Humeral Condylar Disorders in the French Bulldog

Feline Infectious Peritonitis (FIP) – now a treatable disease

Congenital Goitreous Hypothyroidism in a Kitten

Pancreatitis in Cats
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FROM THE DIRECTOR

Like many people, I am a fan of cookbooks. I like to flick through them, be inspired by a photograph and go on to prepare the dish. I make notes in the margins of adaptations I made that may or may not have worked. Sometimes I’ll remember a previously loved dish I’ve not made for ages and go searching through my books, with a mental image of a picture or page placement but not recalling the recipe name.

For me, the C&T in print is a bit like a recipe book. I have, on quite a few occasions, been presented with a case that the pattern-seeking recesses of my brain recognise as having encountered in some way before—and I’ve gone flicking through past editions of C&T looking for the particular photo I recall to locate the article and treatment info. For me, the digital version just doesn’t activate that same memory trigger.

With our brains hard-wired to make connections, I also often get that feeling on reading the C&T of recognising a presentation—usually a difficult or peculiar one—that I’ve seen in the past. Hopefully, it elicits the ‘I’ve treated a case like that before!’ feeling rather than the slightly deflating ‘oooh, that’s probably what was going on in that patient I saw a few years ago…’. Either way, it’s a learning experience. Speaking of connections, I find it a striking coincidence that both of the cats with a similar presentation of skin necrosis in the perineal area are called ‘Louis’ (but maybe that’s just me?).

So, it’s exciting to have the printed C&T back in clinics, homes and cars. The FIP article—and the concept of viewing this as a treatable disease—is a ‘must read’ for anyone in small animal practice. As is the discussion of yet another unfortunate breed disposition in French bulldogs. And for pictures that will remain etched in your memory – well, try forgetting Dinky and his penis. We’ve also introduced a new section—‘Specialists’ Corner’—where you can get to know a little more about a contributing specialist.

Happy reading!
Simone
FELINE INFECTIONOUS PERITONITIS (FIP)—NOW A TREATABLE DISEASE

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Clinical bottom line
Recent advances in anti-viral therapy are showing that effective treatment for FIP is no longer a desperate hope, but a reality. The search for reliable and efficacious treatment for cats suffering from FIP has eluded our profession since it was first documented in the 1960s. Many treatment approaches have been explored and have, until recently, been ineffective. In 2018, the feline medicine world took note as published studies on the nucleoside analogue GS-441524 (Murphy et al. 2018; Pedersen et al, 2019) and protease inhibitor GC 376 (Pedersen et al, 2018) showed disease inhibition and remission states in cats with confirmed FIP. Currently, these products are not legally available for cats with FIP; however, other potential antiviral drugs, namely another nucleoside analogue Remdesivir and the antimalarial drug mefloquine are currently registered in Australia with the Therapeutic Good Administration (TGA). Remdesivir has shown great promise in the field, with an increasing number of treatment successes cured within Australia (now >100 cases). Both drugs are now the focus of prospective treatment trials through the Sydney School of Veterinary Science. These studies aim to further investigate the short- and long-term outcomes of both remdesivir and mefloquine as FIP therapeutics in formal in vivo clinical trials and are actively recruiting new cases of confirmed FIP Australia wide.

Introduction
Feline infectious peritonitis (FIP) is a fatal immune-mediated disease in cats caused by virulent pathotypes of feline coronavirus (FCoV) known as feline infectious peritonitis virus (FIPV). FIPV has an increased ability to replicate in large quantities in monocytes and macrophages. There are two major forms of FIP: effusive (wet) form and non-effusive (dry) form. The effusive form is characterized by rapid onset and widespread vasculitis with fibrinous-granulomatous serositis, with protein-rich effusions in the thoracic or abdominal cavities. The non-effusive form is typified by pyogranulomatous lesions found within multiple body organs. The median life expectancy ranges from days to weeks in the effusive form, and weeks to months in the non-effusive form. Once disease commences, few cats are reported to survive.

In reviewing previous FIP treatments, the major challenge lies in the lack of well-controlled clinical trials and the use of some treatments that are mostly based on in vitro studies. In addition, some older literature describes potential treatment success in cases without a confirmed diagnosis of FIP. Furthermore, despite recent breakthroughs in treatment of FIP using a nucleoside analogue (GS-441524) and protease inhibitor (GC 376), these products are not registered for veterinary use and are not commercially available. Even with recent access to remdesivir (which is metabolised into GS-441524), the high cost for these newer antiviral agents mean that some treatments may remain cost prohibitive for some clients.

Treatment Approaches for FIP
Eliminating feline coronavirus from our global population of domestic and wild cats has not been possible to date. Similarly, the development of successful vaccinations has been unrewarding although the recent global pandemic in humans caused by SARS CoV2 has provided a framework for new approaches to coronavirus vaccination. When faced with an infection that we cannot currently eradicate, effective treatment is our current focus.

When considering treatment approaches to FIP, there are three major elements to consider: firstly, the virus endogenously mutates and acquires an increased capacity to replicate within macrophages/monocytes; secondly, there is an ineffective host immune response and failure to clear the virus from infected macrophages; and thirdly, the immune-mediated inflammatory cascade that ensues in response to activated macrophages results in the pathology seen with FIP.

The three main targets for therapeutic intervention therefore have aimed to:

1. Directly inhibit the viral replication using targeted antiviral drugs (e.g. nucleoside analogues and protease inhibitors);
Improve the efficacy of the host’s immune response with immunomodulators used to stimulate the patient’s immune system non-specifically (e.g. interferons and polypropenyl immunostimulants); and

Dampen the immune-mediated response with immunosuppressive drugs, to attempt to ameliorate the clinical signs (e.g. corticosteroids and cyclosporin).

The following describes the pros and cons of the immunomodulators, immunosuppressive drugs and antiviral drugs.

**Antiviral therapeutics**

Antiviral treatments aim to target cellular mechanisms to inhibit viral replication or a specific aspect of virus activity related to infection.

**GS-441524 and Remdesivir (GS-5734)**

Nucleoside analogues GS-441524 (not currently legal or commercially available in Australia) and its prodrug Remdesivir (GS-5734, legal and accessible via BOVA Compounding Pharmacy), were developed to treat human viral infections such as Middle East respiratory syndrome virus and Ebola virus, for which they have subsequently been found to be ineffective. Fortunately for the veterinary world, these drugs are now showing efficacy against FIPV.

Nucleoside analogues mimic endogenous nucleosides and become incorporated into viral DNA and RNA, thus inhibiting viral replication. For GS-441524, its active metabolite acts as a competitor of the natural nucleoside triphosphates in viral RNA synthesis by inhibiting RNA-dependent RNA polymerase-mediated transcription (Murphy et al, 2018). Murphy et al, 2018 reported effective inhibition of FIPV in vitro using GS-441524. In this study, the pharmacokinetics of GS-441524 in cats was also determined. This drug was then tested in experimentally infected FIP cats and led to the rapid reversal of clinical signs and remission in all 10 cats, without apparent signs of toxicity. In the following year, Pedersen et al, 2019 reported the use of GS-441524 in cats with naturally occurring FIP. In this study, 26 cats completed the 12-week course of treatment with 18 out of 26 cats going into remission after the first round of treatment. Eight cats relapsed, and five of the eight cats were treated at a higher dosage (from 2.0 to 4.0mg/kg q24 h). These five cats then went into remission. Three of the eight cats were treated at the same dosage, however, relapsed. One cat was euthanized while the other two relapsed cats were treated a third time at higher dosage. Overall, the study produced 25 long-term survivors. However, a higher dosage of GS-441524 was required to treat cats with neurological FIP (Dickinson et al, 2020).

Although GS-441524 has shown to be successful in treatment of FIP, it is not currently registered for veterinary use and is not approved by the Australian Pesticides and Veterinary Medicines Authority (APVMA). It has been available on the ‘black market’. However, all Veterinary Practitioners Boards in Australia have warned that clients and veterinarians involved in the unlawful importation of unregistered veterinary chemicals may be subject to significant financial, civil or criminal penalties. Despite this, some owners have accessed black market channels via Facebook groups and inject their animals unsupervised at home.

One of the rare positives to come out of the COVID-19 global pandemic is the chain of events that has resulted in Remdesivir (GS-5734) now being registered in Australia by the TGA. Off-label use of this drug is now available to veterinarians via BOVA Compounding Pharmacy. Reports of its use have been very positive in cats with naturally occurring FIP, with increasing numbers of Australian cats responding incredibly well, even those with wet FIP.

Dose rate starts at 10mg/kg/day SC or IV for the first 4 days with close monitoring and hospitalization recommended. Then 6mg/kg/day SC for 12 weeks.
In neurological cases, keeping the dose at 10mg/kg is advised. Discussions are occurring to determine if these drugs are suitable and feasible for oral administration (Xie et al., 2021 in submission).

**GC 376**

The coronavirus 3C-like protease processes viral polyproteins into functional proteins. As this is an essential step for virus replication, inhibition of 3C-like protease is an attractive target for therapeutic intervention. In Kim et al. 2016, there was a reduction in viral titre and recovery in experimental infected FIP cats after GC 376 treatment. A field trial (Pedersen et al., 2018) reported inhibition of disease development and remission after the course of GC 376 treatment with seven out of 20 cats surviving after the treatment course. A minimum course of 12 weeks was recommended in this study. Side effects of this product were pain and subcutaneous inflammation at injection site, retention of deciduous teeth and delayed development of adult teeth. This drug remains experimental and is not registered for veterinary use, nor approved by the TGA.

**Other antiviral compounds**

Other compounds have been trialed for their antiviral activity against FIPV. McDonagh et al. 2014 reported inhibition of FIPV replication by three compounds: chloroquine, mefloquine and hexamethylene amiloride inhibit viral load of FIPV. Although chloroquine inhibits FIPV replication in vitro, this drug had poor antiviral efficacy in experimentally induced FIP cats, together with its side effect of causing increase in alanine aminotransferase (ALT) in cats which could suggest hepatocellular injury (Takano et al., 2013), suggesting that this drug is a less than an ideal candidate for FIP treatment. A closely related drug hydroxychloroquine (Takano et al., 2020) when tested in vitro showed increased antiviral activity against FIPV infection when used with recombinant feline interferon-omega. Hexamethylene amiloride is yet to be further investigated and mefloquine is described in more detail below.

Itraconazole has demonstrated antiviral activity against FCoV (Takano et al., 2019). A case report describing the use of itraconazole (Kameshima et al., 2020) showed initial reduction in pleural effusion when treated with prednisolone, but with subsequent neurological manifestations and was eventually euthanized due to status epilepticus after 38 days of treatment. A case series (Doki et al., 2020) describing itraconazole use in three cats with experimental induced FIP resulted in two of the three cats showing improvement in clinical signs, increase in lymphocyte count, and a decrease in alpha-1-acid glycoprotein after treatment. Further investigation of this drug is needed to determine its long-term efficacy in naturally occurring FIP.

Dunowska et al. (2021) found dose dependent inhibition of FIPV in vitro using doxycycline 24 hours post infection supporting the notion of future clinical trials in this registered antimicrobial. Furthermore, there were also other agents that had shown to have in vitro antiviral activity towards FCoV, such as cholesterol transport inhibitor U18666A, a vacuolar ATPase blocker (diphyllin) and its nanoformulation, a circular triple helix forming oligonucleotide RNA and concurrent use of Galanthus nivalis agglutinin and nelfinavir. There are currently no in vivo studies reported with these compounds.

Mefloquine

As mefloquine is a human antimalarial prophylactic and treatment, it is registered by TGA, is commercially available in Australia and is easy to access. The pharmacokinetic profile, plasma protein binding and hepatic metabolism of mefloquine in cats have been investigated (Izes et al., 2020, Izes et al., 2020, Yu et al., 2020). In Yu et al. 2020, when mefloquine was given at 10-12.5mg/kg orally twice...
weekly, a plasma concentration exceeding the dose required to inhibit FIPV in vitro was reached. Side effects of the drug included vomiting when given without food. Mefloquine could be used as a potential treatment for cats with FIP. It is early days yet, but some local cats have responded to oral mefloquine medication. This is also available via Bova compounding pharmacy as an off label use of this TGA registered drug.

Our research group is currently conducting a clinical trial to investigate the efficacy of mefloquine with or without feline omega interferon (Virbagen®) in FIP-confirmed cats.

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Product to stop faecal FCoV shedding
Besides FIP treatment, it is worth mentioning a product that has been reported to stop faecal FCoV shedding. Mutian®X is a synthetic adenosine analogue described by Addie et al 2020 to stop faecal FCoV shedding in chronically infected cats. Its potential use in treatment of FIP is being investigated. This medication is not currently registered for use in Australia.

FIP is now generally considered as a potentially treatable disease, but there are some caveats. Although a nucleoside analogue (GS-441524) and protease inhibitor (GC 376) have been successful in treating cats with FIP, these products are not registered for veterinary use. Whilst pending the regulatory approval of these agents globally and through our APVMA, other commercially available agents, such as remdesivir and/or mefloquine, need to be investigated more thoroughly through in vivo studies to assess their true potential as FIP antivirals.

Immunomodulators
Interferons and polyprenyl immunostimulant are common types of immunomodulators in some countries. Interferons have been used for immune response modification with limited success in FIP. There are two types of interferons that have been used on FIP cases to date: human interferon-alpha and feline interferon-omega. Subcutaneous injections of human interferon-alpha had little effect in an experiment trial of FIP (Bolcskei & Bilkei, 1995). Feline interferon-omega is licensed in some European countries, as well as Australia and Japan. This product was initially reported in an uncontrolled trial with glucocorticoids (Ishida et al, 2004). The study yielded promising results with 67% of cats achieving complete or partial remission; however, some cats did not have a confirmed diagnosis of FIP. Ritz and colleagues (Ritz et al, 2007) showed that there was no significant difference in survival and quality of life in cats treated with feline interferon-omega versus a placebo group. However, the efficacy of interferon in this study was difficult to assess because all cats in this study were treated with glucocorticoids and antibiotics concurrently. Polyprenyl immunostimulant is a plant extract that enhances T-lymphocytes to promote cell-mediated immunity. There has been some reported success in the treatment of non-effusive FIP with this product. Polyprenyl immunostimulant was initially reported in Legendre & Bartges, 2009 and long-term survivals were seen in three cats with dry form of FIP using this product. A field trial (Legendre et al, 2017) with 60 cats also showed promising results, with eight cats surviving longer than 200 days and four cats surviving more than 300 days. Authors of this study did not believe that this product was efficacious for severe or effusive form of FIP.

Another example of an immunomodulator is anti-feline TNF-alpha monoclonal antibody. Doki et al, 2016 reported a slightly prolonged survival time in two out of three cats using this product compared to a controlled group (65 days compared to 25–27 days in controlled group). Propionibacterium acnes, a gram-positive bacterium, has been used as an immunomodulator; however, its use has been ineffective (Weiss et al, 1990).

