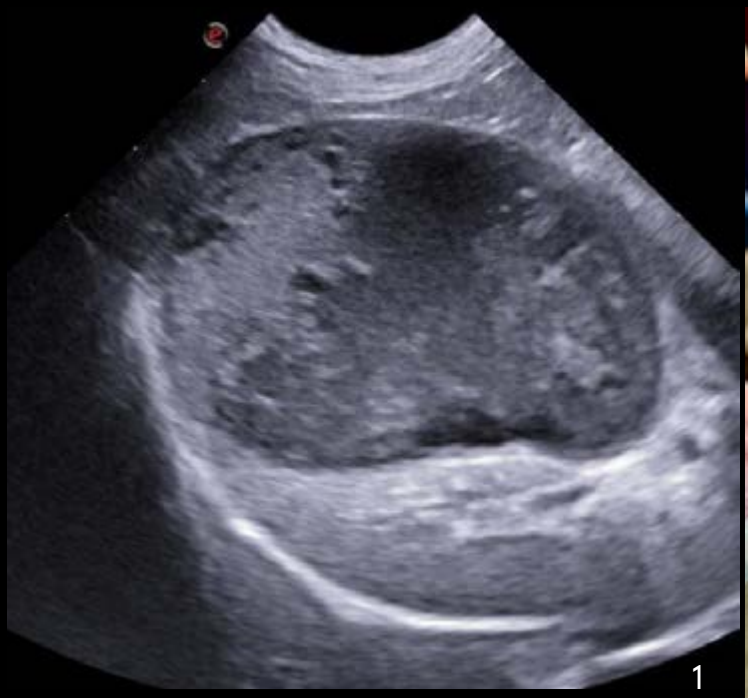
C&T

THE UNIVERSITY OF SYDNEY
Centre for Veterinary Education

Control & Therapy Series
Issue 311 | June 2023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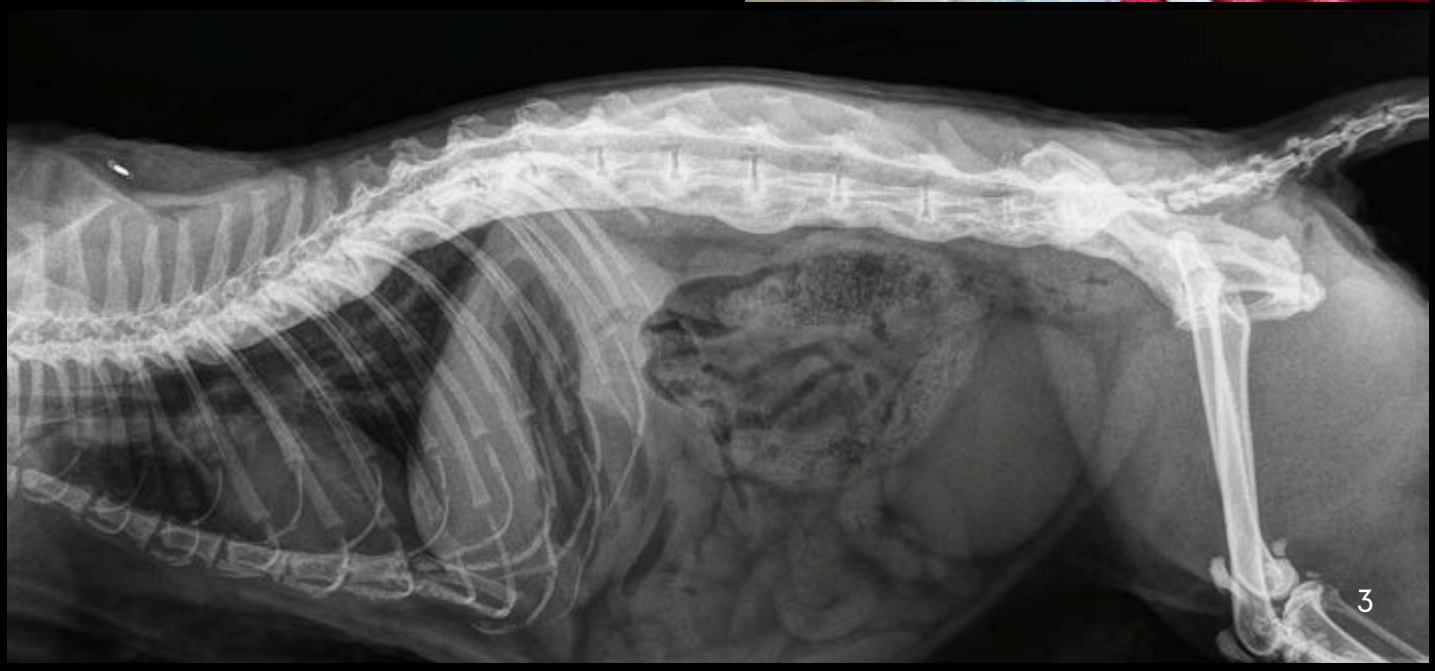
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***Control & Therapy* Series**

