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5 Ways to Get Started With a Fear-Free Practice

Decontaminating the Poisoned Patient

Accredited CPD

Canine Atopic Dermatitis - A Practical Approach A to Z

Also in this issue:
Thyroid Hormone Assays | Vit B_{12} Deficiency | Thoracostomy Tube Placement

Editor's Note

Reducing patient fear in our practices seems a self-evident concept - yet many small adjustments which could change things radically for the patient are not being made. Purely I think, because we are used to things in a certain way. Think out of the box. See things from a dog or cats perspective. Put yourself in your patients' position and then see how small changes can make a great impact on your staff and your clients.

The CPD article in this edition is a real marathon - the A-Z of the practical diagnosis and treatment of canine atopy. We all have these animals in our practices and I know the sense of futility felt when the poor dog just cannot stop itching and scratching no matter what we try. Persevere, read the article.

The trick is in being thorough...not something impatient vets are always good at! Advances in the evaluation of the barrier function of the skin as well as new immunomodulating agents other than cortisone are highlighted.

Again - thank you to those who contributed to this edition.

Regards Liesel

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- Canine Adverse Food Reaction (CAFR)

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5 Ways to Get Started with *Fear-Free* Practice

Aug 01, 2015
By Jessica Vogelsang, DVM
DVM360 MAGAZINE

"Fear and anxiety are nosocomial infections, and they are zoonotic. We created this problem. Patients acquired it at our hospital and it was spread by other pets and staff, then the owners catch it." - Dr. Jonathan Bloom

This Toronto hospital has clients beating down the doors requesting a lower-stress approach to veterinary care.

In an uncertain economic climate when veterinary visits are on the decline, Willowdale Animal Hospital in Toronto is enjoying unprecedented success. Clients are raving, staff morale is the highest it's ever been and business is booming. Instead of cajoling and pleading with clients to bring their pets through the doors, Jonathan Bloom, DVM, has them lined up with smiles on their faces.

Many practices with excellent staff and medicine are struggling these days, but Bloom's hospital has a secret ingredient: It has embraced the concept of Fear-Free practice, and as far as Bloom is concerned, you should too.

"I started about seven or eight years ago," says Bloom, who will be speaking at CVC Kansas City Aug. 30. "It started with x-rays. I looked at my staff and saw that they'd have to take a whole bunch of views to get two good images."

Wondering how he could improve their efficiency, he investigated. "I realised dogs and cats don't like having x-rays taken. There's nothing natural about being in a dark room, on a hard, cold table, having your limbs held in four different directions with the viselike grip of lead-lined gloves by people wearing aprons and collars." He decided to try a different approach.

Bloom implemented a policy that no pet would be restrained for radiographs. "They're either so sick they don't move, or they're going to be sedated," he says. The results were immediate. "There were no retakes, the pets weren't stressed, and it was better for the staff," who reduced their radiation exposure while becoming more efficient.

Happy with the results and the owner responses, Bloom made his next decree: "Don't give owners an update from the phones in the ICU. No one wants to hear screaming cats." About an hour later, Bloom says, he reflected on what he had just said and realized that was the wrong approach. "We shouldn't be letting pets get that upset in the first place," he says.

Bloom started to ask himself and his team the following: If the pet could talk, what would he or she say? It wasn't that hard to figure out. "We create mental health problems," he says. "If you need two 150-pound people to trim the nails on a 10-pound pet, that's a problem." Bloom and his staff began experimenting with various techniques to give pets a less stressful experience, and they were buoyed by a receptive client response. "Pet owners may not be able to identify dental disease or obesity, but they are experts at identifying fear and anxiety in their pets," he says. "Owners are on it. They are grateful for any effort made to help their pets."

He finds the veterinarians harder to convince than the owners—at least at first. "Here's the problem with training vets: They will spend a day learning the best insulin for the two diabetic pets they see a year. But five of the next 10 patients will be suffering fear and anxiety," he says.

It took some time, but the other veterinarians at Willowdale also came on board. When they did, Bloom says, the wait was worth it. "It's fun to watch them get it," he says. "They go from skeptical to 'That was sort of neat!'"

During the last seven years, Bloom's team has developed a multimodal approach to Fear-Free veterinary practice. With nine other veterinarians on staff, finding common ground and common purpose was challenging at first. "Not everything works every time," he says. "If Plan A doesn't work, it doesn't mean there's no treatment; it just means we need a different approach."

Here is Bloom's toolbox of techniques that have worked wonders in his own clinic.

1 Create a Fear-Free culture

In Bloom's practice, everyone has a role to play in creating the right environment for pets and owners, and every employee is encouraged to give input on how to minimise patient stress. No part of the patient-client experience is left unexamined, from the choice of vaccines to the way appointments are structured. Here's what Willowdale has done to maximise calm most successfully.

- Designate dog- and cat-only examination rooms. Pets, especially cats, are often overstimulated when visiting the veterinary hospital. Imagine the experience of an indoor-only cat from the car ride to the hospital—different lighting, different colors, different sounds and different smells. Let's at least try to keep cats in an as familiar environment as possible by providing cat-only rooms. If you're a small practice with limited number of exam rooms, there's an alternative, Bloom says: cat-only appointment hours. "There's no reason a one-doctor practice can't designate 2 to 4 p.m. once a week as cat-only appointment hours," he says. Many cat owners will choose to come in at other times, but those owners with highly fearful cats will appreciate that option.
- Keep doctors on time. Veterinarians at Willowdale make running on time a priority so as to reduce clients' and patients' waiting time in the exam rooms. But if a delay is unavoidable, a few minutes for pets and owners in the quiet environment of an exam room is preferable to the waiting area.

2 Have the appointment begin at home

The front desk team at Willowdale sends out reminders a week in advance with a list of ways for clients "to make their pet's visit an enjoyable experience." Bloom has found that empowering clients to take action ahead of time creates a very effective partnership. Owners are instructed to do the following:

- Bring pets hungry. When medically appropriate, withhold food for several hours before an appointment so the patient is highly food-motivated.
- De-stress the carrier. Make the carrier a fun place to be: use it as a regular place to feed treats, deliver toys and spray it with pheromones such as Adaptil or Feliway.
- Use tools. Bring calming items such as Thundershirts along to the appointment if clients own them.
- Bring prescribed pills and chewables. Be ready with sedatives if the pet normally requires them.
- Acclimate nervous patients. If Bloom senses that a pet is anxious during an appointment, he encourages the owner to bring the pet to the hospital between appointments to play and eat a few treats to create a positive association. Another idea—one that Bloom says his practice doesn't do but should—is to have clients bring all new patients into the clinic several days before the appointment to let them play in the hospital.

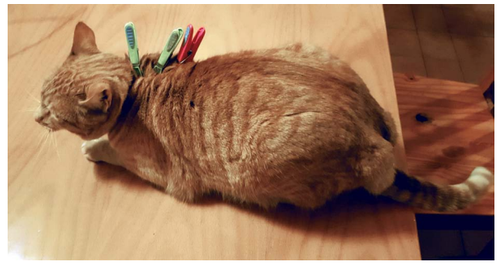


Figure 1: "Clipnosis": non-painful restraint using pegs. Effective only in cats

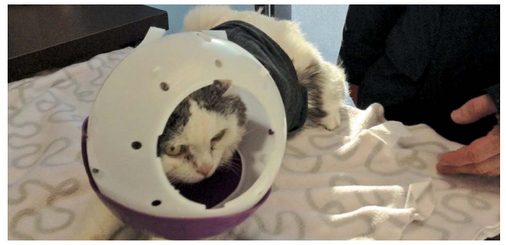


Figure 2: Air muzzles are effective in cats and small brachycephalic dogs

3 Control pets by minimising stress instead of maximizing restraint

As far as Bloom is concerned, the less restraint, the better. Here are some of his favorite environmental modifications and tips:

- Lots of food treats throughout the entire appointment.
- Calming music, such as "Through a Cat's Ear," in the exam rooms.
- Pheromone diffusers placed throughout the hospital.
- Massage on the pet's head.
- Clipnosis, a small plastic scruff clip that resembles a chip bag clip. (Figure 1)
- Vaccines that lower stress during administration—for example, the new oral transmucosal Bordetella vaccine rather than the intranasal or injectable forms.
- Air muzzles for cats and small brachycephalic dogs: instead of a tightly fitting piece of cloth that covers the eyes, the air muzzle is a ball that resembles a deep diver's helmet, with an opening in the front so the pet can see while keeping the head safely enclosed
- Early use of sedation in those pets who need it.

4 Use effective calming products

Over the years, Bloom's team has accumulated a variety of products for use in the hospital and at home. As an added bonus, when clients see how well pets respond to them, they often purchase the items to use on their pets at home. Two of his favorites are

5 Administer anti-anxiety medications

For pets that don't respond to the above techniques, Bloom doesn't hesitate to use anti-anxiety drugs, which reduce his patients' stress levels without making them less alert.

His preferred medications are:

- Trazadone for dogs.
- Gabapentin for cats—"100 mg two hours before the appointment and that cat will be a different pet when they arrive," Bloom says.
- Diets such as Hills c/d Multicare Feline Stress
- Natural products such as Zylkene® (a casein-derived relaxation agent) and Anxitane® (L-theanine).

For veterinarians who are resistant to the idea of changing the pet experience, Bloom reminds them that pet fear and stress are one of the major factors keeping clients away from the hospital, and it's one of the easiest to fix.

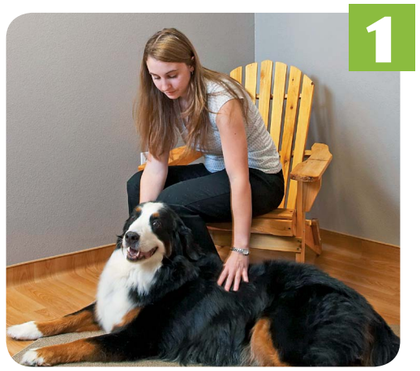
"Fear and anxiety are *nosocomial infections*, and they are zoonotic. We created this problem. They acquired it at our hospital and it was spread by other pets and staff, then the owners catch it," he says.

He likens the change to what happened in pediatric human medicine in the last several decades, when doctors realised the patient's experience was just as important as the medicine provided.

"When puppies come into my practice, they aren't coming in nervous," he says. "We can make it the best experience possible, or we can make it what 40 to 50 percent of pets are experiencing today."

Dial it DOWN

You're ready for easy

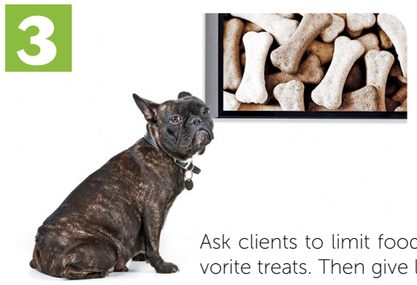


1 Offer dogs physical examinations and treatments on the floor, if they prefer, instead of up on the exam room table or behind the scenes in the treatment area

2

For cats eliminate visualisation of other cats and photorealistic cat images.

3



Ask clients to limit food before an appointment and bring their pet's favorite treats. Then give lots of those treats during the appointment.

5

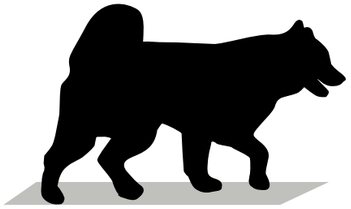
Add shelves or plants as visual barriers in the reception area.

4

Play pet-pleasing music (that may be different for cats or dogs) at low volume in areas with animals. Include a volume control in the room.

You're ready for a little effort

6



Install non-slip flooring for dogs. Too pricey? Start with non-slip mats (yoga mats) on exam room tables.

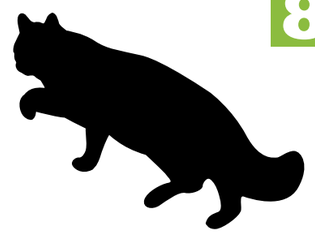
7

Three ways to make exam room tables low-stress:

- Towels with pheromone
- Ramps or stairs up to the table
- Heated surfaces.

8

Make sure cage latches, hinges, cabinets and clipboards all open, close and move quietly.



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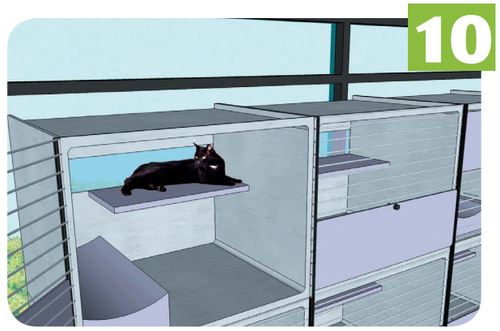
Install enrichments and climbing structures for cats in exam rooms.

10

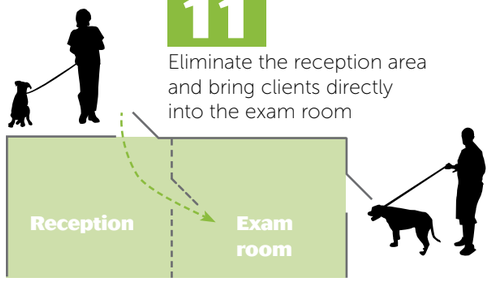


Offer resting platforms for cats in cages and runs.

You're ready for a totally low-stress clinic

11

Eliminate the reception area and bring clients directly into the exam room



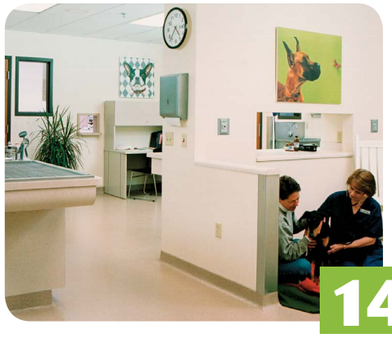
12

Allow clients to enter directly from outside

13

Offer a quiet space for clients to spend time with hospitalised pets, especially in critical care situations.

Provide daylight in animal wards, treatment areas and exam rooms



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Decontaminating the Poisoned Patient

Justine A. Lee, DVM, DACVECC, DABT
Pet Poison Helpline, Minneapolis, Minnesota

Decontamination is meant to inhibit or minimize further toxicant absorption and to promote excretion or elimination of toxicant.[1]

Types of decontamination include ocular, dermal, inhalational, injection, GI, forced diuresis, and surgical removal.[2]

Ocular decontamination

- If ocular exposure to a corrosive agent (eg, drain or oven cleaners) occurs, instruct owners to flush the eye at home with physiologic saline or tap water for 15 –20 minutes before seeking veterinary attention.
  - This is imperative to avoid secondary corneal injury.
  - The necessary pet restraint methods required often prevent owners from adequately performing ocular decontamination.
- If ocular exposure to an irritant occurs, instruct owners to flush the eye at home as described above.
  - Any change in condition (eg, ocular discharge, pruritus, rubbing, pupil size, blepharospasm, inflammation) warrants immediate veterinary attention.[2]
- Instruct owners not to apply salves or ointments and to prevent any eye rubbing until the pet receives veterinary care.