Immunosuppressive drugs
Glucocorticoids are the most common type of immunosuppressive drugs used for FIP treatment. As FIP is an immune mediated disease caused by FIPV, the use of a glucocorticoid as an immunosuppressive drug dampens the immune response and reduces the clinical signs of infected cats. Despite the popularity of its use over many decades, glucocorticoids remain a palliative treatment options that improve the patients’ clinical signs and quality of life temporarily, but are not curative for infected cats as the viral replication continues unabated.

Other immunosuppressive drugs including cytokine inhibitors (pentoxifylline and propentofylline) (Fischer et al, 2011), cyclophosphamide (Bocskei & Bilkei 1995), cyclosporin A, chlorambucil (Addie et al, 2009, Bilkei 1988) and thromboxane synthesis inhibitor (Ozagrel hydrochloride) (Watarai et al, 1998) and all have been reported as potential FIP treatments. Fischer et al, 2011 investigated propentofylline in FIP
infected cats. However, as glucocorticoids were co-administered with propentofylline in this study, it is difficult to determine the efficacy of propentofylline as a single therapeutic agent. Similarly, Bozskei & Bilkei 1995 used cyclophosphamide with prednisolone and ampicillin, whereby 76 out of 151 cats were regarded as ‘healthy’ at the end of the study; however, not all cats had confirmed diagnosis of FIP. The use of chlorambucil was mainly based on anecdotal evidence (Addie et al., 2009). Watari et al., 1998 explored a thromboxane synthesis inhibitor (Ozagrel hydrochloride) and showed beneficial effect in two cats; however, FIP was not confirmed in these cats.

Cyclosporin A has been reported to inhibit FCoV replication in vitro (Pfefferle et al., 2011, Tanaka et al., 2012, Tanaka et al., 2013). Cyclosporin A was used to treat a cat with FIP (Tanaka et al., 2015) with a reduction in pleural effusion and viral load seen initially but subsequent death due to respiratory failure from relapsed pleural effusion on day 264. Hence, the efficacy of cyclosporin A in vivo remains questionable.

References


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DIAGNOSIS AND MANAGEMENT OF CANINE STERILE NODULAR PANNICULITIS

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Introduction
Sterile nodular panniculitis is an inflammatory process characterised by nodular inflammation of the subcutaneous fat. Whilst the defining feature is clear in established cases, in the early stages it may not present clearly with these fatty changes.

Such was the case with 'Ash', a 4-year-old desexed female red heeler. Subsequently, as is the case with many complex chronic diseases, the approach to her case was a very involved period of investigation to identify the disease as well as possible underlying pathologies, before varying success with management and monitoring.

Presentation
Ash first presented in March 2020 following intermittent lameness on her right hind leg. Examination revealed some right thigh muscle atrophy and possible discomfort in her right stifle. Assessment concluded most likely cruciate disease, she was placed on meloxicam and nutraceuticals (Antinol) and asked to monitor for any deterioration in clinical signs. Ash re-presented the next month having responded well to treatment, but she now seemed painful on her left hind leg. As Ash is a nervous patient, it was recommended that she be booked in for sedation, examination and radiographs following a general health profile (GHP).

In May, Ash presented for further diagnostic workup. The GHP revealed mild blood abnormalities:

- leukogram: mild stress response
- very mildly low Na: K (25.7)
- mildly elevated P 2.2mmol/L (0.8-2.1)
- elevated AST 136 IU/L (18-80)
- elevated CK 1311 (73-510)

At the time, these changes were thought to be related in part to artifactual changes secondary to haemolysis and to mild muscle trauma. When the results were relayed to Ash’s owner, she mentioned that Ash seemed somewhat dull, her appetite was intermittently reduced, and she was occasionally aggressive towards her companion dogs. The owner had stopped meloxicam treatment feeling this may have contributed towards these changes.

There was a period of delay before Ash was admitted for sedation and imaging, during this time it was noted that she had developed a lump on her back and chest. It was difficult to determine how long these had been present. Both were approximately 40mm in diameter; the dorsal mass was midline between her shoulder blades presenting the possibility of an injection reaction; the ventral mass was poorly defined being obscured by the fat of the cranioventral thorax. In-house cytology showed the FNA to be fatty in nature with multiple nucleated adipocytes. External cytology diagnosis was lipoma.

A few days following this, our patient had become lethargic and inappetent. On the morning of her re-presenting she had been given meloxicam again as she seemed sore. On examination, Ash had a
temperature of 40.3°C (anxiety, pain, infection, inflammation/meloxicam reaction??). Under sedation it was determined that she had restricted range of movement in both hips, especially on abduction, demonstrating some discomfort on manipulation and had developed some atrophy of the left thigh musculature. Pennhip rads were not taken, ventrodorsal hip extension radiographs revealed shallow acetubulae and flattening of the femoral heads. A presumptive diagnosis of hip dysplasia and mild degenerative osteoarthritis was made.

At this point in time, it was felt that Ash may not be tolerating the meloxicam well and her signs could be due to hip pain. After a 48hr ‘wash-out’ period, she was started on a standard 4 injection course of pentosan, firocoxib sid and omeprazole bid. Her owner was asked to take Ash’s temperature daily at home where she was more relaxed. Over the course of the following week, Ash’s condition did not improve. At home she was intermittently pyrexic, dull, her appetite was variable, and she had vomited up either food or fluid on several occasions.

Further investigation was clearly required, so Ash was booked in for an ultrasound. Her ultrasound was not revealing; our in-house ultrasonographer could find no pathologies with the only possibly significant finding being that she was extremely difficult to ultrasound; visualisation was uniformly poor. At this point, it was also discovered that there were now multiple firm subcutaneous (SC) masses and Ash’s SC fat felt firm and slightly nodular. It was recommended that Ash return for biopsies.

Prior to surgery, PGA bloods were performed. There were no remarkable changes but of note her BUN was now 9.9mmol/L (previously 4.8) and SDMA 7 (ref: 2.5-10) and her ALP was 441 (previously 46, ref: 23-212). Ash had now stopped NSAIDs prior to surgery allowing for the possibility of her requiring corticosteroids depending on histo results. The biopsy results all showed lesions with a similar histological appearance with a conclusion of multifocal granulomatous dermatitis/panniculitis. No microorganisms were seen in the examined sections.

A diagnosis of nodular panniculitis was made; we now began the process of eliminating causal infectious agents. With the benefit of retrospection, deep fat biopsies for culture should have been made at the time that samples were collected for histo. Fortunately, these samples were able to be harvested under sedation and local anaesthetic. Anaerobic/aerobic culture results were all negative as were fungal cultures and mycoplasma PCR results that were received over the ensuing 15 days. We could be confident now that we had a case of sterile nodular panniculitis.

Treatment and Response

It was now early June, almost 3 months from when Ash first presented. Over this time, she had lost nearly 3kg (13% BW). Although we did not have comprehensive understanding of all the disease process occurring, we had achieved a diagnosis. Once the initial diagnosis of panniculitis was made, Ash was started on prednisolone 1mg/kg bid. Unfortunately, within days of starting the prednisolone, Ash became increasingly inappetent, was vomiting more frequently, was increasingly dull and at times, ataxic. She was admitted to the clinic for blood work and nursing care.

Significant findings from her blood work on the 11th of June as follows:

- PCV 36.2% ref 37–62% mild anaemia of chronic disease/liver disease
- Leukogram: stress response
- BUN 15.5 mmol/L ref 2.5-9.6 (on 4/5 4.8, on 2/6 9.9) uraemia due to pre-renal causes (dehydration, cachexia) or hyperCa
- Ca 3.55mmol/L ref 1.98-3 (on 4/5 2.5 and 2/6 not performed). Possibly due to reduced GFR, granulomatous inflammation but concerns re malignancy. Cortisone and fluids should help reduce level
- ALT 126 U/L ref 10-125 (on 4/5 46 and 2/6 79)
- ALP 589 U/L ref 23-212 (on 4/5 46 and 2/6 441). May be exacerbated by prednisolone therapy but started prior to this.
- Previous elevations of CK and AST were not sustained
- Spec CPL normal

Based on the above assessment, our patient was started on intravenous fluid therapy, buprenorphine and maropitant. Her prednisolone...
was reduced to 1mg/kg sid with the plan to increase again within the next few days if her health allowed. Within hours, Ash appeared brighter. Over the following days, this improvement proved to be sustained with improvements in her demeanor, appetite, vomiting, temperature and weight gain. The nodular feel of her SC fat was slightly better, but the original thoracic masses remained the same. Repeat bloods were undertaken. Significant findings from blood work 25/7:

- **BUN 12.5 mmol/L** (on 11/6 15.5)
- **Ca 3.73 mmol/L** (on 11/6 3.55)
- **ALP 126 U/L** (on 11/6 589)
- The total Ca was followed up with an ionised Ca 2.07 mmol/L (1.25-1.5)

The assessment at this point was that the hypercalcemia was high enough and sustained enough to risk damage to the kidneys plus other systemic pathologies. Plans were made for urine cytology looking for calcium oxalate crystals; none were found. A process of elimination then ensued to determine the cause of the hypercalcemia.

1. Hypercalcemia of malignancy—Plans made for repeat abdominal US, chest rads and lymph node (LN) biopsies or fine needle aspirate (FNA)
2. Granulomatous inflammation—generally milder hypercalcemia and should reduce not increase as condition improves
3. Primary hyperparathyroidism—possible but rare and would be an unfortunate unrelated co-morbidity. Plus, phosphorus levels normal
4. Addison’s—some suggestive presenting signs but ambiguous response to cortisone and bloods had shown a mild stress response, normal K and ratio >25

When Ash returned for these further diagnostic procedures, her external LN were found to be small and difficult to palpate and, at times, impossible to distinguish from surrounding fat. Chest radiographs were all clear. Abdominal U/S was much easier than previously. The only detected change was that the consolidation of abnormal fat in the mesentery was found to be more widespread throughout the entire abdomen. Cranial mesenteric lymph nodes were normal size. Urine was collected for culture and susceptibility and blood for repeat ionised Calcium (Ca).

Ionised Ca was persistent at 2.07 mmol/L (previously 2.07 so checks were made that this was indeed correct). Urine culture revealed mod growth *E. coli*, susceptible to amoxyclav. Lymph node cytology was non-diagnostic.

On the advice of the IDEXX pathologists, I contacted Richard Malik for some guidance. Should we keep looking for a malignant disease process or just focus on treating the sterile nodular panniculitis? What more could be done with this persistent hypercalcemia? Thankfully, Richard’s response was swift and clear:

1. Start combination therapy of prednisolone with either azathioprine or, preferably, cyclosporin. Likely 2 months of maintenance then try to wean off over another month
2. Increase prednisolone again to 2mg/kg until combination therapy starts
3. If no reduction in Ca after 7 days then add in Fosamx

Following the above directions, Ash soon started on cyclosporin 3mg/kg bid and prednisolone 1mg/kg sid (after a short period on 2mg/kg). Ash remained well in herself during this transition period. Seven days later her ionised Ca was measured to be 1.23 mmol/L (1.25-1.5).

In later July, after two weeks of combination therapy, the prednisolone was reduced from 20mg to 10mg. Within a few days, the fatty lumps had become more obvious. Although disappointed by this setback, bloods performed at the time were all within normal range except for BUN of 12.4. Ash’s significant weight gain was also noted; she was now 32kg, 10kg heavier than when she was at her lightest. Ash’s prednisolone dose was increased again to 1.5mg/kg and her cyclosporine dose increased to maintain an accurate and appropriate mg/kg dose. Dietary and management advice was given to help Ash’s owners control her weight.

Ash was allowed to settle longer on the combination therapy; it was now early September.
Ash seemed happy at home although at times stiff and sore in the back end. Repeat bloods showed things were going OK with all parameters within normal limits except cholesterol 10.42mmol/L (ref 2.84-8.26). With Richard’s recommendation; ‘the cyclosporin needs to do the heavy lifting, not the pred’; the prednisolone dose was to be halved every two weeks. He suggested performing a trough cyclosporin to help guide dosing. Ash would also commence a pentosan course for her hip osteoarthritis.

The trough cyclosporin (Vetnostics) returned as 88µg/L, well short of the 300-500 that is considered therapeutic. A couple of weeks after starting cyclosporin, based on financial considerations, we had decided to change Ash from Atopica® to a compounded product. Because we had not previously done an assay, I had no way of determining if the change in formula had any effect on absorption. Richard suggested increasing the dose from 3 up to 4mg/kg bid; this would not push us up to 500 but may be more effective in controlling the disease and allow Ash to stop the prednisolone. At the same time as blood collection for the cyclosporin, blood was collected for a canine C-reactive protein (IDEXX); I had not performed one of these before and was interested to know if it would give us a more objective way of monitoring Ash’s condition. This returned at 4mg/L (ref <10).

**Conclusion**

By early September, Ash was being managed on a cyclosporin mono therapy of 4mg/kg bid. Her subcutaneous fat still had a slightly nodular feel but there were no obvious focal fatty masses. As Ash was weaned off the prednisolone, her owner initially reported her to be less energetic and was experiencing an obvious decrease in appetite; these signs have since settled. Repeat blood work has generally been favourable with only mild increase in ALT 136U/L and CHOL 10.42mmol/L, mild lymphopenia 0.76 (x 10^9 /L). Her Ca is within normal range as were all other measurements.

Since ceasing prednisolone therapy, Ash has exhibited more obvious hind limb discomfort. Failing to respond to a pentosan course and gabapentin therapy, guidance again was sought from Richard Malik. Based on his advice, Ash is about to commence a trial of a low dose of meloxicam (0.05mg/kg) then a trial of paracetamol (or paracetamol-meloxicam bitherapy) if meloxicam fails to provide sufficient analgesia. The longer-term plan is to keep Ash on cyclosporin for approximately 6 months and until full resolution of clinical signs.

**Discussion**

Panniculitis is an uncommon condition in dogs characterised by inflammation of fat. Predominantly, it is the subcutaneous fat that is affected but abdominal fat, intramedullary fat and epidural fat can also be inflamed. As outlined by O’Kell et al (2010), sterile nodular panniculitis is an idiopathic condition often secondary to other disease processes such as pancreatic nodular hyperplasia, pancreatitis, pancreatic adenocarcinomas, other abdominal neoplasia and autoimmune conditions such as systemic lupus erythematosus and rheumatoid arthritis. Panniculitis more generally can develop as a result of infectious agents (viral, bacterial, fungal, protozoal and parasitic), drug/injection reaction, insect bite reactions, vitamin E deficiency and following foreign body or trauma. It can also occur as a primary process.

Dachshunds, Poodles, Pomeranians and Chihuahuas appear over represented in affected dog breeds.¹ The study of Contrary (2015) did not find that there was any greater likelihood of these breeds not suffering from any co-morbidities which may have otherwise suggested a genetic predisposition. There is no age or sex predilection.

