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Welcome...

Welcome to the Winter 2012 Vetnostics newsletter. In this edition we focus on the topical subject of cutaneous mycobacterial diseases in dogs and cats as well as the continuing series' on Adrenal disease, frustrating histopathology cases/diagnoses and 'What's Your Diagnosis?'. I hope you find it all interesting.

As always, please contact me, tel: 02 9005 7272 or email doug.hayward@vetnostics.com.au, if you have any requests/ideas for future newsletters or any other queries.

CUTANEOUS MYCOBACTERIAL DISEASE IN DOGS AND CATS (PART 1):

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Laboratory diagnosis and treatment of non-cultivable mycobacterial infections in dogs (Canine Leproid Granulomas) and cats (Feline Leprosy Syndromes)

Ulcerated and non-ulcerated cutaneous lumps and bumps, sometimes discharging, on animals are a common signalment in veterinary practice. Elucidating the aetiopathogenesis of these lesions can sometimes be difficult – particularly when investigating some microbial pathogens. The successful treatment of some of these microbial pathogens (e.g. mycobacteria and fungi), may also require further rigorous laboratory investigation, utilizing "cutting edge" laboratory techniques (e.g. PCR), to identify these organisms to the species level.

This article is the first of a series that will outline the range of ancillary laboratory tests that are now being used to more precisely define the various manifestations of cutaneous mycobacterial infections in dogs and cats and explore therapeutic options accordingly for each disease manifestation.

Cutaneous mycobacteriosis in dogs and cats is generally caused by infection with one of the following three groups of Mycobacteria (*Algorithm 1*).

Group 1	**Rapidly growing mycobacteria**
Group 2	**Non-cultivable or poorly cultivable mycobacteria**
Group 3	**Slow growing mycobacteria**

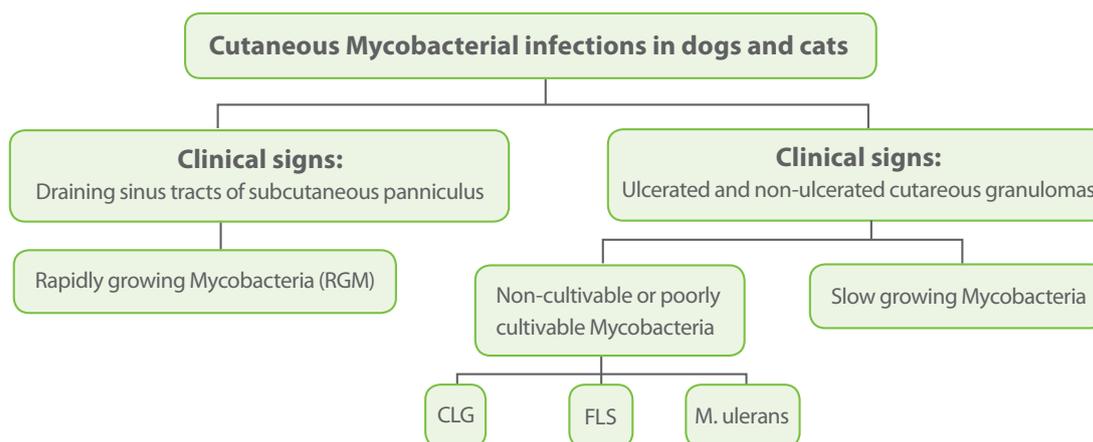In this article we will concentrate on the group 2 (non-cultivable) mycobacteria. These are saprophytic species of mycobacteria which are extremely fastidious and generally non-cultivable on current culture media in the laboratory. These bacteria are involved in 2 specific animal disease syndromes associated with ulcerated or non-ulcerated cutaneous granulomas: canine leproid granuloma (CLG) and feline leprosy syndrome (FLS).

Canine Leproid Granuloma (Canine Leprosy)

CLG usually manifest as single or multiple, firm, granulomatous to pyogranulomatous, well-circumscribed ulcerated or non-ulcerated nodules in the skin or subcutis (2 to 50 mm in diameter). Nodules are typically located on the head, particularly the dorsal fold of the ears (Figure 1), but may be located elsewhere, e.g. the trunk or rump. Extension of the infection to lymph nodes, nerves and internal organs does not occur. CLG appears to have no zoonotic potential for people.

