C&T

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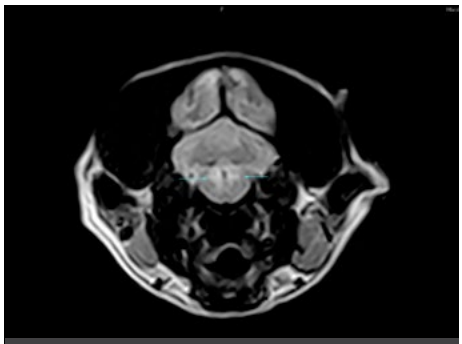

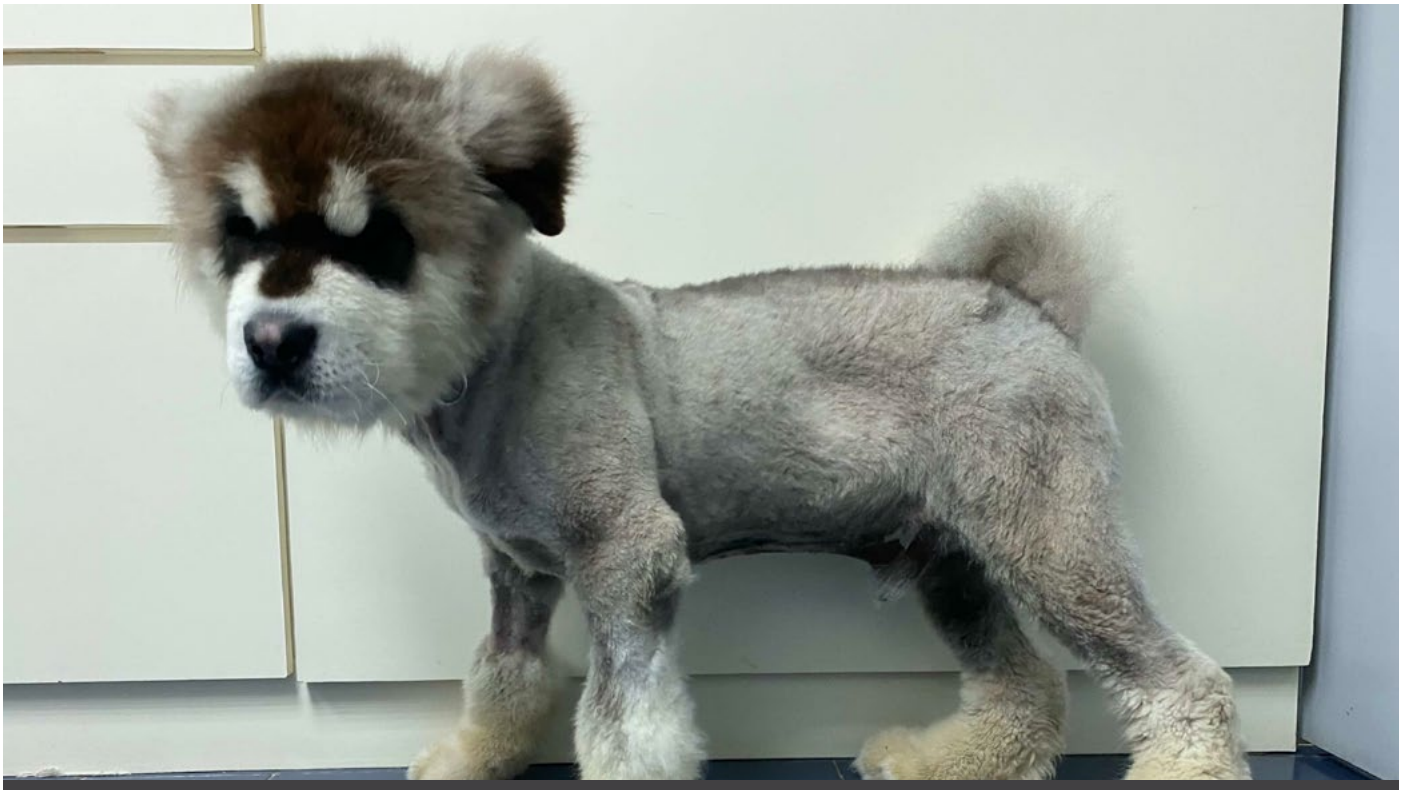

... **saliva testing** for FIV antibodies in a clinic or shelter setting is often easier to perform than blood testing...

... lymphocyte numbers gradually decline, causing **progressive dysfunction** of the immune system...

... **83% of cats in Australia** currently have, or have had, some level of access to the outdoors...

...the importance of **management and housing conditions** on the outcomes of FIV infection...

Expert panel addresses the **HOT TOPIC** of FIV

Despite the passage of over 30 years since its discovery, the importance of feline immunodeficiency virus (FIV) infection on health and longevity still remains a hotly debated topic. However, thanks to the dedicated work of a team of feline and infectious disease experts, Australian veterinarians now have access to part one of the **Australian Feline Retrovirus Management Guidelines** covering FIV. Find out what all the chatter is about - download your copy from Animal Health Academy or use the QR code below.


Dr Mark Westman (Panel Chairperson)


Dr Sally Coggins


Dr Moira van Dorsselaer


Prof Richard Malik


Prof Jacqueline Norris


A/Prof Richard Squires


A/Prof Mary Thompson

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FROM THE DIRECTOR

One of my 'side gigs' here at the University is facilitating clinical communication skills workshops for DVM1 and 2 students. Numerous studies demonstrate effective communication is a critical contributor to job satisfaction and patient outcomes as a vet. Expertise in communicating is a skill that doesn't always get the recognition and celebration it deserves—but I'm convinced these things play just as critical a role in animal welfare as surgical and medical skills.

We're fortunate to have some incredibly gifted communicators contributing to this edition of the *C&T*. Tunbi Idowu's Perspective on IMPA is a perfect example—scholarly research presented in a conversational way. Likewise, Olivia Clarke's discussion of gastrointestinal stasis in rabbits is a must-read for anyone seeing the occasional (or frequent) bunny.

Kim Kendall's musing on cats and their capacity to bend us to their will is pure gold. The takeaway? Don't take 'pee-mail' personally.

The gallery of images collated by Ildiko Plaganyi is stunning—a pictorial representation of the decomposition of a dead sperm whale washed up on a Phillip Island beach. It doesn't sound so appealing when I put it that way—better to see for yourself on page 36.

From large life forms to small, Rick Atwell ponders the reasons contributing to the decrease in tick paralysis cases. Rick considers the multitude of factors impacting tick movement and population size, from disruption of natural habitat to the distribution of bandicoots (carriers of female ticks), climate change, and the shift in the way dogs are viewed and cared for by their owners. To me it seems a metaphor for the interconnectedness of all things.

Happy reading.

Simone

Winner

Entitled to a CVE$100 voucher

Exotic

RABBIT GASTROINTESTINAL STASIS SYNDROME

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C&T No. 5917

Introduction

One of the most common presentations of rabbits in general practice, emergency and exotic referral services is the rabbit that has stopped eating and defecating. Whilst these cases can present with varying severity, they frequently represent true emergencies and may deteriorate quickly or even become fatal without prompt treatment.[1] Gastrointestinal stasis is a syndrome with a complex pathogenesis and multifactorial aetiology.[1,2] It encompasses a spectrum of physiological consequences[3] with a common theme of hypomotility of the gastrointestinal system. This article discusses the pathophysiology, diagnostic process, and management of this common syndrome.

Figure 2. A rabbit eating parsley after recovering from gastrointestinal stasis.

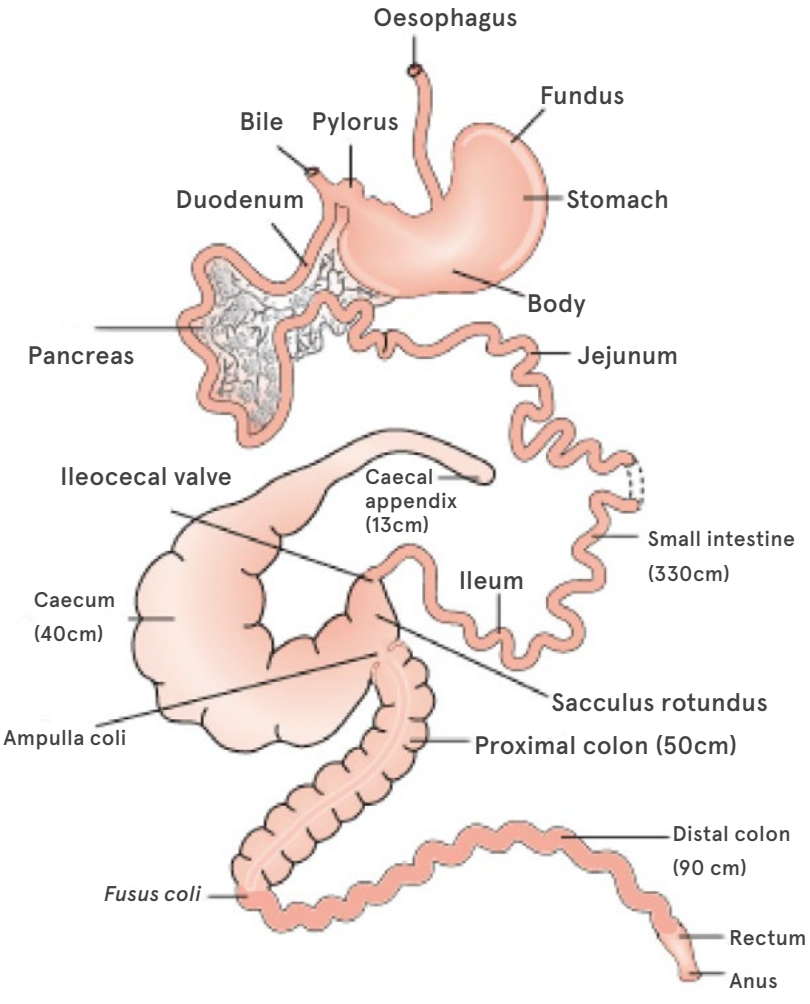

Figure 1. Diagram of the rabbit digestive tract, illustrating the complex hindgut.

GIT: Anatomy and physiology

To understand gastrointestinal stasis syndrome in rabbits, it is important to understand the complexity of their digestive tracts. Rabbits are hindgut fermenters, adapted to thrive on a high-fibre diet. Hindgut fermentation refers to digestion in the caecum by microorganisms. The powerhouse of digestion comprises the caecum and colon.

Rabbits have a simple stomach with a pH of 1-2.[4] It is normal for the stomach to always contain food and fur, even 24 hours after feeding.[4] Rabbits have a muscular pylorus and well-developed cardiac sphincter that prevents vomiting.[5] The stomach and small intestines are similar in anatomy and physiology to other monogastric species.[5] The

small intestines are responsible for digestion and absorption of proteins and sugars as well as vitamins and fatty acids from the caecotrophs[4] and hence have only a small role in digestion.

The terminal ileum dilates into a spherical, thick-walled dilation called the **sacculus rotundus**. This forms the ileocaecocolic junction. The sacculus rotundus opens into the **ampulla caecalis coli** at the ileocaecocolic junction.[5] A valve here, the ileocaecal valve, controls the movement of digesta from the ileum into the sacculus rotundus and prevents reverse flow into the small intestine.[4] The ampulla caecalis coli, caecum and proximal colon are specifically adapted for mixing and separating large quantities of fibrous particles. Large indigestible fibre particles are separated and directed distally through the colon, whilst small particles and fluid are directed proximally into the caecum to undergo bacterial fermentation.[5]

The caecum is a large fermentation chamber that holds approximately 40% of the digesta.[4] It contains a plethora of microorganisms involved in fermentation of plant material including bacteria, protozoa, yeast and amoebas.[1] The predominant bacterial genus is *Bacteroides*. These bacteria process plant fibre into digestible nutrients.[1] During the fermentation process, caecal bacteria produce volatile fatty acids (VFAs) that are absorbed across the caecal epithelium into the blood stream and are used as an energy source.[4]

Caecotrophs are the product of fermentation of amino acids, VFAs and vitamins.[5] They are formed in the proximal colon and caecum.[4] Caecotrophy is the process of ingesting caecotrophs directly from the anus.[4] This process is essential for rabbit health and lack of this process leads to nutrient deficiencies and reduced availability of protein and B, C and K vitamins.[4,6] Caecotrophy is driven by high fibre diets.[4]

The colon is divided functionally into the proximal and distal colon.[4] A key component of the proximal colon is the **fusus coli**, a highly innervated and vascular region, which is influenced by the autonomic nervous system and hormones such as aldosterone and prostaglandins.[4] It is often referred to as the pacemaker of the colon and controls the normograde passage of ingesta into the distal colon[4] and retrograde peristalsis of fermentable particles.[6] The fusus coli controls three types of intestinal motility; segmental, peristaltic and haustral and these differing contractions are involved in the production of hard and soft faeces (caecotrophs).[4] Because colonic motility is under the influence of the autonomic nervous system, any source of stress or pain, which increases adrenaline and noradrenaline release, can inhibit motility.[4]

Pathophysiology and causes

There are a wide number of potential causes of this syndrome. **Known causes of gastrointestinal stasis include stress, pain, ileus following anaesthesia and/or surgery and dietary factors such as high carbohydrate diets, rapid diet change and inadequate dietary fibre.[7]**

Gastrointestinal motility is influenced by many factors including indigestible fibre, pain, stress, disease, volatile fatty acids produced in the caecum, motilin and some pharmaceuticals such as opioids and prokinetics.[5] **Indigestible fibre is the greatest stimulant of motility.[1] It is essential for stimulating caecocolic motility.[1] Low fibre diet results in increased caecal retention time and hypomotility.[4] For pet rabbits, the primary source of indigestible fibre is hay or grass. Therefore, diets low in indigestible fibre are a major cause of gastrointestinal stasis.[1]**

VFAs produced by caecal fermentation also have a major role in gastrointestinal motility. The proportion of VFAs in the caecum influence motility and appetite. Acetate is the predominant VFA produced, followed by butyrate and propionate. Low fibre diets result in reduced acetate and increased butyrate, which inhibits normal peristalsis.[5]

Motilin is a polypeptide hormone secreted by the endocrine cells of the duodenum and jejunum and helps to stimulates motility by stimulating smooth muscle in the small intestine, colon and rectum.[5,6] Motilin secretion is inhibited by high carbohydrate diets.[4]

The fusus coli is under autonomic control.[5] Stimulation of the sympathetic nervous system in response to stress and/or pain causes release of adrenal hormones. Adrenaline and noradrenaline act on the enteric nervous system to inhibit intestinal motility.[2,5] Potential stressors for pet rabbits include changes in household routine, introduction or loss of pets or human companions, exposure to predatory species (dogs, cats) and inclement weather.[1]

Diseases that involve the gastrointestinal tract directly can alter motility, including caecal dysautonomia, coccidiosis and gastroenteritis.[5] However, any disease can result in decreased intestinal motility.[5] **Examples of painful conditions that may lead to anorexia and hypomotility include dental disease, otic disease, urinary tract diseases, respiratory infections, hepatic and renal conditions.[1,5]**

Consequences of hypomotility

There is a myriad of secondary consequences of hypomotility including dehydration, electrolyte, and acid base disturbances, hepatic lipidosis, hypoglycaemia, dysbiosis, gas accumulation and pain, enterotoxaemia, acute renal failure, and functional obstructions secondary to severe ileus or mechanical obstructions caused by trichobezoars.[5,7,8]

Water and electrolytes are continually exchanged along the digestive tract. Dysmotility alters this cycle of secretion and absorption. Decreased water and electrolyte reabsorption from the colon results in rapid dehydration and electrolyte imbalances.[5] Impaction of the stomach and/or caecum may occur due to disruptions in motility and consequential reduced water secretion into the stomach, caecum and colon resulting in desiccation of contents.[2,5]

Anorexia and reduced gastrointestinal absorption causes hypoglycaemia and puts rabbits in a catabolic state that leads to lipolysis and hepatic lipidosis.[5] Obese rabbits are at particular risk of developing hepatic lipidosis due to accumulated triglycerides in the hepatocytes.[5] Once hepatic lipidosis is established, lipid infiltration into kidneys may occur causing acute renal failure.[5] Rabbits lack renal ammonium buffering pathways for correcting acidosis[5] and are particularly susceptible to the effects of ketoacidosis. In one study, 40% of ill rabbits had acidaemia.[9]

Gastric ulceration is a common consequence of anorexia and hypomotility[5] and has been found in many rabbits at necropsy examination. In a survey of 1000 post-mortem examinations, 7.3% of rabbits were found to have gastric ulceration.[5] It is likely that the ulcers develop in response to stress as well as accumulation of gastric fluid increasing acidity.[2,7]

Hypomotility impairs caecal fermentation. This causes changes to caecal pH and VFA production[3,5] as well as disruption to normal enteric microflora.[2] The result is overgrowth of potentially harmful bacteria, namely *Clostridium* species and *Escherichia coli*[1] causing dysbiosis. These bacteria increase gas production in the stomach and intestines, leading to distension and pain.[1] In severe cases enterotoxaemia may ensue.[5]

In severe cases of dehydration and hypomotility a trichobezoar may form, causing gastrointestinal obstruction.[7] **It is important to recognise that trichobezoars are the result of anorexia and hypomotility rather than the cause.[5]** Gastric dilation and tympany causes cardiovascular and respiratory compromise and can result in hypovolaemic shock, hypotension and obtundation.[2]

Clinical presentation

The primary clinical signs associated with gastrointestinal stasis, not surprisingly include anorexia, and decreased faecal output. Faeces that are produced are generally smaller and dry[1] and some may be adjoined by fur. Other clinical signs include changes in demeanour such as withdrawn behaviour and not wanting to socialise. Rabbits may assume a hunched position and engage in bruxism, both indicators of pain.[3] Uneaten caecotrophs may be seen.[3] Generally, in non-obstructive cases there will be a gradual decline, progressing over more than 24 hours.[1,7] This is in contrast to gastrointestinal obstruction, which presents acutely, with rapid deterioration usually in less than 24 hours.[7]

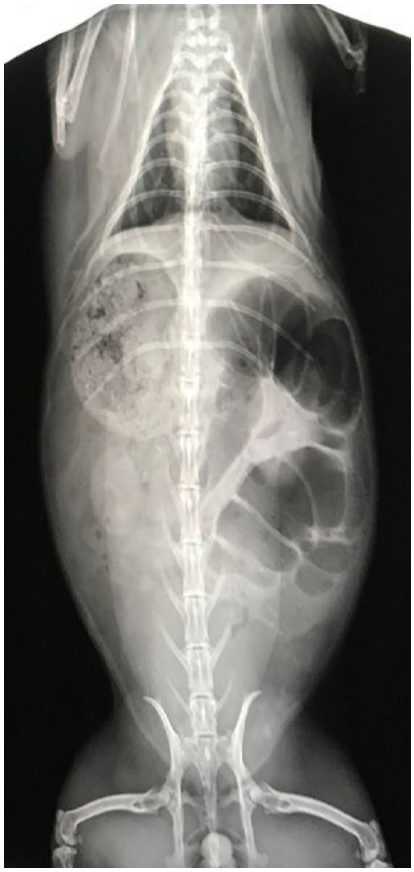

Figure 3. A ventrodorsal radiograph of a rabbit with gastrointestinal stasis, demonstrating severe gaseous dilation of the caecum.

