C&T

Control & Therapy Series

Issue 308 | September 2022

THE UNIVERSITY OF SYDNEY

Centre for Veterinary Education

Leaping ahead

Advances in Dog & Cat Lameness Management

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Dr Claire Adams

Online now

Dr Henry Jones

I hear you guys switched to a mucosal vaccine?

Dr Claire Adams

Yes, we're using Bronchi-Shield 👅 Oral

Dr Henry Jones

What prompted the change?

Dr Claire Adams

We all agreed the benefit of increased mucosal immunity to protect against respiratory disease was worth it!

BTW, mucosal vaccines are also strongly recommended by WSAVA for high risk environments such as shelters[1] 🏠

Dr Henry Jones

We considered it but it's a hassle to change protocols

Dr Claire Adams

We got the rep in, they sorted out the vaccination protocol and trained our staff. The new protocol worked out to be more cost effective too! 👍

Dr Henry Jones

We're also not sure if our clients would like it... 🤔

Dr Claire Adams

Hmmm...if the flu vaccine came out as an oral mucosal form, how many people would still stick with injections?

Dr Henry Jones

...

Simply administer 1 mL of Bronchi-Shield Oral in the buccal cavity for 12 months' protection against *Bordetella bronchiseptica*. Protects puppies with a single dose from 8 weeks of age. Over 2 million doses sold in Australia[2].

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Reference: 1. Day, M., et al (2016) WSAVA Guidelines for the vaccination of dogs and cats. J Small Anim Pract, 57(1), E1-E45. 2. BIAH Data on file.

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***Control & Therapy* Series**

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The *C&T* is not a peer reviewed journal. We are keen on publishing short pithy practical articles (a simple paragraph is fine) that our readers can immediately relate to and utilise. And the English and grammar do not have to be perfect—our editors will assist with that.

Join in—write up that interesting case

C&T authors agree that it is extremely satisfying to read their articles in print (and the digital versions) and know they are contributing to veterinary knowledge and animal welfare.

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FROM THE DIRECTOR

I very much feel like we are witnessing a moment in history. No, I'm not talking about pandemics or climate change – but rather a cure for feline infectious peritonitis.

It's extraordinary to consider that a very short time ago, cats suffering FIP had a grim prognosis. And now with timely intervention and reasonably accessible medications, long term remission is possible.

What makes this breakthrough even more incredible is the generosity of vets in sharing their experiences and protocols to empower more clinicians to save the lives of their feline patients. Noone is 'knowledge hoarding' the information for gain, but rather freely disseminating it to others, including practical pearls and tips to make treatment more successful and accessible. What an incredible contribution to animal welfare. So thank you David Hughes for the update to your previously published FIP treatment protocol in this edition of the ***C&T*** – and to the many others (particularly Jacqui Norris, Sally Coggins and Richard Malik) at the forefront of this revolutionary approach.

Speaking of generosity, Tim Portas and RSPCA Queensland have allowed us to publish their comprehensive guide to veterinary management of wild possums and gliders—a fantastic resource to keep readily accessible within your clinic. And if you are looking to improve your approach to wildlife care, make sure your nurses enrol in our Wildlife Friendly Clinics: Fundamentals and Triage and Care WebinarPLUS NurseEd courses (free to members), presented in collaboration with WIRES.

Finally, don't miss Moira van Dorsselaer's story on nursing a cat recovering from head trauma – it's honest, uplifting and heart-warming.

Happy reading.

Simone

MAJOR Winner

The prize is a CVE$400 voucher

Small

UPDATE ON C&T NO. 5896: A FELINE INFECTIOUS PERITONITIS (FIP) TREATMENT PROTOCOL

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C&T No. 5940

eBook download
C&T No. 5896 A Feline Infectious Peritonitis. Treatment Protocol, Issue 304, Sept 2021

Please note: Coronavirus titres or faecal analysis for the diagnosis of FIP are not recommended or required.

Please refer to the previous article C&T No. 5896 for a discussion on the disease and diagnostic options for FIP.

The Concord Veterinary Hospital Treatment Protocol

This article will discuss the use of a drug called remdesivir injectable liquid and the legally compounded GS-441524 tablets. Many clinicians will be familiar with GS-441524 from its use in Australia and overseas to treat FIP and a time when cat owners would have to source this drug independently and on the black market.

What are Remdesivir and GS-441524?

Remdesivir and GS-441524 are broad-spectrum antivirals. Remdesivir was originally developed to treat Hepatitis C and Ebola Virus; however, it was fast-tracked and given approval in many countries worldwide for the treatment of COVID-19. Its use in COVID-19 patients is somewhat controversial; however, it is showing amazing promise for the treatment of FIP in cats. GS-441524 was developed for human use; however, allegedly because of oral bioavailability issues, the manufacturer concentrated on other molecules, so its use is confined to coronaviruses in animals.

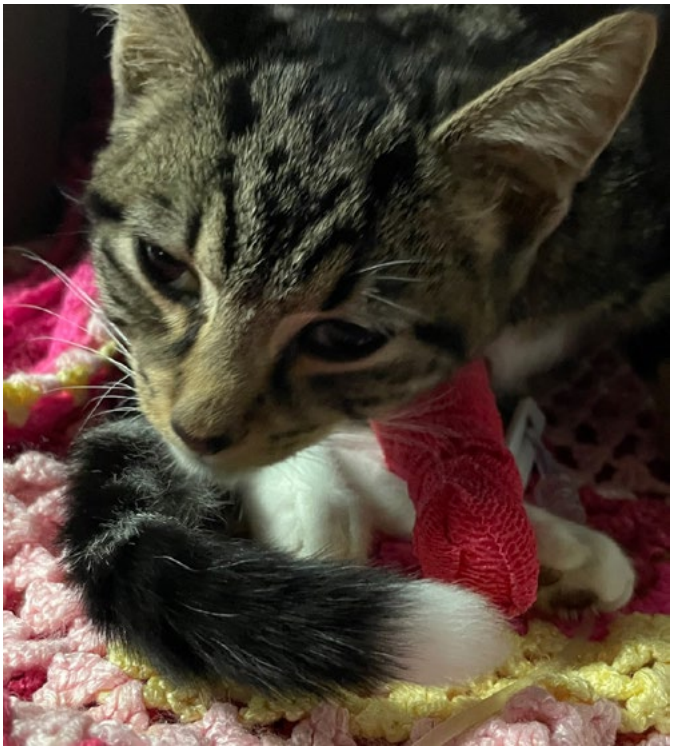

Figure 1. Before treatment

Figure 2. After treatment

Treatment With Remdesivir and GS-441524

Previously, treatment with remdesivir was for 84 days by subcutaneous injection. Remdesivir was the first commercially available anti-viral provided in Australia for treatment of FIP and it was highly successful in treating this disease; however, it was not completely effective in all cases—especially neurological and ocular FIP. We have driven many cats into permanent remission with remdesivir alone; however, there were a few patients where FIP remission was not achieved, or only achieved after many months.

Remdesivir can be obtained from BOVA Compounding who ship Australia-wide and usually overnight. The vials are $275/vial inclusive of GST and the final concentration is 10mg/mL. Dose recommendations range from 10–20mg/kg once to twice daily.

GS-441524 was available for owners to purchase on the black market as an injection and as tablets and capsules. In NSW at least, sourcing GS-441524 by a veterinarian was not permitted.

BOVA Compounding now produces a commercial supply of GS-441524 tablets. The tablets are 50mg, 10 tablets per bottle, at a cost of $660/bottle including GST. Dose range is 10–20mg/kg daily, either given as a single dose or divided and given twice daily. GS-441524 is slightly more economical than remdesivir to use. There are no injections and no need to purchase needles, syringes and EMLA cream.

The First 1 to 4 days

At this stage, Concord Veterinary Hospital is still using injectable remdesivir for 4-14 days prior to the introduction of GS-441524. The rationale behind this is that intestinal absorption of drugs is potentially limited in the first 2 weeks of treatment. A systemic route is preferred for this reason. However, GS-441524 is relatively cheaper when compared with remdesivir, so should a client be financially limited, it is certainly reasonable to commence with GS-441524 tablets.

The earlier treatment is commenced—the better the outcome—and this cannot be overstated. Most cases (there have been very few who have not made complete remission) that did not proceed to permanent remission were treated very late in the course of the disease.

Here are 3 options for commencing treatment. Like most things in life, it often comes down to budget.

1. **Subcutaneous Injections**
   If a patient is stable and eating, subcutaneous injections can be commenced at a dose rate of 10-20mg/kg once to twice daily, (wet FIP requires lower doses than dry FIP and abdominal FIP requires lower doses than neurological and ocular FIP). The patient is checked daily for the first 3–4 days. This is a perfectly acceptable routine for a well-hydrated patient who is eating and is the more financially sustainable option.

2. **The intravenous (IV) Route**
   If a patient is unwell, or where neurological or ocular FIP is suspected, then they should be hospitalised, placed on IV fluids run at a quarter to half maintenance rates (Hartmann's or similar is fine). The reason for such a low rate will become clear shortly.

   Remdesivir is administered SLOW IV (over 10 minutes is fine) once to twice daily for 3–4 days. We have found if you can maintain your catheter to a 4th day it is great to get a fourth intravenous dose into your patient; however, if there is pain at the cannula site or thrombophlebitis, then 3 days is also OK.

3. **The Oral Route**
   If a patient is stable and eating, or where finances dictate, oral GS-441524 can be administered from day 1 of treatment at a dose of 10-20mg/kg daily, given as a single dose or as divided doses. Once again—higher doses are required for dry, neurological, and ocular FIP.

Pros and Cons

The downside to administering remdesivir intravenously is:

- having to hospitalise the patient
- the added expense to the owner, which is not insignificant
- an increased risk (perhaps 10% risk) of developing or worsening of a pleural effusion with the use of intravenous remdesivir plus IV fluids

This is an issue mostly in wet forms of FIP, which then require close monitoring by either regular thoracic radiography or T-Fast ultrasonography daily or twice daily (we prefer twice daily radiographs).

The benefits of intravenous remdesivir are that;

- you get a high anti-viral dose to target the virus for the initial 3–4 days
- you get to observe your patient and often get to see the fever break within 1-2 days

and near moribund patients 'come back to life' and show signs of improvement daily

Obviously, this is a conversation to be had with each individual owner and for the clinician to make on a case-by-case basis. See below for other considerations before deciding which patients would or would not benefit from initial IV or subcutaneous remdesivir therapy *versus* oral GS-441524.

Cats receiving intravenous remdesivir do not need to be in a 24-hour facility—remdesivir could be administered intravenously in the mornings so a clinician then has the whole day to observe their patient, and then send home for overnight care with the indwelling canula in place for the following days treatment.

Days 5-14 to 84

Patients should ideally be switched from remdesivir to oral GS-441524 on day 4-14, depending on how systemically well they are. The minimum treatment course is 84 days in total, with some severe cases requiring longer. We would suggest that dry, ocular and neurological disease is dosed (GS-441524) at 20mg/kg and wet forms at 10-15mg/kg. In our experience there is a tendency for vets to reduce the dose of antivirals. We recommend not dosing to lean body mass or minus their effusion weight estimations.

Remember – kittens metabolise drugs faster and require a relatively higher dose on a mg/kg basis.

Monitoring

There is little point doing blood work on days 1–4 after commencement of treatment—nothing will have changed significantly.

We routinely perform blood work at weeks 4, 8 and 12 of treatment, and decide on cessation of treatment at the 12-week mark. We then perform blood work 4 weeks into the observation period and 12 weeks into the observation period.

If results are excellent (i.e., normal) then they are considered cured.

Ideally, we perform CBC and biochemistry panel including bilirubin and globulins. We are looking for improvements and resolution of anaemia, neutropenia, lymphopenia, hypoalbuminaemia, hyperglobulinaemia and hyperbilirubinemia. A mild peripheral eosinophilia is a favorable sign.

In theory, you could also perform a PCV, TPP and a globulin level. This will give you an idea of the anaemia, the colour of the serum to check if your patient is still jaundiced, the albumin and the globulin level by simple calculation.

Treatment ends when all analytes are normal and critically, globulin levels are well within the normal range.

A few patients have ended treatment when globulins are high upper end normal and have rapidly come out of remission.

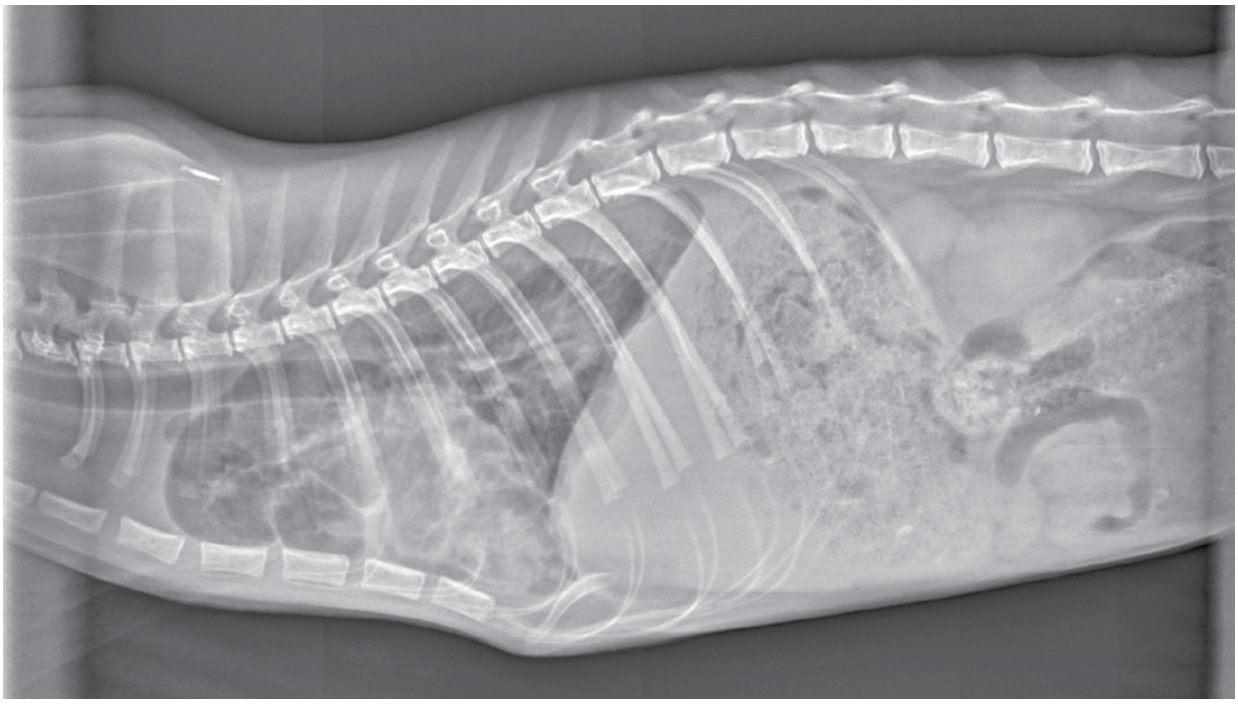

Figure 3. Radiograph showing patient's pleural effusion

But How Long Do They Last and How Many Have Died?

We get asked this a lot.

Since changing our protocol to include GS-441524—the remission rate is approximately 95%.

Timely diagnosis is vital. The cases that have not survived have presented to us moribund or have had comorbidities such as concurrent FIV or lymphoma.

It is worth noting—we have two cases currently, one ocular and one simple dry FIP—where we have been unable to reduce the globulins to a normal level beyond 84 days using remdesivir alone but once switched to oral GS-441524 both cats are now in full remission.

Other unsuccessful cases are predominantly ones we have seen as second opinions where the dose of antivirals has been too low.

To date, we have seen no side effects from remdesivir or GS-441524, although self-limiting increases in SDMA, urea, creatinine and ALT activity have been reported. Cessation of therapy is not normally required or recommended in these cases.

10 Important Points About the Use of antivirals in FIP cases:

1 THE MOST IMPORTANT POINT TO NOTE: 10% of cats receiving initial treatment (first 1–4 days) with intravenous remdesivir or oral GS-441524 can develop life threatening pleural effusion—especially in cases of wet FIP. These will require drainage. Therefore, it is recommended that cats receiving these antivirals should have serial radiographs and/or skilled T-Fast ultrasonography on a daily or bi-daily basis. If a patient is hospitalised for IV fluids and IV remdesivir, pleural effusions are more likely if a clinician pushes the intravenous fluid administration beyond maintenance—we prefer to keep the fluid rate below half maintenance. It is hard to resist the temptation to increase fluid rates in unwell and often dehydrated patients, but we would recommend against this.

2 Many patients will become quiet for 1–3 hours after receiving remdesivir intravenously—the exact mechanism is unknown.

3 There is no need to be alarmed if this occurs. It is generally a good sign. It is recommended to NOT drain the abdominal fluid unless to get diagnostic samples or for urgent therapeutic reasons (e.g., if dyspnoea is severe due to pressure onto the diaphragm). Also, somewhat bizarrely—pica has been reported in many cases of FIP where treatment has been suboptimal (usually dose related) or where remdesivir therapy is insufficient. Eating of cat litter is common, especially if they are anaemic. This is often the first sign that FIP is coming out of remission, and the dose should be immediately increased, and drug switched to GS-441524 if not already being administered.

4 Globulins will often go up at the first blood test before they begin to come down. Do not get disheartened. It's the protein in the fluids appearing in the circulation.

5 Many vets are concerned about the reports of elevations in kidney and liver analytes with the use of remdesivir. There have been a few reports of elevations in SDMA and occasional reports of elevation in ALT with doses of remdesivir as high as 15mg/kg that resolved once the dose was returned to normal. The authors have had 2 cats on 15mg/kg of remdesivir and has not personally seen elevations in ALT (but have not checked SDMA for various reasons).

6 Antibiotics: Often FIP cats have co-morbidities with infections which require treatment with antibiotics. Infections such as haematrophic Mycoplasmas are common. Antibiotics such as doxycycline or marbofloxacin are often a good choice. Other antimicrobials—if indicated—do not appear to be contraindicated. Some cats have translocated enteric bacteria in effusions.

7 NSAIDs to bring fevers down are often NOT required with the use of remdesivir.

8 The concurrent use of corticosteroids with remdesivir is not strictly contraindicated; however, it is not recommended. If your patient is already on corticosteroids, it is recommended you taper them off as quickly as possible.

9 Remdesivir and GS-441524 are well tolerated by cats and GS-441524 does not appear to be bitter when administered orally or crushed in food. Both can be obtained from BOVA Compounding and are usually shipped overnight Australia-wide.

10 Often it can be beneficial to a patient to commence a treatment trial of remdesivir to see if a presumptive diagnosis can be made of FIP or whilst waiting for further diagnostics to return from the lab. We would encourage early treatment and intervention prior to a formal diagnosis as the few days waiting for results can make or break a case ◆

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We look forward to you joining us online!