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

Join in—write up that interesting case

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

Winners

Major Winner
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FROM THE DIRECTOR

There are few of us unfamiliar with that pulse-racing feeling of being faced with an unusual case that presents a treatment challenge. Hopefully, your approach works like a charm, but we all know that's not a certainty. Rachel Korman (lead tutor in our Feline Medicine Distance Education (DE) course) has written about a case exactly like that—'The Hardest Dental Extraction You'll Ever Do', page 7—I could feel my heart rate increasing just reading about it!

Another of our tutors, Xander Huizing (who delivers our Diagnostic Imaging: Musculoskeletal DE course) takes us step by step on page 3 through the unusual but life-threatening diagnosis of a greyhound presenting with pyrexia and acute lethargy. Xander has an incredible capacity for breaking down radiographic interpretation (his webinars in our Webinar Library are well worth a look) and true to form, he takes the same approach here. Once Xander points something out, it becomes so clearly apparent you can't unsee it.

Jeremy Rogers' submission on anaemia and weakness in lambs is interesting even if (like me) you're not seeing ruminants on a regular basis. Also worthy of note, is veterinary pathologist Neil Horadogoda's comment that *Mycoplasma ovis* is an emerging zoonotic pathogen.

There are a few articles you will definitely want to bookmark and return to in this edition. The team from the Animal Poisons Centre have presented a fantastic summary of the clinical presentations and approaches to management of common recreational drug toxicosis. Also, Terry King and Julian Lunn's article on gall bladder disease (which starts with the delightful 'for a trivial little organ that seems unnecessary and dispensable...') is brilliant—particularly their break down of the pathophysiology of gall bladder disease. We also introduce in this edition some content courtesy of the International Society for Feline Medicine—Sam Taylor gives a considered, evidence-based opinion on the use of continuous glucose monitors in cats, and 'Research Roundup'—a summary of the latest in feline research.

Add to that a great case study on a French bulldog with behaviour problems and you've got some great reading. Enjoy!

Simone

Small

ACUTE LETHARGY IN A GREYHOUND

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C&T No. 5972

Xander is a board-certified specialist in Veterinary Diagnostic Imaging (DipECVDI). As such, he spends his days working with ultrasound, radiography, CT, MRI and fluoroscopy trying to make other clinicians look good. He has worked in referral and academia in Australia and Europe. He is the Director of The Austin Vet Specialists and provides an imaging service to the vets of Adelaide and South Australia (and anybody else who will offer him a cup of tea and biscuit). Xander is passionate about radiology and education. He strongly believes in the synergistic value of a veterinary radiologist and the added value they bring to a hospital. Xander loves helping people improve their imaging skills (and becoming a better vet than he is). He has taught numerous vets, nurses, students, interns and residents whilst working as a clinical radiologist.

Signalment and Presentation

A seven-year-old male neutered Greyhound with a sudden onset of inappetence and lethargy.

History

A seven-year-old male neutered Greyhound who was rescued at the age of two years was presented with a sudden onset of inappetence and lethargy to the referring veterinary surgeon. Clinical examination revealed pyrexia (40.1°C) which was non-responsive to non-steroidal anti-inflammatory drugs (meloxicam). No other abnormalities were found on clinical examination. Thoracic radiographs were obtained and were considered abnormal and consequently the patient was referred.

Upon presentation at our referral centre, the owners reported a history of progressive lethargy and panting but otherwise eating well. An increased water intake and polyuria were reported but no previous illnesses. Upon clinical examination there was no coughing or breathing difficulties, but heart auscultation revealed muffled heart sounds with harsh lung sounds on both sides. Thoracic radiographs were taken as part of the work-up and the left lateral is seen in Figure 1.