Diagnosis

Attaining a diagnosis begins with performing a thorough physical examination. It is important to be able to discern between signs of stasis and obstruction and to be alert to the signs of shock in rabbits. In cases of hypovolaemic shock, rabbits may be bradycardic (<180) or tachycardic (>280) or they may have a normal heart rate as arterial

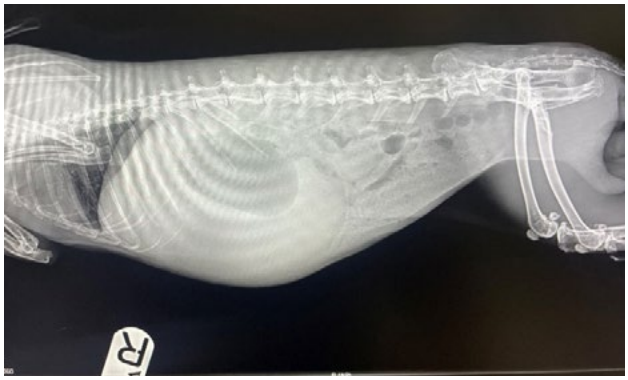

Figure 4. A lateral radiograph of a rabbit with gastrointestinal obstruction. The stomach is markedly dilated with fluid and gas (fried egg appearance) and there is minimal gas in the intestines.

baroceptors are often stimulated simultaneously by both vagal and sympathetic fibres.[3,8] They are often hypothermic (normal 38.5-40°C), they may have pale mucus membranes and an altered mentation.[3]

Rabbits in hypovolaemic shock are hypotensive (systolic blood pressure <90mmHg) but attaining an indirect blood pressure measurement may not always be possible.[3] Signs of shock are compatible with severe disease including gastrointestinal obstruction.[3]

The stomach must always be palpated. In a normal rabbit the stomach is soft, compressible and does not extend beyond the last rib.[9] A firm, dilated, tympanic stomach is compatible with an obstruction and radiographs should be performed as soon as possible in this instance. With gastrointestinal stasis, stomach, caecum, and intestines will often feel doughy on palpation. This is due to dehydration of gastrointestinal contents.[1] Peripheral dehydration is assessed as for other domestic species (i.e., delayed skin tent, endophthlamos).[9] Clinicians should auscultate the abdomen for borborygmi, which is usually reduced or absent in rabbits with gastrointestinal stasis.[2] Provided the rabbit is stable, a full examination should be performed including otoscopic dental and aural exam as dental disease and otitis are common pain sources that lead to inappetence and gastrointestinal stasis.[2] This is not a priority if the rabbit is compromised.[9]

As a minimum base-line, a packed cell volume (PCV), total solids (TS) and blood glucose (BG) should be run. The normal PCV is 30-45%.[5]

Increased PCV indicates dehydration, and this can be assessed alongside the TS. Normal blood glucose in rabbits is between 4.2-7.8 mmol/L.[5,9] Pain and stress cause elevations in blood glucose.[9] A study by Harcourt Brown demonstrated a correlation between marked hyperglycaemia and obstruction when glucose levels were 24.7 ± 3.9mmol/L.[10]

Other blood tests that can prove useful include acid base profile, electrolytes, and biochemistry to check hepatic and renal function. Rabbits may have a renal or pre-renal azotaemia.[3] They may have elevated liver enzyme activities due to hepatic lipidosis or liver lobe torsion.[2] Blood work can help to guide fluid therapy and provide prognostic indicators[5] as well as rule out other diagnoses such as liver lobe torsion.[9] Lactate levels may also be useful in monitoring response to treatment and the author has used this in her own practice; however, data in this area is limited.[9]

Orthogonal abdominal radiographs should be performed in all cases.2 Radiographs serve

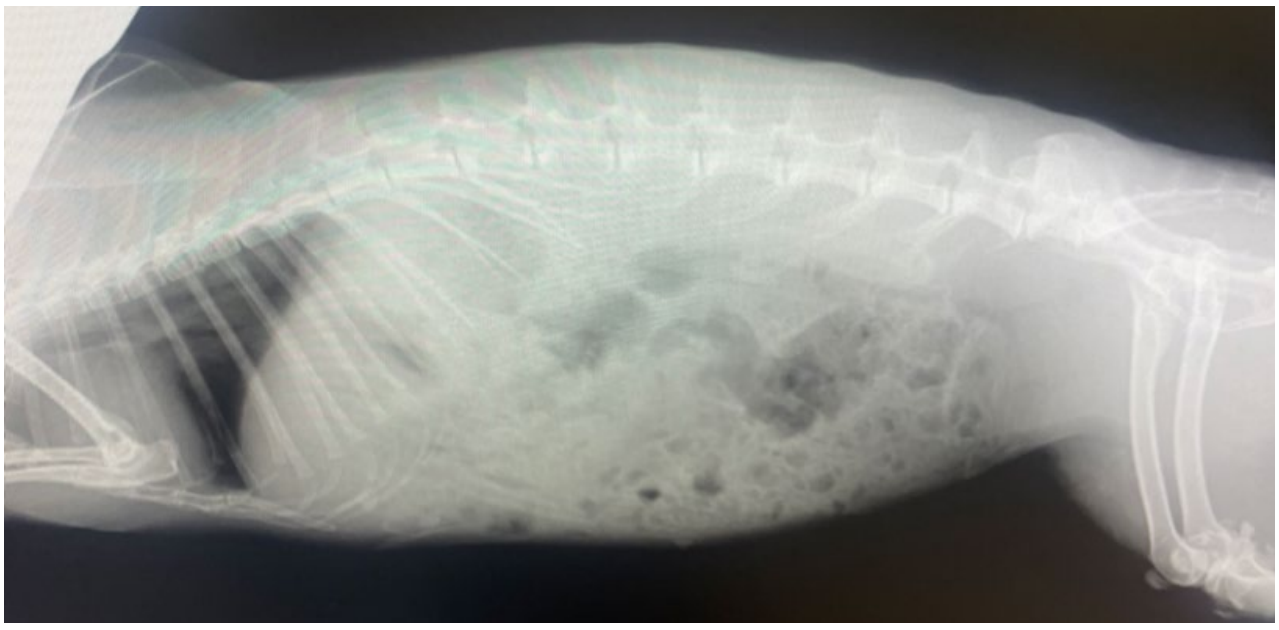

Figure 5. A lateral radiograph of a rabbit with gastrointestinal stasis. This stomach is only mildly enlarged but there is increased gas accumulation in the intestines.

the dual purpose of assessing the gas pattern in the digestive tract and discerning between obstructive and non-obstructive gastrointestinal stasis.1 In many rabbits that are unwell, these can be performed conscious. Otherwise, sedation is used. In the author's practice the typical sedation protocol is midazolam (0.25-1.0 mg/kg IM/IV) and buprenorphine (0.02-0.05mg/kg SC/IM). In normal rabbits, the stomach contains ingesta, it is reasonably small and does not extend beyond the most caudal rib.3 Gas is dispersed evenly in small pockets throughout the intestines, the caecum contains ingesta and there may be evidence of faeces in the colon.[3]

Rabbits that are obstructed have a largely dilated stomach with fluid and a gas cap (fried egg appearance), often with gas ending abruptly in the proximal duodenum.[1,3] Rabbits with gastrointestinal stasis may have a dilated stomach but it contains ingesta and there is increased gas accumulation or larger gas pockets seen throughout the gastrointestinal tract, compatible with ileus.[3,7] Radiographs are often performed serially to monitor response to therapy.[3]

Urinalysis can aid in diagnosis of ketoacidosis and metabolic acidosis. Rabbit urine is normally very alkaline, due to their herbivorous diet.[5] The normal pH is 7.5-9.0.[9] In cases of severe acidosis associated with hepatic lipidosis and anorexia there will be aciduria[9] and possibly ketonuria.[5]

It is important to identify and address the underlying cause(s) whenever possible[5], yet in many cases, the underlying cause is not determined.[3] Despite this, many rabbits will respond favourably to symptomatic and supportive therapy as described above.[3]

Management of non-obstructive gastrointestinal stasis

In order to manage these cases appropriately, it is imperative that clinicians be cognisant of the problems that need addressing. This can include anorexia, pain, dehydration, electrolyte imbalances, ileus, hepatic lipidosis, ketoacidosis and dysbiosis. The majority of rabbits will need to be treated as in-patients initially.[2] Rabbits should be hospitalised in a quiet, dimly-lit area away from predatory species.[2]

Nutritional support is instrumental in providing fibre for caecal nutrition and colonic motility and keeping the rabbit in a positive energy balance.[5] This is also important for managing consequences such as hypoglycaemia, dehydration and hepatic lipidosis.[5] **Rabbits should be syringe-fed a supplement that is calorie dense, high in non-digestible fibre and low in fats and carbohydrates.[3] Suitable enteral diets include Oxbow Critical Care, EmerAid Herbivore or Burgess Dual Care. An assortment of appropriate, palatable foods should also be provided *ad libitum* to encourage voluntary intake.[3]**

Gastrointestinal stasis is a painful condition[1], therefore analgesia is required. Mu opioid agonist drugs are used as first line.[9] Whilst it is true that opioids cause dose-dependent ileus they also inhibit pain and pain is a potent suppressor of gastrointestinal motility; therefore, rabbits with stasis typically respond favourably to opiate analgesia.[3] Buprenorphine can be used at a dose of 0.02-0.05 mg/kg SC, IM, IV or transmucosal every 6-12 hours.[7] A fentanyl CRI or transdermal patch can be used for severe pain such as in obstructive cases.[9] NSAIDs should be avoided until the patient has been rehydrated for at least 24 hours. Meloxicam can then be used at 0.5-1.0 mg/kg PO or SC every 12 hours.

Rabbits with gastrointestinal stasis are both viscerally and parenterally dehydrated.[9] Isotonic crystalloid fluids such as Hartmann's or Plasmalyte-148 are fluids of first choice.[9] Intravenous or intraosseous fluid therapy is generally required. **Subcutaneous fluids are not suitable for most unwell patients as the absorption will be poor due to hypovolaemia.[5]** Maintenance fluid rates for subcutaneous fluid therapy is 50-100mL/kg/day[9] and in the author's practice this is usually divided into 8-hourly doses. The maintenance fluid rate for intravenous or intraosseous fluid therapy is 3-4mL/kg/hour. Fluid rates are calculated based on maintenance requirements and estimated dehydration.[9] **Intravenous catheterisation sites include the lateral marginal auricular vein, the cephalic vein, and the lateral saphenous vein.** Application of EMLA 10-20 minutes prior to catheterisation can greatly facilitate placement. Sometimes fluid supplementation with potassium, glucose or bicarbonate may be required.[9] Hypoglycaemia can be treated with a bolus of 50% dextrose diluted 1:1 with saline at 0.25 mL/kg. If hypoglycaemia is persistent then a CRI of 1.25-2.5% dextrose is recommended.[9]

There is considerable debate regarding the efficacy of motility-modifying agents as an effective remedy for gastrointestinal stasis. In the author's practice they are used in some cases as adjunctive treatments but rarely as first line therapy. Cisapride promotes motility throughout the gastrointestinal tract.[7] The dose is 0.5mg/kg PO every 8 hours. In a recent study, cisapride demonstrated no effect on gastrointestinal motility.[11] Metoclopramide

stimulates gastric emptying, relaxes the pyloric sphincter, and promotes aboral movement of stomach chyme in humans.[12] It is less potent than cisapride and its effects are limited to the proximal gastrointestinal tract. The dose is 0.5mg/kg SC, IV or PO every 6 hours or can be delivered as a CRI at 2mg/kg per 24 hours.[1] There is no current reliable data to confirm the efficacy and safety of this drug in rabbits.[12] In rabbits that are stable enough, daily exercise outside of the hospital cage i.e., on the treatment room floor is also helpful for stimulating motility.[1]

Gastroprotectant medication is indicated in anorexic rabbits, especially if they have been anorexic for more than 48 hours[5,7] as gastritis and gastric ulceration are likely underdiagnosed. In the author's practice the H2 antagonist famotidine is frequently used. Additional anti-ulcer medications that can be used include omeprazole and sucralfate.[5]

Antibiotics are only indicated in cases where there is strong evidence of dysbiosis or enterotoxaemia.[1,2] Metronidazole (20mg/kg PO or IV every 12 hours) is most commonly used due to its efficacy against *Clostridia* species.[2] Cholestyramine can also be used to bind clostridial cytotoxins.[2,7]

As rabbits do not have *Lactobacillus* species as part of their normal microbial flora,[6,7] in cases of dysbiosis, traditional probiotics designed for other species need to be avoided.[12] Caecotrophs from a healthy rabbit can be used for transfaunation to re-establish a healthy caecal microbiome.[7]

Long-term management of gastrointestinal stasis involves recognition and treatment of the underlying cause[5] and ensuring the rabbit is on an appropriate diet. The optimal diet for a rabbit should comprise 80-90% hay (or grass). Oaten, timothy, meadow, orchard, and botanical varieties and teff hay are suitable. We generally recommend avoiding lucerne hay in rabbits > 6mo as the high calcium content can predispose to urinary sludge and calculi.

Approximately 10-20% of the diet should comprise leafy green vegetables, herbs, and weeds such as dandelion and 5% of the diet can comprise a grass-based pellet.

Patients can be discharged from hospital once they are eating and defecating.[1] It is important to note that some animals become so stressed in an unfamiliar environment that they will not eat in hospital, and many will gradually return to a normal appetite once at home.

Management of acute gastrointestinal obstruction or 'bloat'

Gastrointestinal obstructions are serious, life-threatening condtions.[1] The onset is rapid and if left untreated, death ensues within 12-24 hours.[1,5] The rabbit with an obstruction will often present moribund and display evidence of profound hypovolaemic shock.[5,7] The stomach will be firm, dilated and tympanic on palpation.[1,5] Many will be persistently hyperglycaemic.[1] These cases are both medical and, frequently, surgical emergencies.[13]

As rabbits cannot eructate or vomit, the stomach rapidly dilates with fluid and gas.[5] The stomach and intestine proximal to the obstruction become dilated with gas, which produces a typical obstructive pattern visible on radiographs.[5] Foreign bodies causing obstruction are most commonly trichobezoars (dehydrated mats of fur and food), but others may include pieces of carpet or plastic or intraluminal or extraluminal compression by abscesses, tumours, adhesions or tapeworm cysts.[1,5,7,13] The most common sites of obstruction are the proximal duodenum, approximately 2-5 cm distal to the pylorus and the ileocaecocolic junction[3,5] but pyloric obstructions, whilst uncommon may also occur.[5] The hugely distended

Figure 6. A patient with a nasogastric tube secured in place with tape and suture.

stomach not only causes severe pain but also compromises respiratory and circulatory function.[5] In some cases, the stomach may rupture.[1]

In some cases, the foreign body is able to pass through with aggressive medical management.[1,5] Initial stabilisation involves analgesia, aggressive IV fluid therapy and gastric decompression via nasogastric or orogastric tube.[5,7] A nasogastric tube (5-8 Fr red rubber) can often be placed conscious in obtunded rabbits or with mild sedation.[1,3,7] Opioid analgesia such as buprenorphine or fentanyl should be commenced as well as crystalloid IV fluid therapy. Rabbits should be started on a shock bolus with either crystalloid fluids at 10-20mL/kg or a combination of hypertonic saline (3 mL/kg) and colloid fluids such as hetastarch (3 mL/kg) administered concurrently.[1,3] Fluid therapy can then be continued as a constant rate infusion with the fluid rate dependent on maintenance requirements and estimated dehydration deficit.[3]

Rabbits that are hypothermic require heat support.[1] Serial radiographs should be performed every 1-2 hours to assess whether the obstruction is passing.[5] Acid base analysis and biochemistry are useful in guiding fluid therapy and providing prognostic information.[7] In cases that are refractory to medical management or are deteriorating, surgery is necessary.[1] A midline exploratory laparotomy is performed. The author will typically localise the obstruction (usually a trichobezoar) and milk the obstruction aborad into the caecum. Whenever possible, avoid enterotomy or gastrotomy. Specific surgical techniques are beyond the scope of this article.

There is a myriad of anaesthetic and surgical complications associated with celiotomy surgery in obstructed rabbits.[14] Surgical complications are not uncommon and include dehiscence (if gastrotomy or enterotomy is performed), peritonitis, adhesions, iatrogenic damage to viscera, ileus and recurrence.[5,14] If surgical intervention is required the prognosis is guarded with a reported survival rate of 30-40%.[3,6]

Gastrointestinal stasis is a complex and multifactorial condition. With an understanding of rabbit gastrointestinal anatomy and physiology, prompt diagnosis and appropriately guided therapy, clinicians can achieve successful patient outcomes in a large number of cases.

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Small

THIAMINE DEFICIENCY IN A DOG

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Case summary

A 6-year-old female spayed West Highland White Terrier presented to University Veterinary Teaching Hospital, Sydney (UVTHS) for acute onset vomiting and inappetence. Physical examination was unremarkable, and history of recent ingestion of material at the park made dietary indiscretion most likely. The patient was discharged following maropitant 2mg/kg IV for symptomatic treatment.

The patient represented to UVTHS two days later for persistent inappetence and newly developed ataxia. The vomiting had ceased following maropitant. There was no history of trauma.

On initial physical examination, the pertinent findings included a left sided head tilt and vestibular ataxia with delayed proprioception in all four limbs. There was no comment regarding physiological or positional nystagmus or strabismus on the attending record. Based on the neurological findings, central vestibular disease was suspected. The patient was admitted to hospital and a haematology, biochemistry and cryptococcal antigen lateral flow assay (CrAg LFA; IMMY) were submitted to Veterinary Pathology and Diagnostic Services (VPDS). The patient was treated with intravenous fluid therapy and intravenous maropitant (1 mg/kg IV).

Haematology and biochemistry revealed a mild hyperglycaemia (10.56 mmol/L; reference interval 4.11 – 7.95 mmol/L) and mild hyperlactaemia (2.9 mmol/L; reference interval <2.5 mmol/L). A cryptococcal antigen lateral flow assay (CrAG LFA; IMMY) was negative.

Overnight, the patient had a grand mal seizure, which resolved with diazepam 0.5mg/kg IV. The patient was subsequently loaded with levetiracetam at 60mg/kg IV.

The following day, the patient was referred to the internal medicine department. Further history was obtained revealing that the dog was fed a commercial, boutique diet consisting of freeze-dried chicken. The other dog in the household was also fed this diet and was clinically well. The owners reported that over the previous month the patient had become progressively lethargic and hyporexic.

On physical examination, the patient was noted to be quiet but responsive. Vital signs were normal. Neurological examination revealed a left-sided head tilt, reduced facial sensation on the left with no spontaneous nystagmus or strabismus appreciated. There was no pain on flexion or extension of the head or neck; however, the patient would become distressed on cervical dorsiflexion. There was noted hypermetria of the left forelimb, noting this was the IV catheter limb. There was delayed proprioception in the pelvic limbs. Patella reflex and withdrawal were reported to be unremarkable. No further seizures had been reported.