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DEVELOPING QUALITY VETERINARY COMPOUNDING SYSTEMS

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Treatment for FIP is challenging, however at Bova Aus we have developed formulations that can improve patient outcomes and help in the treatment of FIP. The GS-441524 tablet and Remdesivir Injectable Liquid discussed in this edition's Update on C&T No. 5896: A Feline Infectious Peritonitis (FIP) Treatment Protocol by Dr. David Hughes, are part of our quality assured range and have undergone a strict batch compounding process so that we can guarantee the quality of both.

Our system to pre-prepare and quality assure products is compliant with the AgVet Code while our quality control methods follow USP or BP pharmacopeia standards and strive to meet international validation standards set by the European Medicines Agency. To ensure quality medicine at Bova Aus, we follow the steps below for our Quality Assured (QA) product range:

Raw Material and Production

Manufacturers of raw materials are accredited GMP, TGA or FDA licensed facilities and all active pharmaceutical ingredients (API) have a "Safety Data Sheet" and a "Certificate of Analysis". An additional "Certificate of Sterility" is required for materials that are to be used in sterile compounding.

Before any batch is made the process is first validated and a Standard Operating Procedure (SOP) is written. The staff preparing the batch are trained and qualified to produce the exact formulation every time and any equipment used is validated to ensure it is fit for purpose. The creation of SOP's and Process Validation is documented by an experienced technician in a controlled lab setting. The Pharmacist in Charge ensures that the compounded batch is in compliance with any state, territory, or Commonwealth law applicable.

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Quality Control

Method Development and Validation

Once a testing method is developed it is validated in accordance with US (USP) or British (BP) Pharmacopoeia standards, alongside the APVMA Guidelines. Parameters assessed during method validation are selectivity, linearity, range, accuracy, precision, robustness, and forced degradation.

Stability Study

Stability studies are performed on all QA formulations, including GS-441524 and Remdesivir Injectable Liquid and they have been tested in accordance with the European Medicines Agency guidelines. These guidelines are used to establish the stability testing protocol for each of our QA formulations and are accepted worldwide. Stability studies are performed by an APVMA accredited lab using samples taken from the primary batch. The stability study protocol may include Real-Time and Accelerated Stability Testing, Freeze, and Cold Stability tests. Upon the completion of all stability tests, a Stability Study Report is produced and cross-referenced against product-defined specifications to determine a suitable beyond use date.

End Product Testing

All QA formulations are end-product tested. The properties analysed during this stage include physical, chemical, and microbiological properties and testing parameters are dependent on the formulation and physical attributes. Chemical tests are used to determine the assay of the end-product whilst physical tests are performed in accordance with USP or BP official monographs using parameters specific to each formulation, for example, GS-441524 tablets are tested on hardness, thickness, friability, disintegration and uniformity of mass.

Summary

Bova Aus has a unique Quality Assurance program that applies to all of its QA products. This ensures the highest possible standard of medicine, as we know that the quality of a compounded medication is a vital component of a successful clinical outcome.

Want to learn more about FIP?

Check out the FIP webinar on our Bova Scholars webpage

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WHAT'S LURKING IN AUSTRALIAN DOG PARKS?

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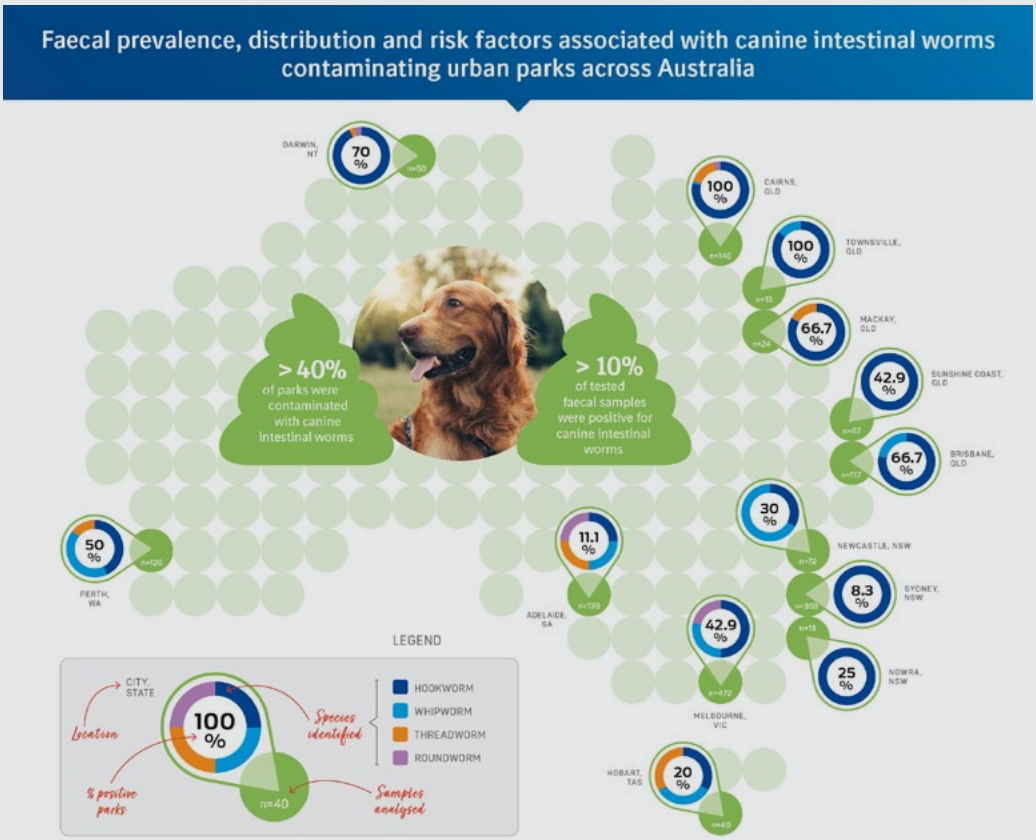

Dog parks represent an ideal opportunity for dogs to be dogs; to run about and socialise in a safe environment while at the same time also providing their owners the chance to mix with other like-minded dog owners.

However, as is the case with any location where animals congregate in numbers, dog parks may increase the risk of exposure to various infectious or parasitic organisms. Of particular concern in dog parks are canine soil-transmitted helminths, including roundworm, hookworm and whipworm. These worms may not only have an impact on animal wellbeing, many of them also have an impact on public health due to their zoonotic potential.

To better understand the risk of exposure of dogs and their owners to these parasites, a research team from The University of Melbourne, led by Professor Rebecca Traub, recently investigated the prevalence of canine soil-transmitted helminths in dog parks around Australia. The study involved the collection of 1,581 faecal samples from 190 different parks across Australia and analysing them with faecal flotation and PCR for the presence of the eight known canine soil-transmitted helminth species in Australia.

The study showed almost half (42.6%) of parks sampled were contaminated with at least one species of canine soil-transmitted helminth, with on average more than 1 in 10 faecal samples (12.7%) testing positive. Hookworm was the most prevalent parasite detected in this study (10.2% of samples), with *Ancylostoma caninum* the most common and widely distributed hookworm species found. Whipworm was the next most prevalent parasite in this study, detected in 1.3% of samples with a wide geographic distribution. This study also demonstrated one of the challenges associated with diagnosing whipworm using faecal flotation as, despite being performed by expert parasitologists, over 75% of positive samples detected with PCR were missed on faecal flotation. Threadworm (*Strongyloides* spp.) was found in 1.2% of samples, and roundworm in 0.7%. Worm prevalence and species breakdown varied across different climatic zones as shown above. Scan the QR code to learn more about the study and implications for your parasite control programs.

Small

TREATMENT TIPS FOR TRICKY AREAS

Dr Áine Seavers MVB MRCVS

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C&T No. 5941

Pedal lesions are notoriously tricky for owners to treat since many dogs hate their feet being touched even on a good day, let alone when their feet are sore and sensitive. Therefore, the access to a product needing just a once per week application for approx. 6 weeks and for that medication to be in a soothing lotion format, cannot be underplayed in these pedal presentations.

For such a product to then also use a low skin atrophy/systemic absorption risk glucocorticoid such as Budesonide, and to marry that with an emollient co-ingredient addressing trans epidermal water loss (TEWL), is to turn this medication a clinician's dream topical product!

The usual caveats about use-i.e. first getting a good diagnosis and avoiding inappropriate use in certain disease presentations still hold true: but the new BARAZONE product from Dermcare seems to offer me a new treatment option for so many in my allergic patient cohort.

I can't remember when I was last this excited about a new product for front-line clinician. I can just visualise my stress- from so many recalcitrant pedal licking cases -drift away, now I have Barazone in my treatment armoury ◆

Further Reading

Ahlstrom L, Cross S, Mills P, 2012, The effects of formulation on the penetration and retention of budesonide in canine skin in vitro, *The Veterinary Journal*.

Small

FUSOBACTERIUM NUCLEATUM INFECTION IN AN EGYPTIAN MAU TOMCAT

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C&T No. 5942

Case summary: An ten-month-old Egyptian Mau tomcat was presented with exophthalmos, swelling of the left side of the face and pterygopalatine fossa and a draining tract just above his left eye. His owner was on therapy for *Mycobacterium tuberculosis*. Samples were taken for 16S rRNA PCR and Sanger sequencing; Aspergillus PCR examination and Mycobacterium infection was excluded by PCR. Sanger sequencing came back with strong signal for *Fusobacterium nucleautum*. NSAID and fluoroquinolone therapy was started and he was improving, but after 14 days, the exophthalmos returned, and central nervous system clinical signs occurred. Antibiotic therapy was changed to cephalexin for 3 weeks and spiramycin with metronidazole fixed combination was given for 1 month. This led to full recovery of the cat and no recurrence of clinical signs.

Relevance and novel information: Use of 16S rRNA PCR and Sanger sequencing in diagnosing the cause of abscesses in cats is quick and useful.

Introduction

Bacterial infections of the orbit are seen relatively frequently in feline practice. Bacteria may reach the orbit in many ways: haematogenous dissemination, traumatic (including iatrogenic) penetrating injuries, migrating foreign body, or spread from contiguous regions such as alveolar tooth roots, the nasal cavity, frontal sinus[1] or chronic otitis externa, media and interna.[2] Another cause could be fungal microorganisms causing orbital infection as extension of sino-nasal

infection, most commonly caused by *Aspergillus spp.*[3,4]

Typical history is acute onset of exophthalmos, globe deviation, nictitans protrusion and reluctance to eat. On examination there is restriction to globe retropulsion, slight prolapse of the nictitating membrane, extreme pain on jaw opening and swelling of the pterygopalatine fossa.[1] Diagnosis is confirmed by ultrasonography, it is important to rule out dental disease, orbital foreign bodies and extension of sino-nasal disease, and for this CT or MRI is sometimes needed.[5,6]

Therapy includes draining of the abscess with ultrasound guidance or by transoral approach, when pterygopalatine fossa is swollen.[1] Samples need to be sent for cytology, and bacterial culture for susceptibility testing. The most frequent genera identified include Pasteurella and anaerobic bacteria such as ***Bacteroides spp.***[7] But if anaerobic bacteria are suspected, PCR testing is superior to bacterial culture unless a specialist anaerobic culture laboratory is available.[8] Systemic antibiotic course should be protracted, at least 1 month, to reduce risk of recurrence. It is also important to provide pain relief.[1]

Figure 1. Photo of the patient at initial presentation

Case description

A ten-month-old Egyptian Mau tomcat was brought from the breeding cattery at the age of 3 months. At the cattery, he was housed indoor and outdoor in the cat pens with many other cats. There were occasional scratch and bite wounds observed. He lives as the only cat and is indoor-only now. His owner was released from the Mycobacterial center in one of the Prague hospitals just before he brought the kitten. His owner was undergoing therapy for *Mycobacterium tuberculosis*.

His vaccinations were up to date, but his current owner had never dewormed him. He was fed with good quality commercial food. His toileting was normal, he did not urinate outside the litter tray. The owner did not report any changes in environment but had observed behavioral changes in the previous 9 days—he was much more sleepy than usual, and he had a slightly reduced appetite and the third eyelid of his left eye was protruded. He had visited another vet 7 days previously, but they did not find any clinical changes. He had also started to sneeze 4 days ago, and his head started to look `different'.

He was a nice young entire tomcat, easy to handle, but was quiet and dull on examination. He was reluctant to move around the room, but when he moved, he did not have any obvious mentation or gait abnormalities. His body condition score was 3/9 and weight 3.5 kg. His body temperature was 39.9 °C, RR 60/min, HR was 150/min without any murmurs and CRT was 2 sec. His right conjunctiva was normal, his left conjunctiva was mildly swollen and dark pink. He had a draining fistula just above his left eye 1 cm deep, the left side of his face was hugely swollen (hard on palpation), and the swelling was apparent also in the left side of his mouth (left pterygopalatine fossa—caudal to the last upper molar tooth on his left side), no bruising was present. He had no evidence of dental disease but opening of the mouth was painful for him. He had very mild protrusion of his third eyelid, ptosis of upper eyelid and exophthalmos on his left eye, together with medial strabismus of this eye (Figure 1). A full neurological exam was not performed, but his palpebral reflexes were present, pupillary light reflex was normal, menace response was normal on both eyes, he was not suffering from nystagmus and his oculocephalic reflex was present and he did not have anisocoria. His left eye was harder on palpation and painful when touched and there was restriction to globe retropulsion. His retropharyngeal and mandibular lymph nodes were enlarged on the left side, other lymph nodes were normal.

Problem list

1. anorexia
2. apathy
3. fever
4. sneezing
5. facial swelling
6. draining tract above the left eye
7. orbital disease (exophthalmos, medial strabismus, third eyelid protrusion)

Diagnostics

Based on history and signalment, hematology, serum biochemistry (Table 1 and 2), and retroviral status was performed and was negative.

TABLE 1

HAEMATOLOGY	DAY 1	DAY 14	DAY 38
RBC (5-10 T/L)	11.4	8.11	10.9
HCT (24-45 %)	53.8	36.4	53.3
MCV (41-51 FL)	47	45	49
PLT (300-600 G/L)	980	401	699
WBC (7-17 G/L)	15.9	33.2	13.3
HGB (80-150 G/L)	136	97	138
MCH (13-18 PG)	11.9	12	12.6
MCHC (310-350 G/L)	253	266	259
NEU SEGM. (50-75%)	77	90	51
NEU BAND (%)	2	2	1
LYMFO (15-50%)	17	5	40
EOS (0-6%)	3	3	6
MONO (0-4%)	1	0	2
BAZO (0-1%)	0	0	0

TABLE 2

BIOCHEMISTRY	DAY 1
FELV/FIV IDEXX SNAP TEST	NEGATIVE
GLU (4,11- 8,84 MMOL/L)	7.06
UREA (5,7-12,9 MMOL/L)	8.3
CREA (71-212 µMOL/L)	144
PHOS (1-2,42 MMOL/L)	2.36
CA (1,95-2,83 MMOL/L)	2.43
TP (57-89 G/L)	90
ALB (22-40 G/L)	27
GLOB (28-51 G/L)	63
ALT (12-130 U/L)	49
AST (0-48 U/L)	71
ALKP (14-111 U/L)	10
GGT (0-4 U/L)	7
TBIL (0-15 µMOL/L)	14
CHOL (1,68-5,81 MMOL/L)	2.54

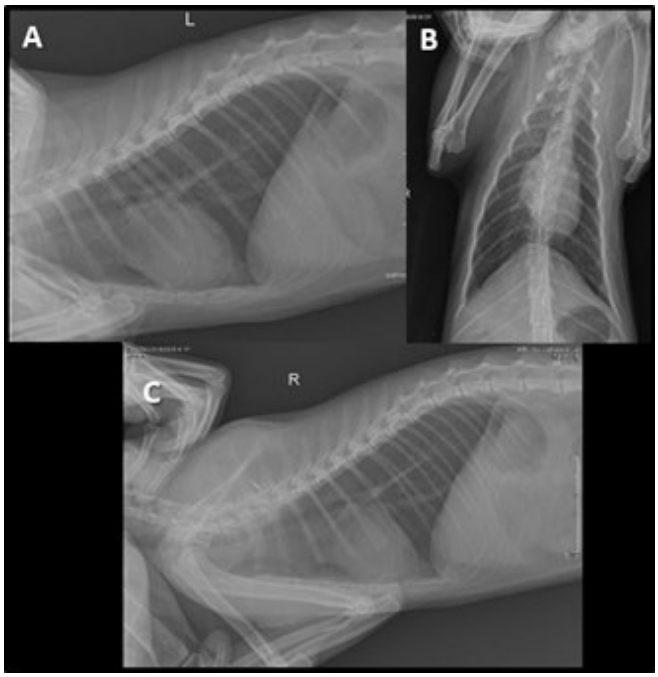

Figure 2. Orthogonal chest radiographs

Hematological changes include mild erythrocytosis and slightly elevated leukocytes with mild neutrophilia and marked thrombocytosis. Biochemical changes included hyperproteinemia with hyperglobulinemia and elevation of AST and GGT. Chest radiography was performed to check for signs of pneumonia and masses (Figure 2 A-C).

A bronchial lung pattern was observed, but no obvious signs of pneumonia, oedema or masses were found.
Histology examination of submandibular lymph node was performed, with result of lymph nodal paracortical expansion with sinuses, histiocytosis and neutrophils with atypical round cells with multifocal plasmacytoid appearance.

The lymph node parenchyma was characterized by a reactive process in association with an unusual population of round cells presenting signs of atypia and multifocally moderate number of mitoses. A neoplastic process, like for example lymphoma, could not be excluded but a severe reactive process was also considered in differential diagnosis.

Immunohistochemical examinations for CD3, Pax5 and Mum1, and PARR test examination on sample submitted was recommended to evaluate the origin, distribution and the phenotype of the cells, and to evaluate the eventual presence of clonal lymphoid proliferation.

Due to financial constraints, however, no further diagnostics were performed on the histology samples. However, the histopathology blocks were kept just in case the treatment outcomes with therapy were poor and PARR clonality was necessary.

PCR analysis to detect *Mycobacterium tuberculosis* and *Aspergillus spp.* from a smear of the deep draining tract and an incision sample of lymph node was negative. 16S rRNA PCR and Sanger sequencing of bacteria revealed infection of anaerobic bacteria ***Fusobacterium nucleatum***. This is an obligate anaerobic bacteria of the type often found in retrobulbar abscesses/cellulitis.

Treatment

Infusion therapy with cold Plasmalyte was started to lower the temperature together with meloxicam (0.1 mg/kg SC).

Since we considered *M. tuberculosis* infection in the DDx, enrofloxacin 5mg/kg was administered s.c., because literature in human patients considers quinolones as a possible choice in treatment of tuberculosis.[9,10] This dose should be safe and not cause retinal damage in cats.[11]

The draining wound above the eye was washed with diluted betadine 1:10 and 0.5 mL of Panalog cream (Nystatin-Neomycin Sulfate-Thiostrepton-Triamcinolone Acetonide Cream) was given into the wound.

Since he was anorectic and painful, a nasal feeding tube was administered for assisted feeding to reach his MER. Vital trunk katze liquid was used, the recommended volume for feeding in 24 hours was 80 mL of the liquid – starting at 1/3 of MER.

