patients can often be observed paddling uncontrollably and vocalizing. The excitation is likely to occur in those patients that recover quickly from anesthetics that are eliminated rapidly from the CNS, such as volatile anesthetics. The goal is to restrain the patient to prevent self-induced trauma. An anxiolytic and or an analgesic drug can be given to smooth out the recovery.

Prolonged Anesthetic Recovery

Ideally the patient should be able to maintain sternal recumbency and lift its head within several minutes of extubation. In some instances, patients will take an unexpectedly long time to recover. There are two general causes for prolonged recovery: either patient or anesthetic related causes. Patient related causes may be due to poor perfusion, hepatic, renal or intracranial disease, and hypothermia. Anesthetic related causes include excessive anesthetic depth and breed predisposition. In the case of prolonged recovery steps can be taken as needed to speed up the patient's recovery. Fluids may be given to enhance perfusion; manual ventilation with 100% oxygen to enhance the elimination of anesthetic gases; or anesthetic drugs may be reversed. Caution should be used with regards to physical stimulation. It is possible to stimulate an animal to the point of extubation and once extubated it returns to sleep. If physical stimulation is used the veterinary personnel needs to be sure that the patient can protect its airway, remain sternal and lift its head.

Airway Obstruction

This is most likely to occur in the patient with an unprotected airway. However, a mechanical obstruction can occur in a patient with an endotracheal tube (kinked, mucous plug). There are a variety of causes for airway obstruction they included soft tissue entrapment such as in brachycephalics, laryngeal paralysis, edema, and spasm.

Extension of the neck and gentle withdrawal of the tongue often opens the airway; this position is maintained until the patient is recovered. A roll of tape can be placed in the mouth to keep the tongue pulled out and to maintain a patent airway. Oxygen is administered if the patient is in respiratory distress.

Aspiration

The initial problem caused by aspiration is airway obstruction with foreign material being inhaled into the airway. Aspiration can occur following vomiting or regurgitation of stomach or esophageal contents. Saliva, blood or mucous can also be aspirated. Patients at risk for aspiration are those that have esophageal (megaesophagus) or gastric fluid accumulation, increased abdominal pressure (pregnancy, ascites, abdominal effusion). When this problem is anticipated, the esophagus and stomach should be suctioned to prevent aspiration. Some drugs may predispose the patient to aspiration. These include volatile anesthetics, anticholinergics, and opioids.

If the animal is in lateral recumbency and begins to vomit, lower the head and neck, and hold the mouth open (take care not to get bitten). Once the vomiting has passed, assess the mucous membrane color, respiratory rate, and breath sounds. In the anesthetized intubated patient, the oral cavity may need to be lavaged and suctioned. If the patient is unconscious but not intubated the patient should be placed in a head down position and the oral cavity suctioned or swabbed.

Aspiration of material into the airway may lead to aspiration pneumonia although the signs of pneumonia may not be present immediately, depending on the severity of the aspiration.

Hypoxemia

Reasons for hypoxemia include low inspired oxygen concentration, hypoventilation, right to left shunts, ventilation perfusion mismatch, and diffusion impairment. The most common reasons that may be encountered in the recovery period include hypoventilation (drug related or airway obstruction), aspiration pneumonia leading to ventilation perfusion mismatch. Low ventilation perfusion mismatch occurs when there is blood flow past poorly functional or non-functional alveoli. Significant pulmonary collapse (atelectasis) may be present in the “down lung” after prolonged lateral recumbency. In most instances the removal of the

airway obstruction, administration of oxygen, positive pressure ventilation with or without positive end expiratory pressure may be used to treat hypoxemia.

Hypoventilation

Hypoventilation is a result of a reduced minute volume due to a reduction in tidal volume and/or respiratory rate. This will result in an increased arterial PCO2. Causes of hypoventilation include depression of the respiratory center by anesthetic agents, pain such that chest expansion is limited, pleural space disease (pneumothorax, pleural effusion), pulmonary disease (pulmonary edema, pneumonia), and restrictive chest bandages.

Treatment is dependent on the cause and may include reduction in anaesthetic depth, thoracentesis, placement of the patient in sternal recumbency to minimize the effects of atelectasis, loosening or removal of chest bandages. In some cases, intermittent positive pressure ventilation will need to be initiated.

Hypothermia

Anesthetic drugs can affect the normal thermoregulatory process. Heat loss through an open chest or abdomen contributes to hypothermia. Smaller patients have a greater surface to volume ratio and are more susceptible to hypothermia. Every effort should be made to return and or maintain a patient in a euthermic state. Warm water circulating or forced hot air blankets and drapes, or towels are effective tools for correcting hypothermia or maintaining euthermia.

Hyperthermia

Hyperthermia may be a result of rough recovery / excessive muscle activity, ketamine administration in dogs or myelography. Placing the patient on a cage floor, wetting with tepid water, or directing a fan at the patient are all options for correcting hyperthermia. An anxiolytic may be helpful in patients that are agitated. Because severe hyperthermia can result in increased oxygen consumption oxygen should be administered. Crystalloids help to improve circulating blood volume and cool the patient.

Hemorrhage

The surgical incision should be monitored during recovery. Excessive bleeding at the surgical site; increase in abdominal girth along with clinical signs suggestive of hypovolemia (pale mucous membranes, prolonged refill time, tachycardia, and poor pulse quality) could be indicative of internal bleeding. The causes for bleeding could be due to a slipped ligature, bleeding from small arteries that were not bleeding during closure or a coagulation disorder. Direct pressure should be applied. The clinician may elect to perform an ultrasound and or perform an abdominocentesis or thoracentesis. Therapy may consist of continued direct pressure, fluid resuscitation and/or surgical reexploration.

Hypotension

Anesthetic drugs can have direct negative effects on the cardiovascular system. However, the most common cause of hypotension is hypovolemia. Induction of general anesthesia can unmask pre-existing fluid deficits. Therapy is indicated if the patient is exhibiting poor perfusion parameters (tachycardia, poor pule quality, poor mm color, prolonged CRT, cool extremities, and decreased mentation) and / or when the systolic and mean blood pressure approach 80 and 60 mmHg respectively. Therapy is directed at correcting fluid deficits either through the administration of crystalloids (10 mL/kg and 20 mL/kg boluses in the cat and dog respectively). Synthetic colloids (10 - 20 mL/kg in dogs and 5 – 10 mL/kg in cats) if hypoproteinemic or if it is difficult to maintain intravascular volume with isotonic crystalloids alone. Consider the potential risk of bleeding with the use of synthetic colloids. 7.5 % hypertonic saline (4 - 6 mL/kg in dogs and 3 – 4 mL/kg in cats) can be given in addition to or as an alternative to isotonic crystalloids. Blood products are given to maintain a packed cell volume greater than 25% and / or the total protein greater than 3.5 g/dl (35 g/L). The patient's perfusion parameters should be reassessed frequently (every 10 – 15 minutes) during rapid fluid administration. In those situations where fluid support is not sufficient sympathomimetics such as dopamine or dobutamine should be considered.

Cardiac Arrhythmias

Perhaps the most common arrhythmias observed in the recovery area are ventricular in origin. Treating the underlying cause and in some instances oxygen supplementation and improved ventilation may help correct ventricular arrhythmias. If ventricular tachycardia is the problem, drug therapy may be indicated. Anti-arrhythmic therapy is indicated if the heart rate exceeds 160-180 bpm, the patient is cardiovascularly compromised, or the patient has multiform premature ventricular contractions (PVC's). Lidocaine is generally considered

the drug of choice for treating ventricular tachycardia. It is initially given as a bolus at a dose rate of 1 – 4 mg/kg over 1 – 3 minutes in the dog. If the patient is responsive to the bolus, it is then followed by a constant rate infusion of 40 – 80 micrograms/kg/minute IV. The bolus dose in a cat is 0.5 mg/kg slowly. cats are very sensitive to lidocaine and the drug should be used with great caution in this species. Response to therapy can be the total abolishment of the PVC's, a reduction in the number of PVC's, a slowing of the rate, or improvement in the overall cardiovascular status.

Cardiopulmonary Arrest (CPA)

CPA is defined as the sudden cessation of functional ventilation and effective circulation. CPA may be a result of any disease process, which disrupts cardiac and / or pulmonary homeostasis. Potential causes of cardiopulmonary arrest include hypoxia, shock, metabolic disorders, trauma, vagal stimulation, anesthetic or other drugs and environmental influences (hypo or hyperthermia).

The existence of cardiac arrest must be recognized early if we are to effectively resuscitate the patient. In the anesthetized patient a declining blood pressure will be one of the first signs you will see. Other signs include the absence of a palpable pulse or audible heart sound, the absence of breathing effort (agonal breaths should not be considered effective breaths) and fixed and dilated pupils. If there is any question that CPA has taken place the patient should be treated as such until proven otherwise.

The goal of cardiopulmonary resuscitation is to provide adequate ventilatory and circulatory support until spontaneous functions return. Once it is determined that CPA has taken place chest compression is begun at a rate of 80 - 120 compressions per minute. An airway is established, and the patient is ventilated once every six seconds. IV access is obtained either peripherally or centrally and, in some cases, intraosseous. Epinephrine (Adrenalin), vasopressin and atropine are used in the treatment of asystole and pulseless electrical activity. Defibrillation is indicated when the patient has ventricular fibrillation or pulseless ventricular tachycardia.

Summary

One prospective multi-center study determined that the postoperative period was the most common time for dogs and cats to die. Further, it showed that the most frequent time of death occurred within three hours of the termination of the

operative procedure . Cardiorespiratory was the most common cause of death in dogs and cats. This reinforces the idea that vigilance is important in the immediate postoperative/anesthesia period. While not ignoring the other body systems the cardiorespiratory system is of great importance.

The goal of postoperative / anesthesia care is to insure a safe and normal recovery of the patient. As discussed, there are several aspects of postoperative / anesthesia care that veterinary personnel must be prepared to manage. It is hoped that this discussion has provided you with the tools necessary to meet that goal.

Xavier Roura

2. Xavier Roura

2.1.1. ¿PODEMOS CURAR LA DIABETES MELLITUS EN GATOS?

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Puntos claves de la presentación:

La diabetes mellitus es una enfermedad que requiere para su tratamiento un esfuerzo conjunto del veterinario y el propietario. Es una enfermedad complicada ya que hay muchos factores que afectan la respuesta al tratamiento, con lo que muchas veces el manejo de la diabetes es complicado. Para tener éxito con esta enfermedad es necesario entender las evidencias científicas publicadas hasta ahora y tomar decisiones clínicas adecuadas para cada gato.

Los signos clínicos clásicos de la diabetes mellitus en gatos son poliuria, polidipsia y pérdida de peso.

La glucosuria ocurre cuando la concentración de glucosa en sangre excede la capacidad del túbulo proximal para reabsorber glucosa del filtrado glomerular (250-290 mg/dl).

El diagnóstico precoz se basa en la concentración de glucosa en ayuno en sangre de ≥180 mg/dl.

No se debe confiar en la fructosamina para diferenciar la hiperglucemia por estrés de la diabetes mellitus, pero valores de fructosamina superiores a 400 µmol/l son altamente sospechosos de diabetes en gatos con signos clínicos compatibles.

El diagnóstico de la diabetes mellitus en gatos debe basarse en las concentraciones seriadas de glucosa en sangre y, si están presentes, en la glucosuria, incremento de la fructosamina y signos clínicos consistentes.

Los principales objetivos del tratamiento son eliminar los signos clínicos y garantizar una buena calidad de vida tanto del gato como del propietario.

Una participación muy activa del propietario en el tratamiento del gato diabético es fundamental: considere el uso de tecnología moderna como algunas Apps, aunque siempre priorizar la facilidad y calidad de vida de propietarios y gatos.

Los clínicos veterinarios deben individualizar los protocolos de tratamiento y seguimiento para adaptarse a las necesidades de cada combinación de gato y propietario.

El tratamiento más eficaz para lograr un excelente control glucémico es la insulina, con varios tipos disponibles para su uso en gatos: insulina de acción intermedia como Caninsulin® (U40), insulina protamina de zinc como ProZinc® (U40) e insulina de acción más prolongada como glargina (Lantus® U100) o detemir (Levemir® U100)

La insulina (preferiblemente una preparación de acción más prolongada) y una dieta baja en carbohidratos y alta en proteínas son los pilares del tratamiento.

Con lo que en gatos que son diagnosticados de diabetes mellitus, utilizar una dieta baja en carbohidratos con insulina glargina 100 U/ml (Lantus®) y buscar un estrecho margen de glicemia (60-160 mg/dL), se asocia con un índice de remisión superior al 78% frente a 14% que se obtiene con el cambio de dosis de insulina en función de los signos clínicos.

Sin embargo, el objetivo es mantener una condición corporal ideal/adecuada y por tanto, valorar el uso de dietas con bajo contenido de hidratos de carbono (teniendo en cuenta la densidad calórica para no producir sobrepeso) y focalizarse, en gatos obesos, en la reducción de peso antes que en el contenido en carbohidratos.

En algunos gatos la insulina glargina 300 U/ml (Toujeo®) tiene mayor duración de acción con menor riesgo de hipoglicemia en comparación con la insulina glargina (U100) en personas.

La insulina glargina 300 U/ml se ha evaluado en gatos sanos, teniendo una acción mas constante y estable en duración y acción, y se ha demostrado que es segura y eficaz en gatos diabéticos.

Lograr un buen control glucémico en las primeras etapas del curso de la enfermedad aumentará las posibilidades de remisión.

Un control de la glucosa exhaustivo, reducción de los carbohidratos dietéticos y eliminación de las causas de insulina-resistencia (perder peso, detener el tratamiento con glucocorticoides y/o tratar las enfermedades concomitantes) cuanto antes posible, puede aumentar las posibilidades de remisión diabética en los gatos.

No obstante, la remisión es más probable en gatos con signos clínicos leves o estadios más iniciales de la diabetes, es decir, con hiperglicemia leve, sin hiperlipidemia y con una corta duración de la enfermedad.

2.1.2. Hipertiroidismo felino: ¡el gran olvidado!

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Puntos claves de la presentación:

La hipertensión arterial sistémica es la patología cardiovascular más importante en gatos adultos-geriátricos.

Esta hipertensión es más frecuente en gatos geriátricos porque está estrechamente relacionada con la enfermedad renal crónica y el hipertiroidismo.

El diagnóstico y apropiado tratamiento de la hipertensión es fundamental en la medicina preventiva de gatos geriátricos.

En gatos la hipertensión puede ser primaria o secundaria a enfermedad renal aguda y crónica, hipertiroidismo, miedo-estrés, hiperaldesteronismo y

acromegalia. Otras causas endocrinas mucho menos frecuentes son diabetes mellitus, hiperadrenocorticismo y feocromocitoma.

En dos estudios, uno en USA y el otro en UK, muestran que la hipertensión primaria es de las más frecuentes en gatos con un porcentaje del 17% (aunque podría llegar al 50% si incluimos a los que no se les dio un diagnóstico etiológico) en el estudio americano y del 20% en el de la Gran Bretaña.

La hipertensión daña seriamente una serie de órganos, influyendo en la calidad de vida del gato. Esto es debido a que cuando hay hipertensión se pierde el efecto protector del "sistema de autorregulación" que permite mantener la presión estable entre 60 y 160 mmHg en estos órganos.

Los principales "órganos diana" de la hipertensión son los ojos, el riñón, el corazón y el cerebro.

Efectos en los ojos: hipema, hemorragia de retina, desprendimiento de retina o ceguera. Debido que en fases iniciales de hipertensión pueden haber cambios más sutiles en la retina como exudados y hemorragias focales o tortuosidad de los vasos, su exploración es muy interesante cuando hay sospecha de hipertensión en el gato.

Efectos en el riñón: progresiva enfermedad renal crónica y proteinuria.

Efectos en el corazón: soplo sistólico, ritmo de galope, reducción de la tolerancia a los fluidos y hipertrofia del ventrículo izquierdo.

Efectos en el cerebro: letargia, cambios de comportamiento, desorientación, signos vestibulares, convulsiones, alteraciones de la conciencia, ataxia y paresia.

La patogénesis de la hipertensión en gatos es compleja, multifactorial y no del todo conocida. Sin embargo parece que podría ser una activación anormal del sistema renina-angiotensina-aldosterona.

El diagnóstico y el manejo de la hipertensión en gatos se basa el valoración o medición de la presión arterial.

La elección de un método u otro de medición de la presión arterial depende de la experiencia y preferencias del veterinario o auxiliar que finalmente la mesure.

Hay diversas técnicas que permiten la medición no invasiva e indirecta de la presión arterial como la doppler o la oscilometría. Lo ideal es que se utilicen aparatos que hayan sido validados en gatos en las mismas circunstancias en las que se utilizará en la clínica.

Igualmente, independientemente del aparato que se utilice, para obtener un resultado fiable en la medición de la presión arterial es muy importante seguir un protocolo estándar basado en el control del ambiente donde se realizará la medición, el estado anímico del gato, el tamaño del manguito, el número de mediciones a realizar o la distancia entre el corazón y la base del manguito.

Varios estudios describen los valores de la presión arterial sistémica para el gato en las diversas situaciones clínicas. Un gato con <140 mmHg es considerado normotenso; con 140-159 mmHg es considerado prehipertenso; con 160-179 mmHg es considerado hipertenso; y con 180 mmHg es considerado hipertenso grave.

Debido que en casi un 80% de los gatos la hipertensión es secundaria, el uso de fármacos antihipertensivos se debe realizar conjuntamente con el tratamiento de cualquier otra condición clínica asociada.

Es muy importante tener claro que para estos "órganos diana" es igual de grave una hipertensión que una hipotensión. Por lo que hay que tener muy claro que el diagnóstico es correcto y valorar el riesgo/beneficio, tanto para decidir el inicio o no del tratamiento como la dosis a utilizar.

Diversos estudios han demostrado que el uso de dietas reducidas en sodio no está asociado a una reducción del riesgo de hipertensión arterial sistémica en el gato.

Los gatos con hipertiroideos con hipertensión se deben tratar con metimazol, cirugía o yodo 131 más el antihipertensivo. En algunos de estos gatos se puede utilizar atenolol (6,25-12,5 mg/PO/día), para reducir la frecuencia cardíaca, aunque este fármaco no ha demostrado un efecto beneficioso sobre la presión arterial en gatos.

En gatos con hiperaldosteronismo e hipertensión en los que no se puede realizar la cirugía, el tratamiento crónico de elección es el uso de espironolactona (1,0-2,0 mg/kg/12-24 h), suplemento oral de potasio y un antihipertensivo.

La mejor opción para el tratamiento médico de la hipertensión en gatos es el amlodipino. Otra buena opción, especialmente en gatos con proteinuria, es el telmisartan. Un tercer fármaco a utilizar junto con los dos anteriores para interferir más sobre el sistema renina-angiotensina-aldosterona es el benazepril (0,5 mg/kg/12h).

Aunque el papel del sistema renina-angiotensina-aldosterona tanto sistémico como intrarenal parece importante en el desarrollo de la hipertensión en gatos, el amlodipino es a día de hoy la primera opción como antihipertensivo porque su eficacia ha sido valorada en muchas más situaciones clínicas que los otros antihipertensivos.

La dosis de amlodipino es de 0,625 a 1,25 mg/gato/día ó de 0,1 a 0,5 mg/kg/24h). En gatos con una presión arterial inferior a 200 mmHg la dosis de inicio más recomendada es la inferior. En gatos con hipertensión >200 mmHg tal vez es mejor iniciar el tratamiento con dosis superiores.

Telmisartan (1-3 mg/kg/24h) es la segunda opción como antihipertensivo porque su eficacia en gatos con hipertensión grave o con hipertensión y alteraciones en los "órganos diana" aún no ha sido demostrada.

Todos estos fármacos se deben usar con cautela en gatos deshidratados ya que sobretodo pueden reducir la funcionalidad renal de forma aguda.

Cuando hay una hipertensión grave o hay evidencias de daño en los "órganos diana" secundarias a la hipertensión, se necesita reducir la presión arterial de forma rápida (en horas) con antihipertensivos. En las otras situaciones clínicas con hipertensión el tiempo permitido para controlar la presión arterial aumenta hasta 3-4 semanas.

Los controles de la presión arterial para valorar la eficacia en los casos graves se deben hacer en 3-5 días del inicio de la medicación, para luego si hay buen control reducir la frecuencia a cada 15 días o más según criterio clínico del veterinario.

2.1.3. Hipertiroidismo felino: ¡el gran olvidado!

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Puntos claves de la presentación:

El hipertiroidismo se diagnostica en un 1-12% de los gatos de todo el mundo, y aproximadamente un 10% de éstos tiene una edad superior a los 10 años. La mayoría de gatos hipertiroideos tienen unos signos clínicos variables y al ser una enfermedad crónica progresiva los efectos clínicos pueden variar de leves a graves. Es importante recordar que la disminución de la frecuencia y gravedad de los signos clínicos asociados al hipertiroidismo es debida a la aplicación de tratamientos efectivos basada en un diagnóstico precoz de esta enfermedad.

Las glándulas tiroideas están constituidas por dos lóbulos separados a cada lado de la tráquea. En ocasiones, se puede apreciar la presencia de tejido tiroideo funcional ectópico, localizado en cualquier lugar entre la base de la lengua y el mediastino craneal. La producción de hormonas tiroideas depende entre otros factores, de la captación de yodo y la función de la enzima peroxidasa tiroidea, dando lugar en mayor medida a la hormona tiroxina (T4) y en menor medida a la tri-yodotironina (T3), que es la hormona más activa. La mayor parte de la producción de T3 ocurre en los tejidos a partir de T4.

El hipertiroidismo se produce en el 98% de los casos debido a una hiperplasia adenomatosa de la glándula o adenoma tiroideo benigno, mientras que en raras

ocasiones (2%) la causa es un carcinoma tiroideo. En la mayoría de los gatos (>70%), la patología es bilateral. Aunque la etiología es desconocida, se han identificado algunos factores de riesgo en varios estudios: edad, consumo de dietas enlatadas y un menor riesgo en gatos de raza Siamés e Himalaya.

Los signos clínicos varían en función de la gravedad de la enfermedad. Los signos clínicos más frecuentes son la pérdida de peso (88%), polifagia (49%), vómitos (44%), poliuria/polidipsia (36%), hiperactividad (31%), disminución del apetito (16%), diarrea (15%), apatía (12%), debilidad (12%), disnea (10%), jadeo (9%) e anorexia (7%). En la exploración física, los hallazgos más frecuentes son un aumento del tamaño de las glándulas tiroideas (83%), baja condición corporal (65%), soplo (54%), taquicardia (42%), ritmo de galope (15%), agresividad (10%) y mal aspecto del pelaje (9%).

El diagnóstico se basa en la sospecha clínica y confirmación mediante pruebas hormonales. Es frecuente encontrar un aumento de las enzimas hepáticas. La mayoría de los gatos hipertiroideos tienen una T4 total por encima de los valores de referencia. Sin embargo, gatos con enfermedades concomitantes pueden tener una supresión de las hormones tiroideas que lleven la T4 total dentro del rango de referencia, generalmente en el límite superior. Recientemente, se ha puesto interés en el uso de la TSH para detectar gatos hipertiroideos en fases preclínicas. Otros métodos de diagnóstico como la gammagrafía son más difíciles de interpretar para el diagnóstico, aunque son muy útiles para localizar tejido ectópico.

El tratamiento del hipertiroidismo ha avanzado en los últimos años. Actualmente, existen cuatro opciones diferentes de tratamiento, teniendo todas ellas sus ventajas e inconvenientes (ver tabla). El tratamiento más ampliamente usado es el metimazol (tiamazol) como son Felimazole® y Apelka®. Se trata de una medicación que inhibe de forma temporal la acción de la peroxidada tiroidea y, por lo tanto, la formación de hormonas tiroideas. Se recomienda empezar con una dosis inicial de 2,5 mg/gato cada 12 horas, y ajustar la dosis en función de los valores de T4. Generalmente se realizan controles a la 3, 6, 10 y 20 semanas, y posteriormente cada 3 meses si se mantiene bien controlado. En algunos gatos es posible reducir la dosis a 2,5 mg/gato cada 24 horas, y en otros es posible que necesiten dosis mayores para controlar la enfermedad. Una vez consigo el estado eutiroideo, es importante revalorar la función renal. En gatos con insuficiencia renal crónica leve, es posible que el hipertiroidismo enmascare la enfermedad debido a que aumenta la tasa de filtración glomerular.

	Ventajas	Inconvenientes
Metimazol (tiamazol)	- Poco invasivo - Efectivo - Pocas contraindicaciones - Evita hospitalización y cirugía	

- Reversible

- Útil en gatos con insuficiencia renal crónica concomitante - Dependiente del cumplimento del gato y del propietario

- Efectos transitorios

- Posibilidad de efectos adversos

- No útil en carcinomas

Medicina nuclear (I131) - Curativo con un único tratamiento en > 90% de los gatos

- Efectivo y seguro

- Efectos adversos mínimos

- Evita hospitalización y cirugía

- Útil en carcinoma (dosis mayores) - Coste

- Disponibilidad sólo en algunos centros de referencia (no disponible actualmente en España)

- Larga hospitalización

- Contraindicado en gatos “enfermos”

- Posible desarrollo de hipotiroidismo

Cirugía - Curativo y rápido

- Económico

- Necesidad de anestesia

- Necesidad de estabilización previa con tratamiento médico

- Posibilidad de hipocalcemia postoperatoria u otras complicaciones

- Irreversible

Tratamiento dietético con dieta restringida en yodo (Hill’s Y/D®) - Evita necesidad de medicación

- Evita hospitalización y cirugía - Dependiente del cumplimento estricto de la dieta

- Sólo el gato enfermo debe comer esta dieta

- No útil en carcinomas

2.1.4. El reto del manejo de la enfermedad renal crónica en perros y gatos

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Puntos claves de la presentación:

Todo tratamiento para un perro o un gato con enfermedad renal crónica (CKD) se debe de individualizar a cada paciente.

Las recomendaciones IRIS para el tratamiento de la CKD en perros y gatos necesitan primero de una clasificación previa de cada paciente mediante en un estadio IRIS de CKD.

Estas recomendaciones IRIS son un buen punto de partida para la mayoría de los perros y gatos con CKD, pero luego se han de ir adaptando en cada caso en función de la respuesta individual al tratamiento.

Hay dos tipos de tratamiento: a) el que enlentece la progresión de la CKD y protegen la función renal remanente; b) el que mejora la calidad de vida del paciente controlando los signos clínicos secundarios a la CKD.

En el primer grupo incluimos el tratamiento de la causa o etiología de la CKD, si se conoce, y los llamados nefroprotectores (dieta prescripción renal o bloqueantes del sistema renina-angiotensina-aldosterona). En el segundo grupo incluimos los tratamientos sintomáticos (fluidos, antieméticos, etc.)

En los estadios IRIS CKD 1, 2 y 3 inicial, hay muy pocos signos clínicos secundarios a la enfermedad renal (PU/PD, pérdida de peso, etc.) con lo que damos más importancia a enlentecer la progresión de la CKD.

En los estadios IRIS CKD 3 avanzado y 4, los signos clínicos secundarios a la CKD son mucho más frecuentes y por tanto, aunque hay que utilizar nefroprotectores si los tolera, son más importantes los tratamientos sintomáticos que mejoran la calidad de vida y aumentan la supervivencia del paciente.

Si es posible, se debe tratar o eliminar cualquier causa que pudiera explicar la presencia de una CKD en el animal, como por ejemplo la urolitiasis, las sustancias o los fármacos nefrotóxicos, la pielonefritis o la leishmaniosis. Esto se debe realizar junto con los tratamientos sintomáticos o antes de utilizar cualquier nefroprotector.

Todos los perros y gatos con CKD han de tener agua fresca siempre disponible, o también se puede aumentar el porcentaje de comida húmeda diaria para conseguir incrementar el consumo de agua.

Se debe alimentar con una dieta de prescripción renal a los perros y gatos en estadios IRIS CKD 1, 2, 3 y 4 antes de que desarrollen hiporexia o anorexia.

Sin embargo, los gatos en estadio IRIS CKD 1 que en general tienen un fósforo normal (<4,5 mg/dl) tal vez tienen un riesgo mayor de desarrollar hipercalcemia con una dieta de prescripción renal. Por tanto es mejor utilizar una dieta para gatos sénior o, si se utiliza una dieta de prescripción renal, monitorizar el calcio sérico para que no exceda los 12 mg/dl. Si esto ocurriera, cambiar la dieta de prescripción renal por una dieta para gatos sénior.

El control del fósforo, y consecuentemente de la PTH, es beneficioso para conseguir un enlentecimiento en la progresión de la CKD. Los objetivos son mantener el fósforo en >2,7 mg/dl pero en pacientes en estadio IRIS CKD 2 en <4,6 mg/dl; en □5 mg/dl en pacientes en estadio IRIS CKD 3; y en □6 mg/dl en pacientes IRIS CKD 4.

El tratamiento ideal para conseguir estos objetivos con el fósforo es el uso de la dieta de prescripción renal.

Si después de utilizar la dieta de prescripción renal durante 1-3 meses no obtenemos estos objetivos, es cuando podemos añadir un quelante entérico del fósforo como hidróxido de aluminio, carbonato de aluminio, carbonato cálcico, acetato cálcico o carbonato de lantano. Iniciarlo a una dosis de 60 mg/kg/día, dividiendo esta dosis diaria al mezclarlo en cada comida.

Para el control del fósforo y del incremento de la PTH no se recomienda en gatos el uso del calcitriol porque no se ha demostrado ningún beneficio con su uso, a diferencia de lo que ocurre en perros donde su uso juicioso podría aumentar la supervivencia en perros en estadios IRIS CKD 3 y 4.

Independientemente del estadio IRIS CKD que tenga el paciente, si hay hipertensión arterial sistémica (160-179 mmHg) persistente en el tiempo, se debe iniciar su tratamiento. Si existe hipertensión (160-179 mmHg) con evidencias de daño en órganos diana como el fondo de ojo, o existe hipertensión grave (>180 mmHg), hay que iniciar su tratamiento sin esperar a que este incremento de la presión sistólica persista en el tiempo.

El tratamiento de la hipertensión arterial sistémica en perros y gatos se realiza mediante el uso de bloqueantes de los canales de calcio como el amlodipino (de 0,125 a 0,5 mg/kg diariamente), con bloqueantes de los receptores de la angiotensina como el telmisartan (de 1 a 3 mg/kg diariamente) o con inhibidor de la enzima de conversión de la angiotensina como el benazepril o enalapril (0,5 mg/kg/12h).

Los perros y gatos con estadios IRIS CKD 1, 2, 3 o 4 y proteinuria (UP/C >0,5 en perros o UP/C>0,4 en gatos) o los gatos con borderline proteinuria (UP/C 0,2-0,4) deben ser tratados con una dieta de prescripción renal. Si después de 1 mes con esta dieta continua la proteinuria o es borderline proteinuria, hay que iniciar la terapia farmacológica.

El tratamiento farmacológico de la proteinuria en perros y gatos se basa en el uso de un inhibidor de la enzima de conversión de la angiotensina como el enalapril o benazepril (0,5 mg/kg/12h) o de un bloqueante del receptor de la angiotensina como el telmisartan (1-3 mg/kg/día).

Aunque no hay evidencias científicas publicadas, parece que los gatos con proteinuria e hipoalbuminemia persistente (<2 g/dl) tienen un riesgo parecido al de los perros en desarrollar tromboembolismos. En esta situación se recomienda la aspirina o clopidogrel tanto en perros como gatos.

El uso de amlodipino, benazepril o telmisartan está contraindicado si el perro o el gato está deshidratado o muestra signos clínicos de hipovolemia. Por eso, en pacientes en estadios IRIS CKD 3 avanzado o 4 hay que utilizarlos con cautela y siempre después de corregir la deshidratación o hipovolemia existente.

En gatos, y tal vez también en perros, en estadios IRIS CKD 3 avanzado y 4 puede ser interesante corregir la deshidratación mediante fluidoterapia SC con Ringer lactato a la dosis de 10-15 ml/kg/día para mejorar la calidad de vida y reducir el número de hospitalizaciones. En estos estadios IRIS CKD avanzados se puede plantear la colocación de un tubo percutáneo esofágico o gástrico para la administración tanto de fluidos como de comida.

Los perros y gatos en los estadios IRIS CKD 3 avanzado y 4 tienen frecuentemente acidosis metabólica (CO_2 total<16 mmol/l), que es una de las causas más probables de la hiporexia o anorexia en estos estadios.

El tratamiento de la acidosis metabólica es necesario en estos estadios mediante el uso oral de bicarbonato sódico o de citrato de potasio si además tiene hipocalemia para conseguir estabilizar el CO_2 total entre 16 y 24 mmol/l.

El vómito y la nausea son frecuentes en perros y gatos en los estadios IRIS CKD 3 avanzado y 4. Los tratamientos más adecuados son la mirtazapina (1,88 mg/gato cada 48h durante unas 3 semanas) o tanto en perros como en gatos, el uso de maropitant (1-2 mg/kg/día durante al menos 2 semanas).

La erosión o las úlceras gastrointestinales no son lesiones típicas en perros y gatos con CKD avanzada, así que si no aparecen nuevas evidencias científicas que lo respalden: ¡no hay que usar moduladores del pH gástrico!

La anemia secundaria a la CKD aparece generalmente en pacientes en estadios IRIS CKD 3 avanzado ó 4.

Es necesario tratar la anemia si se considera que está afectando la calidad de vida del gato, generalmente esto ocurre cuando el hematocrito es inferior al 20%.

Si es posible obtenerla, la eritropoyetina (100 U/kg SC 3 veces por semana hasta que el hematocrito sea ≥25%, luego 50-100 U/kg SC 1 ó 2 veces por semana según el valor del hematocrito) o la darbepoetina (1 µg/kg SC 1 vez por semana hasta que el hematocrito sea ≥25%, luego 0,5 µg/kg SC semanal ó 1 µg/kg SC cada 2-3 semanas según el valor del hematocrito) son los tratamientos recomendados para el control de la anemia, aunque con la

darbopoetina los efectos son más duraderos y se producen menos efectos secundarios.