CLG should be strongly suspected when typical lesions described above are found in a short-coated breed (with Boxers and their crosses remarkably over-represented although Staffordshire bull terriers, Fox hounds and Dobermans are also commonly affected).[1] If only a single solitary nodule is present it also important to exclude neoplasia (especially mast cell tumours).

Algorithm 1
Overview of cutaneous mycobacterial infections in dogs and cats

```
          Cutaneous Mycobacterial infections in dogs and cats
                    │                              │
        ┌───────────┴────────────┐      ┌──────────┴─────────────┐
        │ Clinical signs:        │      │ Clinical signs:        │
        │ Draining sinus tracts  │      │ Ulcerated and          │
        │ of subcutaneous        │      │ non-ulcerated          │
        │ panniculus             │      │ cutaneous granulomas   │
        └───────────┬────────────┘      └──────────┬─────────────┘
                    │                   ┌──────────┴──────────┐
        ┌───────────┴────────────┐   ┌──┴──────────────┐  ┌───┴──────────────┐
        │ Rapidly growing        │   │ Non-cultivable  │  │ Slow growing     │
        │ Mycobacteria (RGM)     │   │ or poorly       │  │ Mycobacteria     │
        └────────────────────────┘   │ cultivable      │  └──────────────────┘
                                     │ Mycobacteria    │
                                     └──┬────┬──────┬──┘
                                     ┌──┴─┐ ┌┴──┐ ┌─┴──────────┐
                                     │CLG │ │FLS│ │ M. ulerans │
                                     └────┘ └───┘ └────────────┘
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CLG is caused by a single, novel, mycobacterium yet to be fully characterised. Infection with this organism may be self-limiting,[1] although it is impossible to predict which cases of CLG will have lesions that regress spontaneously.

Diagnosis:

Performing fine needle aspiration (FNA) cytology would be, in the first instance, the easiest and fastest way of establishing a definitive diagnosis or raising the index of suspicion for the possibility of infection as well as helping dictate the next course of action in the further investigation of this mycobacterial infection which would usually include qPCR (see below).

Mycobacteria appear as negatively-stained bacilli (NSB) in routine cytology preparations as their lipid cell wall prevents penetration by Romanowsky-type stains (e.g. DiffQuik). Further special stains such as the Ziehl-Neelsen (ZN) stain, where the bacilli take up the carbol fuchsin to appear pink - so called acid-fast bacilli (AFB) can also be performed. In smears, CLG is characterised by the presence of numerous, often spindle-shaped macrophages, variable numbers of lymphocytes and plasma cells, lower numbers of neutrophils and variable numbers of medium length AFB, either intracellularly (within macrophages or giant cells), or extracellularly.[2] While some reports suggest that AFB are easy to locate in lesions, organisms can be extremely difficult to find in some instances, depending on the stage of the infection.

Cytology preparations negative for AFB do not rule out CLG, and should prompt examination of histologic sections or submission of material for PCR. Culture of the CLG organism in vitro is not possible because its growth requirements have not been determined.

Histopathological examination of these lesions within the dermis and subcutis reveals pyogranulomas composed of epithelioid macrophages, Langerhans-type giant cells with scattered neutrophils, plasma cells and small

lymphocytes. The number and morphology of AFB in ZN-stained tissue sections is highly variable. Bacteria ranged from long, slender filaments in parallel sheaves to short, variably beaded bacilli or highly beaded to coccoid forms. Morphology may be more uniform in DiffQuik stained smears than in fixed-tissue sections. Sections may require a lengthy search, even by experienced pathologists, to locate foci in which AFB are evident.[2] Further PCR testing could also be performed (see below) on further re-cuts of histology sections examined to attempt to identify the mycobacterial organisms seen.

The advent of PCR testing has helped immensely in achieving a definitive diagnosis of mycobacterial spp infections. PCR is performed by amplifying regions of the bacterial 16S rRNA gene,[3] 16S-23S internal transcribed spacer (ITS) region, or a region within the hsp65 gene,[4] using mycobacterium-specific primers. Species identification is made via nucleotide sequence analysis of the amplified DNA fragment and comparison

Figure 1: Typical CLG lesion

with known sequences in the GenBank database. Although PCR testing is more sensitive when performed on DNA extracted from fresh tissue, it has also been used successfully on paraffin-embedded, formalin-fixed (PEFF) tissue.