What Abnormalities Can You See on the Radiographs?

A mild to moderate amount of pleural effusion is present with visualisation of pleural fissure lines. The effusion has an asymmetric distribution in the right hemi thorax, particularly collecting around the apex of the cardiac silhouette and the consolidated right middle lobe.

The terminal 1/2—1/3 of the right middle lobar bronchus is air-filled (air bronchogram) with a branching pattern to the periphery. The proximal 1/2 of the right middle lobar bronchus is not

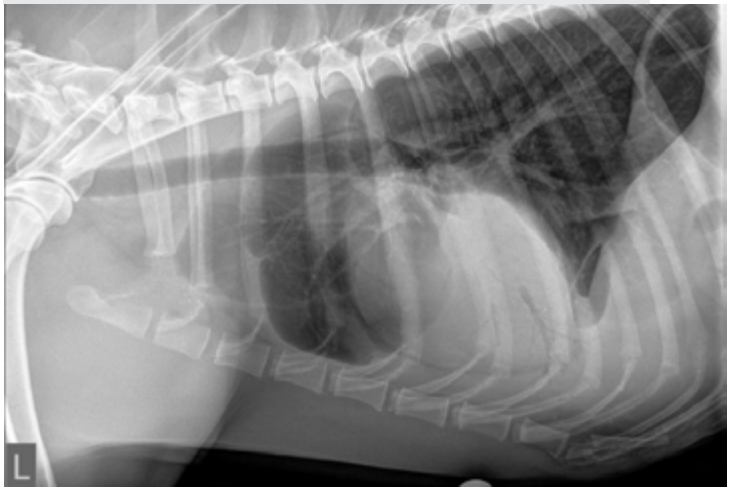

Figure 1.Left lateral radiograph

visualised or at least completely narrowed—the transition to air bronchogram is abrupt. The origin of the bronchus is not visualised.

Ill-defined foci of gas are present within the consolidated lung but it is not a typical emphysematous lung pattern but more similar to a vesicular pattern. The cardiac silhouette and pulmonary vasculature appears within normal limits.

Key Radiological Findings

1. Moderate volume of pleural effusion
2. Right middle lobe consolidation
3. Loss of visualisation of the origin of the right middle bronchus

Differential Diagnosis

- lung lobe torsion
- neoplasia (mesothelioma haemangiosarcoma, adenocarcinoma)
- focal pneumonia
- lung infarction (thrombosis)

What Further Investigation Could you Consider to Try to Obtain a Diagnosis?

Biochemistry and haematology showed mildly decreased number of white blood cells and platelets but no other abnormalities.

A conscious thoracic ultrasound was performed and a representative image is shown as Figure 2. It shows the consolidated right middle lung lobe surrounded by free pleural fluid. Within the lung lobe, distinct foci of gas causing distal acoustic shadowing can be seen. This corresponded to the radiographic findings and a lung lobe torsion, possibly secondary to neoplasia, was suspected. The pulmonary changes were deemed quite severe and the dog was sent to surgery immediately.

At thoracotomy, a lung lobe torsion was confirmed and the removed right middle lung lobe was submitted for histopathology. The pathology report stated that the pulmonary tissue was compatible with haemorrhage and pulmonary haemangiosarcoma could not be excluded. The pericardial and pleural tissue were consistent with inflammatory granulation tissue. The patient recovered well from the surgical procedure and did not show any further signs of dyspnoea or coughing. Further investigation of a possible haemangiosarcoma (echo and abdominal ultrasound) did not reveal a primary focus or metastatic disease. The patient was discharged after seven further days of hospitalisation. The fact that the patient recovered so well made an underlying disease of the lung lobe less likely but cannot be excluded at this point in time. Furthermore, follow-up three years later revealed no further clinical signs which also makes a neoplastic aetiology less likely.

Lessons From This Case

Although lung lobe torsion does not occur commonly, the implications of this condition are serious. Without swift intervention, it can be fatal and usually surgery is the treatment option of choice.