La hemodiálisis, el trasplante renal o la aplicación de células madre tal vez podrán ser de utilidad en el tratamiento crónico de la CKD en perros o gatos. Sin embargo, son necesarias más evidencias científicas para su recomendación en la clínica veterinaria rutinaria, especialmente la aplicación de las células madre que aún está en un estadio muy preliminar de investigación y por tanto, ¡no se debe usar actualmente porque no están probadas ni su utilidad ni su eficacia como tratamiento de la CKD!

Elizabeth J. Colleran

3.1.1. THE ROLE OF STRESS IN FELINE ILLNESS: UNIQUENESS OF THE MESOPREDATOR

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SIGNIFICANCE

In a UK report on Animal Wellbeing in 2015, it reported that of the estimated 11.1 million owned cats 24% were living an indoor only lifestyle up from 15%. Over 4.5 million were living in multicat households, 50% of whom shared their litter tray and 58 % shared food and water bowls with other cats.

42% were fearful of loud noises, 37% of unfamiliar people and 60% of domestic noises like the vacuum cleaner. 63% of cat owners said their cat displayed one or more behaviors owner would want to change. The average number of cats was 1.68 and 43% of cats 4.7 million lived with at least one other cat. 2.3 million 21% were recognized by their owners as not getting along with a at least one other cat.

Hence, it should not be surprising to find that both multi-cat households and chronic stress are key issues in feline welfare.

Even the owners of highly sociable cats will describe the frequent tendency of their cat to actively desire periods of solitary independence. This level of freedom to remove themselves form human social environment can often leave owners with the impression that cats actively choose to encounter the stimuli with which they come in contact and hence that their lives should remain free form the distress associated with the negative emotions of anxiety/ fear or frustration.

IMPORTANCE OF MESOPREDATOR

In contrast to this assumption, the cats are highly susceptible to these emotional states.

The domestic cat is not hundreds of generations form their wildcat ancestors and consequently display an innate behavioral repertoire associated with being both a predator and prey.

- Limited tolerance of other cats co-habiting within its home range and even less capacity to cope with cats within its core resting area
- Limited requirement to develop a communication system for close encounters with other cats and hence a limited social signaling system.
- Limited capacity to cope with sudden or prolonged encounters with other cats or to diffuse social tensions
- Limited capacity for sharing access to resources or for coping when access to resources is denied or prevented through barriers or social competition.

- Limited competency with losing control over its environment and hence lacking in an ability to cope outside the area with which the cat is familiar
- Enhanced responses to sudden, small; movements and immediate predatory responses to potential prey.

Yet the cat also experiences a strong motivation to ensure that it remain safe form potential harm from predators or other threats requiring:

- Avoidance of non-prey animals by gaining height to enhance observation opportunities and conceal itself
- Avoidance of the source of sudden noises or significant movements
- Avoidance of novel stimuli that do not smell familiar

All these traits are essential to the cat's survival. When cats find themselves in situations where these criteria are in some way prevented from assisting with their survival, they immediately experience anxiety and as a result of this, distress.

In addition, by thwarting the cat's from meeting its needs frustration ensues. Anxiety is potentially the predominant negative emotion experienced by cats, as their primary motivation for survival drives behaviors of avoidance and escape that frequently prevent cats from familiarizing itself with the diverse range of stimuli surrounding the home environment.

As a result of the many novel or unfamiliar social and environmental stimuli smelled and heard each day, the difficulty of co-existing with co-habiting cats, the problems in exhibiting a full range of natural behaviors if remaining indoors and the problems of avoiding neighboring felines if wandering outdoors, the cat is frequently exposed to stressors. As each stress inducing incident creates neurochemistry that takes many hours to degrade, the frequent exposure to stressors creates an accumulation of neurochemicals that maintain the brains of many cats in a chronic state of distress.

The cat's ability to express distress is also affected by its dual role as predator and prey. It is east to forget that the majority of the world's cats live a feral lifestyle and that every year many domestic cats enter the stray population due to an inability to cope with their domestic situation. Hence cats must retain their ability to hunt efficiently which requires solitary hunting activity and sole control of access to an area capable of sustaining their hunting attempts many times a day. This has reduced the need of cats to develop a wide range of obvious close range communication signals. Cats simply have not evolved to be close to one another. In addition, as a prey species they need to avoid the attention of predators by remaining very still and quiet when in pain or very fearful. For these reasons, feline distress is often conveyed in a subtle enough way not to be noticed by owners.

Comparison to other species can be made in various ways, one of which is encephalization quotient (EQ) which is defined as the enlargement of the brain beyond that expected by the size of the body. Large bodies need large brains because they have larger muscles and more extensive somatic sensory systems. The brain of the domestic cat is very similar to that of other members of the genus Felis. Two of the most striking features of the feline brain are the enlargement of the cerebellum, coordinating balance and movement, and the large portion of the cortex devoted to controlling movement. The part of the cortex that deals with hearing is well-developed but the olfactory bulbs are compared to other carnivores, small. They have little room on their skull for large bulbs While the vast majority of species are closely adapted to their given niches, those with mostly instinctive behavior repertoire will adapt very slowly to changes. Those with extensive learning ability can alter their behavior

patterns more rapidly; they possess the capacity to solve problems and cope with unexpected change.

The domestic cat is able to move from total dependence on man to semi-independence and back, within a lifetime or at most a few generations. However, cats are not infinitely flexible, for there is ample evidence that their learning abilities are species specific.

CAREGIVER AWARENESS

In a study of 194 Italian cat owners, a questionnaire sought to evaluate whether owners recognized stress in their own cats. Most owners correctly identified the features of stress. When asked, however, the overall stress level of their cats 56.7% (110) chose low, 38.1 % (74) chose medium and 5.2 % (10) chose high, while 9.8% (19) did not think stress was a of any consequence in their cat. 4 components identified were: body posture, social avoidance, house soiling and self-directed behavior. Unrecognized were: scratching furniture, freezing, mydriasis, and recurrent cystitis. Only very prominent dramatic behaviors were recognized by more than 2/3 of owner: excessive vocalization, posture with ears back, urinating out of litterbox. These findings suggest that owners tend to overlook certain signs and to, thus, overlook situations of poor welfare.

Mild stressors defined as brief in duration and mild-to-moderate in magnitude can help develop coping skill. Mild stress responses are part of normal development when they occur in safe predictable environments of stable and supportive relationships. Examples of events resulting in positive stress responses in young animals include nonthreatening veterinary visits and exposure to novel environments and foods.

EFFECTS OF CHRONIC STRESS

In the most threatening circumstances, severe stress responses can result. Toxic stress responses – strong, frequent, prolonged – are the most dangerous to longterm health and welfare. Examples of events that can result in severe stress responses include chronic abuse, severe or chronic disease, early adverse life events like nutritional deprivation, maternal separation or significant maternal threat during pregnancy.

The sequence of events that has emerged from research proposes that when a pregnant female is exposed to a sufficiently severe stressor, the neuroendocrine products of the ensuing stress response cross the placenta and affect fetal development. The biological “purpose” of transmitting such environmental cues to the fetus may be to guide the development if the fetal CTRS and associated behaviors to increase the probability of survival. The fetus may “use” the information in utero to make adaptive response decisions. If a threatening, nutrient-limiting environment is perceived, the development trajectory of the fetus may change in response to the available information to enhance fitness in the predicted ex utero environment.

Although sensitization of the CTRS is more likely to occur during growth and maturation of the neural, endocrine and immune systems, it is not restricted to the developmental period. Moreover, sensitization of the CTRS may be unmasked by another adverse experience later in life, possibly associated with another round of epigenetic modulation of gene expression.

The stress system orchestrates body and brain responses to the environment, thus sensing danger and at the same time aiming to maintain homeostasis. This response ensures survival of organisms in situations in which they face exogenous and endogenous stressors. In conditions of acute aggression, the body elicits a “fight or flight” reaction by activating a set of neuroendocrine and inflammatory responses with certain similarities between infection, trauma and psychosocial stress. The defense reaction of the body involves not only the activation of hypothalamic-pituitary-adrenal axis, (HPA) but also proinflammatory transcription factors, cytokines, coagulation factors and vasoconstrictors. Thus the response to psychosocial stress bears a close relationship with inflammation.

Both major and minor stressful events can have direct adverse effects on a variety of inflammatory and immunological mechanisms that can have detrimental consequences to health. One molecular link between psychosocial stress and organ function is provided by the activation of proinflammatory transcription factor NF-kappa beta. Norepinephrine is released into nerve fibers in response to psychosocial stress to initiate a response to an immediate threat. There is ample evidence for the influence of CNS modulation through inflammatory cellular reactions to psychosocial stress. These might be of major influence not only for metabolic and vascular disease but also for autoimmune diseases for which stress has been reported as a risk factor.

Stress affects the length and quality of a cat’s life. Particularly affected in cats are the lower urinary tract, gastrointestinal system and immune system, In addition, stress may predispose cats to endocrine diseases and skin disease. The interrelationship between stress and health can become a “chicken or egg” debate. Although there is no doubt that stress can predispose cats to medical problems, it is inevitable that illness and discomfort will initiate or intensify stress. In addition, any condition resulting in a cat experiencing pain will also initiate or increase stress. This can become a serious welfare problem for the older cat whose owner may assume that their cat’s reduced activity is merely associated with old age, leading to a failure to alleviate either stress or physical discomfort.

CLINICAL EVIDENCE

The objective of this study was to quantify the effects of owner separation and physical examination location on fear anxiety and stress behavior indicators in cats. 21 healthy cats were enrolled. Owner separation coupled with physical examination location can result in clinically significant increases in perceived stress in cats and compromise vital sign assessment. Less than ½ of all cat owners in the US seek routine preventative veterinary care and wellness examination for their cats. Two common reasons cited for not taking their cats for routine care relate to anxiety and stress experienced by the cat during transfer there, or previous stressful experiences at a veterinary establishment. The “circle of behavior” illustrates how cats sense and respond to events in the environment. Starting at the top, when a cat acts (jumps up on the counter) the environment responds (a swat or shout to remove cat) The sensations arising in the cat’s nervous system enter the cat’s brain to form a perception of the response which is compared to events in the cat’s history (genetic, epigenetic, environmental) the context in which the response was received and its expectation of future events. These result in subsequent acts, depending upon the threat or reward potential of the response. The circle is completed on a time constant of milliseconds throughout the life of the cat.

Sickness Behaviors in response to unusual external events in healthy cats and cats with feline interstitial cystitis JAVM 2011

This study has 4 noteworthy findings. First no difference in mean number of SB was identified between healthy cats and cats with FIC under the enriched housing conditions implemented in the colony. This is despite the fact that all the cats had been donated to the colony as an alternative to euthanasia because of severe LUTS and previous efforts to enrich the home had failed. Second exposure to UEE significantly increased the risk for an increase in the total number of SB in both groups of cats, suggesting that SB were more closely associated with UEE than with disease status. Features that can activate stress responses leading to sickness behaviors in cats include intrinsic unpleasantness (cold, barren, loud) unfamiliarity (unknown caretakers) discrepancy from expectation (change in schedule, feeding) and decreased capacity for control (lack of hiding, perching, abrupt change in diet)

Third, the most common sickness behaviors associated with UEE included decreases in food intake and elimination behaviors and increases in defecation and urination outside the LB are quite common in cats housed in other environments. These are also frequently complaints that prompt an owner to bring the cat to see a veterinarian. This suggests that when a cat is presented for these clinical signs, the possibility that the signs resulted from external events as well as internal ones should be considered.

Fourth, an increase in age conferred a significant risk for an increase in SB and for an increase in UGI signs and avoidance behavior. “Wear and Tear” associated with chronic stress similar to that postulated in stress-related diseases in humans.

SUMMARY

1. The number of companion cats is quite large and growing. A species understanding of cats and their unique needs has not kept up with the growth of the population.
2. Dramatic evolutionary changes have not taken place in the feline species despite profound changes in the environment and circumstances in which they live
3. There is ample evidence across species that chronic stress has negative physical and psychological consequences
4. There is considerable evidence of the effects of chronic stress in cats, that stressors often go unrecognized by caregivers and that stressors are different for mesopredators as a result of their evolutionary adaptation to this role.
5. Scientific evidence of the characteristics of environments that cause chronic stress in cats are useful tools in the modification of human behavior

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3.1.2. UPDATE ON FIP: REASONS FOR OPTIMISM

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Feline Enteric Coronavirus. (Fecv)

Feline enteric coronavirus is an enveloped single strand RNA virus that is associated with asymptomatic, persistent and enteric infections. It is found worldwide and is ubiquitous in domestic and wild felines. There are 2 serotypes: 80% to 95% are serotype 1 (cat-like), 5% to 20% are serotype 2 (dog-like). Serotype 2 has a higher incidence in Asia than in North America and Europe. Clinically inapparent infection of kittens can start after weaning at 9 to 10 weeks of age. Virus shedding in feces, can persist for many weeks months or longer. Fecal-Oral transmission is facilitated by shared litter boxes. The virus displays tropism for the mature, apical epithelium of the lower intestine. Immunity develops slowly. Immunity is lost after shedding ceases, and recurrent infections are common.

Kittens can make IGM antibody and mount cell mediated immune responses from birth. IGG and IGA antibodies are absorbed from colostrum during the first few hours of life. Passive, systemic immunity results, from IGG and IGA from colostrum. Passive local immunity is derived from IGG and IGA antibiotics in the milk. Passive, systemic and local immunity protects kittens until their immune system matures. IGG and IGA production does not start until passive antibody is gone at 6 to 8 weeks of age. Kittens' immune systems do not reach adult levels until 12 to 16 weeks of age.

Kittens born to FECV exposed Queens, will have maternal immunity until 9 weeks of age. Primary infection will evoke both a systemic and a local antibody response. IGA goes from blood, transmitted across the intestinal epithelium into the mucus. There is no evidence of cell mediated immunity or any changes in lymphocyte populations. The IGA antibody response slowly leads to the cessation of virus shedding. Antibody titers wane as virus, shedding, ceases and cats, become susceptible to reinfection. Immunity to FECV infection is transient and recurrent infections are common.

Feline Infectious Peritonitis Virus. (FIP)

FIP arises from FECV, infection by mutation. FIPVs occur in regional lymphoid tissues of the lower intestine and 10% of FEC V, infected cats. Mutations result from positive selection pressures favoring replication in macrophages. FIPVs acquire tropism for peritoneal macrophages and lose tropism for intestinal epithelium. Tropism change results from mutations in Spike s and accessory 3C genes. FIP V mutation is unique to each cat. If FIPVS are strictly cell associated and local and systemic spread is in monoocytes and macrophages. FIPV is confined to infected macrophages which are not spread from cat to cat. There are rare cases of cat to cat spread (epidemic form) Serotype II FIPV.

FEC, be infections occur in virtually all cats and kittens. FECV to FIPV biotype conversion, occurs in eleven percent of cats. Only 1 in 10 to 1 in 30 cats with mutant viruses develop FIP. Worldwide feline mortality among all cats due to FIP is thought to be point 3 percent to 1.3 percent. FIP favors, multi-cat environments: Catteries, foster homes, rescue groups, shelters and cats living in dense, urban areas. if there is additional stress in these environments, the odds of FIP occurring increases. 95% of cases occurring cats, less than 7 years of age. 70% in cats less than 1.5 years of age. 50 percent in cats, less than 7 months of age. Pedigree Cats have 3 times greater incidence of FIP than random bred cats. Males are slightly more susceptible.

Pathophysiology

Macrophages are infected by immune complex, virus through their FC receptors. FIP is typical of other macrocphage infections, such as tuberculosis, leprosy , and deep mycosis. FIP is mediated by cytokine responses of infected macrophages. Th 1cell mediated cytokine responses are protective. Protective immunity is innate at onset and then becomes adaptive. Failure to establish protective immunity leads to th2 inflammatory response and disease. Affected cats have delayed apoptosis of infected macrophages, allowing for increased virus production. The incubation period from first replication in macrophages, to clinical disease is days to months. The actual disease course, varies from days to months and rarely a year or more. Once typical disease signs appear, historically, fewer than 5 percent of affected cats have survived to 1 year.

There are 2 major disease forms effusive and non-effusive. Wet and dry FIP Resemble the lepromatous and tuberculoid forms of human leprosy respectively. The 2 forms of FIPV are dependent on the relative balance of cellular and humoral immune responses. Wet FIPV is characterized by a predominance of immediate hypersensitivity reactions, vasculitis, And high levels of virus replication and macrophages. Dry FIP is characterized by a predominance of delayed hypersensitivity reactions and low levels of virus replication in macrophages. The disease form in a cat, may change from wet FIPV to dry FIP, or dry to wet during the course of the illness.

Diagnosis

In most cases a diagnosis of FIPV can be made by obtaining a complete history and performing a thorough exam of the patient and using the following diagnostic tools to build a diagnostic wall" brick by brick.

- High index of Suspicion and playing the odds.
- Signal meant age, breed origin Typically shelter / sanctuary or other dense multi-cat situations.
- Sudden loss of activity, lethargy, anorexia weight loss.
- Failure to thrive. Smaller than normal, poor hair, coat thin.
- Recent stressful event: Vaccination, surgery, new home, relinquished to a shelter or foster home, other illnesses.
- Physical exam and presence of signs associated with FIPV: Jaundice, ascites, pleural fluid uveitis or retinitis neurologic sign, palpable abdominal masses.

Basic nondefinitive tests.

Complete, blood count may show anemia of chronic disease Leukocytosis or lymphopenia. Serum proteins Often have a total protein high with albumin and globulin, an Albumin:globulin, ratio of less than 0.6 With the lowest albumin:globulin ratios tending to be in cats with wet FIPV, rather than dry. Bilirubin is elevated, in 21% to 63% of FIPV cases, often without marked elevation in hepatic enzyme activity. Feline coronavirus antibiotics Titer cannot determine if antibody titers are against FECV or FIPV. Abdominal and or thoracic effusions, maybe, yellow tinged, mucinous with partial clots., cloudy, containing non-degenerate neutrophils, monocytes, macrophages, or large foamy mcrophages and lymphocytes. Fluid protein may be 2 to 10 plus grams per deciliter. A Rivalta Test, should not replace a complete fluid analysis. but the test is inexpensive and easy to perform. Images for evidence of effusion and or organ and/or central lymph node, involvement include radiographs, abdominal ultrasound, MRI scans, with contrast and complete ophthalmic exam.

Definitive diagnosis occurs, when FECV / FIPV RNA is identified in effusion or diseased tissue. Histopathologic or microscopic appearance of lesions is not pathognomonic unless combined with the rest of the clinical picture. Immunohistochemistry positive for Coronavirus antigen in macrophages within effusions of diseased tissues. Polymerase chain reaction (rt-PCR) must have enough FIPV RNA in the effusion sample. FCOV 7b RNA PCR Is most sensitive. FIPV s mutation RNA is less sensitive. Positive 7B RNA test is diagnostic, even if the S mutation test is negative. PCR on blood is not highly sensitive. If you confirm the presence of FIPV virus antigen or RNA within peritoneal type macrocephages within typical effusions or lesions, you have made a definitive diagnosis. However, this is compounded by all the false negative test results that are made by laboratories using poor techniques, or that have been given non-representative samples.

Treatment

Current non-FDA-approved, antiviral drug status. FIPV is now considered a curable disease. Anecdotally, thousands of cats worldwide have been treated and possibly cured with antiviral compounds. Reversal of severe signs for both wet and dry forms of FIPV Can be seen within days of starting antiviral treatment. Recurrent known drug with the most significant Success for curing, FIPV is GS – 441524, Nucleoside analog produced by Gilead Sciences, which is under patent and not available for veterinary use. AniVive LifeSciences Incorporated is working to gain FDA approval of GC 376 as a treatment for FIPV.

Several companies in China, have developed what they suggest are similar or the same compound drug products and are b marketing these products worldwide as dietary supplements to treat FIPV. The compounds have not received, generally recommended as safe status (GRAS). Under us, FDA restrictions the use of non GRAS compounds in veterinary health care is illegal. Those treating cats with FIPV are acquiring a version of GS4 441524 Through online or non-Veterinary resources such as members of the public referred through Facebook groups. No assurances are available as to bioactivity ,safety, toxicity, and identity of the compounds being used in these non-FDA approved substances. The course of treatment recommended can be expensive. Prices vary among companies and whether cost is related to the quality of the product remains unknown. Suppliers of GS441524, should be chosen with care. No

centralized data is being maintained a reviewed under scientific supervision. Veterinarians cannot prescribed, or dispensed these non-approved compounds. they can though choose to provide supportive and monitoring care for cats undergoing treatment. Establishing a good veterinarian client patient. Relationship is in the best interest of the patient.

Complex Risk

Stressful conditions have a tremendous impact on virus shedding. There are multiple mutations that cause disease from reservoirs of FECV in crowded conditions. Every virus shed is an experiment in potential mutation and exposure of cats to that mutation. Reducing the environmental viral load is a critical component in prevention. Stress appears to trigger disease in some cats. However, tools for accurately analyzing stress triggers, may be somewhat lacking. Nutrition, lack of passive immunity for orphaned kittens, nd gastrointestinal dysbiosis are all worthy of investigating as possible stressors. Distinguishing, the role of stress from the role of comorbid conditions is not yet possible.

Strong Immune response and Virus elimination may occur for only a period of time. FECV does not induce durable immunity. Immunity wanes and cats become susceptible again. Passive, maternal antibodies are highly protective and may be useful in creating more durable immunity. Innate cellular immunity. is required in turn, requires growing, FECV serotype 1, in cell culture for vaccine development.

Eleven percent of cats, develop the FIP biotype mutations but only a fraction of those become sick. Identifying, the genetic component for this is crucial. A pedigree cats have increased susceptibility, it appears that all cats of a particular breed do not seem to be susceptible. Particular lineages, within a breed have high susceptibility. The reverse must then be true. There are lineages that have genetic resistance. There may be an interferon gamma gene that could be a marker for increased risk. Other markers may also exist given the complexity of this disease.

According to a recent study, certain Bentonite based cat litters can decrease viral shedding and environmental contamination. However, the clinical significance has not yet been demonstrated. Early, weaning of kittens is not desirable as passive immunity from colostrum And lactation can effectively protect kittens from coronavirus infection. A Corona virus Free environment is Not Practical as reinfection is nearly impossible to prevent.

In well-run shelters today FIPV is not a common problem. A well-functioning shelter should see less than 1% FIPV cases. Shelter should balance intake with healthy outcomes by reduced crowding, thus decreasing virus shedding and exposure to infectious disease. One study showed FECV shedding increased millions fold in some cats after a week in the shelter. Developing fostering programs. No bottle raised kittens or Queens with kittens, should not come into the shelter, but go straight into foster homes. This lessens the odds of exposure to all infectious diseases, including FIPV. Using double compartment and closures with 8.5 square feet of floor, space leads to a 50% decrease in the exposure to upper respiratory infections. Placing portal between compartments, decreases the incidence of respiratory disease by 90%. Group housing is not ideal for cats. If grouped place no more than 3 to 5 cats in each group. Keep the group, stable note in, and out movement of cats. Don’t mix long and short stay cats.

Our mission to end FIP is not finished. Research must continue in such areas as improving diagnostic methods, developing additional antiviral drugs and especially strategies to prevent FIPV infection in cats.

Summary

1. Feline Enteric Corona virus (FECV) and Feline Infectious Peritonitis virus (FIP) are very different viruses that cause much different forms of infection
2. FIP diagnosis is not as difficult as clinicians make it. Because until recently, it has been a fatal diagnosis, clinicians have tried to find other causes of signs that are more promising
3. FIPV is now considered a curable disease. Anecdotally, thousands of cats worldwide have been treated and possibly cured with antiviral compounds.
4. Veterinarians cannot prescribed, or dispense these non-approved compounds. they can though choose to provide supportive and monitoring care for cats undergoing treatment
5. Stressful conditions have a tremendous impact on virus shedding

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3.1.3. THE KIDNEY AND THE PARATHYROID

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The interrelationship between calcium, phosphorus, parathyroid hormone, activated vitamin D and fibroblast growth factor has a profound impact on the progression of chronic kidney disease (CKD) in dogs and cats. Beneficial effects of calcitriol treatment during CKD have traditionally been attributed to regulation of parathyroid hormone (PTH). New analysis of information emphasize direct renoprotective actions independent of PTH and calcium. It is now apparent that calcitriol exerts an important effect on renal tubular Vitamin D which may be important in maintaining adequate circulating Vitamin D. This in turn may be vital for important actions of Vitamin D on peripheral tissue.Limited information is available reporting the benefit of calcitriol treatment in dogs and cats with CKD. However, a survival benefit has been shown in dogs with CKD treated with calcitriol compared to placebo. The concentrations of circulating Vitamin D have recently been shown to be low in people and dogs with CKD and are related to survival in people. In 2015, there will be compelling data regarding the benefit of calcitriol use in cats with CKD.

Rather than focus on the dearth of evidence for several forms of intervention, this talk will focus on a historic review of the use of dietary therapy, phosphorus binding agents and calcitriol over a ten year period. These are all client-owner cats. Therefore, these are not randomized, blinded, controlled studies. Rather, these cases are a demonstration of practical interventions that have prolonged good quality of life in cats who may not have agreed to all of the recommendations made in the literature.

ASSESSMENT

In assessing renal disease in cats, the most sensitive indicator is the loss of urine concentrating ability. The use of an early morning urine sample to assess urine specific gravity (USG) may help to counter effects of diet or drugs on a tested sample. Using the International Renal Interest Society (IRIS) values for classification of renal disease can be helpful in planning therapy. In some classifications IRIS 2 is divided in to 2a (Cr. 1.6-2.4 mg/dl) and 2b (2.5-2.8 mg/dl). In our practices the classification of 2a with USG less than 1.030 eating a mostly dry diet formula, for example, are started on treatment for chronic progressive renal disease (CPRD). Early intervention prolongs quality of life, good body condition score and wellbeing in a number of key ways. We use ultrasound guided cystocentesis in every cat from whom urine is obtained. This allows a quick early morning visit by the owner, a sterile sample for culture if indicated, a full assessment of the appearance of the urinary bladder and observation of complications such as uroliths.

In a recent study, the predictive value of Symmetric Dimethylglargine was retrospectively examined. Cats with CKD under consideration were IRIS Stage 1 (sCr < 1.6 mg/dL) or Stage 2 (sCr 1.6–2.8 mg/dL) and nonproteinuric (UPC ratio ≤ 0.2) or borderline proteinuric (UPC ratio 0.2– 0.4) at the time of diagnosis. All healthy geriatric cats had serum SDMA and sCr

concentrations in the normal reference interval. Serum SDMA represents a promising biomarker for early detection of CKD in cats with compromised renal function. Early detection is desirable because it may be beneficial to initiate dietary or other interventions even earlier in order to slow progression of CKD, as was shown for cats with IRIS Stage 2 or 3 CKD.

Analysis of big data demonstrated that increased TT4 concentrations correlated with a decrease in CREA concentrations . This finding is consistent with previous observations that kidney dysfunction may be masked by the hypermetabolic state of hyperthyroidism and that reduced muscle mass results in reduced production of CREA. By contrast, SDMA concentrations did not significantly decrease with increasing concentrations of TT4, suggesting that SDMA is more resistant to the effect of reduced muscle mass and increased GFR associated with increased TT4 concentrations than is CREA.

DIET

One of the most frustrating aspects of treating this and any condition requiring lifelong therapy in cats is the difficulty clients have complying with our recommendations. Cats resist contact or intervention they haven't agreed to and clients want to preserve the relationship they have with their cat, often at the expense of appropriate therapy. It is essential then to choose the most effective forms of therapy, to provide options when resistance is experienced and to communicate a willingness to the client to assist in preserving the relationship they have with their beloved cat.

While it has been shown that dietary modification has the most positive long-term effect on outcome, the relationship between survival and protein restriction or the attendant restriction of phosphorus has yet to be illuminated fully. Strong evidence, however, supports dietary phosphorus restriction in animals with kidney disease. Serum phosphorus is an independent predictor of disease in cats with chronic kidney disease. Cats with induced renal disease fed phosphorus –restricted diets had less severe histological renal changes than cats fed normal diets.

Phosphate retention and hyperphosphatemia are primarily due to impaired renal phosphate excretion. If renal function is normal, clinically significant hyperphosphatemia seldom develops. In the early stages of CPRD increased levels of PTH can keep serum phosphorus within the reference range by decreasing expression of the sodium-phosphate transport system in the proximal tubule resulting in increased urine phosphate excretion. This allows for normalization of serum phosphorus at the expense of hyperparathyroidism.

As cats are quite specific about preferences in taste, texture and flavor, the use of renal formulated diets may not always be possible. Alternatives may not have been thoroughly tested to the extent that prescription diets are but the truth of the statement "It is more important THAT he eats than WHAT he eats" is undeniable. Treatment goals of dietary modification start with maintaining body weight and a normal body condition score. If renal diets are not tolerated, warm canned diets diluted with some form of flavored moisture are a good choice. Other alternatives include adding other forms of moisture to food to increase fluid intake, providing flavored waters to encourage moisture consumption, water fountains and multiple drinking places throughout the house.

IF a renal diet is not fed, most cats will tolerate low doses of aluminum hydroxide in food to act as a phosphorus binder, before serum phosphorus levels leave the normal range. Serum phosphorus should remain in the 4-5 mg/dl range, especially if calcitirol is considered. Low body condition scores and malnutrition are negative prognostic indicators in dogs and the same is likely to be true in cats. If adequate caloric intake and preservation of lean body mass does not occur, quality of life will decline.

Studies done to confirm preservation of lean body mass in cats fed a low protein diet, about 28% on an as-fed basis, were, as one would anticipate, time restricted to around 4 months. With the advent of a better plan for managing renal patients, they are living for years with stable renal values and hematocrits within the normal range. The effects of protein restriction on the body condition scores of cats with CPRD should be evaluated. Until then, we all have observed the protein cachexia of our renal patients. It is crucial to preserve adequate caloric intake and adequate protein for these patients.

The effects of uremia on appetite are well known, particularly in human renal patients. However, uremic gastritis has not been demonstrated in cats, making H2 blockers commonly used not indicated. Transdermal or aoral mirtazapine and oral or injectable maropitant seem to have some synergistic effect. Transdermal mirtazapine is well-tolerated by most cats.. Strategies for oral medication Administration should be included in client education including the use of “sticky” high value food like cheese in a can, cream cheese or pill pockets and other soft treats.

The U.S. Food and Drug Administration has approved Elura (capromorelin oral solution), the second drug approved for management of weight loss in cats and the first drug approved specifically for the management of weight loss in cats with chronic kidney disease. Cats with chronic kidney disease (CKD) may begin to lose weight prior to diagnosis and typically continue to lose weight as the disease progresses. This weight loss can worsen cats’ prognosis and shorten their lifespan. Capromorelin is a ghrelin receptor agonist known to increase appetite and weight gain and is approved as ENTYCE for appetite stimulation in dogs. Elura is the second product that the FDA has approved for the management of unintended weight loss in cats

VITAMIN D AND CALCITRIOL

Calcitriol has long been reported to provide benefits to the human uremic patient by lowering parathyroid hormone concentration. This has also been reported in dogs and cats. Oral calcitriol has been shown to increase survival in human patients with CPRD including those treated prior to dialysis. The antiproteinuria effects of Vitamin D analogs are of crucial significance because proteinuria is a major risk factor for the progressive decline of renal function in both dogs and cats. Podocytes are critically important in overall glomerular fuction and structure. Injury to podocytes commonly leads to proteinuria and glomerulosclerosis. A marker for podocyte injury, desmin, was lowered by calctriol in one model of CPRD in rats. Fibrosis as either glomerulosclerosis or tubulointerstitial fibrosis is a common sequelae in CPRD. Calcitriol in physiologic doses interfered with glomerular proliferation and growth, lessening glomerulosclerosis in a rat model. Calcitriol treatment of an experimental glomerulonephritis model in rats inhibited medangial cell proliferation, glomerulosclerosis and albuminuria.

The renin-angiotensin-aldosterone system (RAAS) is a major mediator of progressive renal injury in CPRD. The RAAS system is present entirely within the kidney and is present in most renal cells including tubular epithelia.

Calcitriol is a negative endocrine regulator of RAAS. Calcitriol suppresses renin biosynthesis and has a protective role against hyperglycemia-induced renal injury in diabetic human patients. Through its effect to inhibit RAAS, calcitriol decreases production of Angiotensin II and thus lessens these fibrogenic consequences as well as other harmful renal effects.

A glomerular mesangial or interstitial inflammatory reaction with marked involvement of macrophages and lymphocytes attends all forms of renal disease. Together with control of RAAS, the ability of calcitriol to control inflammation are hallmarks of renoprotective actions.

In our practices, early diagnosis of CPRD at the IRIS 1 or 2a level is the key to successful management. A cat with or without proteinuria, with or without hypertension with a USG less than 1.030 and normal Calcium and Phosphorus will be started on Calcitriol at a dose of 2.5-3.5 ng/Kg per day. This is compounded into a chicken or fish flavored oil base by a compounding pharmacy licensed to produce compounded pharmaceuticals for the human market. Calcium, Phosphorus and their product will be measured in 2 weeks.

While the literature is clear that iCA is a far more accurate measure of total body calcium, it is an expensive test. Our protocol calls for frequent testing of renal values including calcium and phosphorus. We would be treating a fraction of the cats we can help if this costly test were included. Instead we use a protocol advocated by Larry Nagode and Dennis Chew, Pathology and Urology professors respectively at the Ohio State University Veterinary College.

One of the benefits of the preservation of renal tissue using this protocol is the preservation of erythropoietin production and the consequent preservation of normal hematocrits. Cats with IRIS Stage 3-4 CPRD are still feeling better, more active and eating better with adequate circulating red cells. Anemia is a quality of life issue.