> An enhanced, relatively inexpensive, real-time PCR (qPCR) assay has also very recently been developed for the CLG organism that is sensitive and specific. We have successfully used this assay on the same cytology slides submitted to our lab for routine examination with DiffQuik staining in which a Mycobacterial infection was diagnosed, to identify the species of Mycobacteria present on those cytology slides. The combination of cytology and qPCR has resulted in a powerful, relatively inexpensive, yet minimally invasive, combination of laboratory investigative techniques to help diagnose CLG.

Treatment of CLG:

The great majority of CLG infections are self-limiting, with spontaneous regression of lesions occurring within one to three months, regardless of treatment, likely due to an effective adaptive cell-mediated immune response. This complicates assessment of efficacy of empiric antimicrobial regimes. In one study of CLG in dogs, 57% of cases recorded a favourable response to doxycycline, 63% had a favourable response to amoxicillin-clavulanic acid, while spontaneous regression occurred in 86% of untreated dogs.[1] Many clinicians believe drug therapy against secondary Staphylococcus pseudintermedius is helpful, less expensive and have fewer side effects than drugs used for specific combination anti-mycobacterial therapy.

Persistence of lesions over a timeframe exceeding that for which spontaneous regression commonly occurs (three to six months) warrants further treatment. Cell-mediated immunity may be compromised in these patients, perhaps due to inherited immunodeficiency affecting major histocompatibility complex expression, innate immunity or the development of adaptive immunity. In this regard, it is of great interest that Boxers are at greatly increased risk for CLG, prototothecosis, and histiocytic ulcerative colitis, diseases that may have in common insufficient innate or adaptive immunity.

In dogs with CLG, marginal surgical resection of nodules is typically curative, even if negligible margins are obtained. In cases with large lesions, or multi-focal lesions medical treatment alone may be a better first-line option.

Medical treatment of mycobacterial disease presents several challenges. Antimicrobials must achieve therapeutic concentrations intracellularly within phagocytes, in a range of tissues and with minimal toxicity to the host. Furthermore, they need to work despite a likely inadequate host immune response (defective innate and/or adaptive immunity). Additionally, Mycobacteria spp. in general are known to readily develop antimicrobial-resistant clones. Therefore, combination antimicrobial therapy with agents known to be effective against slow-growing non-tuberculous mycobacteria, such as rifampicin, clofazimine, clarithromycin and moxifloxacin/pradofloxacin, may facilitate disease resolution. Selecting appropriate antimicrobials is complicated by the fact that most slow growing mycobacteria known to causes these diseases have yet to be successfully cultured on synthetic media or in tissue culture. Even those species that can be isolated on laboratory media take months to grow.

Topical formulations of clofazimine in petroleum jelly have been used as an adjunct to oral rifampicin and doxycycline to treat dogs with CLG successfully.[4] More recently we have had good results with silver sulphasalazine, with or without DMSO.[5] Historically, radiation therapy was also found to be effective, although of course this is no longer recommended. A combination of rifampicin (10-15 mg/kg PO q 24hr) and clarithromycin (7.5-12.5 mg/kg PO q 8-12hr) is recommended for treating severe or refractory CLG,[4] in concert with topical silver sulphasalazine (see *Table 1* following). Some dogs with CLG develop lesions subsequent to therapy or self-cure, often some years later; it is not known whether the new lesions represent new infections or disease recrudescence.[1]

Feline leprosy Syndromes (FLS)

FLS is generally seen in cats with access to outdoors. Young male cats are overrepresented in some studies, likely because of their tendencies to fight and hunt.

FLS may present with single or multiple cutaneous nodules ranging from 2-40 mm in diameter (*Figure 2*), typically accompanied by peripheral lymphadenomegally. In cats with focal disease, lesions are often found on the face, head, limbs or trunk – areas subject to penetrating injury. The location of lesions on cats suggests inoculation of organisms through insect bites, rodent bites or (most likely) fight wounds. Fight wounds are normally ascribed to cats, but may include injuries from prey species such as rats or possums.