The next day he started to eat on his own and his temperature was normal (38.7°C). His owner was unable to take care of the feeding tube properly (washing with the water), so it became obstructed and the next day it was removed. He started to play at home and was more active. Enrofloxacin SC SID was changed to safer quinolone pradofloxacin 5 mg/kg given p.o. SID. Meloxicam 0.1 mg/kg p.o. SID was continued. His wound was treated with betadine and Panalog at home once daily.

He was much improved by day 3, eating on his own, the facial swelling was much reduced, the draining wound above his left eye was smaller too, but exophthalmos was still present after 4 days. On Day 7 an abscess had formed on his left cheek (*Fusobacterium nucleautum* was the suspected cause), there was no exophthalmos present, the draining tract above his left eye was already closed, covered just with a small crust, no swelling of the face was present (Figure 3A). He was active, playful, eating his full meal and his temperature was normal (38.6°C). Debridement of necrotic tissue was performed together with lavage with betadine (Figure 3 B).

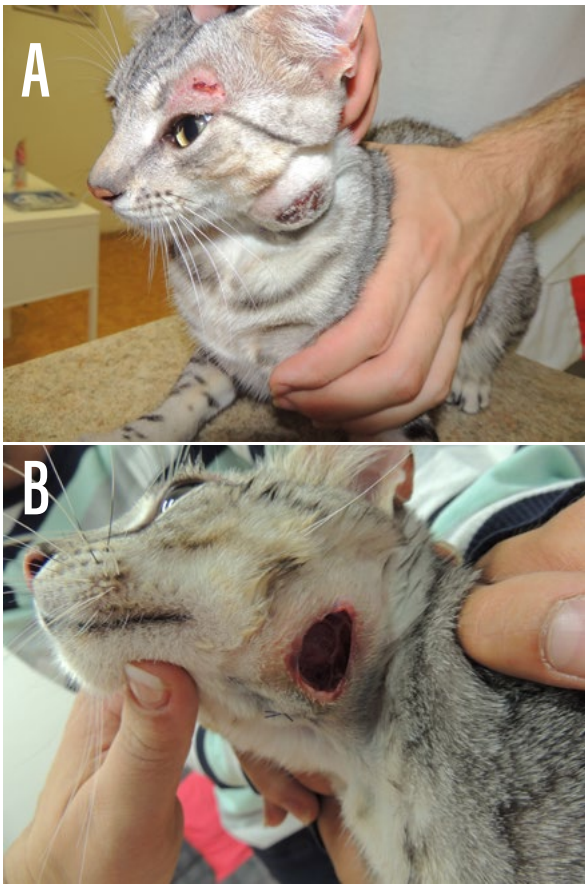

Figure 3 A & B. Lesion progression

Since susceptibility of *F. nucleautum* to 3rd generation quinolones should be good,[12] sole therapy with pradofloxacin was continued together with meloxicam and local treatment of the wound with betadine and Panalog SID.

On day 14 he was presented for a checkup. He had gained some weight (currently 3.88 kg). His owner reported that he started to have diarrhoea that day—liquid stool with no mucus nor blood and was much more subdued and did not want to eat or drink.

On physical examination, he seemed to have slight ataxia, was reluctant to move at all and had altered mentation. Marked miosis was present in his right eye (Figure 4A), which did not dilate even in darkness. A full neurological exam was not performed, but his palpebral reflexes were present, pupillary light reflex was normal in his left eye and his left pupil reacted to light, albeit with a less marked reaction because of the miosis, menace response was normal on both eyes, he was not suffering from nystagmus and his oculocephalic reflex was present. He had protrusion of the third eyelid of his left eye. His left eye was harder on palpation and painful while touching and there was restriction to globe retropulsion, exophthalmos was present on his left eye again. Mild swelling was apparent also in the left part his mouth (left pterygopalatine fossa—caudal to the last upper

molar tooth on his left side) and opening the mouth seemed to be painful for him. His mandibular lymph nodes were both enlarged. His ears were both clean and tympanic membranes appeared normal. His temperature was normal (37.8°C). The wound on his cheek seemed not to be healing properly; it was smaller, but still present. Minimal contraction of the wound had occurred and a small amount of granulation tissue was present (Figure 4B).

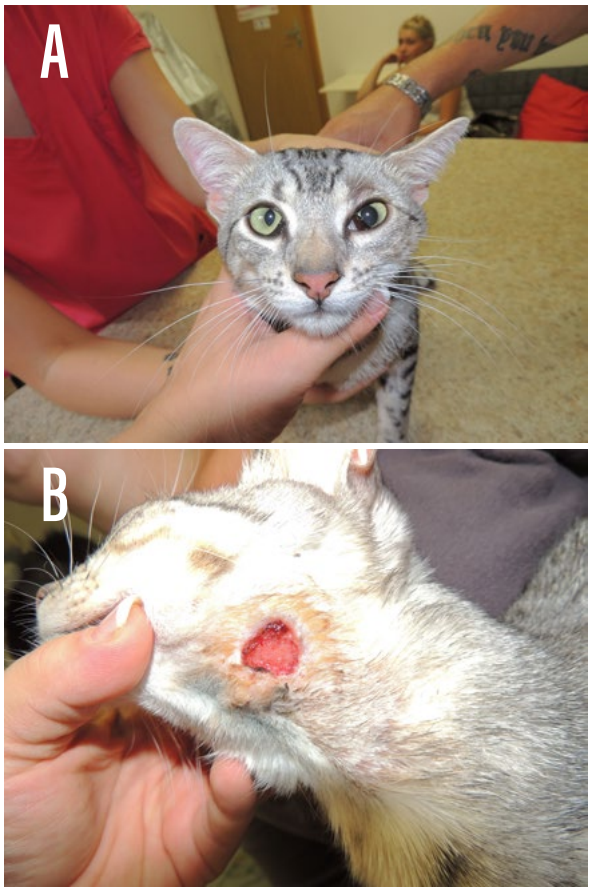

Figure 4 A&B. Lesion progression

Reoccurrence of orbital disease, particularly retrobulbar abscessation and further spread of the infection was suspected, together with development of resistance to the antibiotic prescribed.

It would have been better to add metronidazole earlier, but since he responded well to the current therapy, we did not do it in this case.

Hematology exam was performed (Table 1) and he was referred to the board neurologist for MRI and PARR clonality from the lymph node was recommended but refused by the owner. Hematology revealed marked leukocytosis with neutrophilia and monocytopenia (Table 1). We suspected reoccurrence of infection, but neoplasia could not be ruled out either.

Since the owner refused referral, we had to start rescue therapy at the practice. It would be better

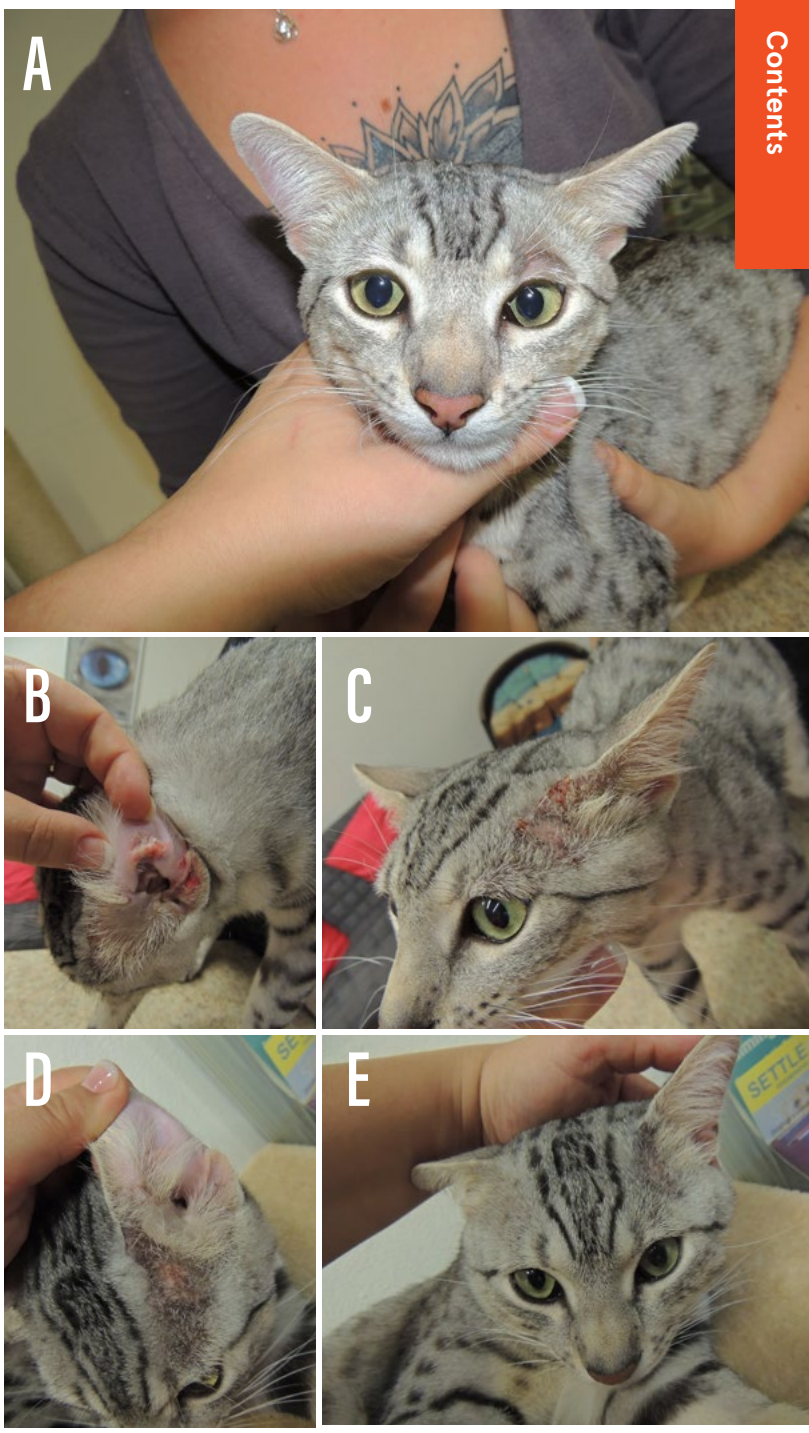

Figure 5. Finally getting a resolution. Note the suspected skin eruption due to the cephalosporin used for rescue therapy.

to give I.V. antibiotics in this case, but it was not possible at that time (Friday evening), with no access to the antibiotics needed according to the literature.[13]

Rescue ATB therapy was started with Cephalexin 30 mg/kg SID (Cefa-cure tbl.), spiramycin 375000 IU and metronidazole 62.5 mg combined (Stomorgyl 10, ½ tbl) SID, since this combination has good results in therapy in human medicine.[14,15]

Prior to antibiotic therapy, maropitant 0,5 mg/kg SCI and ranitidine 2.5 mg/kg SC were administered. Maropitant was used to prevent possible vomiting because of antibiotics and ranitidine to support bowel movement and slow down the production of gastric acid and therefore reduce the likelihood of the antibiotics irritating the stomach. He was also given fluid therapy—50 Plasmalyte SC, because he did not drink that day.

Vitamin C (300mg/toto), thiamine, riboflavin and B12 (Milgamma 0,5 mL pro toto) were given SC to support the immune system and vitamin C has proven to have potential to block lipid peroxidation in the plasma and improve cellular immune function in humans so I used it in this case too.[16,17]

Since the cat appeared to be in pain, buprenorphine was given IM 0.05 mg/kg (instead of meloxicam) and continued at home BID buccally.

Debridement of the wound on his left cheek was performed along with lavage with diluted betadine liquid and application of manuka ointment with honey.

Probiotics were added to the therapy too (Probican paste 1mL per day) with a 2-hour interval after antibiotics.

Feeding tube placement was refused by the owner, so that only offering whatever food he would like to eat was possible. Luckily, the cat started to eat on his own the next day and his pupils looked almost the same according to the owner's report.

On day 17 anisocoria was not present, nor exophthalmos. Protrusion of third eyelid of his left eye was very mild (Figure 5A). Mild medial strabismus of his left eye was still present. The wound above his left eye was completely healed (Figure 5A). He was active, his gait was normal, he had no altered mentation and he was playful. The wound on his cheek started to heal properly (size was 25% smaller). He gained weight (3.92 kg); his temperature was normal (38.7).

The owner came for a recheck in 3 weeks (day 38 of therapy). The wound on the cheek was almost completely healed—only a 3mm crust was present. No abnormality of pupils was present, no swelling of the face or exophthalmos, no third eyelid protrusion. He had gained weight (currently 4.41 kg) and his BCS was 5/9, his temperature was normal (38.6°C). Finally, a full neurological exam was performed, and no abnormalities were found. He was hard to examine, high in energy. His left mandibular lymph node was enlarged.

The owner reported reddish discharge with eschars on the inner part of his left ear and on the skin above the left eye (Figure 5B,C) which occurred 4 days after the antibiotic rescue therapy was started. It was not itchy, and pedal reflex was negative. Both ears were clean without any pathology. His haematology was completely normal, including differential white blood cell count. (Table 1).

Since the skin changes occurred shortly after the therapy started and cephalexin is known to be a cause of adverse skin reactions,[18] this antibiotic was disconnected, only Stomorgyl tablets were continued and manuka honey gel was applied SID on the pinnal lesions.

The cat was checked 7 days later. His skin had healed completely (Figure 5D,E), and he had no more third eyelid protrusion nor medial strabismus (Figure 5E). His lymph nodes were not enlarged at all.

Two months later he was still a healthy young entire tomcat.

Discussion

According to the clinical signs, a retrobulbar abscess was suspected in this case.[1] Since a draining tract was already present, surgical draining or needle aspiration was not performed. Mild erythrocytosis might be caused by splenic contraction in this case,[19] because no signs of dehydration were present. Marked thrombocytosis was thought to be present due to the inflammatory process in the body.[20] Since inflammation was suspected, hyperglobulinemia supported this hypothesis, but hyperglobulinemia might be present in some types of neoplasia such as lymphoma.[21] Elevation of AST was hypothesised to be caused by muscle damage due to inflammation present in his head/muscles.[22] GGT is elevated in cholestasis and bile duct inflammation or neoplasia, since this enzyme has higher sensitivity but much lower specificity than ALP elevation in cats,[23] hepatobiliary disease was not suspected in this case.

Radiography of the skull was not performed, because of lack of experience in positioning and

experience with skull radiography. CT scan or MRI of the skull would have been much better for evaluating the disease of the orbit,[5,6] but since tuberculosis was suspected, referring for such investigation was not possible on first presentation and the owner later refused due to financial reasons.

Histological examination of the mandibular lymph node can help to differentiate between inflammation and neoplasia and lymph node incision was the least invasive way to distinguish neoplasia and inflammation,[24] probable identification of presence of bacteria and obtain samples for PCR examination. In this case, neoplasia could not be ruled out by regular histopathology stains, so further examination was recommended but refused by the owner. Since the cat reacted well to the given therapy, further examination was omitted.

16S rRNA PCR and Sanger sequencing of bacteria was performed, because anaerobic infection was highly suspected,[8] with a possible old bite wound in anamnesis. Moreover, aerobic bacterial culture is not capable of detecting anaerobic infection. *Fusobacterium nucleautum* is a Gram-negative anaerobe, an oral commensal and a periodontal pathogen associated with a wide spectrum of human diseases.[25] Quantitative PCR examination was preferred over bacterial culture, because of the need to quickly exclude or confirm transmission of mycobacterial infection from the owner and identify *Aspergillus* infection if present. Both results were negative and no signs of such infection were observed in histopathology of the lymph node.

Conclusions

In this case 16S rRNA PCR and Sanger sequencing was very quick and helpful in identifying the cause of abscessation when anaerobic bacteria were the suspected cause. Quantitative PCR and histology helped to rule out *Mycobacterium* and *Aspergillus* infection. Ultimately, the tomcat was cured with an antibiotic combination found in literature with no recurrence ◆

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Small

LEFT-FIELD TIP: OCULAR MEDICAL ACCESSORIES IN A CHILD'S TOYBOX- THANKS TO DRS BARBIE & BRATZ.

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C&T No. 5943

I have found the Barbie and Bratz doll range to have a wonderful assortment of combs suitable for cleaning off epiphora crusts from blocked tear ducts. The clients are all enthralled when I show them this use—they can't wait to dash home and raid the kids' or grandkid's toy box.

The varied reasons that cause the dog to have epiphora in the first place still need to be addressed, resolved, and managed, but in the interim, the combs provide a soft and gentle way to maintain peri-ocular hygiene.

Using a Barbie *et al* comb is certainly a better way to clean the dried, often microorganism-teeming matted fur than the option of owners dragging/ picking dried crusty debris off the medial canthus area with their own (possibly unclean) fingernails, often leaving scratches and abrasions anew on the delicate skin of that area.

- The small toy combs are tiny enough to fit into the medial canthus/nasal area making it easier for the owners safely use rather than the client wielding a a large steel grooming comb or, horrors of horror, scissors!
- The comb teeth have rounded tip ends so less likely to scratch the delicate skin or eyes
- The combs are soft plastic so they bend and 'give' if you apply too much force. That *'give'* reduces the risk of avulsion of the tear ducts—caused when the skin around the lid and lid is forcibly yanked on or up. Having personally suffered damaged tear ducts from a post-op sticky drape being pulled hard off my own eyelids by a human theatre nurse—I would not wish this painful condition on anyone or any pet...
  If the matted crust has dried and stuck firmly to the hair and skin, I usually just initially apply some artificial tears, warm compress, Lubrithal or the eye ointment I am going to use on the said patient's eye issues-then at the end of the consult, I finish up by combing off the remaining recalcitrant debris.
  At home, I have the client apply a little dab of Vaseline in the overflow area of skin which both protects the skin and allows the crusts to be combed off more easily.

In clinic, the combs can be washed easily in shampoo or soap, dried and sprayed with disinfectant between uses. At home, I just have the client wash or rinse the comb clean under running water and allow to dry ◆

Figure1. The style and size of combs on the left of photo are of the style that I prefer to use peri-ocular.

These combs are flexible and light so harder to do damage.

I reserve the larger combs on the right of photo to use on general skin areas such as small-medium infected crusts or hotspots or matted skin wounds. These larger combs are single use in these cases then throw away. The level of microorganism contamination in those heavily infected skin as opposed to blocked-tear duct presentations is too great for me to consider reusing that comb again.

Winner

Entitled to a CVE$100 voucher

Small

AN INCREDIBLE CASE THAT REINFORCES WHY I LOVE BEING A VET!

SEVERE HEAD TRAUMA IN A CAT

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Presentation

Valentine presented to our afterhours practice After Hours Veterinary Emergency Clinic (AHVEC) with head injuries consistent with a motor vehicle accident. His owners saw him by chance, in their paddock, as they were driving down their long driveway. They live an hour and half from AHVEC.