Dermal decontamination

- Remove toxicant from fur or skin to prevent reexposure (secondary to grooming) and transdermal absorption.
- Advise handlers to use protective gear when necessary (eg, around corrosives, organophosphates, pyrethrins) while bathing the patient.[2]
  - Many owners are unable to restrain cats for adequate bathing, and veterinary attention is often required.
- Use degreasing dishwashing liquid to fully remove oil-based substances (eg, pyrethrins, glow sticks, essential oils).
- Do not use neutralising agents (eg, acid for alkaline exposure), as these can cause a chemical reaction that exacerbates dermal injury.[2]
- With acidic or alkaline exposures, decontaminate gently; use copious tepid water for 15–20 minutes.
  - Do not use high-pressure sprays and scrubbing.
- Thermoregulate the patient following decontamination - monitor the tempurature.

Inhalational decontamination

- Remove patient from the source of exposure.
- Evaluate patient for severity of hypoxemia (based on pulse oximetry, arterial blood gas analysis)[2]
  - Often, removal from the source and adequate ventilation are sufficient.
  - If necessary, provide oxygen therapy.
- Ensure that the area of exposure is appropriately ventilated to prevent re-exposure.
- Be aware of public health risks associated with certain toxicants.
  - During zinc phosphide rodenticide toxicosis decontamination, pet owners, support staff, and veterinarians can be exposed to phosphine gas in the dog's vomitus. Emesis induction must occur in a well ventilated area.
- Educate clients on the effects of inhalational toxins (eg, fragrances, Teflon) on birds.
  (See Vet360 Feb 2015)

GI decontamination

- For GI decontamination, which is the most common, consider emesis induction (Table 1), gastric lavage, whole bowel irrigation, and the use of activated charcoal and cathartics.

- Take the history of exposure (eg, timing, toxin) and evaluate the patient (eg, for status, medical history) to determine whether emesis is appropriate (Table 2).
- Consider appropriate use of and contraindications for emesis induction.
- Salts, liquid dish soaps, mustards, and syrup of ipecac are not generally recommended.
- For dogs, the use of hydrogen peroxide and apomorphine is appropriate.
    - Hydrogen peroxide, which acts as an emetic by direct gastric irritation, is the only recommended at-home emetic agent for owners.
    - The use of α-adrenergic agonists as an emetic agent in dogs is not routinely recommended, as they are typically not as effective as hydrogen peroxide or apomorphine.
- For cats, there are no current recommended at-home emetic agents.
    - The use of hydrogen peroxide is not recommended because of risk for hemoptysis.
- In cats, an α-adrenergic agonist (eg, xylazine, dexmedetomidine) should be used.
    - Yohimbine or atipamezole can be used to reverse severity of sedation.
- If emesis induction is unproductive or contraindicated (eg, from severe clinical signs such as obtundation, seizures, coma), consider gastric lavage to remove gastric contents
- Although labor intensive, gastric lavage is warranted in severely affected patients, for life-threatening ingestion, and with certain toxicants that have a narrow margin of safety (eg, calcium channel blockers, baclofen, metaldehyde, organophosphates).
    - It is vital to ensure the correct placement of a well fitting, cuffed ET tube to avoid aspiration. *(Editor)*
    - neuromuscular weakness can sometimes complicate entubation. *(Editor)*
- Consider use of activated charcoal and cathartics that prevent further systemic absorption of the toxicant.[2]
- While rarely used in human medicine, AC is considered a primary treatment for the poisoned veterinary patient.
- Administration of AC is contraindicated with toxicants unable to bind to it, such as alcohols (eg, ethylene glycol, xylitol, methanol) and heavy metals.

Table 1: Emesis Induction: Indications and Contraindications

Emesis <u>Only</u> Should Be Performed	Emesis Should Not Be Performed
With recent ingestion (<1 hour) in asymptomatic patients	With corrosive toxicant ingestion (eg, lye, ultra-bleach, batteries, oven-cleaning chemicals)
With unknown time of ingestion in asymptomatic patients	With hydrocarbon toxicant ingestion (eg, tiki-torch oil, gasoline, kerosene)
When a product known to stay in the stomach for a long time (eg, grapes, raisins, chocolate, xylitol gum) is ingested by asymptomatic patients	In symptomatic patients (eg, tremoring, agitated, seizuring, hyperthermic, hypoglycemic, weak, collapsed)
	In patients with underlying disease predisposing them to aspiration pneumonia (eg, megaesophagus, history of aspiration pneumonia, laryngeal paralysis)

AC = activated charcoal

- AC is also contraindicated for such toxicants as hydrocarbons or corrosives.
- Multiple doses of AC (with additional doses devoid of a cathartic) are warranted with toxicants that undergo enterohepatic recirculation when the product is sustained-release (eg, certain prescription medications) or when the toxicant has a long half life (eg, naproxen, 72 hours).
    - Patients receiving multiple doses of AC require periodic electrolyte monitoring because of the rare risk for hypernatremia.
- Patients at risk for hypernatremia include dehydrated patients or those with free water loss (eg, diabetes mellitus, renal disease, excessive panting, vomition, polyuria).
    - Administer antiemetic (eg, maropitant) or fluid therapy (SC or IV) to patients receiving AC to prevent fluid losses contributing to hypernatremia.
    - Obtain baseline sodium levels.

Decontamination should be considered a primary treatment. Knowledge of the underlying mechanism of action, pharmacokinetics (eg, absorption, distribution, metabolism, excretion) and toxic dose of the toxicant is imperative in determining appropriate decontamination and therapy for the patient.

Table 2: Appropriate Emetic Agents & Toxicology - Drugs Commonly Used

Emetic Agent	Where It Works	Dose	Use in Indicated Species Only
Hydrogen peroxide 3%	Local gastric irritant	1 –5 mL/kg PO, up to 2 times, not to exceed 50 mL in dogs	Dogs
Apomorphine	Chemoreceptor trigger zone	0.02 mg/kg IV; 0.04 mg/kg IM; crushed tablet subconjunctivally. Can be repeated at titrated dose. If excessively sedated, can consider reversal with naloxone.	Dogs
Xylazine	Centrally acting	0.44 mg/kg IM	Cats
Activated charcoal	Binds directly to the toxicant in the GI tract	1–5 g/kg PO. The first dose should ideally contain a cathartic (eg, sorbitol); however, with multiple doses, the remaining doses should not contain a cathartic	Dogs Cats
Yohimbine	Reversal of α-adrenergic agonists	0.1 mg/kg IV slow to effect	Cats
Atipamezole	Reversal of α-adrenergic agonists	0.05–0.2 mg/kg IM or IV	Cats

REFERENCES - available on www.vet360.vetlink.co.za

Comments

GI Decontamination in Organophosphate and Carbamate Toxicity

By: Dr Liesel van der Merwe BVSc Hons MMedVet (Med)

The following aspects on the management of organphosphate toxicity have been acquired as a result of much exposure in clinical practice ...

Activated charcoal is more effective than the tablets as it is the surface area which determines the binding capacity.

Organophosphates and carbamates are lipid soluble and are excreted through the bile. They undergo an enterohepatic circulation and will be re-absorbed in the ileum if no activated charcoal is present to bind the toxin.

All poisoned animal must be given AC to avoid reabsorption. Unless the animals are eating by themselves, I prefer to place a naso-oesphageal tube and administer the mixture through the tube. AC has no taste. Even cats will eat it if mixed into the food. Just don't add too much to make the texture powdery.

Organophosphates affect the nicotinic receptors of the neuromuscular junctions and even if the dog is not paralysed or does not have obvious dysphagia or megaoesophagus - the function of the pharyngeal muscles (striated) and oesophagus (striated muscle in the dog) is often suboptimal and they tire easily . This may result in aspiration if syringe feeding the mixture.

Placement of naso-oesophageal tubes can be complicated in a poisoned dog as they cannot swallow properly and the feeding tube is inclined to pass into the trachea.

- I will give the patients a light GA, pass the tube through the nose into the pharynx and then, opening the mouth and using a laryngoscope, direct the tube into the oesophagus with an instrument of sorts.
- To avoid aspiration and volume overload - I make quite a concentrated slurry and rather dose smaller volumes more frequently. If the patient is recumbent make sure that the entire thorax and head are on a slop of about 30°) to prevent reflux and aspiration

AC does not bind the toxin permanently - so the charcoal and toxin must be passed from the body via the colon. Constipation can and will result in the re-appearance of clinical signs as "released" toxin is once again absorbed through the colon.

Atropine causes ileus - thus constipation is a reality in poisoned animals .

- Addition of lactulose to the mix is essential
- Stool production must be monitored and enemas may be required

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Emesis & Activated Charcoal

Valentina Merola, MS, DVM, DABVT, DABT
ASPCA Animal Poison Control Center

Decontamination is frequently a first consideration in toxicosis cases, but induction of emesis or administration of activated charcoal may not always be indicated—in some cases, it can be harmful.

Profile

- Major human clinical toxicology associations have noted that neither the use of ipecac to induce emesis nor administration of a single dose of activated charcoal is routinely recommended.[1,2]
  - There is no evidence that emesis or activated charcoal improves clinical outcomes was found.
- In veterinary medicine, however, emesis and activated charcoal are still viable options for decontamination.
  - Dogs commonly consume large enough amounts of toxicants to make gastric decontamination a valuable option.
  - In dogs and cats with successful emesis induction, at least some of the ingested toxicant was recovered in 68% of cases.[3]
  - Induction of emesis or administration of activated charcoal should be evaluated on a case-by-case basis.

Treatment

Considerations

- Stabilization is the first priority.

! If the patient is dehydrated, activated charcoal administration can increase risk for electrolyte disturbances, and emesis can worsen dehydration.

  - If the patient is dehydrated, activated charcoal administration can increase risk for electrolyte disturbances and emesis can worsen dehydration.
- If the animal has already vomited multiple times, encouraging additional emesis may not be beneficial.
  - Administration of activated charcoal should be postponed until vomiting has been controlled.
- GI ulceration or perforation should be ruled out before administration of activated charcoal, as the charcoal could impair healing of ulcers or, in cases of perforation, contaminate the abdominal cavity.
- If the patient is at risk for developing or suspected to have ileus or GI obstruction, activated charcoal should be avoided until these conditions are ruled out.
- Activated charcoal is of limited benefit in cases of poisoning with xylitol, alcohols, heavy metals, strong acids/bases, and many other small molecules because of poor binding ability.
- Some toxicants (eg, marijuana, antihistamines) have antiemetic properties; immediate administration of activated charcoal may be more beneficial.[5]
- Because some toxicants (eg, grapes, chocolate) remain in the stomach for long periods, emesis may still be beneficial several hours after exposure.
- Some medications come in extended release formulations for longer or slower absorption times.
  - Decontamination may be effective later than typically expected.
- If the toxicant undergoes enterohepatic recirculation (eg, bromethalin, naproxen), activated charcoal may be beneficial for up to 72 hours following exposure.

Outpatients

- At-home emesis is only indicated if the patient is stable.

Dogs

- Owners should offer dogs bread first; emesis may be more productive with a full stomach.[5]
  - In toxicity cases involving zinc phosphide or aluminum phosphide, no food should be given.
  - Food stimulates production of stomach acid, which causes release of toxic phosphine gas.[5]
- Walking the pet or allowing it to move around may help stimulate vomiting.[4]
- Hydrogen peroxide 3% at 2 ml/kg PO should be administered up to 45 ml.
  - Further administration is likely of little benefit and can cause injury to the esophageal and gastric mucosa.

Cats

- Emesis is only recommended at home in extenuating circumstances.
- Hydrogen peroxide is effective in 30% of cases,[3] and adverse effects (eg, severe, bloody vomiting) can occur.

Inpatients

Dogs

- Apomorphine at 0.03 mg/kg IV or 0.04 mg/kg IM or a portion of a tablet crushed and dissolved in water for administration into the conjunctival sac
  - This acts by stimulating dopaminergic receptors in the chemoreceptor trigger zone (CTZ).[5]
  - Sedation can be reversed with administration of naloxone, but it will not stop the vomiting.[4]
  - The conjunctival sac needs to be rinsed after emesis.
- Hydrogen peroxide may be considered if toxicant has antiemetic effects at the CTZ (eg, phenothiazines).
- Both hydrogen peroxide and apomorphine are >90% effective in dogs.[3]

Cats

- Xylazine (0.44 mg/kg IM) is effective in ~57% of cases.[3]
  - Sedation can be reversed with yohimbine.
- Apomorphine is rarely effective in cats.[3]

Follow-up

Complications

- Marked or bloody vomiting can result from administration of hydrogen peroxide.
- CNS depression is possible with administration of apomorphine or xylazine.
  - Depression may be reversed with naloxone (with apomorphine) and atipamezole or yohimbine (with xylazine).
- Electrolyte disturbances (ie, hypernatremia, hypermagnesemia, hypokalemia) can occur following administration of activated charcoal.
  - Monitoring electrolytes is always recommended with multiple-dose activated charcoal and suggested with a single dose.
- Activated charcoal can increase serum osmolality, osmolal gap, and serum lactate concentration.[6]
- Aspiration of charcoal or stomach contents can occur.
- Vagal events or bradycardia may occur.

Contraindications for GI Decontamination

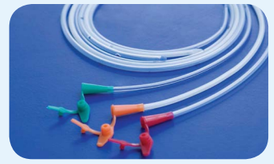
In some situations, emesis should not be induced and activated charcoal should not be given, including if:

1. Patient has moderate to severe CNS depression or stimulation or is having seizures (because of risk for aspiration); unless the CNS signs are readily reversible (eg, with administration of atipamezole for amitraz toxicosis), emesis should not be induced and activated charcoal should only be given with a cuffed endotracheal tube in place.
2. Patient has underlying health problems that may compromise ability to protect the airway.[4]
3. The toxicant is caustic or corrosive. Reexposing the esophagus by inducing emesis can cause further damage.[5] Activated charcoal may not bind well to caustics or corrosives and can impair healing.
4. The agent is a volatile hydrocarbon or petroleum distillate (causing risk for aspiration).
5. The agent is likely to cause rapid onset of serious signs (eg, seizures, coma) before vomiting occurs.
6. If it is suspected that the patient may require enterotomy or gastrotomy, activated charcoal should not be given.