Presentation can vary depending on severity, causal and associative disease processes. As described by O’Kell et al, most often the condition is associated with malaise, pyrexia, and inappetence. Fewer cases also present with swollen joints and shifting lameness, skin lesions and discharging sinus tracts, heart murmurs and abdominal masses. All will have singular, multiple or dispersed subcutaneous nodular changes ranging in size from 0.5-7cm in diameter. ⁸

Common laboratory changes include elevations in alkaline phosphatase (ALP), hypoglycaemia, hypocalcaemia, hypoalbuminemia and a leukocytosis with mature neutrophilia. Less common abnormalities include increases in alanine aminotransferase (ALT) and (AST), hypercalcaemia, other electrolyte derangements, increases and decreases in blood urea nitrogen (BUN), increased serum creatinine and increased blood clotting times.²

Immunosuppressive therapies such as prednisolone 0.5-2mg/kg, cyclosporin 3-6g/kg, azathioprine and chlorambucil as either mono or combination therapies.¹ ¹¹ can be successful in management of the condition. Ultimate response to treatment may be reliant upon severity and presence of primary condition or co-morbidities.¹

Cyclosporin is a potent immunosuppressive
drug that has been shown to provide a high level of success in treating a range of autoimmune diseases. It has a marked variation in oral bioavailability\textsuperscript{11,12} and this variation is further exacerbated by different formulations. The presence of food will act to diminish absorption and increase variation in bioavailability.\textsuperscript{13} For this reason it is recommended to be administered two hours prior to feeding.

Cyclosporin is a lipophilic compound; originally available as Sandimmune, a vegetable oil-based formula. More recent advances have seen ultramicronised preparations become available; initially as Neoral\textsuperscript{®} on the human market and Atopica\textsuperscript{®} approved for use in dogs and cats. This ultramicronised formula has led to better and more predictable absorption. The use of compounded ultramicronised products may not have this same level of bioavailability.\textsuperscript{11} Because therapeutic drug monitoring was not undertaken in Ash prior to changing from Atopica\textsuperscript{®} to a compounded formula, it could not be determined if this is a causal factor in Ash’s low plasma concentration. On later discussions with the compounding pharmacist, the compounded formula of the cyclosporine used on Ash was found to be a non-ultramicronised, triglyceride-based formula. The decision to swap formulas was based on economics, the compounded suspension being 25\% of the cost of the branded ultramicronised preparation. It is possible that the lower bioavailability of the fat-based suspension is as little as a quarter of the ultramicronised one with the trough blood concentration achieved being \textit{88µg/L}, approximately a quarter of what is considered therapeutic levels (300-500µg/L).

The expense of long-term high doses of cyclosporin have led to the decision to use a compounded product in this particular patient. Once Ash had further stabilised on cyclosporin monotherapy, attempts are to be made at increasing absorption through the concurrent use of other pharmaceuticals known to enhance absorption or decrease clearance\textsuperscript{11,12}. For this, cimetidine\textsuperscript{11}, ketoconazole\textsuperscript{10} and grapefruit juice are known to have positive effects. Ongoing therapeutic drug monitoring will be required to monitor the effectiveness of this approach; the cost of therapeutic drug monitoring (TDM) may negate any financial benefit of the cheaper formula; such is our dilemma. This is an ongoing case; whilst we have come far with Ash, we clearly have some way to go.

Acknowledgement
During this time, I have had the guidance of Richard Malik, for which I am very grateful. I’ve also enjoyed his quick and punchy replies. As with many multiplex cases, Ash’s care was given by the whole team of vets and nurses I work with at Manuka Veterinary Hospital; this was in every respect—professional advice and input, undertaking of procedures, reporting of results and nursing care. Also to Ash’s owner, Elia, she has been a delight to work with.

Postscript
I hadn’t seen Ash for some months as life had got rather busy for her owner. Ash had slowly kept improving and had been living disease free for 4-5mths; the plan had been to slowly wean her off cyclosporin by reducing the dose by 25\% every 2 months. However, because of unrelated factors, her owner had left her on a low dose of compounded cyclosporin of 4mg/kg once daily. Despite this, two weeks ago, palpable subcutaneous changes were detected along with the occasional vomit and more irritable behaviour. She has now started back onto cyclosporin & prednisolone combination therapy and Atopica has been ordered-in to trial a replacement of the compounded product. A trough cyclosporin concentration measure will be performed once she has stabilised on the new compound.

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Small

PRE-BREEDING VAGINAL CULTURE SWABS: DISPELLING THE MYTHS

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The vagina of the bitch is not a sterile environment. Open to the outside world, a thriving microbial ecosystem exists to maintain the health of this region. Despite this, many owners of breeding bitches and studs still request vaginal cultures to be obtained by a veterinarian prior to breeding on the premise of assessing prospective fertility or present infections.

The following summary will assist veterinarians in explaining to breeders the major reasons that vaginal culture is commonly NOT a helpful diagnostic test, thereby preventing unnecessary antibiotic therapy which can do more harm than good.

1. Low specificity of culture results

Normal, healthy bitches harbour aerobic bacteria throughout the vagina. Proestrous cultures from healthy bitches of the caudal vagina will result in an average of 2.3 isolates grown, and an average of 1 isolate if taken from the cranial vagina. It is not a sterile environment. Therefore, returning a positive vaginal culture result is very common (around 60-90%) and is not confirmatory of an infectious process occurring that requires treatment.

2. Treatment of ‘positive’ results will alter the true, healthy microbiome

As veterinarians we are expected to interpret vaginal culture results when they are requested by the owner; however, in the absence of clinical signs indicative of infection (i.e. purulent vaginal discharge) treatment is superfluous and can lead to negative results including any of the following:

- An imbalance in normal, protective vaginal commensal flora
Pathogenic bacterial overgrowth and potential increased risk of most likely metritis/pyometra

- Disturbance of the host metabolism and vitamin absorption
- Alteration of susceptibility to host infections
- Overgrowth of yeast and/or Clostridium difficile
- Extrapolating from human studies, neonates can be affected by gestational antibiotics with increased susceptibility to infection and antibiotic resistance.3,4

3. Culture swab of the vagina is not representative of the true microbiome

Nearly all published data pertaining to flora of the canine vagina has previously been obtained using culture-based sample techniques (i.e. culture swab aerobic culture and sensitivity). Though this technique is practical and easy to perform, recent DNA extraction (metagenomic) techniques have shown that they fail to detect >90% of microflora present in the sampled environment.5

To further demonstrate this disparity, the most commonly reported bacteria cultured from the canine vagina via culture swab techniques are Enterococcus, Streptococcus, Pasteurella and Escherichia genera,3 yet further analysis with DNA extraction did not confirm a single one of these organisms in the top 5 genera represented.6 Furthermore, culture results have been shown to significantly underestimate bacterial diversity and concurrently overestimate the role of isolated bacteria as they are more ‘visible’.7

4. No correlation between vaginal and uterine microbiomes

The prevailing reasoning for pre-breeding treatment with antibiotics indicted by vaginal culture results is a correlation between the uterine and vaginal microbiomes. However, despite the uterine microbiome recently being confirmed as non-sterile, there is no association between microbes present in the vagina, and those in the uterus in individual bitches.6,7 Given the ‘goal’ of treatment is to remove bacteria in the uterus that may be affecting fertility, treatment specific to those cultured from the vagina will therefore be unrewarding.

In fact, the culture of Streptococcus spp. in proestrus has shown to negatively correlate with the development of uterine infections, indicating it is likely this organism plays a protective competitive role against more dangerous pathogens affecting fertility of the bitch.3

5. Stud dogs carry the same organisms

A common scenario is for stud dog owners to request bitches be tested prior to a natural mating on the premise that the bitch may infect the stud dog during the breeding. Stud dogs, however, harbour the same organisms as the bitch and exposure to normal vaginal flora is not harmful nor does it have a negative effect on conception.

When is a vaginal swab acceptable and what technique is best?

Despite all of the above, there are circumstances where a vaginal swab IS a necessary component of the diagnostic process! In a bitch presenting with a purulent vaginal discharge thought to originate from the uterus, be it during diestrus (most likely pyometra) or post whelping (most likely...
endometritis), sampling for a cranial vaginal culture is indicated as you have concurrent clinical signs of disease. A concurrent vaginal cytology sample will also allow for assessment for neutrophils and presence of estrogenic superficial cells to aid the diagnostic process.

For the reasons discussed above, sampling for culture requires sample collection of uterine discharge, rather than vaginal discharge (or normal bacterial). Because of this, we want to take our sample from the very cranial aspect of the vagina (i.e. at the cervix; pictured in Figure 1. with yellow star) without contamination from other regions. Contrary to popular belief, the cervix is NOT palpable on digital vaginal examination in the dog. The distance from vulva to cervix ranges from 12 – >36cm depending on the size of the bitch (as a side note, remember this next time you are ‘checking’ if the cervix is open at whelping!).

The best way to facilitate cranial vaginal sampling is through the use of a guarded swab. Given the frequency of this procedure in the horse industry, readily available equine guarded culture swabs (pictured) are the ideal piece of equipment for this process in medium to large breed dogs. Regardless of device used, the goal is to guard the culture swab through its journey through the vulva, vestibule, and vagina and to only expose it for sample collection at the level of the cranial vagina. In the case of the equine culture swab, the swab is contained within the outer tube for its journey, then pushed out for sample collection and returned to the tube for extraction.

Even with the correct technique, commensal contamination can still occur and blur interpretation of cranial vaginal culture results. Additionally, if the bitch is already on antibiotics the results can of course be skewed, so it is important to remember to take the sample prior to starting pre-emptive antibiotic therapy. Results depicting a heavy growth of a single bacteria are very suggestive of this being the causative agent of your discharge and therefore treatment should be aimed at such (based on sensitivity testing). However, if your results return a mixed growth, or a mix of a few commonly cultured bacteria mentioned above, it is less likely that the culture is representative of the uterine cause of infection and likely just contamination. If this occurs, continued use of a broad-spectrum antibiotic (usually amoxicillin clavulanic acid) is recommended if diagnosis of pyometra/endometritis has been confirmed by imaging.

Conclusion

With the few key points above we can educate clients asking for vaginal pre-breeding swabs on why they are not indicated nor beneficial in a normal, healthy bitch. In cases of suspected uterine infection or infertility where cultures may be indicated, care still needs to be taken in interpretation of the results; in general, only a moderate to heavy growth of a single organism is likely to correlate with a potential causative agent.

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Frequency, Stressfulness and Type of Ethically Challenging Situations Encountered by Veterinary Team Members During the COVID-19 Pandemic

Ethically challenging situations (ECS) are common in veterinary settings and can lead to moral stress. However, there is no published information about how a global pandemic affects the frequency and types of ECS encountered by veterinary team members. An online mixed methods survey was developed to determine the frequency, stressfulness and types of ECS experienced by veterinarians, animal health technicians and veterinary nurses since the advent of the global COVID-19 pandemic in March 2020. Responses from 540 veterinary team members from 22 countries were analysed.

HOW DO YOU PROCESS YOUR SURGICAL INSTRUMENTS?

Are there room for savings? Is your current process sustainable? Can processes be improved?

Have you ever really looked at the way your practice processes its sterile equipment?

Do you know what the financial, environmental or labour impacts are of these processes?

Your clinic is likely sterilising Surgical Instruments and implants using at least one of the following methods:

- single-use blue wraps
- single-use paper or sealable plastic pouches
- re-usable cloth drapes
- re-usable rigid sterilisation containers

Single-use wraps are inexpensive and commonly available. However, they are designed to be used once and then discarded. They are easily perforated, therefore compromising sterility.

Single-use paper or sealable plastic pouches are also inexpensive and commonly available. They serve the purpose of allowing small quantities of instruments to be contained and sterilised, but are also discarded after each use and are also easily perforated.

Like many single-use products in the clinic, blue wrap and sealable pouches will contribute to a significant percentage of a practice’s waste that will end up as land fill. Australians produce approximately 21.6 billion tonnes of land fill each year; however, there are other options to help reduce the waste and the impact it has on our environment.

Re-usable cloth drapes are relatively expensive. Although they are disposed of infrequently, they need to be washed and dried after each use. They can still be perforated and hold contaminated waste in the fibres. Consider the water, detergent, electricity and nursing time required to clean them.

Reusable rigid sterilisation containers are typically made of aluminium. While they require a larger upfront cost, there’s no comparison in lifespan as rigid containers can last for more than a decade. If you calculate the processing cost per kit, a practice can see savings as soon as year 2 of ownership, simply by comparing to the cost of consumables that would have been used instead.

For more information on Aesculap reusable container systems, visit


or email us at vetcare.au@bbraun.com for personalised assistance.
Small

A KICK IN THE BUM?

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Louie is a MN DSH that lives an indoor-outdoor lifestyle on a semi-rural property. He is owned by one of our lovely clients who runs a small feline rescue organisation that we perform veterinary work for. His owner has a busy household with her own clowder of cats, rambunctious young children, and a constant rotation of foster kittens and cats. As well as a small assortment of farm animals on the property, there are also horses, sheep and cattle residing in the neighbouring paddocks. Louie was originally found dumped with his litter mates at 8 weeks-of-age and taken into care by the rescue organisation. The kids fell in love with him, and instead of being adopted out with his siblings he became a permanent member of the family. Louie visited The Cat Clinic regularly as a young kitten, and had completed his full course of kitten vaccinations, and been microchipped and de-sexed.

When Louie was 6 months-of-age, his owner went away for a well-deserved overnight holiday, and recalls seeing Louie happily playing with butterflies on the lawn in her rear vision mirror as she drove away. On return 24 hours later, Louie was discovered sitting in the bathroom, and was uncharacteristically quiet and lethargic. A strange, slightly pungent jelly-like substance was found on the bathroom floor. Louie was reluctant to move and noted to be straining to defecate with his tail sticking straight back and mucus on his bottom.

Louie was taken to the local afterhours vet and hospitalised for treatment. On physical examination, he was found to be dull, with a HR of 240bpm, RR 60bpm, and temperature of 41°C. He weighed 3.75kg with a body condition score of 4/9. A moderate amount of flea dirt was detected in his coat. He had generalised discomfort on abdominal palpation. His perianal tissue was found to be severely thickened, but no specific mass or wound was detected. He had no obvious anal tone, and was not reactive when a rectal examination was performed. He was able to walk, but was slow and crouched in the hind end.

Problem List:
- Dehydration approximately 10% and hypovolaemia
- Pyrexia
- Perianal swelling
- Abdominal pain moderate–severe

The main differential diagnosis considered at this stage was cellulitis of an unknown cause. Possibilities included a cat fight wound, anal gland disease, rectal perforation or trauma.