When Valentine presented to the emergency centre he was yowling with obvious altered mentation. He had a heart rate of greater than 280 bpm, muffled heart sounds, respiratory rate of 60+ (increased effort), and upper respiratory stertor and bilateral epistaxis. Orally he had gravel and blood, both fresh and dried. His upper left dental arcade (maxilla) was obviously fractured and displaced laterally *(see images taken 48 hours later)*. There was an obvious laceration to the soft palate and his upper right canine tooth was fractured. He had marked facial swelling, dorsal head crepitus and his right eye was proptosed, desiccated with zero apparent neurological function. His mandible was swollen with multiple grazes. There was no obvious limb/spinal pain or trauma noted.

Initial treatment

Included placement of an intravenous catheter, flow by oxygen, methadone 1 mg per cat IV and a Hartmann's 10mL/kg IV bolus given over 10 minutes. His heart rate reduced to 240 bpm and a further 5mL/kg IV bolus of Hartmann's was given. He was then started on an intravenous fluid rate of 1.5 x BMR (basal metabolic rate) hourly. A fentanyl CRI at 1µg/kg/hr was initially started. He was admitted for overnight care, baseline imaging and arterial blood gas (ABL) testing was done. His

Figure 1. The patient anaesthetised and being examined

owners were adamant that he be resuscitated in the case of cardiorespiratory arrest. The results of his ABL test showed marked stress hyperglycaemia and marked metabolic lactic acidosis with respiratory compensation. Baseline radiographs showed an intact diaphragm. He had a normal bladder silhouette with no rib or spinal fractures noted. His lung fields, cardiac silhouette and pleural space appeared normal. An aFAST ultrasound was performed and no evidence of abdominal effusion was evident, and his bladder appeared normal. Valentine was placed in an oxygen tent.

Valentine was head pressing in the oxygen tent, his right eye had marked hyphaema and appeared to be bulging more. His left pupil was fixed and dilated with absent PLR. He was treated with a 3mL/kg bolus of 7% hypertonic saline over 5 minutes and continued IVFT at 2xBMR. His fentanyl was increased to 3µg/kg/hr, and Ketamine was added at 0.3mg/kg/hr. IV Ampicillin was started in the morning.

Transferred by AHVEC ambulance to our clinic

He was examined on the consult room floor as while his mentation was not normal, he would become frantic and attempt to jump out of his cage or off the bench. It was noted that he could walk but that he was ataxic. His rectal temperature was 37.4°C, his heart rate was still 240 bpm plus, and his respiratory effort was increased with a loud URT noise. He had significant facial swelling,

bilateral epistaxis and had thick saliva and blood in his mouth. He resented any examination and continued to make a groaning sound.

Decision to wait

On discussion with his owner, it was decided that we should give him another 24 hours before attempting a general anaesthetic for assessment of his jaw and eye, dental radiographs, eye enucleation and oesophagostomy tube placement.

Day 1

He remained unchanged throughout the day on his current therapy. He urinated and defecated with no awareness. He would become distressed and unsettled with noises in the clinic. He no longer required supplemental oxygenation. He was transferred back to AHVEC for ongoing monitoring overnight. AHVEC repeated his ABL blood test which showed improving hyperlactatemia and hyperglycaemia. His tachycardia was persistent, and they repeated a fluid bolus with hypertonic saline of 10mL/kg over 5 minutes. There was concern from his owner about whether he had sustained contralateral ocular trauma and was going to be blind in his left eye; they would consider euthanasia if that were the case.

Day 2

Valentine was on a fentanyl CRI 4µg/kg/hr plus a ketamine CRI 0.45 mg/kg/hr. We elected not to give him a pre-med given his fentanyl and ketamine CRI. He was induced with Alfaxalone® IV and intubated with an uncuffed ET tube and maintained on isoflurane and oxygen. His anaesthetic was without complication despite it going for close to 2 hours by the time we had completed everything.

Skull radiographs did not show an obvious fracture of the mandible. Assessment of the cranium was difficult due to the superimposition inherent to skull radiographs. Dental radiographs confirmed the fractured maxilla on the left side with a large hard and soft palpate wound. This was sutured closed with absorbable 5-0 suture.

A Braun 14g oesophagostomy tube was placed without complication and we proceeded to enucleate his traumatised eye. The globe was completely ruptured caudally and the optic nerve while intact was obviously stretched.

We attempted an ECG post-surgery but there was too much electrical interference, and we did not want to remove the oxygen while he was in recovery. His recovery was smooth, and he returned to laying laterally in his cage *(see figure 1 on previous page)*.

His post op medications were fentanyl plus ketamine CRI's, amoxicillin (20mg/kg 8q) and maropitant (1 mg/kg IV 24q). We started a metoclopramide CRI in recovery in preparation for starting his enteral feeding. I also had a discussion with Dr Sam Long regarding his potential head trauma and he agreed that we could give him a single dose of dexamethasone (0.4 mg/kg IV).

Transfer back to AHVEC

Valentine was again transferred for overnight monitoring post-procedure. The attending clinician was concerned about his persistent tachycardia and the potential for undiagnosed cardiac disease was considered. The clinician also thought that they could potentially hear a cardiac murmur. On discussion with them, we decided to wait on working up the possible cardiac disease until a later date when we had a better idea of what his brain function and vision would be like. His altered mentation/head pressing remained unchanged and vision in his remaining eye was questionable with a dilated fixed pupil with no menace response. He continued to urinate without awareness and would rest quietly with intermittent periods of restlessness. His level of analgesia was adequate.

Day 3

The next day at our clinic, Valentine had improved slightly. The most distressing thing was that he would not settle at all, he circled his cage constantly only to stop and head press in the corner of the cage. He only vocalised occasionally, and we thought his analgesia was adequate. He did continue to have episodes of being frantic and opening and closing the cage was a challenge.

We started tube feeding him with watered down Hills A/D® using a syringe driver (3mL/hr) and an IV giving set. On advice from Dr Richard Malik, we did not hurry to get him to his expected Resting Energy Requirement (RER) too quickly. Our main goal initially was to avoid Valentine vomiting as we introduced the A/D®.

Valentine could not be left with a litter tray due to his constant circling in the cage; he would just knock it over. Urination was noted but not measured. He had not defecated for 48 hours. He was unaware of his own urination. He showed no interest in food or water. These were not left with him at any stage initially.

All medication was swapped to oral via the oesophagostomy tube other than his fentanyl and metoclopramide CRI's. He was currently on Clavulox® 62.5mg BID, maropitant 4mg SID, Celluvisc® eye drops q4-6 hourly due to a reduced blink and menace response.

Day 4

Valentine was transferred to AHVEC for ongoing care over the long weekend The plan was to wean Valentine off his IV medications and IVFT. We decided to place a fentanyl patch and start oral meloxicam (plus omeprazole because of the dexamethasone injection given earlier) now that he was being fed regularly. Four days post initial trauma he still had altered mentation/head pressing, he could stand but was extremely ataxic and would fall over after less than a couple of steps, he had questionable vision in his left eye and had an ongoing tachycardia with a gallop rhythm. There was still concern about his persistent tachycardia and there was discussion about contacting a specialist about this.

Day 5

Valentine was weaned off all IV requirements. His medications consisted of a fentanyl patch (25µg/hr), amoxyclav 62.5mg BID, maropitant 4 mg SID, gabapentin 25mg BID, meloxicam 0.2mg SID and omeprazole 4.3mg SID all given via the O tube. He was initially given 25% of his RER using A/D and this was slowly increased to 50% of his RER. He tolerated the tube feeding very well and there was never any sign of regurgitation noted in his hospital charts. His mentation remained altered but responsive. He started to respond to handling/smooching and voices. He was able to walk *(see video)* and no longer head-pressed for extended periods. He still had no menace or dazzle response and had a weak PLR. He appeared to navigate some objects on the floor while walking around the ward. Valentine urinated frequently, still without obvious awareness and defected after he was treated with a Microlax® enema.

Valentine's owners were going on a 6-week overseas trip that had been planned for many months prior to his accident. As we board cats, it was decided that he would spend those 6 weeks at our clinic; in particular, because he still required tube feeding and close monitoring. As he no longer needed intensive care, he was not returned to the after-hours practice again and remained an inpatient at our clinic for the next 6 weeks.

10 days post-accident

We thought that Valentine had developed some vision in his left eye and that he could distinguish light from dark but did not feel that he could actually see at this point. He also defecated on the clinic floor, although did not have any awareness that he was toileting. He would just defecate while he was walking around the clinic.

His owners also visited him for the first time after his accident (they lived over an hour and half away and were preparing for their 6-week overseas holiday). Valentine was so obviously excited to see them and absolutely remembered who they were. It was at this point we knew that we needed to persist.

Over the next 6 weeks

Relearning

We observed Valentine re-learn pretty much everything from learning how to eat again, use a litter tray and even sleep. We had to make many adjustments to his boarding cage to assist him to re-learn how to 'cat' again.

It took him approximately 3 weeks to learn how to eat again without assistance or requiring any enteral feeding. We had to raise his food bowl, placing it inside a litter tray as he was so messy and have a non-slip mat to stop the bowl moving around while he was eating. Valentine would put the entire right side of his face in the food (*see figure 2)*.

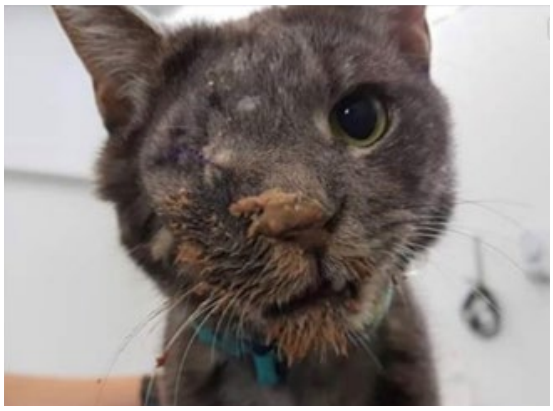


Figure 2. Neurological function during recovery was deficient—he was a messy eater and didn't realise his face was covered with food

Kitten-like behaviour

He behaved as a kitten would when first introduced to wet food. We would have to clean him up after every meal because he had no sense to groom himself. He would also be 'hangry' in the morning but not realise it was because he wanted food; he could not make the connection and although he was left with food at night he never ate until we arrived in the morning and fed him. We eventually got him onto Royal Canin Pediatric growth® wet and dry watered down into a slurry as Valentine was not able to drink water from the bowl himself. He only got water from what we put into his food each day.

Relearning to sleep curled up

Another very interesting thing to watch was that he had to re-learn to sleep curled up. This was

Figure 3. Litter tray lined with bluey

complicated by the fact that if we left him with a bed overnight, he would urinate and defecate in them. Ideally, we wanted to re-train him to use a litter tray so that his owners would not want to consider euthanasia due to him not being toilet trained as the option of him being an outside cat again was just not possible. Again, it took almost 6 weeks for Valentine to allow himself to sleep curled up. He was always alert, and we were never able to get a photo of him actually sleeping, because the minute he heard anything he would be straight back up. To stop him using his bedding as a litter tray overnight, we would only allow him a curled-up towel during the day on the puppy pads that lined his cage. He thought it was very special and would curl up inside the rolled-up towel during the day.

Relearning the litter tray

In order to re-teach him how to use the litter tray we started by lining his litter trays with blueys *(see figure 3)*, followed by blueys and litter, with eventually there just being litter.

Dental procedure

This was also required to remove the many fractured teeth that Valentine had sustained when he was hit by the car. We did postpone this for several months as we didn't want anything to set him back; we also wanted him to gain weight before doing something painful in his mouth which may prevent him from eating well for a period time.

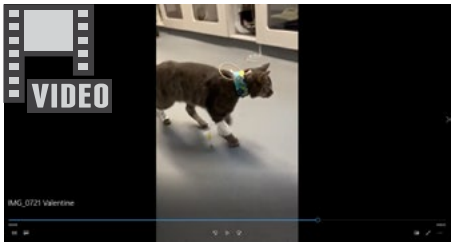
Going home

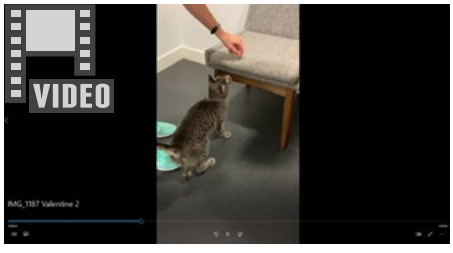
The day Valentine went home was bittersweet. We had all grown incredibly fond of him; he was such a great cat and such a character. He had also become very bonded to us but he was ready to go home and seeing him remember his owners again after a further 6 weeks of hospitalization with us made it a little easier to say goodbye to him. There were tears.

Figure 4. Staff and carers had all bonded with Valentine

This was one of those incredible cases that reinforces why I love being a vet!

Valentine suffered a severe brain injury; it would not have been wrong to euthanase him and, honestly, it was considered more than once in the first few days. However, he made a complete recovery because of the dedication of many veterinary professionals, in particular my team at *The Cat Clinic Hobart*. In a case like this a holistic approach was essential, and it highlighted to me what an amazing profession we are when we can be a criticialist, a surgeon, a neurologist, a dentist, a physiotherapist, an occupational therapist, a nutritionist, a psychologist and the list goes on... and that was all just for Valentine ◆





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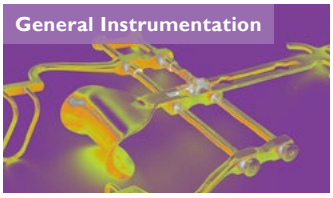

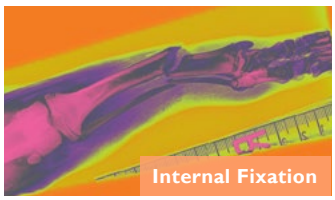

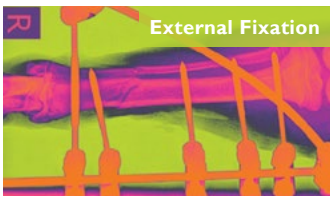

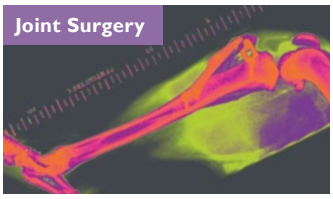

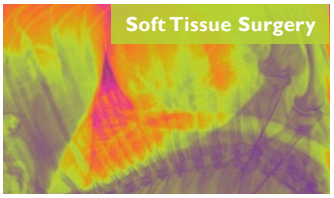

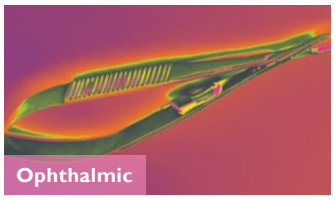

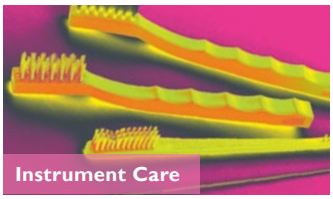

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FUNGAL DERMATITIS IN A PYTHON

Dr Izidora Sladakovic BVSc (Hons) MVS DACZM

Avian & Exotics Service

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C&T No. 5945

A 5.5-year-old male carpet python was presented for skin problems. On physical examination, the python had multiple approx. 1-3cm diameter skin lesions characterised by depigmentation, mild erythema and crusts. The lesions were present predominantly over the cranial two thirds of the dorsum. Additional diagnostics were declined at this stage and an empiric course of antibiotics was prescribed for a possible bacterial dermatitis.

The python presented approximately 2 months later as the lesions had worsened; they were moderately erythematous, ulcerated and painful on palpation *(Figure 1)*.

Skin biopsies were recommended. Apart from mild monocytosis and creatine kinase elevation, haematology and biochemistry were unremarkable. The python was anaesthetised, and multiple skin biopsies were taken and submitted for histopathology.

Histopathology report from Dr David Taylor at Vetnostics:

Microscopic examination

Multiple skin sections are ulcerated and covered by thick serocellular crusts. Within the crust and associated debris are numerous slender 4-5µm basophilic, septate fungal hyphae that show occasional acute angle branching together with scattered round to cylindrical arthroconidia. Some of the hyphae invade the superficial dermis and are accompanied by infiltrates of heterophils, lymphocytes, plasma cells and macrophages. Also present in several areas of the crust are colonies of tiny coccobacilli. Hyphae are also noted within keratin and epidermis in areas that are not ulcerated.

Summary

Severe ulcerative and necrotising fungal dermatitis

Comment

Extensive fungal infection is confirmed. Some of the hyphal features resemble infection by *Ophidiomyces ophiodiicola*, but culture and possibly PCR testing is necessary for confirmation.

Treatment

While awaiting PCR results, treatment was started with terbinafine nebulisation (as described in Kane *et al*, 2017), using terbinafine powder dissolved in sterile saline. This treatment is used in snakes, including vipers, in North America for treatment of *Ophidiomyces ophidiicola*. However, the owner found that there was a large amount of residual terbinafine powder in the nebuliser chamber. Therefore, the treatment approach was changed to dissolving the powder in a small spray bottle, spraying the contents all over the python and allowing it to dry, which resulted in a more complete delivery of the terbinafine. In addition, terbinafine cream was applied to the worst affected lesions.

Multiple PCR tests were attempted, including for *Ophidiomyces*, however, these failed to identify the fungus species.

Eight weeks after treatment, the skin lesions were significantly improved *(Figure 2)*. Terbinafine was continued for a total of 3 months by which time all the lesions had healed, apart from a few scars that remained where the lesions were most severe.

It would have been great if the fungal species in this case was identified. It would have been great if a sample was submitted for fungal culture and a repeat sample obtained to demonstrate that an end goal of treatment was achieved. However, we will take this one as a win! Since this was a non-venomous snake that was easily handled, terbinafine spray was a practical option.

Over a year since treatment there has been no recurrence of the skin lesions *(Figure 3)* ◆

Reference

Kane LP, Allender MC, Archer G, Leister K, Rzadkowska M, Boers K, Souza M, Cox S. Pharmacokinetics of nebulized and subcutaneously implanted terbinafine in cottonmouths *(Agkistrodon piscivorus)*, *J Vet Pharmacol Ther.* 2017 Oct;40(5):575-579.

Figure 1. Lesions prior to treatment

Figure 2. The patient, 8 weeks after treatment

Figure 3. 1 year post treatment. Looking good!

Large

SUDDEN DEATH IN SOWS CAUSED BY *CLOSTRIDIUM PERFRINGENS*

Jeremy Rogers,
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C&T No. 5946

Introduction

From time to time there are sporadic cases of sudden death in sows on some well managed piggeries, and clinical signs including rapid carcass decomposition and discolouration are suggestive of Clostridial infection.

In one well managed piggery, 3 sows had died suddenly over a period of 4 to 6 weeks, but no samples or investigations were able to occur, leaving the diagnosis uncertain. The otherwise healthy sows were found dead a week or so post-farrowing—carcasses were noted to 'blow up' and decompose very quickly.

A diagnosis of *Cl Perfringens* was made in this case from samples collected soon after death, and recommendations about vaccine use were made to the owner. Serious exotic diseases were ruled out as well.

History

Over the past 2 months, 3 sows had been found dead in the farrowing pens about 1 week after farrowing. Sows had been eating normally, with normal litters, the evening prior. Bodies were noted to start decomposing more rapidly than usual, and 'blow up' quickly.

