Feature Article
A cat with jaundice

Australia’s Poisonous Plants, Fungi and Cyanobacteria

Tick Paralysis - Round Table Discussion

CALL FOR ASSISTANCE - Q fever (Coxiella burnetii) – has anyone been sick in your practice?

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Received a further 10% discount on Howie Sein’s and CVE DVDs and CVE proceedings at a CVE Event.

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CVE WEBINARS

Looking for a quick educational pick-me-up? Watch out for the CVE Webinars in 2012. One hour of education with Dr. Pete Laverty on Wound Management & Reconstruction, Dr. Diane Shelton on Small Animal Neuromuscular Diseases, and Dr. Arthur House on Wound Management & Reconstruction.

CVE Clinical Competency Awards for 2011

Congratulations to Rebecca Ruby who is the Massey University 2011 recipient. Rebecca is entitled to $1,000 worth of CVE products/events due to being chosen by Massey Faculty as being the most competent in clinical skills over the clinical portion of her undergraduate years.

BOOK REVIEW

Reviewer: Anne Fawcett BA(Hons) BSc(Vet)(Hons) BVSc(Hons) CMAVA

Anne Fawcett is a small animal general practitioner, based at Sydney Animal Hospitals Inner West. She is an associate lecturer in the University of Sydney Faculty of Veterinary Science and contributes to a variety of publications including The Veterinary Magazine and The Sydney Morning Herald.

BSAVA/VPIS Guide to Common Canine and Feline Poisons

Authors: BSAVA/VPIS

Publisher: BSAVA

Pages: 174 (softcover/spiral bound)

Year: 2012

ISBN: 97819053195-1

The BSAVA/VPIS Guide to Common Canine and Feline Poisons is a handy (but not quite) lab-coat pocket-sized tool for triaging small animal poisoning cases. The book, a collaboration between the British Small Animal Veterinary Association and the UK’s Veterinary Poisons Information Service, provides key information on a range of toxins known to affect dogs and cats.

It is organised alphabetically, by toxin, and divided into 2 colour coded sections, blue for dogs and marron for cats, which are cross-referenced (for example, under the heading ‘Benzodiazepine exposure in dogs’ the reader is directed to the relevant page on benzodiazepine exposure in cats). The feline section is not surprisingly shorter, reflecting the cat’s more discretionary eating habits.

Information is provided in point form under subheadings for each toxin. These include: alternative names, description/source, toxicity/risk factors, clinical effects (omst, common signs and other signs), treatment and prognosis.

Each potential toxin is designated with a traffic-light symbol, with those marked red likely to cause severe and life threatening signs and those marked green considered of low or negligible toxicity. Many agents in the latter category are included as the VPIS – and no doubt Australian private practitioners – receive many queries about these. It is a crude tool and I found myself on occasion disagreeing with the rating based on clinical experience, but more often than not I felt it was very accurate and spoke to my experience. Perhaps because cats are fussier, there were fewer toxicants designated with a green light for cats (10) as opposed to dogs (21).

The book is not designed to be used as a sole reference on managing poisoning. The sections on treatment and prognosis are by necessity very brief and therefore it may not be enough to enable a practitioner to manage a complex, critical case. You will still need a comprehensive toxicology textbook or (even better) access to peer-reviewed journal articles on management of particular toxicities. However, The BSAVA/VPIS Guide is
Australia's poisonous plants, fungi and cyanobacteria

A guide to species of medical and veterinary importance

Ross McKenzie

CSIRO Publishing
(www.publish.csiro.au/pid/6507.htm)
ISBN: 97806430932679

Australia’s Poisonous Plants, Fungi and Cyanobacteria (APPFC) is a rare example of a magnificently presented book replete with valuable information, authoritative, comprehensive, and, amazingly, just readable. It is the literal equivalent of the signature dish from a 4 hat chef. The reader will be struck by the number and clarity of the photographs of plants and fungi associated with significant possibility of poisoning.

The author, Dr Ross McKenzie, now retired to tend his garden of toxic plants, is one of the very few veterinary toxicologists in Australia and well known for his passionate and infectious interest in the subject. This passion and dedication to teaching and learning are fundamental characteristics of this monograph, imbuing it with extraordinarily high value.

At the very start the aim is captured by the Chinese proverb that it is ‘better to light a candle than to curse the darkness’. Indeed this is the first reference book shining a light on plant, fungal and cyanobacterial poisonings of humans and animals (wildlife and domestic) throughout Australia. Information is provided to allow the reader to recognise the main potential poisonous plants, fungi and cyanobacteria and the circumstances leading to poisoning.

The author states that he has written the book to help ‘householders, gardeners, parents of young children, child-care workers, school teachers, Bushwalkers, pet and livestock owners, landholders, land custodians, medical and paramedical professionals, veterinary nurses, agricultural advisers, botanists, park rangers and everyone else interested in the subject’. This is a very broad reach and an ambitious goal – a goal that I believe has been easily achieved, especially from the perspective of veterinarians, pet and farm owners.

The opening sections of APPFC provide an excellent description of what the book includes and does not include (for example, mycotoxins of stored food, produce and plants causing contact dermatitis are not included), an outline of how to use the book followed by illustrative chapters on understanding plants and plant poisonings (including plant, environment and animal factors that increase risk of poisoning) and how to confirm tentative identifications.

Now better armed with an enhanced appreciation of toxic plants, Chapter 3 (54 pages in length) is devoted to common poisoning profiles. A total of 47 syndromes are described and importantly only those that are likely to be seen in practice (‘street list’) and likely to be effective treatment. The syndromes described include Acute liver necrosis (15 sources of toxins identified), Cardiac glycoside poisoning (21*), Cocaine tripterygine poisoning (4*), Cyanide poisoning (27*), Diethylthiocarbamoyl poisoning (5*), Fern norepinephrine-glycoside (4); Ergot alkaloids (5); Fluoroacetate (20); Gastrointestinal (4); Glucosinolate (4); Grayanotoxins (4); Iforrestine (2); Irritant diterpenoids (4); Macrofungal gastrointestinal syndrome (5); Macrofungal neurotoxic syndrome (4); Mucous membrane irritation (4); Nicotine and other pyridine alkaloids (5); Nitrate–nitrite (15)*; Osseous ulceration of horses (Chillogale horse disease); Oxalates (iodine) (27*); Oxalate-crystalline intoxications (big head of foal); Paraquat (4); Phenothiazins (lupinosis) (1); Photosensitization (15)*; Pigeon glycosides (5); Protokarotoxins (4); Phytotoxins (2); Pyrrolizidine alkaloids (2); Seconal (2); Simplexins (6); Steroidal glycoalkaloids (35); Stramonium (2); Sulphur (8)*; Sulphur containing organic compounds (e.g. S-methyl-L-cysteine sulfoxide SMCo & N-propyl disulphide) (13); Sodium and calcium cyanides (9); Tamin (5); Thiaminases (4)*; Toxalbumin (4); Tropaeoline (2); Tropaeolins (8)*; Urushiol (3); Vamps (6); Zamia staggers (5).

The next 570 pages provide profiles of the individual poisonous plants, fungi and cyanobacteria. Part 1 Poisonous cyanobacteria (blue-green algae), Part 2 Poisonous fungi, and Part 5 Poisonous vascular plants (with chapters on ferns, grasses, sedges and mat-rushes, grasses, grass-like herbs (iris and lily families), forbs (non-grass-like herbs); vines (climbing plants and creepers), shrubs, and trees).

The author attests that all profiles are evidence based, meaning that all information is obtained from actual documented cases of poisoning. For each genus or species the profile contains a colour photo and information on the following subjects: Names (scientific, common, etymology, family). Description (plain language, flowering and fruiting seasons; Main distinguishing features). Confusing species. Distribution and habitat (including map). Weight of evidence for toxic effects. Degree of danger (scored on palatability, dose and concentration, rapidity of action, severity, effectiveness of treatment – very dangerous – 9.10, low danger 6.5); Toxic name; Toxic plant parts; Animal species affected; Conditions of poisoning. Toxic dose; Clinical signs; Post mortem changes. Management of intoxication.

The final chapter presents what is entitled ‘A Digest of poisonous cyanobacteria, algae, slime moulds, macrofungi and plants found in Australia’ which is a section summary of other information on toxins, animals at risk, toxic syndrome, weight of evidence of toxicity and classification of degree of danger for all known species of poisonous species in Australia – a total of approximately 2,000 entries.

As if this is not enough, the book is completed with 6 appendices, a comprehensive glossary, references for further reading and a detailed index.

The appendices provide information on aids to identifying flowering plants. The top killers (the most dangerous plants, fungi and cyanobacteria in Australia). Poisoning hot-spots (in the home, garden and on the land); the major species poisoning animals; Body systems affected by the major poisonous species; and Australian states with major poisonous species.

The author includes many key messages. For example, the importance of accurate identification of suspected toxic plants, fungi and cyanobacteria is emphasised on many occasions. Such identification will frequently require consultation with experts in other fields (including botanists and mycologists). Clearly no diagnosis can be certain unless an accurate identification of the source of exposure is known.

The author also notes that ‘unpublished data are data lost’ and entreat those encountering poisoning episodes to ensure that the identity of the implicated toxic plants, fungi, cyanobacteria etc is established in perpetuity by placing a voucher specimen in an appropriate institution. Finally, McKenzie’s ‘Maxim – the animal species does not determine the circumstances make the poison’ is certainly worth recalling.

Inevitably when the objective is to provide information to a large and varied audience the book cannot be expected to be definitive. However, guides to identifying poisonous species and an atlas of poisonous species and a web-based database of poisonous species are expected to help establish an accurate diagnosis. It would be invaluable to have access to the vast literature underpinning the evidence base summarised in the book. However, with around 2,000 species of poisonous organisms included there could well be 45,000 species which might occupy another 200 pages.

Australia’s Poisonous Plants, Fungi and Cyanobacteria is an ambitious and excellently produced book which very well bridges a gap of 30 years since the last edition of Selwyn Everist’s ‘Poisonous Plants and Fungi of Australia’ (1981) which provided descriptions of around 350 species (with few photos and no distribution maps) and Alan Seaward’s ‘Common Poisonous Plants and Fungi of Australia (Volume 2, 1982)’ which focused on 204 species.

I believe that Australia’s Poisonous Plants, Fungi and Cyanobacteria should occupy an important and prominent place on a bookshelf readily accessible to every veterinarian in clinical practice as well as those supporting clinicians or with other interests in animal health.

Editor’s Note: We give the last word to Ross – I have included this as an example of a book that does not flow like a single entity, but one where ideas and information are not linear. There are no literature citations, just a short, ending list, and I have tried to write in a plain language style so that non-professionals will have a chance of understanding. Any poisonous species and the profile contains a colour photo and information on the following subjects. Names (scientific, common, etymology, family). Description (plain language, flowering and fruiting seasons; Main distinguishing features). Confusing species. Distribution and habitat (including map). Weight of evidence for toxic effects. Degree of danger (scored on palatability, dose and concentration, rapidity of action, severity, effectiveness of treatment – very dangerous – 9.10, low danger 6.5); Toxic name; Toxic plant parts; Animal species affected; Conditions of poisoning. Toxic dose; Clinical signs; Post mortem changes. Management of intoxication.

Readers should realise that it has 2 functions: (1) information source and (2) exercise machine (it weights 3.8 kg and repeated use will strengthen your arm muscles).
Examining the afterbirth of the mare

CAT No. 5243

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We can tell many things by examining the afterbirth (the placenta) of the mare after it has been dropped following birth of the foal.

• Is it complete or has some been retained?
• Is the mare healthy and has she had a healthy pregnancy?
• Is the placenta normal as expressed in appearance and weight?
• Is there evidence of placentitis?
• Is the placenta small and inadequate for the foal?

What can we learn from this?

The immediate application is the health of the mare following foaling. The presence of pus, excessive blood, or a retained piece of placenta are issues requiring immediate attention. Postparturium infections, haemorrhage or laminitis can very quickly become life threatening.

Information derived from the placenta can be essential for periparturient care of the neonate. A foal with an infected placenta may rapidly become septicaeic. Once they are clinically ill, reversal of the situation can be very difficult. Early intervention can be critical. On the other hand a foal that has been undernourished in the uterus will need extra care and the sooner it gets it the better. Neonates exhibiting signs of IUGR are not only smaller but are more likely to succumb to infection. Also, premature maturation of the stressed membranes will include the lining of the gut reducing absorption of immunoglobulins. Parenteral supplementation of immunoglobulins may be necessary.

Presentation of a healthy placenta without any retention is a good start when looking to get the mare back in foal, especially if looking to breed on the foal heat, but any deviation from this is not only important in achieving pregnancy but maintaining it.

Figure 1. Afterbirth (placenta) of the mare

How to check the placenta

Firstly check that the tips of both horns are present. Retained pieces of placenta are usually at the tips of the horns and it is almost always the tip of the non pregnant horn that fails to separate. If you look carefully at the end of the horn it is possible to see the small ‘star’ where there are no villi and this is the point at which the placenta is adjacent to the papilla of the oviduct. If the star is not visible and the placenta is torn at that point you must re-examine the uterus as the tip of the horn is probably still in there.

How do you know which is the horn of the uterus? If you hold up the uterus by the tips of the horns the pregnant horn will hang down the way it was in the uterus as it is bigger and more pendulous. The placenta will hang down with the shorter side being the upper side. You will then be able to tell if the missing piece is in the right or left horn.

The tear where the foal has come through the placenta is usually at the cervical pole – rarely if ever at the tip of the horn.

The placenta may rapidly become septicaemic. Once they are sick it can become life threatening.

If you look at the placenta you will see the area where the cervix and the placenta are in apposition. Placentas that have had an infection are often oedematous and so will be thickened and heavier. On the other hand a small underweight placenta may result in inadequate foetal nutrition and intrauterine growth retardation (IUGR). A normal placenta with a healthy mare is a good indication for the next pregnancy as well as the health of the current offspring.

The uterine surface of the allantochorion should have a rich red velvet appearance with healthy villi. Uterine abnormalities such as cysts which have no villous attachments, will be mirrored on surface of the allantochorion, as will be the site of the endometrial cups.

Figure 2. Checking the placenta

Figure 3. Close-up of the placenta

Large Animals

The C&T and Perspective Series is the brainchild of Dr Tom Hungerford, first Director of the PFG (1968-1987), who wanted a forum for uncensored and unedited material. Tom wanted to get the clinicians writing.

‘…not the academic correctness, not the theoretical niceties, not the super correct platitudes that have passed the panel of review… not what he/she should have done, BUT WHAT HE/SHE DID, right or wrong, the full detail, revealing the actual ‘blood and dung and guts’ of real practice as it happened, when tired, at night, in the rain in the paddock, poor lighting, no other vet to help.’
**WINNER OF BEST FILM CLIP**

Compiled at the Currumbin Sanctuary Wildlife Hospital by Mimi Dona © 2010

Part 2: Wildlife Flashcard Series - Birds
C&T No. 5244

This series is the result of collaboration between Mimi Dona & Dr Michael Pyne of Currumbin Wildlife Sanctuary Veterinary Hospital. Film clips courtesy of Lincoln Williams, Fotomedia (www.fotomedia.com.au). Non CVE members can access these flashcards and videos at www.cve.edu.au/candt2012

**BIRDS**

Be Aware of:-

- Many birds are capable of defending themselves using either: their beak, claws, wings or a combination of these.
- Never put in wire cages as they can damage their feathers; line with cardboard or shade cloth if no other options. A cardboard box or pet carrier is more suitable.
- Some birds have heavy feathering e.g. nocturnal raptors and waterbird_swan.
- Preferred enclosure temperature is between 28º - 35º degrees celsius.
- Some birds have heavy feathering e.g. nocturnal rapto and sea birds so take care not to over heat. Keep below 28ºC unless young or unfeathered.
- If unsure of the species, the safest food to feed a baby bird is soaked dog kibble.