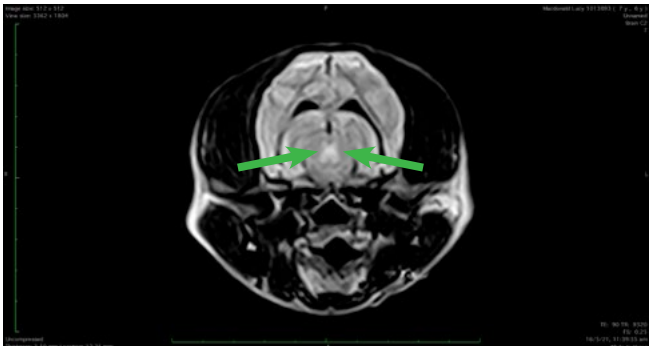
Multifocal disease was considered likely, given forebrain signs (seizure) and vestibular changes (vestibular ataxia, left head tilt). Differential diagnoses included meningitis of unknown aetiology (granulomatous meningoencephalitis, necrotising meningoencephalitis), infectious disease (including protozoal diseases (toxoplasmosis, neosporosis) and fungal diseases (cryptococcosis)), metabolic diseases (such as thiamine deficiency), infiltrative disease (primary or metastatic neoplasia), less likely cerebrovascular accident, idiopathic epilepsy or congenital or storage diseases given the age of presentation, neurological exam findings and rapid deterioration.

A fasting ammonia was obtained prior to anaesthesia, which was within reference limits. Serology for toxoplasma IgM / IgG and neospora IgG was not submitted, with the view to interrogate these differentials pending results of the MRI.

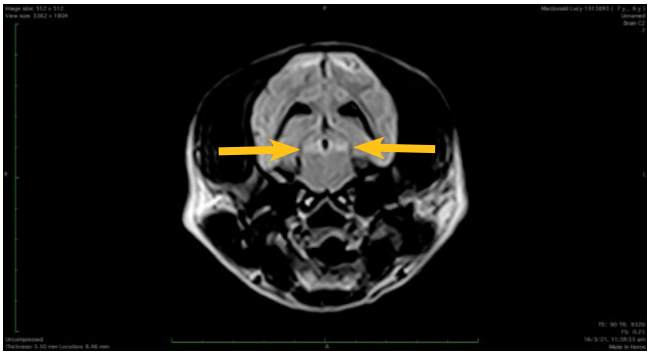
An MRI (0.25T) revealed bilaterally symmetrical T2W hyperintense lesions rostral colliculus, caudal colliculus and pons *(Figure 1)*. The bilaterally symmetrical changes made a toxicity or metabolic disease, such as thiamine deficiency or an inborn error of metabolism more likely than inflammatory, infectious, idiopathic or neoplastic disease processes. A cerebrospinal fluid (CSF) sample was obtained from the cerebellomedullary cistern and had a normal nucleated cell count with normal cell morphology and differential distribution. A whole blood sample (EDTA) was collected for thiamine levels, which was submitted to IDEXX laboratories. Based on low likelihood of protozoal disease based on these imaging findings, toxoplasma

and neospora serology was not submitted. A neurological PCR was declined at the time of submission.

The patient recovered uneventfully and was treated with Hartmann's fluid therapy, thiamine supplementation 10mg/kg IM q12 hours in addition to levetiracetam 20mg/kg IV q8 hours. Overnight, the patient became tachypnoeic and dyspnoeic. Thoracic radiographs were performed revealing unstructured interstitial pattern in the left cranial lung lobe, raising concern for early aspiration pneumonia. Following radiographs, the patient was noted to be hypertensive (Doppler blood pressure of 170 mmHg) and bradycardic suggestive of the Cushing's response and increased intracranial pressure. Amoxycillin-clavulanate 20 mg/kg IV q8 hours and mannitol 0.5 g/kg dilute slow IV were administered by the attending veterinarian for possible aspiration pneumonia and increased intracranial pressure, respectively.



(a)



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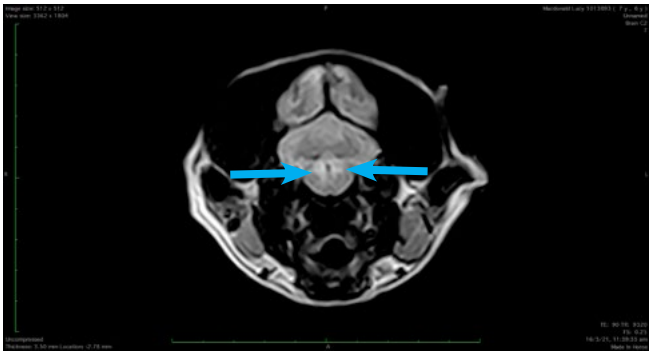

(c)

Figure 1. Transverse MR images at the level of the rostral colliculus (a), caudal colliculus (b) and pons (c): FLAIR sequences. FLAIR images show bilaterally symmetrical focal hyperintensities in the region of the mesencephalic aqueduct (green arrow), caudal colliculus (orange arrow) and pons (blue arrow).

Over the following 24 hours, the patient continued to deteriorate. Repeat thoracic radiographs revealed alveolar changes of the left cranial and right middle lung lobe and oesophageal dilation. Due to the critical status of the patient and prolonged treatment, the owners elected for humane euthanasia.

Following euthanasia, thiamine deficiency was confirmed based on red cell thiamine levels of 34.0 nmol/L (reference interval 113.0 – 309.0 nmol/L).

Given the suspicion of dietary-related thiamine deficiency, the bag of food was obtained for evaluation by the manufacturer *(Image 1)*. Thiamine and sulphite concentrations were tested on the patient's bag, a bag from the same batch, and a recent batch of food. The thiamine levels detected in the patient's bag were negligible at 0.2 mg/kg dry matter (DM). Thiamine levels from the same batch, and more recent batches were variable, ranging from 3.6 – 5 mg/kg DM. The minimum thiamine level recommended for dog food is 1.9 mg/kg DM. Sulphite levels in the patient's bag and new batches of food were assessed returning with levels of 75mg/kg DM and <5mg/kg DM respectively.

Discussion

This is an interesting case of presumed dietary-related thiamine deficiency in a dog. Both dogs and cats have an absolute dietary requirement for thiamine (Vitamin B1). Once absorbed across the gastrointestinal tract; it plays a vital role as a cofactor in energy metabolism and neural function, including neurotransmission. The nervous system is particularly vulnerable to deficiency due to the high metabolic demand.

Dietary thiamine can be supplied in the form of thiamine rich ingredients or the addition of synthetic supplements such as thiamine mononitrate or thiamine hydrochloride. Thiamine deficiency can occur as a result of dietary deficiency through anorexia, ingestion of thiaminase-rich products (such as fish), addition of sulphites as preservatives which degrade thiamine, or destruction of dietary thiamine by cooking or processing. Absorption-related deficiencies can involve a defect in one of the two thiamine receptors (THTR1 or THTR2) in the small intestine, with the most recognised form Alaskan Husky Encephalopathy. Rarely reported, excessive renal thiamine excretion can also occur.

Image 1. Pet food in question

There are three clinical phases of thiamine deficiency. Phase one consists of non-specific signs including vomiting, lethargy, hyporexia with or without weight loss. Phase two consists of acute onset neurological signs which can include ataxia, paraparesis, delayed pupillary light reflex, blindness, poor proprioception, or seizures. In cats, cervical ventroflexion and dyspnoea are more common presenting complaints. The final phase consists of neurological deterioration and eventual death. Thiamine is a co-factor for pyruvate dehydrogenase, which is required for the conversion of pyruvate to acetyl-CoA and entry into the mitochondria for the citric acid cycle. A deficiency of thiamine results in an accumulation of pyruvate, and subsequent hyperlactaemia. This biochemical change was appreciated in this case.

The dietary history, subtle biochemistry changes, and neurological deterioration, in addition to the bilateral MRI changes, served as the flags for thiamine-related disease in this case. MRI changes associated with this disease include symmetrical lesions in the brainstem affecting grey matter, reflecting bilateral necrosis and haemorrhage in susceptible brain nuclei. The most common regions affected include the red nuclei, caudal colliculus and medial vestibular nuclei (Garosi *et al* 2003).

There are numerous reports of dietary-related thiamine deficiency in Australia and elsewhere. The author highlights the case series by Singh *et al* (2005), describing five dogs of variable age with suspected thiamine deficiency. These authors identified that non-specific gastrointestinal signs including inappetence and vomiting were noted in the two adult dogs with the disease, similar to this case, and other cases reported in the literature. More recently, a case of thiamine deficiency was reported in a five-year-old Maltese dog, being fed a diet exclusively of sweet potato (Song & Jung 2020).

Treatment of thiamine deficiency involves supplementation with thiamine, initially intramuscularly followed by PO at a dose of approximately 10 mg/kg for four weeks, or until clinical signs resolve. Intravenous supplementation is not advised due to dose-dependent hemodynamic effects such as acute hypotension, cardiac arrhythmias, apnoea or death (Markovich *et al* 2013). Clinical signs tend to improve within one week of treatment; however, full recovery can take from several months to up to two years.

The minimum thiamine requirement for adult dogs is 1 mg/kg DM, with dietary thiamine recommended to be 1.9 mg/kg DM, significantly higher than noted in the patient's bag of food (0.2 mg/kg DM). The presence of sulphites coupled with low thiamine levels could suggest sulphite-related thiamine deficiency. Historically, sulphites have been used to extend the shelf-life of food. A random evaluation of 13 pet food products commercially available in Australia in 2005 found some with levels in excess of 1000 mg/kg DM. The authors noted that only three of the 13 products indicated the presence or absence of preservatives (Malik & Sibraa 2005).

Currently there is no government regulation of the pet food industry. Australian pet food is underpinned by the Pet Food Industry Association of Australia (PFIAA) Code of Practice. While the PFIAA Code of Practice recommends the listing of preservatives, including sulphites, the presence or concentration is not required by law. The exception to this is Queensland, with a label requirement for the inclusion of preservatives. The label of this food did not include an indication of the absence or presence of preservatives.

The manufacturers of the food noted that they routinely added thiamine to their products. Following investigations by the manufacturer, batches of food had variable, and sometimes low, levels of thiamine. The origin of the sulphites within this batch is unknown, and remains under investigation by the manufacturer. Following this case, mixture and processing requirements of the food have been updated, with pre-distribution tests demonstrating thiamine levels well above the recommended 1.9 mg/kg DM. Rapid on-site sulphite testing has been undertaken, and sulphite levels have remained low.

In retrospect, this patient was demonstrating phase one signs for approximately one week,

before deteriorating rapidly to stage two disease. Given the other dog was currently well, further history was obtained including previous `B vitamin injections' in the previous month for non-specific disease. Whole blood thiamine levels returned within normal limits (256 nmol/L). It is postulated that this dog may too have shown signs of early thiamine deficiency at this stage.

This case serves as a reminder that although uncommon, dietary-related nutritional deficiencies can occur. The authors recommend lodgement of any dietary-related disease with PetFAST Reporting, moderated through the Australian Veterinary Association. This provides means of recording and identifying any possible dietary associated diseases.

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Winner

Entitled to a CVE$100 voucher

Small

CAT CHEMOTHERAPY

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This particular case (and Richard Malik) taught me that chemotherapy is just a recipe. There are multiple recipes that you can use, you just have to find the right one for the patient. Jinx hated coming to our clinic and being hospitalised for chemotherapy, so we adapted our recipe, and how we delivered it, to suit Jinx. Possibly, we got lucky with Jinx; however, it did show me that as clinicians we need to be more flexible, work with our patients, their owners and just have a go. Not having a go can have dire consequences for our patients.

Jinx is a 5-year-old neutered male Tonkinese, `inside only' cat that lived with one older (13-year-old) Burmese male cat. He presented to our clinic because he missed his litter tray whilst urinating earlier that day, was off his food for a period of a week and a half and had possibly lost some weight. On initial examination he had a good body condition score (BCS), had halitosis with grade 2-3 dental disease, normal TPR, hind leg weakness and a large, firm, and painful mass within his abdomen.

Jinx was admitted for further work-up including routine screening bloods, ultrasound (+/- fine needle aspirate biopsy (FNA) of mass if possible) and intravenous fluids. He was sedated using

Zoletil® (4mg/kg) and an in-house ultrasound was performed. He was placed on 3mL/kg/hr Hartmann's for the procedure. Ultrasound revealed that his left kidney was > 8.5 cm and his right kidney was approximately 5.5 cm; a FNA was performed with in-house cytology suggestive of renal lymphoma.

Smears were sent to Veterinary Pathology and Diagnostic Services, The University of Sydney, with the findings consistent with malignant round cell neoplasia—renal lymphosarcoma (large lymphoid cells).

We began treatment for renal lymphoma working on the theory that the sooner we began treatment the more likely we would see if he were going to respond favourably. We continued with intravenous fluid at the current rate of 3mL/kg/hr with 20mmol/L KCl added. We gave Jinx vincristine IV (0.03 mg/kg), dexamethasone 1 mg (total dose) IV, maropitant (1 mg/kg) IV and buprenorphine (0.015mg/kg). The plan was to give Jinx a dose of L-asparaginase IM as soon as we could source the drug and he would remain in hospital on IVFT to support his kidneys and monitor him for a response.

We received Jinx's biochemistry results the next day with his SDMA, creatinine, urea and total calcium all being elevated *(see table 1)*. He also had a high WBC count with elevation mostly of his neutrophils. A urine culture was not done at the initial work-up, and because Jinx was becoming a little fractious, we opted to just begin intravenous amoxicillin 22 mg/kg IV q24h. We placed a fentanyl (25 µg/hr) patch on a hind leg and continued with intravenous maropitant at 1 mg/kg.

Jinx had eaten all the food that was left for him overnight and was urinating well. Initially when Jinx presented, he was quiet and anxious; however, by the third day of hospitalisation he had become quite cross (understatement!) and was now hissing and striking whenever we approached the cage. Examination became impossible and all medications were given intravenously. His owner was able to visit, and he was much more relaxed in her company. It was decided that once Jinx had received his L-asparaginase dose (under sedation) we would send him home with his owner on all oral medications.

Four days post initial presentation, we sedated Jinx again with Zoletil® (2 mg/kg). We administered L-asparaginase (450 IU/kg) intramuscularly and removed his indwelling intravenous catheter. He was discharged with oral Clavulox® (50mg bid), prednisolone (10mg sid), mirtazapine (1.88 mg prn) and maropitant (4 mg sid).

Due to 'owner reasons' Jinx did not return for his next dose of chemotherapy until 4 weeks after his initial treatment. He was dropped off by his owner who reported that he was amazing and totally back to normal. Jinx was unexaminable and was sedated again with Zoletil® (4mg/kg) IM with good effect. He had lost 60 grams since his initial presentation. On abdominal palpation, his kidneys were no longer enlarged. A 24g IV catheter was placed in the right cephalic vein, and he was given 50 mLs of saline over 40 minutes. He received vincristine IV (0.03mg/kg), maropitant IV (1mg/kg) and we repeated his IM dose of L-asparaginase (450 IU/kg). Please note that we had purchased Sanofi Aventis Leunase® Inj (10,000.U : 2000U/mL) and due to the high cost to the client and the fact that we had so much left in the vial we chose to repeat the dose of L-asparaginase, as the drug is said to have reasonable activity for up to 4 weeks after being reconstituted. We had informed the client that it may not have the same efficacy as freshly made drug, but she was happy to use what was remaining instead of wasting the unused medication.

At the same time, we took bloods (routine screen) in readiness for giving him doxorubicin the following week. He was sent home on maropitant (1.5mg/kg sid), mirtazapine (1.8 mg) and Clavulox® (50mg bid).

Blood results revealed that Jinx was anaemic with no evidence of regeneration *(see Table 1)*. He also had a low WBC count; although all results apart from the lymphocytes were low normal. He also had a slightly low total protein. However, his SDMA, creatinine and urea (*see table 1*) were all back within normal limits.

On discussion with his owner, we decided to treat Jinx with darbepoetin and Ferrum H® iron injections. His owner also reported that Jinx had been miserable (lethargic and anorexic) since his chemotherapy the day prior; she was also struggling to medicate him orally. We gave his owner gel capsules to allow for multiple medications to be administered simultaneously and dispensed gabapentin (100 mg capsules) to be given prior to treatment with darbepoetin and iron injections, as both can sting, and Jinx was really not co-operating on visits to the clinic. It was decided that we would no longer give Jinx more than one chemotherapy agent at a time as it really made him feel miserable after dosing.

It should also be noted that we only had 'out of date' darbepoetin available and due to the owners very limited finances we decided to give Jinx the out-of-date darbepoetin.

Jinx's owner had given him gabapentin (100mg) 1.5 hours prior to his visit at our clinic. His owner held him while we gave him 5 µg of darbepoetin and 1 mL Ferrum H iron IM. He tolerated the injections extremely well and we then took a small blood sample from his medial saphenous vein while his owner held him for an in house PCV (28%) and TS (78g/L). Jinx had lost a further 150 g. No further examination was performed at this consultation.

His owner reported 7 days later that he had improved significantly; he was eating dry food again and was basically back to normal. Jinx returned for a full check up with his owner 3 weeks later. He had gained 200g in weight since his last visit, he was reactive but allowed gentle examination while his owner was holding him. His kidneys were still a normal size and non-painful. His owner had stopped his prednisolone and he was currently taking no medication. We started him back on prednisolone, which continued for the duration of his chemotherapy.

At 10 weeks post his diagnosis of renal lymphoma, he had received two doses of vincristine, two doses of L-asparaginase, prednisolone, darbepoetin and Ferrum H iron injections. We again repeated his L-asparaginase injection (450IU/kg subcutaneously this time) as we still had some left in his vial. He was sent home on Clavulox (50 mg bid).

Jinx returned the following week, again his owner had given him gabapentin (100mg) prior to his visit. His owner held him while he was given vincristine (0.03 mg/kg) intraperitoneal (IP) using a 19g butterfly catheter and flushed with 3 mL NaCl. He was given 100 mLs subcutaneous fluids (0.45% NaCl & 2.5% glucose). Bloods were taken from his jugular for repeat routine screening while his owner held him in her arms. We decided to give him two weeks to recover and sent him home with oral maropitant (1.5mg/kg), mirtazapine (1.8 mg) and Clavulox (50mg/kg).

His blood results showed a slight elevation of SDMA and urea *(Table 1)* and low total WBC, although individually all were within normal limits. His RBC and HCT were normal.

From here, we developed our own protocol for Jinx giving him vincristine IP with his owner holding him every 6 weeks. He was always given SC fluids at the time of IP vincristine and then his owner would give him oral cyclophosphamide (50 mg tablet) one week following his IP vincristine dose. We repeated this cycle roughly every 6 weeks depending on Jinx and his owner's health. We never repeated his darbepoetin/Ferrum Iron injections again and he never received doxorubicin either. Jinx received vincristine (IP) /cyclophosphamide (PO) /prednisolone (PO) for just short of 12 months, roughly every 6-8 weeks before we ceased it completely.

Almost 12 months after his initial diagnosis of renal lymphoma we did his dental procedure and removed 8 teeth uneventfully. The most recent bloods were taken 19 months after his initial diagnosis and at this stage he remains in remission. A very satisfactory result for what was basically COP chemotherapy with some L-asparaginase. Sometimes you can be lucky!