An observation by the manager was that deaths seemed to occur when there had been a warm day, but cold night.

On post-mortem examination, the carcass was bloated, with purple discolouration on extremities, and some blood-stained foam from the nose and rectum. Internally there was advanced autolysis, despite the cold weather and only approximately 8 hrs after death. Blood-tinged fluid in the abdomen was noted and an enlarged and friable liver and spleen, both dark in colour. Loops of small intestine were distended with gas, and there was some food material in the stomach. The brain appeared very soft, almost liquefied.

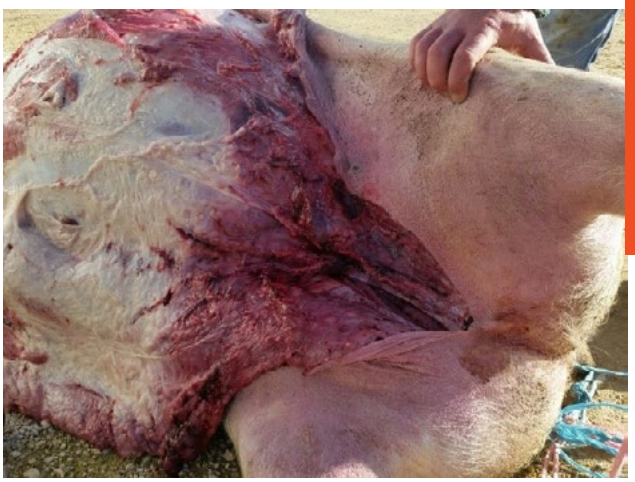

Figure 1 Rapid discolouration and 'blown up' carcass

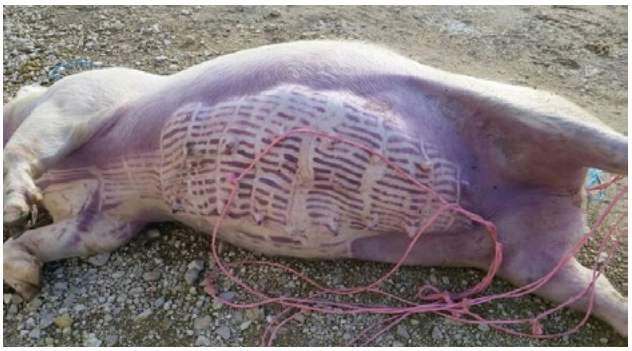

Figure 2.Purple discolouration

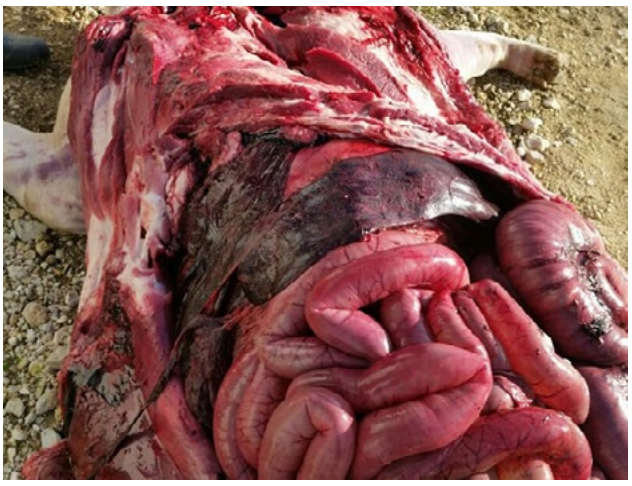

Figure 3. Distended bowel loops, enlarged and friable spleen and liver. Fibrin tags from abdominal exudate on abdominal organs, rapid autolysis

Results from samples summary

'Fixed tissues were too autolysed to be very helpful, but pathologists comments below are useful. African and Classical Swine fever tests were negative, and Clostridium perfringens *was cultured from colon contents, furthermore Epsilon toxin was detected from unfixed bowel samples submitted.'*

Comments

'There is autolysis in the organ tissues examined, with the greatest degree occurring in the liver and spleen. The occasional bacteria detected within blood vessels, vascular channels or sinusoids in

the absence of vascular changes or inflammatory reaction infer either a peracute infection or post mortem invaders. Furthermore, tissue autolysis can promote overgrowth of post mortem invaders such as the Clostridium perfringens *isolated on culture. Unfortunately in this case, tissue autolysis compromises histological analysis and prevents assessment of ante-mortem damage to blood vessel endothelium typically associated with angiotoxin liberated by* Clostridium perfringens *type D.'*

Discussion

References to *Clostridium Perfringens* Type D (producing Epsilon toxin) in pigs are difficult to find in the literature. Some older texts say that these deaths are `unsubstantiated'. However, consultant veterinarians in the pig industry do say that cases occur, and that vaccination with vaccines intended for ruminants is beneficial.

The clinical signs, history and PM findings are characteristic of Clostridial disease here, and the detection of Epsilon toxin would support that diagnosis. However, the diagnosis of Clostridial disease in pigs is complex, and it is possible that other toxin types may be operating, that have been undetected so far.

Industry veterinarians recommend the use of commercial Clostridial vaccines in pigs , where there has been a history of deaths with characteristic symptoms, but in some cases the vaccines appear to be less effective than in others.

The reason why some pigs are affected and others are not, is unknown ◆

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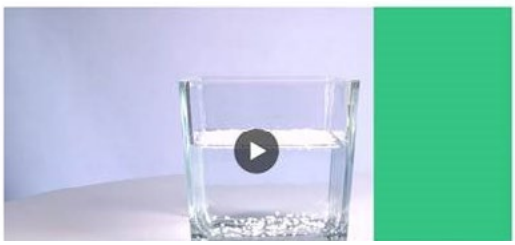

There is a plastic granulate on the surface.

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Perspective No. 155

FINDING THE WHITE RHINOCEROS *(CERATOTHERIUM SIMUM)* NASOCONCHAL PARANASAL SINUS

Mathew Gerard[1] BVSc PhD DipACVS, Anthony Blikslager[1] DVM PhD DipACVS, Johan Marais[2] BVSc(Hons) MSc

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Mathew Gerard

Mat is a Teaching Professor of Veterinary Anatomy at North Carolina (NC) State University, College of Veterinary Medicine. He is a 1992 Sydney University graduate and completed a large animal internship at Ontario Veterinary College, University of Guelph, Canada and then a large animal surgery residency at NC State University. Returning to Sydney Uni (Camden) for postgraduate work, Mat earned his PhD in equine exercise physiology in 2001. Back to the USA, he subsequently joined the large animal surgery faculty at NC State. Then in 2012 he followed his passion for teaching and clinically applied anatomy by moving into the role of full time veterinary anatomy instructor. No more on call duty and reliable time to travel home were desired bonuses to the transition from clinics. Mat was awarded membership to the NC State University Academy of Outstanding Teachers and received an NCSU Alumni Association Outstanding Teacher Award, in 2019.

2014 The Conversation

In 2014, Blikslager travelled from the North Carolina State University, USA to the University of Pretoria, Faculty of Veterinary Science, Onderstepoort, South Africa with every intention of establishing a collaborative research program in equine colic. Blikslager had a solid contact at Onderstepoort and was geared up to launch studies in enterocolitis. The bloke Blikslager was meant to collaborate with didn't show. Instead, a serendipitous meeting in the corridor with Dr. Johan Marais occurred. Marais was a professor of equine surgery at the time and had developed a strong interest in working on large pachyderms. In particular, Marais was attending to the severe wounds sustained by rhinoceros which were poached for their horns. Most poached rhinoceros are killed at the time of, or die shortly after, the brutal attack *(Figure 1)*. A few survive and Marais was confronted with managing extensive facial traumas *(Figure 2)*. Marais's contribution to saving injured rhinos was part of a rapidly growing effort by conservationists, veterinarians, public and private wildlife parks and reserves, celebrity animal lovers, and certain government officials to mitigate the poaching-induced rapid decline in white and black rhinoceros populations in Africa. Marais co-founded the organization *Saving the Survivors* (savingthesurvivors.org) in 2012, a non-profit entity dedicating all its resources to providing medical and surgical care of Africa's wildlife, with a focus on the endangered rhinoceros.

Figure 1.Recently poached white rhinoceros, found deceased. The white markers are placed by police to identify the hacking wounds on the face. These may later help to narrow down the poaching syndicate involved, based on the type of axe used to make such wounds.

Returning to the `conversation'—from that chance corridor chat, Blikslager learnt that Marais did not have a detailed understanding of the anatomy of the rhino head—there simply was no relevant resources published on the topic and Marais

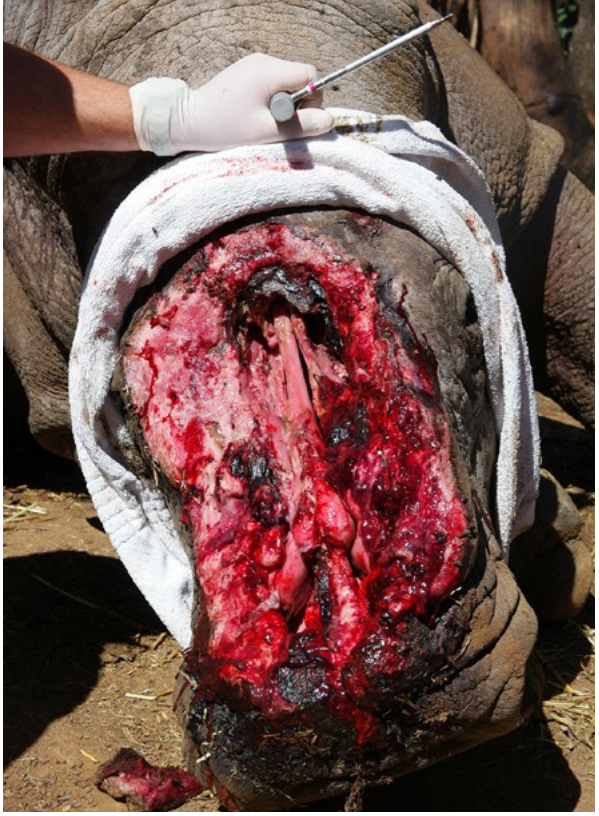

Figure 2. Extensive facial trauma in a white rhinoceros bull, approximately three weeks after poaching of his horns. An elephant skin wound covering was removed shortly before this image was taken. The elephant skin was secured with screws and washers, drilled into the frontal bone (hence the screwdriver), and elsewhere with orthopaedic wire sutures where purchase in skin was possible.

was assimilating knowledge on the fly from case experiences. Would Blikslager be interested in helping to study this anatomy? 'Absolutely' was the answer and, by the way, *'I know a bloke back at NC State who is an equine surgeon and now teaches anatomy. He would be good to include in this project…'* And so, in lieu of equine enterocolitis, as the South Africans say, `just now', a collaborative rhinoceros anatomy project got under way.

2016 Dissection #1

Gerard and Blikslager made their first combined trip to SA in May 2016 and alongside Marais they dissected a normal sub-adult female cadaver head. Use of the pathology facilities of the Faculty of Veterinary Science at UP was instrumental to the studies performed. In particular, a reliable bandsaw and somewhat fearless personnel willing to operate the bandsaw to its limits and beyond, was critical for cutting through rhino skull and teeth. The nasal (rostral) and frontal (caudal) horns had been completely removed from the rhino specimen, before we had access to it. This was done to avoid any temptation of horn poaching. It was quite remarkable to recognise that the horns are literally perched on the surface of the facial bones, anchored via cutaneous tissues and with some give to this attachment. Unlike in cattle and other ruminants, there is no bony cornual process extending into the rhino's horns—they are conical constructs of modified epidermis.[1] The realisation that horn removal can be performed

Figure 3. Skeletonized rhinoceros skulls. Upper shelf – limb bones and three intact skulls showing bulbous nasal bone rostrally. Lower shelf – skulls of poached rhinoceros showing the typical degree of facial bone removal undertaken. The skull on the right had the nasal horn removed only.

with only a sharp knife, albeit a robust one, was eye opening. Poachers opt for machetes, axes and saws presumably with the expectation that bone has to be cut to remove the entirety of the horn—turns out this level of destruction to the head is categorically and tragically unnecessary *(Figure 3)*.

Our work to understand the underlying sinus anatomy intensified as we felt the pressure of the need to learn what we could to help save the few rhinos that live through a poaching trauma. During this first dissection event we identified pneumatization of the left and right nasal bones, forming a cavernous paranasal sinus, separated on the midline by a bony septum *(Figure 4)*.

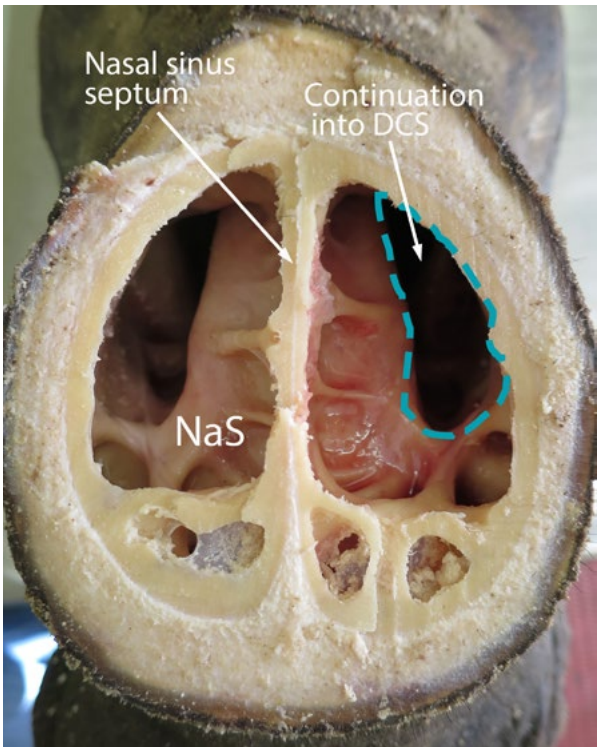

Figure 4. Dorsal view of nasal sinuses separated by the nasal septum. The nasal sinus (NaS) on each side continues with the ipsilateral dorsal conchal sinus (DCS) through a large communication (outlined by dashed line). Because of the convex surface of the rostral nasal bone, the nasal sinuses are opened when the nasal horn is sawn off at its base.

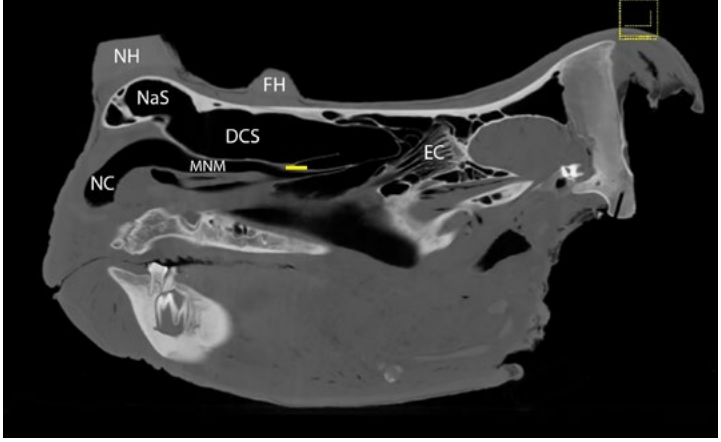

Figure 5. Sagittal CT image of rhinoceros cadaver head. The nasal sinus (NaS) and dorsal conchal sinus (DCS) form one large nasoconchal sinus. NH – nasal horn; FH – frontal horn; NC – nasal cavity; MNM – middle nasal meatus; EC – ethmoidal conchae; yellow bar – location of conchonasal opening between DCS and MNM.

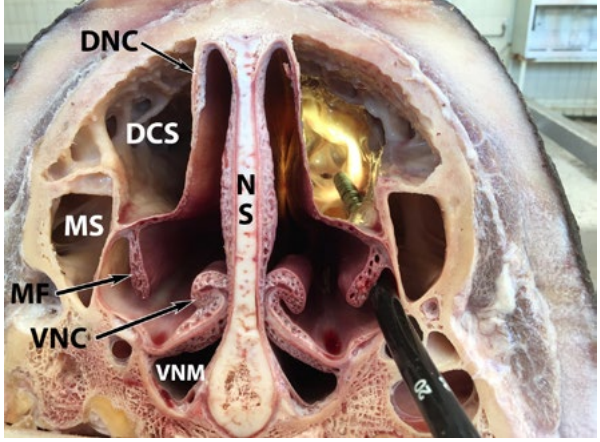

Figure 6a. Rostrocaudal view of transversely sectioned cadaver head, focused on nasal cavity and paranasal sinuses. This section was made between the nasal and frontal horns. A flexible endoscope has been passed through the left conchonasal opening and it is illuminating the caudal extent of the dorsal conchal sinus (DCS). Note the mucosal flap (MF) the endoscope must pass lateral to, as the scope is directed along the middle nasal meatus towards the opening. DNC – dorsal nasal concha; MS – maxillary sinus; NS – nasal septum; VNC – ventral nasal concha; VNM – ventral nasal meatus.

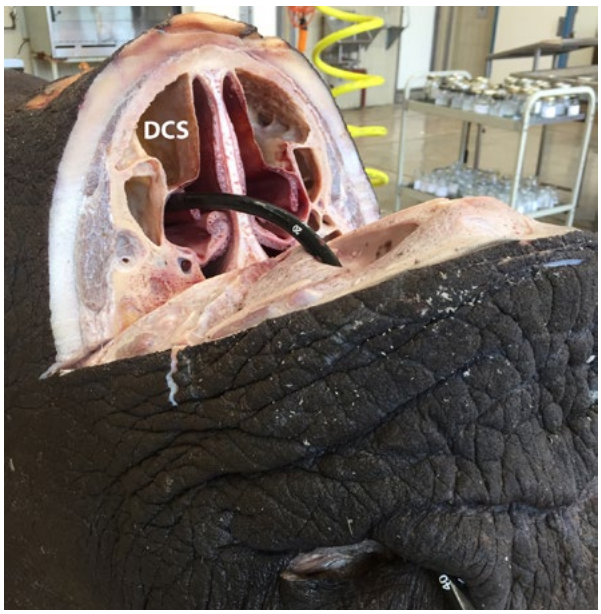

Figure 6b. View of pathway of flexible endoscope direct via the right nares towards the right conchonasal opening and dorsal conchal sinus (DCS). Note the significant arc through which the endoscope needs to pass. This is due to the steeply inclined nasal vestibule extending from the nares up to the ventral nasal meatus.