**Emergency diet**

- Bird have specific dietary requirements that may not be able to be met in a Veterinary Clinic. The following emergency diets are not long term.
- Smaller or baby birds have a high calorie requirement and need to be fed frequently. This varies with species and age.
- If unsure of the species, the safest food to feed a baby bird is insects.
- Many birds will not eat in hospital and will need to be assist fed or crop fed.

**Assessment under gaseous anaesthetic**

- Water bird and pigeon like species (columboforms) are slow to induce.
- Fasting is required for birds prior to an anaesthetic. Take care with very small birds under 20 grams not to fast for more than 2 hours.
- Native vegetation and flowers are essential to many birds.
- If unsure of the species, the safest food to feed a baby bird is soaked dog kibble.
- Give IPPV routinely throughout the procedure to reduce the risk of mucous plugs in the endotracheal tube.
- Many birds will not eat in hospital and will need to be assist fed or crop fed.

**Housing the sick or injured bird**

- Some sea and water birds can have pointy or knife like beaks.
- Some species, particularly birds of prey, will require a high level of fitness prior to release and may need specialised training or a large flight aviary.
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**Intubation**

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- If unsure of the species, the safest food to feed a baby bird is soaked dog kibble.

**Fluid Therapy**

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**Euthanasia methods**

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**Anaesthetic agents**

- Propofol/Antithal (10mg/kg) IV – Often used in large waterbirds (Pelican, Swans).

**Housing the sick or injured bird**

- Some sea and water birds can have pointy or knife like beaks.
- Larger birds can be handled by gently securing the head with one hand and holding the body in the other, be careful that the wings are contained.
- The best temporary set up is a box (not ideal long term for parrots who can chew their way out) with air holes away from their eye line. Waxed cardboard pet packs are ideal and easy to clean. A rolled up towel will provide a perch and keep the bird elevated protecting its tail feathers.
- Young birds not perching need a nest – use a bowl with a small towel. A feather duster can be used to mimic mum. If the bird has no feathers it will require warmth.
- The debilitated bird should be placed on a doughnut shaped towel until it is able to perch.

**Medium sized birds**

- Can be handled by holding your hand around the wings with the feet secured between your fingers.
- Parrots by using a pistol grip to hold their head.

**Housing the sick or injured bird**

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- The debilitated bird should be placed on a doughnut shaped towel until it is able to perch.
Feline Herpesvirus-1 as a differential diagnosis for feline facial pruritus

Joseph A. Bernstein DVM Diplomate ACVD Long Green Animal Dermatology Center, PC, Consultant Antech Diagnostics Baltimore, MD USA djose@longanimalderm.com

This recurrence may present as a striking eosinophilic dermatitis of the face and/or nasal planum (commonly on the extremities) that is easily mistaken by both clinicians and histopathologists for allergic eosinophilic dermatitis or eosinophilic granuloma complex. While the literature indicates pruritus may be moderate to absent (Gross et al., 2005), some cases have marked pruritus and resulting self-mutilation which compounds the clinical confusion with allergic pruritus. This commonly results in a fruitless diagnostic work-up for allergic disease as well as ineffectual and possibly aggravating immunosuppressive therapeutics.

Histopathology of biopsy specimens reveals a necrotizing eosinophilic dermatitis. Intracellular viral inclusions may not be present or may be easily missed by the histopathologist. Both PCR and immunohistochemical techniques have been investigated and validated as screening tools for herpetic dermatitis (Hargis et al., 1999, Persico et al., 2011). However, the discovery of the safe and effective use of the viral antiviral, Famciclovir, has revolutionized our ability to both diagnose and treat this disease (Thomasy et al., 2006, Malik et al., 2009, Thomasy et al., 2011). The author recommends a 2 to 3 week empirical course of Famciclovir as a diagnostic rule out in cases of feline facial dermatitis and pruritus where herpes may be suspected (125 mg PO BID). In the author’s experience, response is noted in many cases of neutralic facial excoriation and dermatitis that were previously misdiagnosed as allergic in etiology and treated unsuccessfully with steroids and/or cyclosporine. The veterinary practitioner should be mindful of this insidious differential diagnosis when confronted with refractory feline facial dermatitis cases.

REFERENCES

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Adenocarcinoma Feline

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A 9-year-old male, neutered, domestic short hair presented because the owner identified a small, discharging wound on the ventral abdomen. The patient weighed 7 kilograms and had a body condition score of 6 out of 9. There was no history of a catfight or trauma. The cat was an outdoor cat and fed a diet of Hills Feline Adult. Four days prior to presentation (05/04/12) serum biochemistry and a complete blood count were performed (see Tables 1 and 2) because muscle wastage was identified when the patient presented for annual vaccination. Results of the biochemistry and complete blood count were unremarkable.

On physical examination a serosanguinous discharge was identified from a superficial wound on the caudalventral abdomen. Examinaiton identified 2 puncture marks 15mm apart. Erythema was present surrounding the puncture marks. Differentials at the time were catfight or an insect bite. The patient was treated with cefovecin 8mg/kg subcutaneously.

The patient returned 14 days later for a recheck. Over that period appetite had reduced, the cat had lost weight (1.4kg) and the patient was lethargic.

Physical examination showed pale mucous membranes. Marked pain was elicited on palpation of the ventral abdomen. Severe bruising and erythema were present on the ventral abdomen (see Figure 1). The lesion felt soft and fluid filled. The cat had generalised oedema.

A complete blood count was performed (Table 1). A mild regenerative anaemia was identified possibly secondary to blood loss from the wound or haemolysis. Blood loss was considered more likely due to a concurrent hypocalbuminaemia. A moderate neutrophilia was present with toxic change suggestive of an inflammatory response possibly related to infection. Serum biochemistry identified hypocalbuminaemia, which was consistent with protein loss through the skin lesion. Electrolytes abnormalities were present due to inappetence or third space fluid loss. Addison’s disease was considered unlikely.

Differential diagnosis at the time was a pansteatitis secondary to atypical infection (actinomycosis, actinobacillosis, mycobacteriosis, nocardiosis), deep mycotic infections, neoplasia, foreign body and steatitis secondary to pancreatitis or immune mediated disease. Nutritional deficiencies secondary to excess consumption of fatty acids or a vitamin E deficiency were also considered.

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Pancreatic carcinomas are often associated with subcutaneous fat necrosis. 11 It has been hypothesised that the aetiology is fat necrosis secondary to systemic release of lipolytic enzymes. Cytology of the fine needle aspirate identified an exfoliated, lobulated carcinoma (see Table 2).

It was hypothesised that the patient had adenocarcinoma of the pancreas with secondary pansteatitis. The animal was euthanised on request of the owner. A post mortem was declined. Culture and sensitivity of the fluid was cancelled.
We need your help...

CALL FOR ASSISTANCE

Q fever (Coxiella burnetii) – has anyone been won’t find in a textbook

University of Sydney researchers, Amanda Shapiro (PhD candidate) and Drs Jacqueline Norris and Karina Bossward, are investigating the potential role companion animals may play in the transmission of Coxiella burnetii to humans. This research has been sparked by an outbreak of Q fever in veterinary personnel in a Sydney companion animal veterinary hospital in 2010, following a cat caesarean section. In the preliminary stages this is being investigated by determining the seroprevalence of previous exposure to Coxiella burnetii in dogs and cats in eastern Australia as well as investigating the potential role of this organism as a cause of disease in our companion animals. We would therefore love to hear from any veterinarians and veterinary nurses who have been confirmed or are suspected of having Q fever resulting from contact with infected and cats. All that we require to further develop our diagnostic assay is a serum sample from the suspected or confirmed animal. All cases will be dealt with strict confidentiality.

Please contact and supply further information to: Jacqui Norris, jacqui.norris@sydney.edu.au

Karina Bossward, karina.bossward@sydney.edu.au or Amanda Shapiro, amanda.shapiro@sydney.edu.au

ADRENALS: What you won’t find in a textbook

Re-published here courtesy of Vetnostics, North Ryde

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Practice Tips

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Muzzles: For clients who get a bit ‘cranky’ when you announce they are going to muzzle their dog, politely advise them it is a Wicket Cover (OHS in Victoria) issue, WHICH IT IS. The difference in their attitude is astonishing! We take no risks with dogs that even MOUTH bite!

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Karina Bossward, karina.bossward@sydney.edu.au or Amanda Shapiro, amanda.shapiro@sydney.edu.au
1. Lack of a stress leucogram in a sick dog can be an indication for hypoA and may be the only clinicalpathologic abnormality in dogs with glucocorticoid deficient (atypical) hypoA. When I ask vets about the leucogram in suspected hypoA cases, the common response is ‘Everything is normal’. Remember, a normal leucogram can be quite abnormal for a collapsed dog and each count should be assessed with respect to the dog.

2. Lymphocytosis is often always present.

3. Lymphocytosis is a good ‘rule-out’ for hypoA. I have never seen a hypoA case with lymphopenia. However, a recent study on lymphocyte counts in dogs with hypoA (Seth et al 2011) did identify few low lymphocyte counts. In this study, 100% of hypoA dogs had a lymphocyte count <0.75x10^9/L and 92% had lymphocyte counts <1.00x10^9/L.

4. Eosinophilia is also not always present.

5. Alkaline aminotransferase (ALT) is commonly increased but could be hyposthenuric, isosthenuric or concentrated. In one report (Peterson 1984), many dogs can present signs; only 82% of 300 hyperA dogs had PU/PD (presumably excessive mineralocorticoid secretion).

6. Urea concentration may be decreased due to polypocephalus.

7. Hypoalbuminemia and hypernatremia may occasionally be seen and are probably more common in dogs with adrenal tumours as the cause of their hypoA (presumably excessive mineralocorticoid secretion).

8. Bile acids test results may be increased in dogs with hypoA (Center et al 1985).

9. Serum lipase may be increased by exogenous corticosteroids.

From my experience (Seth 2011) in my own patients or Vetstudent cases. Whilst hyperglycaemia can occur, it would seem to occur at a much lower rate in our patients.

Tumours as the cause of their hyperA (presumably excessive mineralocorticoid). Again, not necessarily. It is not usually increased to the same extent as ALP.

Hyperadrenocorticism (HyperA)). All which will have familial hypertriglyceridaemia; Miniature Schnauzers, not of this breed (Seth et al 2011).

1.  Lack of a stress leucogram in a sick dog can be an indication for hypoA and may be the only clinicalpathologic abnormality in dogs with glucocorticoid deficient (atypical) hypoA. When I ask vets about the leucogram in suspected hypoA cases, the common response is ‘Everything is normal’. Remember, a normal leucogram can be quite abnormal for a collapsed dog and each count should be assessed with respect to the dog.

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Replies to Tick Paralysis in the cat

C&T No. 5193, Dec 2011, Issue 264

Dr Gasch’s nicely summarised an approach to tick paralysis in cats which is very similar to the way I personally treat these patients. The one exception was his comment regarding the pulmonary edema, ‘consider chest radiographs (dx: pulmonary oedema), tx frusemide 1-2mg IV q6hrs’. It is my view, and that of many of my friends and colleagues in Sydney, that we do not observe cardiological pulmonary oedema in cats. Only dogs caused by paralysis tick envenomation. Therefore, the use of frusemide is not only unwaranted, it potentially compromises renal perfusion in dehydrated, sedated cats.

Dr Campbell’s PhD studies in 2000 looked at cardiovascular function in canine tick paralysis patients and concluded that cardiological pulmonary oedema does occur in dogs and postulated that it is the likely contributor to death in severe cases. She claimed the heart failure was due to diastolic dysfunction; however, this was based on M-mode measurements (2DMD) and not more accurate measures of diastology like mitral inflow studies or tissue Doppler imaging (TDI). I suspect mitral inflow studies were not routine then, and certainly TDI was in its infancy. However, in 5 patients where BALs were performed, the fluid collected was low in protein and therefore not consistent with cardiological pulmonary oedema. Her study was well designed and certainly suggests cardiological pulmonary oedema occurs; however, we just don’t seem to see it clinically.

Figure 1. lodoes holococlic paralissis tick in water  (Courtes of Anne Fawcett)

Figure 1. Iodoes holococlic paralysis tick in water (Courtesy of Anne Fawcett)

My mentor, Dr Richard Churche, began investigating this hypothesis because of the discrepancy between what was reported and what we were seeing in practice and performed echocardiography on several dogs with acute tick paralysis. No abnormalities were found using mitral inflow studies. We contemplated conducting a prospective controlled study, but we considered it too challenging for many reasons to undertake full echocardiography while experiencing severe respiratory compromise and aborted the idea.

In our referral hospital, we may treat up to 5-10 tick paralysis patients on any given day during the tick season, usually with 1-2 receiving mechanical ventilation, and never have I used frusemide to treat any patient in Sydney (especially those treating large numbers of tick paralysis patients) that use frusemide routinely, if at all. I believe the only worthy indication is in the combination of respiratory muscle paralyis/paralysis (including upper respiratory tract structures causing upper airway obstruction), aspiration pneumonia, severe haemorrhage, severe dehydration, or severe renal oedema (perhaps secondary to upper respiratory tract obstruction – especially in cats). In severe cases, we provide mechanical ventilation, continuous positive airway pressure (CPAP), extensive respiratory physiotherapy, reoblution, antimicrobials if indicated, frequent arterial blood gas analyses and careful nursing care; very rarely do we lose patients (if costs are not an issue...). I believe the cause of death in most cases is severe respiratory acidosis (due to ventilatory failure) and occasionally severe aspiration pneumonia or ventilator associated infection. Patients can rapidly become acidemic from hypoventilation, often with pH values below 7.0. We presume such patients, if untreated, develops cerebral oedema, followed by death. With increased monitoring of arterial blood gases, we are much more aggressive with ventilation and as a result very few patients die (I would estimate less than 1 patient each season). Furthermore, all those where food and water are withheld (all but the very mild cases) receive maintenance intravenous fluid support from admission.

I am not suggesting there are no cardiac effects from the toxin; however, I do think practitioners need to be well aware of the dangers. Dr Gasch’s reported dose of frusemide is very low, which I think may be sub-therapeutic. The concern is that vets will use increasing higher doses in patients that are seemingly not responding when perhaps they require intubation to relieve the obstruction (especially cats) or artificial ventilation for worsening hypercapnia (especially dogs). In short, I don’t believe cardiological pulmonary oedema is likely in dogs and certainly not cats.

Q & A (Qs by Richard Malik/As by Karina Graham)

1. Have you read the likw papers?

I skimmed them, they are quite old so I didn’t read them in detail. To be honest, if they said ‘without a doubt’ that congestive heart failure is caused by tick paralysis I still wouldn’t take too much notice – because we don’t see it clinically. It MAY have existed, I don’t know. Now you have me more interested, I will review them further... Perhaps the best thing is to ask Fiona Campbell her thoughts? She is an outstanding cardiologist who used to (and may still does evaluate) many tick paralysis patients. Do they treat any tick paralysis patients for heart failure? If so, how come we go away without treating them? 

Editor’s Note: Dr Campbell’s Comment will be published in our December issue.

2. Can you extrapolate from dogs to cats?

There are differences i.e. cats appear to suffer from upper respiratory tract obstruction more than dogs, and often you can just sedate them and intubate them. No frusemide though!”

Round Table Discussion

What's YOUR Diagnosis?

The alarming drops in HCT from the mid-20’s to around 10% seemed inexplicable without a source of haemorrhage. Indeed it was felt that the check for constipation had triggered the drop, indicating a friable-blood filled lesion in the abdomen.

While she is described as a DSH, she had the appearance of a British Shorthair cross and this made it statistically more likely that FIP might be a problem, however, it was ruled out at the end.

Conclusion

Other than the obvious of always providing yourself with a thorough minimum database, there is much to be learnt from this unusual case.

Upon consultation with specialists Dr Vanessa Barrs from Sydney University and Dr Richard Malik from the CVE, it seems likely that the right kidney and the liver were indeed affected, whatever their gross appearances. I am also told that a renal lymphoma can bubble out in size very rapidly, explaining why this obviously huge kidney was not felt until very late. Dr Malik also reported that cases of feline renal lymphomas diagnosed in cats older than 18 months of age have a survival time of about 3-4 months. The younger cats respond better to multi-agent chemotherapy and may survive for many years. So the outcome was never likely to be a good one. However, there is a lingering sense of not having risen to the occasion when presented with the unexpected.