Full bloods were performed and found marked haemoconcentration, moderate–severe metabolic acidosis, moderate neutropenia and monocytosis, hyperbilirubinemia and azotaemia. Later that evening a small bladder was palpable, and cystocentesis and a urinalysis were performed.
Louie was transferred from the emergency clinic to The Cat Clinic Hobart the next morning. His temperature had decreased during his stay from 41 to 39.3°C. He was quiet, and had persistent abdominal pain. He had produced a small volume of urine overnight, but not defecated or been seen to eat or drink. On arrival at the clinic, significant tenesmus was noted and Louie passed a large 10cm string of thick mucus. He had significant abdominal pain, and his abdomen was very doughy on palpation. His TPR findings were; HR 200bpm, RR 80bpm and temperature 39.5°C. He was pain scored according to the Glasgow Feline CMPS pain scale as 13/20 which was higher than his last assessment earlier that morning at the emergency vets. An intravenous Fentanyl CRI was commenced at 3µg/kg/hr. Amoxicillin IV was administered, and IV Hartmann’s fluids with potassium supplementation continued at maintenance rate. A Nitenpyram (Capstar) tablet was also administered orally due to the presence of flea dirt.

Louie remained quiet throughout the day, sleeping in his bed but with a persistently high respiratory rate. Due to cost constraints and being relatively stable his owner elected to leave Louie overnight unattended at The Cat Clinic rather than being transferred back to the emergency clinic. His final examination for the evening found his HR 240bpm, RR 40bpm, temperature 40°C, and his mucous membranes were noted to be icteric. He had been tempted to eat a small amount of fishy flavoured wet food.

The next day Louie was brighter, purring, and had eaten all the food left for him overnight. Due to cost constraints his owner was reluctant to perform too many diagnostics or procedures, but consent was obtained for radiographs. A general anaesthetic was performed using intravenous Alfaxalone slowly dosed to effect (total of 1.2mL IV), he was intubated with a V gel size 3 and maintained on isoflurane 1-2%. The ventral abdomen and
perianal area were clipped and revealed bruising on the caudal ventral abdomen, significant perianal swelling and several small wounds close to the anus. On digital rectal examination the anus and rectum were severely thickened, particularly dorsally, and no anal tone was present. A blood sample was taken and his PCV was 25 and TP 65.

Several radiographic views were taken; lateral abdominal, VD abdominal and lateral thorax. There were no fractures evident, the diaphragm was intact, and his bladder also appeared intact. The main finding was soft tissue swelling in the caudal ventral abdomen, and fortunately there was no obvious disruption to the abdominal wall on either the lateral or VD view.

Louie had a smooth anaesthetic and recovery, and was bright and affectionate with a good appetite. He had not defecated but had urinated in his litter tray, and the nurses noted that it was bright yellow. Due to the ANZAC day public holiday he was transferred back to the emergency vets for ongoing care. His IV fluids, Fentanyl CRI, and Amoxicillin IV were continued, and he was also commenced on Metronidazole IV.

On return to the clinic from the emergency vets, Louie was significantly brighter than previously, purring and very affectionate. His pyrexia had resolved and his mucous membranes were now pink. He had urinated but still not defecated. He was assessed on the floor in the consultation room and was bright and ambulatory, but crouched in the hind end, and irritated by the bandage on his front leg from his IV catheter. A small amount of anal tone was now present. The bruising on his abdomen had progressed to a large open skin wound. A blood sample was taken and his PCV was now 32 and TP 74.

Careful consideration was given to the best method of treating Louie’s wound. His owner was financially limited, and although very capable and experienced with feline medications, had a very busy household and was juggling numerous commitments. She also resided over an hour away from the clinic making multiple revisits an unrealistic option. The wound was located in an awkward place close to the prepuce and anus, which meant that bandages or dressings in this area would have been difficult, and likely highly unsuccessful. The use of therapeutic Manuka honey was also determined to be problematic due to the sticky nature and messy application. I decided that the simplest treatment option in this case was silver sulfadiazine (Flamazine) ointment to be applied twice daily and left on for 10 minutes before gently removing with saline. This was performed multiple times at the clinic with Louie falling asleep in the nurses’ arms during treatment as she performed special cat whispering. I also opted not to place an e-collar on Louie. He was showing no interest in the wound, and given that...
there was significant necrotic tissue that would require debridement, I was not concerned if he did lick the wound at this stage of healing.

The next day Louie was discharged to his owner. She was happy to continue his treatment at home and keep him quiet and contained in a collapsible crate. He went home with several medications: Fentanyl (Durogesic) 25µg transdermal patch, Amoxicillin/Clavulanic acid tablets (Clavulox) 50mg BID, Metronidazole (Metrogyll) 50mg in gel capsules BID, Polyethylene glycol 3350 (Miralax) powder to be added to wet food as a stool softener, and silver sulfadiazine (Flamazine) cream to be applied to his wounds twice daily and left on for at least 10 minutes. I discussed that careful monitoring of the wound was necessary, and that surgical debridement may be required in the future if healing was not progressing.

Louie returned 5 days later for a recheck (12 days post injury). He was doing extremely well at home, and his owner was happy with his progress. He had been kept confined to his cage and allowed supervised time out of an evening when the kids were in bed......other than one time when he had escaped out of the bathroom window! His appetite was excellent, and although he was not keen on the oral medication his owner was able to administer this, and had no difficulties with his wound treatment. On examination Louie was very bright, purring loudly and affectionate. He was extremely active and even jumped off the consultation table. His mucous membranes were pink and moist. He was slightly tender on gentle abdominal palpation and vocalised when palpated in the inguinal region. The perianal swelling had reduced significantly, and anal tone was present. The skin sloughing had progressed considerably, with a small island of skin remaining around his prepuce. Given the excellent progression of his wound, the current treatment regime was continued and the owner advised that the wound would look worse before it would look better. His Fentanyl transdermal patch was removed and replaced, oral Meloxicam commenced, and a re-examination was planned for a week later.

The following week the owner reported that she had found a piece of dead skin in Louie’s bedding, and his wound was granulating and starting to contract already. Louie continued to be bright and active, with an excellent appetite.

Louie was again re-examined (19 days post injury) and there had been considerable progress with his wound granulating and reducing in size. On examination he was still slightly tender in the caudal inguinal area, but otherwise bright and comfortable. His Fentanyl patch was removed and his oral antibiotics and Meloxicam were continued. At a further re-examination on Day 35, the wound had continued to heal well, granulating and contracting. The Flamazine ointment was continued as the main wound treatment. Louie’s wound continued to contract and heal well, and at a quick check up on Day 57, only a tiny scab remained.

Discussion
It is not known exactly what had happened to Louie in the 24 hour period that his owner was away, and the cause was not evident at initial presentation at the emergency vets. After clipping his abdomen on Day 3, and seeing the pattern of skin bruising, I was highly suspicious of blunt force trauma. The pattern was not consistent with a horse or cattle hoof, and was in an unusual location. Louie was a very sociable and friendly cat, and it is suspected that he may have visited the neighbour who is not keen on cats, and received a forceful kick up the backside. This would explain the acute presentation, pattern of bruising and severe trauma to the abdomen and perianal area. The hyperbilirubinemia and bilirubinuria were likely to have occurred from red blood cell destruction due to severe tissue damage.

The management of this case is an example of where less is sometimes more, particularly with cats. The awkward location of the injury, and the inability for frequent revisits to the clinic prevented the use of bandages or dressings. The decision not to use an e-collar as part of the wound management may be seen as a controversial
one. E-collars can be highly distressing for cats, inhibiting essential grooming behaviours, and often restricting access to food and water, as well as causing discomfort and anxiety. At the clinic we infrequently use e-collars in cats, and reserve their use for cases such as managing blocked cats in hospital to prevent removal of catheters, or those with nasogastric tubes. In the initial stages of wound management I was not concerned about Louie debriding his own wound, as surgical intervention would have been required if this had not adequately occurred. Once the skin had sloughed Louie continued to be an excellent patient and was not interested in the wound. If complications had arisen during the healing phase, then a review of the treatment plan would have been necessary which likely would have included the use of an e-collar.

Louie recently visited us for his annual vaccination and routine check-up. He is living a fabulous life, and has no lasting physical or behavioural issues after his traumatic episode. All that remains to show for the incident is a scar on his caudal ventral abdomen.

An interesting differential for this case suggested by Dr Richard Malik is Fournier’s gangrene. In humans this disease is a rare form of necrotising fasciitis that affects the genital and perianal region. It is most common in middle-aged to older men, and associated with other co-morbidities. Infection involves both aerobic and anaerobic bacteria from the normal flora of the anorectal, urogenital tract or skin of the perineum.

A review in JFMS of three suspected cases describes young, otherwise healthy indoor cats with no identifiable co-morbidities presenting with perianal swelling, tenesmus and skin sloughing. Louis presented with very similar signs to these cases, although he was allowed free access to the outdoors which increases his risk of other differentials such as trauma, and infection from fight wounds. Biopsy and culture of the wound would have assisted in achieving a diagnosis, however this was not an option at the time due to owner financial constraints. In addition, antimicrobial therapy had also been commenced prior to the discovery of the skin sloughing. Fortunately this would not have changed the treatment or outcome for Louis if he did have Fouriners gangrene, as conservative management with broad-spectrum antibiotics and daily wound care was a successful treatment option in the other suspected cases.

Further Reading


Figure 13. 10 months later only a scar remains
Small

FOURNIER’S GANGRENE IN A CAT

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Louis is an indoor-only cat with an outdoor enclosure. He presented to me after his owner noticed an unusual event. Louis was sitting in his enclosure when he suddenly leapt in the air as if bitten by something. The leap was so violent, he crashed into his litter box, vomited, then ran and hid. Over the next few hours, Louis’ owner noticed progressive lethargy and depression. Being a dedicated and astute owner, he immediately brought Louis to the emergency clinic as he was worried about a spider or snake bite.

At presentation, he was pyrexic with a right sided Horner’s syndrome (protruding third eyelid, constricted pupil). No other clinical signs were identified. Blood chemistry, complete blood count and thoracic radiographs were performed and were unremarkable. He received intravenous fluids and an antibiotic (cefazolin). After 24 hours, as the Horner’s syndrome, pyrexia and lethargy were persistent, he was referred.

Louis’ presenting problems at this time were pyrexia and a unilateral Horner’s syndrome. His neurological examination revealed some subtle conscious proprioceptive deficits to his left forelimb; however, his intravenous catheter was in this limb, so the changes were of questionable significance.

Differential diagnoses for the Horner’s syndrome and pyrexia included infectious disease (e.g. otitis media/interna, feline infectious peritonitis, toxoplasmosis), immune mediated disease (e.g. meningitis), cerebrovascular accident (CVA) or trauma. Pyrexia would be unusual with CVA and there were no vestibular or otitis signs suggestive of otitis. After discussion with his owner, an MRI of his brain and CSF analysis was performed.

Figure 1. The patient developed mucoid diarrhoea in hospital. Gastrointestinal symptoms are a common theme in patients with Fournier’s gangrene.

The MRI, spinal fluid cytology and total protein concentration were all normal.

Cryptococcus, Toxoplasma and Coronavirus RNA PCR were negative. FeLV and FIV serology were negative. Toxoplasma serology (IgM and IgG) was negative.

Three days after Louis’ initial presentation, the Horner’s syndrome resolved; however, he remained pyrexic and developed mucoid diarrhea. He continued to receive intravenous cefazolin. Repeat biochemistry and CBC revealed a neutrophilia (neutrophils 12.4 x 10⁹/L [3.8-10.1]) with moderate toxic change, a mild thrombocytopenia (platelets 125 x 10⁹/l [200-700], confirmed on blood smear) and mild increases in muscle enzymes (AST, ALT and CK).

Physical examination now revealed some curious perineal changes. A small, ulcerated region of skin had developed immediately adjacent to his rectum, measuring approximately 1cm. Louis was sedated and the fur around his hindquarters clipped. Things now became even more curious.

The skin of his hindquarters was firm to touch and there was a very clearly delineated line of erythema creating a clear junction between normal and abnormal skin. The tissues of his anus and prepuce were completely unaffected. The skin lesions were bilateral and roughly symmetrical.
Louis was anaesthetised and a contrast cystourethrogram performed to ensure the lesions were not due to subcutaneous urine leakage from urinary tract trauma. This was normal and so skin biopsies were obtained from the border of affected and unaffected tissue. A sterile swab was inserted deep into the subcutaneous tissues and submitted for culture. An oesophagostomy tube was placed to facilitate enteral nutrition.

Histopathology revealed extensive necrosis involving the epidermis but also extending into deeper tissues and the panniculus. There was multifocal neutrophilic inflammation within the dermis and panniculus at the interface of the necrotic and viable tissue. Occasional fibrin thrombi were seen. The ultimate histopathological diagnosis was widespread epidermal, dermal and subcutaneous necrosis with ulceration and neutrophilic dermatitis and panniculitis. What a mouthful. Possible causes for this included thermal or chemical burns; however, this was considered unlikely given the controlled environment that Louis lived in. Immune mediated disease (e.g. toxic epidermal necrosis, erythema multiforme) can yield a neutrophilic infiltrate, but do not extend so deeply into other tissues such as the dermis and panniculus. A necrotising toxin or cutaneous infarct (e.g. secondary to a localised septic vasculitis or ‘sharp intake of breath’ a spider bite???) was considered most likely.

A gram stain performed on the deep swab revealed gram-positive cocci, numerous gram-positive bacilli and a few gram-negative bacilli. Culture yielded a mixed growth including *Corynebacterium* species, anaerobes and a multidrug resistant *Enterococcus faecium* with sensitivity only to chloramphenicol. *Corynebacterium* is considered part of the normal skin flora. Anaerobic bacteria may exist as part of normal flora in the mouth or digestive tracts but are also seen in necrotising and suppurative lesions. Genera include *Bacteroides* and *Fusobacterium* (gram negative bacilli) and *Peptostreptococcus* (gram positive cocci). Further identification of anaerobic isolates to a genus or species level is not typically performed in commercial laboratories.

So now we are edging ever closer to a diagnosis.

The pyrexia and skin lesions are now explained by a localised septic infarct with a population of anaerobic bacteria, *Corynebacterium* and *Enterococcus faecium*. Treatment of infections caused by anaerobic cocci consist of antibiotic therapy and wound debridement and management. In general, penicillins are the drug of choice, although some anaerobic cocci are resistant to penicillin but more sensitive to metronidazole.
Louis was anaesthetised again after 72 hours and the wounds around the perineal region were debrided and the eschar removed. Multiple pockets of purulent exudate were trapped under the eschar. The necrotic fat was trimmed and the wound lavaged and left open due to its proximity to the penis and anus.

Louis is making a slow but gradual recovery. We are now almost three months out and his wounds are almost healed.