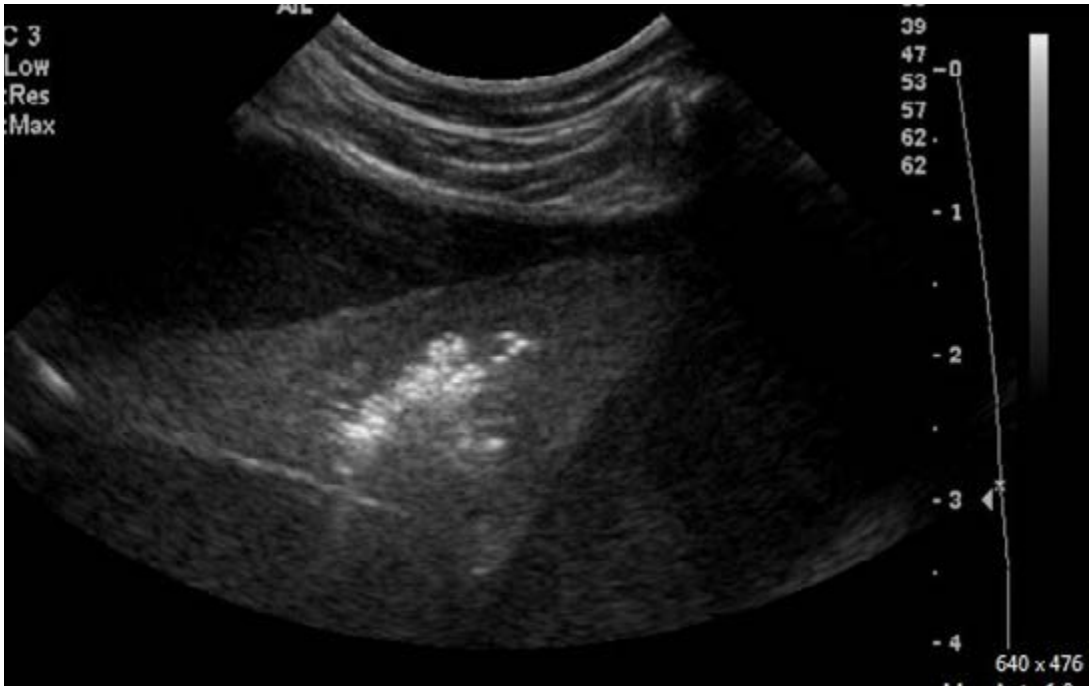

Figure 2. Right middle lung lobe

Lung lobe torsion occurs when a lobe rotates around its own axis, usually at the hilus. As a result, the vasculature is occluded. The venous and lymphatic return is occluded first, leading to congestion, oedema and pleural effusion. However, in later stages the arterial supply can also be compromised usually leading to necrosis and acutely life-threatening complications. Another sequela is that the air inside the torsed lobe is trapped which shows as a characteristic vesicular pattern. Various imaging modalities can be used to diagnose a lung lobe torsion, including radiography, ultrasound and (contrast-enhanced) computed tomography (CT).

Two distinct populations of breed have been described to get spontaneous lung lobe torsion. Large breed dogs with a deep, narrow chest (such as Afghan Hounds) are predisposed to right middle lobe torsion whereas small barrel-chested breeds, Pugs in particular, are more prone to left cranial lobe torsion. Usually lung lobe torsions are spontaneous but occasionally underlying pulmonary pathology can progress to torsion. An example would be a bronchial neoplasia causing bronchial luminal occlusion and subsequent air resorption leading to a smaller lung lobe which will be susceptible to torsion. However, usually an inciting cause for the torsion is not found.

Radiography

Radiographically, the most common sign is pleural effusion. In one study of 22 cases between 1981-1999, all 22 dogs had pleural effusion either unilateral or bilateral (Neath *et al*, 2000). Other common radiographic findings are increased lobar opacity and a pattern of small dispersed air bubbles within the affected lobe known as a vesicular pattern. The air bubbles are a sequela of the bronchial occlusion which causes air trapping. However, this vesicular pattern was variably extensive and not always present.

The bronchi leading into the affected lobe are usually not completely visible and can appear irregular, focally narrowed or even blunted. Other common radiographic findings included mediastinal shift and a dorsally displaced trachea with axial rotation of the carina (d'Anjou M-A *et al*, 2005). The pulmonary vessels in the affected lobe are usually not visible or, when using cross-sectional imaging such as contrast enhanced CT, can be observed running in an abnormal direction.

Ultrasound

Thoracic ultrasound can help in the diagnosis and can usually be performed without the necessity of sedation. Particularly if the radiographs are complex or inconclusive, it may be an aid. The easiest manner to ultrasound the thorax of these patients is in sternal recumbency as these are usually dyspnoeic patients who usually do not tolerate prolonged periods in lateral or dorsal recumbency.