These sinuses occupy the bulbous rostral aspect of the nasal bones and bony septa provide scaffolding support across the space. The large nasal horn is parked on the dorsal surface of this part of the face. Each nasal sinus communicated through a large opening at its caudolateral aspect with a massive dorsal conchal sinus *(Figures 4 & 5)*. The nasal sinus and dorsal conchal sinus are one continuous cavity and we coined the term 'nasoconchal sinus' to label this anatomy *(Figure 5)*. *Nomina Anatomica Veterinaria*, the international tome cataloguing veterinary anatomy terms, as of its 2017 6th edition[2], does not list 'nasoconchal sinus'. It became clear we were describing a feature of anatomy that was somewhat unique and a literature search at the time revealed one other mention of a nasoconchal sinus, in a long ago new late Miocene American badger![3] Following the identification of the nasoconchal sinus, the next step was to figure out where it drained to. That was a relatively straightforward exercise, thanks to a transverse section of the head made just rostral to the opening we needed to discover. An approximately 6 cm longitudinal opening was viewed in the ventrolateral floor of the nasoconchal sinus, specifically located in the middle third of the dorsal conchal sinus and communicating directly with the middle nasal meatus of the nasal cavity. We labeled this opening the 'conchonasal opening', given it connected the dorsal conchal sinus part of the nasoconchal sinus with the nasal cavity, and one might agree that 'nasoconchonasal opening' is a bit on the wordy side! Once the sinus opening was recognised, the transverse sections were lined up surface to surface and the pathway between the nares and the conchonasal opening was mapped. From the nasal cavity perspective, the conchonasal opening was concealed by a mucosal flap hanging vertically from the dorsal nasal concha *(Figure 6a)*. We wondered if it would be possible to direct a flexible endoscope to the conchonasal opening via the nares and nasal cavity, and once there would it be reasonable to pass the scope through the opening and into the nasoconchal sinus? In this sectioned cadaver it was not a problem to place the endoscope along the anticipated path *(Figures 6a, 6b)* and we were keen to try this procedure on an intact cadaver head and eventually live rhinos.

The most relevant experience we brought to this novel dissection was Marais's field experience with rhino head wounds, and our familiarity with equine head anatomy, gained from clinical cases and the intensive teaching of veterinary anatomy. The horse and rhinoceros (and tapir) belong to the same Order, Perissodactyla ([G] *perisso* - odd, uneven, and [G] *dactyl* - toe). It was reassuring to dissect the rhinoceros head and to readily identify the comparative anatomy as we would expect to see it in the horse and then to also pick up on the differences, the nasoconchal sinus being the

most striking to date *(Figure 7)*. In the horse, the nasal bone is a thin plate that tapers to a point rostrally, and it is not excavated into a sinus. The dorsal nasal concha of the horse has a rostral scrolled component, enclosing a conchal bulla, and a separate caudal sinus component, i.e. the dorsal conchal sinus. In the white rhinoceros the entire dorsal nasal concha encloses a relatively vast dorsal conchal sinus that communicates rostrally with the nasal sinus, forming the so-named nasoconchal sinus. In contrast, the equine dorsal conchal sinus communicates caudally with the frontal sinus, forming a continuous space, the conchofrontal sinus. The conchofrontal sinus drains into the caudal maxillary sinus in the horse and from there via the nasomaxillary opening, into the nasal cavity, whereas the nasoconchal sinus of the rhinoceros drains directly into the nasal cavity, via the conchonasal opening.

During this 2016 trip, Gerard and Blikslager joined Marais on a recheck examination of a poaching wound of a rhino named Hope. Hope was one of the first white rhinos to be attended to by Marais and his *Saving The Survivors* unit and she had sustained brutal trauma to her dorsal and lateral rostral face. Hope managed to survive the poaching attack and was found by park rangers approximately 2 days later. With our enhanced understanding of nasal cavity and nasoconchal sinus anatomy, we could appreciate how much tissue was hacked from Hope's face during horn removal. Her nasoconchal sinuses were absent, along with the dorsal third to one half of 75% of the length of the nasal septum. Laterally, maxillary sinuses were damaged and exposed *(Figure 8a)*. Hope provided the team the opportunity to gain extensive experience in wound management and a long list of methods and devices were employed over a 2 year period *(Figure 8b)*. Very sadly, Hope was found dead one morning in her boma, and a peracute clostridial enterocolitis was determined to be the cause of death. Her facial wound had been progressively contracting and healing, although a sizeable defect remained.

2017 Dissection #2

Gerard and Blikslager returned to the USA from their 2016 SA trip and were immediately thinking about when they could go back. There was a sense of urgency to continue the work as the head count for poached, slaughtered rhino continued to rise at an alarming and disturbing rate. During an interview regarding their experiences, the question was asked: '*When are you going back?*' Gerard and Blikslager looked at each other right there and then and said 'Well, it will be as soon as possible! We have to study another head and confirm our first dissection findings'. N=1 was not likely to get published, and the thought was N=2 would convince us, and hopefully an editor.

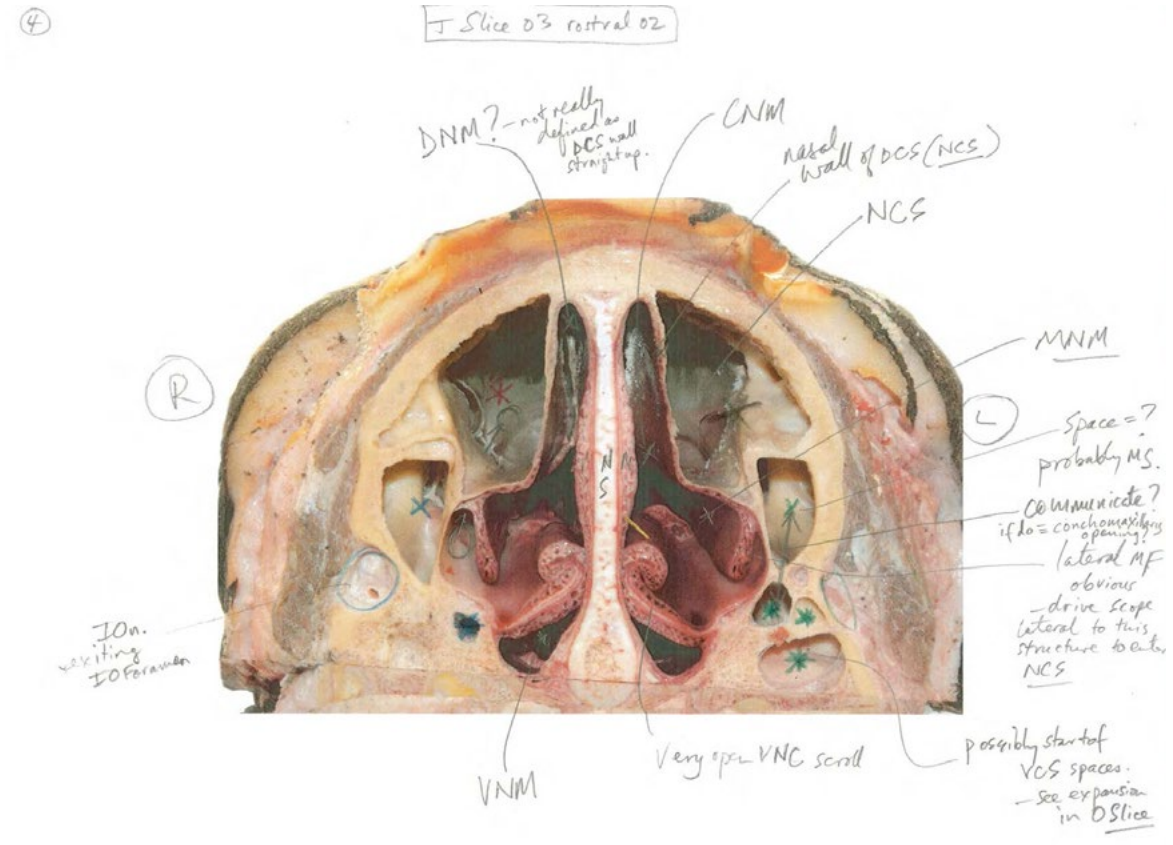

Figure 7. Example of annotated image of transverse section of first cadaver dissected, identifying recognized anatomy and questioning areas to be confirmed.

Figure 8a. Hope, during her early treatments, only weeks after being discovered alive following a brutal poaching attack. The nasoconchal sinus, and much of the nasal septum is absent. Laterally, maxillary sinuses were exposed and have sealed with granulation tissue. The left and right ventral nasal concha remain intact alongside the nasal septum.

Figure 8b. Hope, following the application of a dynamic wound closure system in an effort to draw wound margins towards midline. This device was adapted from human medicine where it is used for closing large body defects.

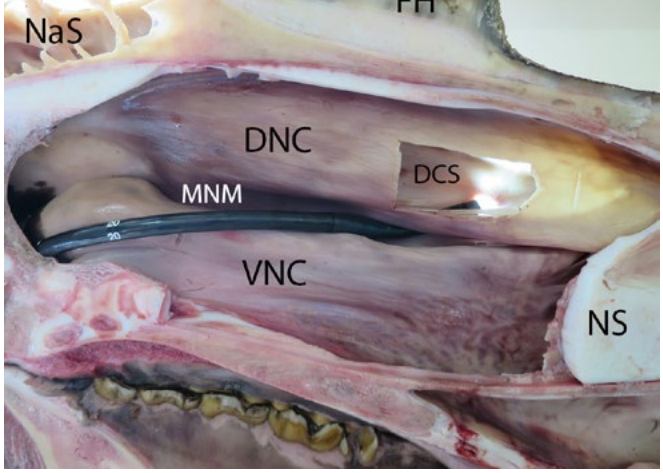

Figure 9. Medial to lateral view of right nasal cavity of second cadaver dissected. Most of the nasal septum (NS) has been excised. A flexible endoscope is passing into the nasal cavity over the rostral part of the ventral nasal concha (VNC) and along the middle nasal meatus (MNM). The endoscope enters the DCS at the conchonasal opening. A window has been cut in the dorsal nasal concha (DNC) to show the dorsal conchal sinus (DCS) and the end of the endoscope. NaS – nasal sinus; FH – frontal horn.

Afterall, gross anatomy is relatively consistent within a species! The 2017 trip was as exciting and rewarding as the 2016 event. Another normal sub-adult female white rhino cadaver head was available for dissection. Brimming with new knowledge from our first dissection we approached this second head from a different plane of view to initially keep the focused areas of interest as intact as possible as we gradually deconstructed the specimen. This dissection was performed in the anatomy laboratory facilities of the Faculty of Veterinary Science and once again our most indispensable tool was a trustworthy and capable bandsaw, guided and coaxed into sawing through a rhino skull by an equally capable anatomy staff member. As one might imagine, there is some size involved, along with substantial density of bone and dental tissues. With anticipation, we confirmed our second nasoconchal sinus in a white rhino and the conchonasal opening was equally irrefragable, it being nicely and fully intact based on our informed dissection approach. To double check there was no other natural pathway draining the nasoconchal sinus, we tilted the head nose-end up and filled the dependent and more intricate caudal sinus space with new methylene blue diluted in a little tap water. It stayed put. Revisiting our 2016 endoscopic questions we once again traced the pathway a flexible scope would need to follow to the conchonasal opening and it was easy to pass a 9 mm diameter endoscope through the opening into the combined nasoconchal sinus and to take a good look caudally, and by retroflexion, a good look rostrally too *(Figure 9)*. For a second time, we concluded the endoscopic application was practical enough in such a specimen but the utility of the technique was certainly not a *fait accompli*. We needed to give it a go in live rhinos.

Beyond 2017

After that second dissection, we wrote up our observations and the *Journal of Zoo & Wildlife Medicine* found the work of publishable merit.[4] In 2019 we made another trip, and were able to apply our endoscopic technique to three live white rhinos. We got it right on the third rhino. Directing the scope as envisioned proved confounding under the field conditions; however, with the sequential experiences the technique was accomplished and we are confident it is a repeatable procedure. We have expanded our anatomy studies to include soft tissue dissections of the distal limbs. Snare injuries are not uncommon and further understanding of the tissues involved is necessary. A very unexpected surgical consult on a black rhinoceros

Figure 10. White rhino bull shown in Figure 2, approximately 4.5 years later. Significant contraction of the facial wound has occurred. Facilitating closure of the remaining defect is an ongoing challenge. This rhino otherwise is behaving normally and has successfully returned to siring calves.

Figure 11. Healing of a conservative poaching trauma, which resulted in opening of the nasal sinus only. A – the exposed nasal sinuses resulted in bilateral nasoconchal sinusitis. B – following lavage of the sinuses and wound debridement a protective cap of casting material is secured with screws. C – at two months the sinuses have sealed over. An infraorbital nerve block is being applied before wound debridement. D – complete healing and early regrowth of nasal horn at 8 months post injury.

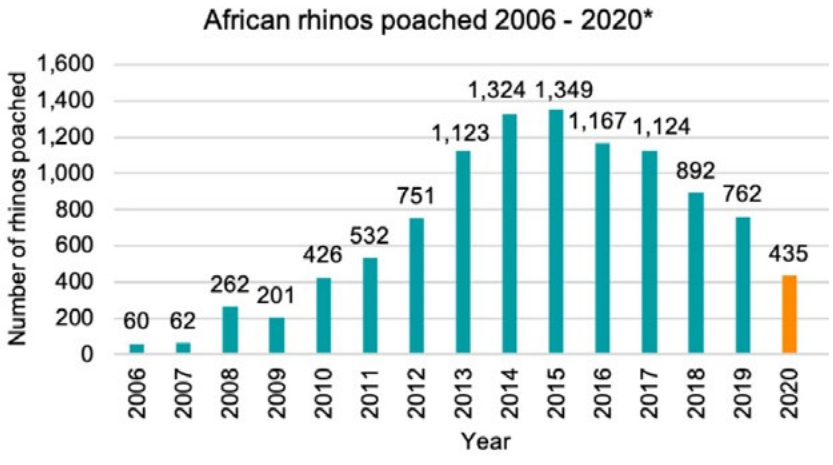

Image credit: Save the Rhino International, sourced from https://www.savetherhino.org/rhino-info/poaching-stats/

Figure 12. Graph showing African poaching numbers for the past 15 years. South Africa accounts for the vast majority of poachings. Zimbabwe, Kenya and Namibia have experienced large losses. *2020 figure is an approximation as not all countries had released data at the time of graph production.

at Chicago's Brookfield Zoo was undertaken in 2018 (https://www.czs.org/LaylaRhino) a direct consequence of zoo veterinarians learning of our anatomy studies. The valuable female early adult breeding animal developed a nasopharyngeal mass that expanded rostrally to occlude her left nasal cavity and most of the right too. Back in SA much success with managing wounds has been realised and the less traumatic poaching wounds are healing in about 4 months with facilitated care *(Figures 10 and 11)*. On the poaching front, the good news is that the numbers have fallen off in Africa *(Figure 12)* thanks to concerted efforts by national and local governments, anti-poaching units and a vast array of concerned groups and individuals funding the protection and long term survivability of this magnificent pachyderm. The dramatic drop in the 2020 poaching figure is tempered by the knowledge that the SARS-CoV-2 pandemic restricted the illicit rhinoceros horn trade. Mid-year 2021 data indicates a resurgence of poached rhino casualties. Our work continues.

Acknowledgements

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The Australian Rhino Project Gala Dinner Fawcett A	Control and Therapy Series Issue 281 - December 2015
A Visit To The Rhino Revolution Orphanage & Rehabilitation Facility Brown G	Control and Therapy Series Issue 284 - September 2016 C&T No. 5573

Perspective No. 156

VETERINARY MANAGEMENT OF FREE-RANGING POSSUMS AND GLIDERS

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Dr Tim Portas is a veterinarian who has worked exclusively with free-ranging and captive wildlife since 1999. He has provided veterinary support to conservation projects for numerous species including the Northern corroboree frog, Eastern bettong, Eastern quoll, Leadbeater's possum, Northern hairy-nosed wombat, Southern brush-tailed rock wallaby, Sumatran rhinoceros and Sumatran tiger. His work involves health evaluation, disease risk analysis, anaesthesia and sedation, assessment of short- and long-term physiological responses to conservation translocations and disease investigation in free-ranging populations. He is currently senior wildlife veterinarian for the RSPCA Queensland Wildlife Clinic where he has worked since 2016.

INTRODUCTION

Twenty-eight species of possums and gliders occur in Australia. Most are nocturnal, although some species may be active during the day and all live a predominantly arboreal lifestyle. More common species that may be presented for veterinary evaluation on the eastern coast of the Australian mainland include the following:

- common brushtail possum *(Trichosurus vulpecula)*
- short-eared possum *(Trichosurus caninus)*
- mountain brushtail possum *(Trichosurus cunninghami)*
- common ringtail possum *(Pseudocheirus peregrinus)*
- sugar glider *(Petaurus breviceps)*
- Krefft's glider *(Petaurus notatus)*
- squirrel glider *(Petaurus norfolcensis)*
- feathertail glider *(Acrobates pygmaeus)*

Common brushtail and common ringtail possums have adapted well to urban and peri-urban environments and are the two species most likely to be presented for veterinary evaluation on the eastern coast. In the Northern Territory the northern brushtail possum *(Trichosurus arnhemensis)* and, less commonly the savanna glider *(Petaurus ariel)*, are the main species likely to be presented. While in Western Australia the common brushtail possum and the western ringtail possum *(Pseudocheirus occidentalis)* are the most common species in urban areas.

This article provides information on husbandry, nutrition, restraint and handling and diseases or conditions that are likely to be seen by veterinarians working in private practice. For a more complete account of diseases and medical conditions seen in possums and gliders see Johnson and Hemsley (2008) and Scheelings (2019).

Disclaimer: Many of the drug doses cited in this module constitute off-label use and have not undergone pharmacokinetic or pharmacodynamic studies in the species described. Physiological differences mean that the absorption, distribution and metabolism of these drugs can vary significantly compared with these drugs in domestic mammals. Clinicians should use these drugs with caution.

Husbandry

All wildlife should be held separately from domestic dogs and cats in a quiet environment, preferably with subdued lighting. Possums and gliders are typically nocturnal so lighting cycles

in indoor enclosures should reflect this. Possums and gliders should be fed at night when they are normally active.

For short to medium term medical treatment, adult possums and larger gliders can be housed in standard domestic animal hospital cages, containing a nest box or drey that can be readily cleaned or replaced as required. A square plastic bucket lined with a pillowcase will suffice and facilitate handling. Smaller gliders will need to be held in soft walled crates with a mesh small enough to prevent escape. For longer term care and rehabilitation, a larger vertically orientated enclosure (such as a commercially available bird aviary) will be necessary.

Hand reared juveniles should be housed in artificial pouches, with an internal liner of natural fibre such as cotton, and placed within a humidicrib set to temperatures appropriate for the animal's age and developmental stage. **Unless being managed for a specific medical condition, juvenile possums and gliders should be transferred to an appropriately experienced and permitted wildlife rehabilitator as soon as possible.**

For more detailed information on the husbandry of possums and gliders see the following resources:

- Jackson S. (2003) *Australian Mammals: Biology and Captive Management*. CSIRO Publishing Melbourne.
- Wildcare. (2017) *Caring for Gliders*. Wildcare Australia Inc. Nerang.
- Wildcare. (2020) P*ossums of South-East Queensland*. Wildcare Australia Inc. Nerang.