Hepcidin excess prevents iron absorption from the diet and blocks iron release from body stores by binding to and inducing the degradation of the iron export protein ferroportin. A mechanism for the EPO sparing effects of vitamin D is suggested by recent data demonstrating a hepcidin lowering effect of vitamin D. In vitro treatment with vitamin D of monocytes isolated from hemodialysis patients downregulated hepcidin transcription. Furthermore, oral administration of vitamin D in healthy volunteers lowered serum levels of hepcidin by 50% compared to baseline levels within 24 hr and persisted for 72 hr. Supplementation with vitamin D has also been reported to have beneficial effects on increasing erythropoiesis and decreasing inflammation. These initial results are promising, and a randomized controlled study is warranted to determine whether correction of vitamin D deficiency can ameliorate ACD.

In a recent study, telmisartan effectively decreased proteinuria and was safe for treatment of cats with CKD. For the primary variable, telmisartan was at least as effective as benazepril. Indeed, telmisartan's antiproteinuric effect appeared to be more pronounced because significant

decreases in UP/C compared to baseline were identified for telmisartan but not benazepril. The results of this study indicated that telmisartan was a safe and effective treatment to decrease proteinuria associated with CKD in cats.

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3.1.4. MANAGING THE COMPLICATED SENIOR CAT

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Old age is not a disease. Both veterinarians and owners must resist the temptation to ascribe signs of illness to aging. Some signs of illness such as chronic pain, dehydration or hypokalemia may lead to clinical signs that owners scribe to “slowing down” with old age. Many problems of senior cats are chronic and progressive so that early diagnosis and treatment is important for pain management and quality of life. It can also be tempting to find a “diagnosis” and treat for that without continually evaluating the “whole” cat. The focus on a single diagnosis and treatment plan can neglect common comorbid conditions that can dramatically affect quality of life. A hyperthyroid cat, for example, may suffer from other conditions more common in older cats; overgrown nails, decreased olfactory sensing, which can impact appetite, muscle atrophy and osteoarthritis or periodontal disease.

For these reasons and a host of others, comprehensive wellness examinations, history assessment and a minimum database are recommended every 6 months for seniors. Health status may change rapidly in this group and early detection and treatment is important to preserve quality of life. Signs of illness in cats are often quite subtle and easy for owners to overlook. The minimum database includes a compete blood count (CBC), a full serum chemistry panel with electrolytes, a full urinalysis and total T4. Early detection of a decline in renal function will be found in declining urine specific gravity before BUN and Creatinine are beyond the normal range, making the urinalysis a critical part of information gathering. Depending upon risk factors, fecal examination and retrovirus testing may also be indicated. Blood pressure measurement should also be included in any cat with known risk factors.

Because senior cats’ response to vaccination is largely unknown and immune function may be affected by both aging and the presence of chronic disease, vaccinations should be given according to the AAFP Feline Vaccine Advisory Panel for all cats.

The home environment is critical to wellness. Staff members should be trained to educate owners about enrichment, stress identification and modification for aging changes. Senior cats may benefit by an additional heat source such as a heated bed or one placed close to a heat source. Resting or hiding areas that are inaccessible to other pets, quiet and easily accessible for the senior may help with stress reduction. Litterboxes should be large and shallow with low sides and placed in quiet locations. If the home has multiple stories, boxes should be placed on each. Night-lights may be helpful with declining vision. Multiple fresh water sources can encourage moisture consumption in cats that may be prone to dehydration because of reduced urine concentrating ability.

In senior cats, cognitive dysfunction (CD) is now recognized as an important problem. Formal diagnostic criteria have yet to be established. It is a diagnosis of exclusion. The most common signs are disorientation in time and space, altered learning, house soiling, altered interactions (e.g. attention seeking, anxiety, irritability) changes in

activity (wandering or pacing) changes in sleep patterns, decreased appetite, decreased grooming and increased vocalization. Medical problems such as hyperthyroidism, hypertension, pain of osteoarthritis, or chronic kidney disease can mimic many of these signs and so must be excluded before presuming CD.

Published studies are lacking on the efficacy of treatment. Therapies extrapolated from studies in humans and dogs include anti-oxidant enriched diets, supplements phosphatidyserine, omega-3 fatty acids, Vitamins E and C, L-carnitine. One ingredient found in supplements for dogs, alpha-lipoic acid, is toxic in cats. SAMe improved activity and awareness in dogs and is commonly used in cats with hepatic disease. No trials with these supplements have been published for cats with CD. Therapies for cats with CD are anecdotal.

There is evidence of cholinergic decline in senior cats so drugs with anticholinergic activity (e.g. some SSRIs such as paroxetine and TCAs) should be avoided. Selegiline (Anipryl), which has been anecdotally reported to be useful in cats and proven beneficial in dogs for CD, should not be combined with SSRIs or TCAs. Environmental enrichment and Feliway have often been recommended but no studies in cats show benefit. In fact, in cats with CD modifications to the environment may be detrimental. Regular and predictable routines are most desirable. Any changes should take place slowly.

HYPERTHYROIDISM AND CHRONIC KIDNEY DISEASE (CKD)

Recent literature suggests that treatment of FHT while avoiding hypothyroidism is desirable in cats with renal insufficiency . The new Hyperthyroid guidelines recommends treatment of hyperthyroid patients regardless of concurrent disease. This includes cats with pre-existing CKD and those that develop azotemia after initiation of FHT treatment. These patients will require careful monitoring in order to achieve and maintain a euthyroid state while at the same time preventing hypothyroidism or mild hyperthyroidism.

Treatment recommendations differ depending on the degree of underlying renal disease. Therefore, it is important to fully determine the renal status of the patient prior to initiating FHT treatment. The new guidelines recommend using the staging guidelines set out by the International Renal Interest Society (IRIS), including determination of blood pressure and urine protein quantification. Note that cachexia will affect the serum urea nitrogen level (elevated due to increased protein turnover) and creatinine level (decreased due to loss of muscle mass) Recording a body condition score and muscle condition score at each physical exam will help to document progressive changes. Cats that are identified as hyperthyroid with concurrent renal azotemia fall into the should be monitored accordingly. Comorbidity of azotemia with FHT is common.

The guidelines recommends treating FHT in cats with pre-existing CKD. Treat both diseases concurrently. Manage IRIS stage 1 and 2 cases as though they are non-azotemic. If the patient responds favorably and renal function is stable using a reversible treatment, then consider an irreversible FHT treatment. IRIS stage 3 and 4

patients warrant a more prudent approach for example, using lower doses of methimazole and more aggressive management of CKD. If a permanent treatment for FHT is pursued, careful monitoring and aggressive kidney support may be required during the period of regeneration of previously suppressed normal thyroid tissue.

Typically the thyroxine nadir occurs 2 weeks after radioiodine treatment, with T4 normalization occurring around 4 weeks after treatment. Supplementation with during this period will resolve iatrogenic hypothyroidism and may be necessary in clinically hypothyroid patients. However, this treatment will also suppress pituitary TSH, which is needed to stimulate regeneration of atrophied thyroid tissue. In such cases, it is imperative to establish euthyroidism in order to avoid renal hypertension and further glomerular damage, while at the same time avoiding iatrogenic hypothyroidism. Just as in those cats that develop azotemia after treatment of FHT, the evaluation of serial concomitant creatinine, T4 and TSH tests may help to determine whether T4 supplementation is necessary. The Panel generally recommends testing post-surgical and post- radioiodine patients at 30, 60, 90 and 180 days after treatment.

HEART FAILURE AND CHRONIC KIDNEY DISEASE (CARDIORENAL SYNDROME OR CRS)

In the cat, the incidence of chronic abnormalities in cardiac function (e.g. congestive heart failure) causing progressive and permanent chronic kidney disease is unknown. A study of 102 cats with hypertrophic cardiomyopathy reported 59% prevalence for azotemia as compared to 20% for age-matched controls.

CRS occurs when worsening renal function limits diuresis despite clinical volume overload associated with heart failure. In cats being treated for chronic heart failure, declining renal function should be anticipated. The diagnostic marker for CKD, isosthenuria, cannot be relied upon in cats being treated with diuretics. Monitoring of Creatinine especially should be used to discern trends in renal function. A progressive rise even within the normal range should alert the practitioner, along with clinical signs: PU/PD, hyporexia, anorexia, weight loss and vomiting.

A minimum database should include abdominal ultrasound to assess for typical changes in renal architecture and to identify underlying causes that may have specific treatments, such as neoplasia, pyelonephritis, and nephrolithiasis. Blood pressure monitoring should be included as well as hypotension from therapy can decrease renal perfusion. The usual diagnostic imaging; echocardiogram, thoracic radiographs are important for type of cardiac disease, risk assessment, and treatment planning.

Goals of treatment are to recognize CRS, reverse it as much as possible and deal with the renal consequences of heart failure and the complex relationship between heart failure and renal injury. The difficult balance is to “dry out” the heart failure and hydrate the kidneys. Different therapeutic strategies are based upon the degree of compromise of each organ.

Ace inhibitors are the mainstay of therapy for CRS especially in the presence of hypertension or proteinuria. Cats with CRS should be hydrated before starting therapy. Low dose benazepril or enalapril 0.25mg/kg Q 24 hours can be increased to proved better control for heart failure. Benazepril is metabolized in the liver, Enalapril in the

kidneys. Therefore, cats with CRS may need a lower dose of enalapril than benazepril. Initiation of therapy may show a transient increase in BUN/Creatinine concentrations. If persistent, lowering the dose is usually sufficient.

If azotemia is becoming a concern, the first step is to lower the dose of diuretics. The goal is to find the lowest effective dose that controls heart failure. The dose must be continuously reassessed. The ideal dose for an individual patient achieves the threshold rate of drug excretion. An individual HF patient that is not responsive to 5mg of furosemide per 24 hour for example will need 10 mg per 24 hours, not 5mg every 12 hours. Adequate natriuresis can be grossly assessed by observation of increased urine volume and decreased specific gravity. Periodic drainage of pleural fluid or ascites can be used to avoid excessive diuretic use.

In the event that diuretic resistance occurs, several options are available to correct fluid balance. A CRI of furosemide (0.3-0.6mg/kg/hour IV inhibits sodium resorbtion more effectively than oral or IV Boluses. Once the volume overload has resolved, most cats will again respond to oral therapy. Another loop diuretic, torsemide has superior diuretic action and long half-life. (0.3mg/kg PO Q 24 hours) It appears to be 10 times more potent than furosemide. Dual-diuretic therapy can be considered when furosemide dose needs to be decrease. Spironolactone (1-2 mg/kg Q 12 hours) may cause severe facial pruritus and must be used with caution. Aldosterone sometimes causes significant hyperkalemia. Each work at different sites within the nephron and if tolerated may be helpful.

Systemic hypertension is common in CKD and by increasing afterload increases the cardiac workload. Hypertension worsens both CKD and heart failure. If present, amlodipine (0.625 mg/cat PO Q 24 hours) should be added. Blood pressure monitoring is critical to avoid the effects of iatrogenic hypotension.

In advanced CRS, a positive inotrope (pimobendan) may improve azotemia, demeanor and appetite and allow reduction in diuretic dose.

Dietary modification should consider both conditions. Sodium restriction is sometimes needed and the extent to which it is required will vary. Distilled or low sodium water may be offered for drinking if more sodium restriction is needed than can be provide with diet. Clients should be cautioned not to feed high sodium treats. Lower phosphorus diets may be helpful in managing kidney disease but may result in the loss of lean body condition. High quality protein should be given to the level that it does not worsen azotemia. Omega -3 polyunsaturated fatty acids have been shown to be beneficial in both cardiac and renal conditions. Many renal diets are supplemented or if given separately EPA 40mg/kg/day, DHA 25mg/kg/day.

Fluid administration is a balance between improving renal blood flow without precipitating congestive heart failure. Fluids should be given slowly to correct azotemia, tailored to the individual's ability to tolerate. Abrupt changes in weight, a new gallop heart sound and/or heart rate may indicate impending congestive event and justify fluid rate reduction. Sometimes a low-dose CRI of furosemide will be indicated concurrently in cats with end-stage CRS. SQ fluids may be less likely to trigger a congestive event and can be given every 24 -48 hours via a balanced electrolyte solution and adjusted to the individual patient's ability to tolerate. In fragile patients, a smaller volume of fluids, as little as 30mls every 48 hours may be necessary, titrating slowly upward if the expected effect on uremia is not evident. Electrolytes should be monitored closely,

especially potassium, as hypokalemia can trigger arrhythmia. Correction can take place through fluid therapy or oral means.

Although renal function may remain stable for a period of time in cats with heart failure, when CRS occurs it leads to frequent hospitalization, difficulty maintaining good quality of life and eventually euthanasia. The therapy described here is directed at improving quality of life for cats with CRS. Whether they contribute to prolonged survival is unknown.

SARCOPENIA AND CACHEXIA

In both people and companion animals, cachexia and sarcopenia are 2 important syndromes that occur in a variety of chronic diseases and aging, respectively. Although cachexia has been recognized in people for over 2,000 years, only recently has it become acknowledged as a common and detrimental finding that is associated with increased morbidity and mortality, and with this observation has come rapidly expanding interest and research. Both of these syndromes are becoming increasingly important in human and veterinary medicine because of their high prevalence and adverse clinical effects, and a better understanding of the mechanisms underlying these syndromes is critical for optimal patient care, whether human or veterinary.

Cachexia is defined as loss of weight and muscle mass secondary to chronic inflammation or disease. Sarcopenia, “poverty of flesh”, is an age-related loss of lean body mass. Sarcopenia is not caused by disease, is a gradual process and progresses with age. Loss of muscle can occur without fat loss or a decrease in Body Condition Score (BCS). Individual cats, particularly those with long coats or a history of obesity may appear to have a high BCS and yet be under muscled.

One of the keys to the management of cachexia and sarcopenia in cats is recognizing it in its earliest stages. To achieve this, BCS and Muscle Condition Score (MCS) must be consistently assessed. The goal for BCS in a healthy cat is 4–5 on a 9-point BCS scale. However, in certain diseases (eg, CHF, CKD), a slightly higher BCS may be desirable (ie, a BCS of 6–7/9), although further research is required to make specific recommendations. Even in animals with these diseases, obesity (BCS > 7/9) should be avoided.

The MCS differs from the BCS in that it specifically evaluates muscle mass. Evaluation of muscle mass includes visual examination and palpation of the head, scapulae, epaxial muscles over the thoracic and lumbar vertebrae, and pelvic bones.

In people, the loss of LBM has direct and deleterious effects on strength, immune function, wound healing, and survival. In fact, cachexia is an independent predictor of survival in people. The specific deleterious effects of muscle loss have not been as well studied in cats although there are studies associating thin body condition with decreased survival.

The weight loss that occurs in cachexia is unlike that seen in a healthy animal that loses weight. In a healthy animal that is receiving insufficient calories to meet requirements, metabolic adaptations allow fat to be used as the primary fuel source, thus preserving LBM. Conversely, acute and chronic diseases alter concentrations of a variety of mediators (eg, inflammatory cytokines, catecholamines, cortisol, insulin, glucagon), which then decrease the ability to make metabolic adaptations required to

switch to fat utilization, and amino acids continue to be used as a primary source of energy. Therefore, muscle and LBM quickly are catabolized.

Numerous other factors can contribute to muscle and weight loss. Maintenance energy requirements vary with age, genetics, health status and gender (intact or altered). In presence of some disease states, maintenance energy requirements increase significantly. Decreased nutrient absorption is another possible mechanism for muscle loss in cachexia and sarcopenia. Studies in cats have shown decreased digestive ability. One investigator showed a reduced ability to digest protein in 20% of geriatric cats with about 33% having a significant reduction in ability to digest dietary fat. Micronutrient absorption, potassium, phosphorus, sodium, choline, B vitamins and Vitamin E, is also decreased.

Cats derive most of their energy requirements from protein and are metabolically less able to handle decreased amounts of protein and increased amounts of carbohydrates to maintain their energy requirements. Omnivores adapt to lower dietary protein by down regulation of their protein metabolism (protein sparing) but cats have been proven to be unable to make this physiologic adaptation. This preferential use of protein for energy can have clinical effects when cats are ill or anorectic as protein malnourishment can occur.

An important problem in cardiac and other forms of cachexia is a decreased calorie intake. The anorexia may be secondary to fatigue, dyspnea, or may be because of medication toxicity or alterations in appetite that often accompany CHF, cancer, and CKD in cats. Absolute food intake may decrease in animals with these diseases, but there also may be altered food preferences, cyclical appetite, and other issues that negatively affect overall food intake. Anorexia, for example, is present in 34–84% of dogs and cats with heart disease.

Increased energy requirements, alterations in nutrient absorption, and decreased energy intake all likely play important roles in the pathogenesis of cachexia by causing a net calorie deficit. However, a healthy animal that has a calorie deficit, either as a consequence of decreased food intake or increased energy requirements, would primarily lose fat. Therefore, these factors are not sufficient to explain the muscle and LBM loss and relative sparing of fat that are the hallmarks of cachexia and sarcopenia. This discrepancy suggests that metabolic alterations also are present.

Because of the important implications of cachexia and sarcopenia on morbidity and mortality in people, there is now extensive research into the prevention, diagnosis, and treatment of these syndromes. There are exciting opportunities for new and effective targets to decrease energy requirements, enhance energy intake, improve nutrient absorption, and modify metabolic alterations to prevent and even reverse the effects of both cachexia and sarcopenia.

A 2008 study on longevity in aging cats studied in a controlled environment for 5 years showed that all cats lost weight over time. However, cats supplemented with dietary antioxidants, prebiotic chicory root and a blend of Omega 3 and 6 fatty acids had a beneficial effect over a commercially fed diet alone or one supplemented only with antioxidants (Vitamin E and beta carotene). Cats in the fully supplemented group lost less weight, lived longer, had better LBM scores, improved fecal flora and fewer diseases.

In many cases, practical methods to help owners manage their animal's appetite are critical to success. This is particularly important because anorexia is one of the most common contributing causes to an owner's decision to euthanize his or her pet.

Any issues that potentially can affect food intake should be addressed, whether physical or environmental. Dental disease, for example, can substantially impair food intake in an otherwise healthy or sick animal. Pain (eg, back or joint) can decrease an animal's mobility and make it more difficult to secure adequate food intake. Environmental issues also can negatively impact food intake. Multipet households may impede the ability of an individual animal to gain access to food (eg, a more frail or timid animal may be crowded out from the food bowl). Stress often can increase for animals after diagnosis of any illness because of lifestyle changes (eg, medication administration, new foods), as well as increased stress on the part of the owner, which may be detected by the animal.

Once environmental issues are ruled out as a cause of weight loss, a nutritional screening is crucial. Older cats may need 5-6 g of protein/kg to prevent protein catabolism. Reduced digestive ability indicate that a high energy, highly digestible diet may be needed. Some kitten formulas may be more appropriate. Folate and cobalamin supplementation may be useful. Commercial cat foods vary quite widely in caloric density. Specific formulas should be investigated for adequacy.

Cachexia should be anticipated in animals with chronic diseases such as CHF, CKD, cancer, and others. Consistently evaluating MCS in all patients will help identify muscle loss at an early, mild stage in aging or ill animals, rather than waiting until muscle loss is moderate or severe, when it may be more difficult to successfully manage. Similarly, as animals age, muscle loss is likely to occur, even in healthy individuals. Therefore, muscle mass should be thoroughly evaluated in geriatric cats and dogs.

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3.1.5. CATS DON'T LIMP: CHRONIC PAIN IN THE SENIOR CAT

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Pathophysiology

Osteoarthritis (OA) is a common and complex progressive disease. Clinically it is defined as a slowly evolving articular disease characterized by the gradual development of joint pain, stiffness, and the limitation of range of motion. Pathologically at has been described as a disorder of synovial joints characterized by deterioration of articular cartilage and by the formation of new bone at the joint surfaces and margins. The median age of affected cats in another study was 10.2 years and increasing age was clearly a risk factor for the development of osteoarthritis and other degenerative arthropathies.

In cats older than 12 years of age one study found a 90% prevalence of all types of degenerative joint disease. (DJD) DJD is not the same as OA though the terms are commonly used interchangeably. OA is a subset of DJD which includes all forms of degenerative pathology of skeletal joints.

The hip and elbow joints are most commonly affected and bilateral disease was invariably a feature. In a group of 100 randomly selected cats aged up to 20 years old almost all of the cats had radiographic evidence of degenerative joint disease (DJD). Affected joints in descending order of frequency were hip, stifle, tarsus, and elbow.

OA associated pain starts at the peripheral join and results in decreased ability to perform daily activities and decreased mobility. This initiates musculoskeletal deterioration due to decreased use and altered body carriage. Additionally, the nociceptive pain input into the system can result in sensitization and more pain. Heightened pain results in further negative affects on the musculoskeletal system – muscle atrophy, trigger point development, muscle pain – which in turn results in a greater burden of pain as a result of decreased bone support. Thus there is concurrent deterioration of the musculoskeletal and sensory systems. Pain also has an effect on cognitive function and on emotional states, resulting in heightened fear, anxiety and poor sleep. These changes in turn feed back and heighten pain. The inability to perform daily activities, resulting from pain and deterioration of the musculoskeletal system, also drives negative affective changes through decreased and altered actions with the cat's environment.

Caregiver Observation

Owners may be completely unaware of subtle changes in their cat's behavior or of their potential significance as indications of pain. A validated musculoskeletal pain index can be helpful in uncovering evidence. (www.painfreecats.com) Among these may be:

- Avoiding other household members

- Increased grumpiness
- Decreased grooming
- Restlessness
- Changes in elimination behavior
- Clumsiness
- Reluctance to jump up or down

In the veterinary setting, assessing a cat's emotional and physical characteristics is often confounded by the stress of taking this territorial animal out of his home range and surrounding him with strangers. It can be especially challenging to assess gait and chronic musculoskeletal pain in an animal whose instinct is to freeze or flee or to distinguish pain from fear. With this in mind, a validated pain score has been developed the Feline Musculoskeletal Pain Index (FMPI) which gives the power of observation to caregivers in the home. Questions are addressed towards specific indicators using accessible language.

Arguably most important role of the caregiver in the diagnosis of OA pain is the use of video and photography. Most households have a "smart phone" with photographic and video capability. In the comfort of the home range, the locomotion of a cat may be most reliably observed and largely absent from the examination room. The quality of the video in particular can be demonstrated to the caregiver and is comprised of view tips and techniques to make it both a useful and efficient component of the diagnostic process. While caregivers will be tempted to provide long video segments, lovingly made, instructing them to limit the clip to a minute or less and from a distance where evaluation of the whole cat is critical to efficient examination time.

Physical Examination

A comprehensive physical examination is important but may yield little beyond assessment of the cat's gait in the examination room unless the circumstances are carefully managed. A quiet, secure and low stress environment is key. The cat should be allowed to acclimate to the room at which time a visual assessment of gait may be possible. A feline facial pheromone diffuser plugged in overnight in the room can help reduce a fear response. Cats do not walk in straight lines, are not usually trained to the leash and generally are more interested in investigating the unfamiliar environment or seeking somewhere to hide. In some cases it is possible to assess their willingness to jump. Palpation and manipulation of the joints must be done gently and it is not unusual for some cats to resent this even if joints are normal and pain-free.

A set of comprehensive videos giving full instruction on the appropriate position and method of physical examination is available online.

With so many complex changes occurring and multiple joint often involved, staging of the OA patient may seem daunting. However, staging is probably best performed by assessing the overall impact on the whole cat. A simple staging of the impact of OA based on activity and mobility could be:

Stage	Activity/mobility
1	Early signs of activity impairment
2	Intermittent signs of activity impairment
3	Obvious activity impairment and some decrease in mobility
4	Loss of mobility with significant pain

Key Therapeutic Points

Multi-Modal Treatment

Targeted multi-modal pain management is intended to reduce the risk of drug toxicities and to target the different components of chronic pain, including maladaptive pain.

Gabapentin

Pain modulation happens in the dorsal horn of the spinal cord. In the dorsal horn, there are dramatic anatomic changes that happen in the face of chronic maladaptive pain. Gabapentin affects the alpha-2-delta subunit of the calcium channel in the dorsal horn. This drug is really part of the gold standard for managing chronic maladaptive pain in humans, and what has recently become available is information that it also can play an important perioperative role in reducing the reliance that humans have on post-operative opiates. The downfall for gabapentin is that it must be dosed appropriately, Somewhere between 5-20 mg/kg two to three times per day. Under dosing patients will not address maladaptive pain. Doses should commence at 50mg/cat at night for 3-4 days because sedation may occur initially and alarm the caregiver. The dose is then titrated until it is effective. The second step is to give a dose every 12 hours. Dose effectiveness may change over time and should be regularly interrogated.

Polysulfated Glycosaminoglycans

Adequan is a polysulfated glycosaminoglycans that can be given subcutaneously in cats and is very helpful and well-tolerated. Use in cats is extra-label but is nonetheless an important mainstay of OA pain management. Owners can be instructed in subcutaneous administration and the entire bottle dispensed. The dose is 4.4 mg/kg twice weekly for 4 weeks, once weekly for 4 weeks and then at an interval that reflects effective duration, every 10-15 days. Owners will recognize the day on which the cat appears less comfortable and administer in a one day shorter interval.

NSAIDs

Nonsteroidal anti-inflammatory drugs (NSAIDs) are indicated in the treatment of pain and inflammation associated with OA, as they produce analgesic and anti-inflammatory effects. However, the potential toxicity of these compounds must be considered. NSAIDs act predominately by blocking the inflammatory effects of prostaglandins through inhibition of the breakdown of arachidonic acid buy cyclo-oxygenase (COX), specifically COX-2, which is responsible for producing inflammatory

prostaglandins. COX-1 also plays an important part in the inflammatory process and pain perception.

Nutriceuticals

Diets rich in omega-3 fatty acids sourced from fish oil are recommended for cats with OA. Not only are the absolute levels of these omega-3 fatty acids important but also the ratio of omega-3 to omega-6 fatty acids. These diets have been shown to improve weight-bearing and reduced inflammation. Cats given a diet rich in omega-3 or supplemented with omega-3 fatty acids may be comfortable on lower doses of NSAIDs after a period of 6 to 8 weeks. Other sources include Wellactin 125 mg/ml (Nutramax) and Nordic Naturals 150mg/ml. Extrapolated dosage for cats is 75mg/kg/day.

Pulsed Electromagnetic Field Therapy

Pulsed electromagnetic field (PEMF) therapy is a non-invasive, non-thermal treatment that involves pulsing electromagnetic fields in tissue to promote healing. PEMF devices have been approved by the U.S. Food and Drug Administration (FDA) to treat non-union fractures and cleared to treat post-operative pain and edema, osteoarthtitis and plantar fasciitis. Implementation of PEMF therapy in veterinary medicine is increasing. Pathologies that are often treated with PEMF devices include bone fractures, inflammation and arthritis, pain, edema, and chronic wounds. Though there is a growing body of basic and clinical evidence in support of PEMF treatment as a therapeutic modality, veterinary practitioners and animal owners report significant confusion about PEMF devices largely due to the number of different types of devices and the varying amounts of evidence that support each type of device.

Feline-Specific Anti-nerve Growth Antibody

Neutralizing antibodies against nerve growth factor (NGF) are analgesic in rodent models, naturally occurring degenerative joint disease(DJD) pain in dogs and chronic pain in humans. Currently, the nonsteroidal anti-inflammatory drug (NSAID) meloxicam is approved in Europe for use in treating chronic pain and cats, but has not been approved for this use in the United States. There are concerns about the use of NSAIDS for long periods of time and cats, especially because of the majority of cats presenting with DJD related pain have evidence of chronic kidney disease. A double blind, placebo controlled randomized pilot study with 12 cats in each of three groups evaluated the efficacy of a fully felinized anti-NGF antibody (NV-02) for the treatment DJD pain and mobility impairment in cats. The results of this study showed a clear positive treatment effect with NV–02 in the study cats given the drug. The beneficial effects were seen for objectively measured activity, and also, despite a large caregiver placebo effect, for owner assessed subjective measures. The duration of affect appear to be about six weeks, based on objectively measured activity. This is similar to the duration of efficacy of 0.2 mgs/kg IV in dogs of at least four weeks. The investigators concluded that the potential impact in veterinary medicine of an injection lasting approximately six weeks for the control of long term pain in the cat is very positive and clinically relevant. Further clinical studies are warranted.

SUMMARY

1. Osteoarthritis is an important cause of chronic pain and loss of quality of life in cats
2. There are multiple tools available to help clinicians and caregivers recognize the behaviors that demonstrate pain, create a sense of urgency around its treatment and evaluate the efficacy of a pain management plan upon implementation
3. A multi-modal pain management plan is necessary to improve mobility and quality of life
4. Future scientific investigations will result in new methods of pain management that will integrated into a multimodal plan
5. Clinicians have an obligation to balance the importance of relieving pain and the impact that their plan may have on the relationship between cat and caregiver

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3.1.6. THE WHEEZING CAT:LOWER RESPIRATORY DISEASE

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CLINICAL SIGNS

Clinical signs of lower airway disease most commonly include coughing with or without periodic respiratory distress. Audible wheezes and prolonged expiration or an expiratory push may be appreciated. Upper airway diseases should result in prolonged inspiratory phase, by contrast. Cats are usually 1 year to about 9 or 10 years when first affected. Kittens with cough should be evaluated for an underlying disease such as parasitic migration or for another cause such as nasopharyngeal polyp. Older cats, while they will continue to have asthma throughout their lives should not develop asthma for the first time as geriatrics. Any time asthma is suspected in an old cat, a complete evaluation should be performed, as a distinct underlying condition is much more likely present

Some cats have a syndrome more consistent with asthma, reversible bronchoconstriction in response to inhaled allergens. Other cats are more similar to chronic bronchitis with cough, excessive mucous and bronchial thickening. Still others are difficult to distinguish. The underlying cause is often unknown and the role of viral respiratory infections, a common cause of asthma in genetically susceptible infants, is unclear. Some cats are affected seasonally and others year round. Certain cats appear sensitive to environmental contaminants such as dusty cat litter, tobacco smoke or aerosol fragrances. Secondary spontaneous pneumothorax has been reported. Clinical signs of affected cats include cough, wheeze and respiratory distress.

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Thoracic radiology is the main source of diagnosis. Affected cats have a bronchial or bronchointerstitial pattern. Hyperinflation due to air trapping and right middle lung lobe collapse from a mucous plug may occur.

Baseline work up is usually unremarkable. In endemic areas, heartworm antigen and antibody testing and Baermann fecal for lungworms is indicated. Bronchoalveolar lavage appears unable to distinguish between chronic bronchitis and asthma but is useful to evaluate for other causes such as neoplasia or infection.

Pre-oxygenate for 10 minutes, intubate using a sterile tube. Infuse 3-6 mls of sterile saline into the tube and collect for cytology.

ACUTE SIGNS

Acute therapy includes supplemental oxygen therapy, glucocorticoids and bronchodilators. Cats with lower airway disease should improve with in 4-6 hours. In acute situations 2-4 mg/cat of dexamethasone IV. 5-10 mg/cat prednisolone may be needed foe a period of time. As bronchoconstriction is common, albuterol 1-2 puffs every 6 hours or terbutaline (0.01 mg/kg SQ) is often helpful.

The distinction between cardiac and respiratory distress may be difficult. Rapid assessment by ECG is ideal, however the cardiac biomarker NT pro-BNP may be helpful to exclude a diagnosis of heart disease. Pulmonary infection is not common in cats but must be excluded before commencing corticosteroid therapy.

CHRONIC

Chronic therapy is aimed at removal of any identifiable triggers and glucocorticoids. Inhaled corticosteroids reduce the risk of systemic effects but require administration via face mask and spacer. (Aerokat, Trudell Medical, London and Ontario).Typically oral glucocorticoids precede inhaled therapy and are given simultaneously for a period followed by tapering of oral form.

Several drugs have been shown to be of no help: cyproheptadine, cetirizine, zafirlukast, and maropitant.

Fluticasone is the inhaled corticosteroid most commonly used. Dosage is not certain. This author starts fluticasone at 110mcg/cat twice daily. Combination products such fluticasone with the long-acting bronchodilator salmeterol improved efficacy in one study. For cats who do not respond well to oral corticosteroids, the inhaled form is unlikely to be helpful. Cyclosporine has been described as helpful (10 mg/kg PO q 12 hours) with improvement in airway hyperresponsiveness and amelioration of cytologic inflammation. It is a potent immunosuppressive so blood levels should be monitored as well as potential for pathogens to gain a foothold with an impaired immune system. Doxycycline or Azithromycin is prescribed if infection is suspected, but this is uncommon.

Future therapeutic options are promising including rush immunotherapy which has been effective in research cats. Masitinib and fish oil may be useful after further research. Stem cell therapy research is ongoing for long-term disease control.

Improving Indoor Air Quality

Key elements of improved air quality are source control, adequate ventilation (especially in shower, laundry, and cooking areas), maintaining relative humidity between 30–50%, changing air filters on a regular basis, and air duct cleaning as needed. To limit dust accumulation, use a quality vacuum with a high-efficiency filter

weekly. Water leaks should be promptly repaired, and after any flooding, areas should be thoroughly cleaned and dried. If materials cannot be dried promptly, they should be replaced. CO monitors should be installed in the home. All gas appliances should be properly functioning or inspected and adequately cleaned and repaired. Gas stoves and heaters should be vented to the outside of the home. Owners should be reminded that gas ranges should never be used as a heat source. Smoke detectors, along with the enforcement of improved building codes, are effective in reducing fire-related deaths. Owners should be cautioned about so-called air purifiers that use ionization as a means of "clearing the air" but actually increase indoor ozone concentrations, at times to levels well in excess of that considered safe. Kerosene heaters should be used only as indicated by the manufacturer's instructions, and should be refueled outdoors using specially manufactured low-sulfur fuel. Cars, lawn mowers, etc., should never be left running inside a garage or shed, especially if the space is ever used to house a pet

HEARTWORM DISEASE

In endemic areas, indoor and outdoor cats are at similar risk for infection. The infective L3 stage larva enters the cat through a bite wound, molts to L4 and L5 stages and migrates to the pulmonary arteries as immature adults 70-90 days after infection. Once infected, the cat's natural resistance results in a short period of microfilaremia. The clinical worm burden is also lower in cats, from 1 to 9 worms, than in dogs. The average time for infective larvae to develop into circulating microfilariae in experimental feline infections is about 8 months. Thus, microfilaremia is uncommon (<20%), inconsistent, and transient in cats, and very low numbers are usually produced. The comparable development period in dogs is 5-6.5 months.