Contraindicated Emetics

- Table salt: Not a reliable emetic and can result in life-threatening hypernatremia[5]
- Syrup of ipecac: Less readily available and less effective than hydrogen peroxide; can be cardiotoxic with high dose[5]
- Mechanical methods (eg, fingers down the throat): Considered to be generally ineffective[4]

Administering Activated Charcoal



- Only medicinal-grade activated charcoal (1–2 g/kg PO) should be used.
- Medicinal-grade charcoal has a large surface area for binding to toxicants.[5]
- Multiple-dose activated charcoal can be beneficial in some circumstances

REFERENCES - available on www.vet360.vetlink.co.za

Shaping the future of animal health

Canine Atopic Dermatitis

A Practical Approach A to Z

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Definition

Canine Atopic Dermatitis (CAD) is defined as an inflammatory and allergic skin disorder, affecting genetically predisposed dogs, with characteristic clinical features. It is generally associated with IgE antibodies most commonly directed against environmental allergies (Type 1 hypersensitivity). The patient becomes sensitised to environmental antigens that cause no reaction in non-atopic dogs. In addition, is now also recognised that CAD is a complex and multifactorial disease involving immune dysregulation, allergic sensitisation, skin barrier defects, microbial colonisation and environmental factors. CAD affects 3 to 15% of the canine population. In some studies up to 50% of dermatology cases are CAD cases.

Pathophysiology

CAD is mainly caused by aeroallergens that gain access to the body via the percutaneous route. Outbreaks of atopy have also been linked to allergens presented via other routes e.g. the digestive tract. Numerous allergens have been identified in the pathogenesis of CAD. These include house dust and storage mite allergens, pollens from grasses, trees and weeds, mould spores, epidermal allergens, insect allergens and miscellaneous allergens such as kapok. The majority of cases result from hypersensitivity to house dust and storage mite allergens, leading to a non-seasonal dermatitis. Pollens usually lead to a seasonal dermatitis.

Atopic dogs are predisposed to penetration of the allergens because they have an inherited dysfunction of the immune system as well as a defective cutaneous epidermal barrier function.

From a practical perspective this means that all of the potential components that contribute to the pathogenesis (immune dysfunction, infection, and epidermal barrier defects) need to be identified and considered in the diagnosis and eventual treatment plan for successful control.

The following steps are important in the pathogenesis:

1. Allergens access the body via the percutaneous route. Alteration of the epidermal barrier function facilitates penetration via this route.
2. Once the allergen enters the epidermis it binds to the epidermal Langerhans cells where it undergoes internal processing and is presented to naïve T cells in the draining lymph nodes.
3. In allergic individuals this leads to Th2 differentiation/polarisation and these Th2 lymphocytes release cytokines including IL-4, IL-5, IL-13 that stimulate IgE production by B cells that bind to cutaneous mast cells.
4. Re-exposure to allergens causes mast cell degranulation, cytokine release along with homing of T cells to the epidermis. This leads to cutaneous inflammation, erythema and pruritus.
5. A variety of inflammatory mediators are involved including histamine, leukotrienes and proteases from mast cells and interleukins from keratinocytes.
6. Atopic dogs have impaired cell-mediated immunity predisposing them to secondary bacterial and yeast infections.
7. It has become clear that in addition to the complex immune response, both epidermal barrier dysfunction and infection play an important and interrelated role in the pathogenesis and severity of disease.

Epidemiology

The peak age of onset is between one and three years of age, with a range of 6 months to 6 years. It is relatively uncommon but not impossible to see the disease first appear in middle aged or older dogs.

It can affect any breed, but there is a greater incidence in pure bred dogs such as Terriers, Golden and Labrador Retrievers, German Shepherd dogs, Dalmations, English Bull dogs and Shar Pei dogs. There are significant variations between breeds affected between different countries. This confirms that although there a genetic component, environmental factors play a more important role.

Epidermal barrier function:

There is increasing evidence that animals with canine atopic dermatitis (CAD have abnormalities in their skin barrier function and that these changes contribute significantly to the disease severity. It is not yet clear if this is innate (genetic) or acquired. The stratum corneum plays a vital role in the barrier function of the skin in mammals. Some of the abnormalities that have been identified include: abnormal intracellular lipid lamellae, abnormal stratum corneum morphology and abnormal and reduced ceramide content.

Atopic dogs are predisposed to penetration of the allergens because they have an inherited dysfunction of the immune system as well as a defective cutaneous epidermal barrier function. Both lipid and protein skeleton (fillagrin) abnormalities have been described which lead to a weakened stratum corneum - which allows both allergens and pathogens (bacteria) access to the deeper layers

The epidermis is composed of 4 or 5 layers of cell types depending on the region. The superficial stratum corneum layer of the skin is made up of 10 – 30 layers of cells (keratinocytes and corneocytes). The cells are flattened and fit together like bricks. They are each encapsulated by water-retaining keratin and ceramide proteins as well as cholesterol and free fatty acids. The cells are held tightly together by a skeleton of protein junctions. In-between the cell layers are multiple stacked lipid layers (lamellae).

The epidermis is an active site of lipid synthesis. Lipids are important in barrier function, stratum corneum water holding and cohesion, desquamation of corneocytes and control of epidermal proliferation and differentiation. Ceramides are the most important lipid component for the lamellar arrangement of the stratum corneum and have an important barrier function as they allow stretching and bending. The multiple layers of lipids prevent trans-epidermal water loss.

The ability of the skin to hold water is due to the stratum corneum and is critical for maintaining a healthy skin. Shifts in humidity may alter hydration status of the stratum corneum. The defective epidermal barrier in addition results in increased trans-epidermal water loss (TEWL) resulting in dry skin with loss of skin flexibility, increased penetration of irritants and allergens, stimulation of the skin immune system and a lower ability to prevent microbial colonisation, which predisposes them to bacterial and yeast proliferation and infections.

The tight cell structure and skeleton makes a physical barrier, the lipids and other proteins make a chemical barrier and immune surveillance cells an immune barrier.

Clinical signs

The main clinical feature of CAD is **pruritus**. It ranges from mild to severe, but most cases are moderately to severely pruritic.

Lesion distribution initially is very typical. (Fig.1) The face (rubbing), ears, paws and distal extremities (chewing) and ventral aspects (scratching) are predominantly affected. As the disease progresses and becomes chronic, the entire body may become affected in about 40% of cases.

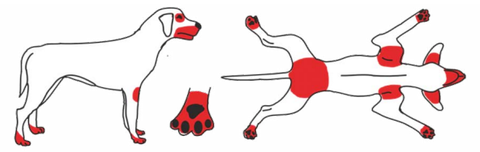

Figure 1: Lesion distribution in CAD

Primary lesions seen are erythematous macules and papules. These are very soon changed into secondary lesions due to self-trauma, secondary bacterial and Malassezia dermatitis and chronic inflammation. (Figs. 2-4) Chronic cases are often characterised by seborrheic skin changes, salivary staining, marked scaling and varying alopecia. Marked seborrhoea is seen in 12% of atopic dogs. 60% of affected cases suffer from secondary pyoderma, usually a folliculitis, and/or Malassezia infections.

Otitis externa accompanies 80% of cases. In some cases this may be the only presenting sign. Initially the inside of the pinna and the vertical canal are affected, but in chronic cases the horizontal ear canal may also

Figure 2 - Peri-ocular dermatitis

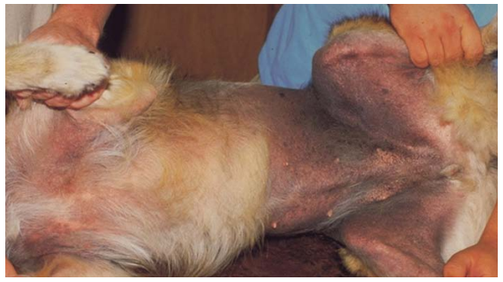

Figure 3 - Axillae and inguinal regions affected

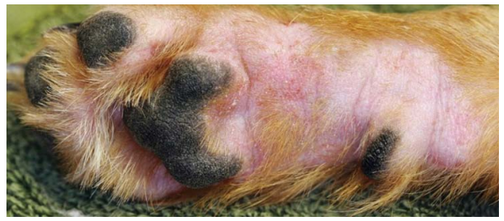

Figure 4 - Caudal aspect of front paw showing affected skin

become affected. Seborrheic skin disease and secondary bacterial and Malassezia infections may all contribute to the objectionable odour of atopic dogs.

Non cutaneous clinical signs reported to occur occasionally in atopic dogs include rhinitis, asthma, urinary and gastrointestinal disorders and cataracts.

Diagnostic approach

Any itchy dog may potentially have CAD, but there are also many other causes of pruritus. A diagnosis of CAD can only be made once all other causes of pruritus have been eliminated. This is done by following a step by step approach to a pruritic dog. It is very important to make a diagnosis as some of the causes of pruritus, e.g. flea bite dermatitis/allergy, mites and cutaneous adverse food reactions (CAFR) are treatable.

It is very important to explain to the owner of the pruritic dog that you are going to work according to a plan to find out why their pet is pruritic. It is very easy to just give a "magic" corticosteroid injection to take the itch away, but obviously the pruritus will return again and again until a diagnosis is made and treatment options can be given.

They should understand that it is in the best interest of their pet that a diagnosis is made from the onset and treated or managed according to the cause. This process usually takes time and the owners and clinician should not expect a cure in one visit for a problem that has been going on for months/years. The relationship should be cooperative and in the long run they will save money and their pet will benefit.

It is important to take time with the initial dermatological examination, to sit down with the owner and take a detailed history. Thereafter it helps to hospitalise the patient for a few hours and do the clinical examination and in house diagnostic tests when there is more time. The owners return later and the diagnostic and therapeutic plan is discussed in detail. Follow up examinations can be done in normal 15 minute consultation slots.

1. Historical features
   Age of onset, breed, chief complaint, where did the pruritus start at first, describe the lesions seen, diet, previous response to treatment (drugs used, dosages, duration of treatments given), lesions present in in-contact animals or owners. It helps to have a form with all the relevant questions which the owners can fill in prior to the first consultation. This helps to get all the necessary information.

2. Clinical examination
   Lesion distribution, types of lesions present, presence of secondary bacterial and yeast infections (pustules, crusts, epidermal collarettes, greasy seborrhoea, offensive odour). Never underestimate the role that fleas can play in any pruritic patient. The presence of fleas or flea dirt is very significant and should be shown to the owners.

3. Skin scrapings
   Skin scrapings should always be done to rule out Sarcoptes and other mites. Sarcoptes mites are not easy to find on scrapings. Crusted papules on elbows, hocks and ear pinnae give the best results. A single mite or egg is diagnostic. Although Demodex is not usually pruritic, it should be ruled out because it may cause a secondary pyoderma that may often be pruritic. Many pruritic dogs have been on chronic corticosteroid treatment which leads to immunosuppression and demodicosis. Cheyletiella is often found on cello tape impression smears of dandruff present on the skin. If a mite is suspected, a therapeutic trial should be performed (see later).

4. Skin and ear canal cytology
   Cytology is very important, easy, quick and inexpensive and can be performed in-house. Samples are taken from all affected areas as the infections are often regional. Ear buds are effective for taking samples from the external ear canal and cello tape impression smears are very effective to determine the presence of secondary bacterial and Malassezia infections on the skin. It is a good habit to grade these infections at the various sites from 0 to 4 +. This helps to determine response to treatment. The presence of the numbers of eosinophils, neutrophils, monocytes, etc. should also be recorded and graded.

5. Therapeutic trial
   At this stage all secondary infections present should be treated for a minimum of three weeks. This is necessary in order to determine what contribution the infections are making towards the pruritus. Often the lesions will resolve with this treatment, but not necessarily the pruritus. Cephalexin at 20 mg/kg BID or amoxicillin-clavulanic acid at 20 mg/kg BID is effective in most cases. Severe cases of Malassezia are treated with systemic ketoconazole at 5-10 mg/kg once daily. In less severe cases topical ketaconazole or chlorhexidine shampoos are often effective. If fleas are present or Sarcoptes or other mites are diagnosed or suspected, these should be treated at this stage.

   Remember the principle of "treat what you see and see what is left". The owners are asked to score the pruritus before initiating any treatment out of ten. Most owners will score 9/10 or 10/10. If at all possible no drugs to suppress the pruritus symptomatically should be given. This will obviously give a false impression that the other treatments are effective. If the pruritus is severe, a short course of prednisolone, 0.5 - 1 mg/kg OID, for 3 to 5 days may be given. This will bring relief to the dog and also help to win the confidence of the owner.

The most important differential diagnoses for CAD are:

a. Flea allergy dermatitis (FAD) – may co-exist in the same patient, typical distribution involves lower back and posterior and inner thighs.
b. Cutaneous Adverse food reactions (CAFR) – difficult to distinguish, similar clinical features. May also co-exist in the same patient. Differences – CAFR can occur at any age, a primary papular eruption is often present, often corticosteroid resistant pruritus.
c. Parasitic dermatoses – rule out Sarcoptes (affects elbows, hocks, ears), Cheyletiella (dandruff), Otodectes (usually in ear canals too), Fleas (flea dirt, fleas present, affects lower back region).

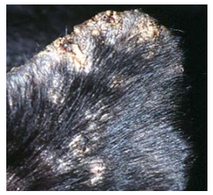

Figure 5: Typical crusting lesions on the edge of the pinna due to Sarcoptic mange in a Dog

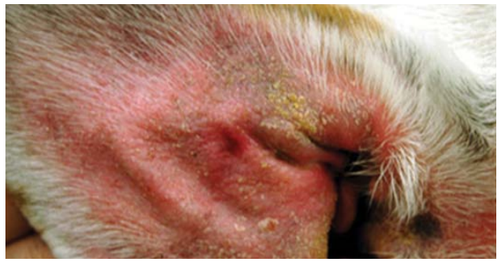

Figure 6: Otitis externa due to CAD affecting the inside of the pinna in a Dog

Major and minor criteria for Canine Atopic Dermatitis

There are important major and minor criteria that can be documented from the history and clinical examination which can aid in the diagnosis of CAD. The more major criteria are met, the more likely the diagnosis of CAD. Three or four key criteria are considered enough evidence. The presence of minor criteria supports the diagnosis. The criteria were put forward by Willemse (1986). *Willemse A 1996. Atopic skin disease: a review and a re-consideration of diagnostic criteria. Journal of Small Animal Practice 27: 771-778*

Major Criteria	Minor Criteria
• Onset of clinical signs between 1 and 3 years old	• Breed predisposition and/or family history
• Pruritus of gradual onset. Face (rubbing) and feet (chewing) typically affected.	• Chronic or chronically relapsing dermatitis for more than 2 years
• Corticosteroid responsive pruritus (initially)	• Secondary cutaneous infections, especially bacterial
• Bilateral front foot pododermatitis	• Bilateral conjunctivitis and/or epiphora
• Otitis externa	• Lesions on the flexor surface of the tarsus or extensor surface of the carpus
• Inflammation of inner surface of the pinnae	• Seasonal aggravation of symptoms
• Facial erythema (around eyes, lips, chin)	• Variation in severity with environmental changes
	• Aggravation when in contact with grass

d. Allergic contact dermatitis (ACD) – very rare in dogs, lesions are restricted to contact areas where hair is absent or thin.