As with radiography, pleural effusion is almost always seen. Due to collapse of the lung lobe, it can become echogenic with usually a characteristic hypoechoic appearance and coarse echotexture (so called 'hepatised' appearance). Hyperechoic foci with shadowing may be seen in the centre of the abnormal lobe, which represent foci of trapped gas. Linear hypoechoic structures without blood flow and with hyperechoic walls have also been described. These are consistent with fluid-filled bronchi and are termed fluid bronchograms. Sometimes the tip of the lobe can be seen orientated in the wrong direction, i.e. in cranial or dorsal direction. Using colour Doppler can be helpful to distinguish a torsed lobe from a fluid-filled lobe secondary to other pathology. In lung lobe torsion, decreased and usually absent flow can be demonstrated within the affected lobe.

Usually the venous flow is occluded first as the veins are more compressible. In later stages, the arterial supply will also be comprised.

Computed Tomography

Computed tomography is considered the cross-sectional modality of choice when examining the thorax. It gives superior contrast resolution compared to radiography and can distinguish between soft tissue and fluid. It has the additional benefit that, besides examining the potential lung lobe torsion, it can also be used to screen for other pathology such as pulmonary or mediastinal masses, lymphadenomegaly and airway disease. An additional benefit is the ability to reformat the images and examine them in customised planes (multi-planar reconstruction). This allows bronchi to be followed along their length making the search for a blunted or abruptly ending bronchus more easy. It can be useful to use an iodinated contrast medium given intravenously, usually at a dose range of 660-880mgI/kg, to delineate the vessels. An abnormal course of the pulmonary lobar arteries and veins is expected with a lung lobe torsion. Virtual CT bronchoscopy has also been reported to aid in diagnosis (Schultz *et al*, 2009). The scientific literature has a few reports of partial lobar torsions. These are more challenging to diagnose since characteristic features associated with complete torsion may be absent.

The findings specific for a lung lobe torsion on CT are similar to radiography. Pleural effusion can be

readily appreciated with abrupt loss of visualisation of the associated bronchus. After contrast administration, the affected lobes show mild or absent enhancement due to vascular occlusion. The torsed lobes are usually swollen with the apex orientated in an abnormal direction and a vesicular pattern is also readily identified. If in doubt, inflation of the lungs can be used to differentiate between the increased attenuation of atelectatic lungs and torsed lobes. Inflation should resolve atelectasis but not affect a lung lobe torsion.

In conclusion, lung lobe torsion is a relatively rare disease but will require swift intervention. The suspicion should be raised in patients with consolidation of the right middle (large breed) and left cranial (small breed dogs) lung lobes and the presence of pleural effusion. A range of modalities can be used for further assessment.

Further reading:

1. d'Anjou M-A, Tidwell A, Hecht S (2005) Radiographic diagnosis of lung lobe torsion. *Vet Radiology and Ultrasound* 46(6): 478-84 doi: 10.2460/javma.2000.217.1041
2. Hecht S (2008) Thorax. In: Penninck D, d'Anjou M-A, eds. *Atlas of small animal ultrasonography*. Blackwell Publishing, Oxford, UK: 138-41
3. Mai W, *et al* (2008) The lung parenchyma. In: Schwartz T and Johnson V, eds. *BSAVA Manual of Canine and Feline Thoracic Imaging*, Gloucester, UK: 276-280
4. Neath P *et al* (2000) Lung lobe torsion in dogs: 22 cases (1981-1999). *Journal of the American Veterinary Medical Association* 217(7): 1041-4 doi: 10.2460/javma.2000.217.1041
5. Schultz RM, Peters J, Zwingenberger A (2009) Radiography, computed tomography and virtual bronchoscopy in four dogs and two cats with lung lobe torsion. *J Small Anim Pract* 50(7): 360-3 doi: 10.1111/j.1748-5827.2009.00728.x
6. Seiler G *et al* (2008) Computed tomographic features of lung lobe torsion. *Vet Radiology and Ultrasound* 49(6): 504-8 doi: 10.1111/j.1740-8261.2008.00435.x
7. Thrall, D (2013) The canine and feline lung. In: Thrall D, ed. *Textbook of Veterinary Diagnostic Imaging*, Elsevier, St Louis: 629-32
8. Wisner E, Zwingenberger A (2015) Small Airways and parenchyma. In: Wisner E and Zwingenberger A, eds. *Atlas of Small Animal CT and MRI*, Wiley Blackwell, Iowa: 459-60