With the benefit of hindsight, Louis’ presentation and lesion distribution are pathognomonic for a condition known as Fournier’s gangrene (FG). In humans, this rare but life-threatening disease appears more common in males with diabetes and long-term alcohol misuse, although it occasionally occurs in patients without obvious immune compromise.

The infection nidus is usually located in the genitourinary tract, lower gastrointestinal tract or skin. Suppurative bacterial infection results in microthrombosis of the small subcutaneous vessels leading to development of gangrene of the overlying skin. The production of various exotoxins and collagenase, heparinase, streptokinase — amongst others — aids in tissue destruction and further infection spread. As the subcutaneous inflammation progresses along fascial planes, necrotic regions appear and progress to extensive necrosis. This can progress rapidly to sepsis, multiple organ failure and death.

The distribution of the lesions is striking— the testes are usually spared as their blood supply originates within the abdomen. The anal margin is spared due to the attachment of the fascia to the perineal body.

On average, at least 3 organisms (aerobes and anaerobes) are cultured from each patient which are typically commensals in the perineum and genitalia. Although anaerobes are not as commonly isolated, this is likely more due to difficulties with isolating anaerobes in the laboratory.

Antibiotics and aggressive debridement are accepted as the standard treatment; however, in humans the mortality remains high reaching 20–30% [Pawlowski et al, Pol Merkuriusz Lek 2004].

A case series describing Fournier’s gangrene in three cats exists (Vaske et al, JFMS 2015). All cats were strictly indoors and had a subtle history of mild gastrointestinal signs (such as tenesmus or vomiting) preceding sudden severe lethargy. All patients were pyrexic, with bloodwork consistent with sepsis (e.g. increased total bilirubin, leukopaenia, elevated bands and toxic morphological changes). All had similar progression and distribution of their skin lesions and all cats survived.

The skin lesion distribution provided the key to Louis diagnosis. Interestingly, none of the cats in the case report showed any neurological symptoms like the Horner’s seen with Louis. My suspicion is this also represented a thromboembolic event.
Figure 6. The lesion is granulating well approximately 2 weeks after presentation.

The take home message from Louis is to commit to memory the distribution of the skin lesions. Give these cats antibiotic treatment, good wound care, time and they will heal.

Figure 7. Louis continues to make a good recovery approximately 10 weeks after presentation. Some self-trauma to the region is suspected to be slowing the healing process but Louis is back on form and loving life.
ANTIMICROBIAL RESISTANT NAILBED INFECTIONS OR SOMETHING MORE SINISTER?

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Penny was a 13-year-old female-spayed Domestic Shorthair who presented to Epping Veterinary Clinic in September 2020 for acute onset lameness in her right front limb with a torn nail on digit III of the corresponding paw. Her owner also reported increased lethargy, but her appetite appeared to be normal. She was an indoor/outdoor cat who shared the household with one other cat.

On physical examination, she was bright, alert and responsive, her heart rate was 232BPM with no audible murmur or arrhythmia, and she had strong and synchronous femoral pulses. She weighed 3.6kg and had a body condition score of 4-5/9, but in-depth examination of her history revealed she had lost 20% of her body weight since 2017. The soft tissue around digit III on her right front limb was swollen with a large black scab surrounding the nail, preventing extrusion of the nail for inspection. Digit III on her right hind limb was also mildly swollen with sanguineous discharge coming from the base of the nail. The nail itself appeared to be damaged with the quick no longer visible within the nail. Closer examination of her digits and assessment of her temperature could not be completed due to temperament but thoracic auscultation revealed no abnormalities, and the rest of her examination was unremarkable.

Initial differentials included
- primary paronychia (bacterial vs fungal)
- trauma (soft tissue vs fracture)
- +/- secondary paronychia

Her owner approved sedation to allow for closer examination and cleaning of her digits with 2.5% chlorhexidine solution and potentially radiographs of the digits as well. She was sedated with an intramuscular injection of medetomidine (50 µg/kg) and butorphanol (0.4 mg/kg). The fur around the affected digits was clipped, revealing ulcerated skin with purulent exudate around the nail beds (Figure 1). The nails of both digits appeared nonviable (the quick was no longer visible in the nail) but remained attached. Given the nature of the exudate, radiographs were not taken at this point. She was given a meloxicam injection (0.2 mg/kg SC) and discharged with 3 additional doses (0.05 mg/kg PO SID). Penny's owner was unable to give tablets due to her temperament, so Penny was given a cefovecin injection as well as an Elizabethan collar to prevent self-trauma. She was discharged with the recommendation of keeping her indoors until the toes had healed and to return for further investigation if there was no improvement.

Penny's owners called the practice 12 days later to ask for more antibiotics as they believed the toes were still infected. They decided to try giving Penny...
Penny returned to our clinic for a week of boarding on October 1st and a brief physical exam was performed on admission. The owners reported they had not been able to keep the e-collar on since her last consultation. At this examination, it was noted that the nail bed on the front limb was ulcerated but there was no active bleeding or discharge at this point. The exposed tissue appeared pink and viable; it had been 14 days since her initial presentation and the nail bed did not appear to be healing. However, as her e-collar had not been on, it was decided that self-trauma could be perpetuating the infection. Another differential we began considering was neoplasia. It was decided that we would put the e-collar back on her during her week in boarding and to continue the amoxicillin/clavulanic acid until we re-examined her the day before she went home. If there was no improvement, it was strongly recommended that we radiograph the digits under general anaesthesia, obtain a biopsy, and send samples off for bacterial and fungal cultures and sensitivity.

I re-examined Penny on October 7th, 20 days after her initial presentation. The right front digit was markedly more swollen relative to my initial examination. The area of ulceration had enlarged and there were white plaques on the ulcer. It was warm to the touch and oozed serosanguinous discharge when pressure was applied. The right hind digit showed similar changes. Progressive deterioration of her toes, despite having a course of amoxicillin/clavulanate as well as cefovecin, created suspicion of an antimicrobial resistant bacterial pathogen, fungal infection, or neoplastic process occurring. As there were two digits on different paws affected, primary neoplasia originating from the toes seemed less likely and feline lung-digit syndrome (FLDS) was added to the list of differentials. I recommended thoracic radiographs in addition to the other tests listed above in order to rule out FLDS and to assess for pulmonary metastasis in case the primary neoplasm originated from the toes. Due to cost constraints, Penny’s owner could not agree to the full battery of diagnostic tests. Photos of Penny’s toes were sent to Dr Malik to see if any of the diagnostic tests should be prioritized. After his input, it was agreed that the gross appearance of the lesions seemed aggressive, placing neoplasia higher on our differentials list and radiographs were recommended.

Penny was sedated with an intramuscular injection of medetomidine and butorphanol. Blood was taken for in-house haematology and biochemistry, both of which were unremarkable. Three views of the thorax were taken. In both the left and right-lateral views, there was a bronchial pattern in the caudodorsal lung lobes and a diffuse interstitial pattern in the accessory lung lobe (Figures 2 & 3). There were also pinpoint radiopacities in the ventral aspect of this lung lobe, most prominent in the left lateral view. The ventrodorsal view was unfortunately slightly rotated and the spinal vertebrae were superimposed on top of the accessory lung lobe (Figure 4). There were also pinpoint radiopacities in the ventral aspect of this lung lobe, most prominent in the left lateral view. The ventrodorsal view was unfortunately slightly rotated and the spinal vertebrae were superimposed on top of the accessory lung lobe (Figure 4). Radiographs of the right front and hind paws were taken as well. There was a moth-eaten appearance to the middle and distal phalanx of digit III on the right hind limb (Figure 5) as well as complete

Figure 2. Left lateral view of the thorax illustrating a bronchial pattern in the caudodorsal lung lobes and a diffuse interstitial pattern in the accessory lung lobe with pinpoint radiopacities in the ventral aspect.

Figure 3. Right lateral view of the thorax illustrating the same changes as in the left lateral view.
Osteolysis of the distal phalanx including loss of the nail on the right front limb (Figure 6).

Radiographic findings of the thorax and digits were consistent with the presumptive diagnosis of FLDS and Penny was discharged with analgesia (buprenorphine oral transmucosal q8h) while her owners decided on the best course of action. We relayed Dr. Malik’s suggestion of palliative care using meloxicam to keep her comfortable for a few more months; however, her owners were concerned about her quality of life and Penny was euthanized 4 weeks after initial presentation.

Case Comments
A definitive diagnosis was not obtained for this case as that would require histopathological analysis of the lesion in the toes and thorax, proving that the same neoplasm was at both locations. A comparable and cost-effective alternative would be cytological examination of fine-needle aspirates from the toes for respiratory epithelial cells, which would suggest metastasis from a pulmonary neoplasm and support a presumptive diagnosis of lung-digit syndrome. As bacterial and fungal cultures were never obtained, it is conceivable that the opacity in the thorax could have been unrelated (i.e. localized pneumonia, abscess) to the pathology in the digits (i.e. fungal or resistant bacterial infection). However, this was less likely based on the normal haematology and clinical examination findings.

In hindsight, more judicious use of antimicrobials could have been demonstrated had we questioned the owners’ willingness to try giving tablets. Amoxicillin-clavulanate is of lower importance than cefovecin and ideally should have been prescribed before moving on to the higher importance third-generation cephalosporin. In addition, we may have reached a more timely diagnosis had we suggested further work-up rather than prescribing a second antibiotic without culturing. Both amoxicillin-clavulanate and cefovecin are beta-lactam antimicrobials with a similar spectrum of activity and mechanism of action, so a methicillin-resistant Staphylococcus infection would be resistant to both. Furthermore, without a fungal culture, we cannot definitively exclude a fungal infection.

Feline Lung-Digit Syndrome
Lung-Digit Syndrome is a term used to describe a clinical presentation unique to cats in which a primary lung tumour metastasizes to the digits. Multiple digits and limbs may be involved, with the weight-bearing digits most commonly affected. The mean age of presentation is 10–14 years of age, with no sex or breed predilections. Primary lung tumours in cats are considered rare, making up less than 1% of all diagnosed tumours in the species. Of the
primary lung tumours diagnosed in feline patients, bronchial and bronchioalveolar adenocarcinomas are the most common.1,2 Respiratory symptoms arising from the primary lung tumour are frequently absent, with the presenting complaints often consisting of non-specific signs such as lameness, lethargy, weight loss and inappetence.1,3

The exact pathogenesis of FLDS is unknown but it has been hypothesized that increased vascularity to the digits as a method of heat dissipation facilitates haematogenous spread of tumour emboli from the lungs.3 Metastatic lesions in the digits typically result in osteolysis of the distal phalanges, and have the ability to cross the intra-articular space to involve the middle phalanges.2

In a 1999 study of 64 cats with digital lesions submitted for histopathology and immunohistochemistry, 56 (87.5%) cats had metastatic lesions from a primary pulmonary carcinoma while the remaining 8 had primary digital carcinoma.4 A more recent study from 2007 found malignant neoplasia in 95% of feline digits submitted for histopathology and 1 in 6 digits contained a metastatic lesion from the lungs.1

Unfortunately, the prognosis of patients with FLDS is poor as it has not been shown to be responsive to chemotherapy and treatment consists of palliative care in the form of pain management2,3 Amputation of the affected digits does not enhance survival time as there may already be metastatic spread to other digits.3 The median survival time (MST) after diagnosis is 67 days, with many owners electing to euthanize due to poor quality of life.3

Acknowledgments
Special thanks to the owners for allowing Penny’s case to be shared. Additional thanks to Dr Richard Malik and Dr Sarah Short for their support and input in the work-up of this case.

References
A 7-week-old male domestic shorthair presented for a 7-day history of constipation that was unresponsive to regular laxatives (unknown type as seen as a second opinion). The kitten was undersize for its age and was obstipated. Radiographs were concerning for megacolon.

The kitten was administered a general anaesthetic and an enema performed to clear the colon. Intravenous fluid therapy, Osmolax, lactulose and cisapride were commenced upon recovery. Unfortunately, obstipation recurred, and another general anaesthetic and rigid enema was performed.

Upon further investigation of chronic constipation in kittens, the possibility of hypothyroidism was discovered (in the 5-minute Vet Consult of all places). Upon closer examination of the kitten, he was found to have short limbs, an enlarged broad head with wide set eyes and a noticeable goitre. The signs were overall quite subtle and were easy to miss without specifically looking for them.

In house lab work revealed a mild non-regenerative anaemia (0.30 L/L), high cholesterol (6.28mmol/L) and low total T4 at 7mmol/L (IDEXX in house < 11). External free T4 was too low to gain a reading (IDEXX laboratories). Total T4 recheck is still pending at time of writing this article.

The kitten was started on thyroxine 25µg twice daily and his symptoms rapidly resolved. Laxatives were slowly tapered—he rapidly started to gain weight and regained normal bowel function in the absence of laxatives within 2 - 3 days.

As he was diagnosed and treatment commenced so early (7-weeks-of-age) and his symptoms were mild (minimal bony changes) his long term prognosis is good with appropriate twice daily thyroxine administration—according to Drs Richard Malik and Andrea Harvey who the authors wish to thank for their assistance with this case.
A CASE OF SUSPECTED LILY (LILIUM SPP.) INTOXICATION IN A YOUNG DOMESTIC KITTEN

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Abstract
Lily plant (Lilium spp.) intoxication has so far been an issue seen specifically in cats. These cats are often presented with vomiting, lethargy, and/or because an owner with knowledge of the plant’s toxicity observed ingestion. The mainstay of treatment is dependent on time of presentation, but is generally gastrointestinal decontamination, fluid therapy and supportive care. The kitten in this case presented after an onset of lethargy and anorexia with suspicion of lily pollen ingestion. Hospitalisation and supportive care were provided for the kitten and led to a complete resolution of clinical signs.

Introduction
Any number of everyday household situations can put companion animals at risk of possible exposure to a variety of toxic substances; from caustic materials such as bleach that the majority of owners would immediately recognize as dangerous, to those that owners may not immediately consider to be life-threatening e.g. human medicines such as psoriasis cream, ibuprofen or paracetamol. In general, domestic cats are thought to be presented to their primary care veterinarians with a history of toxin ingestion significantly less frequently than their canine counterparts who are more likely to indulge themselves in dietary indiscretions. However, young kittens are naturally playful, curious and inquisitive generally placing them at higher risk of toxicity than adult cats.

Ornamental house plants belonging to the genus Lilium and Hemerocallis (Easter lily, Tiger lily, Rubrum, Japanese Show Lily, Stargazer Lily, Oriental Lily and Day Lily) are nephrotoxic to domesticated feline species. Studies have shown that the ingestion of any part of the plant, pollen or leaves can be fatal for cats.1-3 Lily intoxication is characterized by kidney tubular necrosis and death is caused by acute renal failure.1-3 Affected cats may start presenting clinical signs between one and six hours after plant ingestion, which may include anorexia, apathy, vomiting, diarrhoea, and drooling.1-3 Signs of renal failure usually occur from 12–72 hours after lily ingestion, and are characterized by polydipsia, polyuria, anuria, and azotemia.1-3 Here we present a case of highly suspected lily intoxication in a young kitten which survived to discharge following hospitalisation and intensive medical care.