After 3 weeks the patient should be re-evaluated. The owner should score the dog again. The dog should be checked thoroughly to see how the pruritus and the lesions have responded to the treatment. Follow up cytology has to be performed to grade infections that may have remained. It is important to evaluate the pruritus score as well as the appearance of the lesions – lesion resolution does not always accompany the resolution of pruritus. Failure to respond to a proper therapeutic trial is not a failure. It simply means that a possible cause has been eliminated. There is no quick fix and most, if not all chronic pruritus cases cannot be controlled in one visit. If the client does not return for the revisit, the initial visit will not have any long term benefits.

There are now three possible scenarios:

a. The patient is much better, pruritus score is now 0/10, both pruritus and lesions have resolved: it could have been FAD with secondary infections, Sarcoptes with secondary infections or non pruritic primary disease that now presents with typical lesions, e.g. auto-immune or endocrine dermatoses. Further work up may be necessary.
b. The patient is better, lesions have improved but the pruritus is between 2/10 and 5/10: the treatment was able to bring allergic threshold (see later) down, the patient more than likely has CAD and it may be possible to control the CAD with antimicrobial pulse therapy and strict control of parasites. The pruritus may become worse again if the allergic threshold changes.
c. The patient is not much better, lesions may or may not have improved or have improved only slightly, the pruritus score is more than 5/10 and the secondary infections and parasites are under control: the 2 main remaining differential diagnoses at this stage are CAD and CAFR. The next step is a diet trial to rule CAFR in/out. If there is no response to the diet trail, a diagnosis of CAD can be made.

6. Diet trial

Since CAFR and CAD have a very similar clinical presentation, it is advisable to put the patient on a hypoallergenic diet trial for 3 to 8 weeks in order to rule CAFR in or out. Many owners will say that they have already done a diet trial. It is important to ask about the trial and if it was done properly. If not, it should be repeated. Serology tests for diagnosing CAFR do not have any diagnostic value. It is important to remember that these 2 conditions may co-exist. If there is a good possibility from the history and initial consultation that a patient may have CAFR, the hypoallergenic diet trial can be started at the same time as the therapeutic trial to save time. It is better, however, to do diagnostic tests one at a time to really get an impression of the response to a specific treatment.

7. Allergy testing

A diagnosis of CAD is made after ruling out other causes of pruritus, NOT by doing any allergy testing whether intradermal or in vitro allergy testing. The diagnosis is made on the basis of history and clinical findings. Allergy testing is done where immunotherapy is a treatment option. The true utility of allergy tests is in the selection of allergens for inclusion into a vaccine for allergen-specific immunotherapy and possible consideration of allergen avoidance measures. There are 2 types of allergy tests available, intradermal tests (not available in South Africa) and allergen-specific IgE serology. Allergen-specific IgE serology identifies specific antibodies for allergens circulating in a patient's blood. It is an easy test as only a serum sample is required.

Prolonged steroid treatment may result in negative tests, but short term corticosteroid therapy does not lower significant levels of IgE. Olivry and co-workers have published guidelines for the optimal withdrawal times of anti-pruritic drugs prior to serologic testing: short-acting oral glucocorticoids, e.g. prednisolone 0 days, long acting injectable glucocorticoids e.g. depomedrol 28 days and oral cyclosporine 0 days. Once the results are obtained, the clinician has to determine which of the positive allergens are of clinical significance. If the allergy test results are negative, the following could be reasons: the patient does not have CAD, drug interference (corticosteroids), wrong time of the year or allergens of significance were not included in the panel. A small percentage of dogs with CAD have persistently negative test results.

Treatment principles

It is very important to remember that CAD cannot be cured. It can only be managed as well as possible, with the aim to give the patient a better quality of life, as in most cases the dog cannot be protected against allergen exposure.

It is important to remember that every allergic dog has an allergic threshold. The allergic load can often be tolerated by an allergic dog without disease manifestations. A small increase in the load may push the patient over the threshold and cause or initiate clinical signs which are called "flare ups".

- The allergic threshold is not fixed and can be raised or lowered by various trigger factors, such as food, infections (staphylococci and/or Malassezia), external parasites and environmental allergens.
- The threshold may therefore be controlled by treatment of the secondary infections, control of ectoparasites and treatment of any associated cutaneous adverse food reactions.
- Each case is different and treatment must be tailored according to each patient and treatment usually involves a combination of treatments.
- Barrier repair is becoming increasingly important in the management of CAD in dogs. This can be facilitated by bathing regularly with appropriate shampoos and dietary and topical fatty acid supplementation.
- Successful long-term management also requires substantial and ongoing owner commitment. The owners need to understand the concept of the allergic threshold so that they can help to determine and avoid triggering factors and adjust long term management accordingly.
- The management and treatment plan may change over time as the disease changes.

1. Avoidance of allergen:

In theory this would be the best possible treatment, but usually this is not possible or practical.
In most cases the aim is to decrease exposure.

- In cases where house dust mites are the cause, keeping pets more outdoors or away from bedrooms and off fabric furniture (where the highest concentrations of mites are) may help. Other suggestions to decrease the numbers of house dust mites include washing bedding in hot water (> 70^{0}C), avoiding stuffed toys, keeping the dog overnight or during the day in a non-carpeted room and running the air conditioner during hot and humid weather. There are products available that are miticidal. A recent study by Christophe Rème and colleagues has shown that good in-house control of house dust mites with acaricidal products can significantly alleviate clinical signs in atopic dogs. Pyriproxifen has been shown to be effective in reducing mite proliferation.
- Suggestions where moulds are the allergens responsible for the allergy are to avoid dusty food, avoid having large numbers of house plants, and to clean the environment and bedding with chlorine bleach solutions.
- For pollens the best ways to avoid the allergens include keeping the dog out of fields, keeping the grass cut short, rinsing the dog off after periods in high grass or weeds, keeping the dog inside at dusk and early morning in heavy pollen season, using air conditioners, and keeping the dog away from the lawn when it is mowed.

2. Control of secondary infections:

Control of secondary infections is vital in the management of a CAD case because infections often contribute significantly to the pruritus induced by allergy. In some cases good infection control may be sufficient to keep the patient below the allergic threshold.

- Pulse or weekend treatment is often instituted after the initial 3 weeks and has been shown to be very effective. Cephalosporins or amoxicillin-clavulanic acid are the most commonly used antimicrobials for this purpose. A study by Didier Carlotti has shown that there is no increase in the development of resistance against cephalosporins when using them for pulse or weekend treatment.
- Chronic Malassezia overgrowth often exists concurrently with the bacterial overgrowth. Pulse therapy with systemic ketoconazole has been shown to be very effective.
- The use of weekly topical antimicrobial shampoo, e.g. chlorhexidine may in some cases be sufficient to control the overgrowth without necessitating systemic medications.

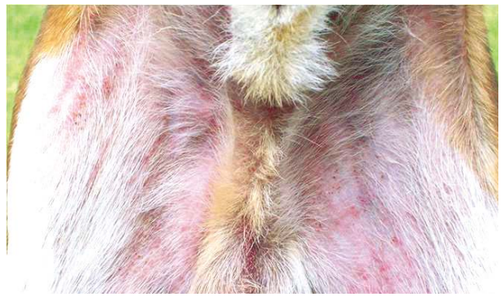

Figure 7: Small superficial pustules indicating secondary infection on medial thighs of a dog

3. Allergen specific immunotherapy (ASIT):

The cornerstone of therapy of CAD is Immunotherapy (hyposensitisation or desensitisation). This is the practice of administering gradually increasing quantities of an allergen extract to an allergic patient to ameliorate the symptoms associated with subsequent exposure to the causative allergen. It "down regulates" the allergic response and may raise the allergic threshold. It alters the balance between TH1 and TH2 cells, which moderates the sensitivity and tolerance to allergens.

ASIT should be considered for young patients; patients where concurrent treatment with topical shampoos, systemic antibacterial and systemic anti-yeast medications is not able to control pruritus sufficiently; cases where corticosteroids have to be used at high dosages for control of pruritus or where side effects are unacceptable.

- There is now a choice between injectable and oral immunotherapy.
- Response to ASIT may take 3 to 10 months.
- The success rate is 65 – 75%.
- It is important to note that only a minority of cases will be totally controlled by ASIT alone. The majority of cases will benefit from ASIT but may require symptomatic treatment, including corticosteroids for some parts of the year.
- It is very important to explain to clients that the target of ASIT is to reduce the amount of immunosuppressive treatment needed. If owners understand what to expect rather than believing that total control is the target, client compliance and satisfaction will be much better.
- Most cases require lifelong control with immunotherapy, supplemented from time to time with other medical therapy.

4. Suppression of inflammation:

Antihistamines:

- H1 antihistamines may sometimes give partial relief to the pruritic patient.
- They are usually used in conjunction with prednisolone to help lower the dose of prednisolone required to control the pruritus and inflammation.
- A selected antihistamine should be administered for 10 to 15 days before evaluating its effectiveness. If the patient does not respond to one type, it may be worthwhile to try another.
- In a recent study by Ewert and co-workers, a combination of hydroxyzine (25 – 100 mg/dog/day) and chlorpheniramine (1 – 4 mg/kg/day) resulted in an improvement of more than 50% in lesion scores in 18% of dogs and of the pruritus score in 30% of the dogs. The investigators judged the treatment satisfactory in 24% of dogs.
- H2 antihistamines have no action on pruritus.
- Antihistamines commonly used, together with dosages, are given in the table below.

Antihistamines	
Chlorpheniramine	0,4 mg/kg q8h
Diphenhydramine	2,2 mg/kg q8h
Hydroxyzine	2,2 mg/kg q8h
Loratidine	0,25-1 mg/kg q24h
Clemastine	0,1-0,25 mg/kg q12h
Cetirizine	2,5-20 mg q24h

Corticosteroids:

- These drugs are highly effective in relieving pruritus, except when fleas or other ectoparasites and/or severe bacterial and/or Malassezia infections are present concurrently.
- Corticosteroids have various anti-inflammatory properties. They are strong inhibitors of the synthesis of pro-inflammatory cytokines with keratinocytes and Langerhans cells their principal targets.
- They are used in many CAD cases and have been one of the most commonly prescribed drugs over the past thirty years for the treatment of CAD.
- Once the secondary infections and other complications have been treated and the patient is still pruritic, it is one of the treatment options for patients where immunotherapy is not a treatment option.

In cases where corticosteroids have to be used, the following rules should be followed:

1. Only short acting, oral products, e.g. prednisolone, prednisone and methyl prednisolone should be used. Prednisolone and prednisone are equally effective in dogs.
2. Ideally an alternate-day regime should be used, often together with antihistamines and oral essential fatty acids, which make it possible to reduce the dose of corticosteroid required.
3. The starting dose is 1 mg/kg once a day and the dose is halved every 4 to 5 days. A maintenance dose of ½ mg/kg every other day or less is considered "safe". Increasing the interval between prednisolone dosing decreases the risk of side effects and pituitary suppression. 1 mg/kg every other day equals 0.33 mg/kg daily.
4. Prednisolone has a half-life of 12 hours, but its biological effect lasts for 4 to 7 days after the last dose. This is the reason why cortisone responsive atopic dogs relapse approximately 5 to 7 days after the last dose of cortisone.
5. The use of injectable forms, especially the "long acting" corticosteroids, is not advised due to the high risk of side effects.
6. A patient on chronic corticosteroid therapy should be examined every three months to monitor weight, inspect the skin for infectious complications and check the urine for urinary tract infections which are very common in these patients.
7. A poor or no response to corticosteroids may indicate that the diagnosis of CAD is not correct, the CAD is complicated by secondary infections or other causes of pruritus e.g. CAFR may be concurrently present.

Oral Essential fatty acids (EFAs):

Omega-6 and omega-3 fatty acids have three main functions in the skin: structural components of cells membranes, maintenance of the epidermal water barrier and precursors for the production of pro- and anti-inflammatory eicosanoids.

- Both omega-6 and omega-3 fatty acids can have immuno-modulatory effects. The main omega-6 fatty acid in cell membranes is arachidonic acid (AA), which is the precursor for the production of prostaglandin E2 (PGE2), leukotriene B4 (LTB4) and 12-hydrodroxyeicosatetraenoic acid (12-HETE), which are all potent inflammatory mediators. When omega-3 fatty acids are given orally or a diet is supplemented with omega-3 fatty acids, part of the AA in cell membranes can be replaced by eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA).

  EPA may then be used instead of AA for the production of eicosanoids, resulting in a different and less inflammatory set of compounds (e.g., PGE3, LTB5 and 15-HETE instead of PGE2, LTB4 and 12-HETE). The substitution of omega-3

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Cyclosporine
in Canine Atopy

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Calneurins are calcium-dependent serine-threonine phosphatases that activate the transcription of cytoplasmic T-cell nuclear factors (NFATc) by dephosphorylating them. The activated NFATc is then translocated into the nucleus, where it upregulates the expression of interleukin 2 (IL-2), pro-inflammatory cytokine which, in turn, stimulates the growth and differentiation of T cell response. IL-2 also promotes the differentiation of T cells into effector T cells and into memory T cells when the initial T cell is also stimulated by an antigen. Calcineurin is the target of a class of drugs called calcineurin inhibitors.

Cyclosporine A is such a calneurin inhibitor. This drug is used as an alternative to corticosteroids to control pruritus. It reduces pruritus and cutaneous lesions. At a dose of 5 mg/kg/day, it was found to be equally as effective as prednisolone. Due to its large size and lipophilic nature availability of cyclosporine is greatly improved (50%) if the micro-emulsion formulation is used.[10] Administration of cyclosporine as micro-emulsion formulation with food decreased the bioavailability by 22% and increased the individual variability of drug absorption – thus dosing two hours before or after a meal is recommended.