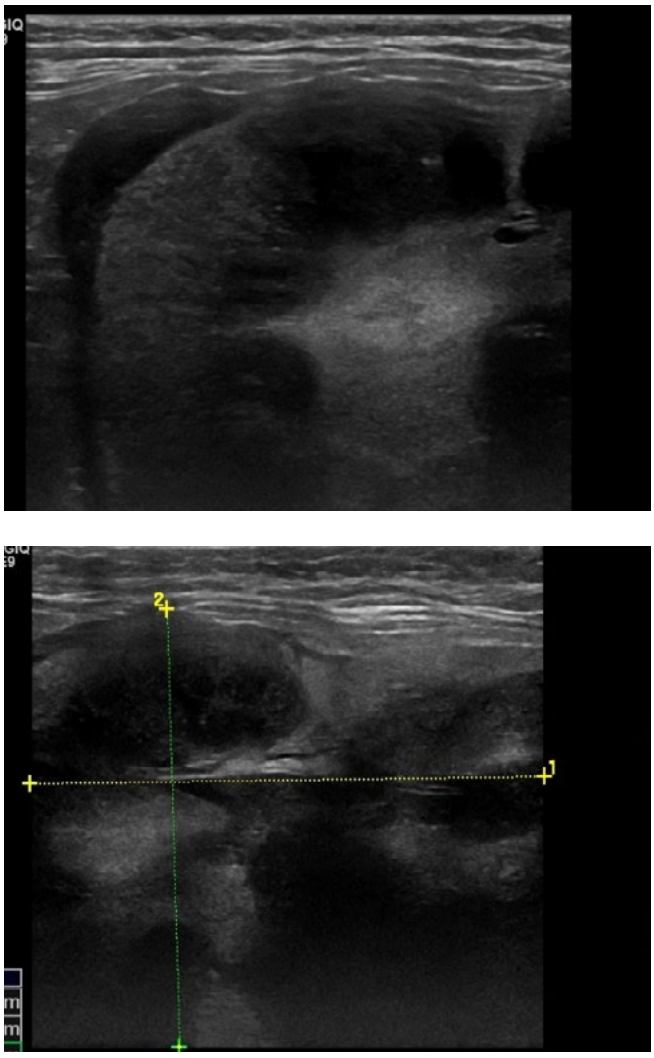


Figure 1. Ultrasound images of kidney on presentation

Table 1

Analyte	22.2.19	22.3.19	10.5.19	22.11.19	29.5.20	25.9.20	Reference Interval
Haematology							
RBC	5.3	3	5.7	6.3	6.6	6.8	**(4.9-10)x10^{12}/L**
Haematocrit	0.31	0.17	0.31	0.43	0.37	0.36	**(0.25-0.48)L/L**
Haemoglobin	89	51	98	116	120	116	**(77-156)g/L**
MCV	58	57	54	68	56	53	**(43-55)fL**
MCH	17	17	17	18	18	17	**(13-17)pg**
MCHC	287	300	316	270	324	322	**(282-333)g/L**
% reticulocyte	0.1	0.1	0.1	0.2	0.2	0.2	**(0.0-0.4)%**
Abs. Reticulocytes	5	3	6	13	13	17.5	**(3-50)x10^9/L**
WBC	22.7	3.3	4.6	7.5	7.7	7.5	**(5.5-19.0)x10^9/L**
Neutrophils	17.9	2.1	2.3	2.7	3	2.9	**(2.0-13.0)x10^9/L**
Lymphocytes	3.2	0.8	1.5	2.4	3.6	2.9	**(0.9-7.0)x10^9/L**
Monocytes	1.4	0.2	0.4	0.4	0.4	0.4	**(0.0-0.6)x10^9/L**
Eosinophils	0.2	0.2	0.4	2	0.7	1.4	**(0.0-1.0)x10^9/L**
Basophils	0	0	0	0	0	0	**(0.0-0.1)x10^9/L**
Platelets		398		291			**(300-800)x10^9**
Platelet Comments	Clumped and adequate	Clumped and adequate	Clumped and adequate	Clumped and adequate	Platelet clumps confirmed, consistent with adequate numbers.		
Blood film	Mild toxic changes	Mild nisocytosis	Mild nisocytosis	Red cell and white cell orphology normal	Mild anisocytosis, occasional echinocytes and small numbers of smudged red cells. White morphology normal		
Evaluation	Platelet estimate from the >300x10^9/L	White cell morphology normal	White cell morphology normal. Many small platelet clumps, platelet estimate from mear>300x10^9/L	Few medium sized platelet clumps	Platelet estimate from the smear >300x10^9/L		
Chemistry							
Glucose (Flouride oxalate)	4	5.7		4.1	4.1	6.6	**(3.2-7.5)mmol/L**
IDEXX SDMA	68	14	15	13	12	12	**(0-14)µg/dL**
Creatinine	0.22	0.18	0.18	0.17	0.15	0.18	**(0.08-0.20) mmol/L**
Urea	23.2	14.9	15.1	15.7	13.9	14	**(5.0-15.0mmol/L**
Phosphorus	1.6	1.3	1.4	1.3	1.3	1.5	**(1.0-2.3)mmol/L**
Calcium	3.8	2.2	2.4	2.4	2.5	2.7	**(2.1-2.8)mmol/L**
Calcium:Phosphorus Ratio	2.4	1.7	1.7	1.8	1.9	1.8	**(1.1-2.3)**
Sodium	152	152	152	158	155	157	**(144-158)mmol/L**
Potassium	4	3.8	4.4	4.9	4.7	4.8	**(3.7-5.4)mmol/L**
Na:K Ratio	38	40	34.5	32.2	33	32.7	**(29.0-40.0)**
Chloride	119	117	114	121	113	120	**(106-123)mmol/L**
Bicarbonate	13	20	22	17	23	21	**(12-24)mmol/L**

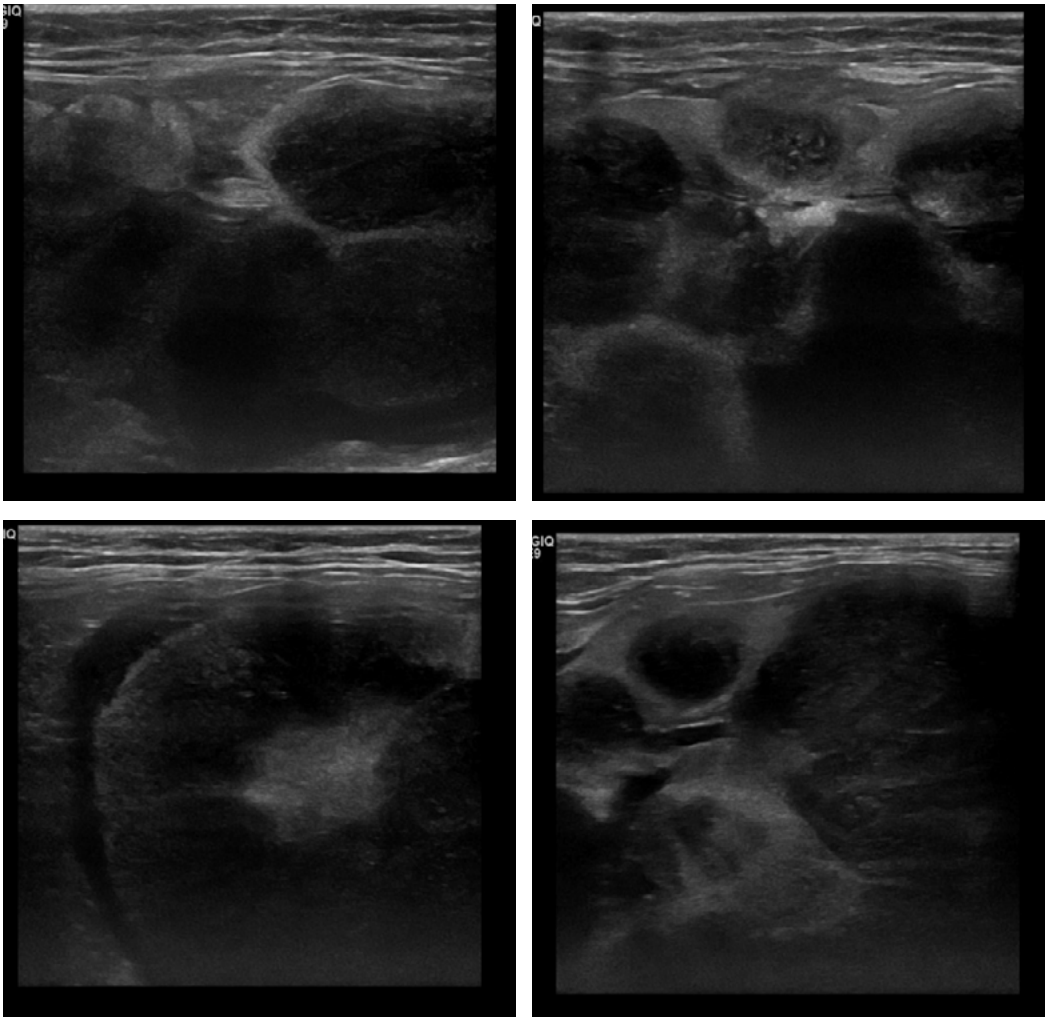
Contents

Table 1 continued

Analyte	22.2.19	22.3.19	10.5.19	22.11.19	29.5.20	25.9.20	Reference Interval
Anion Gap	24	18.8	20.4	24.9	23.7	20.8	**(15.0-31.0) mmol/L**
Total Protein	71	58	72	77	75	77	**(60-84)g/L**
Albumin	28	28	30	34	33	35	**(25-38)g/L**
Globulin	43	30	42	43	42	42	**(31-52)g/L**
Albumin:Globulin Ratio	0.7	0.9	0.7	0.8	0.8	0.8	**(0.5-1.1)**
ALT	38	24	41	52	55	52	**(19-100)IU/L**
ALP	4	17	34	21	20	30	**(5-50)IU/L**
AST	84	26	20	42	32	27	**(2-62)IU/L**
GGT	2	0	0	1	0	1	**(0-5)IU/L**
Bilirubin Total	5	3	3	3	2	3	**(0-7)µ/mol/L**
Cholesterol	3.9	3	4.1	3.6	4.2	4.7	**(2.2-5.5)mmol/L**
Creatine Kinase	208	244	95	228	185	194	**(64-400)IU/L**
Haemolysis Index	Nil	Nil	Nil	Nil	Mild haemolysis	Nil haemolysis	
Lipaemia Index	Nil	Nil	Nil	Mild lipaemia	Mild lipaemia	Mild lipaemia	
Total T4	15	35	28	37	31	30	**(10-60)nmol/L**

Pathologist's note: You will notice that there is an apparent non-regenerative anaemia in March; however, there is recovery of the anaemia by May. Reticulocyte counts do not always reflect likelihood of an adequate bone marrow response in cats.

Figure 1. More ultrasound images of kidney on presentation



Winner

Entitled to a CVE$100 voucher

Small

CONGENITAL OR JUVENILE-ONSET SECONDARY HYPOTHYROIDISM IN AN ALASKAN MALAMUTE

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History

Bear was rescued from a breeding facility (as is common in Hong Kong) with another individual Alaskan Malamute, believed to be a littermate, or a related individual of similar age.

It was immediately noted that compared to the companion dog, Bear was smaller in stature, with a persistent juvenile coat.

As well as these obvious physical features, Bear was rapidly found to have multiple additional health problems by his carer, including a very weak and stiff gait, apparent pain on multiple joints, pruritus, flaking discharge of the skin/coat, entropion, associated eye discharge, regurgitation, coughing, and selective appetite.

For these reasons, he was presented for veterinary assessment.

Clinical Exam

On clinical examination, Bear was noted to be short in stature and small for his stated age, which was estimated to be between 12 and 18 months. His mentation was dull, and he was immobile unless urged and assisted to stand and walk. His gait was stiff, and suggestive of lameness and pain of multiple appendicular sites. No gross neurological abnormalities were evident.

Integumental examination revealed a retained puppy coat, with soft/downy fur (lanugo), and a crumbly, malodorous yeasty discharge. There was severe bilateral entropion, resulting in chemosis, epiphora and blepharospasm.

Chest auscultation revealed increased adventitious sounds, crackles, and there was a mildly increased costo-abdominal respiratory effort.

Heart rate was 100 BPM, and body temperature was 37.2 °C.

Provisionally, a congenital or juvenile-onset

Figure 1. Bear in comparison to his littermate – Image courtesy Dr Wallis Chan-Benami

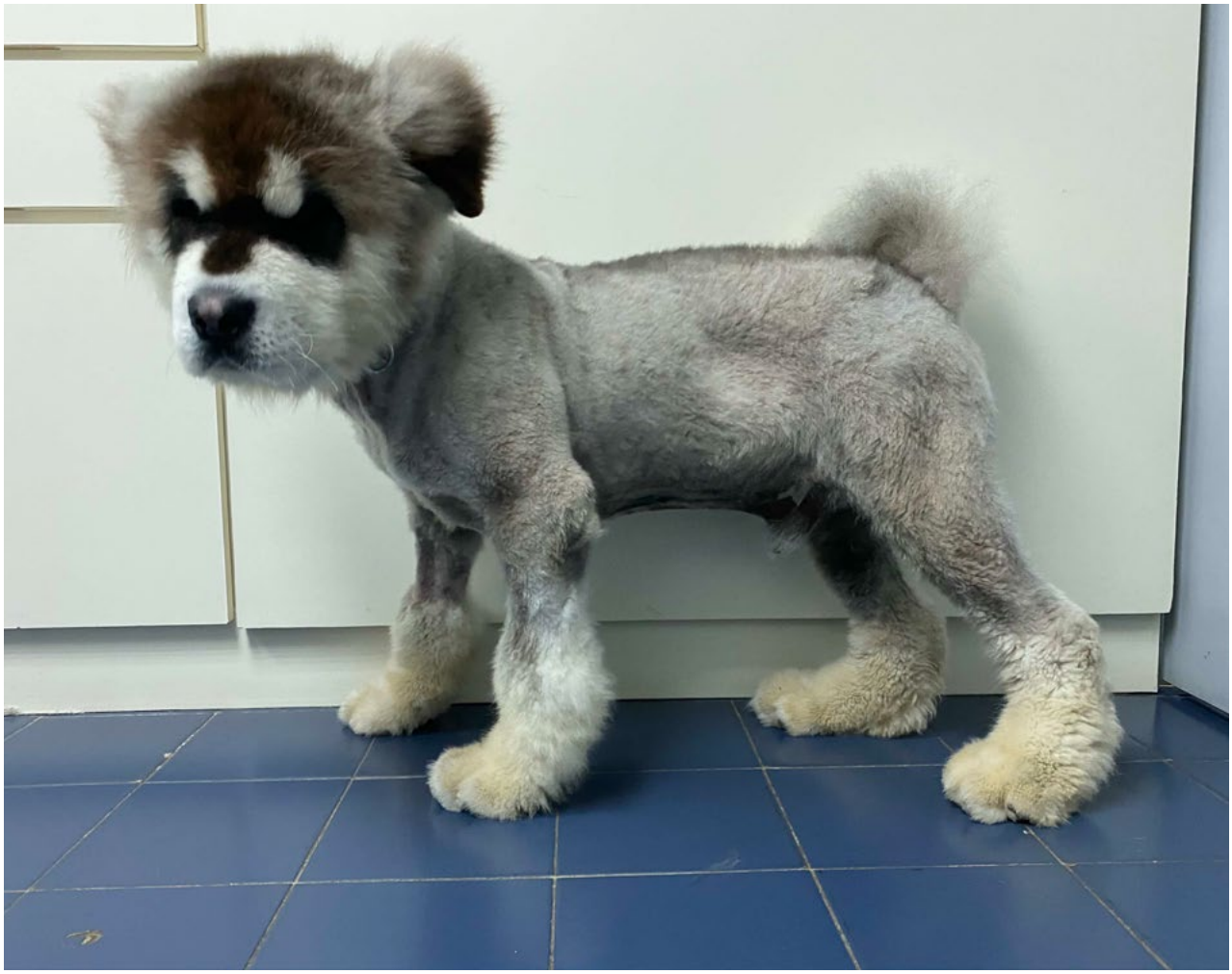

Figure 2. Bear – initial appearance after clipping away the lanugo – Image courtesy Dr Wallis Chan-Benami

endocrinopathy was suspected, potentially resulting in a dwarf phenotype, and appropriate diagnostics were performed to pursue this suspicion.

Test Results

In-house biochemistry revealed a mild BUN and mild cholesterol elevation. CBC revealed a mild non, (or pre)-regenerative anaemia. Urinalysis revealed a USG of 1.008, with inactive sediment and no other abnormalities.

In-house total T4 concentration was undetectably low.

Plain radiographs of his elbows revealed bilateral incomplete union of the anconeal process.

A whole-body CT scan was performed, which revealed multiple abnormalities:

1. Suspected delayed physeal closure of multiple/many axial and appendicular sites (based on dentition and given anamnesis)
2. Bilateral humeral OC/OCD
3. OC/OCD of the left humeral condyle (medial)
4. Bilateral incomplete ossification of the greater tubercle of the humerus and of the trochanter
5. Bilateral incomplete union of the anconeal process
6. Bilateral irregularity of the medial femoral epicondyle
7. Shortening of the short bones—particularly vertebrae
8. Incomplete closure of the T1 and L7 spinous processes
9. Mild deformation of the ribcage
10. Mild subcutaneous oedema (axillary and groin regions)
11. Bilateral mild shoulder and stifle effusions
12. Bilateral lung changes suggestive of aspiration or other pneumonia
13. Mild bilateral pleural effusion or pleural thickening
14. Possible megaoesophagus
15. Prominent axillary lymph nodes
16. Mild peritoneal effusion
17. Prominent jejunal and medial iliac lymph nodes

Test	Results	Reference Interval	LOW	NORMAL	HIGH
Catalyst One (April 6, 2021 10:58 AM)					
TT4	< 6 nmol/L	13-51	LOW	<	

Diagnostic Interpretation for TT4

< 13	nmol/L	Low
13-26	nmol/L	Low Normal
13-51	nmol/L	Normal
> 51	nmol/L	High
27-69	nmol/L	Therapeutic

Dogs with no clinical signs of hypothyroidism and results within the normal reference range are likely euthyroid. Dogs with low T4 concentrations may be hypothyroid or 'euthyroid sick'. Occasionally, hypothhyroid dogs can have T4 concentrations that are low normal. Dogs with clinical signs of hypothyroidism and low or low normal T4 concentrations may be evaluated further by submission of free T4 (fT4) and canine TSH. A high T4 concentration in a clinically normal dog is likely variation of normal; however, elevations may occur secondary to thyroid auto antiboides or rarely thyroid neoplasia. For dogs on thyroid supplement, acceptable 4-6 hour pot pill total T4 concentrations generally fall within the higher end or slightly above the reference range.