Discussion

Names clearly have been changed or suppressed to protect the innocent!

The relatively swift change from clindamycin to doxycycline followed by seemingly erratic changes in laboratory results raised my suspicion of a false negative smear examination and hopefully inducing a more thorough search of the pathogenesis of her illness.

There were a number of mistakes made that stemmed from the sheer disbelief of a problem of this magnitude in a young healthy cat. Common things occurring commonly, the pyrexia was ascribed to an upper respiratory tract infection.

Maxine, a 4 year old neutered female cat, presented to the practice after having been in the yard for 4 hours. The owners were very concerned due to the fact that she had strayed. Upon examination she appeared to have a high fever (HCT 45%) with submandibular lymphadenopathy, an enlarged and abnormal left kidney; 53.4 mm length (Normal: 36.6 +/- 4.0mm) and 40mm height (H: 22.1 +/- 2.8mm) and mixed echogenicity in the left kidney, (Figure 1). I performed a moderately experienced rural ultrasonographer the rest of the abdomen, including the liver and right kidney, appeared normal.

A decision was made to use the time-honoured ultimate diagnostic tool of a laparotomy, while she was stable enough. A decision was made to use the time-honoured ultimate diagnostic tool of a laparotomy, while she was stable enough.

She had been on fluids since the transistion, and was urinating though not otherwise dramatically improved.

A routine midline laparotomy was performed. There was no trace of current or past haemorrhage and the fluid collected was straw-coloured and formed a stable foam when shaken. As per the ultrasonography finding was of a grossly abnormal and enlarged left kidney with extensive adhesions (Figure 2). A nephrectomy was performed and the abdomen was routinely closed.

The following day the samples were sent to the laboratory. The fluid came back negative for FIP. The kidney histopathology showed a poorly differentiated lymphoma.

Before the results were back Maxine crashed again (HCT 10%) and her owners elected euthanasia. She was buried next to her mate the dog, 38 days after first examination.

Dr Gasch has nicely summarised an approach to tick paralysis in cats which is very similar to the way I personally treat these patients. The one exception was his comment regarding the pulmonary edema, ‘consider chest radiographs (dx: pulmonary oedema), tx frusemide 1-2mg IV q6hrs’. It is my view, and that of many of my friends and colleagues in Sydney, that we do not observe cardiological pulmonary oedema in cats. Only dogs caused by paralysis tick envenomation. Therefore, the use of frusemide is not only unwaranted, it potentially compromises renal perfusion in dehydrated, sedated cats.

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Round Table Discussion

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Dogs more often require mechanical ventilation. However, other species can have it the other way around too.

3. What about measuring pulmonary wedge pressure and cardiac output? Maybe. We would need to organise a prospective study to sort this out.

4. How many dogs did you or Dr Churcher echo?
I’ll ask him. I’m not entirely sure. I’m guessing a handful of cases, but they were before my time; that’s why I confidently don’t use any heart failure medication. However, it is very difficult to restrain these patients for thorough echocardiography.

5. Do you use CPAP or PEEP?
Both. PEEP when using the ventilator (for many patients) and CPAP mainly when we’re weaning them from ventilation. CPAP is a great tool when we want them conscious but they’re not strong enough yet to adequately ventilate, or have ventilation related respiratory fatigue. It has many other indications too, some use for recovery of brachycephalic patients.

6. Why do you do so many arterial blood gases - why not capnography?
We have capnography constantly monitored; however, it is not nearly as accurate as arterial blood, and it doesn’t tell us what the pH is. Together they are great.

7. Do you have many cats that just need an endotracheal tube and not ventilation, like with a laryngeal problem?
Yes.

8. Why do cats and dogs have an end-expiratory grunt?
Good question − not entirely sure. Several thoughts, not sure which is correct.

9. Have you not treated any cases where you actually see edema fluid come back out of the tracheal or the endotracheal tube?
No. I have seen many times with fulminating congestive heart failure, however not with any tick paralysis patients. Any fluid I have seen has been more of an exudate, related to aspiration pneumonia or saliva (usually aspirated) because they can salivate excessively and have a weak gag reflex and megaesophasus.

10. Do you think the tick anti-toxin works (TAS) − is there any evidence?
Yes. I do; I can’t imagine a double blind, controlled study being ethical. I’m not going to NOT treat them. My own dog had a tick a month ago above his eye and he had palpental paralysis for 3½ weeks! That’s without TAS because I was lazy and because I watched him closely (he didn’t develop any other signs). My point is, I haven’t seen paralysis last that long (especially without other signs). I assume if I gave him TAS, it would not have lasted that long − but who knows?

Comment courtesy of: Frank Gaschk evhs
Vetwell Veterinary Group, Brisbane QLD
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E. franko@grimminebear.com.au

First up, I’m a bog standard general practitioner with an interest in cats, who qualified in WA, so it’s hilarious to me to find myself talking about an east coast parasite and disease. But I do practice and practice in Brisbane, so it’s not so hilarious when I think about it. I’ve got no extra post-noms, I just read things that interest me or that I can use in general practice. I do love a cat conference and enjoy trying to communicate information through cartoons or a practical conversation. So, most definitely my knowledge always needs updating and improving, so I thank you for your excellent response. I do know how long things take to type, re-edit and type, so thank you also for taking the time.

I do hope we stir up some discussion with the readers of the CVE Control and Therapy Series.

I’m glad we follow a similar protocol; I caught that part. I love knowing what other vets do. Let’s then focus on the pulmonary oedema and frusemide.

The dose of frusemide is correct. I’m sure our colleagues will double check the most recent literature. I have used it in only a few cases where I thought I saw pulmonary oedema radiographically and interestingly enough those cats were also on carefully monitored IV fluids based on the earlier steps in the protocol. Quite a few of the cats are dehydrated purely because they haven’t been able to get up to have a drink. I don’t know if the frusemide in those X-rayed cases made any difference or if it made things worse; there is so much going on in the multi-modal approach to treating a tick paralysis case, it’s hard to tell. The poor critters are there, unable to resist our ministrations, and what vet is unable to resist a fully compliant feline patient? So we try to do everything and that may be a mistake in itself.

No cats died in the formulation of the protocol − at least I can say that. I know one cat I recently put to sleep with end stage renal disease after treating him for tick paralysis in 2002. I don’t think I used frusemide on him but I remember he almost died of breathing related problems during the tick paralysis treatment. I was only new to Brisbane at the time, so it stays in my memory. I do know I probably used too much ACP because that was the way the Queensland vets treated cats at the time. Thankfully we have moved on from that, even though we cycle through it every few years. That cat only comes to mind because of his recent death. I love an anecdote as an antidote to EBM.

The more severe tick paralysis cases I see these days are sent off for 24 hour care at various emergency centres, so I haven’t used frusemide in the last few seasons. Not in this particularly active tick season either.

Like you, I really didn’t use it that often at all. Its inclusion in the protocol mainly stems from continuing education talks. The continuing education talks were in heavily canine based case and biased toward canine therapeutics. There seemed to be no protocol for treating cats. So the questions and answers sessions winkled out the cat information into my notes, passed through a few cat practitioners’ brains in Brisbane, and then into the protocol that I set out for myself, that found its way into the X-rayed case I referred to above. I knott in terms of a talk at a local cat practice (2004) and morphed into a talk at a conference based on some retrospective studies... and eventually was published in the C&T Series.

You are correct in detecting where the foundation information comes from. It is a handover from question and answer sessions with Dr Rona Campbell and Prof Rick Atwell. It would be interesting to hear what Rick Atwell and Rona Campbell would have to say about the pulmonary oedema these days, its genesis and treatment.

Your preventative care instincts are spot on. Protect those kidsies! In the wrong situation, with the wrong practitioner, harm can be done, so if the frusemide has no therapeutic benefit, then it should be removed from the protocol. You’ve got me thinking of the stressed vet who just looks at a protocol and gives everything, may not even taking an X-ray.

I’d just make the observation that general vet practitioners, on X-raying a cat’s chest and seeing a pattern that suggests oedema, will have taken the usual half way to the frusemide before the X-ray is dry. So there may be a better way I could word it?

As not mentioning frusemide to GUI well schooled in its use; in cardiac disease/chronic (tick paralysis), combined with its potential risks in tick paralysis patients may prove equally deleterious to the cat.

It will be interesting to see this dialogue stimulate something beneficial for our patients.

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Figure 2. Boedis hypodisculus paralysis tick showing scale (Courtesy of Anne Fawcett)
IN APPLYING IT as a treatment when the animal comes in paralysed or if you can’t find a tick – the alcohol base is what kills the tick, but also drops the body temperature and probably scars animals (smell, noise, sticky, cold etc) who are already fearful.

The other ‘tick’ is to give the cats cyproheptadine (Paranex®) as a premed – 4 orally but may come as injection – as a big part of their anaesthesia is serotonin mediated rather than histamine mediated, according to Annette Lister who studied cat lungs specifically. It also calms them.

And their shock organ is the lung, not the liver like dogs. It’s all about cats...

Editor’s Comment

1. 2

Rollover to read Kim Kendall’s C&T No. 5147, Dec 2010, Issue 261 ‘Paralysis tick tricks’

Editor’s Note: We will be canvassing the views of a number of vets in Australia and publishing a ‘Round Table Discussion’ on the Treatment of Tick Paralysis in our upcoming December 2012 issue. Rob continues the discussion here.

Comment courtesy of:

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I have been a full time emergency vet in the Brisbane and Gold Coast region for the last 12 years. I have just completed an alternative training program in emergency and critical care and I hope to sit the fellowship examination of the Australian College of Veterinary Scientists next year. Our practices focus on referral management of the worst cases of tick paralysis and we use 5 intensive care ventilators between the 2 practices. My clinical and research interest is management of severe tick paralysis and the causes of respiratory failure.

Critical care of patients with tick paralysis

Until one year ago I would have completely agreed with Dr Karina Graham’s comment published above. We just did not recognise pulmonary oedema clinically in our patients (dogs or cats) with severe tick paralysis (TP). Last year however, we started performing histopathology on lungs of patients which died from TP, and I was shocked by both the number of patients which had lung disease, and the number where lung disease consisted of pulmonary congestion and oedema. The results of that study are submitted awaiting approval for publication.

Administration of tick anti serum (TAS), and removal of embedded ticks are the 2 most important aspects of treating tick paralysis, but severely affected patients ultimately die from respiratory failure, and this needs to be managed objectively to improve the chances of successful treatment.

The separation of TP and respiratory failure is a key concept in critical care of affected patients. The neuromuscular syndrome can result in multiple causes of respiratory failure, such that it becomes a guessing game if you just chuck different treatments into the patient hoping that something will work. We need to be looking for the reason behind the respiratory difficulty rather than trying to treat it as part of one disease syndrome.

Dr Rick Atwell’s research on large numbers of ‘regular’ tick paralysis patients did not show any really significant differences in survival of patients regardless of the way we perform the basic treatment strategy. He has looked extensively at how much tick anti serum we (veterinarians) administer, which route of administration we use, whether we pre-medicate the patient, and if we administer anxiolytic medication. He also looked at clipping, bathing, or spraying with frontline. None of these things really made much difference to actual survival, with the only really important thing being administration of ‘some’ TAS.

I advocate minimal intervention (apart from TAS and tick searches in mild cases of TP), but early, goal oriented measures to support respiration and sustain life when respiratory difficulty is noted.

In general, patients with worsening TP either fail to breathe sufficiently to support life because of advanced neuromuscular paralysis, or they become hypoaemic because of pulmonary parenchymal disease. If you keep an animal breathing long enough, they almost all eventually recover from tick paralysis. The use of clinical examination, arterial blood gas analysis, and thoracic radiographs will permit logical decision making regarding oxygen therapy and ventilation.

Respiratory Assessment

To recognise deterioration in a TP patient, frequent respiratory assessment is required. This involves physical examination and whichever diagnostic tools are available. The deteriorating patient will exhibit one of 3 types of breathing:-

- Hypoventilating patients have reduced respiratory rate and effort. Patients with respiratory rates below 20 and advanced tick paralysis are likely to be exhibiting severe enough hypoventilation to warrant treatment. Quantifying hypoventilation involves either capnography to measure end tidal CO₂ (ETCO₂), or blood gas analysis (preferably arterial, but venous is acceptable; it should be about only 5mmHg higher than PaCO₂). The critical limit we use for PaCO₂ (arterial or venous) is 60mmHg. Levels higher than this indicate severe hypoventilation and mechanical ventilation is required. Some animals will survive much higher PaCO₂ levels than 60mmHg, but this is unpredictable.

- Restrictive breathing patterns which involve increased rate and work of breathing are consistent with pulmonary parenchymal disease. TP patients are known to develop pulmonary oedema, and aspiration pneumonia. Hypoaemia often occurs, but the patient will work very hard to prevent this, and may respiratory arrest due to fatigue while the SPO₂ level is adequate. The level of hypoaemia can be quantified using arterial blood gas analysis or pulse oximetry. The critical limits for these measurements are: SPO₂ >90% and PaO₂>60mmHg. Management of hypoaemia and increased work of breathing involves oxygen supplementation initially. Mechanical ventilation is required should this not be effective in maintaining adequate oxygenation and reducing the work of breathing to a sustainable level.

- Upper airway obstruction occurs frequently in TP patients especially brachycephalic animals and those intubated for a prolonged period. The classic clinical signs of stridorous breathing and inspiratory dyspnoea may not be apparent if the patient is showing marked paralysis. Additional clinical signs include gagging, retching, regurgitation, and anxiety. The suspected upper airway obstruction can be confirmed the signs resolve after placement of an endotracheal tube. Management of upper airway obstruction involves maintaining an endotracheal tube under anaesthesia until the sign resolve, or placement of a tracheostomy tube.
What’s YOUR Diagnosis?

CAT No. 5253
Usrina Schlittke – DE Feline 2011 participant
Kleintierpraxis Hirschkuh (Vet Clinic)
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What’s YOUR Diagnosis?

Perspective 92
Part 2 – Gastrointestinal Endoscopy

Andrea Harvey
RCVS Recognised Specialist in Feline Medicine
European Veterinary Specialist in Internal Medicine
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Andrea Harvey graduated from University of Bristol Veterinary School, UK in 2000, and, after a couple of years in small animal practice, returned to Bristol Vet School to undertake a residency in feline medicine, funded by the Feline Advisory Bureau (FAB). She then remained at Bristol Vet School as FAB Clinician in Feline Medicine until 2010 and has since been doing a variety of work throughout Europe, contributing to numerous textbooks, and working closely with the International Society of Feline Medicine (ISFM). In 2006 she developed and monitored the ‘Friendly Clinic’ scheme. Andrea obtained the RCVS Diploma in Feline Medicine and European Diploma in Internal Medicine in 2005 and is a RCVS Recognised Specialist in Feline Medicine and European Veterinary Specialist in Internal Medicine. Andrea has wide interests in all aspects of feline medicine, and is passionate about both providing the best care for her patients, and helping to support other veterinarians to do the same.

Part 1 – Introduction & Respiratory Endoscopy
(June 2012, Issue 267)

Part 2. Gastrointestinal endoscopy

Equipment

For gastrointestinal endoscopy, a flexible endoscope is required with 4 way tip deflection (and ability to retroflex > 180˚ in one plane), and must have the mechanical functions of insufflation, irrigation and suction. These functions should always be checked prior to anaesthetising the cat. For cats, a single gastroscope can generally be used in all sized cats for oesophagogastroduodenoscopy, gastroduodenoscopy and colonoscopy. The larger the biopsy channel, inevitably the larger the biopsies that can be collected, and therefore the more likely they are to be of histological quality. The compromise, is that the larger the distal tip size, the more difficult pyloric intubation will be. The instrument channel should be at least 2mm in order to be able to obtain diagnostic biopsy samples. This is achievable with a 5-6mm distal tip (e.g. Olympus Lucera video-gastroscope GIP-X200 6.5mm insertion tube with 5mm distal tip and 2mm instrument channel, Olympus SlimGIF GIF-XIF180N 5.5mm outer diameter, 2mm biopsy channel, 1.1 working length), and this can be intubated into the pylorus relatively easily in small cats. A 1.7mm working length, 7.0mm outer diameter gastroscope is available with a 2.9mm biopsy channel (Storz, 60714PK) which will allow procurement of significantly larger biopsy samples. An experienced endoscopist will be able to intubate the pylorus of most larger cats with a 7.3mm distal tip, but an inexperienced endoscopist may struggle, and it may not be possible in smaller cats. The 1.4mm insert tube is quite long for cats, which can make manoeuvering more challenging as the insert tube outside the patient tends to loop.