This case report was first published in the *UK-VET Companion Animal* April 2016. ◆

Press Release

MSD Animal Health today announces the Australian Pesticides and Veterinary Medicines Authority (APVMA) has approved a novel injectable form of BRAVECTO® (fluralaner) for the control of *Ixodes holocyclus* (paralysis tick) for 12 months, the treatment and prevention of *Ctenocephalides felis* (flea) infestations for 12 months, the control of *Rhipicephalus sanguineus* (brown dog tick) for 11 months, and the control of flea allergy dermatitis (FAD).

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General Manager, Australia and New Zealand, MSD Animal Health, Eric Flanagan

'MSD Animal Health has always been committed to ensuring veterinarians are at the forefront of pet owner education and advice when it comes to our products. BRAVECTO QUANTUM is no exception; we are excited that Australia is the first country in the world to launch this game-changing treatment that can only be administered by veterinarians.'

Small

THE HARDEST DENTAL EXTRACTION YOU'LL EVER DO....

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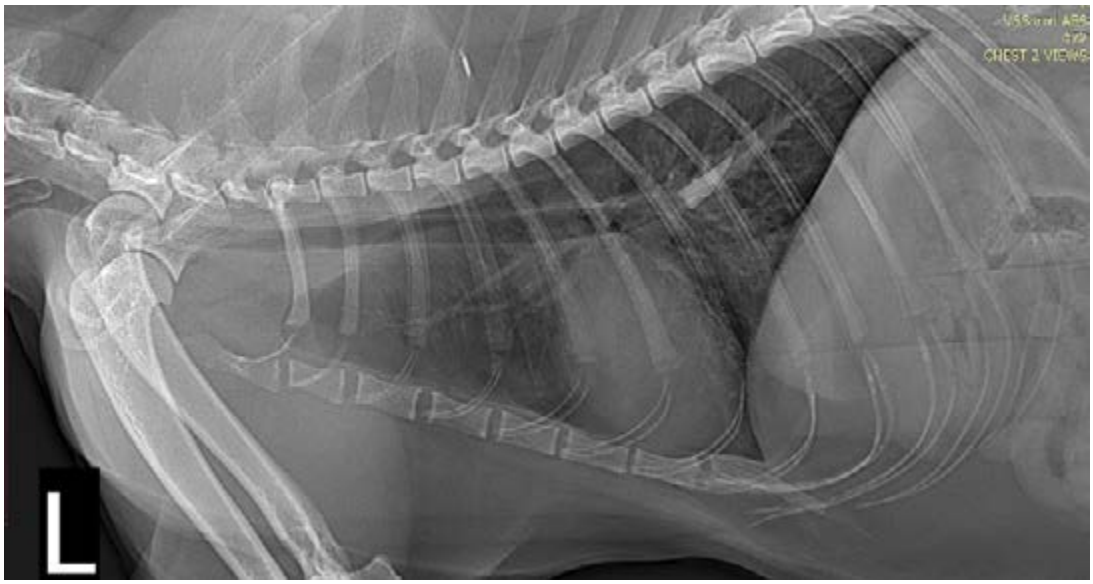
C&T No.5973