Figure 3. In house T4 result

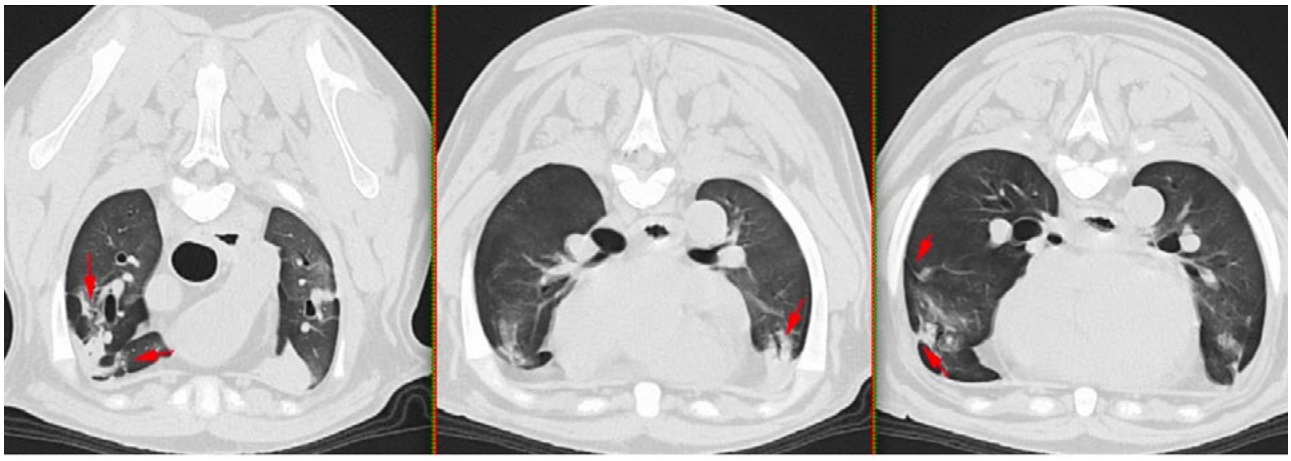

Figure 5. Lung CT, showing areas of suspected aspiration or other pneumonia. Image courtesy Dr Wallis Chan-Benami

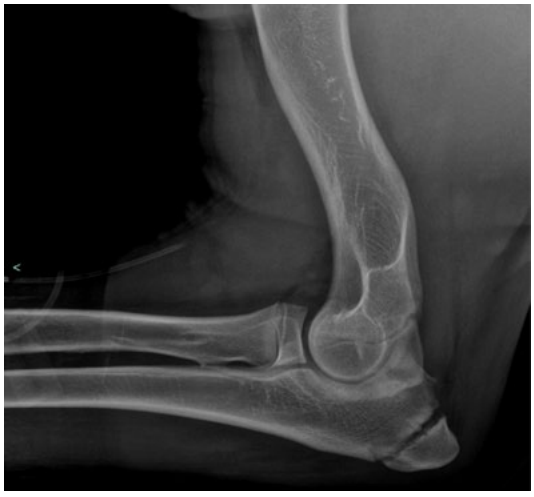

Figure 4. Lateral radiograph of the elbow

Differential Diagnoses

Based on a phenotype suggestive of delayed growth and disproportionate dwarfism, a congenital or juvenile-onset endocrinopathy was suspected.

The two main differential diagnoses under consideration were congenital hypothyroidism or pituitary dwarfism (growth hormone deficiency), though other differentials considered were storage disease such as mucopolysaccharidosis and GM1-gangliosidosis, which has been reported in the Alaskan malamute breed.

To attempt to reach a definitive diagnosis, serum was sent to DCPAH at Michigan State University for an expanded thyroid hormone panel + insulin-like growth factor 1 assay.

Based on a strong clinical suspicion of hypothyroidism, combined with the low in-house total T4 concentration, treatment with supplemental L-thyroxine was commenced pending the endocrine panel results.

In addition, joint pain was managed with oral meloxicam, and suspected aspiration pneumonia was treated with amoxycillin/clavulanic acid and ofloxacin.

Repeat chest radiographs taken one week after L-thyroxine was commenced showed no evidence

of a megaoesophagus, and apparent marked improvement or resolution of the pneumonia.

Diagnosis

The results were consistent with secondary (pituitary-dependent) hypothyroidism, and not supportive of pituitary dwarfism (growth hormone deficiency).

Treatment

Treatment with L-thyroxine was continued. Joint pain was managed with oral meloxicam, rest, and physiotherapy. This regimen resulted in a significant clinical improvement, and when sufficiently stable, entropion surgery was performed under general anesthesia.

Results

After one month of L-thyroxine supplementation, a repeat in-house total T4 concentration was 36 nmol/L (10-50)

Repeat elbow radiographs demonstrated a marked degree of closure of the anconeal physeal growth plates.

Bear's overall clinical demeanor brightened, his ambulation improved, his eyes were markedly improved after entropion surgery, and his appetite normalised, with no signs of dysphagia, regurgitation, or coughing. At the time of writing there is minimal or modest improvement in coat quality, without current development of a normal adult coat.

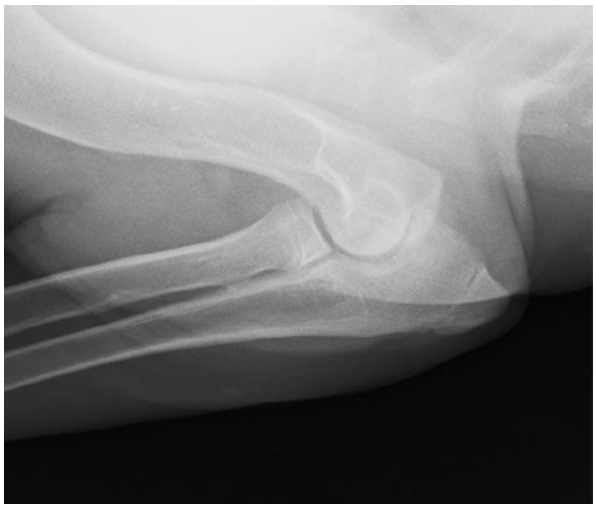

Figure 8. Lateral elbow radiograph showing fusion of the anconeal process

Discussion

Bear's apparently stunted growth and appearance, especially when compared to his littermate, prompted consideration of a congenital or juvenile-onset endocrinopathy in this case.

Figure 9. Bear after replacement thyroid therapy

Differentials included congenital or juvenile-onset hypothyroidism, pituitary dwarfism, and storage disease such as mucopolysaccharidosis and GM1-gangliosidosis, which has been reported in the Alaskan Malamute breed.

The results of the endocrine panel were supportive of secondary (pituitary-dependent) hypothyroidism.

Hypothyroidism was a good explanation for multiple clinical abnormalities encountered in this case, including the disproportionate dwarf phenotype, persistent puppy coat, delayed physeal closure, mental dullness, hypothermia, megaoesophagus, and associated suspected aspiration pneumonia.

The test results suggested secondary hypothyroidism because TSH levels were inappropriately low given very low total T4, total T3, and free T4 concentrations.

It can be very difficult to distinguish congenital from juvenile onset hypothyroidism. Congenital hypothyroidism is reported in dogs and cats and may be associated with a wide range of defects involving various points along the hypothalamic-pituitary-thyroid (HPT) axis.

(Bojanić *et al, New Zealand Veterinary Journal* 2011 – Congenital hypothyroidism of dogs and cats – A review).

Treatment with L-thyroxine is warranted regardless, so the exact localisation of the defect is neither possible nor necessary in many cases.

Prognosis in cases of congenital or juvenile-onset hypothyroidism is variable, and largely depends on whether the diagnosis is made in a timely fashion, particularly prior to closure of the physes. Delayed diagnosis is likely to result in permanent and irreversible skeletal deformities, which may be refractory to subsequent management. In addition, persistent megaoesophagus increases the risk of serial aspiration events. Finally, hypothyroidism is associated with renal insufficiency, which may become intractable if intervention is delayed.

The other main differential diagnosis in this case was pituitary dwarfism. Pituitary dwarfism is the name given to the syndrome of insufficiency of multiple hormones of the anterior pituitary gland, including growth hormone (GH), and thyroid-stimulating hormone (TSH).

Pituitary dwarfism is most reported in the German Shepherd Breed, where it is a well-defined entity with a known genetic basis.

(Voorbij & Kooistra *Journal of Veterinary Clinical Science* 2009 – Pituitary dwarfism in German shepherd dogs, Voorbij et al PLOS Onee 2011 – A Contracted DNA Repeat in LHX3 Intron 5 Is Associated with Aberrant Splicing and Pituitary Dwarfism in German Shepherd Dogs)

Results in this case were not supportive of pituitary dwarfism.

Insulin-like growth factor 1 (IGF-1) is mostly measured in the diagnosis of dwarfism, due to the pulsatile secretion of GH, and its physical instability, making it unsuitable for shipping from Hong Kong to the US for testing.

In Bear's case, a diagnosis of hypothyroidism was considered preferable to pituitary dwarfism, as treatment for the latter condition is much more challenging and expensive. Growth hormone replacement therapy is used to treat pituitary dwarfism. Canine GH is not available, and the administration of human GH is associated with formation of antibodies against it. Porcine GH is identical to canine GH, so is the treatment of choice, but is very expensive and not widely available.

L-thyroxine therapy is widely established in dogs, is safe, relatively inexpensive, and provisionally highly effective in Bear's case.

Endocrine Results

Collected Date/Time (If Provided)	05/03/2021 10:20:00	04/06/2021 8:12:00		
Procedure			Ref Range	Units
Insulin-like Growth Factor 1 (RIA)		57	[4-95]	nmol/L
Total Thyroxine (TT4) (CLIA)		3 L	[9-45]	nmol/L
Total Triiodothyronine (TT3) (RIA)		0.3 L	[0.8-2.1]	nmol/L
Free T4 by dialysis (RIA)		2 L	[6-42]	pmol/L
T4 Autoantibody (RIA)		14	[0-20]	%
T3 Autoantibody (RIA)		6	[0-10]	%
Thyroid Stimulating Hormone (CLIA)		0.01	[0.00-0.58]	ng/mL
Thyroglobulin Autoantibody (ELISA) *		6	[0-35]	%

Figure 7. Endocrine panel from Michigan

Small

WHY HAVE TICK PARALYSIS CASE NUMBERS DROPPED SO MUCH IN SOME AREAS?

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Ixodes holocyclus (Image courtesy of Anne Quain)

In recent tick seasons (especially in October of 2019 and 2020), there have been very few cases of Tick Paralysis (TP) in dogs, or in all species in general.

Usually a busy weekend (late Friday to early Monday) would see up to about 20 cases (mainly dogs at our reference research hospital at Manly Road) in various stages of toxicity/paralysis, with several on O_2 support or ventilators. The information below outlines some possible causes of this apparent change in the epidemiology of this disease, caused by *Ixodes holocyclus*, in the Manly Road Veterinary Hospital client draw area, when compared to, say, 2010 case numbers data (Case severity during the 2010 tick season, *C&T*, University of Sydney).

Owners

Tick Paralysis treatment is understandably expensive, and preventives are effective (to a large extent; even beyond their claim times). Provided they are used correctly, fewer cases should be seen. The compliance rate you would expect to be increasing as animals are increasingly more important in households (especially with Covid-19)—puppies are now 'babies' and pet decisions are a major family issue. This 'humanisation' of pets could be the incentive to the increasing use of tick prevention topicals/tablets, etc on monthly or longer dosing timetables. So, people are more attached and are probably more likely to protect their pets! (Also, accurate owner compliance has increased—seen in two large tick trials ten years apart: n > 1,000 dogs).

Homes

Most homes do not have any natural bush, usually have 'ground to high' fences, and bandicoot presence is dropping—cars and cats, e.g., having a major effect on these 'pass-by' travellers. Bandicoots need a certain close-by bush acreage to be viable, and need easy access to yards if they are to help maintain any local tick presence. So, the places where pets are mostly (yard and home) are now less likely, in general, to have bandicoot passage, as suburbs become so urbanised and congested, with smaller blocks and less accessible native vegetation in general.

Dogs

Dogs are usually leashed (Council by-laws) when they go out and people tend to walk on cement paths or on a well-defined journey, devoid of bush contact. Dogs don't regularly escape and wander through any local cool, moist creek beds. They instead are controlled (no wandering off—straying) on defined pathways into (less-natural) dog, off-lead, council areas and back again to an enclosed home yard with less and less chance of natural tick exposure—i.e., to tick micro-environments. Such enclosed, dog-friendly areas do not (usually) have the vegetation conducive to tick life cycles—more mowed parkland style park areas with scattered trees and grasses.

Development

As existing green acreage areas are split up for density housing, less TP cases are seen locally and as 'further-out' acreage areas are usually very developed, fewer people and animals in general are likely to have tick access. But the numbers involved tell the story—generally one large acreage in a TP suburb, e.g. The Gap, Brisbane, becomes cut up—changing yard sizes—lessening one original large area as a likely source but gaining many unlikely sources—multiple small, restricted backyards. Even the previous open range experiences for dogs are less likely in these higher density suburbs, whereas the new acreage areas are further out and involve far fewer people overall usually with horse paddocks and cleared mini-farms—so more and more pets are less and less likely to be exposed to

the original traditional areas where ticks are more likely to be. There are also now less 'cow/horse' agistment paddocks (replaced by density housing) that used to be publicly accessible, and which still had native vegetation.

Practices

The dilutional effect of new practices in the draw area of the reference hospital may be a reason. However, there are many more housing developments in the area, e.g., taking over old horse stable agistment paddocks, market gardens, etc. Both staff and client numbers have increased in the reference practice. (Theoretically, owners have less distance to travel now should their pet be a TP case).

Carriers & Tick Cycles

Bandicoot access is now limited as, historically, the main dispersal agent of egg-laying female ticks. Hard ground (e.g., concrete edging) surfaces and high fences have ensured no easy inter-house access for the bandicoot's foraging travel. (In fact, such fencing limits social interaction, for both people and pets, reducing the chance of more supportive, friendly streets/suburbs).

Client Cost Factors

With the increasing standards of care (and associated costs), clients are aware of the costs of not using prophylactics, compared to the cost of treating a severe case of TP. So protective use may have increased, both due to the increasing importance of (more humanised) pets and to the cost of care for a TP case, compared to the relatively inexpensive use of very effective, longer-term products.

People are also more aware of the real cost of modern animal care, (as opposed to the catch-cry of the 'cost of vets') whereas the real cost of human medicine is seldom apparent or even discussed. Look at the number of veterinary programs on the general access TV channels; they are helping people to see the real and high cost of appropriate care, without tax levies and the extensive health insurance cover available for people. (In fact, there is a real ignorance of medical costs—a small survey showed that medicos did not know the cost of routine PET scans nor that those costs are mostly non-refundable).

Local Weather Effects

As 2019 was the hottest and driest year to date, Sep/Oct 2020 was also hotter and drier than, say, 10 years ago when September to November TP cases peaked in October with about the same TP numbers for each of these three months. In fact, September 2020 was the hottest ever recorded in Brisbane. Ticks can't afford to stay exposed to such heat—they are very susceptible to dehydration and so do not quest (search for hosts), but rather stay in moister, inaccessible microclimate, micro-niche areas. Rainfall had been delayed and local suburbs (lawns and yards) were affected. Humidity, too, was lower. So, less moisture, hotter and (as a result) drier—thus tick exposure is far less likely because of local 'day by day' weather changes in the local climate, i.e., creeks are dry, soil temperatures are high. Paradoxically, if rainfall is extensive over many days, ticks become susceptible to lethal fungal infections, further reducing their likelihood of animal exposure. Additionally, nights are relatively cool, e.g., early November 2020; these night temperatures may also have had an effect on tick activity—i.e., still cool at night and very hot spells during the day? (The 'kangaroo ear tick' will not quest for hosts when night temperature is below 12°C).

Tick Behaviour

Due to the effect of local micro-weather (on tick behaviour), ticks probably stay in the most protected areas they can find to avoid dehydration, etc. (their only 'water' supply is their own recycled saliva). Once more favourable conditions commence (e.g., warm and wet), they begin to quest, seeking, for example, the tips of grasses and the outer lower edges on bushes, where they have easier access to hosts: waiting and hopefully avoiding exposure to unfavourable conditions. This approach would maximise both tick survival and host exposure, while minimising the dangers of excessive environmental effects. Recall they can't see and rely on infrared light detection, CO_2 levels and vibration to find a host.

Predictions

Based on the local tick season (2020) of very few TP cases, it is likely that fewer eggs will be deposited and so the probability of fewer TP cases in 2021-2022 (provided the practice client catchment area is similar for each year). (A pilot study of 'kangaroo tick' infestations at Augathella in 38 animals showed no ticks in their 'ear canals'—normally they can be heavily infested, with engorging ticks, seen easily about the ears).

Record series of high daily 2020 temperatures may have a bearing on tick cases in 2021. Similarly, the levels of the Colorado river (USA) can be predicted out two years by looking at existing weather patterns—perhaps our tick seasons will be more predictable if we source last year's (e.g., 2020) local (to practice) climate data as a predictor for 2021 tick case numbers (these being related to tick-egg-life cycle issues from 2020).

Reverse but Supportive Data

Observations over 60 years (*G. Greenup BVSc, Pers Comm* 2021) at a property near Stanthorpe (NSW/Qld border) have revealed a micro example of changing biology. There has never been bandicoots or TP cases up until last year—where cattle died from TP and local sheep dogs developed classical TP signs. Bandicoots are now seen routinely in this controlled, closely observed space—a reflection of adaptive arrivals and ticks moving successfully into a more friendly climate-altered environment.

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SEPTEMBER 2014

HOW MUCH DO YOU KNOW ABOUT HUMAN TICK-RELATED DISEASES?

For example, have you heard of red meat allergy? if not, then this information will prove useful for your clients and you

Sheryl van Nunen Clinical Associate Professor and Senior Staff Specialist, Department of Clinical Immunology and Allergy, Royal North Shore Hospital & Paul Canfield Professor Emeritus, Faculty of Veterinary Science, University of Sydney

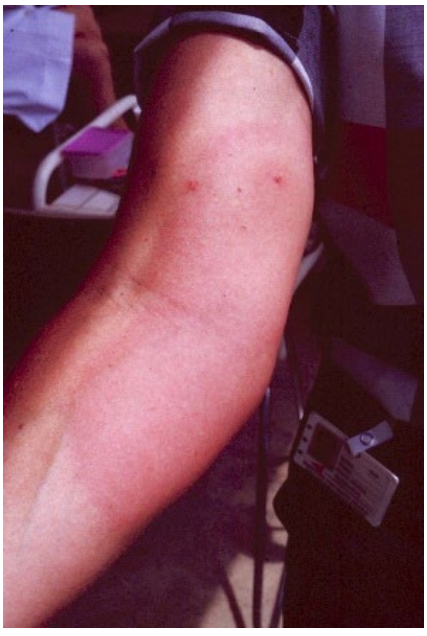

Figure 1. A very large local reaction to a tick bite on the inner aspect of the upper arm.

Small

WHY DO CATS STILL BAFFLE US?

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Why do people keep pets?

Pet keeping in general is a bit baffling. Philosophers, ethicists, zoologists, anthropologists all wonder why people keep pets at all. But they do, even in poor farming and hunter-gatherer societies. Apparently, humans have an over-active nurturing gene along with our empathy gene. Interestingly, there are numerous recorded cases of individuals of other species raising an orphan of a completely different species, and there are many interspecies friends on the internet. If you are tired of watching cats on the internet, have a look at those.

Source: Times of Israel
timesofisrael.com/loveless-monkey-adopts-chicken-at-israeli-zoo/

Cats have hung around with us for 9,500 years or more, and they have not changed much up until the last 50 years. The Egyptians worshipped cats and pretty much just let them be cats. Only after 1947, with Ed Lowe's invention of cat litter, did large numbers of cats come in from the cold. Still, it took until the 1990's for research to move to studying the behaviour and social systems of the house cat. Until then, we knew more about the lions of the Serengeti and farm cat colonies than our fluffy

Source: Pinterest
pinterest.ca/pin/21251429461419465/

feline companions. In many places in the world, *Felis catus* still is considered an outdoor or rural pet, and therefore their behaviours do not come under owner scrutiny.