The main forces required for feline gastrointestinal endoscopy are biopsy forceps. Grazing forceps and basket forceps are also useful, for removing gastric foreign bodies, although these are less commonly encountered in cats compared to dogs.

Basket forceps are also required for placing percutaneous endoscopically placed gastrostomy (PEG) tubes. There are many different types of biopsy forceps, varying in shape (oval, round, edge, smooth, serrated), fenestrated or unfenestrated, those with a central spike, and swing jaw forceps. The author prefers oval fenestrated forceps without a central spike. The fenestrated cups reduce crush artefact, and the oval cups tend to collect more tissue. The type of edge used depends on the toughness of the tissue being sampled. The author tends to mainly use smooth edged cups, but if the tissue is tough, these can slip off, in which case switching to serrated edged cups is advisable. When biopsying the intestine, swinging jaw forceps help turn the cups into the intestinal wall, but are not easy to use.

Prior to anaesthetising the patient, ancillary equipment should also be ready (protective clothing, mouth gag, biopsy forceps, formalin, paraffin, saline), and the equipment should be checked that it is fully functioning (including air pump, suction unit and valve, air/water valve, tip deflection, light source image, biopsy forceps, recording equipment).

Uses and indications

The most common reason for performing upper GI endoscopy in cats is to assess for oesophageal disease in cases of regurgitation or dysphagia (where megaesophagus has first been excluded on radiography), panting, swallowing, excessive salivation, and for balloon dilation of oesophageal strictures, and to assess for gastric and small intestinal disease in cases of chronic vomiting, unexplained weight loss, unexplained anorexia, haematemesis or melaena, chronic diarrhoea, ultrasonographically detected thickened folds of the stomach or small intestine, and for PEG tube placement/removal. Unless there is a specific oesophageal disorder (e.g. stricture) which may make advancing beyond the oesophagus unnecessary, gastros-astro-duodenoscopy should always be performed.

Colonoscopy is indicated for investigation of large intestinal diarrhoea, haematochezia, tenesmus, dyschezia, constipation and for investigation of a palpable rectal mass or stricture. Upper GI endoscopy should always be performed whenever colonoscopy is being performed, since lesions are rarely confined to the colon even in the presence of a large and relatively large intestinal mass.

GI endoscopy has the advantages of allowing examination of the mucosal surface of the GIT, being a much less invasive...
The cat should be positioned in left lateral recumbency for not become excessively hypothermic during the procedure. An ET tube should be placed. Cuffed ET tubes are usually advised in place, and IV fluids administered throughout the procedure. Intubation easier, and it may reduce the risk of vagally induced inappropriate, maintaining on halothane, isoflurane or sevoflurane, where non-mucosal disease is suspected, or disease is known to be beyond the reach of an endoscope, or where there is evidence of intraluminal disease (e.g. mesenteric lymphadenopathy, ascites, hepatic/pancreatic abnormalities), then surgical biopsy is indicated.

Patient preparation
For upper GI endoscopy, patient preparation simply requires withdrawal of food for at least 12 hours prior to the procedure, to ensure that the stomach is empty. If any barium contrast studies have been performed, this duration should be not be performed for at least 24 hours. For colonoscopy, withdrawal of food for 24 hours is required, in addition to ‘cleansing’ the large intestine. There are no specific guidelines, but the author’s preference is to administer 10-15mg/L of polyethylene glycol laxative (e.g. Kleanprep), via a naso-oesophageal tube (just placed for each administration and then removed in between), on 2-3 occasions within the period approximately 4-20hours prior to colonoscopy, and then to administer a sodium phosphate rectal enema (Microlax) 2-4 hours prior to the procedure.

Pre-medication prior to induction of anaesthesia is routine (e.g. ACP, buprenorphine combination). Atropine is not used routinely, although some advocate that it can make pyloric intubation easier, and it may reduce the risk of vagally induced bradycardia during the procedure. An IV catheter should be in place, and IV fluids administered throughout the procedure. Induction of anaesthesia is achieved using standard methods for the species. Usually appropriate, maintaining on halothane, isoflurane or sevoflurane with oxygen. Nitrous oxide should be used since insufflation of the stomach permits diffusion of nitrous oxide and causes gastric overdistension. Intravenous midazolam or diazepam can be useful if there is airway anxiety, but the catheter should not be advanced beyond this point. Air should always be suctioned from the viscus before withdrawing the endoscope.

Technique for upper GI endoscopy
Delay in intubating the pylorus, and insufflation of air, makes pyloric intubation more difficult, therefore it is ideal to only quickly visualise the pylorus and immediately withdraw the scope. It then be advanced along the greater curvature until the pyloric antrum is in view. Attention should be paid to keeping the insertion tube as straight as possible to assist in more accurate steering. The lens will often become obscured by blood, mucus and GI contents, and this requires flushing by depressing the air/water button. Any fluid pooling in a viscus requires suctioning to examine the underlying mucosa. Air should always be suctioned from the viscus before withdrawing the endoscope.

Technique for colonoscopy
Colonscopy is more straightforward to perform than upper GI endoscopy, provided that the patient has been adequately prepared. The preparation is essential, otherwise colonoscopy will be a waste of time if significant amounts of faecal material remain within the colon. The insertion tube should be lubricated with KY Jelly (baking care not to get lubricant over the lens), and an assistant is required to gently pinch the anus around the insertion tube to prevent air from escaping. Once inserted into the rectum, air should be insufflated until the mucosa of the descending colon is in view, before advancing further. The junctions between the descending and transverse colon, and transverse and ascending colon can be readily detected as obvious bends, prior to rising to the ileocecal junction, identified by the author into the caecum and the prominent raised appearance of the ileocecal sphincter. It is possible to pass biopsy forceps through the sphincter to biopsy the ileum using the same protocol. As the colon is explored, biopsies should be taken from all parts of the colon, prior to interrupting the air out and withdrawing the endoscope and examining the rectum on the way out.

The most common pathology observed in the feline oesophagus is oesophageal strictures or oesophageal foreign bodies are suspected, the cat should be kept in sternal recumbency with the head elevated in order to reduce risk of aspiration. For oesophageal biopsies, the cardia above, and the pyloric antrum below (Figure 6), and then slightly reducing the retroduction to bring the lesser curvature into view. Some insufflation is required to visualise these landmarks, but over insufflation will make pyloric intubation difficult, and this is the most common mistake made. In cats, the angle of the lesser curvature is quite acute, and a slide-by-technique can be useful for passing the endoscope into the antum. This involves gently advancing the endoscope along the mucosal surface of the greater curvature, the endoscope tip will be impinging on the gastric mucosa and so red-cut will occur, but provided this is moving and the endoscope is not advanced against any resistance, it can be continued to be advanced along the greater curvature until the pyloric antrum comes into view. Ensuring that the pylorus is in the centre of the screen, and suctioning as the insertion tube is advanced towards it, assists with pyloric intubation.

Once through the pylorus the insertion tube is advanced past the cardia (Figure 7) and then slightly reducing the refraction to bring the lesser curvature into view. The pyloric antrum may even be reached in cats. It is common for the mucosa to appear macroscopically normal even when significant microscopic disease is present. Multiple biopsies (approximately 6-10) should therefore always be taken from different regions of the small intestine (see below for biopsy technique). The most common small intestinal disorders in cats are inflammatory bowel disease (Figure 9) and intestinal lymphoma. Once the duodenum has been assessed and biopsies taken, all the air should be suctioned out as the endoscope is withdrawn. The proximal duodenal phase should be evaluated on the way out for lesions (e.g. ulcers) before withdrawing the endoscope back into the stomach for more complete evaluation. Retortion and rotation of the endoscope along its long axis allows full inspection of the cardia and the fundus. Multiple biopsies (total approximately 6-10) should be taken from all areas of the stomach, in addition to any macroscopic lesions. In cats, the most frequent macroscopic lesions identified are neoplastic disorders, most commonly gastric lymphoma. The mucosa is often pale, lumpy and friable, however this is very variable and there is no pathognomonic appearance (Figure 10).

The cat should be positioned in left lateral recumbency for routine GI endoscopy (upper and lower) (Figure 1), so that the gastric antrum is uppermost allowing air to fill it and make the pylorus more visible, and that the descending colon lies ventrally, while the sigmoid colon is cephalad. The most common pathology observed in the feline oesophagus is oesophageal strictures or oesophageal foreign bodies are suspected, the cat should be kept in two carnivores of the world: cats, dogs and other carnivores; immediate corticosteroid treatment can be started if indicated.

Limitations and contraindications
There are relatively few contraindications for gastrointestinal endoscopy. The main contraindication is, however, where systemic causes of clinical signs have not been excluded and there have been a large number of investigations performed prior to considering endoscopy. Other contraindications may be a priori anaesthetic risk, presence of a bleeding disorder, any patient that has had recent GI surgery, and where food has not been withheld, or the stomach has been histologically abnormal.

Whilst being a very valuable way of assessing the GI tract, there are limitations of endoscopy. Firstly, it does require the appropriate and fully functioning equipment together with a competent endoscopist, with good biopsy technique, to enable adequate evaluation of the GIT and collection of diagnostic quality biopsies. It is also not able to assess functional and motility disorders, histologically and non-histologically, of the GI tract, submucosal lesions or intraluminal lesions. When endoscopic biopsies have failed to produce a diagnosis, where endoscopic biopsies have failed to produce a diagnosis, where endoscopy. Other contraindications include patients that are a poor anaesthetic risk. In cats, the distal oesophagus has distinct circular folds and the lower oesophageal sphincter may be performed for at least 24 hours. For colonoscopy, withdrawal of food for 24 hours is required, in addition to ‘cleansing’ the large intestine. There are no specific guidelines, but the author’s preference is to administer 10-15mg/L of polyethylene glycol laxative (e.g. Kleanprep), via a naso-oesophageal tube (just placed for each administration and then removed in between), on 2-3 occasions within the period approximately 4-20hours prior to colonoscopy, and then to administer a sodium phosphate rectal enema (Microlax) 2-4 hours prior to the procedure.

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Biopsy technique

An assistant is required to operate the biopsy forceps and it is important that they are familiar with how to operate them before starting. Instruments must always be passed through the biopsy channel in a closed position and never forced against resistance. Squeezing too hard to close the cups can break the wire of the forceps so must be avoided. Care must be taken when passing instruments through the deflected tip of the endoscope, as forceful passage can easily damage the inner lining of the instrument channel. The instrument channel also serves as a suction channel, so suction will be much reduced when an instrument is within the channel, and if the instrument channel cap is open. When the scope has passed several intestinal flexures, the forceps can be difficult to open as the wire may be bent, and straightening the endoscope will help with this.

The quality of the biopsies obtained are determined mainly by the size of the forceps (dependent on the size of the scope), and the pressure exerted on the tissue by the operator. This is a big limitation in feline endoscopy as the patient size limits the size of endoscope, and therefore size of biopsy forceps that can be used. Therefore in cosmetic endoscopy with pressure grading, there is no room for poor operator technique. Exerting maximal pressure can be achieved by positioning the biopsy cups perpendicular to the tissue being sampled (Figure 9). Being able to do this effectively, and knowing how much pressure can safely be applied, comes with experience. Deflating the viscous before biopsy also helps increase the size of the sample by reducing stretching of the mucosa.

Once the biopsy has been collected the forceps are removed from the biopsy channel, the cups opened and immersed in 10% formalin releasing the tissue. The forceps must be rinsed in water before reintroducing into the endoscope. Alternatively, tissue samples can be placed on card or tissue cassettes prior to being placed in formalin.

Complications and aftercare

Serious complications associated with GI endoscopy are rare. The most common problem that care should be taken to avoid is gastric overinflation, which as well making the endoscopy more difficult, particularly pyloric intubation and biopsy collection, also leads to impairment of venous return, and can quickly result in severe cardiovascular and respiratory compromise. It is important to avoid overdistension, and to remember to remove air with suction prior to withdrawing the endoscope at the end of the procedure. Other potential complications include tachycardia—which may occur as a vagovagal reflex, usually in cats with severe GI disease, and is usually resolved with atropine—and gastrointestinal perforation which, when it occurs, is usually the result of severe GI disease, accompanied by forceful use of the endoscope without adequate visualisation, or poor biopsy technique. Occasionally with severe GI disease, perforation can occur just with overinflation. Emergency laparotomy is required if perforation is evident or pneumoperitoneum develops. Significant mucosal haemorrhage associated with biopsying is rare, and it is not necessary to routinely prescribe gut protectants following endoscopy. Most cases of routine endoscopy require no specific aftercare.

Figure 2 – The endoscopic appearance of the normal feline oesophagus (courtesy of University of Bristol)

Figure 3 – Endoscopic view of an oesophageal stricture (courtesy of University of Bristol)

Figure 4 – A balloon catheter is being advanced through the lumen of the oesophageal stricture to allow balloon dilation (courtesy of University of Bristol)

Figure 5 – The normal endoscopic appearance of the area at the junction of the body/fundus of the stomach on the greater curvature. Note that the parallel rugal folds are running towards the pyloric antrum beneath the angularis incisura (courtesy of University of Bristol)

Figure 6 – Endoscopic view of the angularis incisura with the fundus/cardia dorsally and the antrum ventrally (courtesy of University of Bristol)

Figure 7 – Here the gastroscope is being retroflexed to examine the cardia (courtesy of University of Bristol)

Figure 8 – Endoscopic appearance of the normal feline duodenum (courtesy of University of Bristol)

Figure 9 – Duodenum from a cat with inflammatory bowel disease, illustrating biopsy technique, positioning the biopsy cups perpendicular to the mucosa (courtesy of University of Bristol)

Figure 10 – Endoscopic view of the stomach of a cat with gastric lymphoma. Note the very pale and “lumpy” appearance (courtesy of University of Bristol)
What’s YOUR differential diagnosis and diagnostic plan?

C&T No. 5255
Donna Peckett – DE Feline 2012 participant
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Donna is a CVE Feline DE alumnus who generously contributed this case to the DE Feline forum.
We invite C&T Members/Readers to formulate a systematic reply to this case presentation. There is an ‘ Isiscript’ in this case i.e. a pattern strongly suggestive of a particular diagnosis. We would like you, the reader, to submit a differential diagnosis and a diagnostic plan based on the information below provided by Donna. The winner of the best answer will win a CVE publication. (Go to www.vetbookshop.com to view our titles.)

Figure 1. Nose before cleaning

Figure 2. Nose

Figure 3. Toe 2

Figure 4. Toe 3

Figure 5. After cleaning toe

Figure 6. Toe showing ulceration

‘Marley’ is a 1-year-old desexed and fully vaccinated DH5 who had access to outdoors. His owner noticed some swelling and crusting of 3 toes (at different feet) and reported that Marley was licking his toes but was not lame and was otherwise well i.e. eating well etc.

A physical exam was unremarkable and aside from the toes I noted a demarcation of the skin on his nose (see Figure 2) which I felt was soon going to ulcerate. On clipping and cleaning the affected toes there was severe swelling and ulceration and we removed lots of thick inspissated pus from the crevices of the nail fold. A swab was sent for culture and a smear for cytology at the cat was commenced on doxycycline.

My concern was that it may have been an immune mediated condition with involvement of the nose and likely the next step would be biopsy and retroviral testing.