There is high mortality of L5 as they arrive in the distal pulmonary arteries in the cat. High mortality of immature adult heartworms is associated with intense pulmonary bronchial and parenchymal response called Heartworm-Associated Respiratory Disease (HARD). Residual pulmonary pathology related to HARD persists even after immature heartworms die. Thus many heartworm infections may be misdiagnosed as feline asthma.

Heartworm disease in cats is characterized by pulmonary eosiniphilic bronchial and interstitial reaction associated with immature adults (3-6 months after infection) chronic lung changes associated with mature adult heartworms (6 months – 4 years) and acute respiratory distress associated with the death of worms at any age.

Lesions associated with HARD are initiated by immature larvae as early as 70 to 90 days after infection. The lesions in HARD are characterized by peribronchial fibrosis, intertstitial myofibroblasts and fibrosis of alveolar struts. Muscular hypertrophy, villous endarteritis and adventitial cellular infiltrates are common findings in all pulmonary arteries, although caudal arteries are most commonly seen radiographically. Infiltrative interstitial lung disease, reduced clearance of mucous and inflammatory debris are the hallmark of this lung disease as opposed to increased bronchiolar wall reactivity as proposed in asthma models.

Wolbachia are gram-negative bacteria belonging to the order Rickettsia that reside within the body of D. immitis and appear within 2 months of exposure to infective larvae. The release of bacteria following worm death has shown to cause upregulation of proinflammatory cytokines, neutrophil recruitment and an increase in specific immunoglobulins, although the role of this intracellular bacteria alone in the pathogenesis of feline heartworm is unclear.

Clinical signs often include coughing or vomiting most commonly associated with immature heartworms arriving in the lungs or death of adult heartworms. The initial arrival of L5 in the distal pulmonary arteries induces diffuse pulmonary infiltrates and often eosinophilic pneumonitis. Clinical signs associated with acute phase subside as the worms mature but lesions remain even in cats who clear infection.

Most infected cats will be asymptomatic during most of infection. Adult heartworm death, even as little as one, may induce acute, potentially fatal disease with acute circulatory collapse or severe respiratory distress. Anorexia and lethargy may be the only presenting complaints. Coughing or intermittent vomiting may occur. The vomiting appears unrelated to eating. Inflammatory mediators and stimulation of the chemoreceptor trigger zone are postulated as the cause.

Positive antibody result indicates infection with L3 which has moulted to L4 and lived at least 2-3 months. Adult heartworms may or may not develop from this infection. ELISA antigen testing is specific for glycoproteins associated mainly with reproductive tract of fully mature female worms, making false-negative results common. Cats presenting with HARD from immature adults will be antigen negative as will those with low worm numbers. Eosinophilic cytology from BAL will be most intense 3-6 months after migration and is intermittent. Thoracic radiology is helpful but not specific. Aelurostrongylus and roundworm infection are the most common pulmonary infections to mimic heartworm radiologic signs.

Year round heartworm prevention prevents both patent infection and HARD. When cats were infected with L3 heartworms experimentally and treated with selamectin monthly commencing 28 days later did not develop adult worms but did seroconvert to antibody-positive status. Another study demonstrated that cats pretreated with selamectin 32 and 2 days before L3 infection did not develop HARD or seroconvert to antibody positive. The role of Wolbachia remains to be illuminated. Heartworm in cats is often confused with lungworm, roundworm, bronchitis, asthma, or in many cases overlooked.

istopathologic and Morphometric Evaluation of the Nasal Airways of Cats with Experimentally Induced Asthma

Christine M. Venema; Kurt J. Williams; Laurel J. Gershwin; Carol R. Reinero; Stephan A. Carey

ACVIM 2011

Allergic rhinitis and asthma are often seen concurrently in human patients. In human allergic airways medicine, the "unified airways theory" has been advanced to explain the coincidence of allergic rhinitis and asthma documented frequently in people.This theory suggests that the upper airways are immunologically linked to the lower airways. Epidemiologic evidence suggests that allergic rhinitis and asthma are comorbid conditions in many human patients.Histopathologically, this finding is supported by demonstration of eosinophilic inflammation in both the nasal and pulmonary airways regardless of clinical signs. Moreover, treatment of rhinitis reduces functional and clinical parameters of asthma in human patients. Also proposed as part of the "unified airways theory" is the idea that different clinical manifestations of global airways disease predominate during particular periods of life while other regress. Allergic rhinitis and asthma may represent the respiratory manifestations of a generalized atopic disease process that begins with atopic dermatitis and progresses over time. This concept has been referred to as the "atopic march.

A similar relationship between the nasal and pulmonary airways of cats has not been evaluated. To investigate the possibility of a "unified airways theory" in cats, we have developed an experimental model of feline asthma that replicates the functional, clinical, immunologic, and histopathologic features of the spontaneously occurring disease.[21] We hypothesized that sensitizing and chronically challenging cats with a relevant aeroallergen would induce morphologic and histopathologic changes in the nasal airways that mimic the changes induced in the bronchial airways. Five mixed breed cats were sensitized to and challenged with Bermuda grass allergen (BGA) while four cats were instead treated with phosphate buffered saline (PBS) and served as controls. The nasal and bronchial tissues of these cats were harvested and analyzed with morphometry to measure the numeric cell densities (NCD) of mucosal inflammatory cells and with image analysis to determine the volume density of mucosubstances within the nasal epithelium. Determining whether the upper and lower airways of asthmatic cats respond to aeroallergen challenge similarly will expand our understanding of the immunopathogenesis of feline allergic airways disease. This information may lead to the development of novel therapies for conditions such as feline bronchial asthma and chronic rhinosinusitis, which can be challenging to manage effectively.

Our findings are similar to those documented in human patients with allergic airways disease. In particular, concomitant eosinophilic rhinitis has been demonstrated in human asthmatics.[9,22] In the experimental models of feline asthma of our study, eosinophilic inflammation was focally distributed in the anterior region of the nasal airways, which is similar to the heterogenous distribution of eosinophilic inflammation noted in biopsy samples obtained from humans with naturally occurring chronic rhinosinusitis.[23] Additionally, the results of our study demonstrate that mast cell rhinitis occurs in cats following artificial aeroallergen sensitization and chronic challenge. A significant mast cell infiltrate in the nasal mucosa also characterizes allergic rhinitis in

humans.[24–26] As in the feline asthma models of our study, a heterogenous distribution of eosinophilic and mast cell inflammation throughout the upper and lower airways has been demonstrated in humans with allergic airways disease.[9,12,22]

This study represents the first characterization of the histopathologic and immunologic alterations of the nasal airways of experimental models of feline asthma and the first investigation into a potential mechanistic link between the upper and lower airways of asthmatic cats. These results provide a basis for future studies to investigate the pathophysiology of feline rhinitis and asthma. Our results suggest that concurrent inflammation within the nasal airways should be considered in the evaluation of asthmatic cats.

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Cecilia Villaverde

4.1.1. PREVENCION DE OBESIDAD

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La obesidad es una condición común en perros y gatos, aunque la prevalencia varía según el área geográfica. Además de frecuente, la obesidad se asocia a varias enfermedades y en perros se asocia a una longevidad reducida1,2. El tratamiento es largo y trabajoso, con un porcentaje elevado de pacientes que no llegan a su peso ideal o lo recuperan3,4. Prevención del sobrepeso es por tanto una estrategia importante para mejorar la calidad de vida de nuestros pacientes.

Para prevenir la obesidad hay que conocer las asociaciones5 (potenciales factores de riesgo) que se pueden identificar durante la visita y la evaluación nutricional. Estos se pueden clasificar en factores del animal y de los propietarios. Estos factores nos permitirán identificar pacientes a riesgo de sobrepeso y las mejores estrategias para prevenirlo.

Factores del animal5

Raza: Tanto en perros como en gatos, estudios epidemiológicos han encontrado que ciertas razas tienen una mayor prevalencia de obesidad, lo que se puede deber a predisposición genética. Estudios en Europa han identificado una prevalencia de obesidad relativamente alta en perros y gatos de concurso6,7, lo que puede indicar que ciertos estándares de raza necesitan ser ajustados. El riesgo de obesidad de ciertas razas puede deberse a necesidades energéticas menores que la media o a una motivación elevada por la comida.

Sexo: algunos estudios han identificado ser macho en gatos y ser hembra en perros como factores de riesgo.

Esterilización: este es un factor de riesgo bien descrito, que se asocia a una reducción del gasto energético y de un aumento temporal del apetito.

Tasa de crecimiento: el crecimiento acelerado en perros8 y gatos9 se ha asociado al sobrepeso en adultos.

Edad: la obesidad es más frecuente en animales adultos de mediana edad.

Factores del propietario5,10

Elección de dieta: palatabilidad, densidad energética de la dieta pueden contribuir al sobrepeso. Un estudio en gatos identifico el alimento seco como riesgo, y otro encontró una asociación con el alimento seco, mientras que otros estudios no encontraron una asociación entre el contenido de humedad de la dieta y el sobrepeso.

Método de alimentación: Instrucciones inadecuadas en la etiqueta, uso de premios y restos de mesa, y alimentación a voluntad se han asociado a riesgo de sobrepeso.

Ejercicio y ambiente: en gatos, la vida en interior se ha asociado a sobrepeso, potencialmente debido a una menor actividad física. En perros, la actividad física protege contra el sobrepeso.

Características de los cuidadores: ciertos estudios han encontrado asociaciones entre obesidad de la mascota y la obesidad de propietarios (en perros), una mayor humanización, y un menor nivel de ingresos. La característica mas importante asociada a la obesidad canina y felina es la subestimación de la condición corporal de su mascota.

Es importante identificar el tipo y cantidad de factores de riesgo en cada paciente para desarrollar un plan de prevención, desde la primera visita (de cachorros o de gatito). Ciertos momentos críticos incluyen el momento de la castración, donde es recomendable reducir el alimento un 20-30% y evitar alimentación ad libitum, cambios de dieta (con diferente densidad energética) y el envejecimiento (reducción de actividad). Las estrategias de prevención incluyen:

El uso de alimentación racionada, y el uso de una balanza de cocina diariamente.

Elección de dieta moderada en calorías (<3.5 kcal/g)

Promoción de ejercicio/actividad física (dar minutos por semana, tiempo diario de juego, y no confiar en que la mascota auto ejercite)

Medir peso y condición corporal una vez al mes (hacerlo siempre en cada visita, y enseñar a cliente a hacerlo en casa). Es recomendable llevar un buen registro de pesos, incluso en forma gráfica, para identificar tendencias de forma temprana y atajarlas.

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4.1.2. TRATAMIENTO DE OBESIDAD

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La obesidad, definida como el exceso de grasa corporal con efectos negativos sobre el organismo, es una condición común en perros y gatos y se asocia a varias enfermedades además de a una longevidad reducida1,2 en perros. El tratamiento es largo y trabajoso, con un porcentaje elevado de pacientes que no llegan a su peso ideal o lo recuperan3,4. Los cuidadores deben ser informados del tiempo y esfuerzo que hay que dedicar para este proceso, y el equipo veterinario debe ofrecer soporte practico y emocional en estos casos. En ciertos casos complicados, la mejora parcial puede ser aceptable, no todos los pacientes van a llegar a una condición corporal ideal, pero reducciones de peso van a ser un beneficio también. El tratamiento principal de la obesidad es la restricción calórica.

Diagnostico

El sobrepeso en la clínica veterinaria se suele hacer con la condición corporal5,6, existen varias escalas, siendo una de ellas la de 9 puntos, donde 5 es ideal (4 en perros también se considera ideal) y valores de 6-7 indican sobrepeso y 8-9 obesidad. Se estima que cada punto por encima de 5 es un exceso de 10-15% de peso. Otros métodos prácticos de estimación de grasa corporal son el BFI (body fat index) y medidas morfométricas7,8. Métodos más objetivos de medida de la grasa corporal, como el DEXA o el uso de isotopos, es costoso y complicado.

Plan de perdida de peso9,10

Los propietarios del paciente deben reconocer que su mascota tiene sobrepeso y también estar decididos a hacer algo al respecto, nuestros esfuerzos serán en vano si no están alineados con nuestro plan. El plan debe incluir elección de dieta, cantidades, pauta y seguimiento.

Dieta: el uso de dietas veterinarias formuladas especialmente para perdida de peso es positivo en dos aspectos: densidad energética reducida (para promover saciedad) y, mas importante, densidad nutritiva alta, para asegurar el aporte nutricional mientras se realiza la restricción calórica. Las dietas de este estilo deben ser altas en proteína para minimizar la perdida de masa muscular.

Cantidad: si se conoce cuantas calorías esta consumiendo el paciente (vía una buena historia dietética) se puede reducir en un 20%. Si no se conoce, se pueden usar formulas, por ejemplo, el RER (requerimientos energéticos en reposo) en perros (70 x peso metabólico) o 80% del RER en gatos. Se puede usar peso actual o peso objetivo. El peso actual es conocido y resulta en restricción mas moderada; el peso objetivo no siempre es conocido y resulta en restricción mas agresiva. Dependiendo del caso, se puede elegir un enfoque u otro. La cantidad diaria se consigue dividiendo el objetivo calórico entre la densidad de la dieta de elección. Esta se debe pesar, ya que el uso de medidas de volumen tiene mucho error, especialmente para cantidades pequeñas11. Si se elige dar premios, una recomendación común es dedicar 90% de las calorías a la dieta y 10% para cualquier item calórico (premios, restos de mesa, aceites, etc.).

Pauta: la alimentación siempre debe ser racionada, múltiples comidas al día pueden ayudar a reducir conductas de hambre. El uso de puzles dispensadores de alimento o métodos de alimentación más lenta, etc. puede ayudar a reducir el aburrimiento. Es importante asegurarse que el paciente solo recibe lo que le corresponde. SI se usan recipientes12, es importante que sean del tamaño adecuado.

Seguimiento: se debe pesar al paciente en la misma balanza, idealmente misma hora del día, en ayudas, cada 2 semanas (mas adelante se puede espaciar a 4). La ración se debe ajustar para promover perdida de peso entre 0.5-2% semanal.

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4.1.3. NECESIDADES NUTRICIONALES DE LOS GATOS SENIOR

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En muchos países, los gatos de edad avanzada son un porcentaje importante de las clínicas veterinarias. El envejecimiento se define como los cambios fisiológicos y metabólicos que ocurren en el cuerpo tras la madurez, que lleva a una reducción de la función orgánica. Estos cambios incluyen cambios en el color de pelo, alteraciones en los sentidos, cambios de comportamiento y cambios menos aparentes en composición corporal, función inmunitaria y fisiología digestiva, entre otros.

No existe una definición especifica de “senior”. Una clasificación1 en gatos los define en varias etapas incluyendo maduros (7-10 años), senior (11-14) y geriátricos (15+). Las necesidades nutricionales en gatos de edad avanzada pueden verse afectadas, pero no de forma homogénea. Por este motivo, las publicaciones científicas2 y regulatorias3,4 no dan recomendaciones específicas para esta etapa de vida (a diferencia de crecimiento, reproducción y mantenimiento). Por tanto, las dietas para gatos “senior” en el mercado van a ser altamente variables y su composición nutricional es dependiente de cada fabricante.

Energía

Las necesidades energéticas en gatos adultos no varían demasiado por edad5, pero la prevalencia de obesidad es mas alta en gatos maduros y senior que en jóvenes. Se ha descrito que los gatos geriátricos pueden mostrar más problemas de delgadez y atrofia muscular6, potencialmente asociado a una menor digestibilidad de la proteína y la grasa. En todos los gatos de edad avanzada, es importante evaluar su masa grasa y muscular durante la evaluación nutricional7 para identificar si el paciente necesita una dieta alta en calorías y mayor acceso al alimento o si se debe realizar un plan de perdida de peso.

Macronutrientes

No hay evidencia que las necesidades proteicas se vean reducidas o aumentadas con la edad, aunque algunos estudios en gatos han mostrado una reducción en la digestibilidad de este nutriente en un porcentaje de la población6,8,9, la digestibilidad de la grasa y el almidón también pueden verse afectadas. En estos casos, es importante evitar moderación proteica innecesaria y el uso de dietas altamente digestibles puede ser positivo.

Otros

La absorción de minerales no se afecta por la edad9. La tolerancia a ciertos minerales como el fosforo o sodio puede ser reducida en presencia de enfermedades renales o cardiacas. Se ha propuesto que el uso de ciertos nutrientes (como antioxidantes, fibra prebiótica y ácidos grasos esenciales) puede contribuir a una mayor longevidad8. Un estudio mostro un efecto positivo sobre la capacidad cognitiva de gatos seniors usando una combinación de antioxidantes, arginina, aceite de pescado y vitaminas del grupo B10.

Elección de alimento en gatos senior

Es importante realizar una evaluación nutricional avanzada en todos los casos. Se debe realizar una historia dietética detallada en todos los casos, para evaluar la idoneidad de la dieta actual. En la evaluación nutricional se van a identificar enfermedades que sean sensibles al manejo nutricional (como osteoartritis, enfermedad renal, enfermedad cardiaca, etc.), y la condición corporal es importante para el cálculo de las necesidades energéticas.

Al no haber recomendaciones de los organismos regulatorios, las dietas senior son altamente variables, y se deben considerar un subgrupo de las de mantenimiento. El equipo veterinario se debe familiarizar con las dietas senior disponibles para identificar sus estrategias: algunas serán altas en calorías (otras serán mas “light”), algunas serán moderadas en proteína y otras serán mas altas, etc. En gatos senior saludables no siempre es necesario cambiar a una dieta senior.

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4.1.4. SUPLEMENTOS: BENEFICIOS, RIESGOS Y COMO EVALUARLOS

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Existen muchos suplementos o nutracéuticos para mascotas en el mercado, incluyendo suplementos vitamínicos y minerales, enzimas, aceites, productos botánicos/de herbolario, suplementos articulares, pre/pro/simbióticos, etc.

El uso de suplementos de forma indiscriminada tiene riesgos. Antes de recomendar suplementos a un paciente es importante la evaluación nutricional1 para identificar los pacientes que se pueden beneficiar de estos. Por otra parte, el suplemento se debe evaluar en dos aspectos principales: seguridad y eficacia.

Cuando se evalúa la eficacia y seguridad de un nutracéutico se deben de tener en cuenta ciertos aspectos:

1. Fabricante: La legislación de suplementos es en general laxa2, con lo que es importante elegir un fabricante/distribuidor de buena reputación. Es importante que el componente activo se encuentre en las concentraciones indicadas, ya que esto puede afectar a su eficacia.

2. Publicaciones y en que especie: Para ciertos suplementos, la evidencia es in vitro, o en modelos (roedores) o en otras especies, y no siempre se puede asumir que el producto será seguro o eficaz en perros o gatos

3. Dosis

4. Interacciones con otros nutrientes (dieta) o con medicaciones.

Los suplementos no son inocuos, y su uso puede conllevar riesgos nutricionales (incluso en productos considerados seguros para la especie). Cada suplemento tiene sus riesgos específicos, pero en general estos se pueden clasificar en:

1. Aporte de calorías no controlado

Algunos suplementos pueden aportar calorías, como por ejemplo los aceites (coco, oliva, pescado, etc.). Estos aportan unas 9 kcal por gramo (8 por mililitro). Los aceites son suplementos comunes, por ejemplo, en dermatosis3. Los ácidos grasos omega 3 del aceite de pescado también son comunes en ciertas enfermedades4 como osteoartritis, glomerulopatías, y enfermedad intestinal entre otras. El aporte de calorías puede promover ganancia de peso indeseada, o, si se reduce la ración diaria para prevenir el sobrepeso, el aporte de nutrientes esenciales se puede ver comprometido.

2. Niveles tóxicos de nutrientes

Este problema puede darse con el uso de suplementos vitamínicos-minerales o con ciertos aceites de pescado (que pueden ser ricos en vitaminas A y D). La adición de suplementos de este estilo sobre una dieta que ya es completa tiene el riesgo que llegar a niveles demasiado alto de vitaminas liposolubles5 o ciertos minerales. Por ejemplo, en cachorros en crecimiento, el exceso de calcio puede resultar en problemas esqueléticos6, y la suplementación en esta etapa de vida puede ser, por tanto, de alto riesgo.

3. Contaminación

Se ha descrito contaminación con metales pesados en aceites marinos7, algas o harina de hueso. Un estudio en Brasil identifico niveles elevados de mercurio en ciertos suplementos vitamínicos8. Es importante preguntar al fabricante los detalles específicos del control de calidad y como se aseguran de que su producto no contiene sustancias indeseables.

4. Antígenos

El uso de suplementos en pacientes con sospecha de alergias o intolerancias alimentarias puede ser causa de fallo de tratamiento. Muchos suplementos incluyen saborizantes que pueden interferir con el manejo dietético de estos problemas.

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4.1.5. ESTRATEGIAS NUTRICIONALES PARA ENTEROPATÍAS CRÓNICAS

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El manejo dietético es importante no solo para el diagnóstico sino para el tratamiento, tanto en EC que responde a la dieta como las que no. En EC que responde a la dieta puede ser la única modalidad terapéutica necesaria2. En todos los casos, un manejo nutricional adecuado es necesario para aporte de energía y nutrientes, soporte del tracto gastrointestinal y control de los signos clínicos.

Los tipos de dietas que se pueden usar en EC incluyen dietas intestinales, de eliminación (o “hipoalergénicas”), y enriquecidas en fibra.

Dietas intestinales:

Las dietas altamente digestibles son usadas con frecuencia en enfermedades gastrointestinales agudas, para facilitar el proceso digestivo y maximizar la absorción de nutrientes. Además de su digestibilidad pueden incluir otras estrategias como ácidos grasos omega 3 y prebióticos. No son la recomendación habitual en EC, pero ciertos pacientes pueden responder a esta estrategia3–5. El uso de dietas intestinales esta también indicado en EC que no responden a la dieta, al ser completas y equilibradas (algunas incluso para crecimiento), fáciles de digerir, y (en general) altas en calorías.

En EC con perdida de proteínas, las dietas intestinales bajas en grasa son una buena opción, en algunos casos el uso de dietas caseras altamente digestibles ultra bajas en grasa es recomendado, como en linfangiectasia intestinal primaria6.

Dietas de eliminación:

Son aquellas con numero limitado de ingredientes en base a ingredientes noveles o en base a proteína hidrolizada y son una de las recomendaciones más habituales en EC. Su uso es indicado para identificar pacientes con EC que responde a dieta (diagnostico) y también para su manejo a largo plazo7. Estas dietas también son altamente digestibles, y pueden incluir fibra prebiótica, ácidos grasos omega 3, etc.

Dietas enriquecidas en fibra:

La fibra puede ayudar al control de la diarrea en colitis8–10 mediante sus efectos físico-químicos y mediante su efecto sobre el microbioma intestinal. Las dietas intestinales y de eliminación suelen incluir pequeñas cantidades de fibra prebiótica; las dietas enriquecidas en fibra para la colitis suelen llevar cantidades mas elevadas.

Estas dietas no se recomiendan en caso de enfermedad del intestino delgado, ya que su densidad energética puede ser bastante baja.

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4.1.6. MANEJO NUTRICIONAL DE LA ENFERMEDAD RENAL CRONICA

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Los pacientes con ERC deben recibir una evaluación nutricional1 avanzada, para decidir el mejor plan de alimentación para cada caso, incluyendo elección de dieta, cantidades y pauta de alimentación.

Elección de dieta

Las dietas renales tienen varias características en común2, incluyendo restricción de fosforo, moderación proteica, moderación de sodio, fortificación vitaminas hidrosolubles (para compensar las pérdidas urinarias), potencial alcalinizante, y adición de ácidos grasos omega 3. En gatos, las dietas renales suelen también estar fortificadas en potasio.

La proteína en ERC es un tema controvertido3 ya que existe la preocupación de perdida de masa muscular debido a consumo proteico inadecuado y/o perdida de palatabilidad. Las dietas renales, sin embargo, no deben estar restringidas en este nutriente sino moderarlo (además de usar fuentes de alta calidad) para reducir la cantidad de productos nitrogenados de desecho. En el caso de proteinuria renal, la moderación proteica puede ayudar a reducir sus pérdidas urinarias4. La moderación proteica también ayuda a restringir el fosforo de forma mas marcada, ya que muchos ingredientes proteicos son altos en este mineral. La restricción de fosforo se considera muy importante para enlentecer la progresión de la enfermedad5. La adición de ácidos grasos omega 3 EPA y DHA ha mostrado reducción de la progresión y de la magnitud de proteinuria en perros con ERC experimental6, aunque aún no existen datos en gatos excepto un estudio retrospectivo7.

Afortunadamente, hay varias opciones en el mercado, tanto secas como húmedas, que pueden usarse en cada caso, y varían en precio, textura, ingredientes, y perfil nutricional. El alimento seco es mas calórico, pero el húmedo es potencialmente más palatable y contribuye a la hidratación del paciente.

En casos con enfermedades concomitantes, o donde la dieta comercial no es apetente, se puede considerar una dieta casera formulada por un especialista en nutrición, individualizada para el caso.

Si se dan premios/extras, u otras comidas no balanceadas como aceites, estos deben seguir las mismas estrategias que la dieta principal, y limitarse a un 10% de las calorías diarias.

Ración diaria

Se pueden calcular las necesidades energéticas del paciente8,9 y dividirlas por la densidad calórica del alimento de elección o se pueden usar las guías del empaquetado. Ambos métodos tienen un error elevado, y la ración diaria se debe ajustar para mantener un peso estable con una condición corporal buena.

Pauta de alimentación

Se puede considerar alimentación a voluntad en pacientes delgados con apetito caprichoso, pero en pacientes obesos o con tendencia al sobrepeso, es recomendable racionar el alimento. Se puede dividir en múltiples comidas al día.

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Elección de dieta

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Ración diaria

Se pueden calcular las necesidades energéticas del paciente8,9 y dividirlas por la densidad calórica del alimento de elección o se pueden usar las guías del empaquetado. Ambos métodos tienen un error elevado, y la ración diaria se debe ajustar para mantener un peso estable con una condición corporal buena.

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4.1.9. NECESIDADES NUTRICIONALES DE LOS GATOS SENIOR

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En muchos países, los gatos de edad avanzada son un porcentaje importante de las clínicas veterinarias. El envejecimiento se define como los cambios fisiológicos y metabólicos que ocurren en el cuerpo tras la madurez, que lleva a una reducción de la función orgánica. Estos cambios incluyen cambios en el color de pelo, alteraciones en los sentidos, cambios de comportamiento y cambios menos aparentes en composición corporal, función inmunitaria y fisiología digestiva, entre otros.

No existe una definición especifica de “senior”. Una clasificación1 en gatos los define en varias etapas incluyendo maduros (7-10 años), senior (11-14) y geriátricos (15+). Las necesidades nutricionales en gatos de edad avanzada pueden verse afectadas, pero no de forma homogénea. Por este motivo, las publicaciones científicas2 y regulatorias3,4 no dan recomendaciones específicas para esta etapa de vida (a diferencia de crecimiento, reproducción y mantenimiento). Por tanto, las dietas para gatos “senior” en el mercado van a ser altamente variables y su composición nutricional es dependiente de cada fabricante.

Energía

Las necesidades energéticas en gatos adultos no varían demasiado por edad5, pero la prevalencia de obesidad es mas alta en gatos maduros y senior que en jóvenes. Se ha descrito que los gatos geriátricos pueden mostrar más problemas de delgadez y atrofia muscular6, potencialmente asociado a una menor digestibilidad de la proteína y la grasa. En todos los gatos de edad avanzada, es importante evaluar su masa grasa y muscular durante la evaluación nutricional7 para identificar si el paciente necesita una dieta alta en calorías y mayor acceso al alimento o si se debe realizar un plan de perdida de peso.

Macronutrientes

No hay evidencia que las necesidades proteicas se vean reducidas o aumentadas con la edad, aunque algunos estudios en gatos han mostrado una reducción en la digestibilidad de este nutriente en un porcentaje de la población6,8,9, la digestibilidad de la grasa y el almidón también pueden verse afectadas. En estos casos, es importante evitar moderación proteica innecesaria y el uso de dietas altamente digestibles puede ser positivo.

Otros

La absorción de minerales no se afecta por la edad9. La tolerancia a ciertos minerales como el fosforo o sodio puede ser reducida en presencia de enfermedades renales o cardiacas. Se ha propuesto que el uso de ciertos nutrientes (como antioxidantes, fibra prebiótica y ácidos grasos esenciales) puede contribuir a una mayor longevidad8. Un estudio mostro un efecto positivo sobre la capacidad cognitiva de gatos seniors usando una combinación de antioxidantes, arginina, aceite de pescado y vitaminas del grupo B10.

Elección de alimento en gatos senior

Es importante realizar una evaluación nutricional avanzada en todos los casos. Se debe realizar una historia dietética detallada en todos los casos, para evaluar la idoneidad de la dieta actual. En la evaluación nutricional se van a identificar enfermedades que sean sensibles al manejo nutricional (como osteoartritis, enfermedad renal, enfermedad cardiaca, etc.), y la condición corporal es importante para el cálculo de las necesidades energéticas.

Al no haber recomendaciones de los organismos regulatorios, las dietas senior son altamente variables, y se deben considerar un subgrupo de las de mantenimiento. El equipo veterinario se debe familiarizar con las dietas senior disponibles para identificar sus estrategias: algunas serán altas en calorías (otras serán mas “light”), algunas serán moderadas en proteína y otras serán mas altas, etc. En gatos senior saludables no siempre es necesario cambiar a una dieta senior.

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4.1.10. SUPLEMENTOS: BENEFICIOS, RIESGOS Y COMO EVALUARLOS

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Existen muchos suplementos o nutracéuticos para mascotas en el mercado, incluyendo suplementos vitamínicos y minerales, enzimas, aceites, productos botánicos/de herbolario, suplementos articulares, pre/pro/simbióticos, etc.

El uso de suplementos de forma indiscriminada tiene riesgos. Antes de recomendar suplementos a un paciente es importante la evaluación nutricional1 para identificar los pacientes que se pueden beneficiar de estos. Por otra parte, el suplemento se debe evaluar en dos aspectos principales: seguridad y eficacia.

Cuando se evalúa la eficacia y seguridad de un nutracéutico se deben de tener en cuenta ciertos aspectos:

1. Fabricante: La legislación de suplementos es en general laxa2, con lo que es importante elegir un fabricante/distribuidor de buena reputación. Es importante que el componente activo se encuentre en las concentraciones indicadas, ya que esto puede afectar a su eficacia.

2. Publicaciones y en que especie: Para ciertos suplementos, la evidencia es in vitro, o en modelos (roedores) o en otras especies, y no siempre se puede asumir que el producto será seguro o eficaz en perros o gatos

3. Dosis

4. Interacciones con otros nutrientes (dieta) o con medicaciones.

Los suplementos no son inocuos, y su uso puede conllevar riesgos nutricionales (incluso en productos considerados seguros para la especie). Cada suplemento tiene sus riesgos específicos, pero en general estos se pueden clasificar en:

1. Aporte de calorías no controlado

Algunos suplementos pueden aportar calorías, como por ejemplo los aceites (coco, oliva, pescado, etc.). Estos aportan unas 9 kcal por gramo (8 por mililitro). Los aceites son suplementos comunes, por ejemplo, en dermatosis3. Los ácidos grasos omega 3 del aceite de pescado también son comunes en ciertas enfermedades4 como osteoartritis, glomerulopatías, y enfermedad intestinal entre otras. El aporte de calorías puede promover ganancia de peso indeseada, o, si se reduce la ración diaria para prevenir el sobrepeso, el aporte de nutrientes esenciales se puede ver comprometido.

2. Niveles tóxicos de nutrientes

Este problema puede darse con el uso de suplementos vitamínicos-minerales o con ciertos aceites de pescado (que pueden ser ricos en vitaminas A y D). La adición de suplementos de este estilo sobre una dieta que ya es completa tiene el riesgo que llegar a niveles demasiado alto de vitaminas liposolubles5 o ciertos minerales. Por ejemplo, en cachorros en crecimiento, el exceso de calcio puede resultar en problemas esqueléticos6, y la suplementación en esta etapa de vida puede ser, por tanto, de alto riesgo.

3. Contaminación

Se ha descrito contaminación con metales pesados en aceites marinos7, algas o harina de hueso. Un estudio en Brasil identifico niveles elevados de mercurio en ciertos suplementos vitamínicos8. Es importante preguntar al fabricante los detalles específicos del control de calidad y como se aseguran de que su producto no contiene sustancias indeseables.

4. Antígenos

El uso de suplementos en pacientes con sospecha de alergias o intolerancias alimentarias puede ser causa de fallo de tratamiento. Muchos suplementos incluyen saborizantes que pueden interferir con el manejo dietético de estos problemas.

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Lowell Ackerman

5.1.1. In-Hospital Diagnostic Testing for Dermatology Patients

Abstract

The successful practice of dermatology relies on performing standardized testing as part of routine consultations. Certain techniques can be used to increase the sensitivity and specificity of such testing, but simply performing them regularly is often sufficient for developing expertise. The tests to be reviewed include skin scrapings, diascopy, trichography, Wood's lamp evaluation, dermatophyte cultures, cytology, and biopsies for histopathologic assessment.

Skin Scrapings

Skin scrapings are, or should be, the most common diagnostic test performed in veterinary dermatology. A dull scalpel blade or similar instrument is moistened with mineral oil and used to scrape away some of the epidermis, in which may reside a number of different parasites. After the scraping is obtained, the blade is then wiped onto a clean microscope slide and a microscope used to scan the slide for parasites. Parasites that might be recovered on skin scrapings include mites (Demodex, Sarcoptes, Cheyletiella, Otodectes, chiggers) and worms (Pelodera, hookworms, heartworms). The depth of the scraping and the locations to be scraped are determined by the specific parasite suspected. For example, Cheyletiella mites are surface dwellers, whereas scabies mites burrow into the stratum corneum. These require superficial scrapings using broad collections of surface debris. Most Demodex mites are follicular in orientation, so scrapings must be deep enough to recover these parasites, and this means scraping deep enough until there is oozing from the dermis (bleeding need not occur, and in fact, blood in the specimen makes it more difficult to find parasites.)