Case Report
A 6-month-old female entire Domestic Shorthair (DSH) cat was presented with a 16-hour history of lethargy and anorexia. The owner reported having
flowers of *Lilium* spp. in the house. While her owner did not see her cat ingest the lilies, the owner suspected intoxication based on her penchant for playing with household plants. Her clinical examination revealed a body weight of 2.35kg, body condition score of 4/9 and a mild yellow staining of the white fur on her front left paw (presumptively lily pollen). Abdominal palpation revealed enlarged, moderately painful kidneys bilaterally. The remainder of the examination was unremarkable but based on the history she was hospitalised for supportive care and further investigations.

Emesis was not induced due to the owner’s suspicion that the intoxication had occurred in excess of 24 hours previously. Abdominal radiography (Figure 1) and ultrasonography (Figure 2) confirmed bilateral renomegaly but found no other abnormalities. Non-steroidal anti-inflammatory drugs were withheld due to the suspicion of a potentially nephrotoxic insult, so opioid analgesia was administered (buprenorphine [Vetergesic, Ceva] at 0.03mg/kg subcutaneously).

Routine haematology and (unstarved) serum biochemistry showed elevated urea at 16.1mmol/L (reference interval 5.7 – 12.9mmol/L) and that the rest of the biochemistry parameters were within reference interval. She was started on aggressive intravenous fluid therapy (IVFT) with Hartmann’s (lactated Ringer’s solution) at four times maintenance rate (12mL/kg/hour) for one hour and then reduced to twice maintenance rate (6mL/kg/hour). After the initial fluid bolus, she was given 2 mg/kg furosemide (Dimazon, MSD Animal Health) intravenously (IV) to assist in the induction of diuresis. She was maintained on twice maintenance fluids through the day and overnight and was voluntarily appetent (Purina EN wet food) within eight hours.

A urinalysis and a repeat haematology and serum biochemistry were done the following day (40 hours post exposure). The urinalysis showed significant proteinuria (>20g/L) and glucosuria (>110mmol/L), a specific gravity of 1.020 and a urine protein-to-creatinine ration (UPC) of >9.63. The serum biochemistry parameters were all within normal reference intervals. The kitten was bright, eating well and never became anuric. She was maintained for another 24 hours (i.e. a total of 48 hours) at twice maintenance fluids and close monitoring on the advice of the Veterinary Poisons Information Service (VPIS).

The following morning the patient was bright, clinical examination was normal, but she had removed her intravenous cannula. Repeat urinalysis was similar to the previous day with the only change being an increased concentration to a specific gravity of 1.025. Repeat serum biochemistry showed azotemia with creatine of 240 µmol/L (reference interval 71-212 µmol/L), urea 16.6 mmol/L, and elevated GGT of 7 U/L (reference interval 0-4U/L). She also had an elevated symmetric dimethylarginine (SDMA) of 17µg/dL (reference interval 0-14 µg/dL). She was discharged that afternoon after 48 hours of IVFT, with instructions for the owner to measure fluid intake and urine output.

The following day the owner reported water intake of 260mL and urine output of 220 grams. She was reported to be bright, eating well, and back to her ‘normal, playful self’. The patient was then lost to follow up.

**Discussion**

The ingestion of lilies is known to be nephrotoxic and sometimes pancreotoxic to domestic cats. The entire plant is known to be toxic, with even small quantities of pollen or leaf ingestion capable of causing toxicity. The toxic component has yet to be identified but it is known to be water soluble leading to ready absorption once ingested. The exact mechanism behind the nephrotoxicity is unknown, but the end result of renal tubular necrosis is likely to have been responsible for both the glucosuria and
severe proteinuria seen in this case. It is reported that, anecdotally, the risk of developing chronic kidney disease (CKD) long-term following lily-induced acute kidney injury (AKI) is probably lower for kittens as their kidneys likely have a greater capacity for tubular recovery than in older cats.\(^5\)

This particular case was challenging as lily ingestion was suspected but both the amount and the timeframe from ingestion to presentation were unknown. Based on the history from the owner, the initial clinical exam, consistent urine output, and initial serum biochemistry revealing mild elevation of blood urea, it was suspected that the patient was presented at least 24 hours after pollen ingestion. Gastrointestinal decontamination was not strongly indicated given this timeframe due to the transition time of food through the feline gastrointestinal system. One study has demonstrated that after approximately eight hours, ingesta (either wet or dry food) will typically have passed from the stomach into the feline small intestine for absorption and so decontamination could be considered indicated for patients presenting sooner than this following ingestion. Initial therapeutic protocols, based on the very first case series of feline lily toxicity, suggested gastric decontamination accompanied by fluid diuresis for cats presented within the first six hours post-ingestion and this was shown to prevent the onset of AKI entirely.\(^6,7\) Decontamination consisted most frequently of the induction of emesis.\(^6,7\) For the majority of cats, emesis can be induced by the administration of adrenergic alpha-2 (\(\alpha-2\)) receptor agonists such as xylazine. Alternatively, medetomidine or dexmedetomidine have been reported to be used where xylazine is unavailable. Further decontamination can be considered by gastric lavage and/or the oral administration of activated charcoal.\(^6,7\)

In the case presented here, the main focus of treatment was to prevent further renal damage and the development of anuria which is known to be a poor prognostic indicator.\(^8\) The initially aggressive rate of fluids and use of furosemide was to induce diuresis and therefore maintain urine production. The use of furosemide or alternative diuretics following nephrotoxic insult can be controversial as it has been proposed that they can further damage kidneys.

More recent literature suggests the importance of not overhydrating acute kidney injury patients and that being too aggressive with fluid therapy can cause renal interstitial oedema and result in reduced glomerular filtration rate.\(^5-8\) Repeat urinalysis demonstrating an increasing specific gravity would usually suggest that the fluid rate wasn’t leading to overhydration; however, with significant glucosuria and proteinuria the specific gravity is artificially elevated and therefore may not be the most reliable measure of hydration status. In this instance, it is probably more useful for clinicians to monitor cats’ resting respiratory rate, effort, and any changes in thoracic auscultation.

It has been suggested that kittens are more likely to ingest foreign objects including toxins than adult cats due to their more playful natures. Young kittens do not necessarily have the same fat reserves.
as adults and it is particularly important that a hospitalised feline patient does not go without eating for more than 72 hours, as this may lead to further complications—such as hepatic lipidosis. This is a condition that results from excessive fat loss from cells that are unable to be metabolised by the liver. Fat accumulation in the liver causes damage due to swelling of the liver cells, fatty deposits, and other processes. Hepatic lipidosis is a serious condition that can result in death of a patient. In persistently anorexic patients it may be necessary to give medication to increase their appetite e.g. mirtazapine (1.8mg per cat per day, given by mouth) and if this fails, place a feeding tube to ensure the patient receives adequate nutrition.

Conclusion

Lily toxicity can be a rapidly progressive and fatal condition of cats. Due to their playful nature, kittens are generally thought to be at greater risk than adult cats but have potentially greater renal regenerative capacity meaning that early identification of ingestion coupled with aggressive therapy can frequently give rise to good long-term outcomes. This case highlights the need for clinicians to take a ‘safety first’ approach to the instigation of therapy when lily intoxication is a differential diagnosis.

Editor’s Note:

The CVE (then the PGF) first alerted our members/readers to Lily Intoxication in the C&T Series in 2005 in a poster that vets were asked to display in their clinics. Over the years, these posters were updated and Dr Frank Gaschk, a long-time CVE member and supporter, designed stickers that were distributed through Australian horticultural education organisations to florists. Download in the eBook.

References:

4. Veterinary Poisons Information Service: Homepage - VPIS (vpisglobal.com)
COPPER POISONING IN SHEEP GRAZING IN A VINEYARD

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Introduction
The owners of a small flock of 60 sheep grazing amongst grape vines became alarmed when 7 died over the course of a few days in May 2019. The sheep had previously appeared healthy and in good body condition, and had not been observed to be unwell.

The final diagnosis was copper toxicity, presumably from the long term use of copper containing compounds in the vineyard.

Sheep are commonly used for grazing amongst vines when they are dormant as a means of weed and grass management, so this case may have lessons for others using small groups of sheep for this purpose.

History
A vineyard owner near Waikerie in the SA Riverland area reported losses of some Dorper sheep to his local vet and PIRSA in mid May. The sheep had not been observed to be unwell or recumbent, no obvious poisonous plants were seen in the area, and no treatments had been applied recently that could be considered toxic to sheep. The sheep, in fact were in very good (prime) condition and most were early pregnant and the onset appeared acute.

Seven sheep died over a period of 2–3 days and two dead sheep were taken to the local veterinary clinic for post-mortem examination. Costs of the examination and lab fees were subsidized by the PIRSA Disease Surveillance Program.

Results
A range of samples including fixed tissues and ocular fluid were tested for ammonia, urea, D Lactate, and Ca/ P levels to rule out some common causes of sudden death. Rumen pH was normal and no internal parasites were seen, faecal egg count (FEC) was 0.

The significant histopathological signs were acute, massive hepatic necrosis. There was marked lobular necrosis and haemorrhage involving the periacinar and mid-zonal regions with only portal hepatocytes unaffected. Changes in lung and intestines were also noted, but not considered to be the cause of sudden death.

The pathologist’s comments were: ‘Consider mushroom toxicity—with wet weather there may be mushrooms amongst grasses and other vegetation between the vines. Other causes of such lesions include: algal toxicity including Microcystis (blue green algae), but there are indications that other as yet unreported or unidentified algae may be toxic.—Cycads, Xanthium species (2 leaf stage) burrs (may be a weed between vines), certain Solanum species, in particular Cestrum (not found naturally in SA , several plant species of the Asteraceae family (but there have not been reports in Australia of toxicity with these). The lung lesions are consistent with verminous pneumonia, most likely due to Dictyocaulus filaria.’
Aqueous humor ammonia levels were very high in one sheep (2636 µmol/L (0 - 200) (possibly artifact?) but Nitrate/ nitrite levels were normal. Copper levels in kidney and liver were measured with all levels high.

<table>
<thead>
<tr>
<th>Kidney copper levels (0.00- 0.20mmol/kg)</th>
<th>Liver copper (0.23 – 3.67 mmol/ kg)</th>
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<td>0.8, 0.61</td>
<td>5.97, 5.43</td>
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**Interpretation/ Discussion**

Both acute and chronic copper toxicity is described in texts 1 and 2. Acute copper toxicity is usually described as producing severe gastroenteritis, abdominal pain, diarrhoea, and central nervous symptoms and death in prolonged cases. On post mortem examination, ascites, hydropericardium, haemoglobinuria and liver lesions may be seen. Chronic poisoning may appear with jaundice, haemoglobinurin and death.

**Clinical signs:** Sheep rarely show clinical signs until the animal is stressed, resulting in a massive liver necrosis and copper release. The released copper then causes intravascular hemolysis of red blood cells, resulting in haemoglobinuria, icterus, anorexia, and death. Urine is dark red (port wine) as a result of the presence of haemoglobin in the urine. Usually only one or two animals in the group die at any one time, while the remainder of the flock appears clinically normal. However, once a stressor affects the flock again, several more animals may die.³

Sheep are more sensitive to copper accumulation in the liver than other ruminants,² as accumulation of copper in liver exceeds the ability to excrete it in bile. During the period of copper accumulation in the liver, associated with subclinical changes to liver tissue, sheep are considered to be in the pre-haemolytic phase of copper poisoning. Following the sudden increase in blood copper concentrations and consequent haemolytic episode, sheep are considered to be in the post-haemolytic phase.

In most field cases in which a haemolytic crisis occurs, death occurs within one to three days of the event. The sudden elevation of blood copper concentration and the acute fatal haemolytic syndrome is the most recognised clinical expression of the chronic intoxication, but cases have also been reported in which copper concentrations in the blood are elevated for several weeks before the acute haemolytic episode. In some cases, milder forms occur from which animals recover.

**Diagnostic Testing:** Serum copper levels can be difficult to interpret. Even though excessive copper is being accumulated in the liver, the serum copper levels often remain normal until hepatocellular necrosis occurs. This elevation may be transient and quickly returns to normal values as a result of renal and hepatic reabsorption if the damage is mild. However, once hepatic reabsorption is lost, the copper is released from the liver into the bloodstream and a toxic amount of copper is detectable in the serum. At necropsy it is best to test both the liver and kidney for copper. In many cases the liver releases enough stored copper so that a non-toxic level of copper remains in the hepatic tissues. However, in these cases the released copper will have accumulated in the kidneys, resulting in markedly elevated levels of copper in the kidneys.³

Chronic forms of poisoning have been described associated with contamination of plants sprayed with fungicides, water due to snail or algae treatment, pasture grazing after some top dressings and other sources.¹

**Management**

Copper toxicity can be managed and treated in sheep both on an individual basis for valuable animals using ATTM (ammonium tetrathiomolybdate), and in groups using Sulphur and molybdate.²

Groups of ewes with chronic copper poisoning have been successfully treated with ammonium molybdate (40 g) and sodium sulphate (1200 g) mixed in water (10 L) and sprayed on hay such that the sheep each received 40 to 50 mg ammonium molybdate per head each day. Treatment was continued for three weeks but deaths in the flock ceased three days after treatment began. There are limited, if any, products for treatment of copper poisoning registered in Australia. Provision of a homemade loose lick consisting of salt,
finely ground gypsum (calcium sulphate) and sodium molybdate in the ratio by weight of 187:140:1 has been recommended and reported to be successful when used in Australia. The salt (75 kg) and gypsum (56 kg) are mixed together on a flat surface and then the sodium molybdate in solution (400 g in 20 L) is sprayed onto the mixture and thoroughly mixed. The product should then be placed in open containers and offered to the sheep in the field, ensuring that all sheep have uninhibited access. *The Practice of Sheep Veterinary medicine* (pg. 560)².

Owners of vineyards that may have small groups of sheep grazing the land in dormant periods should be aware of the possibility of copper toxicity in these sheep when using copper containing products to control diseases on the vines.

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WHAT IS WRONG WITH DINKY’S PENIS?

Answer to C&T No. 5882
(Issue 302 March 2021)

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What is your differential diagnosis?

How would you investigate this case further and what are the treatment options?

The photographs show the penis of an approximately 15-year-old Brumby gelding from Guy Fawkes National Park after sedation with detomidine/butorphanol/acepromazine. This was the second time the horse had been sedated for hoof trimming, and similar changes had been present 12 months earlier. At that time warm soapy water was used to peel the abnormal tissue from the shaft of the penis. The material was hard and apparently keratinised. It would peel off the penis in large chunks, with surprisingly little bleeding, leaving relatively healthy mucosa.