Why is it so hard to herd cats?

Cats have been intensely researched for brain structure, function, and neurophysiology, partly because they are cheaper than dogs or primates to keep. Additionally, many of the concepts of behaviour and learning were studied specifically with cats. So theoretically, we know EVERYTHING about how their brain works and their bodies react.

While comparisons are odious, we still make them, and cats have come off second best when their behaviour patterns are compared with dogs, because people think they understand dogs. Research in dog cognition shows that dogs certainly know people! Your cat, on the other hand, already thinks you know what they are thinking, so your feline is a bit baffled when you do not do what THEY want.

I can think of three reasons why cats might baffle cat owners, vets, dog people and cat haters:

1. People think they are the centre of the universe, when clearly cats are.
2. Cats do not do as they are told; it is easy to forget that felines are not domesticated (yet). They lack the Williams Syndrome/ Domestication mutations, have personalities and grow into independent adults who might even think people are irrelevant.
3. Cats are quick at learning, and particularly learning to MANipulate their owners.

Cats are the centre of their universe

They do not have enough brain power to develop the concept of 'other'. And Schroedinger himself, with his conceptual cat—being alive and dead at the same time—believed that there is only one consciousness (a very religious concept from a very scientific mind).

I like the idea of one consciousness that, in cats, means they think you know what they are thinking, because you are not 'other'. In the same way, toddlers do not lie until about 2 years old because they, too, think you already know what they are thinking. We know all humans tell fibs and lies, and we have taught horses and dogs to lie—but that is another story. Cats never learn to lie, as they never figure out that we do not know what is going on in their brains.

Editor's Note: I disagree. I know cats who pretend they have been fed when they ask food from the 2nd carer!

Reply from Kim: HAH! However, they are still 'honest signalling' if only for attention. Lying in this context (just have to be quick with your reactions :)) means foreshortening signals and suppressing communiCATion. Cats ALWAYS tell you what they are going to do.

Skilful and careful observation of cats will give you, the human, some clues, although we can never be in a cat's skin, as it were, because we lack the strong sense of smell, the vibrissae, and whiskers to connect to vibrations and movement, and ultrasonic hearing to name a few cat features. Ignoring the clues, though, puts you in peril of receiving more potent messages—pee-mail is just a message, do not take it personally. Some cats will even shred themselves to try to cope with their environment. These cats, in a recent article, responded only to a qualitative improvement in their living environment. In particular there is a very interesting QOL chart they have developed in the article, read here: frontiersin.org/articles/10.3389/fvets.2018.00081/full

Paradoxically, B. Skinner's levers and Thorndike's cat-in-a-box have left scientists and people in general with a more mechanistic view of cat behaviour. And some scientists question whether non-primates even have emotions, or indeed feelings. We've not come far from the vivisectionist who, back in the 1800's, stated 'the crying of a dog (under the knife) is no more anime than the squeaking of a door'.

Being tamed is not the same as being domesticated

Another part of the answer to why cats seem to baffle us, is that the other companion animals we

have—dogs, rabbits, ferrets, pet rats and mice, and also donkeys, pigs, horses, and goats, will all look towards humans—although only dogs 'ask' for help. All these species have been domesticated, not just tamed. In silver foxes, the evolution from the very wild anti-human fox to the rather corgi-like domesticated fox that seeks human attention, took less than 50 years of directed selection by the Russian researchers.

Peter G. Fisher writing in ***Exotic Pet Behavior***. 2006 : 163–205. Published online 2009 May 15. doi: 10.1016/B978-1-4160-0009-9.50011-6 has described the process cogently for ferrets:

'It must be emphasized that the behavior of domesticated animals in captivity differs from that of tamed wild animals and that these behavioural differences have arisen as a result of selection by humans. The exact form taken by [deliberate] selection will, however, depend on the role of the particular domesticated animal in relation to people. Behaviourally there are major differences between domestic ferrets and the ancestral polecat. Polecats tend to be solitary and very territorial with fighting between males having been observed, presumably over territory and sexual domain. The domestic ferret on the other hand is very social and gregarious, enjoying play activities with conspecifics and preferring to sleep with other ferrets of the same or opposite sex.'

So, once *Felis catus* is comprehensively domesticated, we will probably end up with a bigger, fluffier ferret. The primacy of the sense of smell is also worth noting. Cats do not see us as much as smell us—all the odorants of perfume, sweat, pregnancy and fear. Their noses are tuned for checking pheromones—a major form of communication for them. Again, another story.

So, is the current cat domesticated? Not so much. Felines start life as sociable kittens—they need food and warmth from someone else—and then they grow into independent adults with variable attitudes toward getting close to people and other species. Every cat is representative, but no cat is average. Associating with humans, but not having their breeding regulated, has given us pets who have variations in temperament from the hyper-social (bold cat) to very shy (or timid) ones, who can nevertheless tolerate the pressure of close association with people. Truly wild cats or feral cats do not want anything to do with people. It also means that every cat born is a hunter at heart (though some of the highly bred ones like doll-face Persians will only be able to hunt out the next person to feed them). Every cat born is equipped to hunt and survive on its own, and therein lies a significant source of bafflement. Humans are tuned to nurture, feed and protect their pets, yet here is one who does not need them. OUCH? A blow to the psyche? In the food chain, cats are part prey, part predator and mostly pessimistic, and they swapped part of their brain—in the prorean gyrus—away from executive function (think before you act) for athleticism. After all, the archetypical, historical cat, has to react quickly either to catch prey or get away from a predator. No time to stop and think. Good swap, as it has allowed *Felis catus* to proliferate in all parts of the world, even competing and interbreeding successfully with the native cats.

Image https://edwardlowe.org/the-1940s-2/

Not just a pretty face

Individual cats do have personalities—as do pretty much any species that moves—vertebrates like us (mammals), birds, fish, and reptiles, through to cephalopod molluscs like octopus and cuttle fish. Those personalities have genetic and environmental forces on them, and individuals learn different behaviours that help them survive in their particular environment. Cat enthusiasts are breeding cats for looks and temperament—in my experience we have the bold Burmese who head butts everything including cars and dogs—to the more aloof Russian Blue and then the hyper-bonding Rag doll. Each breed has a set of physical and temperament parameters that selective breeding is stabilising in the population, which makes them a reliable choice of pet for people so inclined to live with a cat with those particular traits. In fact, there is genetic evidence that the highly selected breeds like Abyssinians and Birmans, have the modified neural crest-related genes associated with behaviour and reward, as predicted by the domestication syndrome hypothesis. These breeds could therefore be considered domestiCATed, although the hunting mind-set has never been bred out. And then there is the learned component.

How does this work? Cats learn fast. But they are hard to train. Their abilities to make connections between separate events has been studied intensely, but we cannot quite believe it in our pets. Cats watch us very closely and are themselves excellent trainers or MANipulators. The purr for yes, the claws for no. The dialect that each cat learns to use for their owner. It has been shown that your cat makes specific sounds, in an individual conversation that your cat has and is just for you. Owners can recognise what their cat's vocal command is, but not what another cat's cry means. I am tone deaf, so my cat gets a bit frustrated that I do not get what they want right away! However, training a cat is tricky because only 1 in 3 will respond to food rewards, so teaching them to differentiate between A and B so you can 'ask' them questions becomes problematic. It also means that cat cognition is poorly represented in the scientific literature because it is so difficult to get a consistent picture of a cat's preferences when it just stops doing anything rather than work for food as a reward for a choice. Only recently was it even confirmed (electrophysiologically) that cats do have colour vision—previous research showed only that they just do not care enough to demonstrate it in preference tests. How Cat!

Note I have not yet talked about the significance of socialisation or habituation on cat personality. An unfortunate trend has emerged that has corrupted the concept of socialisation in cats. Socialisation only means humans and other species have to be in a kitten's environment, preferably in a friendly and supportive manner, for 20 - 40 minutes a day between the ages of 2 – 7-weeks-old to teach them that non-felines can be OK. That has morphed into taking them from their mother, sometimes even isolating them from other kittens, at 2 – 7-weeks-old so they can bond more strongly with humans. This CANNOT make an emotionally stable adult cat. The research was conclusively done in 1959. Get over it—kittens need their mother (or another friendly feline who will lick the kittens' perineal regions) to get their brains hard wired correctly. Even raising 2 kittens together from that age only partially compensates. In the alternative, if there is no human contact for kittens between 2 and 7 weeks, then humans become either irrelevant or frightening. It is a big reason the kittens brought in from the streets avoid people (and disappoint their owners). They run from people not because they have been badly treated, but because they did not have positive interactions while in the safe care of their mother during that time. As prey animals, running and hiding is a good option—and that becomes the embedded reaction. Again, much to the disappointment of their owners. A cat that runs from visitors is actually the normal cat. The one who head butts everything and everyone is at the extreme end of the boldness spectrum. The cat who hides from all people including the owner, and never relaxes in their presence is the shy / timid / unsocialised (to people) one and is not having a good time as a pet. I have seen 2 kittens who were removed to their new, totally indoor, home at 8-10 weeks change from being the frighteningly fractious cat to become an engaging and co-operative feline companion. However, it took 6 to 8 years, and included regular stays in good boarding facilities to convince them that there are other humans worth snuggling. Both cats were bold temperaments to start with, so they were prepared to risk some interactions with non-owners to see what they could get out of them. All this becomes somewhat moot if the poorly socialised cat gets to roam about outdoors, where they have choices, take risks, and are usually dead by 3 years old.

In conclusion, I re-present the case that cats are the centre of the universe and they command us at their will. Orcas and cats are excellent trainers of people. Humans can browbeat dogs and horses into doing what is wanted and accept ferrets for what they are and do. Orcas, elephants and all manner of 'wild things' will perform for food rewards. But cats—not so much. Unless you have trained the sociable, behaviourally flexible kitten, or own the 1 in 3 who like to perform for food rewards, you will probably only succeed in training your cat to do what it wants when you want it to. I maintain most owners (including myself) are not adept enough to train a cat or even a kitten. Our timing is off and our patience and purr-sistence is poor. If you have the right cat and take the time, you can teach a cat like Gus—he won his inaugural swim in the Dog Race in 2019. Read about him here: theguardian.com/australia-news/2019/dec/23/glenn-druerys-toughest-race-how-the-preference-whisperer-set-the-cat-among-the-dogs

If you do put in the time and have the observational capacity, you can be one of the exceptional trainers like Catmantoo—he has convinced Didja to do some spectacular things—but it is a looong process.

youtube.com/watch?v=YRbakPKgU5Y

Kim is well-known to C&T readers, being a long-term CVE member and generous contributor and supporter of the C&T Series.
Members are reminded that there is a wealth of information in the CVeLibrary. If you'd like to read other others written by Kim, visit: cve.edu.au and search by author.
A sample below:

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Lower respiratory tract disease in cats

Small

OSTEOSARCOMA IN A YOUNG CAT

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C&T No. 5923

An 18-month-old MN DSH cat presented for left hind limb lameness. He had a palpable distal femoral swelling. Radiographs confirmed a lesion in the distal femur (see Figure 1.) and needle biopsies were taken. We were concerned about osteomyelitis/ fungal infection/ non-union fracture or osteosarcoma.

The needle biopsies were non-diagnostic and he was started on NSAIDs, antibiotics, and a plan to re-evaluate in a few days.

Three weeks later after missing his re-evaluation appointment, he was presented with non-weight bearing lameness with a leg swollen from suspected pathological fracture and a large haematoma resulting in PCV of 12%.

His owner was overseas, so it took a day or two to get consent for amputation, but one week post-surgery the cat is going very well.

Histopathology was performed on the bone lesion and an osteosarcoma was diagnosed.

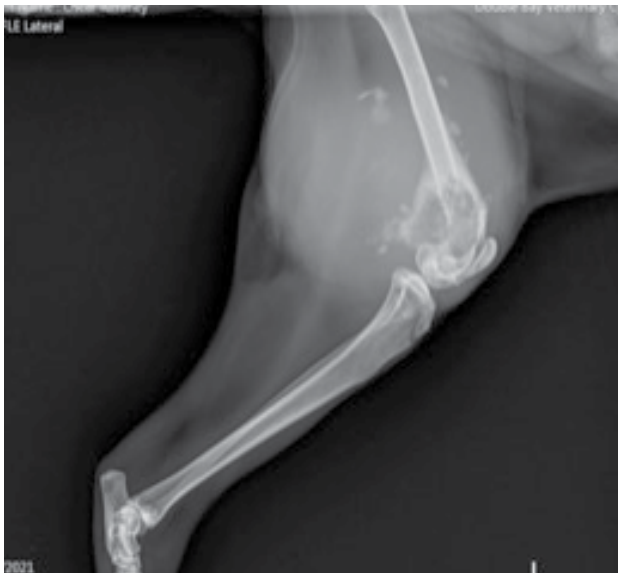

Figure 1.

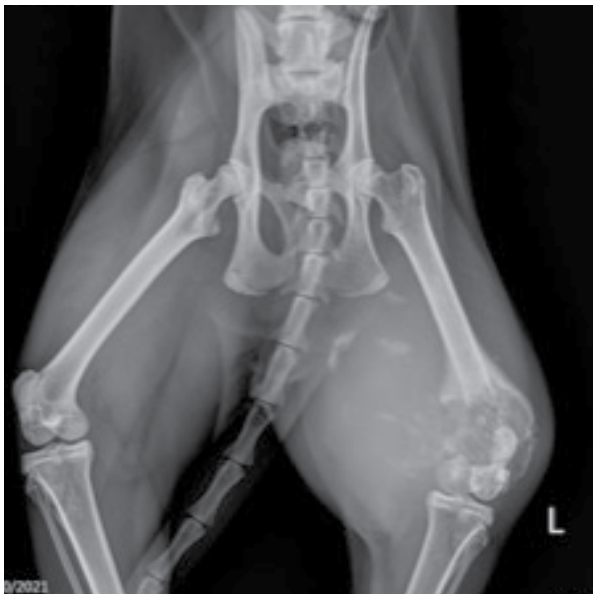

Figure 2.

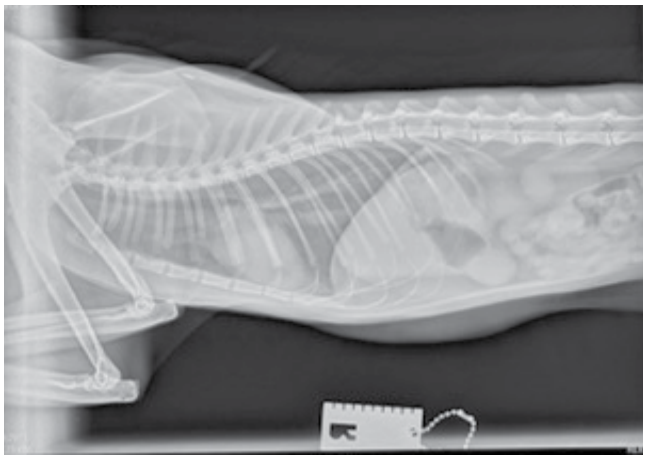

Figure 3.

COMMENTS COURTESY OF

David Taylor
Veterinary Pathologist
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Osteosarcoma accounts for approximately 70% of the malignant bone tumours in cats and the disease is much less common than in dogs. Cats appear to develop osteosarcoma at an older average age than dogs, but as in dogs, the age range is broad. The reported mean age of cats diagnosed with appendicular and axial osteosarcoma was 8 years and 10 years respectively in a retrospective study. No sex or breed differences were observed.
The clinical course of osteosarcoma in cats is slower than in dogs, especially those involving the appendicular skeleton. A median survival time of 49.2 months was reported in 12 cats with appendicular osteosarcoma treated by amputation. Four of the 12 cats survived for 5 years or more after diagnosis. Cats with axial osteosarcoma had a much shorter median survival time (5.5 months), presumably reflecting the difficulty of complete surgical removal of tumours in axial sites. The incidence of metastasis in cats with osteosarcoma is considerably less than in dogs and the prognosis appears to be slightly more favourable.

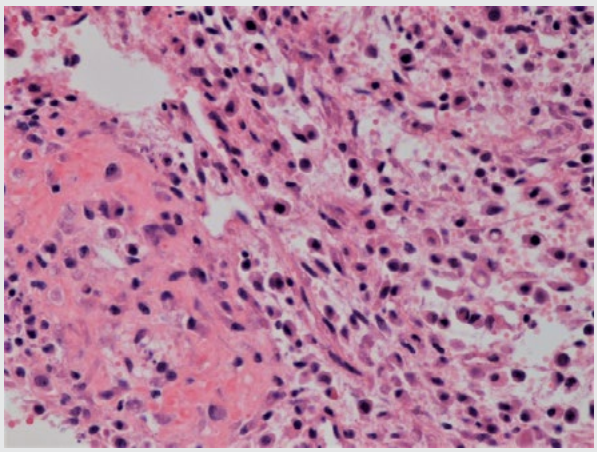

Figure 4. Neoplastic spindle and round cells producing osteoid (left)

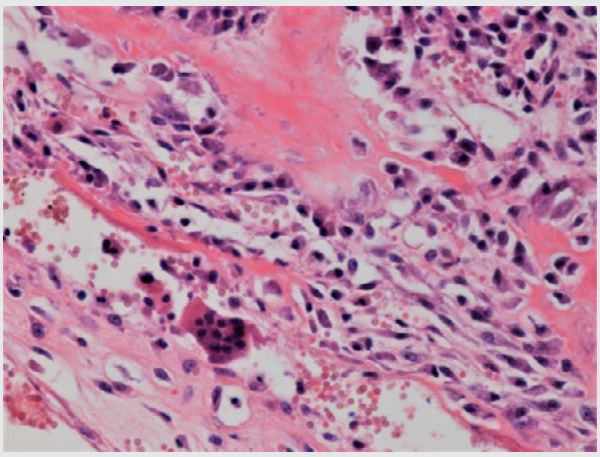

Figure 5. Neoplastic spindle and round cells producing osteoid (top and right)

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ALPACA ABSCESS GRANDE

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C&T No. 5924

I was in Year 9 at Armidale High School, when I went to a property at the end of the school bus run to babysit some kids. The local vet was preg-testing horses. We talked as he worked, and when he drove off I knew what I wanted to do with my life. I chewed it over for two days, and announced to the family at Sunday lunch that I was going to be a vet. Out of the blue. 'Why a vet?' asked dad. 'It's better than being a horse', I replied. The family resumed their meal.

I enrolled at the University of Sydney in 1978, and graduated in 1986, after repeating second year and working/travelling for two years. Graduated with First Class Passes.

I returned to my home-town in 1989 and purchased a single vet mixed practice. I am still here. Best thing I ever did, becoming a vet in my home-town. Out in the winter sun on a Sunday morning doing a caesarian on a cow, stopping to gaze at the Milky Way in the mountain sky on the way home from a long colic, or having a dog jump up for a pat when it was all but dead the week before from snakebite.

Best job on the planet.

Several months ago, I examined a 2-year-old female alpaca, which I estimated to weigh about 75 kg. The presenting complaint was a pendulous abdomen. On examination, the findings were a low body condition score (2/5) and a large abdominal mass.

There was no ascites or anaemia present. An attempted aspirate of the mass with an 18-gauge needle produced very thick pus, too thick to travel down the needle. I diagnosed an intra-abdominal abscess approximately 30 cm in diameter. As much as I hate to admit it, I was floundering a bit.