NUTRITION IN CAPTIVITY

Common brushtail possums are more frugivorous and less folivorous than common ringtail possums which subsist principally on a diet of *Eucalyptus* spp. and other native leaves (see below). Captive diets for common brushtail possums should include free access to browse from a variety of native species including *Eucalyptus, Acacia, Banksia, Syzygium and Grevillea (Figure 1)*. Small amounts of vegetables and fruits can be offered and are usually readily consumed.

Common ringtails feed predominantly on *Eucalyptus* spp. but also consume leaf from species of *Syzygium, Callistemon, Grevillea and Banksia* (Hume 1999). The young growing tips are preferentially selected and these should be provided to possums in care. Supplements such as Wombaroo High Protein Supplement (wombaroo.com.au/product/high-protein-supplement) are usually readily accepted.

Gliders have more specialised species-specific dietary requirements ranging from greater gliders, which are obligate folivores, feathertail gliders which consume a high proportion of nectar, through to sugar gliders which are omnivorous consuming a wide range of foods including plant saps, gums, pollen and arthropods. The diet offered in captivity should reflect the natural diet as much as possible. Artificial nectar products are commercially available (wombaroo.com.au/product/nectar-shake-n-make) and can be used as a dietary component for sugar, squirrel and feathertail gliders.

In smaller hospital enclosures, non-browse food can be offered in bowls. Browse should be placed in water filled containers that are secured to the side of the enclosure to prevent tipping. Water should be provided *ad libitum* for all species.

Detailed information on the nutritional requirements and techniques for hand-rearing juvenile possums is beyond the scope of this document and readers are referred to the following resources:

- *Australian Mammals: Biology and Captive Management (Jackson 2003)*
- wombaroo.com.au/product-category/native-wildlife/possum
- Rich G. (2014) *Nutritional considerations for hand-rearing possums*. Proceedings of the Australian Wildlife Rehabilitation Conference. Hobart.

Figure 1. Fresh browse

RESTRAINT AND ANAESTHESIA

Manual restraint and handling

All possums and many gliders will attempt to bite and scratch when restrained. Towels facilitate restraint in larger species, reducing the risk of injury to handlers. Possums should be restrained with one hand firmly but gently grasping the neck immediately caudal to the head, and the other

grasping the base of the tail *(Figure 2)*; to prevent injuries from the hind claws. Hospitalised possums tend to retreat into their nest box during the day. Anaesthesia can be induced usually with minimal physical restraint with possums contained inside a hospital nest box; a mask can be gently introduced over the face to induce anaesthesia using isoflurane in oxygen.

For short term restraint (such as for weighing or transfer between enclosures) possums and gliders can be restrained in a calico bag or cotton pillow slip. Larger possums such as *Trichosurus* spp. can readily tear through light weight cotton bags with the claws of the hind limbs so use of such bags should be limited to short term restraint in enclosed/secure areas. For transportation, animals can be housed in standard domestic animal pet packs with visual barriers to reduce stress.

Figure 2. a) Restraint technique for an adult common brushtail possum. b) Restraint technique for a juvenile common ringtail possum; clinical examination can generally be performed in juveniles without excessive manual restraint. c) Restraint technique for a sugar glider.

Figure 3.a) Mask induction of an adult common brushtail possum restrained in a cotton pillowslip. b) Maintenance following intubation with a 2.5 mm endotracheal tube.

CHEMICAL RESTRAINT AND ANAESTHESIA

Fasting is not generally required as possums and gliders are not prone to regurgitation; however, it is prudent to fast hand-reared juveniles for 1-2 hours to prevent passive regurgitation of milk. In the wildlife rehabilitation setting, induction (via mask) and maintenance (via mask or endotracheal tube) of anaesthesia using isoflurane in oxygen is adequate for most procedures *(Figure 3)*. Premedication with diazepam (0.5 mg/kg IM) or induction with tiletamine/zolazepam (5 mg/kg IM) may be indicated for fractious individuals. Recoveries following induction with tiletamine/zolazepam can be prolonged and rough so diazepam at 1 mg/kg IM is probably better. Tiletamine/zolazepam has been implicated in the deaths of squirrel gliders so should be used with caution in glider species (Holz 1992). The use of alfaxalone (8 mg/kg) in combination with medetomidine (0.12 mg/kg) has also been reported; alfaxalone used alone tends to produce variable results (Vogelnest 2019a).

VENEPUNCTURE SITES IN POSSUMS AND GLIDERS

The following veins are accessible in possums and gliders for blood collection, intravenous injection and (in some cases) catheter placement:

- jugular vein
- lateral tail vein
- ventral tail vein
- femoral vein
- lateral saphenous vein
- ventral tail vein (small gliders)
- tibial artery (small gliders)

DISEASES AND CONDITIONS SEEN IN FREE-RANGING POSSUMS AND GLIDERS

Dermatological Disease

Exudative Dermatitis

- Aetiology

  An idiopathic ulcerative dermatosis that occurs in free-ranging common brushtails and, to a lesser extent, short-eared and mountain brushtail possums (ARWH 2005, Vogelnest 2019b). The aetiology is unknown but is thought to be multifactorial. Environmental stressors may be important contributing factors and dispersing sub-adult males, predominantly those in urban areas, are commonly affected. A range of often commensal bacteria have been cultured from skin lesions including Staphylococcus, *Streptococcus, Corynebacterium* and *Pseudomonas* spp. Secondary yeast infections can occur. Anecdotal evidence suggests an increasing incidence of this disease.

- Clinical signs

  Clinical signs vary from mild alopecia, erythema and excoriations to extensive exudation and crusting with full thickness ulceration in severe cases *(Figure 4)*. Lesions occur most commonly on the face, distal limbs, neck and rump. Conjunctivitis and mucopurulent discharge may be evident in some cases. In cases complicated by secondary yeast infections, the skin can have a greasy appearance. Animals with advanced disease may be severely debilitated.

- Diagnosis

  Diagnosis is based on the characteristic clinical signs. Microbial culture of lesions is generally not indicated as the isolation of a particular organism from an open wound does not indicate causality.

- Treatment

  The decision to treat or euthanise affected animals depends upon the extent and location of the lesions and the animals' age and sex. Mild cases typically respond well to treatment with cefaclor (12.5 mg/kg PO bid) or amoxicillin-clavulanic acid (15 mg/kg IM or SC sid). Where there is a secondary yeast infection, anti-fungal therapy with fluconazole (20 mg/kg PO bid) or itraconazole (10 mg/kg PO sid) is indicated. Removal of crusts and bathing of lesions with dilute chlorhexidine solution under isoflurane anaesthesia is recommended. Euthanasia is indicated in possums with extensive or deep ulceration, the loss of eyelids or lip margins, or where lesions occur over joints. Relapse of disease following treatment and release of sub-adult males has been observed.

Figure 4. Examples of severe exudative dermatitis in common brushtail possums that warrant euthanasia

Lumbosacral Dermatitis

- Aetiology

  A poorly understood and defined disease of common and mountain brushtail possums associated with damage to the pelage and, sometimes the underlying skin, over the lumbosacral region (Hufschmid *et al*. 2010). The clinical changes indicate mechanical damage (commonly bites from territorial conflicts, particularly in males) and an association with *Trichosurolaelaps* spp. mites has been proposed but causality not proven.

- Clinical signs
  Alopecia, decreased coat length, broken hair shafts and scaling of the underlying skin predominantly in the lumbosacral region *(Figure 5)*. Mites may be evident on affected possums.

- Diagnosis
  There are no diagnostic tests and diagnosis is based on the clinical signs.

- Treatment
  Ectoparasitic treatments such as ivermectin (200 µg/kg SC or PO) or selamectin (6-18 mg/kg topically) may be effective in animals with ectoparasites.

Poxvirus

- Aetiology
  Localised skin lesions associated with an *Orthopoxvirus* have been reported from common ringtail possums (Vogelnest *et al*. 2012). Arthropod vectors have been proposed to play a role in transmission and an increase in cases may be seen when favourable climatic conditions increase arthropod activity.

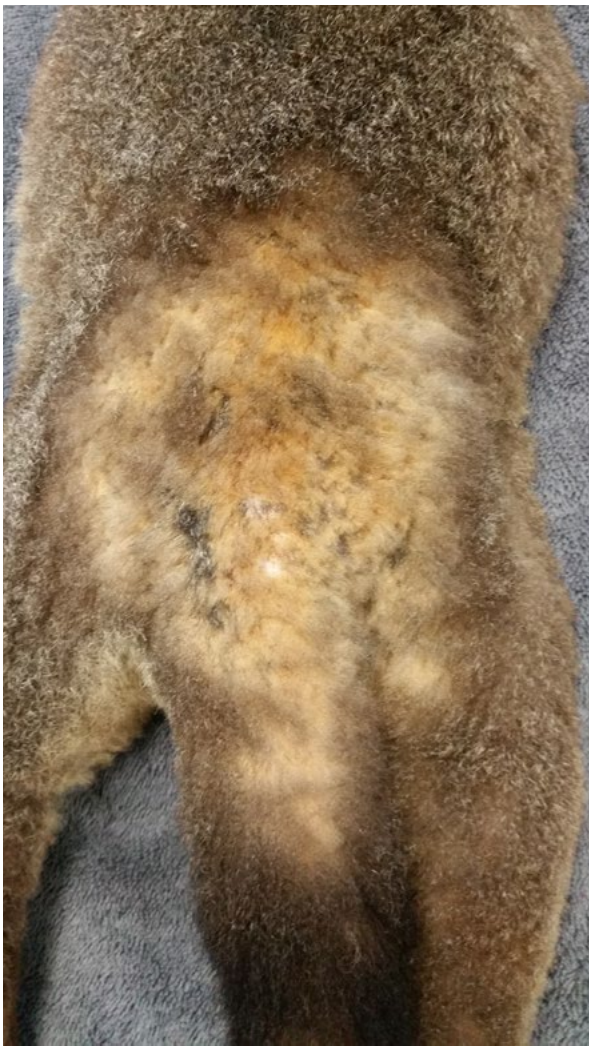


Figure 5. Lumbosacral dermatitis in a common brushtail possum

- Clinical signs
  The disease in possums is characterised by small, multifocal, raised nodular exophytic lesions on the tail and distal limbs.

- Diagnosis
  The suggestive clinical signs can be supported by characteristic histological changes and electron microscopy findings.

- Treatment
  Lesions are typically self-limiting and resolve within several months. The use of topical and systemic antibiotics and anti-inflammatories to provide analgesia and control secondary bacterial infections has been reported.

Mycobacterium ulcerans (Buruli ulcer, Bairnsdale ulcer)

- Aetiology
  Mycobacterium ulcerans is the causative agent of Buruli ulcer, a severe necrotic disease of the skin and associated soft tissues, affecting animals including humans (Scheelings 2019). The disease is reported in common ringtail, common brushtail and mountain brushtail possums which are thought to be important reservoirs of the bacterium (O'Brien *et al*. 2014). Sporadic outbreaks occur predominantly in Victoria and less in northern Queensland. Cases in possums have only been described from endemic areas in Victoria (Bairnsdale). The mode of transmission is unknown but biting insects could be important vectors. The organism has been identified in the gastrointestinal tract of possums and allocoprophagy in common ringtail possums is proposed as a mechanism for reinoculation and amplification of the organism in this species (O'Brien *et al*. 2014).

- Clinical signs
  Disease is seen most commonly in adult possums and presents as single or multifocal cutaneous ulcers on the face, limbs or tail. Common ringtail possums (with males most commonly affected) appear significantly more susceptible to the development of clinical lesions compared with the two brushtail possum species. Systemic disease involving the liver and lungs has been reported in common ringtail possums.

- Diagnosis
  Diagnosis is by PCR, histopathology or culture of the organism.

- Treatment

Treatment has not been described nor is it recommended in free-ranging possums. Spontaneous resolution of lesions has been observed in common and mountain brushtail possums.

Protozoal Diseases

Toxoplasmosis

- Aetiology

Australian marsupials are regarded as especially susceptible to toxoplasmosis. However, despite reports of morbidity and mortality the true effect of this disease in free-ranging populations is unknown (Portas 2019). *Toxoplasma gondii* is an obligate intracellular parasite for which domestic felids are the only known definitive host. Marsupials are infected after consuming sporulated oocysts (deposited in the environment by infected domestic cats) or bradyzoites (and tachyzoites) in the tissues of infected hosts. The morbidity and mortality rates in marsupials are unknown but the presence of antibodies in a range of species indicates infection is not invariably fatal.

- Clinical signs

Disease ranges from per-acute to chronic and the wide range of potential clinical signs reflect the organ systems affected. Neurological signs include blindness, ataxia, circling, incoordination, nystagmus, head tilt and paralysis. Ocular lesions include keratitis, uveitis, chorioretinitis and cataract. Other signs include respiratory distress, lymphadenopathy and depression.

- Diagnosis

A presumptive antemortem diagnosis can be made by demonstrating an elevated immunoglobulin M titre with a low or negative immunoglobulin G titre (acute disease) or rising/elevated immunoglobulin M and G titres (chronic disease) plus clinical findings.

- Treatment

There are few accounts of successful treatment of toxoplasmosis in marsupials. Pyrimethamine/sulfonamide, trimethoprim/sulfadiazine and clindamycin have been used with variable efficacy. Atovaquone (100 mg/kg PO sid) has been used to treat macropods but the cost is significant.

Neonatal diseases

Orphaned Juveniles

- Aetiology

Orphaned and abandoned juvenile possums and gliders are frequently presented for veterinary evaluation. Possums and gliders may be orphaned following traumatic injuries (vehicular trauma, domestic animal attack) to the dam, misadventure or where the dam is euthanised due to advanced disease.

- Clinical signs

Juveniles are frequently hypothermic, dehydrated and hypoglycaemic at presentation. Juveniles that are severely dehydrated frequently have corneal ulceration. Juveniles should be examined closely for signs of cat attack (obvious bites to cryptic puncture wounds) as this is a common reason for presentation.

- Diagnosis

Diagnosis is based on history and physical examination.

- Treatment

Correct hypothermia by placing the juvenile in a humidicrib and warm gradually. Correct dehydration by administering warmed subcutaneous fluids at 10% of the animal's body weight. Once the animal is normothermic, offer a glucose feed followed by hand rearing formula. Lacerations or minor punctures associated with cat attacks should be cleaned and treated with amoxicillin/clavulanic acid (20 mg/kg IM or SC sid); concurrent administration of nystatin (50000 IU/kg PO tid) is recommended as candidiasis is a common sequela to antibiotic administration in juvenile possums and gliders. Euthanasia is recommended where there are multiple lacerations or deep puncture wounds; *Pasteurella*-associated septicaemias are common in these cases *(Figure 6)*. Corneal ulceration can be treated with topical antibiotics and lubricating eye drops.

Abdominal Distension

- Aetiology

Abdominal distension is common in hand-reared common ringtail possums. There are numerous potential causes including gastrointestinal dysbiosis and caecal stasis resulting from inappropriate diets or bacterial or fungal overgrowth secondary to poor hygiene or antibiotic therapy (Johnson and

Hemsley 2008). Additionally, adult females provide caecal pellets to their offspring which are critical in establishing a normal gastrointestinal microbiome. Common ringtail possums are predominantly folivorous and weaning on to diets that contain large amounts of carbohydrate or fruit can predispose to the development of bloat and caecal stasis.

- ### Clinical signs
  Weight loss (or failure to gain weight), weakness and lethargy followed by abdominal distension, signs of abdominal pain, reduced faecal volume and pellet size and inappetence. Bilaterally symmetrical hair thinning and alopecia (stress-related) can also be observed.

- ### Diagnosis
  Diagnosis is based on the clinical history and physical examination findings including necropsy.

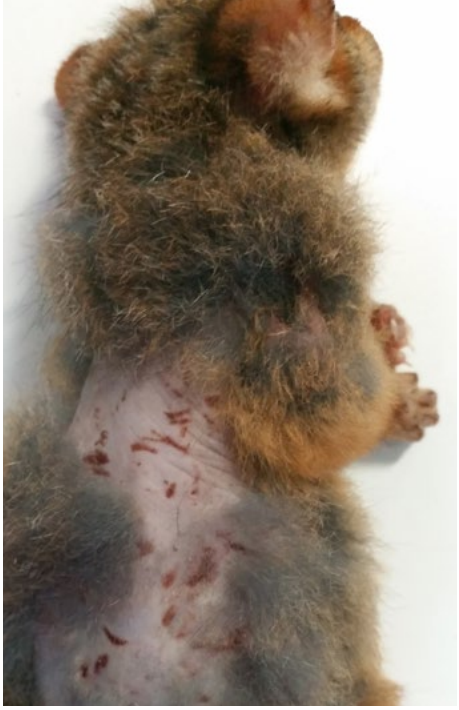
- ### Treatment
  Treatment is frequently unrewarding as possums are often presented at an advanced stage of disease. Treatment includes correcting the diet, transfaunation with caecal contents from a dead adult possum, administration of nystatin (50000 IU/kg PO tid) if candidiasis is present and analgesia. Various prokinetics (cisapride, metomide) and products containing ducosate and simethicone have been advocated but the efficacy of these drugs in treating bloat is unknown. Prophylaxis includes providing caecal pellets, or, less effectively, `poo shakes' (faeces from healthy adult RTP mixed with formula) and/or a good source of fibre such as Oxbow Critical Care Herbivore.

Candidiasis

- ### Aetiology
  Candidiasis is a common disease in hand reared marsupials, including possums. Predisposing factors may include poor hygiene and husbandry and antibiotic therapy.

- ### Clinical signs
  Clinical signs include inappetence, loose or poorly formed faeces with a characteristic yeast-like smell and straining. While *Candida* species typically colonise the gastrointestinal tract mucosa, in severe cases the skin around the mouth and cloaca may also be colonised.

- ### Diagnosis
  The presence of budding yeasts on gram stains of faecal smears and/or mouth swabs in conjunction with suggestive clinical signs supports a diagnosis of candidiasis.



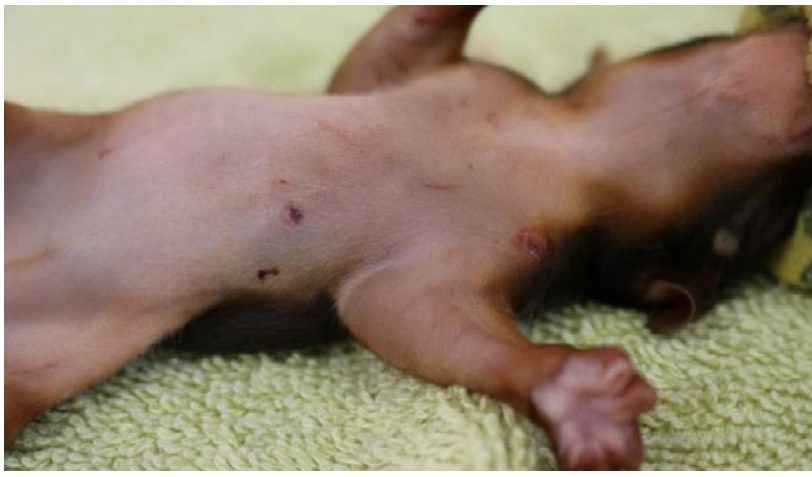


Figure 6. a) Multiple lacerations inflicted by a domestic cat on a juvenile ringtail possum; euthanasia is indicated in this case. b) Deep punctures to the thorax of a juvenile ringtail possum inflicted by a domestic cat; euthanasia is indicated in these cases.