Cyclosporine is mainly absorbed from the small intestine and is metabolised in the intestine and liver by the cytochrome P450 enzyme system.[11] The drug accumulates in the skin, liver, kidneys and fat in dogs. In controlled blinded trials 5mg/kg/day – the approved atopy dose - of cyclosporine was shown to cause a similar reduction in canine atopic dermatitis extent and severity index (CADESI) compared to 0.5 or 0.75 mg/kg of prednisolone or methyl prednisolone respectively. [1,9] In another controlled blinded trial cyclosporine out performed placebo in a dose dependent fashion. [8,10] The tolerability and safety of oral cyclosporine and prednisolone also appeared similar. Cyclosporine treated dogs presented with a higher frequency of gastrointestinal disorders, mainly vomiting, but also diarrhoea and anorexia, but prednisolone treated dogs tended to be more susceptible to infections.[1,8,9] A dosage reduction to alternate day or twice-weekly treatment after an initial phase of daily treatment is usually achievable.[11]

No significant correlation was found between clinical improvement and cyclosporine blood concentrations.[11] Therefore a reduction in the dosage is based on the clinical response to therapy rather than the measurement of serum levels of cyclosporine. A short tapering course of prednisolone therapy expedited the efficacy of cyclosporine A in resolving pruritus and associated clinical signs in the initial week or two of treatment. A lag period of about 2-3 weeks in which no response is seen, occurs after cyclosporine treatment is started. Significant reduction in pruritus is expected in 75 – 85% of cases within 1 month of treatment. After six weeks, alternate day therapy and even twice weekly treatment has been effective.

Allergen-specific immunotherapy (ASIT) offers an alternative to either glucocorticoids or cyclosporine therapy. Identification of putative allergens is required for the formulation of ASIT and cyclosporine has been shown to have no statistically significant effects on either intradermal or serum IgE allergy tests when administered at therapeutic dose rates of 5 mg/kg orally once daily for 30 days – so you can test your patient whilst they are on treatment. [3] Its place as an alternative to, or in combination with, prednisolone therapy is considered well established.[3] It is safe in dogs and does not cause nephrotoxicity or arterial hypertension as in humans. Vomiting and diarrhoea are the most commonly seen adverse effects in dogs. This is seen in 14 – 42% of cases, but is mostly mild to moderate. Papillomatous eruptions and gingival hyperplasia are occasionally seen.

Cyclosporine has been used as an aid in the treatment of numerous dermatological conditions in animals in addition to atopy, including perianal fistulation, sebaceous adenitis, pododermatitis, chronic otitis externa, and pemphigus foliaceus[3].

Studies looking at the pharmacodynamics of cyclosporine show that ketoconazole decreased the systemic clearance of cyclosporine.[3] Ketoconazole causes no significant changes in cyclosporine steady state volume of distribution, or plasma unbound fraction. Ketoconazole does not significantly alter the excretion of cyclosporine and various cyclosporine metabolites in the bile/urine mixture. As a result of these findings ketoconazole has been promoted as a cost saver to reduce the dose of cyclosporine. [1,6] Clinicians need to understand that this is extra label use of a registered veterinary product. It also may represent false economy because the use of ketoconazole requires biochemical monitoring for a hepatic insult and the dose of cyclosporine may in any case be reduced once disease remission is established.[6]

REFERENCES - available on www.vet360.vetlink.co.za

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for part of the omega-6 fatty acids may therefore lead to reduced inflammation which is beneficial in inflammatory conditions.

- Many dermatologists consider an omega-6: omega-3 ratio of between 5: 1 and 10: 1 optimal for treating patients with skin disease. This 5-10: 1 ratio may be more applicable to dogs with a normal skin and coat without any underlying pathology. Lower ratios, which are created by adding more omega-3's, may be more beneficial for dogs with underlying pathology.
- In their practical guidelines paper, the Canine Atopic Dermatitis Task Force conclude that oral EFAs have a role to play in the management of chronic CAD, but there is still no consensus regarding particular combination, dosage, ratio and formulation.
- EFAs require up to 2 months of supplementation before any benefit might be seen.
- They are not suitable for monotherapy of CAD, but rather as part of the management programme.
- EFAs are considered corticosteroid reducing agents, because when used in combination with antihistamines, antimicrobial agents, topical medications and medicated shampoos, they have been shown to reduce the dosage corticosteroid required.
- Many CAD patients benefit from specially formulated diets for skin disorders that typically have increased EFA levels. Several publications have reported that veterinary diets with a novel protein and carbohydrate combination with omega-6 and a high level of omega-3 fatty acids (providing a ratio of 2.7:1 omega-6:3) can result in a significant improvement of pruritus and lesion scores in CAD, as well as a significant improvement of skin and coat condition. The incorporation of high levels of omega-3 fatty acids that help to reduce the inflammation, and highly digestible ingredients, such as high quality protein and fats needed for repair of the epidermal barrier, are all possible reasons for why such diets are beneficial in CAD.

Cyclosporine: *(See Information block opposite)*
Oral cyclosporine is a thoroughly evaluated drug for the treatment of CAD. It is a calcineurin inhibitor.

- Cyclosporine acts mainly on T-helper lymphocytes. It also acts on mast cells, eosinophils and Langerhans cells, reducing their antigen presentation functions and inhibits the synthesis of keratinocyte associated cytokines and prevents delayed hypersensitivity reactions.

5. Stress control:

- Stress and anxiety can be triggers that can cause a flare up in an allergic patient's condition.
- Examples include boarding, family going on holiday, loss of a family member, a new baby or pet or moving to a new home.
- Some of the anti-histamines (e.g. hydroxyzine, cetirizine, clemastine) or serotonin reuptake inhibitors (e.g. fluoxetine, clomipramine) may be helpful in treating CAD because of these anti-anxiety effects.

6. Topical treatment:

Topical treatment is a very important component in effective management of CAD and includes shampoos, topical lipids, sprays, creams and ointments.

Shampoos

- Shampoos rehydrate the skin and result in the patient looking, smelling and feeling better.
- They help to remove allergens from the skin surface, help to restore the epidermal barrier and help to control inflammation and secondary skin infections.
- There are a variety of shampoos available that may be helpful in the management of an allergic skin disease:

Cleansing, non-irritating, barrier restoring shampoos

- These shampoos are used to improve or restore the epidermal barrier, e.g. EFA treatment shampoos, hypoallergenic shampoos, shampoos containing ceramides.

Soothing shampoos

- Colloidal oatmeal is a safe and effective soothing antipruritic agent, commonly used in shampoos. The exact mechanism of this effect is poorly understood. This agent is safe, only provides short term relief (24-48 hours) of mild pruritus and has no antimicrobial properties.
- EFA treatment shampoos have soothing effects because they are rich in essential fatty acids and essential oils, with a natural soothing agent from pumpkin seeds. These shampoos also rehydrate the skin and reinforce skin barrier function.

Antimicrobial shampoos

- Antimicrobial shampoos may be used to control secondary bacterial and /or yeast infections and should be used every 7 to 14 days, depending on the current situation. Antimicrobial agents include:
- Chlorhexidine digluconate: An antiseptic effective against most bacteria, fungi and Malassezia pachydermatis. It is bactericidal by action on the cytoplasmic membrane, which causes leaking of intracellular components, is characterised by a rapid kill, has a 36-hour residual activity and is non-toxic and non-irritant. In a study by Jasmin et al a 3% chlorhexidine shampoo was highly effective in the treatment of Malassezia dermatitis and concurrent bacterial pyoderma when present.
- Povidone-iodine: An iodophore which slowly releases iodine to tissue. It is an effective broad-spectrum antimicrobial and is useful for local lesions. It has a prophylactic effect because of its

persistence on the skin, but should not be used repeatedly for generalized skin problems due to its irritant and staining properties.
- Benzoyl peroxide: It is metabolised in the skin to benzoic acid. Much of its microbicidal activity derives from the lowered skin pH which disrupts microbial cell membranes. It is also an oxidizing agent, which releases nascent oxygen into the skin and produces a series of chemical reactions resulting in permeability changes and rupture of bacterial membranes. It has an excellent prophylactic effect and its follicular flushing, keratolytic degreasing and comedolytic activity are additional benefits. It is generally used in concentrations of 2 to 3%, which are well tolerated, but irritation can occur at higher concentrations (erythema, pruritus and pain).
- Quaternary ammonium compounds: They are surface acting agents. They have less effect and are only useful for limited bacterial involvement. They have no residual effect and have to be applied often for good effect.

Keratomodulating shampoos
These shampoos are indicated in cases with allergy induced keratoseborrhoeic changes. Keratomodulating agents include:
- Salicylic acid (0, 5 – 2%) is keratolytic. It causes a reduction in skin pH, which leads to increased hydration of keratin. These actions help to soften the corneal layer. It acts synergistically with sulphur and is often present in small quantities in shampoos.
- Sulphur (0, 5% - 2 %) is mildly keratolytic (forms hydrogen sulphide in the stratum corneum), keratoplastic (has a direct cytostatic effect) and has numerous anti-seborrheic effects.

Topical lipids
- Topical lipids have been used with success in improving the epidermal barrier in human atopic dermatitis.
- There are a few published studies in the veterinary literature that have documented the effects of topical fatty acids on the barrier function in dogs. In one study the topical application of either a fatty acid containing spot-on (applied weekly) or spray (applied daily) improved both the lesions and pruritus in dogs with CAD. Another study showed that the spot-on formulation applied weekly for 8 weeks was beneficial in alleviating the clinical signs of both mild and severe cases of CAD.
- A study by Piekutowska and Pin demonstrated an increase in epidermal lamellar lipids. This suggested that treatment with topical lipids stimulated the production and secretion of endogenous stratum corneum lipids, contributing to the formation of an improved epidermal barrier.
- Topical lipids are more effective in restoring the hydrolipidic film faster when compared to oral EFAs.
- Topical lipids also moderate sebaceous gland activity and sebum production, rebalance dry or oily coat and skin, are skin barrier enhancing and reduce TEWL for optimal skin hydration.

Topical sprays:
- A steroid-free topical spray has been developed for the symptomatic treatment of CAD. It has 100% natural active ingredients which have a synergetic efficacy and does not have any side effect of steroids. The spray is soothing, reduces skin inflammation, regenerates and repairs the epidermal barrier, decreases TEWL and has antimicrobial properties.
- A corticosteroid spray that is not absorbed systemically is also available.

Other topical treatments:
- Topical glucocorticoids are useful in Veterinary Dermatology. They are the sole ingredient or part of combination formulations with antimicrobial and other agents and are useful for localised lesions. Tachyphylaxis, atrophy and microbial infections can occur in cases of overuse.
- Immunomodulators: Tacrolimus, a calcineurin inhibitor has been shown to be effective in the treatment of localized lesions of CAD.
- Antibiotics: formulations containing fusidic acid and mupirocin are useful for treating localized lesions of pyoderma.
- Antifungals: products containing azole derivatives or nystatin can be used on localized lesions of dermatophytosis, Malassezia dermatitis or candidiasis.

Conclusions
- The control of CAD requires combination therapy.
- The concept of allergic threshold and "flare ups" is very important.
- The basis of the therapeutic approach is hyposensitisation, together with concurrent medical therapy including antimicrobials, EFAs and the frequent use of topical shampoos.
- With good management the use of corticosteroids will be considerably reduced.
- Successful management depends on a thorough understanding of the pathogenesis and of the potential complications, and on a willingness to modify the therapy in the light of a changing situation

REFERENCES - available on www.vet360.vetlink.co.za

CPD Questions

AC/1398/15

1. Which one of the following statements (a-e) regarding Canine Atopic Dermatitis (CAD) is most correct?
   a. CAD is mainly caused by aeroallergens which are inhaled.
   b. CAD dermatitis is mainly caused by contact allergens
   c. CAD is mainly caused by percutaneous exposure to aeroallergens.
   d. CAD is mainly caused by a food allergens
   e. CAD is mainly caused by superficial bacterial and fungal infestation.

2. Which one of the following statements (a-e) regarding CAD is incorrect?
   a. Epidermal langerhans cells bind allergens and process and present them to naïve T cells.
   b. Macrophages in the sub-dermis bind allergens and present to the Langerhans cells..
   c. Atopic dogs have impaired cell mediated immunity.
   d. Re-exposure to allergens causes mast cell degranulation and cytokine release.
   e. Allergic dogs have T-lymphocyte response in a TH-2 response.

3. Which of the following statements (a-e) regarding lesion distribution in CAD is most correct?
   a. Lesion distribution is diagnostic in canine allergic dermatitis.
   b. Lesion distribution for atopic dermatitis and flea allergic dermatitis are similar.
   c. Lesion distribution for atopic dermatitis and food allergy dermatitis are similar.
   d. Lesion distribution is of no diagnostic value in canine allergic dermatitis.
   e. Lesion distribution is the same for acute and chronic canine allergic dermatitis.

4. Which one of the following statements (a-e) regarding flea infestation and CAD is most correct?
   a. Flea allergic dermatitis has no specific lesion distribution
   b. In contact animals will also exhibit severe pruritis due to flea infestation.
   c. Fleas must be seen to make a diagnosis of flea allergic dermatitis.
   d. The presence of flea dirt is sufficient to make a diagnosis of flea infestation.
   e. If flea control treatment is being used - flea allergic skin disease is not a differential.

5. Which one of the statements (a-e) regarding the clinical signs of CAD is incorrect?
   a. Otitis externa accompanies 80% of atopic dermatitis cases.
   b. Otitis externa due to atopic dermatitis is always bilateral.
   c. Primary lesions of canine atopic dermatitis are erythematous macules and papules.
   d. Face and ear rubbing and chewing of paws and distal extremities is typical of CAD.
   e. Chronic cases have seborrheic skin changes

6. Which one of the clinical signs listed (a-e) is not a major criteria of atopic dermatitis?
   a. Bilateral front feet pododermatits.
   b. Facial erythema.
   c. Inflammation of inner surfaces of the pinnae.
   d. Elbow and hock show crusting and pruritis.
   e. Otitis externa.

7. Which one of the statements (a-e) listed below is most correct regarding serum allergy testing in CAD?
   a. Allergy testing is important in making a diagnosis of CAD.
   b. Allergy testing is done when avoidance of the cause or immunotherapy is an option, once a diagnosis of CAD is made.
   c. Prolonged prednisilone therapy may result in negative tests.
   d. Cyclosporine must be removed 7 days prior to sampling for allergy tests.
   e. Allergy testing will probably result in cure of the patient

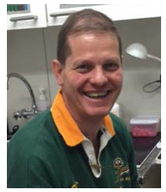
8. Which one of the following statements (a-e) regarding secondary bacterial infection in CAD is incorrect
   a. Control of secondary infection is an important therapeutic trial when diagnosing CAD.
   b. Secondary infections should be treated with antibiotics for a minimum of 3 weeks.
   c. Pulse or weekend treatment can be instituted after the initial 3 weeks has been shown to be effective.
   d. Chronic malassezia infection often exists concurrently with bacterial overgrowth.
   e. Topical therapy will never be sufficient to control secondary bacterial infections.