Diascopy

Diascopy is a technique that is used to determine whether redness in the skin is due to erythema associated with increased blood supply to the area, or whether the reaction is due to blood cells or pigment present extravascularly. It is accomplished simply by taking a microscope slide and pressing it firmly against the red-colored skin. With erythema, the redness disappears as the tissue blanches under the pressure of the slide. With blood cells or pigment present in the tissues, the color remains in the tissue.

Trichography

Direct microscopic examination of plucked hairs can be a very rewarding diagnostic experience. It is particularly helpful in cases of endocrinopathies, follicular dysplasias, telogen defluxion and traumatic hair loss. The hairs are evaluated for integrity of the shaft, stage (anagen, catagen, or telogen), and pigmentation. If most of the hairs have been sheared off, this is likely the result of licking, especially excessive grooming in cats. Damage to the shaft can be seen with several uncommon conditions, but dermatophytosis is the most common cause. It is imperative to understand the stages of the hair cycle when evaluating trichograms. Anagen is the growth phase, and the

hair bulb is typically hooked and broad. Catagen is a transitional stage, and the hair bulb is often straight and fist-like. Telogen is a resting stage, and the hair bulb is spear-like, with little there to hold it in place. Hairs transition between stages as part of normal hair growth, as well as a reflection of different disease processes. For example, with endocrinopathies (such as hypothyroidism) many hairs get arrested in the telogen stage, so when samples are collected, telogen hairs predominate. With hair loss involving many follicular diseases (such as demodicosis, bacterial folliculitis, and dermatophytosis), hair loss is noted even while many of the hairs are in anagen. Finally, many of the follicular dysplasias cause hairs to be stuck in the transitional (catagen) stage, so there become more prevalent when hairs are assessed with trichography.

Wood's Lamp (Ultraviolet Light, Black Light) Evaluation

A Wood's lamp uses ultraviolet light filtered through nickel oxide to cause some fungi to glow green in a darkened room. A tryptophan metabolite is the fluorescing material, not the fungi or spores themselves. This metabolite is only seen when the fungus is growing on hair shafts; it is noticeably absent on scale, claws or material growing on a culture plate. Only Microsproum canis fluoresces (also M. distortum, M. adouinii and T. schoenleinii in humans), and then only about 50% of the time. The diagnostic value of the Wood's lamp is limited to a screening test for Microsporum canis. Negative fluorescence does not rule out M. canis because fewer than 50% of these infections routinely fluoresce, and is not useful for the diagnosis of dermatophytosis caused by other organisms, including M. gypseum and Trichophyton mentagrophytes.

Direct Microscopic Examination for Dermatophytes

The microscopic examination of hairs to identify characteristic spores and hyphae is a relatively specialized test in animals and is not commonly performed in general veterinary practices. However, when viewed by an experienced individual, a diagnosis may be rendered about 60 to 70% of the time. The inexperienced may find it difficult to distinguish spores from pigment and hyphae from keratin. In animals, direct microscopic examination can be made with saline or mineral oil alone, potassium hydroxide (KOH), KOH-dimethyl sulfoxide (DMSO), chlorphenolac solution (chloral hydrate, phenol, lactic acid) or a solution of KOH, DMSO, and chlorazol fungal stain. This last stain is often helpful in practice because hyphae stain green against a gray background and thus are clearly visible.

Dermatophyte Culture

Two types of media are used routinely for dermatophyte culture: dermatophyte test medium (DTM) and Sabouraud's dextrose agar. Dermatophyte Test Medium (DTM) is just a form of Sabouraud's medium to which has been added an antibiotic (such as chloramphenicol), an antifungal capable of inhibiting fungal contaminants (such as cycloheximide), and a pH indicator (phenol red). Since dermatophytes tend to consume the protein in the medium first (whereas many non-dermatophytes consume the carbohydrate first), dermatophytes produce metabolites that often cause a pH change in the medium that in turn leads to a medium color change from amber to red. This is a useful indicator, but on its own is NOT diagnostic. A sample for fungal culture is obtained by selecting individual, representative hairs or scale. Broken hair shafts, hairs that fluoresce with a Wood's lamp, or scale from nails are satisfactory specimens. A culture positive for dermatophyte growth must show colony growth and color change from yellow to red simultaneously, which can occur from 1 to 14 days but usually occurs within the first 7 days. Everything that turns the medium red is not a dermatophyte. Some non-dermatophytes also preferentially utilize the protein in the medium first, and virtually all fungi will cause color change by switching to protein ingestion once they have utilized the carbohydrate in the medium. All DTM culture media that have fungal growth, whether contaminant or pathogen, change from yellow to red over time.

Yeast Prep

Cytology is a quick and easy way to demonstrate yeasts. Material swabbed, scraped, or collected with clear adhesive tape from the ear canals, skin surface, or interdigital area can be applied to a clean microscope slide and heat-fixed for a few seconds. Samples can also be collected by impression smear, acetate tape collection or skin scrapings. Yeasts are usually seen adequately with the high dry objective, but oil immersion may be necessary to clearly depict them.

Cytology

Cytology is the study of free cells from tissues. Samples may be obtained in various ways, including fine-needle aspiration, impression smear, exfoliative procedures, and swab techniques. Each technique is determined by the ultimate availability of the tissue being sampled. Fine-needle aspiration is used primarily to sample papulonodular and vesiculopustular lesions. Impression smears are used primarily for erosive-ulcerative lesions, or where there is active "ooze" that can be readily sampled. Exfoliative procedures are used to gently scrape cells from the surface of lesions and swabs are used for exudative lesions in areas where impressions cannot easily be achieved (e.g., ears, lip folds, etc.)

Biopsies

Biopsy for histopathologic examination (microscopic tissue evaluation) is especially important in dermatology, since the tissue to be sampled (skin) is so readily accessible. Biopsies are valuable diagnostic tools but should not be expected to tell the entire story. They reveal the changes only in a small region of skin surface at a particular point in time. Biopsies can be taken by excising the entire lesion, by incising into the lesion and removing a representative section or, most commonly, with biopsy punches. When biopsying tissue, it is important to preferentially sample primary lesions or early secondary lesions. The most profound changes, such as ulceration, fibrosis and

complete alopecia are less likely to be diagnostic. For this reason, it is best to submit several sections for evaluation, from a variety of different lesions and stages of development. Along with this it is important to submit to the pathologist a reasonable clinical history, along with clinical differential diagnoses.

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5.1.2. The Pattern Approach to Dermatologic Diagnosis

You might have wondered how dermatologists always seem to come up with some fantastic diagnosis or a comprehensive list of differential diagnoses after seeing a pet only briefly during a referral visit. These dermatologists then seem to know exactly which tests to run, and these tests are always ones that you didn't run before you referred the case. Many clinicians falsely believe that the diagnosis was reached because the dermatologist had seen cases just like theirs many times before. That may be true, but most of the time the reason is far more pragmatic; veterinary dermatologists quickly get into the habit of identifying cutaneous lesions properly and that provides critical information necessary to compile differential diagnoses and perform standard tests. There's nothing magical about it.

To do this effectively, you must know your primary and secondary lesions and be able to associate them with a predominant morphologic pattern. If you can't describe the morphologic pattern correctly, you can't diagnose the case correctly either in most cases.

To make a correct dermatologic diagnosis, we need to approach each case in three distinct steps. Of course, patient history and signalment are critical to narrowing down the diagnostic possibilities. However, this approach deals exclusively with the evidence you see during a clinical examination. If we follow these three steps, we'll arrive at a diagnosis in most cases.

Step 1	**Determine Pattern**
Step 2	**Formulate Differential Diagnostic List**
Step 3	**Perform diagnostic testing**

Step 1. Determine Pattern

Take your knowledge of primary and secondary lesions and carefully evaluate the animal to determine the predominant pattern. This is done by asking yourself a series of questions in a specific order. Remember to select the predominant pattern. A dog with profound scaling and 3 pustules doesn't have a vesiculopustular disorder; it has an exfoliative dermatosis. A cat with a large hairless mass on its back has a papulonodular pattern, not alopecia.

INITIAL ALGORITHM

1. Are there pigmentary changes ?
YES = Pigmented Lesions and Dermatoses
Red, White, Dark or Skin-colored?

If NO,

2. Are the lesions raised?
If YES,

a) Are the lesions fluid-filled?
YES = Vesiculopustular dermatoses
Primarily vesicular/bullous or pustular?

If NO,

b) Are the lesions solid and raised?
YES = Papulonodular dermatoses
Primarily nodules, plaques or vegetative lesions?

If NO,

3. Are the lesions flat, depressed, or only mildly elevated?
If YES,

a) Is hair loss prominent?
YES = Alopecic disorders
Primarily focal/multifocal, patchy, regional or generalized?

If NO,

b) Are breaks in epithelial integrity prominent?
YES = Erosive-ulcerative disorders
If NO,

c) Is scaling very prominent?
YES = Exfoliative dermatosis
Primarily patchy, regional or generalized?

If NO,

d) Is the skin palpably thickened?
YES = Indurated dermatoses
Primarily solid or turgid?

NO = Maculopapular dermatoses
Primarily macular or papular?

Lesions in Veterinary Dermatology

Lesion	Description	Pattern	1° or 2°
Macule	A circumscribed, flat non-palpable circumscribed area of change (discoloration) of the skin up to 1 cm in diameter	Maculopapular; pigmented	Primary
Patch	Macules greater than 1 cm	Maculopapular; pigmented	Primary
Papule	A circumscribed, elevated, superficial, solid lesion up to 1 cm in diameter	Maculopapular; papulonodular	Primary
Plaque	A circumscribed, elevated, flat-topped, superficial solid lesion > 1 cm. A papule that has enlarged in 2 dimensions	Papulonodular-plaque	Primary
Wheal	An edematous, transitory papule or plaque	Papulonodular-plaque; indurated-turgid	Primary
Nodule	A palpable, solid round or ellipsoidal lesion with depth. A papule that has enlarged in 3 dimensions	Papulonodular-nodule	Primary
Cyst	A closed epithelial-lined cavity containing fluid or semi-solid material	Vesiculopustular; Papulonodular-nodule	Primary
Vesicle	A circumscribed elevation of the skin, up to 1 cm in diameter, containing serous fluid	Vesiculopustular	Primary
Bulla	A vesicle > 1 cm in diameter	Vesiculopustular	Primary
Pustule	A circumscribed elevation of skin containing purulent fluid	Vesiculopustular	Primary
Petechia	A circumscribed deposit of blood or blood pigment up to 1 cm in diameter; the purplish discoloration noted is called purpura	Pigmented-red	Primary
Ecchymoses	A circumscribed deposit of blood or blood pigment > 1 cm in diameter; the purplish discoloration noted is called purpura	Pigmented-red	Primary
Scale	Shedding dead epidermal cells that may be dry or greasy	Exfoliative; maculopapular	Secondary
Epidermal collarette	A circular peeling rim of scale	Exfoliative; vesiculopustular	Secondary
Follicular plugging	Occlusion of the hair follicle (pore)	Exfoliative	Secondary
Erosion	An excavation in the skin limited to the epidermis and not breaking the integrity of the dermal-epidermal junction	Erosive-ulcerative	Secondary
Ulcer	An irregularly sized and shaped cavitation in the skin extending into the dermis	Erosive-ulcerative	Secondary
Crust	Variously colored collections of skin exudates	Erosive-ulcerative	Secondary
Excoriation	Abrasion of the skin, usually superficial and traumatic in origin	Erosive-ulcerative	Secondary
Fissure	A linear break in the skin, sharply defined with abrupt walls	Erosive-ulcerative	Secondary
Induration	Palpable thickening of the skin	Indurated	Secondary
Scar	A formation of connective tissue replacing tissue lost through injury or disease	Indurated	Secondary
Sclerosis	Hardening of the skin	Indurated	Secondary
Atrophy	Thinning or depression of the skin, due to reduction of underlying tissue	Indurated; maculopapular	Secondary
Lichenification	A diffuse area of thickening and scaling, with resultant increase in the skin lines and markings	Indurated; maculopapular	Secondary
Hyperpigmentation	Darkening of the skin	Pigmented-dark; Maculopapular	Secondary

Step 2: Formulate Differential Diagnostic List

Once you've pigeonholed the case into one of the eight major categories, you can create a comprehensive differential diagnostic list from the tables. This not only increases your awareness of possible dermatologic conditions, but it is extremely helpful to include the list when submitting samples for diagnostic testing, especially biopsies for histopathologic assessment.

The eight major patterns can be subdivided to render more specific differential diagnoses. The categories and subcategories are as follows:

1. Pigmented
    - Red
    - White (Depigmented)
    - Dark
    - Skin-colored
    - Other
2. Vesiculopustular
    - Vesicular
    - Pustular
3. Papulonodular
    - Nodular
    - Plaques
    - Vegetative
4. Alopecia
    - Focal/Multifocal
    - Patchy
    - Regional
    - Generalized
5. Erosive-ulcerative
6. Exfoliative
    - Patchy
    - Follicular
    - Regional
    - Generalized
7. Indurated
    - Turgid
    - Solid
8. Maculopapular
    - Macular
    - Papular

Differential Diagnosis: Pigmented

Category	Subcategory	Canine	Feline
Pigmented	**Red**	Drug Eruption Petechiae Purpura Vasculitis Contact Dermatoses Lupus Erythematosus Photodermatitis Erythema Multiforme Fold Pyoderma Pyotraumatic Dermatitis Histiocytoma Demodicosis Flushing Syndrome Lyme Borreliosis Hookworm dermatitis Acute eosinophilic dermatitis	Drug Eruption Petechiae Purpura Vasculitis Contact Dermatoses Lupus Erythematosus Photodermatitis Erythema Multiforme Eosinophilic Plaque Linear Granuloma
	White (Depigmented)	Lupus Erythematosus Albinism Uveodermatologic Syndrome Morphea Vitiligo Tyrosinase Deficiency	Lupus Erythematosus Albinism Waardenburg Syndrome Chediak-Higashi Syndrome Periocular Leukotrichia
	Dark	Basal-cell Tumor Melanoma Post-inflammatory change Hypothyroidism Hyperadrenocorticism GH-responsive Dermatosis Acanthosis Nigricans Adrenal sex-hormone dermatosis Lentigines Vascular Nevi Hemangioma/sarcoma Organoid Nevus Melanocytic Nevus Melanoderma and Alopecia	Basal-cell Tumor Melanoma Bowen's Disease Post-inflammatory change Feline viral plaques
Skin-colored		Epidermal Nevus Scar Papilloma Morphea Sebaceous-gland Hyperplasia Callus Sebaceous Nevus	Epidermal Nevus Scar
	Other	Dalmatian Bronzing Syndrome Acquired Aurotrichia Tyrosinase Deficiency Waardenburg-Klein Syndrome	Xanthomatosis Waardenburg-Klein Syndrome Chediak-Higashi Syndrome

Differential Diagnosis: Vesiculopustular

Category	Subcategory	Canine	Feline
Vesiculopustular/Vesicular		Pemphigus Pemphigoid Erythema Multiforme Dermatomyositis Epidermolysis Bullosa Dermatitis Herpetiformis Mucinosis Idiopathic Ulcerative Dermatosis Vesicular LE	Pemphigus Pemphigoid Lupus Erythematosus Epidermolysis Bullosa Cat Pox Herpesvirus infection
	Pustular	Demodicosis Bacterial Pyoderma Dermatophytosis Subcorneal Pustular Dermatosis Sterile Eosinophilic Pustulosis Lupus Erythematosus Acne Linear IgA Dermatosis Adverse food reactions Pemphigus Collaretting syndrome	Demodicosis Bacterial Pyoderma Dermatophytosis Abscess Acne Lupus Erythematosus FIV Infection Adverse food reactions

Differential Diagnosis: Papulonodular

Category	Subcategory	Canine	Feline
Papulonodular	**Nodular**		
		Parasitic	Abscess
		Deep Pyoderma	Acne
		Atypical Pyoderma	Atypical Pyoderma
		Dermatophytosis	Dermatophytosis
		Intermediate Mycoses	Intermediate Mycoses
		Deep Mycoses	Deep Mycoses
		Lupus Profundus	Parasitoses
		Neoplastic	Neoplastic
		Dermoid Cyst	Dermoid Cyst
		Nodular Panniculitis	Nodular Panniculitis
		Juvenile Cellulitis	Lupus Profundus
		Mucinosis	Xanthoma
		Eosinophilic Granuloma	Eosinophilic Granuloma
		Sebaceous Adenitis	Leprosy
		Sterile Pyogranuloma	Opportunistic mycobacteria
		Opportunistic mycobacteria	
		Acral pruritic nodule	
		Calcinosis Circumscripta	
		Nodular fasciitis	
		Protothecosis	
		Dracunculiasis	
	Plaques		
		Dermatophytosis	Dermatophytosis
		Urticaria	Urticaria
		Lymphoma	Lymphoma
		Bacterial Hypersensitivity	Sporotrichosis
		Lupus Profundus	Eosinophilic Plaque
		Viral Papillomatosis	Mast-Cell Tumor
		Calcinosis Cutis	Linear Granuloma
		Calcinosis Circumscripta	Vitamin E Deficiency
		Histiocytoma	Mucopolysaccharidosis
		Histiocytosis	Xanthomatosis
		Keratoses	Tumoral Calcinosis
		Nevi	Nevi
		Lichenoid Dermatoses	Lichenoid Dermatoses
		Mucinosis	Erythema Multiforme
		Erythema Multiforme	Papillomavirus infection
		Acanthosis Nigricans	Perforating Dermatitis
		Dermatitis Herpetiformis	Feline viral plaques
		Urticaria Pigmentosa	
		Acral lick dermatitis	
		Malassezia dermatitis	
		Acute eosinophilic dermatitis	
	Vegetative	Mast-cell Tumor	Mast-Cell Tumor
		Cutaneous Papilloma	Squamous-cell Carcinoma
		Fibroma	Fibroma
		Nevi	Nevi
		Sebaceous-gland Hyperplasia	
		Transmissible Venereal Tumor	
		Pemphigus Vegetans	

Category	Subcategory	Differential Diagnosis: Alopecia Canine	Feline
Alopecia	**Focal/Multifocal**	Demodicosis Bacterial Pyoderma Dermatophytosis Alopecia Areata Cutaneous Asthenia Traction Alopecia Morphea Injection Site Reaction Cicatricial Alopecia	Demodicosis Bacterial Pyoderma Dermatophytosis Alopecia Areata Cutaneous Asthenia Traction Alopecia Injection Site Reaction Cicatricial Alopecia
	Patchy	Demodicosis Cheyletiellosis Lice Infestation Dermatophytosis Bacterial pyoderma Lupus Erythematosus Telogen Defluxion Protein Deficiency Drug Eruption Sebaceous Adenitis Bronzing Syndrome Color-Mutant Alopecia Spiculosis Leishmaniasis Familial Benign Pemphigus Mucinous mural folliculitis Pseudopelade	Demodicosis Cheyletiellosis Lice Infestation Dermatophytosis Drug Eruption Lupus Erythematosus Telogen Defluxion Hyperadrenocorticism Pseudopelade
	Regional	Discoid Lupus Erythematosus Hypothyroidism Hyperadrenocorticism Growth Hormone-responsive Adrenal Sex-hormone Dermatosis Seasonal Flank Alopecia Hyperestrogenism Hypoestrogenism Pattern Baldness Testicular Neoplasia Dermatomyositis Follicular Dysplasia Toxicity (e.g., Thallium) Post-clipping alopecia Pinnal alopecia Benign Familiar Chronic Pemphigus Melanoderma and Alopecia Waterline Disease Ischemic folliculopathy	Discoid Lupus Endocrine Alopecia Hyperadrenocorticism Psychogenic Alopecia Post-clipping alopecia Pinnal alopecia Preauricular alopecia Symmetrical Alopecia ParaneoplasticAlopecia
	Generalized	Dermatophytosis Lupus Erythematosus Drug Eruption Demodicosis Hypotrichosis Telogen defluxion Post-clipping alopecia	Dermatophytosis Lupus Erythematosus Drug Eruption Alopecia Universalis Hypotrichosis Telogen defluxion

Differential Diagnosis: Erosive-Ulcerative

Category	Subcategory	Canine	Feline
Erosive-Ulcerative		Fleas	Fleas
		Demodicosis	Demodicosis
		Sarcoptic Mange	Notoedric Mange
		Skin-Fold Pyoderma	Superficial Pyoderma
		Pyotraumatic Dermatitis	Systemic Mycoses
		Perianal Fistulae	Cat Pox Infection
		Bacterial Granuloma	Bacterial Granuloma
		Mycetoma	Mycetoma
		Mycobacteriosis	Mycobacteriosis
		Pemphigus	Pemphigus
		Pemphigoid	Pemphigoid
		Cutaneous Vasculitis	Cutaneous Vasculitis
		Toxic Epidermal Necrolysis	Toxic Epidermal Necrolysis
		Drug Eruption	Drug Eruption
		Lupus Erythematosus	Lupus Erythematosus
		Lupoid Dermatosis	FIV Infection
		Vesiculopustular dermatoses	Indolent Ulcer
		Leishmaniasis	Squamous-cell Carcinoma
		Thallium toxicosis	Bowen's Disease
		Cutaneous asthenia	Sporotrichosis
		Epitheliogenesis imperfecta	Hyperadrenocorticism
		Ectodermal defect	Vesiculopustular dermatoses
		Burn	Burn
		Contact eruption	Contact eruption
		Septicemia/Toxemia	Septicemia/Toxemia
		Dermatomyositis	Cutaneous asthenia
		Erythema multiforme major	Erythema multiforme major
		Cutaneous T-cell lymphoma	Ectodermal defect
		Familial Benign Pemphigus	Dermatophilosis
		Familial Vasculopathy	Epidermolysis Bullosa
		Dermatophilosis	Acquired Skin Fragility
		Candidiasis	Herpesvirus infection
		Metabolic Dermatoses	Idiopathic neck ulcer
		Epidermolysis Bullosa	
		Idiopathic Erosive Dermatosis	
		Acrodermatitis	
		Ulcerative Dermatosis	
		Acute Neutrophilic Dermatitis	
		Acute Eosinophilic Dermatitis	

Differential Diagnosis: Exfoliative

Category	Subcategory	Canine	Feline
Exfoliative	**Patchy**	Ectoparasitism Dermatophytosis Drug Eruption Pemphigus Foliaceus Fatty Acid Deficiency T-cell Lymphoma Pagetoid Reticulosis Sjøgren's Syndrome Hyperestrogenism Vit-A responsive Derm. Sebaceous Adenitis Generic Dog Food Disease Subcorneal Pustular Dermatosis Chronic Maculopapular Dermatoses Parapsoriasis Adverse Food Reactions Hypothyroidism Lupoid dermatosis Leishmaniasis	Ectoparasitism Dermatophytosis Drug Eruption Pemphigus Foliaceus Fatty Acid Deficiency Protein Deficiency Vitamin-A Deficiency Vitamin-E Deficiency Biotin Deficiency Lynxacariasis Adverse Food Reactions Perforating Dermatitis
	Follicular	Follicular keratosis Sebaceous adenitis Acne Comedo syndrome Milia Bacterial folliculitis Demodicosis Dermatophytosis Vitamin A-responsive	Acne Comedones Milia Folliculitis Demodicosis Dermatophytosis Sebaceous adenitis Pseudopelade Thymoma dermatitis
	Regional	Pemphigus Foliaceus Pemphigus Erythematosus Discoid Lupus Erythematosus Hypothyroidism Zinc-responsive Dermatosis Tyrosinemia Nasodigital Hyperkeratosis Leishmaniasis Malasseziasis	Pemphigus Foliaceus Pemphigus Erythematosus Discoid Lupus Cheyletiellosis Thymoma dermatitis
	Generalized	Dermatophytosis Drug Eruption Systemic Lupus Pemphigus Foliaceus Keratinization Disorders Demodicosis Hypothyroidism Vitamin E Deficiency Ichthyosis T-cell Lymphoma Metabolic Disorders Leishmaniasis Graft-versus-Host Disease	Dermatophytosis Drug Eruption Systemic Lupus Pemphigus Foliaceus Keratinization Disorders Cheyletiellosis Hypereosinophilic Syndrome Lynxacariasis T-cell Lymphoma Metabolic Disorders Paraneoplastic syndrome

Differential Diagnosis: Indurated

Category	Subcategory	Canine	Feline
Indurated	**Turgid**	Urticaria	Urticaria
		Angioedema	Angioedema
		Myxedema	GH-secreting Tumor
		Juvenile Cellulitis	Mucopolysaccharidosis
		Mucinosis	Relapsing Polychondritis
		Nephrotic Syndrome	Plasma Cell Pododermatitis
		Urticaria Pigmentosa	
		Hookworm dermatitis	
		Acute eosinophilic dermatitis	
	Solid	Cellulitis	Cellulitis
		Bacterial Granuloma	Bacterial Granuloma
		Fungal Granuloma	Fungal Granuloma
		Calcinosis Cutis	Calcinosis Cutis
		Tumoral Calcinosis	Scar
		Scar	Neoplasia
		Neoplasia	Amyloidosis
		Amyloidosis	Intermediate Mycosis
		Scleroderma	Chronic Maculopapular
		Chronic Maculopapular Derm.	
		Sebaceous Adenitis	

Differential Diagnosis: Maculopapular

Category	Subcategory	Canine	Feline
Maculopapular	**Macular**		
		Allergic Inhalant Dermatitis	Allergic Inhalant Dermatitis
		Food Allergy	Food Allergy
		Allergic Contact Dermatitis	Allergic Contact Dermatitis
		Irritant Contact Dermatitis Irritant	Contact Dermatitis
		Drug Eruption	Drug Eruption
		Bacterial pyoderma	Endo/Ecto Parasitism
		Erythema Multiforme	Erythema Multiforme
		Lupus Erythematosus	Lupus Erythematosus
		Alopecia Areata	Alopecia Areata
		Endo/Ecto parasitism	
		Acanthosis Nigricans	
		Acute eosinophilic dermatitis	
Papular/Papulocrustous			
		Parasitic Dermatoses	Miliary Dermatitis
		Vit A-responsive dermatosis	Parasitic Dermatoses
		Bacterial Folliculitis	Bacterial Folliculitis
		Drug Eruption	Drug Eruption
		Food Allergy	Food Allergy
		Dermatophytosis	Dermatophytosis
		Comedones/Acne	Pemphigus foliaceus
		Pemphigus foliaceus	Comedones/Acne
		Erythema Multiforme	Erythema Multiforme
		Hormonal Hypersensitivity	Hypereosinophilic Syndrome
		Dermatitis Herpetiformis	

Step 3: Perform Diagnostic Testing

One now has not only a pattern diagnosis, but also a list of potential differential diagnoses. At this time, the considerations can be prioritized on the basis of history, specific clinical presentation, breed predisposition and a variety of other clues. If one is not comfortable taking that next step, in information below can be used to create a minimum database; this will uncover the most common conditions with that presentation. If the minimum data base doesn't suggest a diagnosis, the next step listed in the charts proposes additional testing.

Classification	Minimum Data Base	Next Step
Pigmented	**Histopathology**	**As per biopsies**
Vesiculopustular	**Skin scrapings** **Cytology**	**Dietary/parasite trials** **Histopathology** **Cultures**
Papulonodular	**Skin scrapings** **Cytology (e.g. fine needle aspirate)** **Histopathology** **CBC/Biochemistry**	**As indicated by MDB tests** **Cultures, blood tests, etc.**
Alopecic		
Focal	**Skin scrapings** **DTM** **Trichogram**	**Histopathology**
Widespread	**Skin scrapings** **DTM** **Trichogram** **CBC** **Biochemistries** **Urinalysis**	**Endocrine profiles** **Histopathology**
Erosive-Ulcerative	**Skin scrapings** **Cytology (e.g., impression smear)**	**Histopathology**
Exfoliative	**Skin scrapings** **CBC** **Biochemistries** **Urinalysis** **Fungal culture**	**Histopathology** **Endocrine profile**
Indurated		
Turgid	**CBC** **Biochemistry** **Urinalysis** **Fecal**	**Histopathology**
Solid	**Cytology** **Histopathology**	**CBC** **Biochemistries** **Cultures**
Maculopapular	**Skin Scrapings** **Cytology** **Fecal(s)** **CBC**	**Dietary Trial** **Parasite-control trial** **Allergy Testing** **Cultures** **Histopathology**

Recommended Reading:

Ackerman, L: Atlas of Small Animal Dermatology, Inter-Medica, 2008

Hnilica, KA; Patterson, AP: Small Animal Dermatology. A color atlas and therapeutic guide, 4th Edition, Elsevier, 2016

Miller Jr., WH; Griffin, C; Campbell, S: Muller & Kirk's Small Animal Dermatology, 7th Edition. Saunders, 2012.

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Jackson, H; Marsella, R: BSAVA Manual of Canine and Feline Dermatology. British Small Animal Veterinary Association, 2021.

Logas, D: Diagnostics and therapy in veterinary dermatology. Wiley-Blackwell, 2022.

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5.1.3. Understanding and Managing the Allergic Pet

Canine atopic dermatitis is one of the most common canine skin disorders treated by veterinarians and the best outcomes are typically associated with early recognition and control when there is the best chance of avoiding long-term complications such as infections, skin barrier disruption, and chronic inflammatory changes in the skin. Atopic dermatitis starts in a genetically predisposed dog that may have a defective skin barrier that allows for the percutaneous overabsorption of environmental allergens. Less commonly, allergens also are absorbed through inhalation and ingestion. The result of this allergen overabsorption is an aberrant T-cell response in which cytokines are produced that promote inflammation and itch. Managing patients with atopic dermatitis requires a proactive approach to manage the underlying pathogenesis, control acute flares, and hopefully prevent chronic inflammatory changes from occurring that would otherwise complicate the management of this condition over the life of the pet. This is preferred over a more reactive treatment plan focusing on repeated courses of intensive short-term treatment of active inflammation and infection. Treatment relies on controlling the itch and inflammatory aspects of the disease, helping to repair barrier function, and down-regulating the abnormal immune response with appropriate medications and/or immunotherapy.

Prevention:

There are no foolproof methods for preventing atopic dermatitis. The risk can be minimized by selecting puppies with no family history of atopic dermatitis, especially in high-risk breeds. It is theoretically possible that supplementation with probiotics and/or omega-3fatty acids rich in eicosapentaenoic acid could be of some benefit when provided to pregnant bitches and puppies of high-risk breeds, but definitive evidence is lacking.

Detection:

Canine atopic dermatitis is a clinical diagnosis and one often made by exclusion of other pruritic dermatoses such as parasitism, adverse food reactions, and associated bacterial (often Staphylococcus pseudintermedius) and yeast (Malassezia pachydermatis) overgrowth/infection. Allergy testing, either by intradermal or serum evaluation, does not confirm a diagnosis but may be useful when selecting allergens for immunotherapy. Since atopic dermatitis is considered a heritable trait, owners of at-risk breeds should be appropriately counseled to recognize early signs of the disorder, such as licking and chewing at the feet, face rubbing, pinnal redness, and rashes in the inguinal and axillary areas. Pet owners should also be advised that pet insurance can be an excellent hedge against the costs of long-term allergy management. Policies should be acquired at the earliest possible opportunity before any allergy condition could be considered pre-existing and excluded from coverage. The policy should cover chronic allergy care since atopic dermatitis is considered a lifelong condition.

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Treatment:

Since most allergic dogs will require lifelong care, it is important that they be re-evaluated often and that year-round parasite control be in effect, secondary microbial infections be quickly managed, and that surface barrier function be restored or at least managed. Glucocorticoids can be tolerated for acute short-term therapy, but are not preferred for long-term itch control due to their relatively high incidence of side effects. The sooner the pet is on appropriate long-term therapy, the better the chance for successful lifelong control of itch, inflammation and secondary infections. Immunotherapy is best considered early in the treatment process when the prospects for successful immune regulation and prevention of disease progression are best, rather than leaving this as an option for the late-stage or end-stage patient.

Treatment for atopic dermatitis in the dog might include the following (see Table 1):

• Oclacitinib, a selective Janus Kinase inhibitor, is fast acting (typically within the first day) and can be used for the treatment of acute or chronic inflammation and pruritus. It works by inhibiting cytokine-mediated inflammatory reactions in the skin and itchspecific nerve signal pathways.

• Lokivetmab is an injectable agent that targets and neutralizes IL-31, a key itchinginducing cytokine. It's the first monoclonal antibody for chronic canine allergic and atopic dermatitis. For maintenance, it is typically given as an in-office injection every 4-8 weeks.

• Corticosteroids (glucocorticoids) are suitable for very short-term "crisis-busting" treatment as they provide good management of acute flares of pruritus, but have many health risks if used for long-term treatment. Options include prednisone or prednisolone or methylprednisolone (initially daily, in divided doses if needed, then tapering to alternate days), or a corticosteroid/antihistamine combination (which may reduce the corticosteroid component needed to achieve relief). Occasionally an initiating injection of dexamethasone sodium phosphate is contemplated, if near-immediate relief is needed. Topical corticosteroids can also be helpful for localized anti-itch treatment, starting with more potent products such as triamcinolone spray on a tapering frequency, then converting to a safer hydrocortisone spray for long-term therapy of known problem areas.

• Cyclosporine is an immunomodulating drug with immunosuppressive effects that is used in the treatment of canine atopic dermatitis. It is typically administered daily at first, then tapered by decreasing the frequency until a minimum frequency is reached that maintains the desired therapeutic effect. Cyclosporine is not preferred for acute pruritus or shortterm therapy since it may take up to several days or even weeks to achieve the desired effect.