- ### Treatment
  Any underlying husbandry problems must be addressed (i.e., improve the hygiene of the carer). Nystatin (50000 IU/kg PO tid) is administered until 3-5 days after yeasts are no longer evident on faecal gram stains. Nystatin must contact the organism to be effective so should be administered well before milk feeds.

Toxicoses

Anticoagulant rodenticide toxicity

- ### Aetiology
  A common presentation in free-ranging brush-tailed possums from urban and peri-urban environments (Boardman 2019). Non-target poisoning of wildlife with anticoagulant rodenticides is an emerging problem worldwide although the extent of

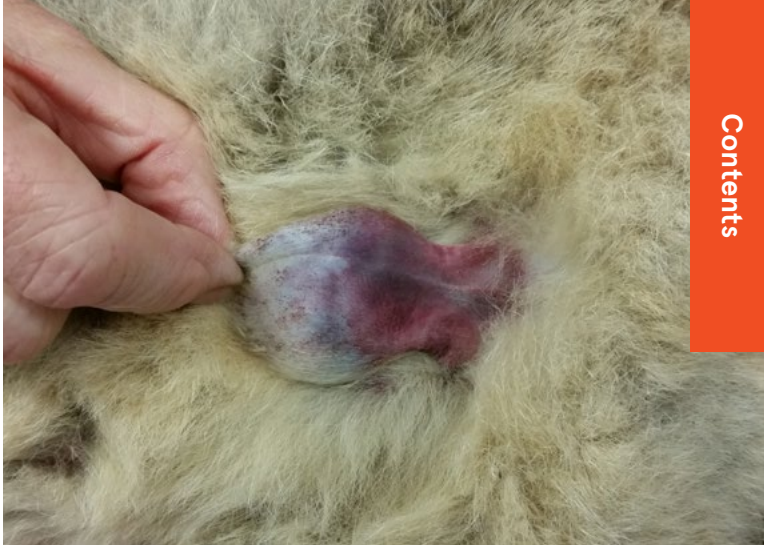


Figure 7. a) Severe epistaxis in a common brushtail possum associated with rodenticide ingestion. b) Subcutaneous haemorrhage is common in brushtail possums following rodenticide ingestion. c) Subcutaneous haemorrhage of the scrotum is a common clinical finding in male brushtail possums following rodenticide ingestion.

the issue in Australia is unknown. Common brushtail possums have a more omnivorous diet and seem to have a greater propensity for consuming baits laid for rodents.

Clinical signs

Clinical signs may not manifest for 3-5 days post ingestion depending on the type of rodenticide consumed. Clinical signs reflect ongoing haemorrhage and may include extensive intramuscular and subcutaneous haemorrhage, epistaxis, pallor of the mucus membranes, anaemia, melena, prolonged bleeding from phlebotomy sites *(Figure 7)* and death.

Diagnosis

A presumptive diagnosis can frequently be based on the history (although this is often not available or provided), clinical signs and demonstration of anaemia (via PCV). If the animal dies, necropsy should be definitive. Evaluation of prothrombin time may help confirm the diagnosis, assess response to treatment and determine when to cease treatment.

Treatment

The mainstay of therapy is administration of vitamin K_1 (3-5 mg/kg PO bid). Duration of therapy depends on the anticoagulant ingested (first generation anticoagulants—14 days, second generation anticoagulants—28 days); as this is often unknown, treatment should be continued for 28 days. Ideally, prothrombin time should be evaluated 72 hours after cessation of vitamin K_1 therapy to assess if ongoing treatment is required. Possums frequently present in an advanced stage of disease and require a blood transfusion and ongoing IV fluid support to stabilise. A single transfusion with blood collected from a healthy conspecific can be performed without cross matching and otherwise follows the principles used in domestic animal medicine.

Parasitic Diseases

Neural Angiostrongyliasis

Aetiology

Angiostrongyliasis, an emerging disease, is caused by the aberrant migration of the larvae of the rat lung worm, Angiostrongylus cantonensis through the central nervous system resulting in a granulomatous encephalitis and meningitis (Johnson and Hemsley 2008, Vogelnest 2019c). Definitive hosts in Australia include various species of introduced rodents. Possums, and other accidental hosts, are infected following ingestion of third stage larvae.

Clinical signs

Clinical signs are variable, although paresis and paralysis of the hind limbs are common findings in Australian mammals. Other signs include nystagmus, proprioceptive deficits, ataxia, seizures, opisthotonos and weight loss (Vogelnest 2019).

- **Diagnosis**
  Antemortem diagnosis is challenging and is generally presumptive after ruling out other differential diagnoses.

- **Treatment**
  Treatment is rarely successful and, if attempted, centres on the use of corticosteroids to reduce the inflammatory response associated with the presence of the parasite. Anthelmintics are generally considered to be contraindicated as the death of the parasites incites a severe inflammatory response and can worsen the clinical signs.

Miscellaneous Conditions

Swollen Paw Syndrome

- **Aetiology**
  A disease of unknown aetiology occurring in common ringtail possums in the greater Sydney region (ARWH 2005), elsewhere in New South Wales, as well as Victoria and Queensland (Spielman pers. comm). All age classes and sexes have been affected. A range of bacteria has been isolated from lesions with *Staphylococcus aureus* predominating. Numerous potential causes have been postulated including photosensitisation, bacterial and viral infections, thermal injuries, various plant toxins and *Laticlerada nidicola* insects and/or leeches (Spielman pers. comm).

- **Clinical signs**
  Clinical signs initially involve oedema of the front paws, progressing to oozing, ulceration with suppurative tenosynovitis and, ultimately, avascular necrosis of the paws. Lesions may develop at other locations including the bridge of the nose, lips and pinnae (Higgins *et al*. 2018).

- **Diagnosis**
  A suspected diagnosis can be further supported by suggestive histological changes in biopsy samples from affected areas. Histological changes include coagulative or caseous necrosis of the epidermis with necrosis and fibrosis of associated blood vessels.

- **Treatment**
  An effective treatment regimen has not been established.

Nonsuppurative Meningoencephalitis

- **Aetiology**
  A neurological disease of common brushtail possums. A viral aetiology is suspected but not proven. A similar disease seen in New Zealand (wobbly possum disease) is associated with a nidovirus and a similar nidovirus has been isolated from Australian possums.

- **Clinical signs**
  Clinical signs include tremors, ataxia, crouched hindlimb gait, blindness with persistently dilated pupils, and incoordination. Progression of the disease occurs over weeks to months.

- **Diagnosis**
  Diagnosis is based on clinical signs and supportive histological findings. Histological findings include haemorrhage, malacia, and fibrinoid vascular necrosis in the meninges and parenchyma of the cerebrum, cerebellum, brainstem and cervical spine (Higgins *et al*. 2018).

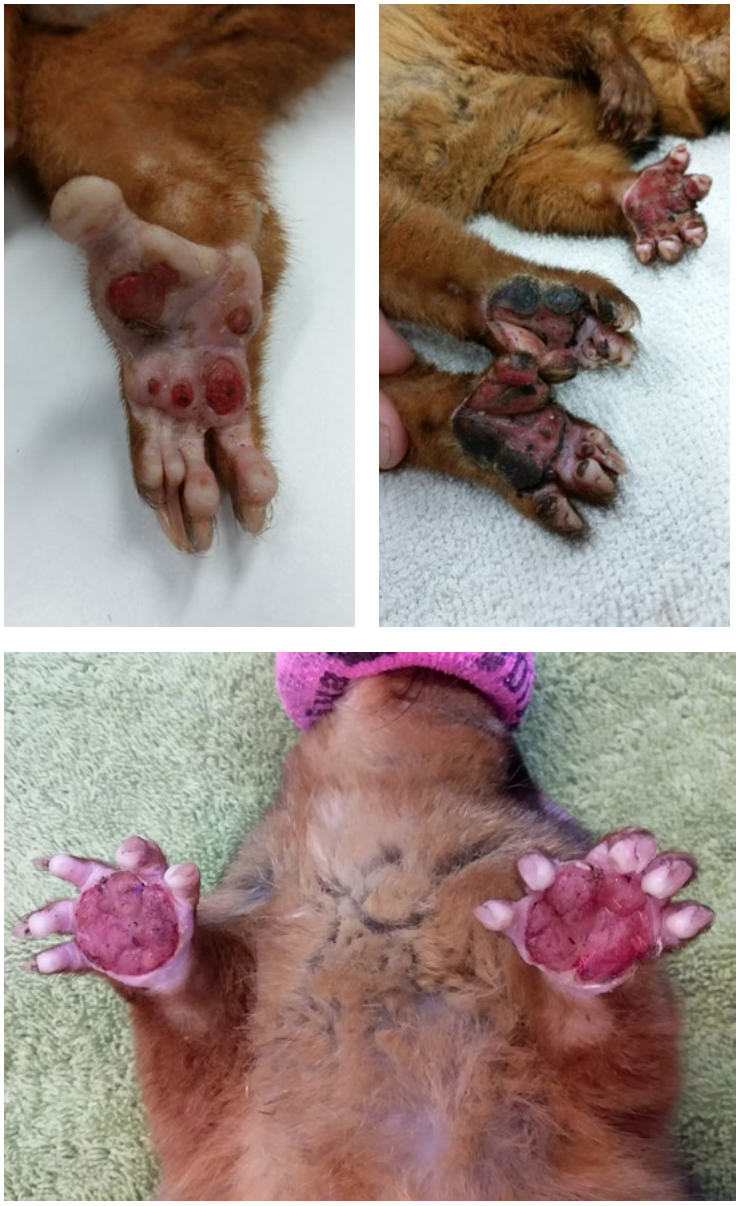
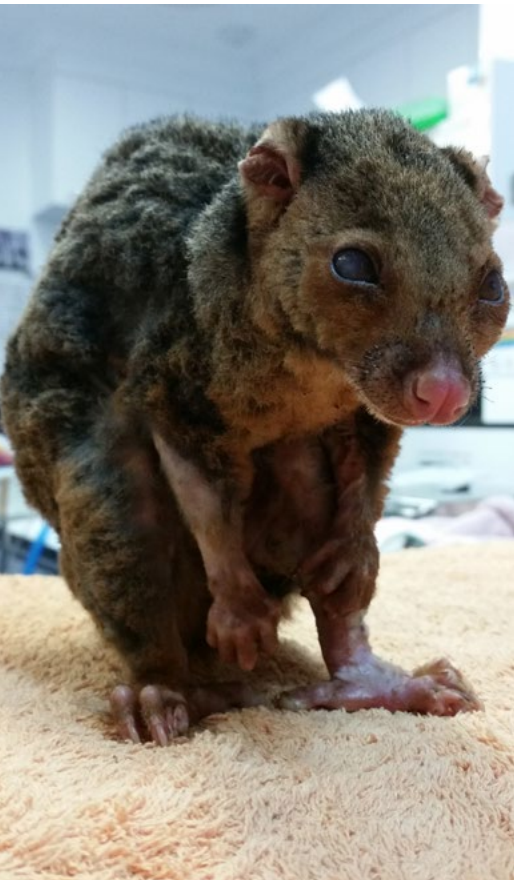


Figure 8. a) Full thickness burns of limited extent on the plantar surface of the pes in a common ringtail possum. b) Extensive full thickness burns on all feet of a common ringtail possums that have been present for several days; euthanasia is indicated. c) Full thickness burns on the palmar aspect of the manus in a common ringtail possum.

- Treatment
  An effective treatment regimen has not been established.

Thermal and Electrical Burns



- Aetiology
  Thermal burns on the palmar/plantar aspect of the manus and pes are common findings during the warmer months in common ring-tailed and common brush-tailed possums due to contact with hot tin rooves, bitumen and barbeques. Electrical burns can occur at any time of the year following contact with power lines; common ringtails seem more frequently affected than other species.

Figure 9. a) Electrocution injuries in a common ringtail possum; note the singed fur, burnt vibrissae and corneal oedema. b) So called current marks on the ventral surface of an electrocuted common ringtail possum.

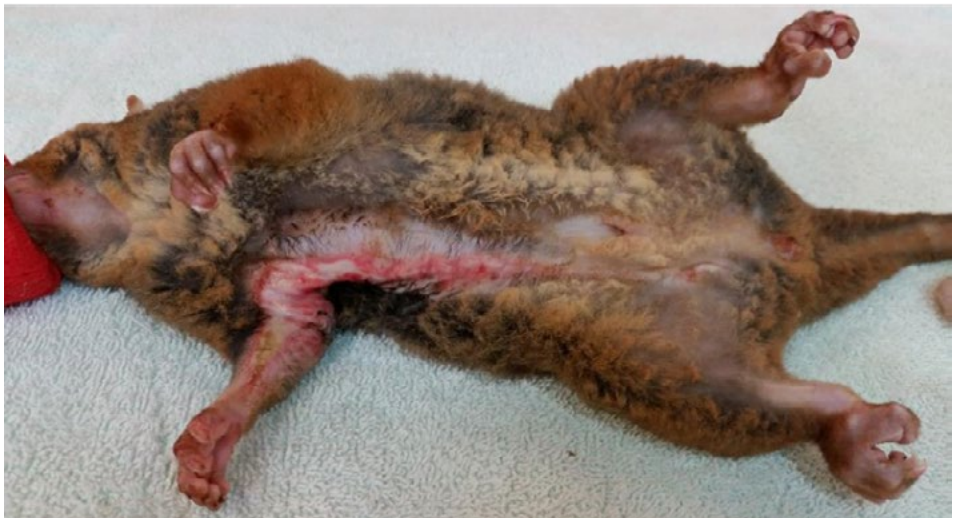


Figure 10. Age related cataracts in a common ringtail possum

- ### Clinical signs
  Possums with thermal burns are frequently reluctant to walk, exhibit weight loss and dehydration and have burns of varying degrees affecting one or more feet *(Figure 8 on previous page)*. Clinical signs associated with electrical burns include singed fur and vibrissae and so-called current marks; frequently on the limbs and/or tail *(Figure 9)*. Complications include pulmonary oedema, rhabdomyolysis, myoglobinuria and cataract development.

- ### Diagnosis
  A diagnosis can be based on the history (if known) and clinical signs.

- ### Treatment
  Treatment of thermal burns follows the principles used in domestic animals. Early debridement of necrotic/sloughing tissue is recommended. Burnt areas should be gently bathed under anaesthesia every 48 to 72 hours. Possums use their forelimbs to prehend food so frequently do not tolerate bandaging of the fore feet well; leave these unbandaged with minor burns. Burn wounds are painful and adequate analgesia for the duration of treatment is imperative. Opioids such as buprenorphine (0.01 mg/kg IM bid) plus carprofen (4 mg/kg SC bid for up to 72 hours) are indicated initially while paracetamol/codeine (15 mg/kg of paracetamol component PO tid) and gabapentin (10 mg/kg PO bid) have been used for longer term analgesia. Prokinetics such as cisapride (0.2 mg/kg PO bid to tid) should be administered concurrently with opioid analgesics.

Trauma

- ### Aetiology
  Vehicular trauma and barbed wire entanglement are common reasons for presentation in possums and gliders respectively. Domestic animal attacks, especially from cats, are common in juvenile possums, especially common in ring-tailed possums, and gliders.

- ### Clinical signs
  Vehicular trauma injuries mirror those in domestic mammals. Clinical signs associated with cat attack include dried saliva on fur, patchy alopecia, scratches and puncture wounds. Dog attacks tend to cause significant crushing injuries and larger puncture wounds.

- ### Diagnosis
  A diagnosis is based on the history and clinical signs.

- ### Treatment
  Treatment for domestic animal attacks and motor vehicle trauma follow the principles used in domestic animals. If extensive soft tissue trauma or deep puncture wounds are present, euthanasia is indicated. Septicaemia associated with *Pasteurella multocida* is a common sequala. Treatment with parenteral amoxicillin/clavulanic acid (15-20 mg/kg IM sid for 5 to 7 days), in conjunction with appropriate analgesia and concurrent administration of oral nystatin (50,000 IU/kg tid) to prevent candidiasis in juveniles, is recommended for treatment of less severe cases.

  Significant tail injuries in common ringtail possums and, to a lesser extent, common brushtail possums are associated with a poor prognosis and euthanasia is recommended. Generally, no more than one third of the tail length should be amputated.

  The prognosis for gliders entangled in barbed wire depends upon the extent of the patagium involved and the location of the injury. The full extent of necrosis of injured patagial tissue may take 3-7 days to become fully apparent so it may not be possible to determine an accurate prognosis until the glider has been in care for up to a week. Surgical resection and attempts to suture patagial deficits are frequently unrewarding. Minor patagial deficits will not preclude release but larger deficits and tears involving the leading edge of the patagium will affect the animal's gliding ability and euthanasia is recommended.

Age-related Changes

- ### Aetiology
  Various degenerative changes are seen in aged possums. These animals typically present in the cold months when food is scarcer and the weather much harsher. Changes include cataracts, dental attrition, spondylosis deformans and degenerative joint disease (Figure 10).

- ### Clinical signs
  Clinical signs vary with the underlying cause but possums frequently present in poor condition with poor pelage associated with reduced vision and/or mobility which limits their ability to forage.

- ### Diagnosis
  Diagnosis is straight forward based on the clinical findings.

- Treatment
  Treatment is not recommended as they will never regain their previous health and vigour; euthanasia is the most humane option.

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Tim regularly writes wildlife-specific papers for veterinarians at mixed practices around Australia.

He and practice member RSPCA Queensland have generously agreed to share these in the *C&T Series* to disseminate this valuable information far and wide ◆

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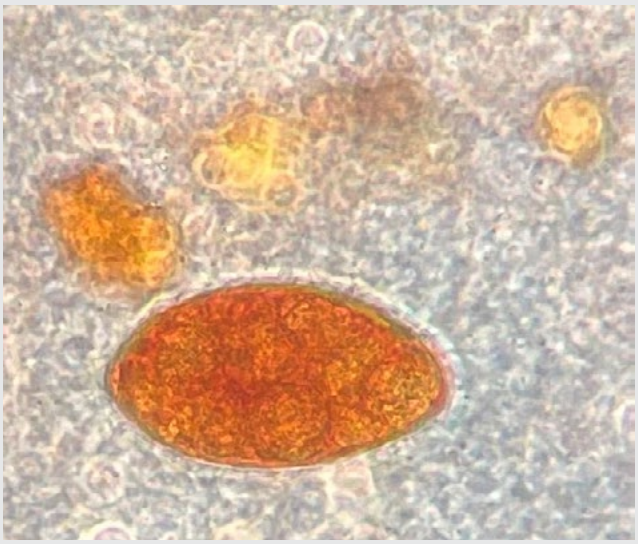
WHAT IS YOUR DIAGNOSIS?

C&T No. 5947

Question: What is your diagnosis and how would you treat this case?



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