The owners did not want to refer it. Then the owner suggested the possibility of marsupialising the abscess. I gave them a 20% chance of success, as I had not been able to explore the abdomen for other smaller abscesses, etc., and I was doing this for the first time! Blood and urine examination was unremarkable. An injection of alamycin was given 5 hours pre-op.

The alpaca was anaesthetised on 11 March 2020 with 200mg ketamine and 25 mg Xylazine IV, then maintained on a Stinger anaesthetic machine with isoflurane. Until the abscess had been drained to some extent, the alpaca was unable to satisfactory oxygenate without positive pressure ventilation. This would have been mostly due to the mass in the abdomen compressing the diaphragm, exacerbated by the small size of the ET tube, which had something to do with my intubation skills.

A 15 cm incision was made through the ventral midline just caudal to the umbilicus. The abscess was not difficult to find. I was able to get my arm in past the abscess with difficulty, and I then palpated

Figure 2. The alpaca during anaesthetic recovery

the liver, kidneys, etc. and satisfied myself there was no reason not to proceed. I then sutured the wall of the abscess to the linea alba with a simple continuous row of No 2 PDS on either side, being careful not to penetrate the full thickness of the wall. (I think this layer may have been a waste of time, put it down to excessive caution—not something I am famous for).

I then sutured the skin to the wall of the abscess with a simple continuous row of 30lb fishing line, then made a 12 cm incision into the abscess. At this point, I loosened ropes and rolled the alpaca onto the right side. In retrospect, the left side would have been safer as it would not have squashed the oesophagus as much, but I had not foreseen the problem. Spontaneous respiration was allowed to resume, and anaesthetic parameters remained satisfactory throughout.

Quite a bit of pus had already flowed onto the ground when we decided to catch and weigh what we could. What we collected weighed 12.5 kg, and I reckon we lost about 1.5 kg. A sphere of pus 30 cm in diameter would weigh 14 kg, and the wall was at this point about 1cm thick. The abscess was flushed out for several minutes with a garden hose and tank water, until the body temperature had dropped by about 2.5°C. The alpaca was placed in the sun to recover, where it lay for 15 minutes before sitting in sternal recumbency.

Figure 1. The alpaca after drainage of the pus

The abscess was flushed with the hose daily for 8 days, by which time the wound was too small to allow entry for the hose, A second injection of Alamycin was given on Day 3.

On 20 October 2020 the alpaca was again anaesthetised, and the expected hernia was closed. The wall of the now 'extinct' abscess was removed from the linea alba. The abscess was now a piece of fibrous tissue 10 cm in diameter, and attempted aspiration with a 16-gauge needle did not produce any pus. The linea alba was closed with a simple continuous layer of 60lb monofilament nylon fishing line, and the skin was closed separately.

On 29 April 2021, I checked the alpaca for the last time. She had a 5-day-old cria at foot and was up for sale. The hernia had opened up again, probably a few months previously. The contents could be easily pushed back into the abdomen without signs of discomfort. The alpaca was in good body condition and, apart from the hernia, had made a complete recovery.

I have since heard that since being sold the alpaca has failed to come into oestrus.

A few comments:

- Try not to make it necessary, but never be afraid to let your client tell you how to do your job.
- Have something like a drum or big esky of hot water ready so several coils of the hose can sit in the drum and warm the water before it is used.
- Wear waterproof shoes or have another pair you can use at the clinic later.

The technique I read suggested a 10 cm skin incision. I was glad I made it bigger, as it allowed me to explore the abdomen first, and the wound closed much quicker than I expected. At no point did it seem that the wound might rupture, or that the wall of the abscess would evert through the incision.

It did not take long, probably 45 minutes of surgery, most of which was spent flushing the wound. It took almost as long to get it on a table, intubate, and tie out in dorsal recumbency. I have difficulty intubating ruminants. My advice is to use an ET tube, which seems ridiculously small (I succeeded with a 6.5mm tube in this case). I know it must increase the work of breathing. I also tell my assistant to bend the head back a bit further than seems reasonable, as though you are about to cut the poor animal's throat. The lady who works with me uses a stilette. **Any hints would be helpful!**

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Wildlife

DEATH OF A SPERM WHALE

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C&T No. 5925

On 6th March 2021, a dead Sperm Whale *(Physeter macrocephalus)* washed ashore near Forrest Caves on Phillip Island in Victoria, Australia. This was unusual, as these magnificent creatures usually live far out in the ocean, where they can dive down a thousand metres while they hunt for their main food source–squid from the depths of the seas.

These cetaceans live in complex social groups and they are cognitively very advanced. The Sperm whale has the largest brain of any mammal on earth (weighing over 7kgs); however, this is not remarkable as a proportion of body size (0.02%, humans and mice are closer to 2%). Their large heads are filled with a giant spermatici organ, which is used for buoyancy control and also for stunning their prey by intense high frequency bursts of sound.

It was a rather sad sight to see the whale's magnificent body on the beach, yet it posed some interesting questions.

The whale was identified as a male, it was about 16 meters long and weighed over 40 tonnes. If you consider the average racehorse to weigh 400kgs, then this whale was as big as about 100 racehorses.

According to the Dolphin Research Institute (DRI), solitary strandings usually indicate that the whale is sick; although other causes include old age, traumatic injuries or being struck by a ship. The presence of sucker marks on this particular whale led to the possibility that a giant squid may have caused injury to the whale. Scientists from Melbourne attended the site and took samples which may provide some additional information; however, an autopsy was not performed in this case.

Authorities were left with the complicated question of how to remove a large whale from a remote beach.

The area where the whale washed up was not accessible by vehicle. Parks Victoria decided that the environmental (as well as sensitive cultural) damage to obtain vehicle access could not be justified in this situation. History has some unfortunate tales of alternative methods of whale disposals that went wrong. This includes the `infamous' video from 1970 in Oregon, USA where they attempted to blow a beached whale up with dynamite.

youtube.com/watch?v=yPuaSY0cMK8

Other options involve burying the whale, but this can be challenging without adequate manpower and equipment as well as the carcass becoming exposed again by shifting sands. Dragging the whale back into the ocean can also be attempted but this requires specialised marine equipment, and the concern that the dead whale could be a shipping hazard as well as attracting sharks to the area.

Eventually, it was decided to leave this particular whale on the beach to decompose naturally. Local islanders would have to tolerate the stench for a while, and the surrounding beaches were closed to swimmers and surfers due to fear of increased shark activity.

A few days after the body was washed onshore, thieves visited the site during the night and removed the lower jawbone of the whale. Sperm whales are toothed whales, and their teeth are valuable like ivory. It is a sad example what length humans go to make a profit in a bizarre black market for whale teeth.

In Australia, whales (dead or alive) are protected by the Wildlife Act and people are not allowed to approach within 300 metres of a whale. This did not stop thousands of visitors and locals from Phillip Island coming to view the washed-up whale, an unusual tourist attraction. Although signs advised people to steer clear of the area and that pathogens were present on the body, these could not be adequately enforced.

The first thing you experience is the smell—from about 1 km away there is an intense pungent smell of rotting flesh and belching gases and it gets worse as you get closer. As a veterinarian, I have smelt awful stuff but this was off-the-scale revolting and emesis inducing. Having a pandemic-ready mask and the wind in the right direction

Figure 1. DAY 6 -Lower Jawbone stolen overnight

Figure 2. Day 10- Decomposing whale

enabled a closer look at the whale. Initially black and bloated with gases, the body slowly began deflating to a pale misshapen creature over several weeks.

It was interesting that initially the whale did not appear to be scavenged by seabirds; presumably it was too noxious? The island has no foxes either so land-based predation wasn't a major issue. Of course, there are the tiny beach scavengers including crabs, worms and other crustaceans taking their share of the food source provided by Mother Nature. There were some reports of sharks spotted in the vicinity though local surfers reported that the sharks are probably too well fed from the seal colony about 20 kms away.

Some huge whale bones started to appear on the beach, each weighing a large amount—hopefully they would remain on the beach to be analysed by palaeontologists of the future? These seemed to disappear over the weeks, either buried by the sands or removed?

Interestingly the Blue Whale skeleton in the Melbourne Museum is from a whale that washed up along the Great Ocean Road in Victoria in 1992. Removing the whale from the beach involved several cranes, a bulldozer and a low loader. The body was taken to Werribee sewerage works to decompose before the articulated skeleton was prepared for display in the museum.

The death of the sperm whale was a cause for lament although Australia only ceased commercial whaling in the 1970s. Sperm whales were highly valued for the large amount of oil in the spermaceti organ in the head, especially the males. Open

Figure 3. Day 19- Further decomposition

boat whale hunting began in the 18th century and continued for two centuries, inspiring literature tales like Moby Dick. The advent of petroleum products at the end of the 19th century gave the whales a reprieve but whaling started up in earnest again after WW2, with mechanised catcher vessels and explosive harpoons killing up to 30,000 whales a year, before the IWC (International Whale Commission) moratorium in 1988. As males were preferred, this led to a shortage of male sperm whales, and the species is still recovering.

Humans have attempted to improve the world for whales but the future will hold more challenges for the species with regards to chemical pollution, climate change, ship collision, plastics in the ocean as well as diminishing food sources.

Over 2 and a half months this whale carcass slowly decomposed; it was washed back into the ocean after a wild storm and then beached a couple of hundred meters away from the original site. Towards the end of May there were still bones and some blubbery bits on the shore. The shearwater chicks were beginning their flight from their nests and this brought Pacific gulls scavenging to the island, now seen feasting on the carcass as well as fledglings who couldn't take off. The death of the whale is a reminder of the fragility of the natural world, and how all living things will be recycled.

Additional Sperm Whale Facts

- Sperm whales can live up to 70 years, with females breeding from age 10, having a gestation period of roughly 14-16 months.
- The females live in a social group with their young. The young males often form bachelor groups when they leave the females and become solitary when they are very large. There are remarkable parallels to that of the African Elephant (large body sizes, large brain sizes, sexual dimorphism, similar life history variables, large ranges and matrilineall- based social systems)
- Females and young spend 75% of their time foraging; however, they spend several hours near the surface of the sea lying closely clustered, apparently resting (as seen in Blue Planet II, episode 4)
- Best locations to view sperm whales include Kaikoura (New Zealand) where they can be spotted all year round. !

References:

1. Encyclopedia of Marine Mammals (Sperm Whale: Hal Whitehead, Dalhousie University, Nova Scotia, Canada. (Page 1163-1172)
2. Wikipedia (Sperm Whale)
3. BBC Blue Planet 11 ; Episode 4
4. Local newspapers: *The Guardian* and *The Sentinel Times*

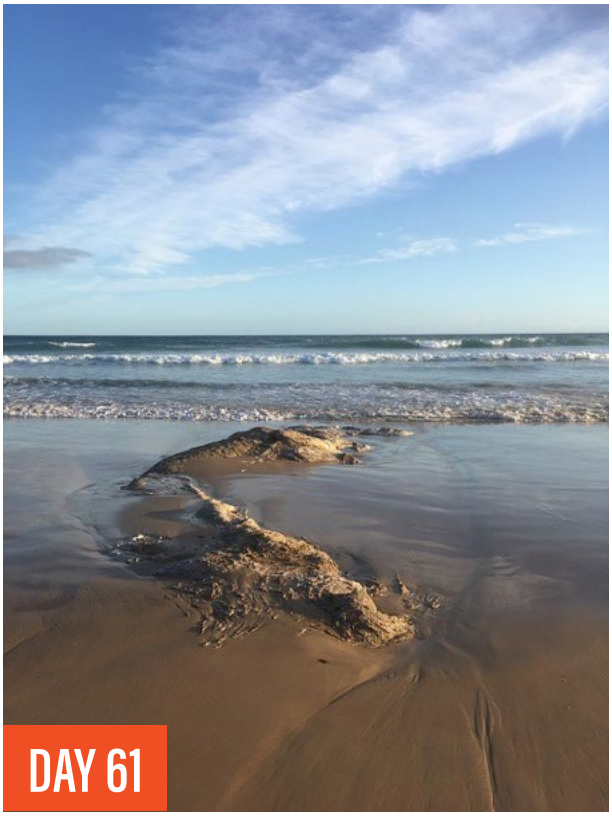
Figure 4: Day 24 - Huge whale bones appear on the shore

Figure 5. Day 30- The white carcass releasing its bony skeleton

Figure 6- Day 43 - Carcass washed back into the sea and out again after a storm

Figure 7- Day 61 - Disintergrated carcass but still pungent !

Figure 8. Day 75- Only a few blobs of blubber left on the beach



VERSATILE VET - LEARN MORE

Sally Grainger, Director

I love the variety and challenge of general practice, the constant problem solving and the excitement of never knowing exactly what case is going to show up next. I love feeling that confidence when I KNOW I have made good decisions and executed best practice techniques. But it can take YEARS to finesse the necessary skills and get a breadth of knowledge in all the required areas to feel this kind of confidence.

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I work with some of the most qualified veterinary specialists in the country, in some of the best veterinary hospitals, on a regular basis. I learn an enormous amount every day, and I love producing engaging videos to help other general practitioners, students and nurses learn as much as they can about every facet of veterinary science. It's a wonderful profession, and I feel very grateful to have the opportunity to see the best of it.

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General

FIRES IN WESTERN AUSTRALIA – SOLASTALGIA IS A NEW WORD FOR OUR LEXICON

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C&T No. 5926

`Solastalgia': a form of environmental grief generated by losing important aspects of one's home environment.

I first read this term a number of years ago and it resonated deeply. My first job as a new graduate Veterinarian was in the South West of Western Australia (WA) in 1999, near my grandparent's farm so I was very familiar with the environment. An area with the mighty Karri and Jarrah forests towering over the roads as I visited farms. I had favourite properties to visit because of the awe and wonder I felt as my tiny car wound its way through the giant trees, which somehow put life as a stressed new graduate into perspective.

I moved on and spent the next 10 years travelling and working overseas. During these years, the family farm burnt in a severe bushfire that also tragically killed a lovely young farmer and Volunteer Fire Fighter whom I had known. My family had to flee with only what they could carry. I always knew going back to see the burnt building and trees on the farm would be hard.

On return to WA, I was excited to show my husband the natural beauty of the area. When we drove along my favourite roads, I became more and more horrified. Many of these areas I loved had been clear felled and as far as the eye could see there were just stumps.

I have not visited the area again as the pain of the loss is too raw for me to process.

Solastalgia is named and described by Professor Glenn Albrecht from Murdoch University in WA.

It is a word we will all become very familiar with as Climate Change brings changes—from the loss of homes with high tides and storms, to the losses that come with fire and flood—which unfolded during the Summer of 2019-2020 in Australia.

I first learned of Climate Change in the early 80s. My Aunt was studying Environmental Science and thought 6-years-old was a good age to start the conversation whilst she babysat me and completed assignments.

Most of the things she told me about have turned out, 40 years on, to be fairly accurate. The days are long, but the years are short. What will the world look like when my children are 40?

It seems so far away, but blink and we will be there.

I have carried many conflicting emotions about Climate Change over the years—often simultaneously, guilt, shame, anger, sadness, despair, hope, denial, resignation.

There have been times I have been in the Climate wilderness – where I just cannot even bear to think about it.

Figure 1. Fire approaching

Figure 2. One of the hardest things was needing to move the animals quickly when they were agitated and nervous

Figure 3. Up close and personal

The moral obligation of our profession

As a practising small animal Veterinarian, I may help 30 or 40 animals per day. By being part of Veterinarians for Climate Action and expediating meaningful Climate Action, I can help billions of animals.

Recognising the impact of Climate Change on biodiversity and animal health has galvanised me.

Please join us
vfca.org.au

As a Veterinarian, I feel our profession has a moral obligation to speak for the billions of animals dead or displaced due to the fires and all animals affected by Climate Change; if we don't give them a voice, then who will?

February 2022 update:

It's been an incredibly dry year in South West WA and there have been lots of fires which have had devastating effects.

My aunt was unable to return to the same farm for a night last week when more fires passed nearby. Thankfully, conditions eased on the Sunday and the fires were controlled.

Small

WHAT IS YOUR DIAGNOSIS?

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C&T No. 5927

A 6-year-old, male desexed Domestic Shorthair cat was presented for re-evaluation of a urolith.

Six weeks prior, the cat had presented for dysuria (specifically, increased frequency of small volume urinations). Urinalysis had shown copious struvite crystals with urine pH of 8 and urinary tract inflammation. A distal urethral urolith was recognised radiographically. The bladder was easily expressed (i.e., the cat was not obstructed). The cat had been started on a urine modifying diet and treated with NSAIDs. The clinical signs reduced notably but the cat continued to dribble after urination for some weeks (attributed to the urolith). After the urine dribbling also reduced, the cat presented to assess if the urolith was still present.

The radiograph from 6 weeks prior was assessed:

Questions:

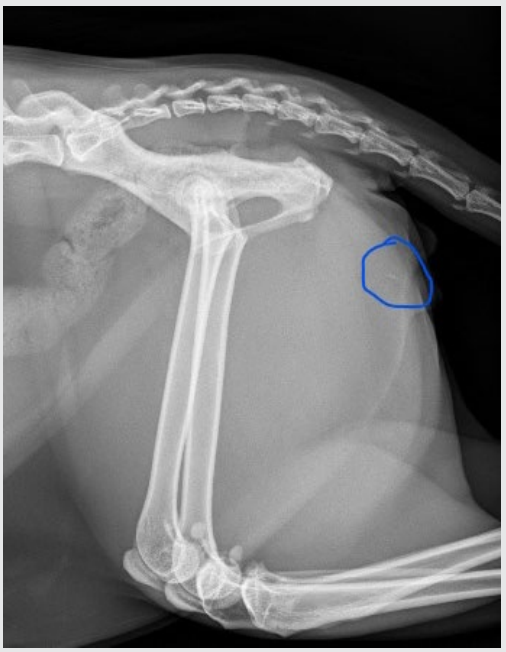
What is your diagnosis?

How would you proceed?

Best Answer Wins a CVE$100 voucher

Email your answer to
Joanne.Krockenberger@sydney.edu.au.

The voucher may be used to enrol in CVE courses or to purchase CVE products, for example a DVD or digitised video from CVeSHOP. Renowned surgeons teach a number of must-learn veterinary procedures available as a DVD or MP4 download.



small

SUPPLEMENTED LIQUID ENTERAL DIET FOR THE DOG & CAT USING JEVITY

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C&T No. 5928

The following recipe is designed to make the liquid human enteral diet 'Jevity' (Abbott Nutrition) a more balanced and complete diet for short-term enteral nutritional support for critically ill dogs and cats. It has the advantage of being a liquid formulation, suitable for administration via small-bore nasogastric or naso- oesophageal feeding tubes.