Replies or Comments
Please send your Replies or Comments to Lisa Churchward, Editor at elisabeth.churchward@sydney.edu.au for publication in the following issue.

A cat with jaundice

C&T No. 5255
Nikki Lough – DE Feline 2012 participant
‘Pets ‘n’ Vets’
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Signalment: A 2½-year-old male neutered Turkish Angora.

History: ‘Mac’ presented with a 3-4 week history of lethargy and dullness at home. He had a poor appetite but was still drinking, and the owners felt that he had lost weight. He was an indoor cat so had no access to toxins, was up to date with his vaccinations (FHV, FCV, FPLV), and had recently been wormed.

Physical Exam: Mac was quiet, alert and responsive on exam. The weight loss was confirmed, with a decrease from 4.74kg to 4.17kg, and he currently had a BCS score of 2/5. Profound jaundice was evident around Mac’s ears, sciera and mucous membranes. No other abnormalities were discovered in the rest of the physical exam.

Problem List and Differentials: Jaundice, weight loss, poor appetite, lethargy. The primary problem was considered to be the cause of the jaundice with the other problems secondary to this.

Differentials for jaundice included: pre-hepatic causes – haemolytic anaemia, hyperbilirubinaemia; hepatic causes – inflammatory hepatopathies, hepatic lipidosis, FIP, sepsis, neoplasia, hepatotoxicity, toxicplasma, amyloidosis, telangiectasis; post-hepatic causes – bile duct obstruction (pancreatitis, cholecystitis, cholelithiasis, hepatobiliary mass, duodenal mass) or bile duct/gall bladder trauma.

The history and age of the cat made hyperthyroidism and neoplasia unlikely. The owners were sure there had been no access to toxins although hepatotoxicity could not be ruled out at that stage. There was no evidence of intra-abdominal haemorrhage, thus making amyloidosis and telangiectasis less likely. There was no history of abdominal trauma as he hadn’t escaped from the owners’ house.

The most likely differentials were likely to be haemolytic anaemia, inflammatory hepatopathy (acute or chronic neutrophilic, or lymphocytic cholangitis), hepatic lipidosis, pancreatitis, cholecystitis or cholelithiasis. The nonneutrophic form of FIP was also possible, although he didn’t appear to have any ocular or CNS signs.

Investigation: Bloodwork was initiated, including a full biochemistry profile to evaluate the severity of bilirubinemia and his liver enzymes, and also to get a general picture of his health status. Haematology was carried out to assess any anaemia present, and liver enzymes, as well as to get a general picture of his health status.

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Investigation: Bloodwork was initiated, including a full biochemistry profile to evaluate the severity of bilirubinemia and his liver enzymes, and also to get a general picture of his health status. Haematology was carried out to assess any anaemia present, and electrolytes were checked in view of his poor appetite.

Mac's electrolytes were within normal limits and his biochemistry revealed markedly raised bilirubin and elevated liver enzymes (ALT much more than ALKP). His urea was at the very low end of the spectrum, but still within normal limits. The haematology showed mild anaemia with a lowered haematocrit and mature blood cell count. The lab results for Mac are as follows:

<table>
<thead>
<tr>
<th>TEST</th>
<th>RESULT</th>
<th>RANGE</th>
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<tbody>
<tr>
<td>ALKP</td>
<td>127</td>
<td>14-111 U/L</td>
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<tr>
<td>ALB</td>
<td>29</td>
<td>22-44 g/L</td>
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<tr>
<td>Ca</td>
<td>2.52</td>
<td>1.95-2.83 mmol/L</td>
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<tr>
<td>CR</td>
<td>127</td>
<td>71-212 umol/L</td>
</tr>
<tr>
<td>CRE</td>
<td>4.3</td>
<td>1.6-6.4 mmol/L</td>
</tr>
<tr>
<td>CHOL</td>
<td>4.3</td>
<td>1.6-6.4 mmol/L</td>
</tr>
<tr>
<td>GLOB</td>
<td>38</td>
<td>28-51 mmol/L</td>
</tr>
<tr>
<td>CREA</td>
<td>127</td>
<td>71-212 umol/L</td>
</tr>
<tr>
<td>GLU</td>
<td>5.04</td>
<td>4.1-8.3 mmol/L</td>
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<tr>
<td>AMYL</td>
<td>1048</td>
<td>500-1500 U/L</td>
</tr>
<tr>
<td>ALT</td>
<td>374</td>
<td>12-130 U/L</td>
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<tr>
<td>TP</td>
<td>67</td>
<td>57-89 g/L</td>
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<tr>
<td>TBIL</td>
<td>174</td>
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<tr>
<td>PHOS</td>
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<td>1.2-4.2 mmol/L</td>
</tr>
<tr>
<td>INR</td>
<td>0.89</td>
<td>0.6-1.6</td>
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<td></td>
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<tr>
<td>MONO</td>
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<tr>
<td>EOS</td>
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<td>0.0-0.7 x109/L</td>
</tr>
<tr>
<td>BASO</td>
<td>0.0</td>
<td>0.0-0.1 x109/L</td>
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<tr>
<td>D DIMER</td>
<td>2.94</td>
<td>0.4-8.6 x107/L</td>
</tr>
<tr>
<td>FLT</td>
<td>445</td>
<td>175-600 KU/L</td>
</tr>
</tbody>
</table>

Mac’s electrolytes were within normal limits and his biochemistry revealed markedly raised bilirubin and elevated liver enzymes (ALT much more than ALKP). His urea was at the very low end of the spectrum, but still within normal limits. The haematology showed mild anaemia with a lowered haematocrit and red blood cell count slightly below the normal range. Neutropenia was also present. The mild anaemia would not be enough to explain the markedly raised bilirubin so is more likely to be anaemia of a chronic disease and so secondary to the main problem. The high liver enzymes suggested a hepatic or post-hepatic cause to Mac’s problems. The neutropenia could have been due to viral infection, autoimmune destruction, or excessive tissue demand, and suggested possible FP or retrovirus infection, although his globulin level is still normal.

Management
At this point, we would proceed to diagnostic imaging – specifically ultrasound – to check for biliary obstruction and also to evaluate the liver and pancreas. I would also like to send off a specific test to check for pancreatitis in case the ultrasound results were inconclusive. I would admit Mac for supportive treatment in the form of fluids and analgesia.

However, in Mac’s case it was decided to investigate the anaemia in more detail so bloods were taken and sent to the lab for a Coombs test and also a POC for Mycoplasma haemofelis. Mac was booked in for a scan and sent home.
When Mac returned to the surgery, the owners reported that he was still the same. A scan of his liver revealed a slightly thickened bile duct. The owners were concerned at this time, although there is no note of whether it couldn’t be seen or just wasn’t looked for. The blood results returned from the lab with a normal red and white cell count, haematocrit, a mild neutropenia, and negative results for the Coombs test and M. haemofelis.

Assessment
At this stage, bile duct obstruction was ruled out as was haemolytic anaemia, the most common hepatic lipodosis, hepatic lipidosis and pancreatitis. The retroviruses and FIP could not be excluded at this stage either.

Management
After discussion with the owners, it was decided to proceed with an exploratory laparotomy and liver biopsies. A FeLV/FIV snap test was carried out prior to the surgery, and the results were negative. Mac was anaesthetised and operated on under the surgery and also given Amoxicillin/Olaclavunate (Augmentin, 37.5mg PO). The liver was reported at the time to look quite mottled in appearance with greyish green and darkened areas throughout. No evidence of the granulomas typical of non-feline FIP was found. Biopsies of the liver and pancreas were taken and it was mentioned that the mesenteric lymph nodes were enlarged. The intestines were examined grossly but no biopsies were taken although the reason for this is unclear. The risks of gut wall dehiscence appears low in the literature, and the variability of the gross tissue appearance with infiltrative gut disease is marked, leading to the opinion that gut biopsies should be taken almost every time endoscopy or exploratory laparotomy is performed. With the potential risk of Mac having traumatic, it makes more sense to take all the potential samples required while the surgery is being undertaken, rather than having further surgery later to collect anything more.

A nasochoanal (NO) tube was placed to aid Mac’s nutrition over the next few days once his abdominal wound was closed. Mac developed respiratory arrest while recovering from the anaesthetic and became cyanotic. He was renaledicated and IPPV was carried out, where convulsion and spontaneous breathing began again.

Mac was given marbofloxin (Marbocyl®, 8.2mg SC), buprenorphine (Vetergesic®, 0.09mg SC), cefalexin (Convenia®, 32mg SC) and also clindamycin (Antirobe®, 25mg bid by mouth). Despite this, the owners reported that he was becoming more aggressive towards them at home, to the extent that they were encouraging the owners was his increasing aggression towards them at home, to the extent that they were asked to stop all his medications.

Two months later, the owners reported that Mac had deteriorated and prednisolone therapy was recommended at 2.5mg bid by mouth. Despite this, the owners reported that he was becoming more aggressive with them at home, to the extent that they were asked to persevere if possible but that the prednisolone and UDCA were of higher importance.

Follow-Up: One month later, Mac appeared to be doing very well. His weight had increased to 4.3kg and he was no longer jaundiced. He was bright and eating well at home but he did seem to be more bad tempered. He was unable to be examined conscious in the surgery and so was sedated to repeat his biopsies. Physical exam was normal at this time.

Mac’s blood results were much improved, with resolution of the anaemia and neutropenia and only slight elevation of the ALT in the biochemistry panel. On the basis of this, it was decided to wean the dose of prednisolone down over the next 2 weeks and then to stop all his medications.

Two months later, the owners reported that Mac had deteriorated and prednisolone therapy was recommended at 2.5mg bid by mouth. Despite this, the owners reported that he was becoming more aggressive with them at home, to the extent that they requested euthanasia 2 weeks later.

Discussion: Lymphocytic cholangitis is recognised histologically by an infiltration of lymphocytes into the portal tracts, and is thought to have an immune-mediated aetiology. It is usually a chronic condition which develops slowly and cats may have had vague symptoms for weeks to months prior to presentation. Younger cats tend to be more affected by this condition as they may develop with concurrent pancreatitis and/or inflammatory bowel disease. Pancreatitis was ruled out with Mac although BUD could not be definitely excluded due to the lack of biopsies. Lymphocytes may not diagnose all conditions – e.g. if an unaffected portion of tissue is sampled – although Mac was not showing other symptoms and the response to his treatment suggests that these other conditions were not features in this case.

The treatment for this condition is considered to be steroids, supported by a wealth of clinical experience. Some reports claim that they have found no benefit from using steroids however and would use UDCA as the first choice of treatment. The prognosis for cats with lymphocytic cholangitis is variable, as responses to treatment can be difficult to monitor. A mean survival time of 3 years has been mentioned. The outcome of this case was very disappointing, and it would have been worthwhile to try and investigate Mac’s condition to prove it was the condition of the risk factor that was more likely to be infected. There was no note of aggression in the literature as an effect of the condition and some cats can be less likely to manage after being hospitalised and undergoing various procedures in the surgery. The problem for the owners was his increasing aggression towards them at home, and to not investigate this or suggest a behaviour therapy as a follow up though we have failed to manage this in the best way for the cat and the owners.

Feline Q&A: What dose of tramadol do you suggest in cats?

CAT No. 5256
Quentin Brown
North Nowra Veterinary Hospital
205 Illaroo Rd North Nowra, NSW 2541
T. (02) 4423 1691
F. (02) 4423 1800
E. admin@northnowravet.com.au
T. (02) 4423 1800
F. (02) 4423 1691
E. richardcatdoctor@gmail.com

I wouldn’t expect the cat’s pain to come under control in the short term as tramadol is a chronic degenerative process. It can take weeks to months to control pain adequately in some patients. Some are just poor responders. Part of the lack of response may be the chronicity of the disease and the neurobiological changes that have occurred, as well as degeneration to muscles, nerves and tendons etc. Some cats feel better on low doses of Metacam® (+/- 0.02mg/kg SID PO), but they don’t show it in marked changes to mobility for quite a while longer. Try low dose PO Tencges® SID (0.01-0.02mg/kg) as an adjunctive or so to the Metacam®. This can be used in conjunction with other treatments and cats have had viable clinical signs for months prior to presentation. Younger cats tend to be more affected by this condition as they may develop with concurrent pancreatitis and/or inflammatory bowel disease. Pancreatitis was ruled out with Mac although BUD could not be definitely excluded due to the lack of biopsies. Cats may not diagnose all conditions – e.g. if an unaffected portion of tissue is sampled – although Mac was not showing other symptoms and the response to his treatment suggests that these other conditions were not features in this case.
Treatment of Diabetes Mellitus in 2 cats

C&T No. 5257

Tracey Tonkin – DE Feline 2011 participant
Alice Springs Veterinary Hospital
73 Bath Street
Alice Springs NT 0871
T. 08 8952 9899
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View Tracey’s excellent powerpoint: How to manage your diabetic cat. (Also available at www.cve.edu.au/CandT/resources)

Introduction:

Two cats were presented to the Alice Springs Veterinary Clinic at similar times, both diagnosed with Diabetes Mellitus. The treatment of these 2 cases was approached in a similar fashion, in an attempt to establish a set protocol for treating diabetic cats with Glargine insulin within the clinic. A set protocol agreed upon by all vets at the clinic would allow consistent management of all feline diabetic cases presented to the clinic in the future.

Case Histories:

‘Barkey’ – a 16-year-old male neutered domestic shorthair was initially presented for mild weight loss. There had been no change in appetite or water intake noticed by the owner at this initial presentation. Physically, Barkey appeared normal except for a slight systolic murmur and periodontal disease. One month later, Barkey developed vomiting and diarrhoea and was now no longer eating and appeared dull and lethargic. On physical examination he was found to be dehydrated, pyrexic and had lost a further 500g in the past 4 weeks.

‘Denis’ – 11-year-old male neutered domestic longhair, was diagnosed as being diabetic 12 months prior to this episode. After his original diagnosis, the owner elected not to treat Dennis, but would not euthanase either. When he presented to the clinic in the future.