Mycobacterial species associated with feline leprosy include M. lepraemurium (*Figure 2*), M. visibile, Mycobacterium sp. strain Tarwin[6] and a novel species found in New Zealand and the East coast of Australia.[7] Some organisms (such as M. sp. strain Tarwin) are associated with localised disease in an immunocompetent host, while others (such as the novel East-coast species) are associated with haematogenously disseminated disease (usually limited to the skin) in an immune-deficient host.[6,7]

Unlike CLG, FLS typically have a progressive and occasionally aggressive clinical course, depending on the causal species, the size of the infective inoculum(s) and the immunologic response of the host. In some instances, widespread lesions can develop. Large tuberculoid lesions may ulcerate. Infection can recur following incomplete surgical excision of lesions and after insufficiently long medical therapy.

Figure 2:
FLS case associated with *M. lepraemurium* infection

Diagnosis

Haematology and biochemistry demonstrate non-specific changes, but are worth pursuing, especially in older cats because co-morbidities such as liver or kidney disease can impact on drug choices and prognosis. Testing for FIV and FeLV should be considered, although FIV positivity does not preclude successful treatment.

As for CLG, performing fine needle aspiration (FNA) cytology would be, in the first instance, the easiest and fastest way of establishing a definitive diagnosis or raising the index of suspicion for the possibility of infection as well as helping dictate the next course of action in the further investigation of this mycobacterial infection which would usually include qPCR (see following page).

Cytologically, numerous NSB, either within macrophages and giant cells or extracellularly, are present on DiffQuik stained smears (*Figure 3a,3b*). AFB are easily seen in ZN-stained smears (*Figure 3c,3d*) from needle aspirates from these lesions as well.

Once a cytological diagnosis has been established, and as most organisms associated with FLS cannot be cultured, PCR testing is recommend for mycobacterial species identification. PCR testing utilises primers that amplify regions of the 16S rRNA gene and ITS region[6,7] followed by sequence analysis of the resulting amplicon (*Figure 4*). PCR testing is very sensitive even when used on PEFF tissues, as long as the contact time of the tissue specimen with formalin is less than 48 hours.

Figure 3 (a-d): Fine Needle Aspirate (FNA) from 14yo cat with multiple firm subcutaneous swellings located on head near nasal bridge.
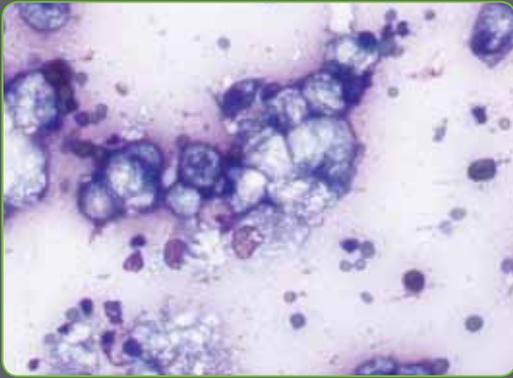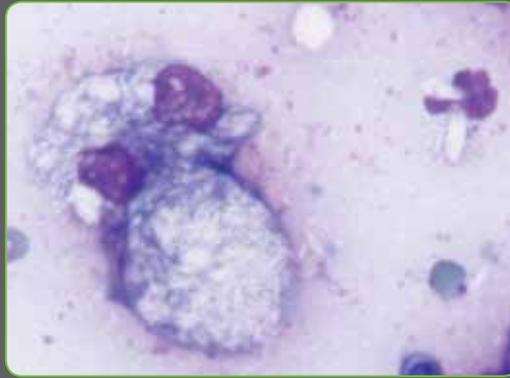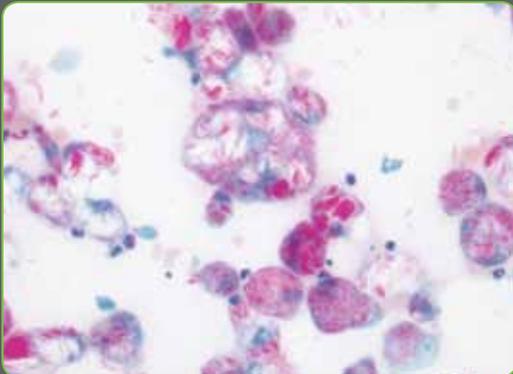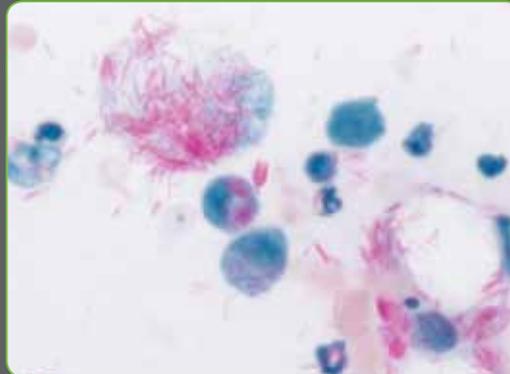
Figure 3A

Figure 3B

Figure 3C

Figure 3D

Recently with the advent of qPCR testing, as for CLG, we have been able to utilise methanol-fixed and DiffQuik stained smears of fine needle aspirates which have been diagnosed cytologically with mycobacterial infection, to amplify DNA in cases where NSB or AFB are abundant; this involves aseptically scraping material from the slides using a scalpel blade into a sterile tube, followed by appropriate DNA extraction procedures. Indeed, this represents the most expedient and least invasive way to obtain a definitive diagnosis, where appropriate laboratory support is available.