• Allergen-specific immunotherapy (hyposensitization) is a method of gradually introducing allergens to which there is sensitivity in a way that may down-regulate the allergic response and raise the allergic threshold. It is the only therapy we have that can actually curb disease progression over time and lessen the need for long-term medications. Because it may take months or years for improvement to be seen, it is best to consider immunotherapy early in the course of the disease rather than as a last resort therapy in an end-stage allergic patient. Immunotherapy can be administered as subcutaneous injections or sublingual drops.

• Bathing and topical therapy are important components of allergy management for controlling inflammation, removing allergens from the skin and haircoat, helping repair barrier function, and decreasing microbial overgrowth on the skin surface. Bathing in cool water is soothing to the skin, and antipruritic shampoo ingredients include colloidal oatmeal, antihistamines, corticosteroids, and topical anesthetics (e.g., pramoxine). Topical therapy with fatty acids and ceramide precursors (e.g., phytosphingosine, Noctadecanoylphytosphingosine) may help restore normal barrier function, although they are rarely sufficient on their own in controlling the condition. Periodic use of antiseptic agents (such as chlorhexidine, ethyl lactate, benzoyl peroxide, micronized silver, and even dilute sodium hypochlorite) can help control bacteria and yeast on the skin surface, which can act as major flare factors and worsen pruritus. Most allergic dogs will require therapeutic bathing several times a week initially to get the itch and infection under control. Bathing can then be tapered to an interval that is medically appropriate yet manageable for owners, such as once or twice weekly.

• Omega-3 fatty acids, specifically eicosapentaenoic acid (EPA), can be beneficial for atopic dogs by helping to restore the skin barrier and reducing inflammation in the skin. They are used more as adjunctive chronic therapy since there is a relatively low success rate when used alone, and it can take up to 2-3 months to see clinical improvement. The relatively high levels needed for benefit are sometimes not well tolerated by dogs and may cause loose stools, flatulence and/or halitosis. Management can be achieved through oral administration of appropriate supplements, or by feeding diets that have been formulated to be rich in this particular fatty acid.

• Antihistamines are typically safe with few side-effects (other than sedation with some products), but have a low success rate in treating itch in dogs with atopic dermatitis, and most represent extra-label drug use. The most commonly used products include hydroxyzine, cetirizine, clemastine, chlorpheniramine, and diphenhydramine. Given the low rates of efficacy and compliance, antihistamines are often not suitable for acute or long-term therapy. If used at all, they should be combined with other more effective therapies in mild cases of atopic dermatitis.

Modality	Acute Treatment	Chronic Treatment
Oclacitinib	✓	✓
Lokivetmab	✓	✓
Glucocorticoids	✓	
Cyclosporine		✓
Immunotherapy		✓
Bathing & Topical Therapy	✓	✓
Omega-3 Fatty Acids		✓
Antihistamines	?	?

Table 1: Appropriateness of various therapeutic modalities for the acute and chronic management of canine atopic dermatitis

Comments:

Atopic dermatitis is a chronic lifelong inflammatory disorder which cannot be cured, but can be safely controlled long term with appropriate vigilance. While most cases can be satisfactorily managed with the treatments mentioned here, it is often worthwhile to involve a veterinary dermatologist early in the process, especially if a pet is not responding to treatment as anticipated, and before owners become emotionally and financially exhausted.

Recommended Reading

Cosgrove, S; Wren, JA; Cleaver, DM; et al: A blinded, randomized, placebo-controlled trial of the efficacy and safety of the Janus kinase inhibitor oclacitinib (Apoquel®) in client-owned dogs with atopic dermatitis. Vet Dermatol, 2013; 24(6):587-e142.

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Pucheu-Haston, CM; Bizikova, P; Marsella, R; et al: Review: Lymphocytes, cytokines, chemokines and the T-helper 1 – T-helper 2 balance in canine atopic dermatitis. Vet Dermatol, 2015; 26: 124-132.

5.1.4. The Pyoderma Prescription

The surface of the skin is not sterile, and it plays host to a variety of resident, transient, opportunistic and pathogenic bacteria. It is important to understand how this occurs, since pyoderma (bacterial infection of the skin) is commonplace, and is often a frustrating condition in veterinary practice.

Resident bacteria live and multiply on normal skin. They are located on the skin surface, and in the most superficial aspects of the hair follicles, and discourage opportunistic infections through effective competition.

Staphylococcus pseudintermedius is the most important organism in pyoderma of dogs, and healthy dogs are a frequent carrier of the organism. Dogs also carry the organism in the following locations: anus; genital tract, buccal mucosae, and; conjunctivae. Most dogs develop their own genetically unique strains of Staphylococcus, present in carriage sites and in pyoderma lesions, suggesting that dogs are not routinely colonized by the staphylococci of other dogs. These staphylococci can also be passed from dam to pup through the milk. If this organism is present on many normal dogs and lives as a commensal organism in most cases, then the logical question should be asked as to why it is responsible for perhaps 90% of canine pyodermas. The answer is that since the organism is not overly pathogenic, in must be host factors that allow the organism to take hold and do its damage.

To cause pyoderma, bacteria must be able to adhere to keratinocytes, take advantage of nutrients on the skin, compete effectively with resident organisms and overcome host defense mechanisms. Bacteria can enter from several different routes, but most penetrate through the hair follicles. There are then a number of host factors to overcome, in order for the bacteria to cause problems.

Any process that disturbs the skin's barrier function will predispose to pyoderma. This includes any abrasion that removes surface barrier functions, any inflammatory process that causes epidermal disorganization, any process that affects local temperature and relative humidity on the skin, or any proliferative epidermal disease that results in keratinocyte disarray. Finally, any underlying disease that impairs immune function can predispose to pyoderma. Therefore, it is most important to investigate underlying diseases that predispose to pyoderma, rather than just responding with antibiotics.

Pyodermas are typically characterized by their depth (superficial, intermediate, deep), or by their particular characteristics. Superficial (surface) pyodermas involve the epidermis and the outermost aspects of the hair follicles. Examples include pyotraumatic dermatitis ("hot spots"), fold pyodermas, and juvenile pustular dermatitis (often incorrectly referred to as impetigo, but much different than impetigo in children). These conditions are so superficial that systemic antibiotics are often not needed. However, it is critical that the predisposing causes be addressed, and that the bacteria are managed, at least topically.

Intermediate pyodermas involve deeper penetration of bacteria, and most often result in folliculitis and perifolliculitis. These most commonly result from secondary reactions to allergies, parasites, keratinization disorders, and metabolic disorders.

Deep pyodermas involve yet deeper structures in the dermis and panniculus, including furunculosis (rupture of hair follicles with their contents discharged into the surrounding tissues), cellulitis, abscesses, and fistulating diseases (including German shepherd dog pyoderma, mycobacteriosis, etc.). These diseases either reflect a more exaggerated reaction to the causes of intermediate pyodermas, a major breach in immune function, or infiltration of an organism more pathogenic than Staphylococcus pseudintermedius.

Regional pyodermas are described based on the part of the body affected, and while they still obey the clinical doctrines of superficial, intermediate and deep pyodermas, they do have their own therapeutic approaches.

Staphylococcus schleiferi (Staphylococcus schleiferi schleiferi and Staphylococcus schleiferi coagulans) is also a significant pathogen in dogs, and many isolates are methicillin resistant and some are fluoroquinolone resistant. Opportunistic infections are not only capable of causing disease in dogs, but also transferring multi-drug resistance. It is therefore important to properly assess and manage cases, so as not to encourage multidrug resistance to important antibiotics.

Superficial (Surface) Pyoderma

Cytologic evaluation of the pustular contents reveals neutrophils and phagocytosed cocci. Further confirmation is rarely needed, but histopathologic assessment often demonstrates a subcorneal pustular dermatitis. Treatment can almost always be accomplished with topical antiseptic agents, and systemic antibiotics are only required if the condition fails to respond to topics alone. Rarely, a short 7-10 day course of antibiotics will resolve the condition.

Intermediate Pyoderma (Folliculitis, Perifolliculitis, and Furunculosis)

These intermediate pyodermas are common in the dog and relatively rare in the cat. The problem starts as a maculopapular to papulocrustous eruption and the distribution depends somewhat on the underlying cause of the problem. There are a variety of underlying causes for the problem, including allergies, endocrinopathies, keratinization disorders and virtually any other process that disturbs the body’s ability to control surface infections.

Clinically, the process starts with macules and papules that evolve into pustules, although the pustular stage may not always be evident at the time of examination. In time, there is an outward peeling rim of scale (epidermal collarette), with central hyperpigmentation, sometimes collectively referred to as a target lesion. When hair follicles rupture and discharge there contents into the underlying tissues, marked inflammation ensues, and the process is known as furunculosis.

At this stage, culture may not initially be required, and empirical selection of antibiotics can be based on the presumption of infection with Staphylococcus pseudintermedius. Beta-lactam antibiotics (such as cefovecin, cefpodoxime, amoxicillin-clavulanate, cefadroxil, and cephalexin) should be considered first-line therapies for empirical therapy; fluoroquinolones should be reserved for use on the basis of culture and sensitivity. Lesions should be cultured in recurrent, deep, or non-responsive pyodermas. Antibiotics alone, however, are not sufficient for long-term management, as the underlying problem must be identified and corrected or the problem is likely to recur once antibiotic therapy has been discontinued. Twice weekly bathing with an antiseptic shampoo and conditioner is often very helpful in keeping surface bacterial numbers under control. Eventually the bathing interval can be weaned to once every 1-2 weeks, if the underlying problems have been resolved. If the underlying problem cannot be identified, pulse therapy with antibiotics and even staphylococcal bacterin immunotherapy can be considered. In this instance, bacterial cultures are indicated, and should be performed on minced tissue collected by biopsy.

Deep Pyoderma

Deep pyoderma occurs when the infection involves the deep dermis, subcutis, and other underlying tissues. It may occur as an extension of furunculosis, secondary to puncture wounds and other methods of microbial inoculation, and as independent entities.

Cellulitis refers to a deep bacterial infection that spreads between tissue planes and fails to localize as an abscess. Abscesses normally result from traumatic inoculation of microbes into the dermis or panniculus. They may appear as firm to fluctuant subcutaneous nodules that may exude pus and may be painful to the touch. With deep pyodermas, it is not unusual to see surface ulceration, and even fistulous tracts.

With deep pyodermas, it is imperative to determine underlying cause(s). In some cases, such as when the pyoderma has resulted from bite wounds or other trauma, the cause is apparent. In other instances, the condition could result secondary to immune compromise, or metabolic diseases. A search for the underlying cause is necessary or the condition is unlikely to ever be completely resolved. Culture is indicated in most cases of deep pyoderma, and the best sample is taken by biopsy, with minced tissue prepared for microbe identification and susceptibility testing.

Antibacterial therapy is best attempted following culture, since the treatment period is often prolonged, perhaps months. Antibacterial resistance is common. In the case of abscesses, surgical drainage, hydrotherapy, and the use of hot compresses should ideally precede antibacterial therapy.

Biofilms

Biofilms are complex communities of bacteria embedded within a slime matrix that adheres to tissue surfaces. This matrix allows bacteria to shield themselves from the host immune response and antimicrobial therapy.

Biofilms are more often associated with deep and persistent infections and Staphylococcus pseudintermedius is a potential biofilm producer. Pseudomonas aeruginosa can be a biofilm producer in otitis cases.

Biofilm-associated bacteria may not be readily accessible for samples obtained using standard culture techniques and growing them on standard culture media can be challenging.

Treating biofilms can be complicated by the fact that antimicrobial sensitivity testing may only test planktonic (non-biofilm-forming) bacteria, and antibiotic doses for biofilm can be thousands of times higher than that needed to kill planktonic bacteria of the same strain. Thus, antimicrobial therapy may resolve clinical signs of infection temporarily, only to have the problem recur after cessation of therapy. The best treatment for biofilmassociated infections is debridement and tissue irrigation preceding antimicrobial therapy.

Non-Pyodermas

It is also important to remember that not everything that looks like a pyoderma is a primary bacterial disease. For example, pemphigus foliaceus may present with pustules and with crusting, and is often initially confused with a pyoderma. The same can be said for zinc-responsive dermatosis, dermatophytosis, demodicosis, sterile eosinophilic pustulosis, juvenile cellulitis, and sometimes even cutaneous T-cell lymphoma.

Recommended Reading:

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5.1.5. Common Mistakes to Avoid in Achieving Long-term Success with Dermatology Patients

Not appreciating appropriate clinical timelines:

While most veterinarians focus on the needs of the client and pet at the time of a specific office visit, it is important to realize that for many dermatologic conditions opportunities exist on a time continuum for intervention and client education. In some instances, the first discussions with the owner may take place long before there is even a skin problem to treat. This might also prompt a discussion of the likely costs associated with long-term management, so the owners might consider their options for risk management strategies, including pet insurance.

Appreciating appropriate timelines also helps in treatment selection. When a two-yearold atopic patient presents with pruritus, the first tendency might be to treat with glucocorticoids (corticosteroids) to achieve short-term relief. However, when you reflect on how that same patient might look at 8 or 10 years of age with such treatment, there is often a different perspective on what the long-term liabilities are for such short-term gains, and alternative more targeted treatments might be selected given the need for sensible long-term solutions.

It's also important to schedule effectively. No one can solve a complicated dermatology case in a single visit, regardless of the appointment's duration. You can address the client's primary concerns, but be clear with the client that the pet's problems will likely not be solved with that single visit. The key to successful dermatology management is scheduling frequent re-evaluation appointments. The success of this strategy is predicated on sharing the action plan with pet owners, so they can understand and appreciate what is expected to be accomplished, and according to what timeline.

We must also be aware that even well-accepted theories of cause and effect can and do change over time, and that we must be prepared to change with them. For example, we once thought of atopic dermatitis as a classic Type I hypersensitivity reaction primarily driven by mast-cell dynamics in response to inhaled allergens. We now know that the major allergen presentation occurs across the skin surface via Langerhans cells and that inflammatory mediators such as interleukin-31 (IL-31) and other pruritogenic cytokines are most responsible for the scratching that we see in our

allergic patients. This helps explain why antihistamines are often of limited benefit, and points the way to more targeted therapies that can and should be considered.

Not appropriately framing client expectations:

Many pet skin problems cannot be cured, regardless of the medication selected, so set realistic expectations with clients early in the process. Fortunately, control of the condition and a better quality of life for the pet can be attained with appropriate management. For the pet with atopic dermatitis, you might explain to clients that this is no different than addressing other incurable but manageable medical conditions such as diabetes mellitus or degenerative joint disease (osteoarthritis). We can often achieve excellent long-term control with ongoing treatment and close monitoring, but it’s unlikely that we’ll be able to “fix” the underlying problem. Usually once owners appreciate that the goal is control rather than cure, their expectations tend to be more reasonable and realistic.

Not viewing the situation in terms of Quality of Life:

Veterinarians might sometimes view dermatologic problems as minor medical issues because often the pet's life isn't at risk, but pet owners may not share that same perspective. Pet owners may not know what a diseased liver looks like, but they are certainly aware when their pet keeps them awake at night while scratching, has bald patches in its fur, or harbours offensive odours, rashes, parasites, or skin colour changes associated with a variety of skin diseases. There might also be a certain amount of guilt associated with friends and relatives asking why they have not done a better job of treating their pet's skin ailments, even when the pet owner has done everything they have been instructed to do. Veterinarians and hospital staff would do well to consider what pet owners find stressful, in addition to what might be causing the pet discomfort.

Not following evidence-based standards of care:

Most pet owners don't want their pets to serve as learning tools, so they appreciate knowing that veterinarians know what they are treating, the chances of success, what problems might be encountered, and the alternatives available should the need arise. Pet owners also appreciate that such knowledge is evidence-based and reflects established expertise, even if not direct experience. They find comfort in consistent messaging from hospital staff, specialists, and even internet resources.

Standards of care should be created by hospitals to provide this assurance, and to guarantee the best care to all patients and all pet owners, regardless of the individual

experience and expertise of the clinician currently treating that pet. Protocols are often used for standardized approaches to basic preventive and acute care (e.g., parasite control, treatment of sarcoptic mange) while care pathways represent a model for approaching problems that might require long-term or even lifelong care (e.g., atopic dermatitis, demodicosis, pemphigus foliaceus, etc.).

When creating standards of care for your hospital, remember to build into the model specific points at which referral to a specialist should take place. The best referral happens as part of a well-conceived plan, not as an afterthought. It helps to prepare clients early on that the first course of treatment may not resolve the problem, and if so, that you may try other options or recommend the assistance of a specialist. Clients will appreciate your efforts to inform them now, rather than when they are frustrated, upset, and depleted of funds.

Underestimating the role of compliance:

When it comes to managing many skin conditions, client compliance is an issue that must be considered.

Before dispensing any medications to owners, be realistic in your expectations for what can be reasonably accomplished at home. Better yet, have that exact conversation with owners, including asking open-ended questions of their previous experiences, so you can plan your treatment accordingly. Owners are more likely to be compliant treating clinical signs they can judge (e.g., control of scratching or pain), and can be less compliant with things that aren't as obvious, such as administering antibiotics orally at specific intervals or remembering to give parasite-control products on schedule when no parasites are seen.

Because compliance is such an important issue in the resolution of most dermatologic problems, clinicians must recognize this and create treatment plans accordingly. If it is medically prudent to do so, injectable medications should be first-line therapy, since they offer convenience to the owner and guaranteed compliance. The next preferred medications are ones that can be administered once daily, followed by those that can be administered every 12 hours. It is unlikely that oral medications needing to be administered more often than twice daily will be given on schedule other than by the most dedicated of pet owners. It is also important to remember that administering a medication twice daily is not the same as administering it every 12 hours, so even clients that remember to give a medication twice in any given day might not give it at the appropriate interval consistent with the drug's pharmacokinetics. If so, the pet is not receiving the true intended benefit of that medication.

Finally, cost might be one consideration in medication selection, but if the goal is for the pet to actually receive all the benefit of any selected medication, compliance should be the main selection attribute. The most costly medication (in terms of time and medical outcomes) is actually the medication that fails to deliver the anticipated benefits because it was not administered appropriately7.

Lacking realistic product and pricing models: Veterinary hospitals benefit from carrying appropriate products in inventory, pricing them competitively, and tracking compliance to ensure that pets and owners are actually receiving the full benefit of products dispensed and administered.

While clients might want to browse retail store shelves for the best bargains, when it comes to treating their pets with medical issues, they typically want a firm recommendation made by the veterinarian, not a list of possible treatments that the owner can consider. Be prepared to make clear product recommendations, and be prepared to explain why this is the best option for a particular pet. Some clients will always want the least costly option, but veterinarians owe it to their clients to make firm recommendations, based on evidence-based criteria of what is in the pet's best interest.

Considering dermatology clients a nuisance: You are in business to serve the needs of your clients. Due to the nature of dermatologic diseases, you will see most of your dermatologic patients and their owners many times during the year, for many years to come. That gives you a great opportunity to bond with these pet owners and help shape a positive and mutually beneficial veterinarian-client-patient relationship. These are exactly the clients that you should crave for your practice. The next time the pollen count rises and your telephone starts ringing frequently from clients with itchy pets -- don't curse -- give thanks! If properly counselled, these are probably the most dedicated clients you will have in your practice.

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5.1.6. Journey to “Yes” – Gaining Client Acceptance of Recommendations

This is an exciting time for veterinary medicine, since the opportunities have never been so great, but also a little scary, because it means that we need to use appropriate business principles and metrics if we are to successfully fend off unwanted competition and reach our true potential.

Interestingly, it appears that it is not pet owners that are setting limits on practice profitability, but veterinarians themselves. Fortunately, this is something that can be remedied, and veterinarians are now starting to heed the call to action.

It might seem that clients are looking for high-quality medicine, and this is certainly a common enough sentiment in practice mission statements, but clients rarely know how to assess this in a practice. So, they make decisions based on the information that is available to them. Accordingly, to keep clients satisfied and coming back for all their pet health care, it is important to give them what they want most.

The value of a client is much more than the sum of any individual invoice. Their true value comes in the relationships that develop that might span years, and the lives of several pets. A loyal client base is the most valuable asset that a veterinary practice possesses. Without it, the level of medicine practiced in the facility is almost irrelevant. Therefore, veterinary practices must endeavor to keep their clients satisfied, and that means giving them what they want most.

It is important to understand why clients accept some recommendations yet reject others. In general, clients take recommendations when they realize they need the service, they understand the service offering, they trust the service being delivered, and they see the value in the offering. On the other hand, clients often reject services when one or more of these features is lacking. That is, they may reject the service if they don’t believe it is needed, they don’t fully understand the reason for the service, they don’t trust the manner in which it is being offered, or they don’t see the value in the offering.

It should be no surprise that clients do not remember everything said to them in the examination room. In fact, their recall by the time they reach the reception area is sometimes suspect. Clients hear and process only a small portion of what they are told, and so this information must be repeatedly validated and education must be considered an ongoing endeavor. If people do not receive an appropriate rationale from their veterinarian, they are just as likely to create their own rationalization or seek other opinions elsewhere.

When speaking with clients, it is advisable to use “message points” rather than rambling conversation styles. Expect that clients will only retain a few points of the discussion, and so deliver those message points as “sound bites” just as is done in news reporting. It not only saves time in the exam room, but it actually increases client retention of facts.

It might seem somewhat artificial, but there are ways of making it more likely for clients to say “yes” to recommend services, and to improve the experience for them as well. To do this, it is necessary to acknowledge that veterinary medicine is a business, that pet owners are clients or customers, and that they do have choices relative to their purchasing experience.

Clients want to do business with practices and doctors that they trust, and sometimes this involves somewhat artificial trust-building exercises. This is especially true for new clients, and for new practices or doctors. While somewhat contrived, it is otherwise difficult to build trust in the short term, when a pet is only seen once or twice a year.

Education is key for client interactions. It may take seven messages on the same topic before clients understand the reason for a recommendation. Do not assume that one discussion during a 20-minute office visit is sufficient to make the point in their minds.

Clients don’t really buy veterinary services. They buy wellness for their pets. For any recommended service, it is important to convey the benefits, as well as any downside if the recommendation is not followed. It is not necessary to be an alarmist for clients to appreciate why something should be done.

In the retail marketplace, salespeople are very familiar with the need to “close” a deal. While veterinarians are uncomfortable with these concepts, clients are left with vague recommendations unless a very specific recommendation is made. Otherwise, you may not see that client again for 6-12 months at which time you’ll like have the same discussion as before.

Retail behavioral science has determined that while clients enjoy a good first encounter, they actually place more value in the last encounter they experience. Accordingly, it is important to finish strong. This is the time to truly impress the client with service, and it is this final experience that will carry them through to their next visit.

If you’ve ever wondered whether to deliver good news or bad news first, from a client service point of view, there is no doubt – deliver the bad news first. The last message the client hears from you should be positive.

There is also an interesting consumer behavior related to good and bad events. When something good is happening, clients would rather segment this happy time into multiple occurrences. On the other hand, when bad things are happening, clients would rather them happen all at once and be done with them.

Clients are much more likely to take recommendations, as well as pay the costs of services, when they have been an active part of the decision-making process. Interestingly, those decisions needn't even be a major part of the medical care issues. When clients play a role, even selecting things such as the time of discharge or the leg in which a catheter is to be placed, they accept a stake in the process. Such clients not only are more accepting if there are problems with the procedure but are more likely to accept financial responsibility for the case management.

Clients often feel that they have so little control in the medical process, that they are even more accepting of standards of care and protocols, since they take comfort in the standardization. Accordingly, it is valuable to clients if the hospital standardizes most care pathways, including the order in which a physical examination is conducted, recommendations for managing periodontal disease, protocols for monitoring a variety of drug therapies, etc.

Veterinarians may assume that their clients have good reason for realizing they are competent, but in reality, it is very difficult for clients to appreciate the level of skill of any physician. They make judgments based on what they know and feel, and most of this is a subconscious decision.

Clients have so little information on which to judge competence that they are excited whenever clues are available to them. Use opportunities to highlight the accomplishments of all professionals and paraprofessionals, such as practice promotional materials, the web site, and newsletters. Prominently display licenses, certificates and diplomas and make sure they are professionally framed.

Finally, it is important for owners to see the value in the services they purchase. This does not mean that discounts are indicated or that the services need to be priced less than competitors. People make value judgments all the time for their purchases, from selecting a brand of milk, to the type of car that they own. However, nobody wants to pay the price of a Mercedes, and receive a Pinto. Fortunately, there is abundant evidence that clients are willing to pay for the value delivered by veterinarians, if only it can be presented appropriately.

Veterinarians are comfortable charging a mark-up on "things" but traditionally have had a difficult time pricing their own services and services performed by paraprofessional staff. In fact, in the classic veterinary scenario, practices discount their professional

services and make up the difference by inflating the cost of products that are dispensed. In a time when consumers are finding other outlets for the buying of products, this is putting increased pressure on the existing veterinary model.

Selling goods, including diets, shampoos, pharmaceuticals and parasite-control products from a veterinary practice should be a matter of convenience for owners, not a measure of

practice loyalty. It is important to be an advocate for clients and for practices to understand what they can sell at a premium – professional services.

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6.1.1. LA IMPORTANCIA DE LA CITOLOGÍA EN EL DIAGNÓSTICO DE LINFADENOPATÍAS

Generalidades

Se ha demostrado que la citología sirve como una herramienta muy útil en el diagnóstico de masas cutáneas/subcutáneas y linfadenopatías en animales. Las interpretaciones a partir de citologías obtenidas por FNA llegan a tener un 97% de precisión comparado a la histología (prueba gold estándar) en casos de linfadenopatías en humanos.

La citología de linfonódulos es mínimamente invasiva y rentable ampliamente utilizada en animales de compañía con fines de diagnóstico e investigación de linfadenopatías e incluso para la estadificación de tumores. Ambas técnicas, FNNA o FNA, son las técnicas mayormente utilizadas para la toma de muestra de linfonódulos sin haber una diferencia significativa en la calidad citológica de los preparados (celularidad, contaminación con sangre, grosor del extendido, preservación celular y fragmentación celular)

Hay que considerar que la citología, como toda herramienta diagnóstica, tiene sus inconvenientes para el diagnóstico de linfadenopatías:

- El muestreo de un linfonódulo que no está completamente poblado por linfocitos neoplásicos: dificultades para el diagnóstico de linfomas de bajo grado.
- Muestreo desproporcionado de un gran linfonódulo reactivo: imposibilidad de colectar suficientes células plasmáticas para la distinción de linfonódulos reactivos de normales.
- Linfomas compuestos predominantemente por linfocitos pequeños (Por ejemplo: Linfoma de células pequeñas): Dificultad para poder diferenciar linfomas linfocíticos de linfonódulo normal.
- Administración previa de glucocorticoides: Reducción significativa de células linfoides.
- Mala interpretación del operario al evaluar las láminas: Debido a la inexperiencia del operario.
- Preparaciones de frotis que carecen de suficientes células intactas o bien diseminadas: Dificultad para llegar al diagnóstico con pocas células o por superposición exagerada de células.

La importancia de la citología en el diagnóstico de linfadenopatías se puede sustentar en 6 pilares:

1. Excelente herramienta diagnóstica para evaluar la morfología celular que constituyen los linfonódulos.

2. Permite el diagnóstico de linfomas, su graduación y tipo de acuerdo a las células predominantes.

3. Permite el diagnóstico de metástasis.

4. Permite conocer el curso de una lesión inflamatoria de acuerdo al tipo celular encontrado.

5. Permite aproximarnos o llegar al diagnóstico de una diversas de agentes infecciosos involucrados.

6. Permite aproximarnos al diagnóstico de diferentes condiciones o patologías.

1. Excelente herramienta para evaluar la morfología celular

La citología ha avanzado mucho como herramienta para el diagnóstico de linfadenopatías y sobre todo, en el diagnóstico de linfomas, siendo muchas de estas células constituyentes, capaces de ser reconocidas por su morfología característica tan bien dilucidada en un buen preparado citológico. Con relación a las células linfoides de pequeño a mediano tamaño tenemos: linfocito pequeño, prolinfocito, centrocito y plasmocito (y su transformación a “célula de mott”); mientras que, las células linfoides de mediano a gran tamaño capaces de ser identificadas tenemos: linfoblasto, centroblasto e inmunoblasto. Es posible reconocer a todas estas células tanto en linfomas de bajo como de alto grado.

2. Permite el diagnóstico de linfomas según el grado y tipo, de acuerdo a las células predominantes

Desde hace varios años se ha venido desarrollando diversos sistemas para la clasificación de linfomas en animales, contando desde la primera clasificación propuesta por Gall y Mallory en 1942 hasta la nueva clasificación propuesta por la OMS el 2001, manteniéndose dicha clasificación histopatológica hasta la actualidad. Sin embargo, existió una clasificación muy completa y que se adecua mucho a las posibilidades que brinda la citología como herramienta diagnóstica de linfomas en pequeños animales. Esta clasificación fue la propuesta por Stanfeld y col. (1988) conocida como la “clasificación actualizada de Kiel” la cual fue propuesta inicialmente por Lennert (1978) mostrando en su época, muchas limitaciones que fueron subsanadas por Stanfeld y col. Hace varios años se viene tomando como referencia la clasificación actualizada de Kiel para trabajos de investigación sobre citología de linfadenopatías en animales permitiendo no solamente el diagnóstico del cancer, sino el establecer el grado ya sea de bajo o alto; y asimismo, los tipos de linfoma (Ej. Linfoma de células macronucleoladas, Linfoma inmunoblástico, etc.) lo cual permite aproximarnos mucho más al posible origen celular del linfoma, la cual debería ser confirmada con la inmunofenotipificación, citometría de flujo y/o diagnóstico molecular.

3. Permite el diagnóstico de metastasis

Como se ha mencionado, los linfonódulos agrandados son frecuentemente examinados citológicamente en caninos y la linfadenomegalia metastásica de cualquier origen es un hallazgo citológico común en estos casos. Los estudios revelan la existencia de tumores metastásicos secundarios entre el 10% al 40% de los perros con agrandamiento de linfonódulos. La precisión y la sensibilidad son dos de los indicadores más importantes valorados en la citología, incluso en un índice mayor a la histopatología. La linfadenomegalia neoplásica es frecuentemente reconocida en perros adultos y gerontes, con predisposición en hembras relacionada a la diseminación de neoplasias mamarias de origen maligno. Los mastocitomas, adenocarcinoma y melanomas son las causas más comunes de linfadenomegalia metastásica; sin embargo, no hay que descartar otras causas comunes dentro del diagnóstico diferencial como son los fibrosarcomas, osteosarcomas, carcinomas de células escamosas, o tumor venéreo transmisible, entre otras.

4. Permite conocer el tipo de lesion inflamatoria de acuerdo al tipo celular encontrado

La linfadenitis es una condición en la cual, los linfonódulos llegan a aparecer inflamados debido a causas infecciosas o no infecciosas. Los neutrófilos son las células inflamatorias predominante y es el primero en actuar en casos de infecciones, otras células que aparecen son los macrófagos en estado activado (espumosos) o inactivos (en reposo), células linfoides entre linfocitos pequeños y plasmocitos, y eosinófilos entre las células más comúnmente encontradas. La linfadenitis neutrofílica y eosinofílica son diagnosticadas de acuerdo al porcentaje del tipo celular encontrado. En caso de las linfadenitis neutrofílica, los neutrófilos superan el 5%, y en las de tipo eosinofílica, los eosinófilos superan el 3% de todas las células nucleadas. Las linfadenitis piogranulomatosas (también llamadas histiocíticas) se definen cuando > 3% del total de células nucleadas son macrófagos o células epitelioides (pueden o no aparecer células gigantes multinucleadas)

5. Permite aproximarnos al diagnóstico de una diversidad de agentes infecciosos

La citología como herramienta diagnóstica también permite la detección, por valoración y descripción de características morfológicas, de múltiples agentes infecciosos, ya sea desde agentes bacterianos donde solo podemos identificar y describir según la forma o distribución que observemos (cocos, diplococos, cocobacilos y bacilos), hasta hongos sistémicos (Cryptococcus, Histoplasma, Blastomyces, etc.) o parásitos (Leishmania, Cytauxzoon, etc.).

6. Permite aproximarnos al diagnóstico de condiciones variadas

El linfonódulo reactivo (también llamado linfonódulo hiperplásico) es uno de los diagnósticos citológicos más frecuentes en medicina veterinaria donde la población de linfocitos pequeños bien diferenciados son la población celular predominante; sin embargo, en muchos casos la población de células plasmáticas y de linfoblásticos se

puede ver notablemente incrementada, llegándose a sumar al diagnóstico la hiperplasia plasmocítica o linfoblástica. La hiperplasia de células plasmáticas es probablemente el resultado de una estimulación antigénica crónica. El diagnóstico de linfonódulo reactivo es común para muchas condiciones de índole infeccioso, en la cual, la estimulación antigénica provoca la hiperplasia de poblaciones linfoides evidenciándose como un incremento del tamaño de los linfonódulos macroscópicamente.

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6.1.2. DIAGNÓSTICO CITOLÓGICO DE LINFOMAS DE ALTO GRADO: UN MUNDO POCO CONOCIDO

Generalidades

El agrandamiento de linfonódulos es un signo clínico común en caninos y felinos, lo cual puede reflejar una variedad de trastornos subyacentes. En tales casos, resulta necesario distinguir entre malignidad, por ejemplo linfomas de alto grado y de bajo grado, y otras causas de linfadenopatías tales como linfadenitis o hiperplasia reactiva en orden a instaurar una terapia apropiada y establecer un pronóstico. Las técnicas citológicas han llegado a ser parte integral de diagnóstico en casos clínicos de malignidad. Siendo un procedimiento simple y rápido, esta técnica emplea pocos recursos para obtener las células representativas de la mayoría de lesiones. Aunque no es posible obtener información sobre la arquitectura tisular; los componentes celulares, las células neoplásicas y no neoplásicas podrían ser interpretadas claramente con la ayuda de la citología.

Lo enigmático de la citología y el linfoma de alto grado

Los linfomas de alto grado han sido poco conocidos hasta la actualidad, y mucho de este desconocimiento a lo largo de los años podríamos evocarlo a cuatro principales razones, entre las cuales se encuentran:

1. Falta de capacitación en temas relacionados a citología de linfonódulos.
2. Falta de adaptación de alguna clasificación para linfomas en medicina veterinaria.
3. Falta de caracterización de linfomas.
4. Falta de exigencia de resultados más precisos por parte de los médicos veterinarios.