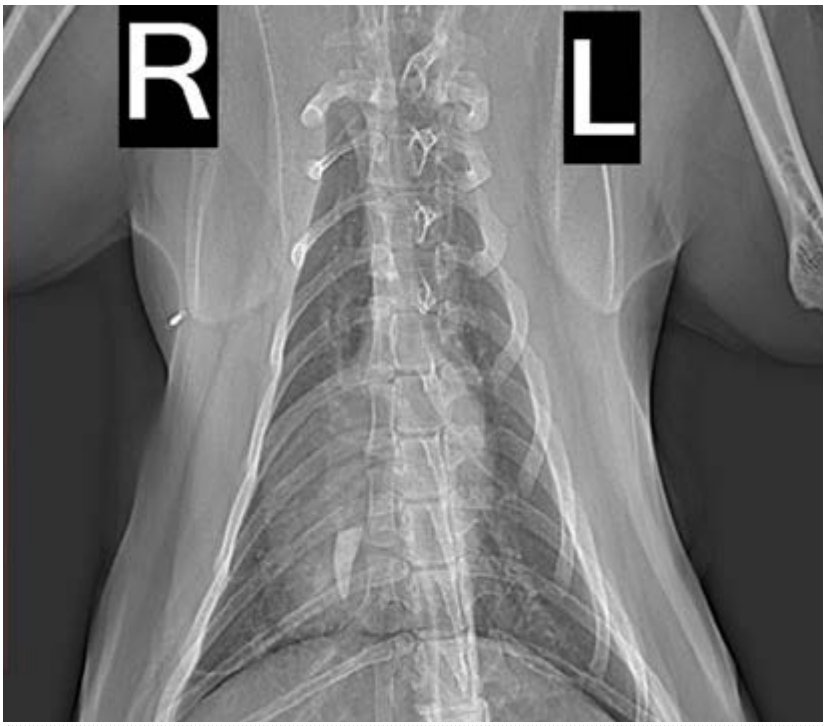
Smudge is a 6-year-old Domestic Shorthair, indoor-only cat with no previous illness. Ten days ago, he fell off his balcony and the owner found him outside on the ground. He ate dinner that afternoon, but over the next 48 hours became progressively more lethargic and inappetant (highly out of character for him).

His owners brought him to the emergency services who identified a mild pyrexia and elevated respiratory rate (60) with a mild increase in expiratory effort.

The following radiographs were obtained resulting in a sharp intake of breath by all (including Smudge).

On closer inspection, Smudge was indeed missing his upper right canine tooth. Smudge was then referred for further management. After arrival, Smudge was placed in an oxygen cage and





Figures 1A and B. These radiographs demonstrate a well-defined mineral opacity superimposed on the right mainstem bronchus, most likely within the bronchial lumen. Importantly there is no evidence of pneumothorax or pneumomediastinum.

given further sedation (methadone 0.1 mg/kg intravenously).

Potential complications that may be associated with tracheobronchial foreign body removal can include pneumothorax and tracheal obstruction necessitating tracheostomy, so all necessary equipment to detail with these complications was set up prior to commencement.

Terbutaline (0.01 mg/kg IV) was given to reduce secondary bronchoconstriction and a conservative dosage of dexamethasone (0.05 mg/kg IV) administered to reduce secondary airway inflammation.

Midazolam (0.2mg/kg IV) was given at induction followed by alfaxalone by titration.

Radiographs were immediately repeated to ensure the foreign body was still in place (it was). The patient's hindquarters and caudal thorax were carefully elevated to try to dislodge the foreign body in a gravity dependent manne; however, no further movement was evident radiographically and this may well have been wishful thinking.

An ET tube was placed and then a Cook airway exchange catheter (8 French) was inserted into the ET tube to provide jet oxygenation into the left bronchus. The ET tube was removed and bronchoscopy commenced with the anaesthesia maintained via alfaxalone and midazolam total intravenous anaesthesia (constant rate infusions with boluses of alfaxalone given if required).

Bronchoscopy was performed using a flexible bronchoscope (Olympus, 5.5 mm insertion tube diameter, a 2 mm instrument channel, 600 mm working length).

The tooth foreign body was identified lodged in the bronchus of the right caudal lobe.

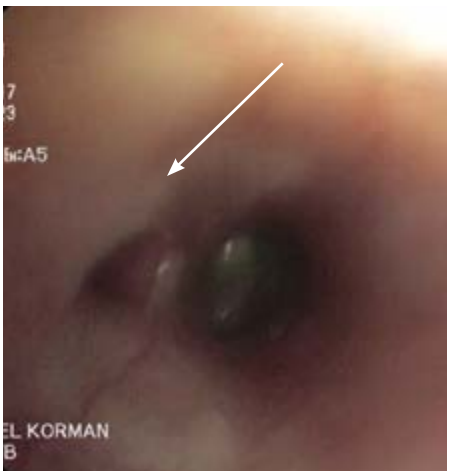


Figure 2. Arrow demonstrates mineralised FB lodged in the bronchi of the right caudal lobe

Attempts were made to grasp the tooth using retrieval baskets, a loop snare and alligator jaw grasping forceps but although a purchase could be made of the foreign body, there was no movement on retraction.

Close monitoring of the patient was maintained throughout the procedure using pulse oximetry, ECG, Doppler and rectal temperature.

If SpO_2 dropped below 90% the bronchoscope was removed and the patient re-intubated and further oxygen administered until oxygenation status improved.