Histologically, FLS is not distinguishable from potentially zoonotic tuberculous infections. In cases due to *M. lepraemurium* and *M. avium* complex (MAC), bacilli are not visible in haematoxylin and eosin (H&E) stained sections. In contrast, *M. visibile* (hence the name) and the novel East coast species can be seen in H&E sections because they weakly take up the haematoxylin.[8] Despite this distinguishing feature, histological features should not be relied on to predict aetiology, the specific organism involved or the prognosis. Indeed, it is desirable to instruct the veterinary laboratory to forward tissue specimens to a mycobacteria reference laboratory for PCR and sequence analysis (to determine aetiology), and for attempted culture of mycobacteria and related organisms (e.g. Nocardia spp.).

Figure 4.
PCR amplicon amplified from a fresh tissue specimen obtained from the cat with cutaneous mycobacterial infection.

TGAGTAACACGTGGGTAATCTGCCCTGCACTTCAGGGATAAGCCTGGGAAACCGGGT
CTAATACCGAATAGGACCTTAAGGCGCATGCCTTGTGGTGGAAAGCTTTTGCGGTGT
GGGATGGGCCCGCGGCCTATCAGCTTGTTGGTGGGGTGACGGCCTACCAAGGCGAC
GACGGGTAGCCGGCCTGAGAGGGTGTCCGGCCACACTGGGACTGAGATACGGCCCA
GACTCCTACGGGAGGCAGCAGTGGGGAATATTGCACAATGGGCGCAAGCCTGATGC
AGCGACGCCGCGTGAGGGATGACGGCCTTCGGGTTGTAAACCTCTTTCACCATCGAC
GAAGGTTCGGGTTTTCTCGGG TTGACGGTAGGTGGAGAAGAAGCACCGGCCAA

Table 1. Antimicrobials used in the treatment of canine leproid granuloma and feline leprosy syndromes.

Drug	Dose (mg/kg) DOG	Dose (mg/kg) CAT	Route of Dosing	Dosage Interval (Hours)	Adverse Effects
Pradofloxacin*	5 (Tablets)	3 (Suspension)	Oral	24	More effective than enrofloxacin, marbofloxacin and orbifloxacin against mycobacteria and is devoid of retinotoxicity even at high doses
Rifampicin	10-15 (Maximum 600mg q 24hr)	10-15	Oral	24	Hepatotoxicity, induction of liver enzymes, generalised erythema and pruritus, dyspnoea, CNS signs, teratogenic
Clarithromycin	5-15 7.5-12.5	5-15 62.5 mg/cat 125 mg/cat	Oral Oral Oral Oral	12 12 12 24	Pinnal erythema, generalised erythema, hepatotoxicity
Clofazimine	N/A	4-10 25-50mg/cat	Oral Oral	24 24-48	Hepatotoxicity, gastrointestinal signs, photosensitisation, pitting corneal lesions
Doxycycline Monohydrate	5-7.5	5-10	Oral	12	Gastrointestinal signs, oesophagitis
Amikacin	10-15	10-15	IV, IMI, SCI	24	Nephrotoxicity, ototoxicity

Treatment of FLS:

In contrast to CLG, FLS syndromes generally have an unrelenting, progressive clinical course. Furthermore, lesions can recur following surgical excision, especially when inadequate margins are obtained and appropriate anti-mycobacterial agents are not given post-operatively. For this reason, medical treatment should be instituted immediately after diagnosis, as a delay may permit lesions to spread to contiguous skin, lymph nodes and even internal organs.