On this occasion—similar changes were present, and again, the thickened hyperplastic material was peeled off the penis.

What is your differential diagnosis?

I guess many different disease processes might account for the observed lesions. Cutaneous habronemiasis is possible, as is onchocerciasis. However, the horse was treated 3-4 times a year with either ivermectin or moxidectin, which would argue against a parasitic aetiology. It might be the horse had an equine squamous cell carcinoma—although the lesion might have been expected to be more proliferative or ulcerated, and certainly one would think it would have progressed over many months. Actually, this is one of those instances where the lesion represents a very characteristic illness script—so the diagnosis is easy, but only if you are familiar with the entity. If you are not—then you have to work from basic principles and get histological assessment of the proliferative keratinous material peeled off the penile shaft, and probably also a biopsy of the underlying mucosa. But if you are familiar with equine papillomatosis due to EcPV-2, then you might just be happy to make a clinical diagnosis or confirm the diagnosis non-invasively by collecting swabs for papilloma PCR testing (available at Massey University).

Equine penile papillomatosis is caused by a horse papilloma virus. One assumes it is transmitted venereally. In the case illustrated in Figures 1 and 2 in the PhD Thesis of Cameron Knight, photographs taken 16 months apart demonstrate lack of lesion progression nor ulceration. The resulting disease seems to run a benign clinical course, without progression to squamous cell carcinoma.

What did we do for Dinky’s penis?

After sedation with detomidine, butorphanol and acepromazine, we cleaned the penis using warm soapy water, gently removing the hyperplastic papillomatosis material. We then applied 5 fluorouracil 5% sparingly over the whole of the
affected penis. The next time the penis was examined, the papillomatosis lesions had largely disappeared, although a small polyp remained. There is a risk of these lesions progressing to squamous cell carcinoma and so frequent treatment with 5 fluorouracil 5% and cryotherapy is often recommended. In the case of Dinky, he doesn’t enjoy handling very much and so we try to strike a balance with managing the disease without too intense treatment. He has now been about 4 years without further progression of the remaining lesion nor any development of further lesions.

Reference

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Pancreatitis in cats is a common condition, likely underdiagnosed. Histological prevalence of 66% has been reported in post-mortem studies and up to 45% in apparently healthy cats. Despite the frequency the disease likely occurs, diagnosis is not always straightforward, and treatment mainly supportive.

Causes of pancreatitis
In the majority of cases, no cause of pancreatitis is found. A few exceptions exist including trauma, pathogens (Toxoplasma gondii), hypotension, thrombosis, medications, and an immune-mediated cause, as seen in humans, is suggested by some authors. Pancreatitis may occur as part of pathology of the cranial abdomen (liver and intestine) in a condition known as ‘triaditis’; perhaps the common biliary and pancreatic ducts found in this species predisposes cats to biliary pancreatic reflux and spread of inflammation to the pancreas from its neighbouring organs. In clinic, a specific cause is rarely identified yet a common process of premature activation of digestive enzymes results in either acute or chronic pancreatitis with fibrosis occurring with the latter.

Clinical signs
Clinical signs are rarely specific, and abdominal pain can be hard to detect in cats. To complicate matters, comorbidities may confuse the clinical picture. Indeed, pancreatitis may present with quite subtle signs in the chronic form with inappetence and lethargy. Vomiting and diarrhoea may be present and in acute cases cats may present as an emergency with fluid volume deficits, hypotension and dyspnoea. On physical examination dehydration is common, along with hypothermia and icterus and abdominal pain may also be present.
Making a diagnosis
Clinical pathology is the place to start in most cases, and a complete blood count may show an inflammatory leukogram or anaemia for example but may be normal. Biochemical changes may include hepatic enzyme elevations and hyperbilirubinemia. The latter may be due to cholestasis, extrahepatic bile duct obstruction or hepatic disease as a comorbidity. Importantly, amylase and lipase elevations in cats are not specific and elevations may be seen in conditions causing for example a reduced glomerular filtration rate. What is more useful is measurement of pancreatic lipase immunoreactivity (fPLI) or DGGR lipase.

DGGR lipase and fPLI
fPLI can be measured using a patient-side test (SNAP fPLI, IDEXX Laboratories) or commercial ELISA. fPLI is highly specific for pancreatitis (few false positives) and sensitivity for moderate to severe pancreatitis is also high. Sadly, sensitivity falls for subclinical to mild disease, hence a normal fPLI does not exclude pancreatitis. DGGR-lipase measurement has been shown to have good agreement with fPLI, and is quicker and more cost effective to measure. However, recent studies suggest a reduced specificity. Confusingly, agreement between fPLI or DGGR lipase with ultrasonography diagnosis of pancreatitis has been poor in published studies, emphasizing our ongoing challenges with diagnosis.

Imaging the pancreas
Radiography tends to have limited utility in the diagnosis of pancreatitis—beyond a loss of detail in the cranial abdomen, or a mass effect if the pancreas is very enlarged. Ultrasound is more useful and has the additional benefit of identifying common comorbidities such as gall bladder disease, intestinal and liver pathology. However, even ultrasound has its limitations in the diagnosis of pancreatitis with a wide range of sensitivities reported for both acute and chronic disease. In acute pancreatitis, the pancreas may be enlarged, hypoechoic with irregular margins, a surrounding bright mesentery and a focal effusion. In chronic disease the pancreas may be more hyperechoic with again enlargement and irregular margins, but given overlap between these signs, ultrasound may not be optimal for distinguishing acute vs chronic disease.

Histopathology
Historically, surgeons have been reluctant to anger the pancreas with handling and biopsy. However, studies show that pancreatic biopsy at exploratory laparotomy or laparoscopy is a low-risk procedure. Hypotension and manipulation affecting blood flow of course are to be avoided. Sadly, even histology has its own limitations. Disease may be patchy (if taking only one biopsy, the left limb is preferred), and mild inflammatory changes seem to overlap with ‘normal’ raising questions regarding how severe changes must be to be make a diagnosis. Results should be reported according to published histological standards for this condition and explained in the recent ACVIM consensus statement on pancreatitis in cats (open access).

The ACVIM Consensus Statement is in the eBook.

Treatment of pancreatitis
Management of pancreatitis centres around supportive measures and treatment of comorbidities, as a cause is rarely identified. A focus on analgesia, anti-emetic therapy, nutrition and treatment of fluid deficits is appropriate (figure 3).

Conclusion
Pancreatitis in cats remains a challenge for clinicians. However, many cats recover fully and are rewarding to treat. Keep an open mind for comorbidities and optimize recovery with early assisted nutrition.

Note: Dr Taylor is presenting at the World Feline Congress.
Figure 3: Management of pancreatitis: a multimodal approach. AP = acute pancreatitis, IBD = inflammatory bowel disease, DM = diabetes mellitus, CRI = continuous rate infusion, EPI = exocrine pancreatic insufficiency. Medications should be given following local prescribing rules and dosage may vary according to the case.

ANALGESIA

Abdominal pain may be hard to detect in cats. Use pain scoring scheme:
- Methadone (0.1–0.2mg/kg IM or IV q 4–6 hrs)
- Buprenorphine (0.02–0.03mg/kg IV, IM or transmucosally q 6hrs)
- Cases of AP may require further analgesia e.g. ketamine/fentanyl CRI
- Gabapentin can be considered as an additional oral therapy

NUTRITION

Do not withhold food, early enteral nutrition a priority:
- Control nausea, pain and stress to encourage voluntary intake
- Treat hypokalaemia if present
- Place a naso-oesophageal feeding tube for short-term feeding
- Consider an oesophagostomy tube for patients with ongoing inappetence
- Mirtazapine may be useful as an appetite stimulant
- Longer-term consider comorbid diseases and appropriate diets e.g. IBD, DM.
- No current evidence of a benefit of reduced fat diet, highly digestible ‘gastrointestinal diets’ appropriate while unwell.

FLUID THERAPY

Correct volume deficits, monitor blood pressure, weight, avoid overhydration:
- Estimate dehydration and correct with crystalloids over 12–24 hours
- If hypovolaemic/hypotensive bolus with 5–10ml/kg crystalloid over 5–15 minutes and monitor response, repeat if required
- Occasionally addition vasopressor support needed e.g. dopamine CRI required to maintain blood pressure in AP

ADDITIONAL THERAPIES

Manage comorbidities, e.g. antibiosis for cholangiohepatitis, cobalamin if deficient, treatment of EPI or DM.
- Corticosteroids rarely indicated specifically for pancreatitis but may be needed to treat other immune-mediated pathology.
- Ileus may be managed with metoclopramide or cisapride.
- Severe AP cases may need additional therapy such as fresh frozen plasma for coagulopathy, management of pleural effusion.
- Surgical management of pancreatic abscess or extrahepatic biliary obstruction may be indicated in certain cases.

ELECTROLYTE ABNORMALITIES

- Hypocalcaemia should be treated with calcium gluconate IV if clinically indicated
- Hypokalaemia should be corrected by supplementing IV fluids

ANTIEMETIC TREATMENT

Assume nausea even if overt clinical signs are not observed. Treatment options:
- NK-1 receptor antagonists: maropitant 1mg/kg SC q24 hrs
- 5HT3 antagonists: ondansetron 0.1–1.0mg/kg slow IV, IM, SC, PO q 6–12 hrs
- Dopamine antagonist: metoclopramide 1–2mg/kg/day CRI (additional prokinetic effect)
The surge in popularity of French Bulldogs is obvious to urban vets in Australia and is a worldwide phenomenon, with the Kennel Club reporting that Frenchies had taken over as the most popular breed registered in the UK in 2018 (demoting Labradors from top spot for the first time on record). This popularity, and the likely indiscriminate breeding that it encourages, have presented several health challenges to veterinarians. The clearest concern is brachycephalic obstructive airway syndrome (BOAS) but spinal conditions are also competing for attention in a breed that seems to spend more time receiving veterinary care than most. As CHOICE notes: ‘Frenchies are really expensive to insure’.

Another breed predisposition, first becoming apparent anecdotally about four or five years ago, is pathology of the humeral condyle. Most frequently manifesting as fracture, this is probably the third most common reason French Bulldogs are presenting for specialist surgical care, after BOAS and intervertebral disc extrusion.

A recent flurry of groups internationally have published research confirming this observation over the last year; finding French Bulldogs to now be over-represented in presentations for humeral condylar fracture.1-4

The other group of dogs considered predisposed to humeral condylar fracture, spaniel breeds, have been known for the last couple of decades to be afflicted by a mid-sagittal plane humeral condylar osseous defect.5, 6 This has been variably referred to as incomplete ossification of the humeral condyle (IOHC) or humeral condylar fissure (HIF) reflecting proposed aetiologies and has been found to be present in 14% of English Springer Spaniels7 which are the most commonly effected breed in the UK.8

The concept of IOHC as a developmental disorder is attractive. The canine distal humeral epiphysis develops as two separate centres of ossification that should fuse by 10 to 12 weeks of age6, 8 so presence of mid condyle lucency radiographically after this age can be considered delayed or failed ossification and leaves the dog vulnerable to fracture, or other associated pain. A genetic mechanism has been proposed in spaniels.6, 8

The difficulty with a failure of ossification aetiology is that serial computed tomography (CT) evaluation has shown development of a sagittal hypoattenuating fissure in the condyle of a Cocker Spaniel twenty-two months after a screening CT had shown a normal condyle in one case report9 and is this considered common amongst surgeons working with this condition. The possibility for a stress-fracture type aetiology must therefore be considered and investigators have proposed an ‘ulnar wedge effect’
based on the cross-sectional conformation of the elbow joint potentially ‘driving’ the condyles apart. Variable modifications of this concept are currently popular with different investigating surgeons: Rotational forces through the elbow have been seen to result in widening of the fissures when assessed on arthroscopic examination by one expert investigator. Rotational instability is also one of the areas currently implicated as an aetiology of coronoid disease, a frequent co-morbidity in these spaniels, lending some support. An early-in-study suggestion that HIF lesions may be healing (on CT) with proximal ulnar osteotomy (PUO) alone could implicate either a rotational or linear force from the ulna on the condyle; however, PUO has been shown to increase rotational instability of the elbow so this finding may favour an axial force overload.

Significant questions clearly remain as to whether there is one primary aetiology acting at different time points or indeed more than one cause. Conformational abnormality, leading to stress and thus fissuring, is a consideration and a chondrodystrophic skeleton is a relatively frequent feature of at-risk dog (and pig) breeds; however, no specific deformities have been identified to date, and radio-ulnar incongruity has been evaluated and found not to be present. This very imperfect understanding of aetiology has led to uptake of the HIF terminology to avoid referencing an uncertain cause.

Presentation
HIF in Spaniels is associated with two types of presentations: I) Fracture, and II) Chronic low to medium grade lameness due to micromotion between the compartments, generally at about four years of age. An important third group of known HIF lesions are those found during screening at the time of a contralateral clinical lesion, reported to be present in up to 95% of spaniels. In French Bulldogs we recognise these three scenarios, with a predilection for young to very young puppies with fractures in our practice, as has been documented elsewhere. Across all breeds, condylar fractures have been roughly divided between lateral 56%, medial 11% and the dreaded ‘Y’ or ‘T’ shaped fracture 34% in one larger study of 133 cases; however, Frenchies appear to develop a higher proportion of medial fractures than other breeds. This is something we have also noticed in practice but there are currently no accepted theories on why the more robust side of the condyle would be vulnerable in the French Bulldog.

Fracture Repair
Identification of fractures using standard radiography
is fairly routine; however, there are some benefits to using CT in these cases, with particular emphasis on assessment of the contralateral condyle. A large, ‘cavernous’ HIF on the contralateral limb can suggest that a perfect reduction, with bone fragments ‘clicking back into position’, might not be an achievable goal and alignment of the bone ends at the supracondylar component might be a more realistic aim. In a departure from traditional desire to achieve perfect radiographic reduction in any articular fractures, the presence of a postoperative radiographic intracondylar gap has been shown in one Frenchie-puppy biased cohort to not affect either short-term outcomes or complications and this has been our finding. That is not to say that perfect anatomic reconstruction of humeral condylar fractures in a ‘normal’, adult dog is not an important goal, only that juveniles and in particular those afflicted with HIF can achieve good post-operative outcomes despite seemingly imperfect radiographic reduction due to their HIF.

The type of stabilisation required for a good outcome appears to vary with age also. There has been recent investigations into the best way to repair humeral condylar fractures, both unicondylar and ‘Y-T fractures’, with a tendency towards recommendations for locking plate fixation in conjunction with trans-condylar screws. A lack of complications in the juvenile dog versus adult subset has been found in one study and stabilisation with Kirshner wires (K-wires) alone found to be a successful modality in another study of skeletally immature dogs. This suggests the potential for different management strategies for different aged dogs.