9. Which of the following statements (a-e) regarding the use of immunosuppressive drugs in CAD is incorrect?
   a. Cyclosporine is used as an alternative to corticosteroids as it has a greater efficacy.
   b. Cyclosporine is used as an alternative to corticosteroid therapy as it has less side effects.
   c. Prednisilone has a biological effect of 4-7 days , thus treatment can be initiated at 5 day intervals.
   d. Side effects of corticosteroids are urinary tract and skin infections.
   e. Cyclosporine and prednisolone can both be tapered after induction doses.

10. Which one of the following statements (a-e) regarding food trails in allergic skin disease in dogs is incorrect?
    a. A hypoallergenic food trial should last 8 weeks
    b. Serology tests for diagnosis adverse food reactions (food allergy) are not of diagnostic value
    c. CAD and food allergy may coexist
    d. The food trial must be of a home-made diet
    e. GIT signs resolve more rapidly than skin signs when a food trial is used.

ANSWER the questions on the Vet360 App. Available from the Itunes/Play store! Alternative system is available through - visit www.vet360.vetlink.co.za and follow the link to CPD Solutions

Cobalamin ($Vit B_{12}$) Deficiency in Longstanding Intestinal Disease



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INTRODUCTION

Cobalamin (vitamin B12) is an essential water-soluble vitamin and is a co-factor for many biochemical reactions. Canine and feline diets contain enough cobalamin, so dietary insufficiency does not occur.

Absorption of cobalamin is complex and requires a healthy digestive system. Cobalamin is bound to dietary protein. After partial digestion in the stomach it is released and immediately binds to R-binder protein synthesised by the gastric mucousa. In the small intestine the R-binder protein is digested by pancreatic enzymes and free cobalamin now binds to intrinsic factor (IF), which is mainly secreted by the pancreas absorption occurs in the ileum.

Diseases that interfere with cobalamin uptake (secondary cobalamin malabsorption) include excessive bacterial utilization of cobalamin associated with bacterial dysbiosis (bacterial overgrowth), exocrine pancreatic insufficiency (EPI), longstanding and severe intestinal disease (IBD, lymphoma) affecting the small intestine in dogs but is seen even with mild GI signs in cats.

- Chronic severe disease of the ileum may lead to destruction or reduced expression of the IF receptors on enterocytes causing malabsorption. Deficiency occurs when stores are depleted – thus indicating a chronic condition.
- Exocrine pancreatic insufficiency results in a lack of pancreatic proteases to separate cobalamin from R-protein. EPI is a major cause of hypocobalaminaemia and B12 levels MUST be checked in these cases.
- IF bound cobalamin is also bound by anaerobic bacteria and thus made unavailable for absorption as they compete. Thus bacterial overgrowth will cause a decrease. In these cases folate will often be increased as some bacteria synthesize folate.

The estimated biological half-life of cobalamin in dogs is 50–100 days. Low serum cobalamin, reflects failing GI cobalamin absorption and a chronic condition. Veterinary laboratories are able to measure cobalamin and the test is not expensive.

Primary (inherited) GI cobalamin malabsorption occurs when gene mutation causes cobalamin malabsorption. These disorders are uncommon but breed-specific and present as poor doers with anaemia and low white cell counts. Giant schnauzers, Beagles, Border Collies, Australian Shepherds, and Chinese Shar-Peis may have a genetic defect.

Clinical Signs

Deficiencies cause abnormalities in the metabolism of the haematological, neurological or gastrointestinal systems, as well as poor response to treatment for gastrointestinal disease. Clinical signs are generally non-specific and vary depending on the age of the patient, the severity of deficiency and the duration of the condition.

Clinical signs are also often mixed with the other signs caused by the primary condition. In young and growing animals there is a chronic, relapsing inappetence, lethargy, and failure to thrive and gain weight and there might also be chronic diarrhoea and/or intermittent vomiting. Seizures may occur if diagnosis and treatment are delayed. In adult animal signs are usually mild until sufficiently longstanding to alter mucosal function.

Treatment

Measurement and/or supplementation of serum cobalamin concentrations in animals with GI disease is important as failure to recognize a deficiency will result in delayed clinical recovery as untreated cobalamin deficiency will lead to progressive megaloblastic and megalocytic changes of the intestinal epithelium that in turn cause generalized malabsorption. Thus chronic intestinal disease of whatever cause may not be successfullly treated until cobalamin stores are replenished by parenteral administration. It is shown that low serum cobalamin concentrations are a risk factor for negative outcome in dogs with chronic en-

teropathies.
Appetite often returns to normal within 12–24 hours, and weight gain occurs over the following weeks. A reticulocyte response will typically occurs 3–4 days post-administration, and there is often a similar burst of neutrophil production.

Red blood cell and neutrophil numbers normalize in 2–3 weeks. I treat all dogs with low B12 levels BUT I treat all cats with levels from mid range downwards as cats are more prone to B12 deficiencies.

Dosages for injectable cobalamin are as follows:
Cats:
For cobalamin deficiency: 250 µg (per cat) SC once per week for 6 weeks, then every 1-2 months based on cobalamin levels or 250 µg SC injection weekly for 6 weeks; then one injection every 2 weeks for 6 weeks; then monthly injections for the patient's whole life.[2]

Dogs:
For cobalamin deficiency - Injectable cyanocobalamin at 25 µg/kg, or practically, 250 – 1200 µg per dog (based on dog's size) SC once per week for 4-6 weeks, then every 14 days for 4-6 weeks, then monthly thereafter to maintain normal serum levels. May take as long as 3-4 weeks to see a response and lifelong therapy may be required depending on the status of underlying cause.

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Protein Losing Enteropathy in Yorkshire Terrier Dogs.

Simmerson SM, et al .Clinical Features, Intestinal Histopathology, and Outcome in Protein-losing Enteropathy in Yorkshire Terrier Dogs. J Vet. Int. Med 2014 (28) 331 - 337

Summarised by Dr Mirinda van Schoor BVSc MMed-Vet (Med)

Why they did it:
Protein-losing enteropathy (PLE) has been diagnosed in many dog breeds including Yorkshire terrier dogs. PLE is characterised by hypoalbuminaemia or panhypoproteinaemia in cases where urinary protein loss or decreased hepatic function were excluded. In Yorkshire terrier dogs and some other breeds PLE has been found to be associated with histopathological evidence of lymphangiectasia and increased mucosal cellularity. Veterinary pathologists interpret histopathologic findings in many different ways, resulting in varied histopathologic diagnoses. The World Small Animal Veterinary Association (WSAVA) published guidelines for the interpretation of histopathologic changes in intestinal biopsies in order to have a more standardised interpretation of findings. Previously published cases of PLE in Yorkshire terriers used non-standardised classification systems for the interpretation of intestinal histopathology samples. This study was performed to investigate PLE in Yorkshire terriers and to describe clinical findings of PLE in this breed.

The study examined intestinal biopsy samples using the WSAVA standardised guidelines for the interpretation of histopatologic changes. The investigators also determined whether any clinical findings or standardised histopathological findings were predictive of long-term survival.

What they did:
They examined the records of Yorkshire terriers that were suspected to have PLE based on clinical signs and hypoalbuminaemia. Thirty dogs were included in the study. The clinical records were examined and clinical findings and clinical pathology results were recorded. Stored intestinal biopsy samples were examined using the WSAVA standardised guidelines. Outcomes were recorded for a period of 4 months after the diagnosis.

What they found:
Most common initial complaints included small bowel diarrhoea (20), vomiting (11), abdominal distention (11) and lethargy (8). The most common clinical findings included ascites (15), dyspnoea and tachypnoea (8), and muffled heart sounds (6). Clinicopathologic findings included mild mature neutrophilia (12) and mild monocytosis (9), hypoalbuminaemia (30), hypocalcaemia (30) and hypocholesterolaemia (24/26). Imaging revealed peritoneal effusions in 15 cases of which 5 had bicavitatory effusions. Histopathologic changes included lymphoplasmacytic inflammation of the lamina propria (30), lacteal dilatation (24/30), crypt lesions (15/17) and villous blunting (17/30). Vomiting was the only parameter found to be a negative prognostic indicator.

Take home message:
Yorkshire terrier dogs can suffer from mild to severe forms of PLE characterised by body cavity effusions, hypoalbuminaemia, lymphangiectasia, crypt lesions and intestinal inflammatory infiltrates. A third of the cases did not have diarrhoea. Vomiting may be predictive of morbidity. Approximately half of the dogs responded to treatment, either completely or partially, while the other half showed treatment failure.

Thyroid Hormone

Interpretation of Assay Results in Dogs

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Reviewed: Prof F. Reyers BVSc Hons. MMedVet(K.Pat).

With the increased availability of in-house hormone assays there is a real risk of incorrect interpretation of test results. Before we go into any details - I would just like to emphasize the title. The definition of the word "interpretation" in the Oxford dictionary is "the action of explaining the meaning of something". I emphasize this, as just looking at the value and using it as a given is not interpretation - it is "reading a result".

Interpretation implies that there are factors which may affect the meaning of a result, and there are many factors which affect thyroid hormone levels in the dog. It is also vital that we realise that use of the thyroid tests may not lead to a definitive diagnosis. Early stages of dysfunction often present with discordant, confusing results.[1]

Most of the time a careful history, physical examination and screening tests and common sense allow the vet to differentiate between non-thyroidal illness/euthyroid sick syndrome (ESS) and true hypothyroidism. Hypothyroidism is common in certain breeds: Dobermans, Schnauzers, Spaniel , Daschunds and Irish setters. It is a disease of middle aged to older dogs, but individuals in predisposed breeds may present as early as 2 years of age.[3] All circulating T4 and only 20% of circulating T3 is from the thyroid gland. The majority of T3 formation occurs outside the thyroid by deiodination (5'-deiodinase). Tissues having the highest concentration of de-iodinating enzymes are the liver and kidney but muscle tissue produces the most T3 due to its volume.[1,3]

Lipid soluble hormones are transported in the plasma by binding to specific proteins, in which there is considerable species variation. Thyroid hormone is highly protein bound in blood (> 99%), T4 greater so than T3. The thyroid hormone binding protein has less affinity for T4 in the dog than in humans - thus the fraction of circulating T4 is higher at 0.1% than in humans at 0.03%.[1,3] The equilibrium is easily shifted by anything impacting the binding proteins. [1,3]

Additionally the dog has only 15% the concentration of thyroxine binding globulin (TBG) of humans thus T4 binds to other protein as well.[1] Thyroid binding proteins in the dog are: thyroid hormone binding globulin (TBG), albumin, transthyretin and apolipoproteins. TBG has a high affinity for T4 (and T3) but low capacity due to low concentrations in the plasma. Albumin has a low affinity for T3 and T4, but a high capacity due to high concentrations in the plasma.[1,3] Tests currently available at veterinary laboratories in south Africa include Total T4 (TT4), canine thyroid stimulating hormone (cTSH) and freeT4 (fT4)

Total T4

TT4 is diagnostic only if the value is normal or elevated. TT4 is about 90% sensitive in diagnosing hypothyroidism in dogs. What this means is that if the animal is hypothyroid - 90% of cases will be correctly identified. However the test is not very specific (75%) – this means that a low TT4 can also occur with non-thyroidal illness. This makes its use, as a single test for the diagnosis of canine hypothyroidism not all that reliable.[1,3] BaselineTT4 can be lowered with non-thyroidal illness and certain medications. The mechanism is thought to be an alteration in the serum proteins which decreases the total binding capacity as the proteins are utilised in the disease process and drug metabolism as well.[1,3]

Sulphonamide drugs can induce true clinical hypothyroidism in a patient as well as laboratory signs of decreased function.[1] Other medications causing a decrease in TT4 are glucocorticoids, phenobarbitone, potassium bromide, propranolol, clomipramine (Clomicalm®) , aspirin, ketoprofen and carprofen (Rimadyl®).[1]

It is thus clear that interpretation of TT4 results should be made with care in patients on chronic phenobarbitone therapy for seizures as well as those on carprofen for arthritis. The effect of other NSAIDS has not been published. A 3 week withdrawal period is required after use of Purbac ® or other sulphonamides as well as glucocorticoids. [1,3]

Certain breeds have also been shown to have TT4 reference ranges which are below the "normal" reference ranges for all dogs - these include sight-hounds (Greyhounds , Salukis , Irish Wolfhounds as well as sled dogs (Huskies , Malamutes).[3] Obesity causes a slight increase in TT4 and TT4 was decreased immediately (24hrs) after a seizure event in patients with seizure disorders.

Canine TSH

TSH is generally elevated only in true hypothyroidism. However elevations above normal also occur with sulphonamides and elevations into the top normal range may occur during recovery from non-thyroidal illness. In humans TSH is an excellent screening test for hypothyroidism - being elevated in almost 100% of cases. Several studies have shown however that about 25% - 40% of dogs with hypothyroidism had normal TSH levels .[1,3] Thus the sensitivity is not great. However the specificity is very good as, apart from recovering non- thyroidal illness patients, TSH is not elevated in euthyroid dogs. A positive cTSH will therefore add specificity to a low TT4 value.

fT4

In the literature you will read about the use of fT4ED - free T4 measured by equilibrium dialysis. This is the most sensitive way of measuring thyroid hormone and is considered the gold standard. In South Africa the laboratories do not use this methodology as it is expensive, time-consuming and require radio-immunoassay. The fT4 you get from your South African laboratory has been done using an immunofluorescence test - and is susceptible to the same interference as TT4. There is thus no benefit in requesting a TT4 as well as fT4 or a fT4 instead of a TT4. It is indicated in differentiating euthyroid sick syndrome (ESS) from true hypothyroidism.Testing fT4-Dialysis would also be helpful in cases on chronic corticosteroids or phenobarbitone therapy where the drug cannot be withdrawn to allow testing.

Antithyroid antibodies (ATA)

The most common cause of hypothyroidism is autoimmune thyroiditis - which eventually results in thyroid atrophy. At the time of thyroid atrophy and hypothyroidism – anti-thyroid antibodies are once again low - as there is no tissue left.[2,4]

In the earlier stages of disease, while the patient is still euthyroid - antibodies will be increased.[4] Some breeds have a very high prevalence of anti-thyroid antibodies and are considered to be high risk for developing hypothyroidism. These are the English setter, Golden retriever, Rhodesian Ridgeback, Cocker Spaniel and Boxer. A familial hypothyroidism has been recognised in Beagles, Giant Schnauzers and Great Danes.[3] The presence of these antibodies provide no information about the function of the thyroid, but they can cause interference when testing for TT4 and fT4, falsely increased readings.[1-4] This may cause a "false" normal result in a patient with clinical signs of hypothyroidism or more commonly an elevated TT4 value in a patient with thyroiditis, which as yet, has no clinical signs of hypothyroidism. The presence of thyroglobulin antibodies in the end stage of thyroid disease where clinical hypothyroidism is present , is in fact very low - so it is is unlikely to cause a false negative finding (normal TT4).[4]

Treatment monitoring.