Medical treatment of FLS should be continued until lesions have resolved completely, and ideally for a further 2-3 months after lesions have regressed, to reduce the risk of recurrence. Some cats with FLS may require lifelong treatment with clarithromycin to prevent recurrence of lesions. Some medications, for example rifampicin, may have serious side effects specifically hepatotoxicity. Animals should be closely monitored for signs of liver dysfunction including inappetance, vomiting or jaundice. Rifampicin hepatopathy can be fatal! A marked increase in serum alanine aminotransferase activity signifies the need to change therapy to a different drug such as clofazimine. As clofazimine can cause photosensitisation, cats should be housed indoors for the duration of therapy.

FLS can be treated with combinations of 2-3 drugs from *Table 1*, typically 1) clarithromycin, 2) pradofloxacin[9], 3) rifampicin or clofazimine. Where pradofloxacin is unavailable the human drug moxifloxacin[10] may be substituted at a dose of 10 mg/kg orally once daily; because of the large tablet size, this drug needs to be compounded for use in cats and small dogs. Occasionally, it can cause vomiting.

Localised infections due to *Mycobacterium* ulcerans

A less well known poorly cultivable mycobacterial species that commonly causes disease in humans (particularly overseas) but has infrequently been diagnosed in animals is *Mycobacterium ulcerans*.

Mycobacterium ulcerans is the causative agent of Buruli ulcer, a chronic localised infection of the skin and subcutis of human patients typically associated with necrotizing skin ulcers with undermined edges. In Australia it occurs mainly in regions of coastal Victoria and Queensland, where it is known as Bairnsdale or Daintree ulcer, respectively. Non-human cases of *M ulcerans* infection have only been reported from Victoria, mainly in marsupial species such as koalas[11], ringtail possums and a long-footed potoroo[12]. Two cases have been described in alpacas. Recently, *M ulcerans* has been diagnosed in a cat[13], two horses[14] , more than five dogs[15] and numerous brush tailed and ring tailed possums[12] .

PCR is exceedingly helpful for rapid diagnosis, although the organism does grow slowly on synthetic media.

Conclusion:

In the majority of cases, conventional mycobacterial culture of CLG and FLS gives a negative result. Consequently, mycobacterial aetiology can only be proven using molecular techniques such as PCR amplification and nucleotide sequence determination of gene fragments, as described previously. PCR has the additional advantage of providing a rapid diagnosis. Fresh (frozen) tissue delivered to a diagnostic laboratory with mycobacterial expertise and PCR facilities provides the optimal sample. Sometimes PCR can be performed successfully on formalin-fixed paraffin-embedded material, although fixation conditions invariably cause some DNA degradation that may limit the success of the procedure.

With the recent advent of PCR & qPCR new laboratory tests, cytological specimens which have been DiffQuik stained and used to diagnose or raise the suspicion of mycobacterial disease can also be submitted to further mycobacterial investigations in an attempt to identify the species of Mycobacteria present on those cytology slides.

Treatment protocols for CLG and FLS will be refined as further investigations yield more information about the ecological niche of the causative mycobacteria involved and the optimal growth requirements for successful laboratory culture and susceptibility testing of these organisms are elucidated.

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ADRENALS:
What you won't find in a textbook

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PART 4a: ADRENAL FUNCTION TESTS

BASAL CORTISOL

1. The gold standard method for cortisol measurement is radioimmunoassay (RIA). Radioimmunoassay (RIA) results are accurate to 10 nmol/L. However, most laboratories today measure cortisol by chemiluminescent assays as these avoid the radiation hazards of RIA. Low and low normal cortisol concentrations cannot be measured accurately by some chemiluminescent assays (Russell et al 2007). The chemiluminescent assay used at Vetnostics was validated against RIA for concentrations <100 nmol/L, using the blood samples from Russell's study (2007) on which another assay was found to be unreliable. There was excellent correlation and no statistically significant difference demonstrated between the Vetnostics assay and RIA (Foster, unpublished data).

2. Prednisolone, cortisone and fludrocortisone all cross-react with cortisol assays. It is difficult to ascertain accurate withdrawal times however, it is safe to use the following:
   - **prednisolone: 36h**
   - **cortisone acetate: 12h**
   - **fludrocortisone: 24h**

   It is widely stated that fludrocortisone does not cross-react with the cortisol assay as it is a mineralocorticoid but that is not the case (Tate 2004) with one RIA cortisol kit insert reporting up to 33% cross-reactivity (GammaCoat Cortisol kit). Dexamethasone does not cross react with the cortisol assay. The withdrawal time for long-acting corticosteroid preparations depends on the actual corticosteroid used and is also likely to vary in individual dogs.