My preference for surgical stabilisation of condylar fractures in adult dogs is to prioritise precise reduction and rigid internal fixation. This typically involves a relatively open approach to the supracondylar component of the fracture to facilitate perfect reduction and plate fixation; however, intra-operative radiographic guidance is typically used to confirm condylar reduction and guide precise transcondylar screw placement. Compression is desirable.

With Frenchie puppies, however, my treatment has evolved to be a largely closed procedure performed with C-arm visualisation. Reduction is achieved manually and maintained first with forceps or a radiolucent condyle-clamp then K-wires placed across the condyle. The trade-off between a lag-screw, providing compression but risking collapse of diseased bone, and a positional screw which allows more threads for security in soft puppy bone is avoided by using cannulated, self-drilling, self-compressing screws. These are well suited to minimally invasive application over K-wires that have been confirmed to be ideally placed using the image guidance. The threads are large and thread pitch varies along the screws length so some compression can be achieved while maintaining good purchase.
in both fragments. Adjunctive K-wires are then placed. A surgical approach becomes necessary where comminution is present and therefore more robust fixation is indicated or where reduction cannot be achieved closed. This is usually due either to complex ‘Y’ or ‘T’ fracture configurations or when chronicity of the fracture prevents fragment movement.

Delayed presentation is an occasional problem, likely stemming from the difficulty of conscious radiography in a wriggly puppy, reluctance to sedate given young age and the fact that condylar fractures can be less palpably obvious than diaphyseal fractures so a wait-and-see approach can be adopted. Chronic fractures can be very difficult to reduce, closed or open, so I encourage a proactive approach to any puppy with lameness localising to the elbow.

This minimally invasive approach is low morbidity and well tolerated. It does not address the potential for non-union, as might be anticipated by possible HIF lesions; however, I have not noted complications associated with this so far. The most common short-term issue is irritation from protruding K-wires, especially those placed medially which is presumably due to ulnar nerve trauma. K-wires can be readily explanted three to four weeks post-surgery if required.

Where plate fixation is performed, some challenges arise from the irregular shape and limited bone stock in the lateral portion of the condyle. In-plane bending of plates is desirable and locking plate-screw constructs to permit adequate fixation from small numbers of sometimes mono-cortical screws are also useful. This difficulty has been specifically addressed by one company that has used multiple CTs to create breed-specific lateral condyle plates, for spaniels and now Frenchies (Lateral Epicondylar Anatomical Plate, Fusion Implants). Medial fractures are more readily stabilised with straight plates.

Management of Clinical HIF

The veterinary literature is yet to catch up with HIF as a cause of lameness independent of fracture in the French Bulldog, but such cases are an occasional presentation at our hospital. Extrapolation from the Spaniel literature is sensible but, with incomplete understanding of the pathophysiology in either breed, care is needed.

Simple transcondylar screw placement in Spaniels has been problematic. Intercondylar instability appears to persist in IOHC despite placement of even relatively large transcondylar screws, resulting in fatigue failure of the implants. Fibrous tissue and dense cancellous bone have been found at the site histologically and could be expected to delay or prevent bone healing.

Two strategies have evolved to address the chronic non-union state and provide long-term stability.

These can be roughly described as biological and mechanical:

A biological approach to spaniel condyles has been attempted for some time, with descriptions of narrow channels being created parallel to fixation screws from the turn of the century. About a decade ago reports arose of techniques whereby a large transcondylar channel was created for the placement of bone graft and adjunctive fixation was applied. Despite results describing achievement of bone bridging using adjunctive cannulated screws this technique has not been taken up by the veterinary
orthopaedic community and another large, multi-fenestrated screw technique does not appear to have advanced beyond biomechanical testing, both presumably for reasons of inadequate efficacy with longer term follow-up.

In more recent years a novel screw (HIRS) has been developed to allow fixation with compression and facilitate trans-condylar bone healing. The ‘dumbbell’ shape of the screw leaves a cavity around the shaft, allowing placement of bone graft or other osteopromotive substances and follow-up CT has shown pleasing efficacy in spaniel patients.

The mechanical approach is of using very large implants, not to facilitate healing, but to avoid fatigue and provide long term stability in spite of chronic non-union. Screw placement has been shown to both provide resolution of elbow pain and protection against further propagation of fissures into fractures but strategies are required to place a screw of sufficient strength to resist fatigue in a relatively narrow safe corridor of bone. Surgeons describe very large screw diameters, such as 5mm in a Springer spaniel, and use of locking screws to maximise core diameter. The 4.5mm shaft screw has found favour due to the lack of thread other than at the trans cortex, providing much higher area moment of inertia at the fissure site compared with a 4.5mm cortical screw. By its nature, this screw provides compression across the condyle; however, the jury is out on the desirability of compression. Screw placement in lag-fashion has been advocated after a finding that this decreased surgical site infection; however, it could be considered preferable to have as much thread-purchase as possible to maintain stability in the long term. Various implants therefore are in popular use; however, there is a common theme of being large to very large in size which makes application challenging.

Surprisingly high complications rates for transcondylar screw placement procedures have been reported, with 60–70% of cases in some studies experiencing infection or seroma. This unacceptable complication rate has been attributed to the lateral-to-medial direction of screw placement following some acceptable results of around 6% infection for medial-to-lateral screw placement presented in abstract only.

It is unfortunate, with the need to place a large screw, that the seemingly superior medial to lateral direction has been found to offer a narrower safe corridor. This makes fluoroscopic guidance appealing and this was the approach used in one of the more recent studies. Disappointingly, medial to lateral screw placement using a cannulated drill system combined with intraoperative imaging still resulted in 36% minor and 21% major postoperative complications in this study, leaving some significant questions. Such complications have not appeared in the small French Bulldog population receiving transcondylar screws in the author’s institution; however, neither over-engineered implants nor a specific effort to encourage healing biologically have been employed so fatigue failure over time remains a possibility. Studies using CT imaging from Frenchies obtained some time post-surgery for clinical HIF will no doubt be produced in time and will help us better understand the course of these lesions better. Specifically, it is important to know whether HIF has any potential for healing in this breed and therefore how analogous the condition is to that of Spaniels.

**Management of non-clinical/ incidental HIF:**

Incidental findings of HIF are largely made during screening of the contralateral limb at the time of imaging for a clinical lesion and the young age of most puppies makes attributing significance difficult. There is little information to guide what is delayed versus failed ossification in the condyle of a three-month-old French Bulldog puppy and there is certainly anecdote to suggest that a proportion (unknown) will proceed to fusion over time. Further unknowns are the potential for healing from screw fixation in a juvenile vs older dog (and Frenchie Versus Spaniel) and therefore the necessity of massive implant use as well as the likelihood of propagation to a clinical lesion over time.

Transcondylar screws frequently violate the physis in juvenile dogs, and the likelihood of this clearly increases with screw size; however, at least when assessed in the fracture situation, this does not result in limb shortening.

A quarter of spaniels with incidental HIF diagnoses were found to progress to a point of requiring surgery in one study (three quarters of those due to fracture) so there is some information on which to make risk based decision-making. Balancing this against the potential for very high complication rates probably works out as a different equation for different dogs/owners.

Many experienced surgeons anecdotally report significantly reduced complication rates with their chosen technique and medial-to-lateral placement and some literature is starting to support this although the reasons for improved outcomes do not appear clear, beyond experience. A manuscript on the HIRS system reports 6% major complications when used for HIF which makes the equation for prophylactic placement more palatable.

A further possibility for these patients that is low morbidity and has apparently found success in some situations is ulnar osteotomy. I have not tried this approach and it remains unproven at this
time; however, the prospect of an easily achieved, relatively low morbidity procedure that results in fusion of HIFs is attractive.

Conclusions

There are clearly many unanswered questions when it comes to HIF in general, and particularly in the French Bulldog breed. Fertile areas for research include:

- The natural progression of HIF in the breed
- Prevalence of HIF in non-clinical Frenchies
- Breed-based analysis of ‘normal’ progression of humeral condylar development
- Potential for HIF healing in juvenile Frenchies with screw placement

Most conjecture at this point in time is in regard to management of ‘incidental’ HIF identified at the time of contralateral fracture. At this point in time, I am inclined to place prophylactic screws in HIF condyles of Frenchies four months or older. I use ‘moderate’ sized self-compressing screws placed minimally invasively and have been happy thus far with outcomes. I expect my approach will change as we develop a better understanding of this disease process.

References


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OBITUARY

Vale Rhondda Canfield

Rhondda Canfield was a passionate teacher, a caring veterinarian, and a wonderful all-round human being.

In celebration of her life, we immediately turn to the effect that she had on others; and they were many.

Being a veterinarian was a source of great pride for Rhondda, coming to it after a stint in another Faculty. She studied Veterinary Science at the University of Sydney from 1968 to 1973. During first year of the BVSc program, she met Paul Canfield and they got married 1971 when they were in 3rd year.

Rhondda spoke of the years of study with great fondness, particularly the camaraderie of studentship, making lifelong friends not just in veterinary science, a time where it seems her quiet confidence and wisdom blossomed.

On graduation, Rhondda worked in Russ Dicken’s practice in Blacktown, first filling in some extra shifts then gradually becoming more and more indispensable. Rhondda worked at Blacktown Veterinary Hospital for a number of years, becoming an accomplished general surgeon during that time, later writing the chapter on Perineal Hernia Repair with Chris Bellenger, in the Textbook of Small Animal Surgery edited by Doug Slatter and published in 1985. Rhondda also contributed to Daria Love’s seminal research into anaerobic infections by providing carefully collected anaerobic samples from cat fight abscesses and cases of pyothorax.

From her first stint living in the United Kingdom in 1979, Rhondda developed a life-long love affair with the English countryside and an important group of friends that she established there. From the early 1980’s Rhondda was afflicted with ankylosing spondylitis, cutting short her career as a surgeon in veterinary practice. However, she redirected her energies to teaching at the University of Sydney as a tutor in the Department of Veterinary Anatomy while completing her PhD on anatomical aspects of perineal hernia in the dog. Rhondda received her PhD in 1986 and continued as a Professional Officer in the Department until transitioning to a Lectureship in 1998.

Rhondda was a focal point of the student experience. She supported and mentored students with great wisdom and compassion, naturally progressing to become Sub-Dean for Student Welfare and then Associate Dean, Students. Rhondda brought to this role her quiet, calm, competent, stable personality. This was coupled with genuine compassion, empathy, and emotional intelligence. She had a substantial and enduring impact on generations of veterinarians in this role, helping students overcome personal difficulties, manage disabilities, and achieve effective learning in veterinary science to become valued veterinary colleagues. Many students would not have got through the program if it were not for Rhondda, and then have gone on to make very capable practitioners. The value of this cannot be underestimated. All graduates of the Faculty of Veterinary Science, who studied between the early 1980’s and 2008, when Rhondda retired, will have

Rhondda on sabbatical in the UK. She loved the spring bulbs.
Some may say the world is a little darker now that Rhondda’s light is no longer shining. But I say no, this is not the case as Rhondda has left behind a part of her flame in all of us, her former students. Knowing that this flame lives on in thousands of her former students worldwide is a fantastic legacy that she leaves behind.

Rhondda, I am so grateful to have been one of your students. Who would have thought as we sat in your class as first-year students thinking you were only teaching us about embryology? You were preparing and shaping us for our future lives. Thank you for your teaching, guidance, and friendship.

-John Waterhouse

Rhondda was an enthusiastic and committed teacher with a genuine passion for veterinary anatomy. She was genuinely interested in developing students’ knowledge and understanding of the discipline to a high standard. As a research collaborator, she was meticulous, skilled, and generous with her time but never one to blow her own trumpet.

Rhondda was always generous in making time to support students and her colleagues, both academic and non-academic. As Sub Dean (Students) she was tireless in her striving to improve support for students in difficult circumstances. She was empathetic, kind and caring but was no pushover when it came to calling out unfairness and would not hesitate to go in to bat for those who needed support in difficult times.

She was a highly respected colleague who also made a positive personal impact on those around her and will be sorely missed by those who worked closely with her.

-Sue Hemsley

Rhondda with a koala patient
Rhondda and Holly. Rhondda had a thing for big dogs – especially Boxers and Rottweilers.

When I started in anatomy, I was taking over some of Rhondda’s practical classes. Truthfully, I was completely overwhelmed, something Rhondda immediately understood and empathised with. While under no obligation to do so, she helped me ‘ease into it’, patiently dissecting alongside me and engendering in me the confidence to take on what at first seemed impossible. As I ‘grew into’ Rhondda’s classes, I could see what a treasure trove they were, designed with skill and a keen awareness of the importance of relating complex anatomical detail in an accessible and practical manner. Even in anatomy practical classes, her concern for student welfare was obvious! Rhondda’s was a rare combination of empathy, generosity, and capability. I am a better teacher for her mentorship, and that is a gift for which I will always be grateful.

-Corinna Klupiec

Rhondda Canfield was a gifted teacher. She was also unfailingly supportive, inclusive, and understanding of students who found themselves suffering from transient or chronic physical, emotional, or mental health issues. She truly appreciated the challenges of the BVSc degree, and the extent to which – if not managed – certain conditions or temporary life circumstances like bereavement had the potential to derail a vet career before it began. She kept many of us ‘on the rails’, often drawing from and generously sharing her own life experiences. Personally, I count her as one of a small group of people without whom I would not be a vet today.

-Anne Quain

Dr. Rhondda Canfield’s role at the University of Sydney may have been that of Associate Dean of Students in charge of Student Support at the Faculty of Vet Science, but Rhondda dedicated her life to so much more than just that role. Not only did she support the current students within the Faculty while she was there, but she continued to also support her former students who had left the comfort of the Faculty, those who had already graduated and moved on with their lives and with their careers. You see, Rhondda was someone who was a supporter of anyone who needed a kind and nurturing soul to talk to. I remember specifically, years after graduation, I visited the campus and ran into Paul and Rhondda in the hallway. We casually chatted about what was going on in our lives. Rhondda, recognizing the complexity and the emotions surrounding a huge decision I had to make within my own life, immediately gave me a hug and held me until she knew I was going to be OK. She cared enough to be human, compassionate and to be that rock for all those who needed a sympathetic friend. Even now, years later, I remember with great fondness, how nurturing and beautiful Rhondda was. I am so grateful for having known, and having learned from, such a genuine, authentic, and caring person. Thank you, Rhondda. We love you.

-Celia Waterhouse nee Romeroso

The personal accounts here show just a small part of the effect that Rhondda had in this world. Rhondda was a pillar of strength, quiet worker against injustice, dedicated teacher, and constant supporter of students in the Faculty of Veterinary Science.

She was the much loved and respected wife of Paul, dearly treasured sister to Ceridwen, David, Patricia, and Vicki. Beloved Aunt of Jamie and Lucy, Charlotte, Estelle and Charlie, and a friend to all. We will miss her

Written and compiled by Mark Krockenberger and Richard Malik for the Sydney School of Veterinary Science.
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