Although one cat had already been diagnosed as diabetic, the following differential diagnosis list could have been considered in both cases:

Metabolic:
- Diabetes Mellitus
- Hyperthyroidism
- Renal Disease

Mechanical:
- Gastrointestinal foreign body
- Intussusception

Results for Dennis – In-house Biochemistry

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
<th>Ref</th>
</tr>
</thead>
<tbody>
<tr>
<td>ALB</td>
<td>34 g/L</td>
<td>(23–39)</td>
</tr>
<tr>
<td>ALKP</td>
<td>45 U/L</td>
<td>(14–111)</td>
</tr>
<tr>
<td>ALT</td>
<td>186 U/L</td>
<td>(12–130)</td>
</tr>
<tr>
<td>AMY</td>
<td>1011 U/L</td>
<td>(200–1500)</td>
</tr>
<tr>
<td>Ca</td>
<td>2.75 mmol/L</td>
<td>(1.95–2.83)</td>
</tr>
<tr>
<td>CHOL</td>
<td>8.48 mmol/L</td>
<td>(1.68–5.81)</td>
</tr>
<tr>
<td>CREA</td>
<td>320 mmol/L</td>
<td>(71–212)</td>
</tr>
<tr>
<td>GLOB</td>
<td>51 g/L</td>
<td>(28–51)</td>
</tr>
<tr>
<td>GLUCOSE</td>
<td>&gt;88.11 mmol/L</td>
<td>(3.94–8.83)</td>
</tr>
<tr>
<td>PHOS</td>
<td>2.87 mmol/L</td>
<td>(1.00–2.24)</td>
</tr>
<tr>
<td>TBIL</td>
<td>9 umol/L</td>
<td>(10–15)</td>
</tr>
<tr>
<td>TP</td>
<td>84 g/L</td>
<td>(57–89)</td>
</tr>
<tr>
<td>Na+</td>
<td>151 mmol/L</td>
<td>(150–160)</td>
</tr>
<tr>
<td>K+</td>
<td>3.9 mmol/L</td>
<td>(3.5–5.8)</td>
</tr>
<tr>
<td>Cl-</td>
<td>111 mmol/L</td>
<td>(112–129)</td>
</tr>
</tbody>
</table>

Results for Barkley – In-house Biochemistry

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
<th>Ref</th>
</tr>
</thead>
<tbody>
<tr>
<td>CK</td>
<td>50–200</td>
<td>U/L</td>
</tr>
<tr>
<td>AST</td>
<td>26–43</td>
<td>U/L</td>
</tr>
<tr>
<td>ALT</td>
<td>6–83</td>
<td>U/L</td>
</tr>
<tr>
<td>ALK</td>
<td>25–93</td>
<td>U/L</td>
</tr>
<tr>
<td>GGT</td>
<td>1–5</td>
<td>U/L</td>
</tr>
<tr>
<td>GLD4</td>
<td>-</td>
<td>U/L</td>
</tr>
<tr>
<td>Bilirubin</td>
<td>&lt; 2</td>
<td>umol/L</td>
</tr>
<tr>
<td>Urea</td>
<td>18–71.7</td>
<td>mmol/L</td>
</tr>
<tr>
<td>Creat</td>
<td>109–159</td>
<td>mmol/L</td>
</tr>
<tr>
<td>Lactate</td>
<td>-</td>
<td>mmol/L</td>
</tr>
<tr>
<td>pH</td>
<td>6.0</td>
<td>-</td>
</tr>
<tr>
<td>Protein</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Urobilinogen</td>
<td>Negative</td>
<td></td>
</tr>
<tr>
<td>USG</td>
<td>1.018</td>
<td>-</td>
</tr>
<tr>
<td>T4</td>
<td>10.9 mmol/L</td>
<td>(15–43)</td>
</tr>
</tbody>
</table>

Biochemistry

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
<th>Ref</th>
<th>Test</th>
<th>Result</th>
<th>Ref</th>
</tr>
</thead>
<tbody>
<tr>
<td>CK</td>
<td>192</td>
<td>U/L</td>
<td>Lipase</td>
<td>3–105</td>
<td>S(Ble 1)</td>
</tr>
<tr>
<td>AST</td>
<td>36</td>
<td>U/L</td>
<td>Amylase</td>
<td>-</td>
<td>S(Ble 2)</td>
</tr>
<tr>
<td>ALT</td>
<td>72</td>
<td>U/L</td>
<td>Glucose</td>
<td>m.mol/L</td>
<td>26.7</td>
</tr>
<tr>
<td>ALK</td>
<td>68</td>
<td>U/L</td>
<td>m.mol/L</td>
<td>0.42</td>
<td>0.00–0.05</td>
</tr>
<tr>
<td>GGT</td>
<td>&lt; 1</td>
<td>U/L</td>
<td>Chol</td>
<td>m.mol/L</td>
<td>7.5</td>
</tr>
<tr>
<td>GLD4</td>
<td>-</td>
<td>U/L</td>
<td>Trig.</td>
<td>m.mol/L</td>
<td>-</td>
</tr>
<tr>
<td>Bilirubin</td>
<td>&lt; 2</td>
<td>umol/L</td>
<td>Na</td>
<td>m.mol/L</td>
<td>145</td>
</tr>
<tr>
<td>Urea</td>
<td>18–71.7</td>
<td>mmol/L</td>
<td>K</td>
<td>m.mol/L</td>
<td>4.2</td>
</tr>
<tr>
<td>Creat</td>
<td>109–159</td>
<td>mmol/L</td>
<td>Ca</td>
<td>m.mol/L</td>
<td>2.56</td>
</tr>
<tr>
<td>Lactate</td>
<td>-</td>
<td>mmol/L</td>
<td>Hb</td>
<td>g/L</td>
<td>110</td>
</tr>
<tr>
<td>pH</td>
<td>6.0</td>
<td>-</td>
<td>WBC</td>
<td>x 10⁹/L</td>
<td>10.0</td>
</tr>
<tr>
<td>Protein</td>
<td>-</td>
<td>-</td>
<td>Neut</td>
<td>%</td>
<td>95</td>
</tr>
<tr>
<td>Urobilinogen</td>
<td>Negative</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>USG</td>
<td>1.018</td>
<td>-</td>
<td>Eosin</td>
<td>%</td>
<td>4</td>
</tr>
<tr>
<td>T4</td>
<td>10.9 mmol/L</td>
<td>(15–43)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Haematology

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
<th>Ref</th>
<th>Test</th>
<th>Result</th>
<th>Ref</th>
</tr>
</thead>
<tbody>
<tr>
<td>HB</td>
<td>g/L</td>
<td>110</td>
<td>WBC</td>
<td>x 10⁹/L</td>
<td>80–150</td>
</tr>
<tr>
<td>HCT</td>
<td>g/L</td>
<td>0.36</td>
<td>Neut</td>
<td>%</td>
<td>69</td>
</tr>
<tr>
<td>PCV</td>
<td>U/L</td>
<td>0.24–0.45</td>
<td>Bands</td>
<td>%</td>
<td>0</td>
</tr>
<tr>
<td>RBC</td>
<td>x 10¹²</td>
<td>6.6</td>
<td>Lymphs</td>
<td>%</td>
<td>21</td>
</tr>
<tr>
<td>MOH</td>
<td>g/l</td>
<td>308</td>
<td>Mono</td>
<td>%</td>
<td>6</td>
</tr>
<tr>
<td>MOH</td>
<td>pg</td>
<td>17</td>
<td>Eosin</td>
<td>%</td>
<td>4</td>
</tr>
<tr>
<td>MCV</td>
<td>L/L</td>
<td>39.0–55.0</td>
<td>Basophil</td>
<td>%</td>
<td>0</td>
</tr>
<tr>
<td>Retic (Cor)</td>
<td>%</td>
<td>Other</td>
<td>%</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>T.S Protein</td>
<td>g/L</td>
<td>60–80</td>
<td>Retic/ABS</td>
<td>%</td>
<td>10</td>
</tr>
<tr>
<td>Nuc RBC</td>
<td>/100 WBCO</td>
<td>1000</td>
<td>Fibrinogen</td>
<td>g/L</td>
<td>1.4</td>
</tr>
<tr>
<td>ESR 20mm</td>
<td>mm</td>
<td>300–700</td>
<td>Platelets</td>
<td>x 10⁹/L</td>
<td>10</td>
</tr>
</tbody>
</table>

Results for Barkley

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
<th>Ref</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood Glucose</td>
<td>21.2 mmol/L</td>
<td></td>
</tr>
<tr>
<td>Total T4</td>
<td>25.7 mmol/L</td>
<td>(15–43)</td>
</tr>
</tbody>
</table>

Urinalysis

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
<th>Ref</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Bilirubin</td>
<td>Negative</td>
<td></td>
</tr>
<tr>
<td>Ketones</td>
<td>Negative</td>
<td></td>
</tr>
<tr>
<td>Blood</td>
<td>Negative</td>
<td></td>
</tr>
<tr>
<td>Protein</td>
<td>Negative</td>
<td></td>
</tr>
<tr>
<td>Urobilinogen</td>
<td>Negative</td>
<td></td>
</tr>
<tr>
<td>USG</td>
<td>1.000</td>
<td>-</td>
</tr>
</tbody>
</table>

Barkey was diagnosed as suffering from Diabetes Mellitus based on the presence of hyperglycaemia and glucosuria. He was not ketoacidotic or ketonuric however, and was classified as being in a stable state. Renal disease and hyperthyroidism were ruled out. The source of the pyrexia was not found, however he was treated with a course of amoxycillin/clavulanic acid and the pyrexia resolved within a few days. It is possible that this may have been related to his periodontal disease, which was quite severe. •

Markers:
- Diabetes Mellitus
- Hyperthyroidism
- Renal Disease

Mechanical:
- Gastrointestinal foreign body
- Intussusception

Neoplastic:
- Gastric or small intestinal neoplasia

Inflammatory/Infectious:
- Acute pancreatitis
- Gastritis/Gastric ulcer
- Septicaemia/bacteriemia
- Panleucopaenia

Toxic:
- Lily ingestion/plant toxicity

Investigations: Blood was taken for biochemistry and haematology to be done at an external laboratory in Dennis’s case, and was suggested in Barkey’s case, but not accepted by the owner. An in-house glucometer reading immediately confirmed that Barkey was diabetic. As Dennis was quite unwell at time of presentation, in-house biochemistry was run immediately, as results from the external laboratory would take 24 hours to return. Urine was collected in both cases to check USG and the presence of ketonuria, and a T4 level was run to rule out the presence of hyperthyroidism.

Barkley was diagnosed as suffering from Diabetes Mellitus based on the presence of hyperglycaemia and glucosuria. He was not ketoacidotic or ketonuric however, and was classified as being in a stable state. Renal disease and hyperthyroidism were ruled out. The source of the pyrexia was not found, however he was treated with a course of amoxycillin/clavulanic acid and the pyrexia resolved within a few days. It is possible that this may have been related to his periodontal disease, which was quite severe.
Dennis presented in a more deteriorated state than Barkley with marginal ketonaemia, although he was not ketonuric at this point either. Whilst Barkley did not require initial stabilisation in hospital, Dennis did, and he received intravenous fluid therapy to correct his hyperhydration status, and was started on Glargine in hospital at 0.5IU/kg. Twelve hourly insulin doses, blood glucose curves and serial electrolyte measurements were carried out in hospital and supportive treatment (including potassium supplementation and intravenous fluid therapy) was provided.

Once stabilised, Dennis was discharged and management of his diabetes by his owner began. Barkley was also started on home management around the same time.

**Discussion:** These cases presented the first opportunity for vets at the Alice Springs Veterinary Clinic to treat a diabetic cat with Glargine insulin. Previously, Caninsulin® was always used in feline diabetic patients in our clinic. Glargine is a long-acting insulin analogue, which can be administered once or twice daily. The advantage of such an insulin over others is that it provides excellent duration of action in cats with inadequate duration of action using intermediate-acting insulins1. This extended duration of action means better glycemic control and therefore higher remission rates than lente, NPH or ultralente insulins2. The dose required for most cats is often only 1–4 IU, and it can be difficult to administer less than 2 IU accurately, even with a 0.3ml insulin syringe3. However, expected duration of action is more important than concentration when it comes to achieving good glycemic control4.

Barkley was started on a dose of 2.5IU of Glargine (0.5IU/kg) BID, and changed to a low carbohydrate, high protein diet (Hils Prescription Diet m/d®). Dennis was started on an initial dose of 0.5IU/kg, however the owner could not be convinced to place Dennis on a prescription diet, and continued to feed him commercial cat food and proteins. Evidence suggests that a high protein, low carbohydrate diet (6–12% of calories from carbohydrates) is best for diabetic cats, because it lowers blood glucose and insulin requirements, and may increase the diabetic remission rates5.

In order to establish a set protocol for treating all diabetic cats in the future with Glargine, the following treatment plan was established for both cats.

Rather than performing glucose curves in hospital, which proved to be a very stressful environment for both cats, pre-insulin glucose blood readings were to be taken by the owners at home, and the dose of Glargine was to be altered depending upon this pre-insulin blood glucose reading, according to the following schedule:

<table>
<thead>
<tr>
<th>Blood Glucose (mmol/L)</th>
<th>Change to Glargine Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 18</td>
<td>Increase by 0.5 IU (if current dose is &lt; 20IU)</td>
</tr>
<tr>
<td></td>
<td>Increase by 1.0 IU (if current dose is &gt; 20IU)</td>
</tr>
<tr>
<td>10 – 18</td>
<td>Increase by 0.5 IU</td>
</tr>
<tr>
<td>6 – 10</td>
<td>Leave insulin dose the same</td>
</tr>
<tr>
<td>3 – 6</td>
<td>Decrease by 0.5 IU</td>
</tr>
<tr>
<td>&lt; 3</td>
<td>Give no insulin AND decrease the next dose by 1.0 IU</td>
</tr>
</tbody>
</table>

The dose of insulin was kept at 2.5IU BID and Barkley’s pre-insulin blood glucose readings continue to remain between 4 and 10 mmol/L. Uncontrolled urine was checked by the owner weekly, and she kept a daily diary of water intake as well as the blood glucose readings. Currently Barkley is clinically well, with a steady insulin dose and good glycaemic control. Although he has not yet achieved remission, this is still a possibility. Remission is likely to occur if the nadir glucose is in the normal range, and the pre-insulin blood glucose is less than 12 mmol/L.

Day 14 showed a blood glucose level which was quite low; however, it was noted by the owner that Barkley did not eat his food as usual on this day, which may explain this particularly low reading. Following this he returned to eating normally the next day.

Dennis’s blood glucose readings also stabilised after keeping the Glargine dose steady (full data not recorded on permanent record however). It was adjusted 2–3 times, and although did not reach the same level of stability that Barkley’s did, the hypoglycaemic episodes stopped, and clinically he remained well.

**Conclusion:** Tracking the progress of these cases allowed a good review of the protocol for treating diabetic cats with Glargine insulin in our clinic. A treatment protocol was established to allow consistent management of diabetic cases between the 9 vets employed at the clinic. The protocol now used in our clinic is outlined in the following box:

- After initial stabilisation in hospital (i.e. cat no longer ketonuric or ketoadidotic, eating well and hydration and electrolytes normal), cat should be placed on an initial dose of Glargine of 0.5 IU/kg SC if blood glucose is >20 mmol/L, or 0.25 IU/kg SC if blood glucose is between 12 – 19 mmol/L.
- Owners are allowed to take pre-insulin blood glucose readings daily before the morning dose of insulin. The dose of Glargine should remain constant during this time.
- Owners also must keep a diary of water intake, and measure urine glucose at home using urine glucose dipsticks.
- Pre-insulin blood glucose readings to be reviewed by vet each week, and changes to insulin dose to be decided weekly based on pre-insulin readings.
- To change the dose of Glargine should be made according to the following schedule:

<table>
<thead>
<tr>
<th>Blood Glucose (mmol/L)</th>
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</tr>
</thead>
<tbody>
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</tr>
<tr>
<td></td>
<td>Increase by 1.0 IU (if current dose is &gt; 20IU)</td>
</tr>
<tr>
<td>10 – 18</td>
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</tr>
<tr>
<td>6 – 10</td>
<td>Leave insulin dose the same</td>
</tr>
<tr>
<td>3 – 6</td>
<td>Decrease by 0.5 IU</td>
</tr>
<tr>
<td>&lt; 3</td>
<td>Give no insulin AND decrease the next dose by 1.0 IU</td>
</tr>
</tbody>
</table>

- Cats should be placed on a low carbohydrate, high protein diet (preferably Hils Prescription diet m/d® or Royal Canin Feline Diabetic Formula®). Cats should be allowed free access to wet or dry food. Feeding does not need to be coordinated with insulin doses, as the post-prandial increase in blood glucose is very prolonged in cats (18 hours or more)6.
- Once stable, monitoring of pre-insulin blood glucose, urine glucose and water intake can be reduced to once weekly.

Although initially it was thought best to alter the dose of insulin daily based on pre-insulin blood glucose reading (similar to human diabetics), it was shown that keeping a set dose initially was more beneficial. Constant changes in insulin can be confusing for some owners to make, and neither cat had stable or consistent blood glucose readings while following this protocol.

Daily recording of pre-insulin blood glucose readings by the owners at home allowed us to monitor the progress of both cats in their normal surroundings, rather than the artificially stressful environment of the clinic. Very good glycaemic control was achieved in Barkley, which provides the best possible chance of remission at some time in the future. Reasonable glycaemic control was achieved in Dennis, although it is likely that Dennis’s diet is not appropriate which does not allow the same level of control compared to a cat fed a low carbohydrate, high protein diet.

The difference in outcomes for both cats seems greatly affected by the level of client compliance, which was excellent in Barkley’s case, but less than ideal in Dennis’s case. It is not expected that Dennis will achieve remission, as he was diabetic for some time before receiving treatment, and his glycaemic control is not good enough to achieve this. Barkley has a fair to good chance of achieving remission however, as his glycaemic control is good, he received treatment very promptly and client compliance is excellent.