Citologías de linfomas de alto grado

Los linfomas de alto grado son también conocidos como linfomas de células grandes y se caracterizan por la presencia de poblaciones de células linfoides medianas a grandes e inmaduras, las cuales presentan un tamaño menor igual al de un neutrófilo y en ciertas células puede alcanzar un tamaño superior. A continuación se detallan las características más relevantes de las células más frecuentemente encontradas en preparados citológicos de linfomas de bajo grado:

1. Centroblasto: representan entre el 1 y el 5% de la población celular linfoide y se origina de las células B foliculares. Presenta un tamaño intermedio a grande, citoplasma intensamente basófilo y granular, diámetro nuclear de 10 a 14 µm, cromatina finamente punteada y nucléolos múltiples.

2. Linfoblasto: representa < 1% de la población linfoide y se origina a partir de poblaciones linfoides de tipo B o T. El tamaño varía entre pequeño y mediano, presenta escaso citoplasma azul claro, diámetro nuclear de 8 a 13 µm, cromatina reticular y nucléolos indistinguibles.

3. Inmunoblasto: representa representan entre el 1 y el 5% de la población celular linfoide y se origina a partir de poblaciones linfoides de tipo B o T. Presenta un tamaño grande, moderada a abundante cantidad de citoplasma finamente granular, forma nuclear y cromatina similar al centroblasto, el diámetro nuclear puede alcanzar los 28 µm, un solo nucléolo prominente de localización central.

4. Célula plasmática: representa menos del 5% del total de células linfoides presentes y se origina a partir de las células B del cordón medular, presenta un tamaño mediano con abundante citoplasma intensamente basófilo, halo perinuclear claro, el diámetro nuclear que varía entre 7 a 12 µm y es de localización excéntrica.

Otras células que pueden ser identificadas en linfomas de bajo grado son neutrófilos, eosinófilos, mastocitos, macrófagos, “hairy cell” y células estromales, los cuales representan menos del 2% del total de células.

Desde hace varios años se ha venido desarrollando diversos sistemas para la clasificación de linfomas en animales, contando desde la primera clasificación propuesta por Gall y Mallory en 1942 hasta la nueva clasificación propuesta por la OMS el 2001, manteniéndose dicha clasificación histopatológica hasta la actualidad. Sin embargo, existió una clasificación muy completa y que se adecua mucho a las posibilidades que brinda la citología como herramienta diagnóstica de linfomas en pequeños animales. Esta clasificación fue la propuesta por Stanfeld y col. (1988) conocida como la “clasificación actualizada de Kiel” la cual fue propuesta inicialmente por Lennert (1978) mostrando en su época, muchas limitaciones que fueron subsanadas por Stanfeld y col. Hace varios años se viene tomando como referencia la clasificación actualizada de Kiel para la clasificación de linfomas. La clasificación de Kiel actualizada presenta varias ventajas, entre ellas, que es fácil de usar, caracteriza muchas entidades biológicamente relevantes, y el linaje es tomado en consideración (linfoma de células B vs linfoma de células T). A pesar de ello, esta clasificación presenta algunas desventajas, entre ellas, que es principalmente aplicada para linfomas de linfonódulos, algunas categorías no son reproducibles, mientras que las neoplasias de células NK no suelen ser reconocidas.

Características citológicas de los linfomas de alto grado

Linfoma centroblástico monomórficoEstá compuesto de más del 60% de centroblastos. Estas células tienen un núcleo redondo, patrón de cromatina fina y 2 a 4 pequeños nucléolos prominentes y basófilos localizados marginalmente. Centrocitos e inmunoblastos pueden estar presentes.

Linfoma centroblástico polimórfico

Número incrementado de inmunoblastos (10 – 90%). Los inmunoblastos son células grandes con abundante citoplasma basófilo, un núcleo que mide al menos 3 veces el tamaño de un eritrocito y un nucléolo grande central. Existen 2 tipos: de células pequeñas y células grandes predominantemente.

Linfoma inmunoblástico

Población de células linfoides constituida principalmente por inmunoblastos. Células linfoides grandes que tienen un único nucléolo grande localizado centralmente.

Linfoma linfoblástico Células de tamaño pequeño a mediano con núcleo redondo o convulado de 1.5 a 2.5 veces el diámetro del eritrocito. Los nucléolos son pequeños y generalmente indistintos. El citoplasma es escaso y moderadamente basófilo. Actividad mitótica es alta.

Linfoma pleomórfico de células grandes y pequeñas mezcladas Población mixta de células linfoides de pequeño y mediano tamaño. Se pueden observar células que contienen un único nucléolo grande y prominente y cantidades moderadas de citoplasma basófilo y otras células linfoides que son pequeñas y medianas con escaso citoplasma.

Linfoma de Burkitt o tipo Burkitt

Linfocitos pequeños/medianos; citoplasma intensamente basófilo, usualmente vacuolizado; núcleo redondeado y no hendido, cromatina variablemente condensada, nucléolos múltiples, índice mitótico alto, macrófagos de cuerpo “tingible”.

Linfoma anaplásico

Células de tamaño mediano a grande con abundante citoplasma anfófilo. Núcleo localizado excéntricamente y varían de redondo a ovalado forma de riñón o herradura (células de sello) o incluso multilobuladas

Importancia de la caracterización citológica de linfomas

Pocas veces se tiene la oportunidad de enviar muestras para diagnóstico histopatológico. Asimismo, el tipo de linfoma diagnosticado citológicamente puede tener implicancia en la respuesta al tratamiento según el tipo de célula, el grado y de ambos:

a) Según el tipo de célula: Cuando se tratan con doxorrubicina, más de un 80% de los linfomas de células B responden con una remisión completa a la doxorrubicina; mientras que sólo el 50% de los linfomas de células T responden a este fármaco y menos del 20% consiguen remisión completa. Sin tratamiento tienen un mal pronóstico (en unos 2 meses el cáncer progresa y la calidad de vida se deteriora mucho).

b) Según el grado: Los linfomas de bajo grado tienen un pronóstico mucho mejor y el tratamiento es distinto. Los linfomas de bajo grado son un 5-30% de los linfomas, pero para su diagnóstico son necesarias pruebas adicionales como la biopsia con inmunohistoquímica o citometría de flujo.

c) Según el tipo de célula y el grado: La mayoría de perros con linfoma de células B de alto grado responden bien al tratamiento consiguiendo una remisión completa en muchos de los casos. Los perros tratados con quimioterapia viven alrededor de un año. Los linfomas de células T de alto grado responden en menor medida y en ocasiones con una remisión parcial al tratamiento y una supervivencia media de alrededor de 5-9 meses.

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6.1.3. DIAGNÓSTICO CITOLÓGICO DE LINFOMAS DE BAJO GRADO: UN RETO AL CUAL ENFRENTAR

Generalidades

El agrandamiento de linfonódulos es un signo clínico común en caninos y felinos, lo cual puede reflejar una variedad de trastornos subyacentes. En tales casos, resulta necesario distinguir entre malignidad, por ejemplo linfomas de alto grado y de bajo grado, y otras causas de linfadenopatías tales como linfadenitis o hiperplasia reactiva en orden a instaurar una terapia apropiada y establecer un pronóstico. Las técnicas citológicas han llegado a ser parte integral de diagnóstico en casos clínicos de malignidad. Siendo un procedimiento simple y rápido, esta técnica emplea pocos recursos para obtener las células representativas de la mayoría de lesiones. Aunque no es posible obtener información sobre la arquitectura tisular; los componentes celulares, las células neoplásicas y no neoplásicas podrían ser interpretadas claramente con la ayuda de la citología.

El reto de diagnosticar linfoma de bajo grado

La citología como herramienta diagnóstica tiene por sí varias limitaciones para poder llegar al diagnóstico de una diversidad de neoplasias. Con relación a los linfomas de bajo grado, se han reconocido algunos retos de significancia, entre los cuales tenemos:

Elevada celularidad de pequeño y mediano tamaño: simula a un linfonódulo normal o reactivo, se debe identificar la morfología celular (forma de la célula, relación N: C, forma nuclear, tipo de cromatina).

Escasez de células blásticas y cuerpos linfoglandulares: falta de experiencia en el operario al tratar de asociar el diagnóstico de linfoma con la presencia de “linfoblastos” y abundancia de cuerpos linfoglandulares en los preparados citológicos.

Diversidad celular y de tipos de linfomas de bajo grado: la evaluación citológica permite reconocer diversos tipos celulares con características propias de ciertas células linfoides incluyendo tamaño celular, relación N: C, forma nuclear, localización del núcleo, número y localización de los nucléolos. Asimismo y gracias a la identificación citológica de diversos tipos celulares se ha reconocido desde hace varios años la existencia de diversos tipos de linfomas, los cuales se ha intentado correlacionar con el éxito al tratamiento y el pronóstico del paciente.

Inexperiencia del operario: este es uno de los retos más importantes y de los cuales se ha venido teniendo pocos avances con respecto al diagnóstico citológico más certero para casos de linfomas.

Citologías de linfomas de bajo grado

Los linfomas de bajo grado son también conocidos como linfomas de células pequeñas, a la evaluación citológica este tipo de linfomas se caracterizan por presentar una población homogénea de linfocitos maduros de pequeño y mediano tamaño, y en ciertas ocasiones, algunas neoplasias llegan a presentar características muy similares a las de un linfonódulo normal. A continuación se detallan las características más relevantes de las células más frecuentemente encontradas en preparados citológicos de linfomas de bajo grado:

Linfocito pequeño: representan más del 70% de la población linfoide y se origina de poblaciones linfoides de tipo B o T. Presenta un tamaño celular y nuclear que varía entre las 7 y 10 µm y está formado de un anillo citoplasmático y un núcleo redondeado o ligeramente indentado.

Prolinfocito: representa ≤ 20% de la población linfoide y se origina a partir de poblaciones linfoides de tipo B o T. El tamaño varía entre 9 a 13 µm con una regular a moderada cantidad de citoplasma, núcleo redondo, ovalado o ligeramente indentado con presencia de nucléolos poco notorios.

Centrocito: representa ≤ 5-10% y se origina de poblaciones linfoides provenientes de las células B foliculares. Es una célula de tamaño intermedio, moderada cantidad de citoplasma basófilo, diámetro nuclear de 10 a 14 µm con indentación. Existen dos tipos de centrocitos (pequeño y grande), ambos con similares patrones de cromatina y núcleo indentado1 o 2 nucléolos visibles., en el caso del centrocito grande, éste presenta 1 o 2 nucléolos.

Célula plasmática: representa menos del 5% del total de células linfoides presentes y se origina a partir de las células B del cordón medular, presenta un tamaño mediano con abundante citoplasma intensamente basófilo, halo perinuclear claro, el diámetro nuclear que varía entre 7 a 12 µm y es de localización excéntrica.

Otras células que pueden ser identificadas en linfomas de bajo grado son neutrófilos, eosinófilos, mastocitos, macrófagos, “hairy cell” y células estromales, los cuales representan menos del 2% del total de células.

Desde hace varios años se ha venido desarrollando diversos sistemas para la clasificación de linfomas en animales, contando desde la primera clasificación propuesta por Gall y Mallory en 1942 hasta la nueva clasificación propuesta por la OMS el 2001, manteniéndose dicha clasificación histopatológica hasta la actualidad. Sin embargo, existió una clasificación muy completa y que se adecua mucho a las posibilidades que brinda la citología como herramienta diagnóstica de linfomas en pequeños animales. Esta clasificación fue la propuesta por Stanfeld y col. (1988) conocida como la “clasificación actualizada de Kiel” la cual fue propuesta inicialmente por Lennert (1978) mostrando en su época, muchas limitaciones que fueron

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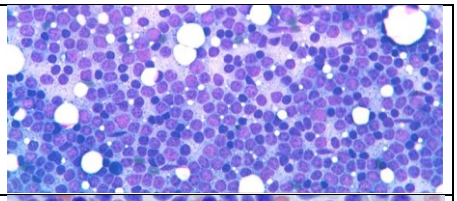
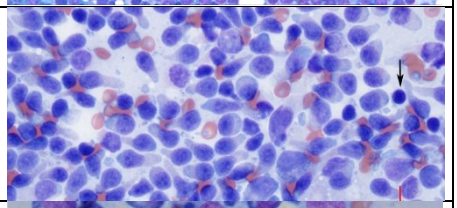
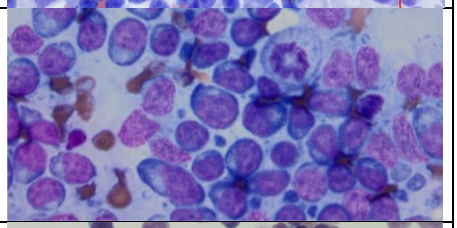
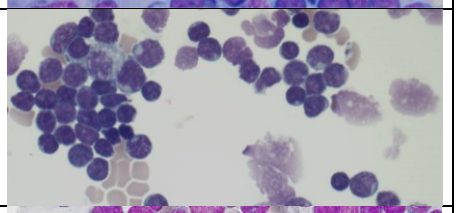
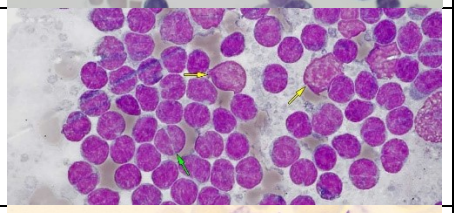
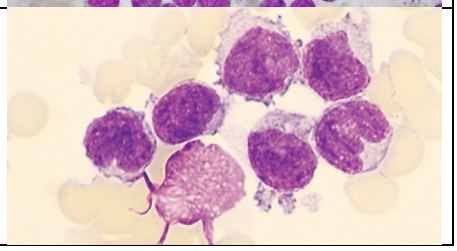
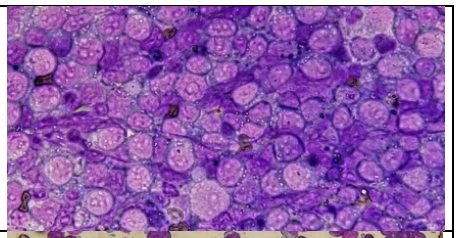
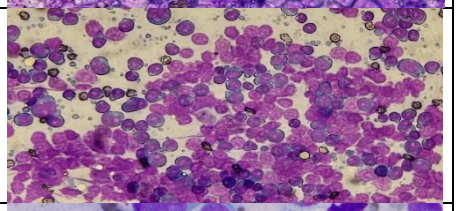
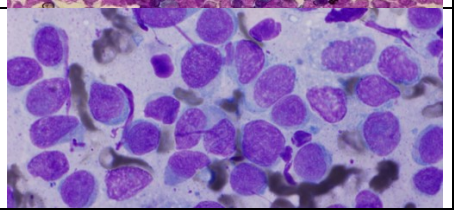
Características citológicas de los linfomas de bajo grado

Linfoma Linfocítico	Población celular dispersa monótona compuesto por linfocitos pequeños con cromatina condensada y ocasionalmente un nucléolo. Las mitosis son raras o ausentes.	
Linfoma de zona T indolente o de células claras	Población cellular compuesta por linfocitos de tamaño pequeño a mediano con un bajo índice mitótico. Frecuentemente se puede observar células con una extension citoplasmática unipolar (hand mirror o células en espejo de mano	
Linfoma Linfoplasmocítico	Es una mezcla de linfocitos pequeños y medianos que frecuentemente tienen una apariencia plasmocitoide. Frecuente se observan cristales astillosos y agujas puntiformes en el citoplasma consistente con formaciones atípicas de cuerpos de Russell.	
Linfoma Prolinfocítico	Población cellular constituida predominante por prolinfocitos (10 a 15 µm), moderada cantidad de citoplasma basófilo, variables nucléolos.	
Linfoma Centrocítico	Población ceular constituida predominante por centrocitos y linfocitos pequeños, el número variable de centroblastos afectan el grado y el pronóstico, bajo índice mitótico.	
Linfoma de células pequeñas pleomórficas	Población relativamente monomórfica de células pequeñas con escaso citoplasma gris. El núcleo está caracterizado por una superficie lisa en un lado y estrías en el lado opuesto.	

Linfoma Centrocítico-Centroblástico	Centrocitoide. Tamaño intermedio entre el pequeño centrocito y el gran centroblasto. El núcleo contiene de 2 a 5 nucléolos pequeños y basófilos localizados centralmente. El citoplasma es escaso y moderadamente basófilo. Las células centrocitoides deberían exceder el 30% de la población celular.	
Linfoma de células de tamaño mediano macronucleado	Centrocitoide. Características similares al centrocítico-centroblástico pero con un único nucléolo.	
Micosis fungoide	Células linfoides de mediano a gran tamaño con leve a moderada cantidad de citoplasma azul grisáceo a ligeramente basófilo, pudiendo presenter finas vacuolas. El núcleo es frecuentemente excéntrico con multiples formas y nucléolos no visibles.	

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6.1.4. NEOPLASIAS EPITELIALES DE GLÁNDULA MAMARIA: ¿CÓMO CLASIFICARLAS CITOLÓGICAMENTE?

Introducción

El tumor mamario es un tipo común de neoplasia que se origina del epitelio glandular de la glándula mamaria. Es un hallazgo común en perras y gatas intactas, representando entre el 50 y 70% de todos los tumores en este grupo poblacional. Asimismo, estos tumores representan una serie problema en medicina veterinaria. El cáncer mamario canino como un buen modelo animal para el estudio de cáncer de mama en humanos. Recientes estudios han mostrado un incremento de la prevalencia de tumores mamarios malignos frente a los tumores mamarios de carácter benigno, similar escenario a lo encontrado en medicina humana.

La citología en el diagnóstico de tumores mamarios

La citología es un método simple, rápido y económico usado para el diagnóstico preoperatorio de tumores mamarios en caninos en la práctica veterinaria. La evaluación citológica tiene importantes beneficios en aclarar algunos aspectos en el diagnóstico temprano de lesiones mamarias. Ya sea la técnica con aspiración o sin aspiración con aguja fina, es más rápida y más económica que la biopsia quirúrgica sin embargo, tiene sus desventajas tales como el muestreo de una baja cantidad de masa tumoral o colección de un tejido inadecuado, pudiéndose minimizar con la experiencia del operario.

Históricamente, la citología diagnóstica ha sido considerada de un valor limitado para neoplasias mamarias; así, mientras algunos estudios indican la inadecuada precisión en el diagnóstico, otros indican su elevada concordancia con los resultados de la histopatología. Esta discordancia podría deberse a la heterogeneidad de los tumores mamarios, las inapropiadas habilidades técnicas o el, diseño de estudio (muestras no diagnósticas). La evaluación citológica tiene una sensibilidad y especificidad satisfactoria para la diferenciación de tumores mamarios benignos y caninos; además la correlación de resultados encontrados entre la citología y la histopatología revelan más de un 90% para tumores malignos y entre un 80 y 90% para tumores benignos.

Las neoplasias mamarias en perras son altamente variables en morfología y frecuentemente están constituidos por más de un tipo celular, por lo que no es tan sencillo poder diagnosticar citológicamente una neoplasia mamaria (sobre todo de origen epitelial) por lo cual conllevará de una mayor observación y criterio por parte del citopatólogo a cargo.

Protocolo de evaluación citológica para el diagnóstico de neoplasias epiteliales mamarias en caninos

Aumento 40x: Búsqueda de áreas con el mayor número de células bien teñidas, bien distribuidas e intactas para su distribución., artefactos.

Aumento 100x: Evaluación de la celularidad, distribuciones celulares, arreglos (formación de “clusters”), sustancia de fondo.

Aumento 400x: Evaluación de los tipos de células encontradas en glándula mamaria (células mioepiteliales, ductales y estromales), células inflamatorias, restos de necrosis y material extracelular. Evaluación de criterios de malignidad nuclear y citoplasmático.

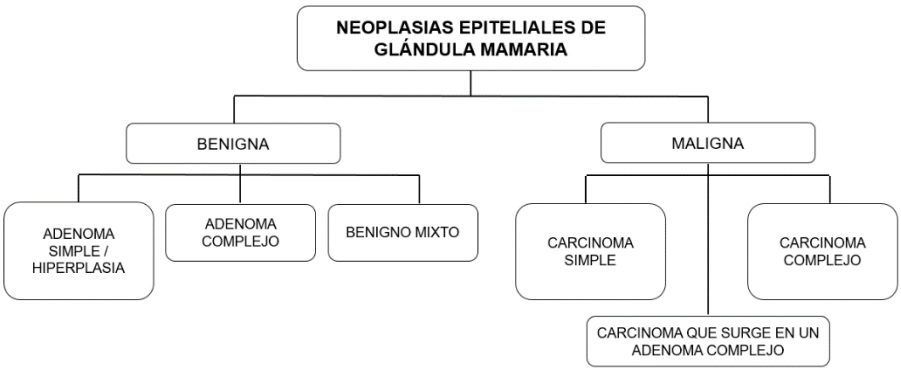
Aumento 1000x: Evaluación de los detalles celulares y profundización en los criterios nucleares de malignidad (nucléolos angulares y multinucleación).

Entre los criterios que tenemos para considerar una muestra inadecuada para diagnóstico citológico se encuentran la falta de células intactas, la excesiva contaminación con sangre, elevada cantidad de debris celular y elevada cantidad de artefacto (precipitados, polen de los guantes, aplastamiento, etc.).

Clasificación, calificación y graduación de neoplasias epiteliales mamarias en caninos

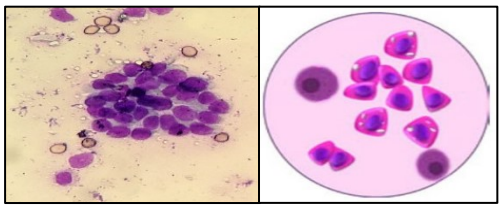
En humanos, la citología por punción aspiración o sin aspiración aguja fina tiene un protocolo estándar, el sistema de graduación de Robinson, el cual tiene una alta correlación con el sistema de graduación histológico de Scarff Bloom- Richardson. Recientemente, se ha podido determinar que el sistema de graduación de Robinson y el sistema de calificación actual basado en el de Robinson y otros sistemas propuestos, puede ser aplicado para tumores mamarios en caninos, concluyendo en un sistema de calificación y graduación citológica eficiente la cual permitiría apoyar a citopatólogos veterinarios en el diagnóstico y pronóstico de esta condición.

Tipos de neoplasias mamarias epiteliales diagnosticadas citológicamente en caninos

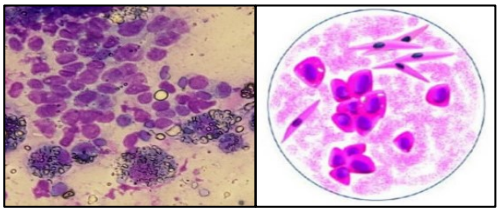


Neoplasias benignas:

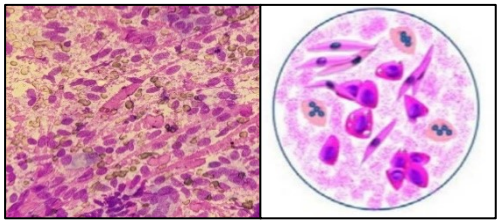
1. **Adenoma simple:** baja a moderada celularidad, grupos de células epiteliales mamarias con leve atipia, o citoplasma vacuolizado, vacuolas secretorias, macrófagos y cristales de colesterol



2. **Adenoma complejo:** dos diferentes poblaciones celulares: 1) grupos de células epiteliales monomórficas con leve atipia; 2) Células mioepiteliales elongadas, núcleos densamente teñidos, escaso a moderado citoplasma pálido, varios núcleos desnudos, material rosado mucosecretorio y macrófagos.

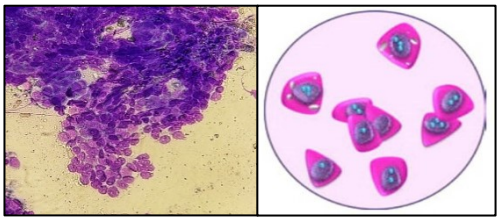


3. **Tumor mixto benigno:** baja a moderada celularidad, células epiteliales con leve pleomorfismo principalmente en grupos pequeños, células mioepiteliales, excesiva cantidad de material extracelular rosado, fondo eosinofílico granular y osteoclastos ocasionales.

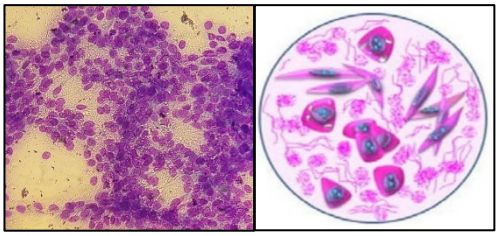


Neoplasias malignas

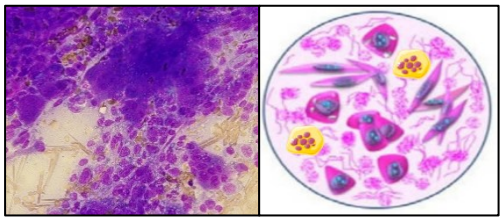
1. **Carcinoma simple:** moderada a alta celularidad, células epiteliales individuales o en grupos, pleomorfismo, anisocitosis, macrocitosis, anisocitosis, macrocariosis, incremento del ratio N:C, nucléolos grandes, prominentes o múltiples, amoldamiento nuclear, cromatina cordonada, presencia de células multinucleadas anormales y figuras de mitosis.



2. **Carcinoma complejo:** moderada a alta celularidad, células epiteliales individuales y en grupos, pleomorfismo, anisocitosis, macrocitosis, anisocariosis, macrocariosis, incremento del ratio N:C, nucléolos grandes, prominentes o múltiples, amoldamiento nuclear, cromatina cordonada, presencia de células multinucleadas anormales y figuras de mitosis. Moderada a gran cantidad de células mioepiteliales, fibras colágenas entremezcladas y matriz extracelular rosada.



3. **Carcinoma que surge en un adenoma complejo:** Características similares al carcinoma complejo pero con matriz extracelular y fondo eosinofílico más abundante y/o presencia de osteoclastos.



Graduación de neoplasias mamarias en caninos

Grado 1: Células en grupos, leve pleomorfismo, márgenes nucleares lisos, nucléolos poco notorios y cromatina fina.

Grado 2: Células en grupos e individuales, moderado pleomorfismo, márgenes nucleares irregulares, nucléolos notorios, cromatina moderadamente granular y pocas mitosis.

Grado 3: Células mayoritariamente individuales, marcado pleomorfismo, márgenes nucleares irregulares, nucléolos prominentes, cromatina gruesa y mitosis incrementada.

Sistema de calificación citológica actual para neoplasias mamarias en caninos (Basado en los sistemas de clasificación de Robinson, Khan, Taniguchi, Howell y Bonzanini) (Kappusamy et al., 2019)

Características citológicas	Score 1	Score 2	Score 3
Celularidad/HPF	10 – 20 células	20 – 50 células	> 50 células
Disociación celular	La mayoría en grupos	Células individuales y agrupadas	Células individuales
Formación de sincitios	1 -2	2 - 4	> 5
Tamaño celular	1 – 2x el tamaño de un eritrocito	3 – 4x el tamaño de un eritrocito	> 5x el tamaño de un eritrocito
Uniformidad celular	Monomórfica / Leve pleomorfismo	Moderado pleomorfismo	Marcado pleomorfismo
Contorno nuclear	Liso	Irregular	Brotes / Hendiduras
Tamaño nuclear	Uniforme / < 3x el tamaño de un eritrocito	3 – 5x el tamaño de un eritrocito	> 5x el tamaño de un eritrocito
Nucléolo	Poco definido	Definido	Prominente
Pleomorfismo nuclear	Ausente	Leve a moderado	Marcado
Cromatina	Fina	Moderadamente granular	Gruesa
Figuras de mitosis	Ausente	1 - 2	> 3
Núcleos tumorales libres	< 3x el tamaño de un eritrocito	3 – 5x el tamaño de un eritrocito	> 5x el tamaño de un eritrocito
Necrosis	Leve	Moderada	Marcada
Células inflamatorias/HPF	3 - 4	5 - 10	> 10
Formaciones tubulares	Marcado	Moderado	Leve a ausente

En resumen, la citología es una herramienta diagnóstica preoperatoria que permite no solamente diferenciar procesos neoplásicos de inflamatorios sino también clasificar, calificar y graduar las neoplasias epiteliales aumentando su potencial para evaluar el pronóstico del paciente. Se requieren más investigaciones de los tumores mamarios en caninos en una mayor serie de casos.

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6.1.5. NEOPLASIAS MULTICÉNTRICAS Y SU DIVERSIDAD CITOLÓGICA

Introducción

Las neoplasias en caninos se han incrementado desde varios años hasta la actualidad, siendo los tumores multicéntricos una de las presentaciones que se presentan con mayor frecuencia. Asimismo, existen muchos factores etiológicos tales como infecciones, trastornos genéticos, agentes químicos e inmunodeficiencias han sido asociados con la fisiopatología de esta afección en estas especies. En el caso de los tumores multicéntricos, éstos se caracterizan por presentar linfadenopatía indolora, así también, puede presentarse anorexia, vómitos, diarreas, pérdida de peso, disnea, poliuria y polidipsia.

Las neoplasias pueden ser focales, multifocales o multicéntricas, éstas últimas difieren de las metástasis ya que no necesariamente una presentación multicéntrica tumoral debería estar caracterizada por una implantación de células tumorales a partir de un tejido neoplásico primario (neoplasia primaria).

Neoplasias multicéntricas

Como se ha mencionado, esta presentación clínica se ha incrementado a lo largo de los años en pacientes caninos, conceptualizándose a dichas neoplasias como neoformaciones localizadas en diferentes regiones del cuerpo (multizonal o multiregional). Este tipo de presentación puede limitarse a neoformaciones que se localizan en la superficie corporal (cutánea o subcutánea), limitado a órganos internos o una combinación de ambos. Entre las neoplasias multicéntricas más comunes en caninos tenemos el linfoma (multicéntrico y cutáneo), tumor venéreo transmisible, mastocitoma, y otros menos comunes como sarcoma histiocítico, sarcoma anaplásico con células gigantes (histiocitoma fibroso maligno), fibrosarcoma, osteosarcoma y carcinoma de células escamosas.

Linfoma multicéntrico

Representa el 75% de todos los casos de linfoma en caninos y es clasificado en 5 estadíos según lo definido por la Organización Mundial de la Salud. La evaluación citológica de un aspirado con aguja fina de un linfonódulo neoplásico es una técnica rápida, sensible y mínimamente invasiva para el diagnóstico de linfomas caninos de alto grado; sin embargo, en ocasiones, la citología podría llegar a ser insuficiente para el diagnóstico de linfomas caninos de bajo grado debiendo apoyarse de otras técnicas de diagnóstico como la histopatología, inmunofenotipificación o de pruebas moleculares.

La hiperplasia de linfoblastos es uno de los hallazgos más complicados en muestras citológicas debido a la dificultad de discernir entre linfoma de alto grado y linfonódulo reactivo; sin embargo, en casos de linfomas de alto grado, la cantidad de este tipo celular debería superar por lo general el 30% de la población total de células linfoides;

mientras que en casos de linfonódulo reactivo con hiperplasia de linfoblastos, estas células han de llegar por lo general a un 5 o 10% como máximo de la celularidad linfoide de la muestra problema.

Linfoma cutáneo

Representa el 3 al 5% de linfomas en caninos siendo más frecuente el de tipo epiteliotrópico (excepto en felinos) y afectando generalmente a caninos geriátricos con un promedio de edad de 11 años. Típicamente este tipo de linfoma se presenta como una enfermedad cutánea diseminada crónica, pero en ocasiones suele afectar las membranas mucosas y mucocutáneas. La citología revela una neoplasia de células redondas pobremente diferenciada donde se muestra una moderada a alta celularidad constituida por una población de células de tamaño mediano a grande, vacuolizadas, algunas bionucleadas y ocasionalmente multinucleadas, con un marcado pleomorfismo nuclear más que celular (núcleos ovalados, arriñonados, de apariencia monocitoide e incluso tipo células renacuajo). Los nucléolos son usualmente poco notorios pero pueden llegar a ser prominentes, el citoplasma es escaso a moderado y ligeramente basófilo. La uniformidad de la población linfoide sin inflamación significativa o infiltración de células plasmáticas es sugerente de linfoma cutáneo.

Tumor venéreo transmisible

Este tumor puede tener presentación tanto genital como extragenital y desde hace varios años se conoce que presenta diversos tipos citomorfológicos, los cuales tienen diferentes comportamientos biológicos incluyendo entre ellos la respuesta a la quimioterapia y agresividad. Los tres subtipos son: el linfocitoide, el plasmocitoide, y el mixto o también llamado linfoplasmocitoide. En el caso del subtipo linfocitoide, éste es diagnosticado bajo las siguientes características: más del 60% de las células redondas presentan citoplasma finamente granular conteniendo pocas vacuolas claras, núcleos centrados, cromatina gruesa y 1 a 2 nucléolos distinguibles. Por otro lado, el subtipo plasmocitoide está compuesto de más del 60% de células ovoides con mayor cantidad de citoplasma, varias vacuolas claras y núcleo excéntrico. Finalmente, el tipo mixto contiene ambos tipos celulares (del linfocitoide y plasmocitoide) sin exceder ninguno de ellos el 59% de la celularidad total.

Mastocitoma

Representa una de las neoplasias más frecuentes en caninos domésticos presentando 3 grados de malignidad en función de su capacidad de extenderse hacia órganos internos. Si bien es cierto la presentación multicéntrica no es frecuente, esta neoplasia es considerada dentro de la lista de diagnóstico diferencial para casos de tumores multicéntricos en caninos. La citología para este tipo de neoplasias es diagnóstica en la mayoría de los casos y la clasificación de los subtipos citomorfológicos está basado en el grado de granulación de sus células. Actualmente, se evalúa citológicamente basado en dos criterios importantes, uno de ellos es la granulación y la otra los criterios de malignidad. Un mastocitoma de alto grado es definido ya sea por la pobre granulación como por la presencia de mínimo dos criterios de malignidad de los 4 que se sugiere (figuras de mitosis, pleomorfismo nuclear, binucleación o multinucleación y

más del 50% de anisocariosis) así no sea pobremente granulado; mientras que, un mastocitoma de bajo grado debe ser granulado y no contener ≥ 2 criterios de los mencionados.