Clinical response is the best judge of efficacy. Steady state concentrations of replacement T4 will generally be achieved with 1 week. It may be advisable to dose larger dogs at 0.5mg/m2 instead of the 0.02mg/kg bid to oid recommended. Most large dogs will maintain well on oid regimen. The target TT4 level is back to the normal range - as this is not needed to manage clinical signs, values from the 15nmol/L are generally sufficient. (pers obs)The most valuable sampling time is trough levels – just prior to pilling. cTSH will normalise within 1 week of starting replacement therapy and there is no need to test for it when monitoring.

Take home principles :

1. TT4 is very sensitive for the diagnosis of hypothyroidism in dogs (89 – 100%) but not very specific due to euthyroid sick syndrome (60 – 87%. So, you will get up to 40% of false positive results, depending on your case selection.
2. cTSH is not that sensitive and is a poor screening test as it is only elevated in about 75% of hypothyroid dogs (sensitivity 60 – 87%) but is very specific (82 – 100%) .
3. The combination of a decreased TT4 and elevated cTSH is specifically diagnostic for canine hypothyroidism.
4. A normal TT4 in a patient with clinical signs of hypothyroidism may, in a small number of cases, be due to interference by anti-thyroglobulin antibodies. An elevated TT4 in a clinically normal dog is likely to be due to this.
5. The fT4 test adds no additional value to the panel unless the equilibrium dialysis method is used.
6. Hyperadrenocorticism and hypothyroidism have similar clinical signs: weight gain, laziness, increased appetite – although a careful clinical examination should differentiate them. If endocrine function tests are required it is important to be aware that hyperadrenocorticism will have an effect on the thyroid tests - causing euthyroid sick syndrome - whereas the thyroid axis does not affect the tests for adrenal function. Thus if there is a low TT4 and normal cTSH - then the adrenal axis may need to be tested as well to exclude cushings disease.

REFERENCES - available on www.vet360.vetlink.co.za

Thoracostomy Tube Placement

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Thoracocentesis and thoracic drain placement are lifesaving procedures in small animal practice and veterinarians should acquaint themselves with the technique and have basic equipment packs available for those emergency situations. These cases are often not initially stable enough to be referred. Emergency treatment is often lifesaving - the cause of the problem can then be determined at greater leisure.

Indications

A thoracic drain serves to remove air or fluid from the thoracic cavity. This will restore the negative pressure essential to normal respiration and alleviate pulmonary collapse.

Initial management of thoracic effusions or pneumothorax is thoracocentesis, this is therapeutic and diagnostic as the fluid or air removed provides valuable information as to the underlying cause. The removal of the fluid and air will alleviate the respiratory compromise that has been caused by the pleural accumulation. In patients where repeated thoracocentesis is required, a thoracic tube should be placed.

In an emergency patient in respiratory distress with fluid or air accumulation in the pleural cavity there are no true contra-indications to placement of the thoracostomy tube. One can consider thoracocentesis to drain some of the fluid to allow stabilisation of the patient prior to placement of the thoracostomy tube. A butterfly catheter is helpful here, especially in smaller animals such as cats, as the needle can be controlled and held still against the patient whilst syringes are swopped over at the other end of the tubing to drain the chest.

Where there is a large amount of pleural fluid or air - there is no longer a negative pressure within the pleural space - so it is not necessary to worry about a 3-way stopcock when you start draining. As the pleural space empties of fluid - you will notice the fluid start to suck back in through the catheter tube - indicative of negative pressure developing. At this point you need to be more careful and place a 3-way stopcock or kink the line when changing syringes if you have no stopcock. By this time however the patient is also more stabilised and the procedure can be more "managed". In a stable patient pleural adhesions and coagulopathies are the only real contraindications.

Chronic effusions from diseases like chylothorax or heart disease can be manage with intermittent thoracocentsis. More recently a product known as a pleural port has been used. This is placed in the thorax under general anaesthesia and a port placed in the sub-cutaneous tissue, which is connected to the drain. This port can then be aspirated under light restraint when needed.

Placement of a Thoracostomy Tube

Thoracostomy tubes can be placed using either an open or a closed technique. Open is performed during a thoracotomy or sternotomy. Open placement will not be discussed further in this article.

Recommended Equipment for an Emergency Thoracostomy Tube Placement

- Needle holder, haemostat, scalpel handle, thumb forceps and suture scissors
- 2-0 monofilament suture material
- Appropriate size thoracostomy tube (Fig 1 & 2).
- Thoracostomy connectors if needed
- Povodine Iodine and sterile gauze
- Two 3 way stop-cocks
- Two 20 ml syringes
- Bandage material

Figure 1: Various sized thoracic drains with trochar and tube

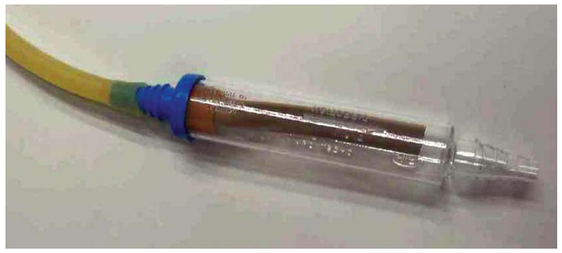

Figure 2: Open flutter valve attached to silicone tubing. Air can move out through the flat rubber tube inside the hard plastic outer casing but cannot be sucked into the chest cavity. Ideal for pneumothorax.

Intercostal Local Anaesthetic

It's preferable to administer local anaesthesia to minimise patient discomfort and movement. If there is no or only mild dyspnoea butorphanol can be administered at 0.05 -0.1mg/kg iv. An intercostal line block should be administered using lignocaine or any local anaesthetic . Diluting lignocaine 1:1 with iv bicarbonate solution will reduce the painful sting caused by the acidity of the solution. The nerves run along the caudal edge of each rib. Inject along your region of interest just as the ribs curve out from the vertebrae. Before injecting, aspirate to ensure you are not in a bloodvessel.

Lidocaine, the most widely used local anaesthetic, takes effect in 3 to 5 minutes and is effective for 60-90 minutes. Mepivacaine (Carbocaine) has a medium duration of action 2-3 hours and fairly rapid onset of about 10 minutes. Bupivacaine (Marcaine) takes longer to take effect (15 to 20 minutes), but its anesthetic and analgesic effects last 6 or more hours. All local anaesthetics cause vasodilation that decreases their duration of action. The duration of blocking agents can be extended by combination with a 1:200,000 dilution of epinephrine. "Washing" the syringe prior to drawing up local anaesthesia provides sufficient vasoconstriction to extend the block and reduce bleeding in the area. This is contraindicated in circumferential limb blocks.

The patient should be in sternal recumbency to reduce respiratory distress. Lateral recumbency is acceptable for pneumothorax and the animal can be rotated in recumbency just as the trochar/tube is positioned. Closed placement is performed under strict aseptic conditions. A generous amount of hair is clipped on the lateral thorax and abdomen. This should be surgically /aseptically prepared.

A small stab incision is made just caudal to the last rib (Fig. 3). The thoracic drain is then passed into the stab incision parallel to the long axis of the dog (Fig. 4). The drain is then tunnelled under the skin a distance of 4-6 rib spaces (Fig. 5). The drain is now lifted perpendicular to the long axis of the body, pulling the skin cranially, with the tip situated in the intercostal space which is going to be penetrated (Fig. 6).

The non-dominant hand is placed around the tube 1 to 2 cm above the skin (Fig. 7). The tube is firmly held by this hand. Using the dominant hand a short sharp bump on the back of the trochar drain is performed. The sharp tip of the cannula will then penetrate the pleural cavity. The non-dominant hand will prevent penetration of the trochar too deep causing damage to the lungs. The drain is then fed along the trochar into the pleural cavity. A thoracic radiograph is taken to ensure correct placement, the tube should run along the sternum to the first rib space (Fig. 10). This is the quickest technique but if done with overzealous force can cause death of the patient.

Alternatively an assistant can pull the skin of the prepared area forwards and the stab incision is made over the desired intercostal space (Fig. 6.). The soft tissue consisting of the intercostal muscles and pleura is bluntly dissected with the tip of the haemostat. Once the pleura are penetrated the tip of the haemostat is opened and the tube passed through the hole created. The skin is now released to created the subcutaneous tunnel. This technique takes more time which one may not have in an emergency.

In a case where there is no available specialist thoracostomy tube a large bore feeding tube can suffice in an emergency. A stab incision is made caudal to the first rib. The tip of the feeding tube is grasped in the haemostat. The haemostat is tunnelled 3-4 rib spaces cranial parallel to the long axis of the body. The tip of the haemostat/feeding tube is placed in the intercostal space and gently pushed through the intercostal

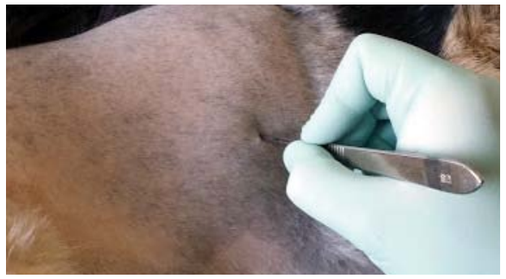

Figure 3: A small stab incision is made caudal to the last rib

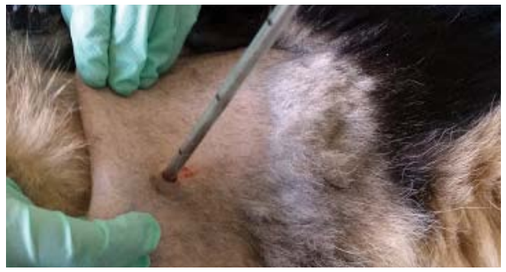

Figure 4: The drain is then inserted under the skin

Figure 5: The drain is then tunnelled under the skin

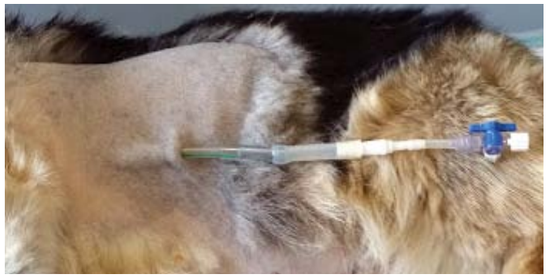

Figure 8: A 3-way stop-cock should be connected to the drain

Figure 6: The drain is lifted perpendicularly, pulling skin forward

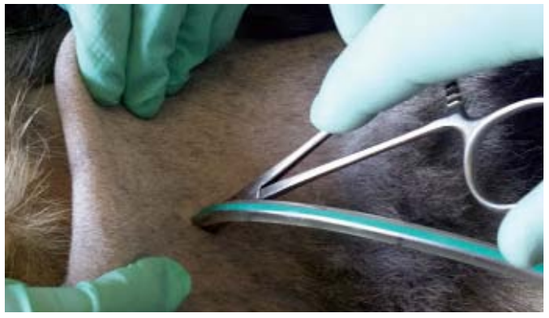

Figure 9: Using a haemostat to blunt dissect and insert tube

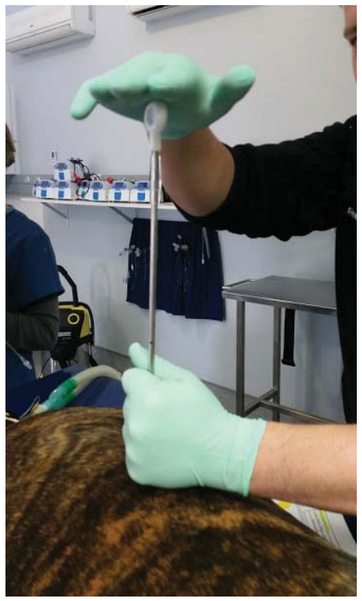

Figure 7: The non-dominant had is placed around the tube 1 to 2 cm above the skin. Using the dominant hand a short sharp bump on the back of the trochar drain is performed.

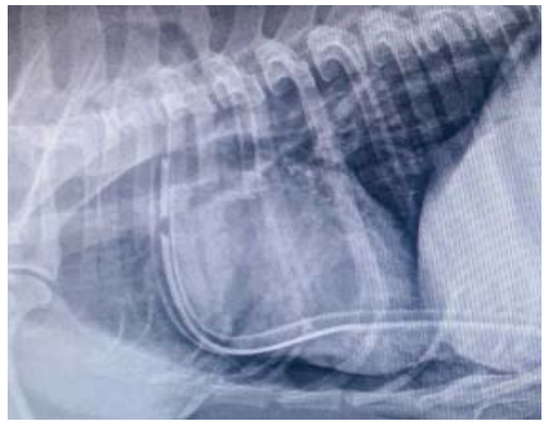

Figure 10: A thoracic radiograph is taken to ensure correct placement, the tube should run along the sternum to the first rib space

muscles (Fig. 9). The feeding tube is now fed into the thorax once the pleural cavity has been penetrated and the haemostat removed.

Once the tube is in place, the air or fluid has been drawn off to stabilise the animal and the placement confirmed with a radiograph. A 3-way stopcock should be connected to the drain (Fig. 8). The tube should be secured with a Chinese finger trap suture. Sterile gauze is placed of the drain skin interface with a blob of antibiotic ointment on the gauze. The drain should be covered with a bandage or Elastomesh to limit patient interference. I will generally superglue all connections to ensure they are not removed.

Drainage

Initial negative pressure should be achieved by aspiration of the drain through one of the 3-way stopcocks. Once negative pressure is established, a decision needs to be made on how continued drainage will be accomplished. The type of drainage selected depends on the cause and type of pleural disease.

Passive drainage relies on the increased intra-thoracic pressure generated during exhalation. This will force air from the pleural space. This has been shown to be very effective in treating spontaneous pneumothorax and traumatic pneumothorax. There is no active suction on the lungs, the theory being this will allow small ruptures in the pulmonary parenchyma to heal where as constant suction could dislodge the fibrin and lead to delayed healing of the tear in the pulmonary tissue.