**Post-script:** We ended up doing a dental on the cat in question.

References
2. Problem Based Feline Medicine Jaques Rand 2006
Multiple Myeloma in an 18-year-old cat

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‘Sleepy’ was a long term patient of our practice and at 15.5-years-of-age sustained severe facial fractures in a road accident resulting in bony distortion of the nasal cavity and enucleation of her right eye. Subsequent to this Sleepy developed a chronic nasal discharge (that was usually serous).

Biochemistry, haematology and urinalysis were performed - abnormal results were:

- Amyl 3143 (200-1100) USG 1.032
- BUN 19 (4-11)   Dipstick 4+ protein; 2+ blood; pH 6.0
- CREA 191 (27-186) No hypercalcaemia (2.39 mmol/L)
- Na 141 (142-164)
- Ca 146 (54-82)
- GLOB 115 (15-57)
- ALP 3143 (300-1100)
- HCT 0.19 (0.28-0.45)
- Mono 0.9 (<0.6)
- Retic 0.5% (abs 20 x 10^9/L)

- Marked hyperglobulinaemia
- Non-regenerative anaemia
- Stress leucogram
- Renal Insufficiency
- Haematuria/proteinuria

Differential diagnosis at this stage included:-

- Hyperglobulinaemia
- Myeloma
- Renal disease
- Hypercalcemia
- Other (reported in humans)

Differential diagnosis at this stage with such marked hyperglobulinaemia included:-

Multiple Myeloma
- Other neoplasia (eg B-cell lymphoma)
- Marked inflammatory process
- FIP
- Chronic infection (reported in dogs - leishmaniasis, ehrlichiosis, chronic pyoderm)
- Amyloidosis
- Other (reported in humans)
- Waldenstrom macroglobulinemia
- MGLUS (monoclonal gammopathy of undetermined significance)
- A protein electrophoresis was requested

Protein electrophoresis
- Alpha-1 Globulins 1 g/L
- Alpha-2 Globulins 20 g/L
- Beta Globulin 3 g/L
- Gamma Globulin 84 g/L

A monoclonal gammopathy suggesting multiple myeloma or lymphoid neoplasm
- Survey thoracic, abdominal and skeletal radiographs were taken
- No mediastinal mass; note thoracic vertebral spondylosis
- Left Radius – centrally there is a small area of bone lysis and 2 smaller lesions distally
- Left Ulna – similar lesion proximally
- Rest of long bones – no obvious lesions

Diagnosis
- The plasma cells were reported as well-differentiated lymphoid neoplasia
- 4 x FNA – 2 x spleen and 2 x liver
- FNAs of the liver and spleen were performed
- Multidisciplinary approach to try and fulfill the criteria needed for definitive diagnosis

Diagnosis – Multiple myeloma

Considering these factors the following diagnostic plan was formulated

- Bone marrow cytology/histopathology was declined by the owners as they did not want Sleepy to have a general anaesthetic.
- There was also the possibility of not achieving a diagnostic sample with a single aspirate - the marrow tumours can be very localised/focal and so multiple aspirates from different sites is recommended – particularly if the affected bone sites can be sampled. This would have been much more invasive and again not what the owners wanted.
- I have little experience with BMA and sample handling in our practice we have more experience with ultrasound guided FNAs and radiography/so this likely influenced my clinical decision making. I used these less invasive diagnostic modalities to try and fulfill the criteria needed for definitive diagnosis of multiple myeloma.
- Sleepy was sedated and abdominal ultrasound and FNAs of the liver and spleen were performed
- 4 x FNA – 2 x spleen and 2 x liver
- All samples contained plasma cells – more seen in the spleens from the spleen than the liver
- The plasma cells were reported as well-differentiated

Diagnosis – Multiple myeloma

Criteria satisfied
- Monoclonal gammopathy
- Lytic bone lesions

A literature review highlighted the following: Patel et al 2 in a retrospective study of 16 cats (2005) showed: Common findings in feline multiple myeloma include atypical plasma cell morphology, hypocholesterolemia, anaemia, bone lesions, and multi-organ involvement.

Based on the results of this study they advocate modifying the classic diagnostic criteria in cats to include:

- consideration of plasma cell morphology and
- urinal tract involvement

They also stated: Although we did not use extramedullary involvement as a diagnostic criterion, it was common finding in the cats in this study, suggesting that assessment of extramedullary sites could assist in the diagnostic evaluation of hyperglobulinaemic cats, especially when marrow aspirates cannot be obtained.

Mellor et al 4 (2008) in their retrospective analysis of 26 cases:-
- Reported that marked extramedullary involvement at initial clinical presentation is significantly more common in the cat than in human myeloma related disorder (MRO) patients.
- Demonstrated that cats with well differentiated tumors more commonly have extramedullary involvement than human myeloma patients with well-differentiated tumors (80% versus 20%)
- Obtained results that contrasted strongly with the human myeloma model of primary Intramedullary neoplastic transformation and suggested that primary extramedullary neoplastic transformation may be more common in feline MRO.

Authors’ views are not necessarily those of the CVE
Lethargy and weakness in a 9-year-old dog

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History: A 9-year-old male neutered Staffordshire Bull Terrier was presented with a history of lethargy and weakness of 1 day’s duration. The dog had a good appetite but had been noticeably losing weight over the past few weeks. No vomiting was reported. Mild diarrhoea had been observed by the owner over the past few weeks, described as voluminous and malodorous, with no mucous or blood. Oily stools had been noted on the dog’s bedding after he arose from rest.

Physical examination findings: Dog was bright and alert on examination. Mucous membranes were pink and capillary refill time was <2 seconds. Chest auscultation was unremarkable, abdomen was palpated as soft and relaxed, HR 200 bpm, pulses normal, temperature 38.4°C and loose faeces were noted on the rectal thermometer.

Initial Problem List
1. Diarrhoea
2. Weight loss
3. Oily discharge
4. Lethargy and weakness

Initial Assessment: This dog had been presented to the veterinarian primarily because the owner was concerned about the dog’s weight and general weakness. However, these are non-specific clinical signs which may have had many causes. The clinical signs that were considered most specific in this case were diarrhoea and weight loss. The non-specific problems were then considered in relation to the specific clinical signs.

Diarrhoea: This patient exhibited diarrhoea of several weeks’ duration, described as voluminous and malodorous, with no mucous or blood. The leading questions were:

• Was the diarrhoea small or large bowel in origin?
• Was the diarrhoea due to primary gastrointestinal or secondary gastrointestinal disease?

The description of the faeces were voluminous suggested that the faecal bulk had increased, which is characteristic of small bowel diarrhoea. The offensive odour of the faeces may have been due to excess gas production, which commonly occurs with small bowel diarrhoea (fermentation of malabsorbed carbohydrates by colonic bacteria). There had been no mucous or blood observed, which made large bowel diarrhoea less likely (but does not rule it out completely).

Small bowel diarrhoea can occur with either primary or secondary gastrointestinal (GIT) disease. There was no vomiting involved; however, this did not directly rule out primary GIT disease. As mentioned above, diarrhoea was not the primary presenting complaint in this case. This raised the suspicion that a secondary GIT disease was occurring. The clinical picture of weight loss, voluminous and malodorous faeces, and an oily discharge on the dog’s bedding made exocrine pancreatic insufficiency (EPI) high on the list of differentials, but is not pathognomonic for EPI. Primary small intestinal disease may also cause similar signs.

Weight Loss: Weight loss accompanied by diarrhoea is often a feature of malabsorption; for example, pancreatic insufficiency (maldigestion) or small bowel disease (malabsorption). If this was in fact the case, then the diagnostic approach was the same as for chronic diarrhoea. However, we couldn’t rule out that the weight loss and diarrhoea may have been 2 separate problems.

When dealing with weight loss, it is important to first consider whether the dog had been receiving an adequate intake of food. This dog had always been fed on a balanced adult dog food, a combination of canned and dry. Additionally, the owner reported that most of the weight loss did not coincide with receiving less food, or with an onset of anorexia. In fact, the dog had a good appetite. Therefore the dietary intake was adequate for the dog to have lost weight.

Weight loss in a dog with an adequate intake of nutrients can be due to:

• Maldigestion
• Malabsorption
• Malutilisation
• Increased nutrient loss - diabetes mellitus, PLE
• Elevated metabolism (e.g. hyperthyroidism)
• Neoplasia
• Chronic infection/Inflammation

Exocrine pancreatic insufficiency and intestinal parasites were high on the list of differentials and easy to rule out before more invasive tests were required.

Initial Plans: This dog’s diarrhoea had persisted for several weeks, therefore a thorough investigation was more appropriate rather than giving symptomatic treatment and observing for a response. Additionally, the fact that the dog was losing weight was cause for concern and an indication for a detailed investigation. The most appropriate first step was to perform a faecal floatation and direct saline smear to investigate for intestinal parasites. These tests are cheap, non-invasive and can be done quickly in-house to rule out the presence of parasites. If there was any doubt, a broad-spectrum anthelmintic could be given regardless of the findings.

Exocrine pancreatic insufficiency was high on the list of differentials, therefore a canine trypsin-1-like immunoreactivity test (cTLI) needed to be carried out to rule in/out this disease. This required an overnight fast and a single serum sample sent to an external laboratory so the result was not available immediately, so...
A complete blood count was important to investigate whether there were any infectious/inflammatory patterns to the white cells. A biochemical analysis allowed measurement of the blood glucose (to rule out diabetes) and liver function test proteins and liver enzymes in the serum in the case of a protein-losing enteropathy.

Electrolyte levels were important to measure any metabolic abnormalities that may have occurred secondary to the diarrhoea and also to survey for hyperadrenocorticism and liver disease.

If a diagnosis was not reached after the above diagnostic tests were performed, the next step would have been faecal culture and sensitivity testing. This would have excluded any parasitic infections.

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‘Bun’, a neutered malelop-eared rabbit who was 6-years-old when I first met him, was an extraordinary rabbit with great owners. The family consisted of 2 young adult owners, 1 geriatric cat, 2 turtles and Bun. When Bun was around 8-years-old, the family expanded to include 2 dogs, both joining the family as puppies. Bun was predominantly an indoor rabbit, which had free access throughout the house and used a litter tray for toileting. He had regular supervised outside time with his owners.

Bun was a confident and friendly rabbit who had a deep bond with his owners. Because of his habits, I was able to accommodate his needs. As a young rabbit, Bun had some issues with chewing electrical cables, walls and furniture but Bun’s owners researched this behaviour and methods to prevent damage both to Bun and to their house. They provided other chewing options and ensured that his environment was enriched and stimulating. He did still occasionally chew furniture or other parts of the house; however, his owners loved him deeply and did not ever consider this behaviour to be a problem.

Bun had a complicated medical history with renal and urinary tract disease issues as well as degenerative joint disease as he grew older. Bun was seen regularly by a veterinarian with a special interest in rabbits and my role was effectively as his general practitioner vet, coordinating his care on the recommendation of his specialists. Bun’s owners spent large amount of time and money on his health care. I was also involved in the care of their geriatric cat, a deceased female ex-stray of unknown age which had severe hyperthyroïdism, hypertension, renal failure and degenerative joint disease. I saw at least 1 member of this family almost every week for several years. There was no difference in their approach to their care of their rabbit, cat, dog or turtles.

Sadly Bun died at the age of 9 years while in the care of an emergency veterinary centre one weekend. I have counselled many owners before, during and after the death of a pet, and the death of this rabbit was no different. His owners were deeply distressed and went through the same grieving process that all bonded pet owners experience. They experienced guilt arising from not being with him when he died and the usual anger and questions that occur in these situations. They chose to have his body cremated. Bun was such an important part of their lives that his female owner placed a small amount of his ashes in her wedding bouquet 6 months after his death. Several years on, we still often talk about Bun when I see other members of their animal family.

The relationship between Bun and his owners highlights the special bond that can occur with particular individuals. I have known these owners for 5 years and have been with them through the death of 2 rabbits, a cat and a turtle. Although these owners are deeply bonded to all their pets and provide each with outstanding care, each experience has been different due partly to different circumstances surrounding the deaths and partly to the personality of each pet. Shortly after Bun’s death, the couple bought 2 young rabbits, 1 of which died unexpectedly at around 1 year of age. Although the owners were greatly upset, they did not have the same deep bond with this rabbit. However, when their geriatric cat was euthanased fairly recently, their pain and grief was palpable as they had a very close bond with their cat, as did I after caring for her for 5 years, and although we all knew that euthanasia was the best option for her, it was still extremely distressing.

Although many owners are reluctant to elect euthanasia for their sick or debilitated animals, the sense of relief afterward is immense for many people. It is incredibly hard to watch your beloved animal in pain and sitting with them during a gentle, painless death can be a strangely positive experience. In this case, the owners felt relief that their cat was no longer suffering.

The experience contrasted harshly with their experience with Bun, as they were not with him at the time and his death was not completely expected.

Relationships with pets occur on many levels and sometimes the particular personality of a pet allows for an especially strong bond, more so than the species of the pet. Although in my professional experience, many owners chose to have a different relationship with pets such as rabbits or guinea pigs than they do with their dogs or cats, there are others for whom the relationship occurs with the individual, regardless of the species.
Granulosa-theca cell tumour in a cat

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‘Whisky’, a 13-year-old, entire female, medium hair cat presented with a 3 month history of aggression and excessive vocalisation. The behaviour changes appeared to coincide with the owner moving house. Significant abnormalities were not identified on complete haematological (CBC), blood biochemistry (MBA) or total 14 analyses. The aggression and excessive vocalisation were diagnosed as behavioural and Feliseq® was prescribed.

Whisky represented 1 month later with continuing behavioural problems. The behavioural problems were characterized by increased howling at night and aggression towards the other cat. The owner reported that there had been no response to Feliseq® and Whisky was re-presented on a Cortisporin® tablet for 1 month, 2 months after initial presentation Whisky was re-assessed for worsening behavioural problems and a more thorough history was obtained. Whisky was bright and alert, eating and drinking normally and was an appropriate body weight. There was no other cat in the household and the owners had owned both cats for over 5 years. Whisky was an entire cat that had never had kittens and until 2004 never showed signs of being on heat. The behaviours described by her owner were classic signs of being in season. These include: presenting her rear for a scratch, a crouched gate, howling and fighting with the other cat.

Clinical findings included; temperature 38.5˚C, heart rate 120 beats per minute, respiratory rate 40 breaths per minute, mucous membranes pink and moist, capillary refill time < 2 seconds, poor hair coat, slightly thin body condition and being fractioned and dangerous to handle. Further examination was limited due to temperament.

Assessment at this stage included behavioural problems with increased intensity despite medication; entire female showing signs of persistent oestrus and possible infertility; a poor hair coat and thin body condition. The differential diagnosis of aggression include behavioural (e.g. fear, control, dominance); hormonal (e.g. functioning ovarian tumour, adrenal tumours, hyperthyroidism); metabolic (e.g. diabetes mellitus, anestrus, intermittent or continuous estrus or masculinisation2. The owner had a detailed water consumption history. Water intake over the next few weeks.

Abdominal radiographs showed a soft tissue opacity caudal to the urinary bladder on the lateral view; all other structures were unremarkable. Abdominal ultrasound findings included an enlarged uterus with thickened walls and occasional cystic components. The left uterine horn had a 0.5cm hyperechoic nodule and the left ovary was almost larger than the right (0.5x0.8cm) with a solitary hyperechoic nodule. The right ovary had 2 hypoechogenic nodules. The urinary bladder, mediastinal lymph nodes, kidneys, adrenal glands, spleen, pancreas and liver were unremarkable. Due to the abnormal findings on ultrasound, thoracic radiographs were taken to look for evidence of metastatic disease. Thoracic radiographs were unremarkable. A CT Scan of the head was also performed to look for multifocal disease, primary intra-cranial disease or malignancy. Pre- and post-contrast CT scans were normal.

Following this extensive work-up, there was no evidence of intra- or metastatic disease and the abnormal uterus and ovaries detected on abdominal ultrasound, it was considered likely that an ovarian tumour was causing the behavioural signs.