Sarcoma histiocítico

Representa una de las neoplasias menos frecuentes en caninos y felinos (más raro en felinos), la cual surge principalmente de las células dendríticas: epiteliales (de Langerhans) e intersticiales en caninos. Entre los órganios y tejidos involucrados se encuentran el bazo, pulmones, linfonódulos, piel, tejido subcutáneo y tejido periarticular. En la mayoría de ocasiones es necesario la histopatología para llegar al diagnóstico de esta neoplasia, y en otros casos el estudio inmunohistoquímico con la detección del marcador CD204 (también conocido como MSR1: macrophage scavenger receptor 1). En muchas ocasiones, el sarcoma histiocítico es confundido con el histiocitoma fibroso maligna y sarcomas pleomórficos. La apariencia citológica de las células neoplásicas revela células de morfología redondeada y fusiforme (apariencia mesenquimal). Las células gigantes multinucleadas pueden visualizarse recordando al sarcoma anaplásico con células gigantes. Las células redondas individuales contienen abundante citoplasma basófilo con fina vacuolización. Con relación a los núcleos, éstos aparecen vesiculados, redondos a identados con 1 o más nucléolos. Los criterios de malignidad más frecuentemente observados son anisocitosis, anisocariosis, macrocariosis, multinucleación y nucléolos múltiples.

Sarcoma pleomórfico

Representa una de las neoplasias menos frecuentes en caninos y felinos (más raro en caninos), 0.34% de todos los tumores en caninos e involucra hasta el 3% de los tumores en felinos. El sarcoma pleomórfico, anteriormente conocido histiocitoma fibroso maligno o sarcoma anaplásico con células gigantes, está constituido por células fusiformes pleomórficas siendo su histogénesis controversial, probablemente su linaje sea de origen miofibroblástico. La presentación clínica puede limitarse a la superficie corporal o podría presentarse también en órganos abdominales, pulmones, linfonódulos y otros órganos. Las muestras citológicas exhiben una moderada a alta celularidad constituidas por tres distintas poblaciones de células neoplásicas interespaciado con una pequeña cantidad de matriz eosinofílico. Una de las poblaciones está caracterizada por células en forma de huso con citoplasma basófilo, núcleo elongado, cromatina reticular y nucléolos múltiples, otra población celular se caracteriza por presentar una forma entre redondeada y fusiforme, células gigantes multinucleadas con citoplasma intensamente basófilo (a veces vacuolizada), 10 a 45 núcleos y múltiples nucléolos. Estas dos poblaciones presentan numerosos criterios de malignidad La última de las poblaciones celulares incluyendo amoldamiento nuclear, relación N:C alta (1:1-2), marcada anisocitosis, anisocariosis y cariomegalia. La tercera población celular es escasa y está caracterizado por células redondas con núcleos excéntricos (semejantes a los histiocitos). También pueden llegar a observarse otras células como linfocitos y células plasmáticas.

A continuación se indican las diferencias citológicas más importantes entre el linfoma multicéntrico, cutáneo, TVT, mastocitoma, sarcoma histiocítico y sarcoma pleomórfico:

Tabla 1. Hallazgos citológicos diferenciales para algunos tumores de presentación multicéntrica

HALLAZGO CITOLÓGICO	LINFOMA MULTICÉNTRICO	LINFOMA CUTÁNEO	TUMOR VENÉREO TRANSMISIBLE	MASTOCITOMA	SARCOMA HISTIOCÍTICO	SARCOMA PLEOMÓRFICO
Tipo de tumor	De células redondas – Linfoide	De células redondas – Linfoide	De células redondas - histiocítico	De células redondas - Mastocito	De células dendríticas- Mieloide	Mesenquimal - Miofibroblástico
Celularidad	Alta	Moderada a alta	Abundante	Variable	Moderada a alta	Moderada a alta
Tamaño celular	Variable	Mediano a grande	Pequeño a mediano	Pequeño a mediano	Mediano a grande	Grande
Forma celular	Redonda a ovalada	Redonda a ovalada	Redondeada	Redondeada	Redondeada a fusiforme	Muy pleomórfica
Relación N:C	Alta	Alta	Alta	Moderada a alta	Moderada	Moderada a alta
Citoplasma	Intensamente basófilo finamente granular	Basófilo finamente granular con vacuolización variable	Azul grisáceo frecuentemente vacuolizado	Azul grisáceo con granulación púrpura	Basófilo finamente vacuolizado	Basófilo
Núcleo	Frecuentemente ovalado con/sin indentación	Ovalado con/sin muesca, monocitoide, pleomórfico	Redondeado	Redondeado	Redondeado con/sin indentación, bi y multinucleación	Redondeado con/sin indentación, bi y multinucleación
Otros hallazgos	Cuerpos linfoglandulares, neutrófilos	Cuerpos linfoglandulares, neutrófilos	Plasmocitos, linfocitos, mastocitos	Eosinófilos	Neutrófilos	Matriz eosinofílica, linfocitos y plasmocitos

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6.1.6. CITOLOGÍA DE TVT: MÁS ALLÁ DE UN SIMPLE TUMOR DE CÉLULAS REDONDAS

Introducción

El tumor venéreo transmisible (TVT) fue propuesto de haberse originado desde hace 200 a 2500 años; sin embargo y con la evidencia molecular, se estima que este tumor tiene entre 6000 y 11,000 años de antigüedad. El tumor ha sido reconocido como un tumor retículo-endotelial benigno (células de origen mieloide) que crece principalmente en genitales externos de machos y hembras y es naturalmente transmitido en el perro por el coito, mordedura o lamido de las áreas que comprometen el tumor. Las células del TVT canino se pueden trasplantar experimentalmente entre perros e incluso a otros miembros de la familia Canidae. A lo largo de la historia, el TVT ha recibido múltiples denominaciones tales como Granuloma venéreo, sarcoma infeccioso, linfosarcoma transmisible, condiloma canino o tumor de Sticker. El TVT canino junto con la enfermedad de los tumores faciales del demonio de Tazmania y el cáncer tipo leucemia de la almeja de concha blanda son los únicos tipos de cáncer transmisibles de forma natural de origen clonal. La metástasis es rara, siendo los grupos más susceptibles los cachorros y los pacientes inmunocomprometidos.

La frecuencia de presentación es similar en machos y hembras, siendo comúnmente reportada en animales de 2 a 5 años de edad, favoreciendo los climas temperados la mayor ocurrencia de esta enfermedad.

Presentación clínica

En machos, el TVT se localiza usualmente en la parte caudal del pene, desde el cuerpo al bulbo, y raramente en el prepucio. En hembras, el desarrollo de TVT se forma mayoritariamente en la pared posterior de la vagina, y entre el vestíbulo y la vagina. Las lesiones aparecen desde pequeñas nodulaciones (1 a 3 mm de diámetro) superficiales variando de rosa a rojo en estadíos iniciales, ya en estadíos más avanzados el volumen puede aumentar considerablemente (hasta los 15 cm de diámetro) y tener la apariencia de coliflor, siendo frágil al tacto y hemorrágico, esto último por ulceración que se puede acompañar de descargas sanguinolentas.

El diagnóstico puede ser realizado dependiendo de la anamnesis, localización de la masa, perineo muy contaminado, descarga sanguinolenta y la apariencia típica del tumor, citología y hallazgos histopatológicos. Otras condiciones deberían también ser consideradas como parte del diagnóstico diferencial como cistitis, prostatitis y uretritis.

La citología en el diagnóstico del TVT

La citología debe ser el método de elección para el diagnóstico de TVT canino dado que la técnica es simple, económica, mínimamente invasiva, poco dolorosa y

asimismo, produce mucha menor distorsión de la morfología celular comparado a las muestras colectadas por biopsia. En general, podemos definir las características citomorfológicas del TVT independientemente del subtipo citológico:

Tamaño de la célula: 15 – 30 µm

Forma de la célula: redonda y/o oval

Contorno: Bien definido

Color del citoplasma: azul pálido y azul grisáceo

Cantidad de citoplasma: Moderada c/s vacuolas

Forma del núcleo: Redondo y oval (indentación variable)

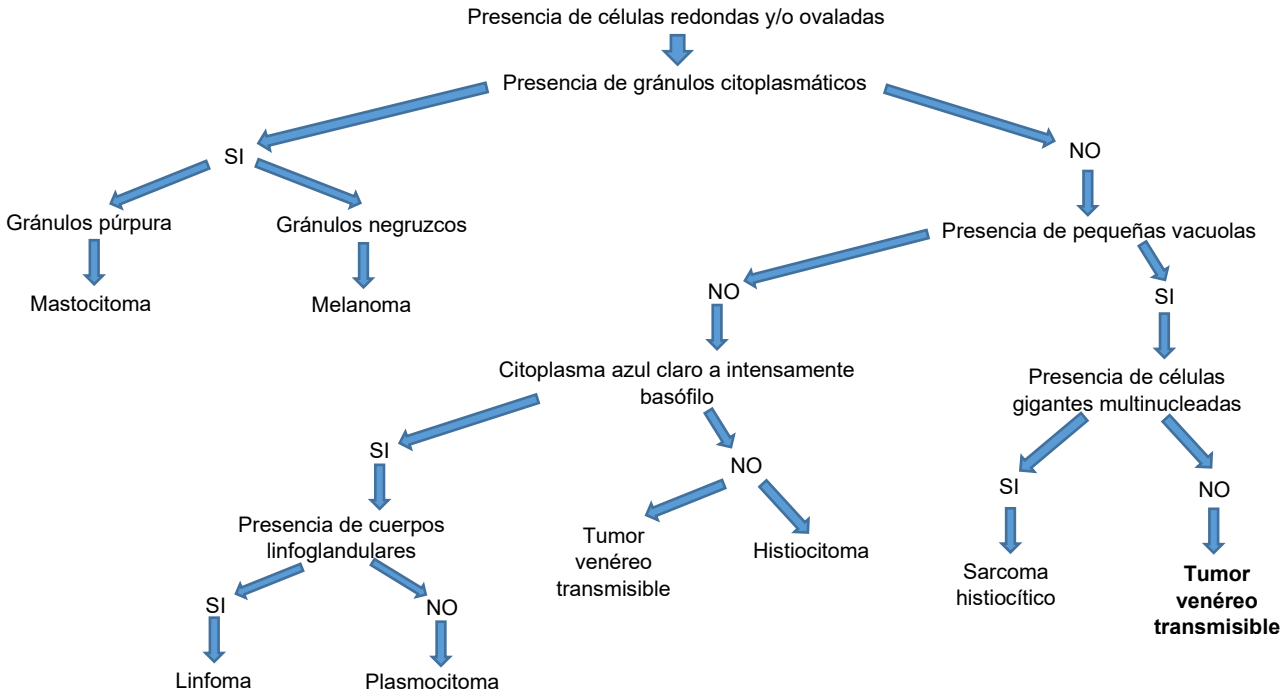
Tipo de cromatina: granular a gruesa

Presencia de nucléolos: 1 a 3

Relación núcleo: citoplasma: Moderada

Las muestras citológicas de TVT canino son generalmente multicelulares, presentando criterios de malignidad como anisocitosis, anisocariosis, figuras de mitosis (bizarras en muchos casos), nucléolos múltiples, basofilia y vacuolización citoplasmática. Otras células que pueden aparecer en los preparados son los linfocitos, macrófagos, células plasmáticas y ocasionalmente mastocitos. Las vacuolas citoplasmáticas juegan un rol importante en el diagnóstico, para distinguir el TVT de otros tumores de células redondas, como mastocitoma, linfosarcoma e histiocitoma; sin embargo, en algunos casos no se evidencian las vacuolas típicas, por lo que el diagnóstico se hace algo más complicado.

Algoritmo para el diagnóstico de células redondas y TVT



¿Por qué se observan linfocitos y células plasmáticas en citologías de TVT?

Experimentalmente, se conoce que el TVT canino trasplantado está clínicamente caracterizado por una fase progresiva (P), estacionaria (S) y regresiva (R). Durante la fase P existe un incremento rápido del tumor que llega a ser una masa exudativa tipo coliflor, pedunculada. Microscópicamente hay abundancia de células mitóticas y pocos linfocitos infiltrantes. Durante la fase S, el crecimiento del tumor es considerablemente lento y microscópicamente hay menos células cancerígenas en mitosis, más células mitóticas y linfocitos infiltrantes. Finalmente, durante la fase R, las células cancerígenas desaparecen, colapsa el estroma tumoral, hay deposición de colágeno y son abundantes los linfocitos infiltrantes.

Diferencias citomorfológicas de los tipos de TVT

	LINFOCITOIDE	**PLASMOCITOIDE**	**MIXTO O LINFOPLASMOCITOIDE**
Celularidad	Más del 60% de células son redondeadas	Más del 60% de células son ovaladas	Ni el tipo linfoide ni el plasmocitoide excede el 59% del total
Cantidad de citoplasma	Escaso citoplasma finamente granular	Moderada a abundante	Variable
Características de las vacuolas	Presencia de vacuolas pequeñas	Marcada vacuolización frecuente	Variable
Localización nuclear	Central	Excéntrica	Central y excéntrica
Relación N:C	Alta	Moderada	Variable
Nucléolos	1 a 2 nucléolos distinguibles	Variable	Variable
Patrón de Cromatina	Reticular a gruesa	Reticular a gruesa	Reticular a gruesa
Mitosis	Relativamente frecuentes	Más frecuente que en linfocitoide	Mitosis frecuente

Según diversos autores, el patrón citomorfológico plasmocitoide es el más frecuente y se asocia a presentaciones más agresivas (Amaral et al., 2007) y suelen ser de mayor antigüedad, siendo su localización más frecuente en tejido extragenital.

Las características citomorfológicas en TVT genital es similar al extragenital y las metástasis son más frecuentes en casos de TVT plasmocitoide; mientras que, el subtipo linfocitoide está más asociado a la presentación genital y los infiltrados mononucleares son más intensos en comparación a los otros subtipos.

Se sabe que por citología existe una mejor caracterización del tipo celular así como de los criterios de malignidad y se preserva mejor la morfología celular (menor distorsión de la imagen). Y aunque se ha avanzado mucho en la caracterización citomorfológica de TVT aún quedan brechas por resolver tales como:

Caracterización citológica e histopatológica para el estudio de la evolución de la neoplasia durante el tratamiento quimioterápico.

Correlación a gran escala entre la citología y la histopatología de los 3 tipos citomorfológicos de TVT.

Estudios con mayor casuística para establecer correlación entre el tipo citomorfológico y la agresividad de la neoplasia.

Estudios de expresión génica de marcadores tumorales en casos de reciente aparición y de mayor antigüedad y su valoración con relación a la respuesta al tratamiento.

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JUAN DIEGO ASCENCIOS

7.1.1. BAJO PRESIÓN: TAPONAMIENTO CARDIACO APRENDE A MANEJAR LA URGENCIA

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GENERALIDADES

El pericardio es la membrana que envuelve al corazón. Está conformado por el pericardio fibroso y seroso (capa visceral y parietal). Entre ellos se encuentra el líquido pericárdico.

El taponamiento cardiaco se define como el colapso diastólico del atrio derecho y puede ser producido por una reducida o abundante cantidad de líquido libre en el interior del saco pericárdico.

Una efusión pericárdica repentina puede taponar potencialmente el corazón, esto debido al poco tiempo que las fibras elásticas que lo conforman tienen para relajarse.

La triada de Beck se define como la presentación de 3 signos clínicos (ruidos cardiacos apagados, venas yugulares distendidas y pulso femoral débil) que nos ayudan a inferir la existencia de un taponamiento cardiaco muchas veces acompañado de ascitis. Además, pueden presentarse otros signos clínicos como hipotermia, colapso y disnea dependiendo del factor etiológico.

FISIOPATOLOGÍA

Las cuatro cámaras del corazón tienen diferentes presiones. El atrio derecho es la más débil con una presión de 5 mmHg, por este motivo es la primera en colapsar en caso de un incremento de la presión intrapericárdica produciéndose el estancamiento del retorno venoso traído por las venas cava craneal y caudal. El resultado es la ingurgitación de las venas yugulares y la aparición de ascitis progresiva.

Causas más comunes: Idiopática y neoplásica (hemangiosarcoma del atrio derecho).

DIAGNÓSTICO

- Radiografía: no es la más precisa, pero es la más extendida. Se puede observar la silueta cardiaca.
- Ecografía cardiaca: Prueba diagnóstica mas recomendada. Se aprecia un halo anecogénico alrededor del corazón indicativo de líquido libre intrapericárdico. Además, se puede apreciar la deformación del atrio derecho (colapso diastólico) confirmándose así el taponamiento.
- Electrocardiograma: Muestra ondas R de distinta amplitud (alternancia eléctrica).

TRATAMIENTO

- La utilización de furosemida está contraindicada en pacientes con taponamiento cardiaco ya que la presión arterial se encuentra disminuida y la deshidratación causada por este fármaco agudizaría esta condición.

- Pericardiocentésis: Procedimiento de extracción de líquido pericárdico.

Materiales: Guantes (de examinación o quirúrgicos), 1 jeringa grande de 20-50 ml, catéter intravenoso y llave de doble vía con extensión.

Método: El paciente debe estar acostado en decúbito lateral izquierdo. El área de trabajo debe estar limpia. Realizar un bloqueo regional infiltrando lidocaína o bupivacaína para tener un buen manejo del dolor. Realizar la punción ecoguiada y la extracción del líquido.

- ¿Cuándo se debe realizar?

1. En caso de taponamiento confirmado por ecocardiografía siempre que haya espacio suficiente para extraer el líquido por punción. Se debe tener al menos 1 cm.
2. Muestreo diagnóstico.

- ¿Cuándo no se debe realizar?

1. Efusión reducida
2. Hemopericardio descontrolado

- Pericardiectomía: Total o subtotal (ventana en el pericardio). El líquido que se producía en el pericardio será drenado por el sistema linfático pleural. La intervención se puede realizar por laparoscopia, un procedimiento mínimamente invasivo y seguro.

7.1.2. CRITERIOS DE ABORDAJE DEL PACIENTE GERIATRA CON TOS

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GENERALIDADES

La tos es uno de los signos clínicos que recibimos con más frecuencia en nuestro consultorio. La evaluación sistemática del paciente debe ser empleada para evitar errores de diagnóstico y tratamiento.

La tos de los pacientes cardiacos se produce cuando el atrio izquierdo crece y comprime el bronquio principal izquierdo. Esto genera un estímulo mecánico que deriva en ataques de tos. Durante la noche los pacientes cardiacos con estas características tosen más porque el tórax esta comprimido, agravando la condición previamente mencionada. La presencia de un soplo no indica inmediatamente que la tos se debe a una cardiopatía. El soplo puede existir sin un atrio izquierdo dilatado al momento de la evaluación.

VLAS – VERTEBRAL LEFT ATRIAL SIZE

Es un índice relativamente nuevo que se basa en la estimación del tamaño de atrio izquierdo en una radiografía para saber si ha crecido o no con el objetivo de poder distinguir la tos de origen cardiaca de la extra-cardiaca.

1 – Se traza una línea desde el centro del aspecto más ventral de la carina hasta el aspecto más caudal del atrio izquierdo donde se intersecta con el borde dorsal de la vena cava caudal.

2 – Se traza una línea de la misma longitud de la anterior desde el borde craneal de la cuarta vertebra torácica hacia caudal y paralela al canal vertebral.

3 – El VLAS es la longitud de de la segunda línea expresada en cuerpos vertebrales (CV) y no debe ser mayor de 2.3 CV.

CAUSAS MÁS FRECUENTES DE TOS

Dentro de estas podemos contar al colapso dinámico de vías respiratorias y a la bronquitis crónica. La primera está referida al cierre brusco de la tráquea, bronquios o ambas estructuras como generadores de tos. La evaluación por endoscopia permite un excelente diagnostico que muchas veces no puede ser alcanzado con estudios radiológicos.

El diagnostico de bronquitis crónica puede ser tomado cuando se tiene un paciente con una tos de más de dos meses de duración habiéndose descartado otras etiologías de manera precisa y sistemática.

ÍNDICE ATRIO-AORTA

Es un indicador que se toma a partir de la obtención de los diámetros de la aorta y del atrio izquierdo realizando un corte de base cardiaca a nivel de la ventana paraesternal derecha. Los valores máximos normales al dividirse el diámetro del atrio izquierdo y el aórtico son de 1.59 para caninos y 1.50 para felinos. El edema pulmonar cardiogénico en nuestra experiencia se suele presentar en pacientes con enfermedad valvular mitral con índices Ai:Ao de 1.8 para arriba.

NEOPLASIAS EN ECOGRAFÍA DE TÓRAX

Pueden presentarse también como factores etiológicos tusígenos. La tos en este caso se produce cuando una neoformación comprime una estructura del árbol bronquial o pulmonar en general produciendo el estímulo de tos.

CONCLUSIONES:

- Debemos realizar más endoscopias de vías respiratorias.
- El VLAS es más efectivo que el VHS para descartar una tos cardiogénica.
- La existencia de un soplo no basta para afirmar que la tos es cardiogénica.
- La ecografía pulmonar puede mostrar hallazgos interesantes.

7.1.3. ECOGRAFÍA PULMONAR: EL ABC QUE DEBES CONOCER

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GENERALIDADES

La ecografía pulmonar es un método confiable y sencillo de poder detectar artefactos a nivel de la exploración pulmonar y torácica que nos permite realizar diagnósticos en poco tiempo.

Las regiones a ser exploradas se dividen en caudo-dorsal, peri-hiliar, media y craneal. Cada una de estas cuatro debe ser revisada en ambos hemitórax, tanto en caninos como en felinos.

La metodología se basa en llegar al punto de interés (espacio intercostal) y mover hacia craneal y caudal un espacio intercostal adicional para obtener 3 espacios evaluados por región.

SIGNO DEL COCODRILO

Conformado por la sobra acústica de las costillas y la línea hiperecogénica entre estas (interfase pleuropulmonar). Esta última debe poseer un deslizamiento o “sliding” bastante apreciable y referido al movimiento de la pleura parietal con la visceral. La ausencia de estos hallazgos podría referir la existencia de una patología.

LÍNEAS A

Estas son normales, ecogénicas, paralelas, equidistantes y representan la presencia de aire dentro del pulmón.

LÍNEAS B

Estas son verticales, ecogénicas, se originan a partir de la interfase pleuro-pulmonar, se mueven con la respiración e impiden ver las líneas A. Se producen por la presencia de una patología intersticial alveolar a nivel del pulmón y por lo general tiene que ver con la presencia de sangre (coagulopatía, trauma), secreción purulenta (neumonía) o edema (cardiopatía, hepatopatía, neoplasia) a este nivel. La detección de líneas B debe ser registrada y cuantificada. Usualmente se divide en la observación de 1, 2, 3, más de 3 e infinito (consolidación de más de tres líneas B).

ÍNDICE ATRIO-AORTA

Es un indicador que se toma a partir de la obtención de los diámetros de la aorta y del atrio izquierdo realizando un corte de base cardiaca a nivel de la ventana paraesternal

derecha. Los valores máximos normales al dividirse el diámetro del atrio izquierdo y el aórtico son de 1.59 para caninos y 1.50 para felinos. El edema pulmonar cardiogénico en nuestra experiencia se suele presentar en pacientes con enfermedad valvular mitral con índices Ai:Ao de 1.8 para arriba.

EFUSIÓN PLEURAL

Se aprecia contenido compatible con liquido libre en la cavidad torácica acompañado de elementos ecogénicos en suspensión posiblemente compatibles con fibrina, coágulos, etc. Se debe tener mucho cuidado con no confundir este hallazgo con una efusión o taponamiento cardiaco.

SIGNO DEL DESGARRO

Se evidencia cuando la interfase pleuro-pulmonar se ve interrumpida. Por lo general se debe a la presencia de tejido pulmonar parcialmente aireado y parcialmente húmedo. Pueden formarse líneas B a partir del área afectada. Se relaciona a la presencia de broncogramas aéreos positivos en las radiografías de tórax. Se sugiere descartar la presencia de neumonías.

CONCLUSIONES

La ecografía de tórax es extremadamente útil e inocua para nuestros pacientes

A través de la evaluación de artefactos ecográficos podemos llegar a conclusiones fiables.

No requiere mucho tiempo de ejecución y es fácil de aprender.

7.1.4. ENFERMEDAD MITRAL: PRESENTE Y FUTURO DE LA CARDIOPATÍA DE TODOS LOS DÍAS

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GENERALIDADES

La sangre pasa por la válvula mitral hacia el ventrículo izquierdo en dirección a la circulación general. Un buen cierre de válvula permite que la sangre siga avanzando y que no retorne. En la enfermedad mixomatosa valvular mitral (EVM) la válvula mitral se vuelve más gruesa produciéndose un déficit de coaptación. Este problema es producido por un defecto genético que al ser expresado facilita la proliferación de proteoglicanos a nivel de la capa espongiosa de la válvula mitral.

SIGNOS CLÍNICOS

Los signos clínicos más frecuentes son tos, disnea, ortopnea, respiración abdominal, edema pulmonar, síncopes (pérdida de conciencia acompañada y de tono muscular con o sin presencia de vocalización y extensión de los miembros anteriores).

FISIOPATOLOGÍA

Disminución del flujo anterógrado causada por el mal cierre de la válvula.

La reducción de cantidad de sangre que esta yendo a los órganos activa el Sistema Renina Angiotensina Aldosterona, esto genera la elevación de la presión arterial, pero a la larga la excesiva retención de líquido en el sistema circulatorio.

DIAGNOSTICO

En el ecocardiograma vamos a tener una relación atrio aorta incrementada (a partir de 1,6 es patológica). La técnica Doppler nos permitirá evidencia el flujo regurgitante mitral a nivel del atrio izquierdo y realizar la medición de la velocidad pico y gradiente de presión correlativos. La evidencia de líneas B concurrente podría indicar la aparición de edema pulmonar cardiogénico en casos avanzados.

Clasificación ACVIM (American College of Veterinary Internal Medicine) de la EVM

- Clase A: Pacientes de raza predispuesta. Deben tener chequeos periódicos.

- Clase B: La válvula no cierra adecuadamente y se ausculta un soplo o B1: Corazón sin cambios remodelatorios. Debe pasar por chequeos periódicos.
  - B2: Corazón con cambios remodelatorios.

- Clase C: Pacientes que han presentado un evento de edema pulmonar.

o Ca: Atención de urgencia en una clínica (Oxigeno, diuréticos, inotrópicos y nitratos)

o Cc: Están siendo tratados en su hogar (Inodilatadores, vasodilatadores y diuréticos)

- Clase D: Individuos refractarios a la medicación de la clase anterior.

Recientemente se ha incorporado como opción terapéutica resolutiva la cirugía de válvula mitral que consiste en la restauración de las cuerdas tendinosas rotas o elongadas junto con la anuloplastía del anillo aurículo ventricular izquierdo. El resultado es la drástica disminución del índice atrio aorta, así como de la fracción regurgitante.

CONCLUSIONES:

La enfermedad valvular mitral es la cardiopatía más frecuente en caninos de raza pequeña a mediana.

Los lineamientos introducidos por el consenso del ACVIM permiten estandarizar el manejo de los pacientes afectados.

La restauración quirúrgica de la EVM es una técnica curativa y confiable que permite extender considerablemente la expectativa de vida de los caninos afectados.

7.1.5. Implantación de marcapasos en caninos: Por qué, ¿cómo y cuándo?

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GENERALIDADES

La implantación de marcapasos en Perú es una técnica reciente en medicina veterinaria que nos permite estabilizar la frecuencia cardiaca de pacientes con cuadros bradiarrítmicos.

Se indica en algunos casos de pausas sinusales, síndrome del seno enfermo, bloqueos AV de segundo grado con presencia de sintomatología clínica y en todos los casos de bloqueos de AV de tercer grado.

Lo pacientes afectados por frecuencias cardiacas bajas suelen presentar episodios de sincope y/agitación durante el ejercicio físico leve, moderado o intenso.

El diagnostico de condiciones de bradicardia se facilita a través del uso de la electrocardiografía y la monitorización Holter (ECG de 24 horas de duración) en caso sea necesaria.

Teniendo en cuenta el peso y talla del paciente se suelen utilizar marcapasos pediátricos monocamerales (estimulación exclusiva a nivel ventricular) fabricados para medicina humana.

Los requisitos necesarios para realizar el proceso de manera exitosa pasan por poseer un equipo de trabajo experimentado, acceso a un arco en C (fluoroscopía) y una familia que entienda los riesgos y beneficios del procedimiento de implantación.

TÉCNICA DE IMPLANTACIÓN:

- Afeitar el área del cuello y el dorso del lomo
- Poner al paciente bajo anestesia
- Realizar una incisión a nivel cervical cercana a la vena yugular derecha
- Exponer la vena yugular derecha
- Introducir un catéter IV dentro de la yugular y pasar la guía de metal del introductor a través de este.
- Verificar por fluoroscopía la ubicación del introductor
- Retirar la guía metálica del introductor
- Introducir el peel-away con el electrodo a través del introductor y verificar su posición por fluoroscopía.

- Iniciar el ingreso del extremo activo del electrodo en el ventrículo derecho. Tener mucho cuidado de no perforar la pared.

- Tunelizar el camino sobre el dorso del lomo para llevar el electrodo hasta el medio de las dos escapulas e introducir el generador en el bolsillo subcutáneo previamente preparado. Unir el electrodo al generador.

- Verificar con el programador del marcapasos que los umbrales y características sean las adecuadas.

MANEJO POSPRECEDIMIENTO

El paciente debe ser internado por un mínimo de 3 a 5 días en la clínica para evitar la formación de seromas o infecciones alrededor del generador del lomo que puedan complicar el proceso. Para estos fines se recomienda el uso de un analgésico, un antibiótico y un antiinflamatorio, así como el cambio periódico de vendajes compresivos.

La revisión electrocardiográfica debe realizarse regularmente en el internamiento ya que uno de los problemas más frecuentes se refiere al desprendimiento del electrodo de la pared del ventrículo derecho, lo que podría significar la muerte del paciente en el peor de los casos.

CONCLUSIONES:

La implantación de marcapasos es una técnica necesaria y factible para corregir arritmias en caninos.

Se debe tener el equipamiento y personal adecuados para lograr el éxito del procedimiento.

Una de las limitantes principales es el costo del procedimiento. Se espera que pueda ir mejorando progresivamente.

7.1.6. DOLOR Y PARÁLISIS AGUDA DEL TREN POSTERIOR: TROMBO EMBOLISMO AÓRTICO FELINO

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INTRODUCCIÓN

- Es la cardiopatía mas frecuente en felinos. Es importante porque está muy subdiagnosticada.
- A la clínica se presenta un felino respirando rapido y con la boca abierta. o Felino con miembro posterior izquierdo arrastrandolo y gritando. o Felino con sincope.
- Felino con problema cardiaco no tosen. Por lo que si el felino tose o tiene arcadas es probable que sea asma felino.

SIGNOS CLINICOS

- Disnea o Sincope o Claudicación

CARDIOMIOPATÍA HIPERTRÓFICA FELINA

Hipertrofia concentrica del ventriculo izquierdo -> insuficiencia diastólica.

Histologicamente las celulas cardiacas se hipertrofian, estan desordenadas y muy poco irrigadas.

CAUSAS

- Primaria: Hay razas más predispuestas a expresar la cardiopatía a partir de cierta edad, sin embargo, los mestizos también pueden expresar la enfermedad.
- Secundaria: Hipertensión arterial crónica (hipertiroidismo, enfermedad renal crónica), sobrecarga de presión (estenosis aórtica).

TIPOS

- Obstructiva: Hay engrosamiento a nivel del septo, haciendo que la sangre acelere y succione parte de la válvula mitral generándose un soplo. (SAM: Movimiento sistólico anterior)
- No obstructiva

CAUSAS DE SOPLO

- Hipertiroidismo
- Fisiológico (obstrucción dinámica del tracto de salida del ventrículo derecho) o Cardiopatías
- Hipertensión
- Anemia

SOPLO VS GALOPE

El soplo es muy ambiguo para diagnosticar cardiopatías, a diferencia del galope que es certero.

El galope se produce porque el ventrículo izquierdo esta tan rígido e hipertrofiado que la sangre choca con este y suena, que produce un tercer ruido cardíaco (se escucha como un caballo galopando en el corazón a la auscultación)

RADIOGRAFÍA

Es el método menos sensible para diagnosticar CMHF, pero en la radiografía en vista VD, la silueta cardiaca se ve como corazón de San Valentín.

El atrio izquierdo va a aumentar de tamaño debido a que hay sobrecarga de volumen (sangre), porque no va a poder entregar toda la sangre al ventrículo izquierdo ya que este está hipertrofiado y sin capacidad de distenderse.

ECOCARDIOGRAFIA

Es el método mas sensible para diagnosticar CMHF.

Se busca medir el grosor del ventrículo izquierdo el cual debe ser mayor o igual de 6mm.

El atrio izquierdo suele presentarse aumentado de tamaño lo cual predispone a la aparición de edema pulmonar y efusión pleural.

Vet blue: Ecografía pulmonar se observa la presencia de líneas B, compatible con una patología intersticial alveolar (edema pulmonar).

TROMBOEMBOLISMO AÓRTICO

- Atrio dilatado
- Daño endotelial
- Activación del sistema de coagulación

La formación de un coágulo en el atrio izquierdo puede pasar al ventrículo izquierdo y salir por la aorta. Este trombo llega hasta las arterias iliacas las cuales irrigan a los miembros posteriores, obstaculizando el flujo sanguíneo.

TRATAMIENTO

- Edema pulmonar -> Oxígeno y furosemida
- Betabloqueantes -> Atenolol
- Atrio izquierdo grande y trombos -> Clopidogrel, aspirina