Passive drains are simple to create, a suction tube is placed on the end of the thoracostomy tube and placed in a bottle of sterile water with at least 15cm of water covering the drain. Another small hole is made in the lid of the bottle to allow the air to escape. Alternatively a fluttervalve can be used. Commercially available pneumothorax drains (Arrow®) use this method and their placement is over the needle catheter requiring very little patient sedation / restraint

Active drainage entails suction consistently placed on the thoracostomy tube. This theory was that this would speed up healing in parenchymal tears. However in current practice it has been found that persistent pneumothorax heals quicker using a passive under water drain as described above. Active suction can be achieved by intermittent suction using a 20 ml syringe on the 3-way stopcock performed by a veterinarian or nurse. There are other devices ranging from expandable bellows to a vacuum pump (portovac) that can provide active drainage. These devices work well in cases of marked pleural effusion or pyothorax.

Care of the Drain

Whenever the drain is handled, the bellows changed, fluid aspirated or the site bandaged it should be done under strict aseptic technique. The bandage should be changed daily and a new dressing placed over the drain skin interface. These patients should be in ICU with constant supervision. Pain control is essential regardless of the underlying cause as the inflammation and the drain cause pleuritis, which is painful. A painful patient will increase its respiratory rate, which in a respiratory compromised patient is not desired.

Removal

Removal is performed once the drain has served its purpose! This varies from patient to patient. For pleural effusion/ pyothorax the drain should be removed when there is a plateau in the production of of sero-sanguinous fluid for about 72 hours at a volume of about 2ml/kg/24 hours. For pneumothorax the drain is removed if there has been no air removed for 24 hours. In the passive water drains, the serous fluid in the suction tube connected to the drain will start to swing and no more air will be seen bubbling in the water. Removal can be done 24 hours after the drains start to swing.

If the air or fluid is still forming after 10 days of having a thoracostomy tube, computed tomography and surgical exploration is indicated.

Complications

The most common complication is improper placement of the tube. Other complications are patient interference with the tube leading to an open pneumothorax, haemorrhage, infection if not handled correctly. If the technique is performed with overzealous force there can be catastrophic penetration and damage to visceral organs resulting in death. However if the drain is not placed death would probably ensue due to the underlying disease.

An Approach to The Anorexic Rabbit

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A history of anorexia is one of the most common clinical presentations in the pet rabbit.

HOW DO WE IDENTIFY THE ANOREXIC RABBIT?

Healthy rabbits graze almost continuously and will produce copious amounts of hard faecal pellets (up to 180) daily. Anorexia will result in a reduction of first the volume and then the size of the hard faecal pellets. A history of poor appetite is an early warning for the clinician that further diagnostics are indicated.[3] Diarrhoea is also a common presenting complaint.

We often try to offer tempting food to rabbits during the clinical examination. A rabbit that shows interest in the food but then ignores it or mouths and then drops the food is often a rabbit with a painful oral condition. A sick rabbit presents immobile, hunched over and oblivious to its surroundings. Be aware that rabbits can initially sit immobile on the examination table, in the "freeze" response and then jump and kick explosively. A rabbit on the table should always be under control to prevent accidents.

Digestive Physiology:

The rabbit is an obligate herbivore and a hindgut fermenter. The digestive system is adapted to a fibrous diet. Digestion in the stomach and small intestine is similar to that of monogastrics. The ingesta reaching the hindgut consists mainly of fibre. This fibre can be divided into two portions: fermentable and indigestible fibre. Both are important for proper gastrointestinal function. The fibre that passes into the proximal colon is divided into two separate portions. Fibres of a greater length than 0.5cm are directed distally and are excreted as hard faecal pellets. These fibres stimulate healthy gut motility. Smaller particles are directed in a retrograde fashion into the caecum. This phase of colonic motility is named the "hard faecal phase". The caecum functions as a bacterial fermentation vat and has a complex and delicate microflora. *Bacteroides spp* predominate in a mixed microflora including aerobic and anaerobic bacteria, both gram positive and gram negative. Small numbers of potential pathogens such as *Clostridium spp* may be present but are not harmful unless changes in caecal conditions allow their proliferation.

Volatile fatty acids are synthesised by the caecal microflora and absorbed as an energy source. The fermentation in the caecum reduces the fermentable fibre to a soft paste containing amino acids, enzymes, microorganisms and volatile fatty acids.

Following a circadian rhythm, usually in the morning and evening, the motility of the proximal colon reverses direction and the caecal contents are expelled and directed towards the anus. This phase is known as the "soft faecal phase". The caecal contents are excreted as soft, odorous clumps of material with a thick covering of mucus. These caecotrophs are reingested by the rabbit directly from the anus and are further digested in the stomach and small intestine. The mucus coating protects the many beneficial bacteria from destruction in the extremely low pH (1-2) of the stomach.[1]

Sequelae of anorexia

Anorexia in the rabbit quickly leads to multiple metabolic derangements. Rabbits are unable to vomit and constantly produce saliva. During normal digestion water is also secreted into the stomach and proximal colon. Re-absorption of water occurs in the caecum and distal colon. For this reason any type of intestinal ileus or obstruction rapidly results in dehydration, electrolyte imbalances and distension of the gut with fluid cranial to the site of obstruction.

As mentioned previously, anorexia can also result in dehydration of the stomach contents forming a so called "trichobezoar". Rabbits with a trichobezoar will benefit from oral fluids and potentially from liquid paraffin. It was previously believed that the oral dosing of pineapple juice helped to dissolve the fibrous mat, due to the proteolytic enzymes in the juice. More recent research indicates that it is likely the extra oral fluid that is making the difference.[3]

Early in the course of the illness rabbits may appear bright and alert but they are predisposed to the development of hepatic lipidosis. During periods of anorexia glucose absorption by the gut decreases and there is a decrease in the amount of volatile fatty acids produced by the caecal microflora. This results in hypoglycaemia which stimulates lipolysis as well as the mobilisation of free fatty acids from the adipose tissue. The liver utilises β–oxidation to metabolise this energy source and ketone bodies are produced. Rabbits do not have effective metabolic pathways to correct acidosis and are particularly susceptible to the effects of ketoacidosis. Triglycerides accumulate in the hepatocytes, further compounding the problem. Hepatic lipidosis occurs most readily in already obese animals.[3]

Liver failure and death are often the endpoint in the chain of events that begins with anorexia. Hypoglycaemia, disorientation and ataxia followed by profound depression may be seen in this terminal stage of the disease.[3]

HOW DO WE APPROACH THE CASE?

It is imperative to establish the underlying cause of the anorexia. A thorough clinical examination including oral exam, abdominal palpation, faecal examination and potentially the use of other modalities such as radiography and ultrasound is indicated. Abdominal auscultation may be used to evaluate borborygmus. Basic haematology and serum chemistries should be run.

One of the most common underlying causes of anorexia in the domestic rabbit is dental malocclusion. The rabbit has aradicular hypsodont teeth that grow approximately 2mm per week. Pet rabbits commonly develop dental malocclusion due to genetic factors, inadequate bone mineralisation (due to a calcium deficient diet or inadequate access to UV light) and inadequate wear of the teeth. A dental exam is a requirement for any rabbit workup. Sharp spurs commonly develop on the cheek teeth that cause pain on mastication and thus secondary anorexia and ileus.[3]

Rabbit dentistry is a speciality on its own so we will cover it only briefly here. Ad-lib access to high energy foods such as pellets often cause the rabbit to eat insufficient amounts of hay as they preferentially select out the most palatable food. The rabbit's dentition is designed for a diet of hard grasses and the constantly growing teeth need to be worn down by the grinding mastication of hay. With inadequate wear, the cheek teeth become overgrown and develop sharp spurs which can cut the tongue or gums. The roots will also proportionately elongate which can cause exophthalmos, sinusitis and lachrymation. The rabbit will then, in a vicious cycle, be even less likely to eat fibrous foods. Eventually the pain causes anorexia.

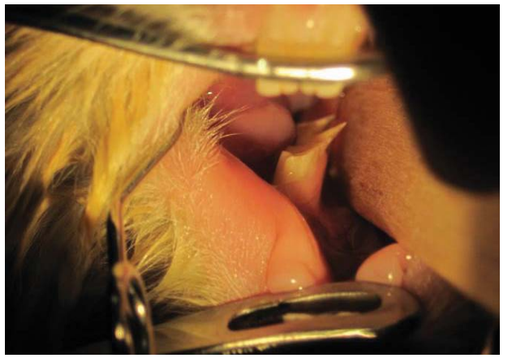

Figure 1: Sharp spurs evident on the cheek teeth

Overgrown cheek teeth are corrected by burring them down to a normal flat occlusal plane. Rabbits that have developed this type of malocclusion should have their teeth re-evaluated regularly as repeated management is likely to be necessary. True diarrhoea is a serious condition, no solid faeces are produced and the rabbit is typically very ill. These animals need aggressive therapy in order to survive.

Common causes of true diarrhoea include intestinal parasites, sudden diet change and bacterial dysbiosis from incorrect use of antibiotics. Antibiotics including Penicillins (especially if dosed orally), cepalosporins, tetracyclines and clindamycin may commonly cause dysbacteriosis. Enrofloxacin, trimethoprim-potentiated sulphonamides and metronidazole are listed among the safer antibiotics.[3]

Owners often mistake uneaten caecotrophs for diarrhoea. These caecotrophs may be found in piles in the cage or may be found tangled in the perineal hair.

Soiling of the perineum will also predispose the rabbit to fly strike. Animals with caecotrophic disorders will typically still pass solid faeces intermittently and will be bright, alert and responsive, often with a good appetite.

Commonly, a gradual increase of good quality fibre (grass hay) in the diet will encourage caecotropy, improve gut motility, control obesity (which can make it impossible to assume the correct position for caecotroph ingestion) and make the gut flora more resistant to sudden stressors.[1,3]

Abdominal palpation and radiography may reveal a distended stomach with gas surrounding a firm (sometimes palpable) mass. Many normal rabbit stomachs

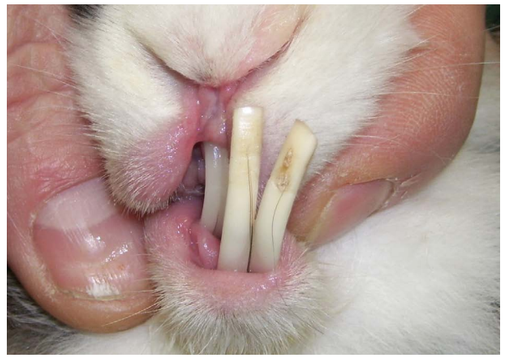

Figure 2: Dental malocclusion due to overgrown incisors

Caecotroph

Faecal Pellets

Figure 3: Normal caecotroph and pelleted faecal matter

contain hair ingested while grooming. In the past it was thought that gastric trichobezoars were a primary cause of anorexia in the rabbit.[1,3]

We now understand that the hard mat of fur and fibre sometimes palpable in the stomach is simply dehydrated normal stomach content and is a sequel to, rather than a precipitating cause of anorexia.[3] Occasionally a rabbit (especially a long haired breed such as the Angora) will develop a true pyloric obstruction. Both gastric and intestinal obstructions are emergencies and typically present with an acute abdomen and a collapsed rabbit. Gastrointestinal surgery on the rabbit is fraught with complications and is considered a last resort. The stomach and intestines are thin walled and fragile and dehiscence of surgical incisions is common. Nevertheless, a truly obstructed animal will need aggressive surgical intervention.[3]

Excess carbohydrate reaching the caecum predisposes the rabbit to bacterial dysbiosis. This occurs commonly in young rabbits in commercial settings where concentrates are fed ad-lib at the expense of adequate fibre. The rabbits typically present a few weeks after weaning with severe mucoid diarrhoea, collapse and death.

HOW DO WE TREAT THE CASE?

Owners should be made aware of the severity and potential fatal outcome of a case of total anorexia and minimal faecal output in a rabbit. These rabbits should be hospitalised for intensive supportive care. Of primary importance is the maintenance of a positive energy balance to prevent excessive lipolysis and hepatic lipidosis. Tasty fresh greens should always be offered to the patient. Good quality grass hay is also needed.[3]

Anorexic rabbits often need assisted feeding in the initial phases of the illness. DO NOT wait too long. Rather begin syringe feeding approximately 10ml/kg of pureed vegetable baby food/soaked complete rabbit diet or Oxbow Critical Care formula for herbivores (most ideal choice) 4-5x daily.[3] Naso-oesophageal tubes may be placed if necessary (8FG works well) but are only used in patients that resist syringe feeding. Most rabbits will need an Elizabethan collar with the NO tube and the stress of the collar as well as the limitations it places on movement and grazing are often counter-productive. Should we need to use a collar at our clinic, we trim it in such a way that the ears are unhindered and the rabbit has easy access to food. We do this by leaving larger flaps of collar laterally and trimming it shorter dorsally and ventrally.

Although the rabbit may not seem to be losing fluids via vomition or diarrhoea, a rabbit with anorexia and gut stasis should be considered dehydrated. Subcutaneous or intravenous fluids (depending on the level of compromise in the patient) should be administered. Lactated Ringers solution is a good choice.[3] Daily maintenance fluid requirements are approximately 60ml/kg/day.

Analgesics are indicated as gas distension of the inactive bowel causes pain which further compounds the problem. Opioids such as Buprenorphine (0.03mg/kg bid) are regularly used and are reported to have minimal effects on gut motility. Non steroidal anti inflammatories such as Meloxicam (0.5mg/kg once daily) are also used for pain control.[2] The use of motility stimulants is a hotly debated topic. Many claim (rightly) that the best stimulator of intestinal motility is long stem unfermentable fibre. Nevertheless, we find that Metaclopramide (0.5mg/kg bid) and Cisapride (0.5mg/kg bid) definitely have a place in our treatment protocol. Naturally prokinetics are contra-indicated in cases of obstruction.[2]

Probiotics have been anecdotally reported to be of benefit in cases of dysbacteriosis. Commercially available probiotics do not contain the normal gut flora of the rabbit but do not seem to be harmful. Caecotrophs can be collected from a healthy "donor" rabbit by placing an elizabethan collar to prevent caecotrophy (ingested) and can then be fed to the patient to re-colonise the caecum.[2,3] Anti-ulcerogenics such as Ranitidine (5mg/kg p/o) and Omeprazole are indicated as gastric ulceration may occur rapidly in a stressed rabbit.[2]

It is important to house ill rabbits correctly. They need quiet quarters, away from possible predator species such as dogs. A bed of hay is often useful both as a good fibre source and as a familiar environment. A hiding box or a covered area should be offered. Rabbits naturally seek out dark, small spaces as retreats when they are frightened. A safe, walled-off area should be available for supervised grazing outdoors. Anorexic rabbits will often be tempted to take fresh growing grass before any other foodstuffs.

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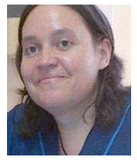

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