Whisky underwent a routine ventral midline ovariohysterectomy. Surgical exploration revealed a firm right uterine horn with a 13x13x8mm moderately firm, irregular, ovoid, right ovary containing a firm, cream, irregular, ovoid 10x7x4mm. The left uterine horn was similar to the right and the left ovary was 12x8x9mm, moderately firm and irregular to ovoid. The entire uterus and both ovaries were submitted for histopathology.

The right ovarian nodule was diagnosed as a granulosa-theca cell tumour and both right and left uterine horns were diagnosed as cystic endometrial hyperplasia.

Follow up over the next 12 months revealed improvement in the behavioural signs. The persistent signs of oestrous resolved and the aggression improved but did not entirely resolve. Whisky’s aggressive behaviour towards the other cat resolved, however she remained aggressive towards the other cat in the household, although to a lesser degree.

Whisky’s body condition and hair coat returned to normal and she continued to do well over the subsequent months. Basic diagnostic tests may reveal persistent oestrus and can still be problematic and may not resolve with medical intervention. It was noted that she appeared to have a lower than normal renal threshold for glucosuria. As she appeared to be well, body weight was stable and water consumption was not excessive, she was allowed to remain at home.


March 2010 – Georgia was returned to the cattery. The owner has noted some water seeking behaviour: licking from the water source. She may have been drinking more water from her bowl but it was not possible to get an accurate reading. Blood samples were taken and sent for a fructosamine level in an attempt to determine stress from diabetes mellitus, see Appendix 2.

Fructosamine was 445umol/L and thus supportive of diabetes mellitus. Georgia was admitted to the hospital with the intention of starting insulin therapy with Glargin. The following blood glucose curve was obtained on day 11: 8.88mmol/L, 12 noon 6.89mmol/L. 4pm 8.8mmol/L

In many cases the diagnosis of diabetes mellitus in the cat is quite straightforward. The cat may display classic clinical and historical signs. Basic diagnostic tests may reveal persistent oestrus and marked elevations in blood glucose and urate may be persistently glucosuric. In this instance, the diagnosis of diabetes mellitus is easily achieved and treatment can be commenced. However, in some cases, the diagnosis is less than clear.

The following case study illustrates the steps taken to establish a diagnosis in a patient with equivocal evidence of diabetes mellitus.

Georgia was a 19-year-old, female neutered, New Zealand cat of mixed breed. Her mother was a Burmese cross.

Case History: Georgia was a 13-year-old, female neutered, New Zealand cat of mixed breed. Her mother was a Burmese cross.

October 2009 – A routine gestational blood screen was run. Blood glucose was 9.9mmol/L in a cat on a glucometer as the profile selected does not include blood glucose. Elevated blood glucose of 18.8mmol/L was detected. Results were otherwise within normal limits. See Appendix 1. This test was taken days into a stay in the cattery. The elevation in blood glucose was thought to represent stress. The owner had not noticed any changes at home.

December 2009 – Georgia was admitted into the cattery for Christmas boarding. No clinical signs had been noted at home. In an attempt to avoid stress hyperglycaemia, blood glucose was re-evaluated immediately. Blood glucose of 12.2mmol/L was detected. Urination showed 2+ glucosuria. USG was 1.021. Stress from the travel to the clinic may have caused this elevation in blood glucose. Editor’s Note: It is very, very rare for stress to cause an elevation in blood glucose in a cat.
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1 week. A blood glucose curve was repeated, see Appendix 5. This curve showed good control of blood glucose. her water consumption had returned to normal, her appetite was preserved, and she had gained weight. At the time of writing, Georgia remains on 3U of glargine SC BID and appears to be doing very well.

Discussion: this case illustrates several points that can be encountered in the diagnosis of diabetes mellitus. Stress can be a confounding factor. In cats, blood glucose can raise significantly in response to stressful stimuli. Fructosamine can be used to differentiate stress from diabetes mellitus in most but not all cases. There is an equivocal range for fructosamine. In this instance, historical and clinical combination with repeated assessment of blood glucose were necessary.

it is interesting to note that Georgia appeared to have a renal threshold for glucosuria that was lower than the usually accepted cut off of 16mmol/L. Georgia developed glucosuria at a blood glucose concentration as low as 12.3mmol/L. It is important to remember that cut off levels and reference ranges do not hold true for all animals. Animals with a normal outside the reference range are common but should be expected. Trends over time may be helpful in determining what is normal for an individual. Georgia consistently displayed glucosuria at lower than expected blood glucose. This consistency in results supports the suspicion that her renal threshold is lower than 16mmol/L.

Stress hyperglycaemia can confound results of blood glucose analysis in cats. Elevations in blood glucose are seen in stressed or ill cats. Well and anxious cats can have blood glucose concentration as high as 16mmol/L and sick, stressed cats can have elevations in excess of 20mmol/L. Elevations of this magnitude would be expected in a severely ill cat with concurrent glucosuria. The elevations in Georgia's blood glucose prior to March 2010 were within the range seen in healthy stressed cats. The absence of any clinical signs suggestive of diabetes mellitus at home or while in the cattery and her feisty nature suggested the hyperglycaemia was due to stress. However, it is important to differentiate between stress and diabetes mellitus. Earlier detection and treatment will increase the chances of diabetes remission to a non insulin dependent state through reversal of glucotoxicity.

it is interesting that Georgia had normal or very near normal blood glucose (normal in non diabetic, unstressed cats taken to be 9.5mmol/L or less). this suggests that diabetes mellitus was not an acute stress reaction in Georgia. Georgia's blood glucose curves in April 2010, once insulin therapy had commenced, also showed a lack of stress hyperglycaemia. A conscious effort was made to keep Georgia as calm and stress free as possible when handling Georgia. All venipuncture was performed by the same technician and 'working with the patient' may all have a significant role in the diagnosis and management of diabetes mellitus in cats, particularly in stressful animals.

Fructosamine may be used to distinguish stress hyperglycaemia from diabetes mellitus. Fructosamines are stable complexes of proteins and carbohydrates. They are produced from the glycosylation and glycation of proteins and carbohydrates. Albinum is the common protein. This reaction is non reversible. An elevation in blood glucose will result in an increase in glycosylation of serum proteins preserving the life of the peptide – usually 1-2 weeks. Thus an elevated fructosamine can be encountered in the diagnosis of diabetes mellitus. Stress can be a confounding factor in cats, blood glucose can raise significantly in response to stressful stimuli. Fructosamine can be used to differentiate stress from diabetes mellitus in most but not all cases. There is an equivocal range for fructosamine. In this instance, historical and clinical combination with repeated assessment of blood glucose were necessary.

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Feline Progressive Dendritic Cell Histiocytosis

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Abstract: A 14-year-old female spayed domestic shorthair cat presented with a papulonodular dermatosis involving her face. Biopsy led to the diagnosis of feline progressive dendritic cell histiocytosis. Over 3 years this disorder acted in an indolent manner. Lesions would wax and wane. In the latter stages of the disorder the papulonodular change worsened and lesions ulcerated. Life quality of the patient deteriorated and euthanasia was performed. This case report will increase awareness of features of this disorder.

Introduction
Feline progressive dendritic cell histiocytosis (FPDH) presents initially as a papulonodular dermatosis (Day M. 2008). In advanced cases, spread may extend to lymph nodes and internal organs (Affolter & Moore 2006). Monocytes, macrophages and dendritic antigen presenting cells are all of the histiocytic cell group. Dendritic cell histiocytosis play an important role in processing and presenting antigen (Affolter 2004).

Histiocytic disorders are classified based on their behaviour (Wellman 2007). Broad classification divides these into reactive or neoplastic disorders (Wellman 2007). FPDH is considered a neoplastic disorder (Affolter & Moore 2006). This classification is based on observed failure of FPDH to respond to immunosuppressive treatment (Affolter & Moore 2006)

Cases of FPDH have been reported infrequently. Affolter (2006) is the only in-depth report readily available that discusses FPDH in a group of affected cats. The aetiology of FPDH is unknown (Affolter & Moore 2006).

No effective treatment is known and because of the progressive course run by the disease, diagnosis carries a guarded prognosis (Affolter 2004). The owner would have been informed that although the lesions had acted benign for several years the long term outlook was poor. Because of the progressive nature of FPDH the prognosis is guarded (Affolter 2004). The owner would also have been aware at an earlier time to monitor the cat for signs of unwellness that may reflect systemic involvement of this histiocytic disorder. As no effective treatment is documented, the final outcome for the patient was not influenced by a delay in making a definitive diagnosis.

The spontaneous waxing and waning nature of FPDH made evaluation of therapeutic response problematic. On 4 occasions the nodular lesions seemed to regress after therapeutic intervention. Nodules reduced in size following use of Elocon, use of Vibravet, performing a fine needle biopsy and performing an incisional biopsy. In the latter case, lesion regression was dramatic (Affolter 2004). The spontaneous waxing and waning nature of FPDH made evaluation of therapeutic response problematic. On 4 occasions the nodular lesions seemed to regress after therapeutic intervention. Nodules reduced in size following use of Elocon, use of Vibravet, performing a fine needle biopsy and performing an incisional biopsy. The owner was informed that no effective treatment was available.

Histologic diagnosis: Atypical histiocytic proliferation most consistent with feline progressive dendritic cell histiocytosis.

Case Report
A 14-year-old spayed female domestic shorthair cat was presented with a facial distribution of a papulonodular dermatosis. The cat’s owner had been aware of this skin disorder for the past 3 years. During this time the lesions had waxed and waned at irregular intervals. There was no correlation between seasonal change and increased size of the lesions. The owner suspected the periods when facial lesions worsened correlated with times when the cat carried large mouldy leaves in its mouth. The owner would see the cat running up the path with an enormous mouldy leaf in its mouth. Direct contact of the leaf with facial tissue was common. Over the 12 months prior to presentation, the owner noted a worsening of lesions with shorter intervals where lesions reduced in size.

Findings at the initial visit were of prominent papulonodular change on and around the nasal area and area above both eyebrows. The largest nodule was 3 x 2 cm. The nodules were solid, raised, non painful, non pruritic and irregular in shape. An impression was gained of generalised papulonodular change to affected sites. Refer to Photo 1 and 2 for appearance of the lesions at the time of presentation.

The aetiology of FPDH is unknown (Affolter & Moore 2006).

The cat was next evaluated 6 months later. The skin condition had markedly deteriorated. Distribution of the papulonodular lesions had not changed but a far greater number of nodules, of larger size, were seen. The cat seemed untroubled by the lesions and she remained well. It was decided to trial a course of Vibravet (50mg tablets. Pfizer Animal Health). This drug was chosen because of its antimicrobial and immune modulating action and its high safety profile. 25mg of Vibravet to be crushed and mixed in food to be given 12 hourly for a 8 week trial.

Initial apparent response seemed favourable with a report of 40% shrinkage of the nodules. However, by the 8th week there was a dramatic deterioration in appearance of the lesions. A fine needle aspirate was performed. As well as routine cytology, the lab was requested to run special stains to check for presence of mycobacteria, nocardia and actinomyces.

Cytology revealed a non suppurative macrophagic/lymphocytic inflammation and no evidence of cause was found.

A month later the largest of the nodules ulcerated and the cat started to scratch the lesion. Under general anaesthetic an incisional biopsy was taken. Refer to photo 3 for appearance of the ulcerated nodule at the time biopsy was taken.

Histologic diagnosis:
Atypical histiocytic proliferation most consistent with feline progressive dendritic cell histiocytosis.

Histologic description: Sheets of histiocytic cells (macrophages) infiltrate through the superficial and deep dermis in a diffuse mass, interspersed by low numbers of neutrophils and occasional small clusters of lymphocytes. The histiocytic cells have large, round, oval or indented nuclei with fine chromatin and prominent single nucleoli. There is moderate, two fold anisokaryosis and mitotic figures are present, approximately 3 per 10 high power fields. The cells have abundant foamy eosinophilic cytoplasm. In some areas the histiocytic cells have a more vacuolated appearance to the cytoplasm.

The spontaneous waxing and waning nature of FPDH made evaluation of therapeutic response problematic. On 4 occasions the nodular lesions seemed to regress after therapeutic intervention. Nodules reduced in size following use of Elocon, use of Vibravet, performing a fine needle biopsy and performing an incisional biopsy. The cat was withdrawn and depressed and often tried to scratch the ulcerating nodules. Euthanasia was performed. Permission to autopsy was not discussed.

Discussion
As the dramatic deterioration in appearance of the lesions was not influenced by a delay in making a definitive diagnosis. The spontaneous waxing and waning nature of FPDH made evaluation of therapeutic response problematic. On 4 occasions the nodular lesions seemed to regress after therapeutic intervention. Nodules reduced in size following use of Elocon, use of Vibravet, performing a fine needle biopsy and performing an incisional biopsy. The owner was informed that no effective treatment was available. Within 10 days of the biopsy being taken the owner reported a dramatic improvement in the appearance of the nodules. Permission did not occur but lesions regressed by 80%.

Figures 1 & 2. Appearance of lesions at presentation.

Figure 3. Appearance of the ulcerated nodule at the time biopsy was taken.
following fine needle biopsy, and also when an incisional biopsy was taken, may have occurred due to mechanical insult inducing an alteration in the behaviour of the dendritic cells. No therapy has been shown to influence the progressive course of FPDH (Affolter & Moore 2006).

The early appearance of skin lesions in all reported cases of FPDH is similar. Waxes and wanes of the lesions is also common to the reported cases. Lesions may appear as a solitary skin nodule then over time may become numerous and coalesce to form large plaques or remain as non painful large skin nodules (Affolter 2004). Lesions up to 1.5cm have been reported (Affolter 2004). In our patient, the appearance of the lesions and the waxing and waning of these lesions fitted previous descriptions of FPDH. Size of the largest nodules in our patient was twice that previously reported. In common with prior reports, our patient experienced ulceration of the nodules in the late stage of the disorder.

Distribution of lesions in published reports suggests a predilection for the head, neck and lower extremities (Affolter 2004). In Affolter & Moore’s study (2006), 21 of 22 cats had leg and foot involvement and 18 of 22 had head involvement. Our patient had lesions restricted only to the facial site.

Further diagnostic investigation by immunophenotyping would have allowed a better understanding of the cell origin of the dendritic cells. A better understanding of the disorder at the time of diagnosis would have prompted this to have been investigated. Immunophenotyping in cats may be limited by the limited availability of useful antibodies or the need for snap frozen fresh tissue (Ordeix 2009).

The cause of FPDH is unknown. In dogs, some cases of histiocytosis are possibly triggered by persistent antigenic stimulation, but no antigen has been identified (Day 1999). T cell driven immune dysregulation is suspected. The canine disorders, however, appear reactive rather than neoplastic and response to immunosuppressive treatment occurs. In cats, no report of resolution on immunosuppressive drugs exist (Affolter & Moore 2006). It is likely the pathogenesis of FPDH differs from canine cutaneous and systemic histiocytosis. It is of interest that the owner of our patient was emphatic that waxing and waning of the disorder correlated with direct facial contact with mouldy leaves. FeLV and FIV status of our patient was not determined. It is therefore not possible to comment on the role these viruses may play in a dysregulated immune response.

The length of time when the lesions act in an indolent manner has previously been reported as up to 3 years (Affolter & Moore 2006). In our patient this indolent period lasted for 3 years. It would have been useful to perform a necropsy as FPDH is poorly understood and the information gained through a necropsy would help understanding of the disorder. Late spread to internal organs could have been checked.

Conclusion

FPDH is a rare disorder. Being aware of its existence is important when considering the differential diagnosis list for papulonodular disease. When history includes the unusual feature of very slow progression of papulonodular lesions with waxing and waning of the lesions, one should rank this disorder high on the differential diagnosis list. Diagnosis early on in the disease allows the owner to be informed of the likely progression of the disorder and to be aware that no effective treatment is available.

Every aspect of FPDH is poorly understood and research into the disorder would be of great benefit. The cause of the disorder, its pathogenesis and possible treatment options are unknown and documenting case reports will increase awareness among clinicians.

References

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