Ingredients

- Jevity
- Whey protein isolate (unflavoured)
- Balanced vitamin/mineral supplement for carnivores e.g. Balance IT™Carnivore Blend
- Di-calcium phosphate

Recipe

- Jevity: 50 mL
- Whey protein: 50 grams
- Multivitamin/mineral 11.5 grams (Balance IT™Carnivore blend used for this recipe)
- Di-calcium phosphate 2.5 grams

Mix

1. Add the ingredients to a bowl
2. Blend well with a hand-mixer or blender until the mixture is smooth and free of sediment
3. Store in a tight-closing bottle and label with contents, time and date
4. Store in a refrigerator
5. Shake well before each use

Nutrient Content (% of Metabolisable Energy):

- Energy Density 1.1 kcal/ml
- Digestible protein 26%
- Fat 26%
- Carbohydrate and Ash 48%

Emeraid is now available in Australia. Were it not for the price, we would have switched over to using it alone, but for price, we are still using Jevity when we don't specifically want to use Emeraid. The advantages are:

1. It is sold as a powder, so you only mix up what you need
2. You can mix it up to higher energy densities, so when you have an oesophagostomy tube in place, you can make up 2kcal/mL, which is more energy dense than a/d or RC Recovery
3. It's very low carb, which for a number of critical patients, that is important. Jevity, even the supplemented recipe, is far too high in carb for critically ill, hyperglycaemic patients.

SPECIALISTS' CORNER

Dr Tunbi Idowu
FANZCVS (Small Animal Medicine)
sashvets.com/our-team/dr-tunbi-idowu/

What attracted you to this discipline?

I have always had an affinity for animals and loved my science subjects at school, so veterinary science made sense. Regarding choosing to specialise in internal medicine, that also was a no-brainer (no pun intended). Based on my overthinking and methodical traits, this drew me to the introspective and problem-solving nature of internal medicine.

Worst veterinary experience?

A client asking to see an Australian veterinarian, with the subtext being that I did not look Australian. This upset me, as I was born in Australia and grew up here and thus I am Australian. It highlighted that some people will judge a book by its cover but you don't have to make it your whole story.

Best veterinary experience?

Hands down, this has been passing my fellowship examinations. I have never studied and sacrificed so much for an exam before. It was not just an exam—it was a lifetime achievement for me.

If you weren't a vet, what would you be?

I would definitely be my dog Kai. He is full of personality, fearless and spoilt so much by my family; plus he doesn't have to work and pay taxes. Pretty sweet life, if you ask me ☺

Perspective No. 153

CANINE IMMUNE-MEDIATED POLYARTHRITIS

WALKING ON EGG SHELLS

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What is Canine Immune-Mediated Polyarthritis (IMPA)?

Canine IMPA is a common, but likely under-recognised, condition in clinical practice. As the name implies, it is an immune-mediated disease directed at the synovial membranes of multiple joints. IMPA is commonly called a polyarthropathy, suggesting that five or more joints are involved. However, many cases of canine IMPA may involve fewer than five joints and therefore should be more strictly defined as immune-mediated oligoarthritis; which involves involvement of two to four joints. Semantics aside, polyarthritis and oligoarthritis are tantamount in definition. Rarely, and again contrary to the strict definition for IMPA, this condition has been reported to involve just a single joint.

IMPA causes a non-septic, suppurative or neutrophilic synovitis and is often accompanied by systemic signs of illness, such as lethargy, fever and inappetence. This condition is suspected to be the result of antigen/antibody immune-complex deposition within the synovium, resulting in a sterile synovitis; that is a type 3 hypersensitivity reaction *(Figure 1)*. However, some forms of IMPA

Contents

may also include a T-cell mediated response; that is a type 4 hypersensitivity reaction, directed against the articular cartilage and causing erosive damage *(Figure 1)*.

Implicated antigens are typically found in the systemic circulation, but can originate from within the joint space itself, creating a true auto-immune disease. Systemic immune complexes (antigen and antibody complexes) can arise from a variety of acute and chronic antigenic stimuli, including viruses, microbial agents, neoplasia, and drug haptens. The deposition or formation of immune complexes in the joint space activates complement along the synovial membrane and within the synovial fluid. Complement fixation results in tissue damage and the release of cytokines, some of which attract neutrophils leading to the suppurative inflammation noted in cytological and synovial membrane histopathological examinations.

How is IMPA classified and what can cause this disease?

IMPA can be classified as non-erosive or erosive. The clinical distinction between the two is primarily determined by radiographic findings of cartilage and subchondral bone destruction in one or more joints; that is evident in erosive IMPA. The biggest underlying cause for canine erosive IMPA, is rheumatoid arthritis. In this IMPA, rheumatoid factors which are autoantibodies against antibodies, are found in the serum in less than 30% of cases.

Non-erosive IMPA is far more common in veterinary medicine than erosive IMPA. Various aetiological classification schemes have been used to describe canine-non erosive IMPA, but a commonly cited classification scheme is outlined in Table 1.

- **Type 1** IMPA is when the source of antigenic stimulation and disease is not able to be determined after thorough investigation. Idiopathic polyarthritis requires ruling out type 2, 3, 4 and other causes for IMPA. This is the most common form of IMPA accounting for approximately 50–65% of all canine polyarthritis cases.
- **Type 2** IMPA is associated with an inflammatory or infectious disease remote from the joint for example pancreatitis or septic peritonitis. However, some people may include pancreatitis induced IMPA in type 3 IMPA. Regarding septic peritonitis, this certainly can cause septic arthritis via hematogenous spread of bacteria to the synovium but to complicate things, these patients can also develop a reactive polyarthritis.
- **Type 3** IMPA is associated with alimentary tract disease, in which I would also include non-infectious hepatic disease and inflammatory bowel disease in this category.
- **Type 4** IMPA is associated with neoplasia remote from the joints and includes lymphoproliferative disease.

- Other classifications for IMPA include juvenile onset polyarthritis of Akitas; polyarthritis syndromes e.g., dogs with IMPA and steroid-responsive meningitis arteritis; drug-induced IMPA in which the most commonly implicated type of drug are sulfonamides, especially in Doberman pinschers.

What does an IMPA dog look like clinically?

Unfortunately, canine IMPA can present in a wide variety of ways that often form a vague, waxing and waning and non-specific clinical picture; thus, obscuring the diagnosis. Due to the fact that IMPA can be secondary to another disease it also means there can be concurrent clinical signs related to the primary disease, thus overshadowing the clinical signs of IMPA.

Typical reported clinical signs of IMPA, include reluctance to walk, pyrexia, a stiff gait, shifting lameness and multiple effused and painful joints on physical examination. However, in one study, only 40% of IMPA cases had joint pain or joint effusion detected on physical examination. This therefore clearly highlights that absence of painful and/or swollen joints in a dog does not sufficiently exclude IMPA from your differential diagnosis list.

In my IMPA research project, only a small number of dogs had overt lameness but the majority of IMPA dogs did have either painful joints and or swollen joints, which increased the index of suspicion of this disease.

Fever, however, was the most common clinical sign in my cohort of dogs with IMPA; with 77% of dogs being febrile. This finding is in line with two studies evaluating causes for fever of unknown origin in dogs, in which IMPA had the highest prevalence.

Other non-specific signs of IMPA include weight loss, GI signs, exercise intolerance and inappetence and I personally have seen that some of these dogs can develop mild respiratory signs and coughing despite normal chest radiographs.

Neck and back pain may also be present, either if vertebral articular facets are involved as part

Type 2 to type 4 IMPA are sometimes collectively referred to as reactive polyarthritis

Type 1	Type 2	Typre 3	Type 4	Other classifications IMPA secondary to :
No identifiable associated cause (uncomplicated idiopathic arthritis)	Associated with an infectious or inflammatory disease, remote from the joint	Associated with gastrointestinal/ hepatic disease (enteropathic)	Associated with neoplasia remote from the joints (neoplastic)	• Systemic lupus erythematous polyarthritis • Polyarthritis-meningitis syndrome • Vaccination induced (little evidence for this) • Drug induced polyarthritis • Breed specific polyarthritis

Table 1.

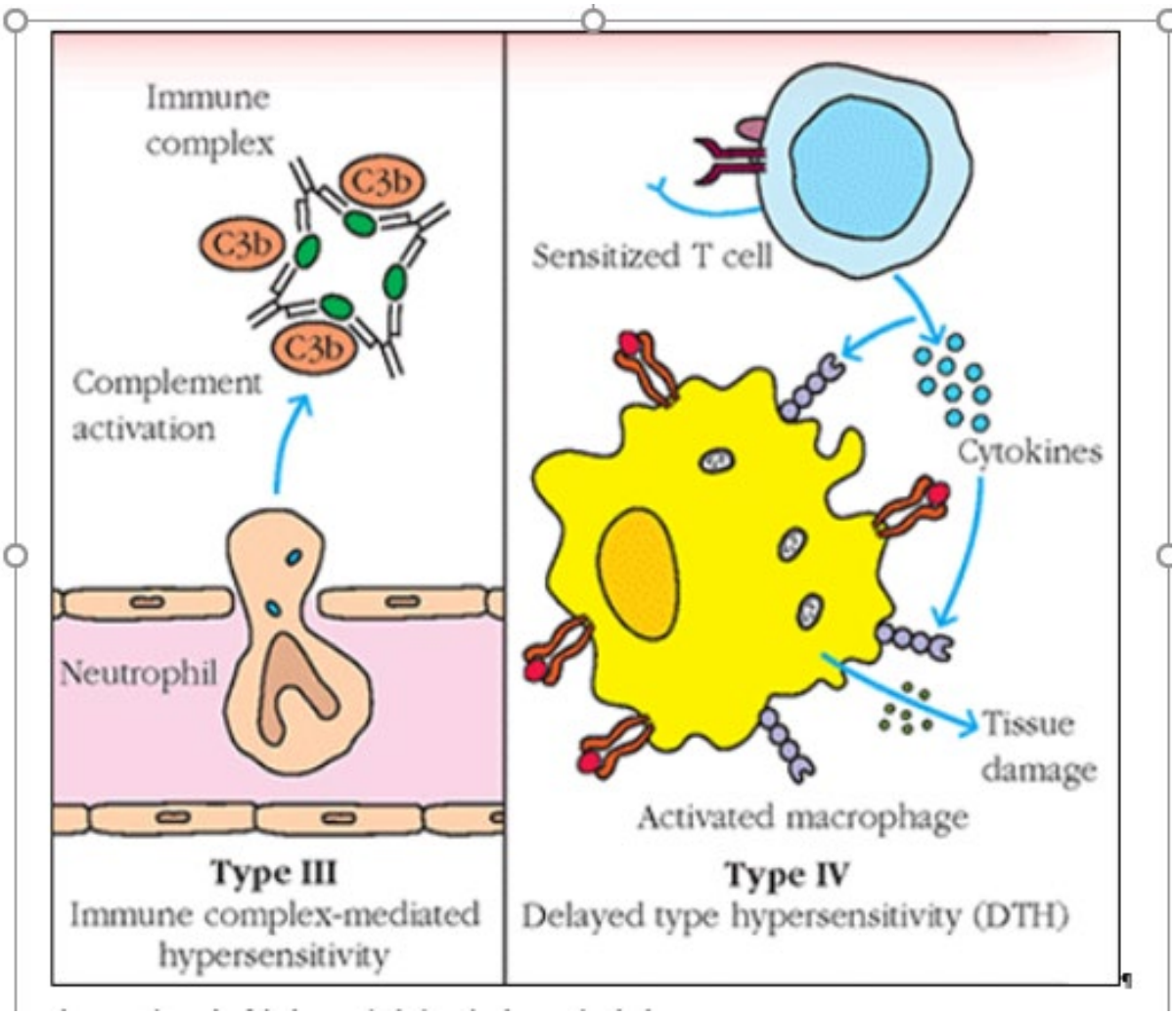

Figure 1. Schematic of the immunological mechanisms underpinning IMPA
https://microbenotes.com/hypersensitivity-type-i-ii-iii-and-iv-in-one-table/

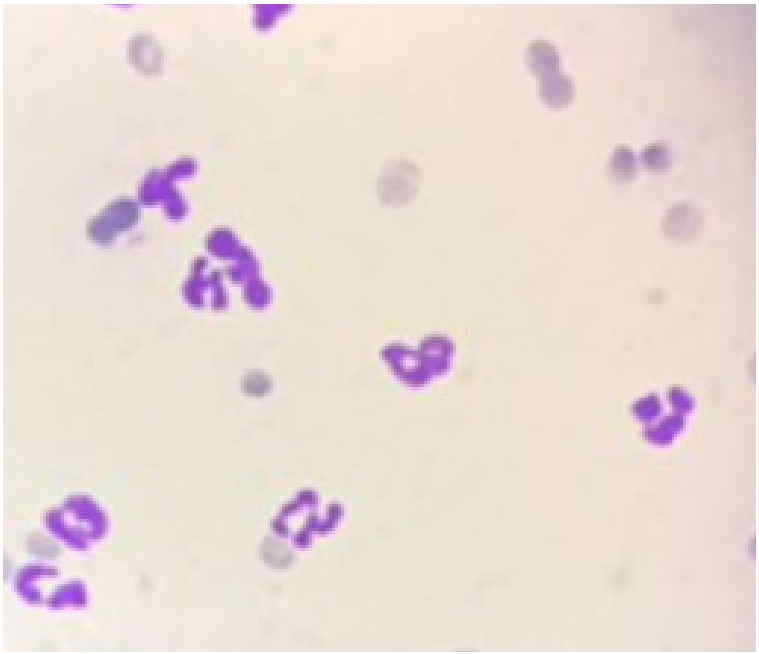

Figure 2. Diff-Quik stained joint fluid cytology from a dog with IMPA

of IMPA or if concurrent Steroid Responsive Meningitis Arteritis is present.

Regarding signalment, any dog can get IMPA. There is no breed predilection, other than with the breed associated IMPAs. There is no disparity between gender but regarding age of onset, dogs tend to be young adult to middle age. It is uncommon for dogs less than 12 months of age to develop IMPA, unless there is a breed predisposition.

How is IMPA diagnosed?

The actual diagnosis for IMPA is pretty straightforward and relies on arthrocentesis of at least two joints in which the synovial fluid demonstrates neutrophilic inflammation (ideally greater than 25% neutrophils of the cellular population, but usually >50%) as seen in *Figure 2*.

I find arthrocentesis of the carpi and hock joints one of the easier procedures I perform. I don't think it is technically that difficult if you have effusive joints but if the joints are only mildly effusive or actually normal then it can be difficult to get synovial fluid or synovial fluid without blood contamination. Performing joint aspirates of the stifle and elbow joints is a little harder. In dogs with IMPA there is often a large volume of synovial fluid aspirated, and the fluid turbid, and watery. Importantly, the normal viscid appearance of joint fluid is much less evident.

Once I obtain synovial fluid, I submit that for cytology. I usually don't submit the fluid for culture unless I am concerned about septic arthritis and even then, joint fluid culture is an insensitive tool. If joint fluid is cultured then a blood culture bottle should be utilised to increase sensitivity.

The next test I perform in my diagnosis of IMPA are joint radiographs or CT of at least two joints to exclude other causes of joint disease and check for erosive IMPA radiographic signs *(Figure 3*).

The third step in the diagnosis of IMPA is to classify it as idiopathic or due to an underlying cause. Depending on patient history and clinical signs, these additional tests are varied and may include complete blood count (CBC), serum chemistry panel, urinalysis abdominal ultrasound, thoracic radiographs, whole body CT, infectious disease testing e.g. leptospirosis, brucellosis.

I will freely admit I don't embark on looking for an underlying cause in every IMPA case. It depends on client finances and my index of suspicion that there may be an underlying cause. I will have this discussion with the owners about how vital it is to look for an underlying cause or concurrent disease. One scenario where I do try to encourage the owner to do additional tests, is when the IMPA patient is presenting with neck or back pain. In this scenario the clinical presentation could be compatible with IMPA affecting the interverbal

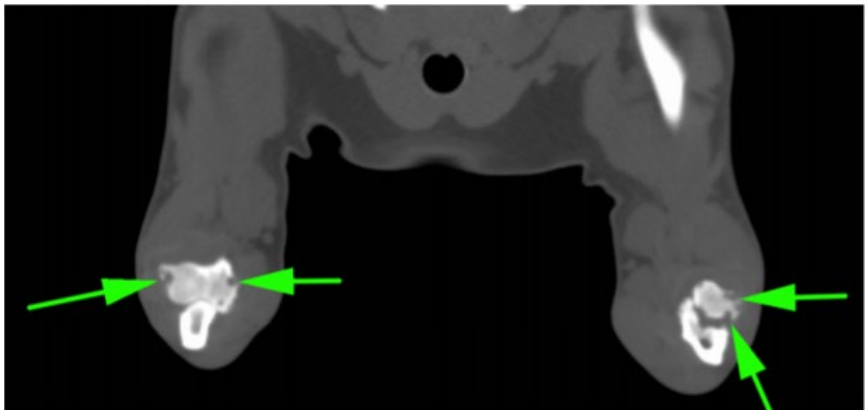

Figure 3. Transverse post-contrast soft tissue reconstruction algorithm image displaying examples of osteolytic lesions, in a dog with erosive IMPA

joints OR polyarthritis-meningitis syndrome i.e., steroid-responsive meningitis arteritis. In this case, I would perform a CSF tap to rule out this condition because if the IMPA patient has concurrent SRMA then I am a lot more careful in how quickly I taper immunosuppressive medications and the prognosis can be less favourable.

What drugs can we use to treat IMPA?

The majority of the dogs can be controlled with single-agent prednisolone therapy. I generally start at 2mg/kg per day for 10-14 days and then slowly taper over 4-6 months. However, an additional immunosuppressive agent may be required but if you are treating a large breed dog with prednisolone, the side effects like muscle wasting are going to be more profound and thus you may need to taper the prednisolone more quickly and have another immunomodulatory drug on board because of this.

In terms of which adjunctive immunomodulator to choose, there is no optimal one! However, cyclosporine and leflunomide have been studied as monotherapy in IMPA with good success, albeit with small case numbers.

How is the disease monitored?

In our armamentarium to monitor resolution and relapse of IMPA we have a couple of tools.

Historical and clinical exam findings—but as mentioned previously, not all these patients will have overt signs of joint pathology e.g., lameness, joint pain, joint effusion. For dogs that presented with fever, resolution of fever does not necessarily mean that the localized joint inflammation has completely resolved. Thus, physical exam is an insensitive method for monitoring IMPA. Obviously if the patient at diagnosis had hallmarks of joint pathology and continues to have these at subsequent rechecks, it is safe to assume we are not yet on top of the disease.

Some clinicians advocate serial arthrocentesis to monitor response to treatment, although often clinical response is used instead, since repeat arthrocentesis usually requires sedation or anaesthesia, along with the concomitant expense.

Recent studies have investigated using C-reactive protein as a surrogate marker for joint inflammation. As this test is now increasingly available in the practice setting, it could provide a good compromise for monitoring, with a measured reduction in C-reactive protein likely correlating with improvement in joint inflammation. However, a recent study by Grobaman *et al*. 2017, *JVIM,* reported results that did not advocate the independent use of CRP in monitoring IMPA remission.

What does the future hold for these dogs?

The prognosis of dogs with non-erosive IMPA depends on the underlying cause, but in general the prognosis for type 1 IMPA or idiopathic IMPA is good, with most dogs responding well to corticosteroid monotherapy or combination immunosuppressive drug therapy. One study of 39 cases of canine type I polyarthritis reported that most responded to therapy: 56% of affected dogs were cured, 13% relapsed but were subsequently treated successfully, 18% required life-long therapy to maintain remission, and only 15% were euthanized or died because of their disease.

Dogs with erosive IMPA will require lifelong therapy (monotherapy is not appropriate) and the response to treatment and long-term prognosis are generally considered poor. While the immunosuppressive agents may control the joint inflammation, the deformation to the bones can result in joint collapse and ongoing pain and mobility issues.

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