HYPERADRENOCORTICISM (hyperA)

1. Basal cortisol concentration has no place in the diagnosis of hyperA unless it happens to be greater than the upper level cut-off for a post-ACTH stimulation test cortisol.

HYPOADRENOCORTICISM (hypoA)

1. Basal cortisol can be helpful as a rapid check for the possibility of hypoA in a collapsed/ill dog although the gold-standard for diagnosis of hypoA is still an ACTH stimulation test. Only one study has looked at basal cortisol concentrations in suspected Addisonian patients (Lennon et al 2007). The abstract states that a result >2 µg/dL (56 nmol/L) excluded hypoA (provided there were no cross-reacting corticosteroids) and that if results were ≤2 µg/dL, no conclusions could be drawn. However, what wasn't included in the abstract was the information that only 2/110 dogs with non-adrenal illness had a basal cortisol of ≤1 µg/dL (28 nmol/L), giving basal cortisol concentration a specificity of 98.2% in ill dogs (specificity of ≤2 µg/dL was only 78.2%). Given that the assay used in the study had 1 µg/dL (28 nmol/L) as its lower level of measurement accuracy, we have no information as to whether the 2 "false positives" were 27 nmol/L or <10 nmol/L (the usual cut-off for RIA). A cortisol concentration of <10 nmol/L is highly likely to be indicative of hypoA in a collapsed/critically ill dog.

2. Relative adrenal insufficiency is a syndrome in which there is insufficient secretion of cortisol in relation to an increased demand during periods of stress, particularly in critical illnesses such as sepsis and septic shock. It is defined as a transiently inadequate response to exogenous ACTH. Differentiating relative adrenal insufficiency from true glucocorticoid deficiency would not be possible using basal cortisol. It may even be difficult with an ACTH stimulation test and sometimes serial testing is required.

3. ACTH stimulation test results from cases of iatrogenic hyperA can mimic those from spontaneous hypoA cases. Topical, aural and ophthalmic preparations can all have systemic effects and result in iatrogenic hyperA with failure to stimulate in response to ACTH administration. Prednefrin Forte® eye drops, for example, are not infrequently associated with iatrogenic hyperA. Even drugs such as budesonide, which theoretically act locally with minimal systemic effects, can significantly affect ACTH stimulation tests and Vetnostics recently noted ACTH stimulation test results of <10 nmol/L pre and post-stimulation in a dog being treated with a standard dose of budesonide. The ACTH stimulation test returned to normal after withdrawal of the budesonide.

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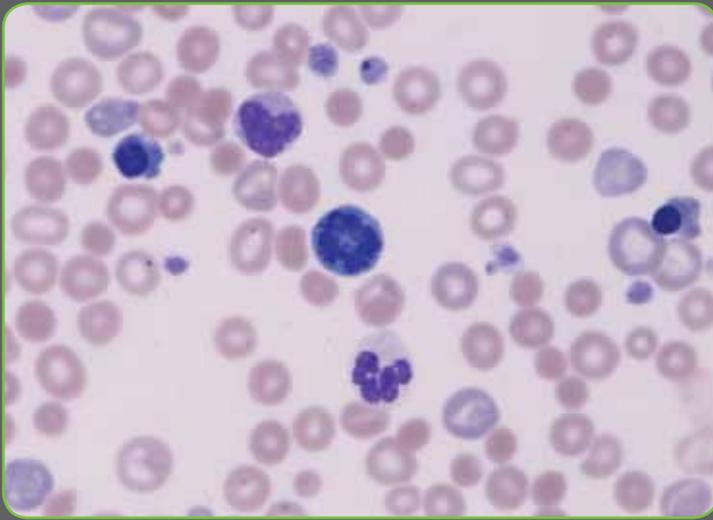
Vetnostics

What is
your diagnosis?

Figure 1: Wright's stained blood film (x100 oil)

In this edition of 'What's your diagnosis?', we have a blood film from a dog which presented collapsed and with pale mucous membranes.

Amongst other significant changes, the full blood count (FBC) revealed a markedly-severe regenerative anaemia. In the image to the left (*figure 1*), what abnormalities are evident, specifically what is the atypical basophilic structure in the centre of the image?

Answer...

The structure in question (labelled 1 in *Figure 2*) is a mitotic figure, presumably erythroid lineage given the basophilic cytoplasm, presence of frequent normoblasts (labelled 2) and further film findings (as described forthwith). This was a case of immune-mediated haemolytic anaemia with evidence of increased polychromasia, increased anisocytosis and spherocytosis on blood film exam (evident in *Figures 1 and 2*). In the absence of haematopoietic neoplasia, the finding of mitotic figures in the peripheral circulation is very unusual however was likely a component of a regenerative erythroid bone marrow response to haemolytic anaemia in this case.
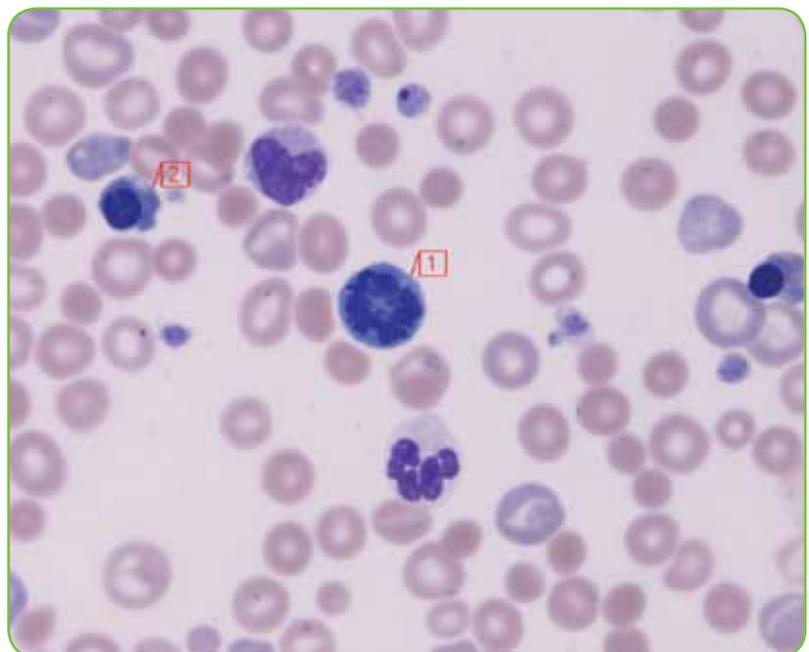

Figure 2: Same case as in figure 1. Number 1 indicates a mitotic figure (presumably erythroid lineage) and number 2 a normoblast. (Wright's stain, x100 oil)

The most difficult and frustrating diagnoses

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CHRONIC DERMATITIS

I am often frustrated with certain skin cases in my ability (or lack thereof!) to reach a specific diagnosis. This is never truer when the clinical history indicates "chronic dermatitis", after various treatments have been trialled and second opinions rendered. Nearly every time I get frustrated reading these cases I'm reminded that exasperation with these very unusual and/or recurrent diseases also involves you as the veterinarian and the owner. Everyone wants an answer yesterday and the best way to get there is to biopsy, right? Wrong! The problem from my perspective is that the more chronic the skin disease, the less specific are the critical-for-diagnosis histologic signposts. While not always an issue with every biopsy, appropriate sample selection and collection plus patient history assume greater significance in these cases, influencing my ability to reach a diagnosis (remember: garbage in = garbage out). Help me to help you by…always taking at least three biopsy samples, preferably 6mm punches; never submit one punch biopsy; never biopsy the edge of a lesion unless the other samples submitted are from the centre of lesions (exception: ulcerative skin disease); in cases of alopecia, biopsy in the worst areas of hair loss; and finally, give me a complete history that includes whether steroid therapy has been used. So, by all means recommend biopsy to your clients, but please temper your promise of a precise diagnosis so I'm not made to look ignorant when the result comes back as equivocal.

Next Newsletter…Hepatic disease

Please note that we have recently released a new Vetnostics Stores Supplies and Blood Collection Tube Guide to simplify stores ordering. Please contact the lab if you have not received and/or require a further copy of the guide. Additionally, included in this newsletter is a copy of our current Stores Order Form (replacing all previous versions) for your use. Many thanks.