A guide wire (Weasel Wire) was introduced through the bronchoscope which was then removed from the airway, leaving the weasel wire . Weasel Wires have hydrophilic technology and allow atraumatic passage into a body cavity of vessels to then facilitate passage of catheters or other devices.

An 8 French urinary foley catheter with an 8 mm bulb was then passed over the Weasel Wire, whilst currently instilling sterile saline over the guidewire to maintain the hydrophilic behaviour (it acts as a lubricant for the catheter). Attempts were made to pass the bulb of the catheter caudal to the foreign body under direct visualisation. The aim was to partially inflate the balloon behind the foreign body (FB) and then slowly advance the catheter to encourage mobility of the foreign body and draw it aborally.

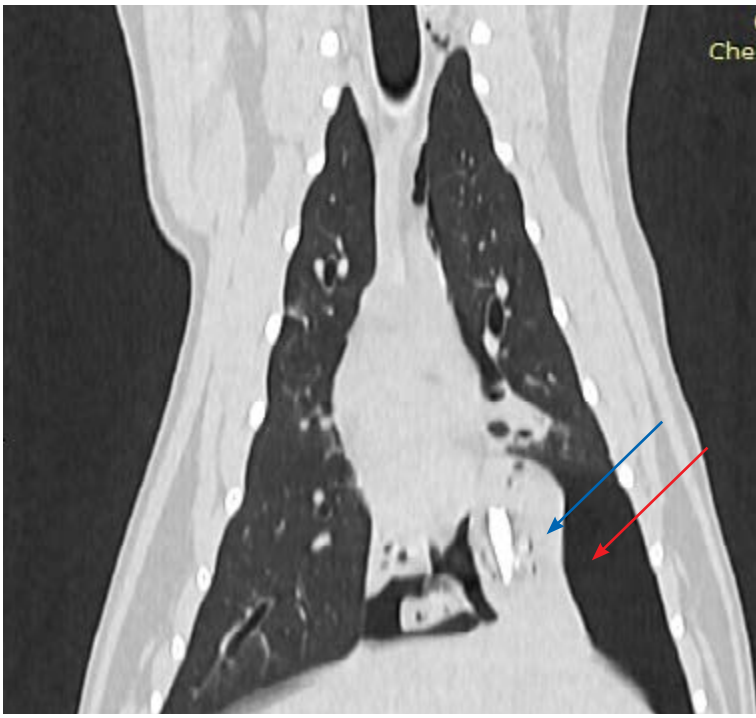


Figure 3. CT of the patient following attempts at bronchial FB removal. The blue arrow highlights the tooth, while the red arrow demonstrates a pneumothorax in the right hemithorax with atelectasis associated with right caudal lung lobe.

Unfortunately, the catheter could not be passed caudal to the FB, as it was extremely well wedged within the bronchus. During the last of these attempts Smudge's oxygenation saturation dropped and after intubation remained around 90%. A CT was performed which revealed a pneumothorax. This was drained via thoracocentesis and the patient again stabilised. After discussion with the owner, thoracotomy was performed.

With gentle palpation the surgeon was able to identify the tooth within the right caudal lung parenchyma but still no movement of it could be encouraged. The site of the leak causing the pneumothorax could not be identified.

Right caudal lung lobectomy was performed.

A chest drain was placed (Mila 12 Fr) and Smudge was hospitalised for a further 72 hours. After 24 hours, the chest-drain was non-productive and was removed. Smudge received fentanyl for analgesia, mirtazapine as an appetite stimulant (he ate within 24 hours) and broad-spectrum antibiotics (amoxicillin 22 mg/kg IV q 8 hours and metronidazole 10 mg/kg IV q 12 hours). His recovery was uneventful and he was discharged with a short course of amoxicillin clavulanate for possible secondary infection from the FB and analgesia (buprenorphine and meloxicam oral suspension) as he was eating well.

Although uncommon, tracheobronchial foreign bodies occur in cats. As with most diseases feline, clinical signs are often vague and with cough being the most common presenting sign. In a recent review of 12 cats with tracheobronchial foreign bodies (Leal et al JFMS 2017) only 25% were presented for acute respiratory distress and the majority of cats showed clinical signs for over a week. Stridor was only found in cats with tracheal foreign bodies and wheezing with bronchial foreign bodies. This is probably because most of the foreign bodies are small and not obliterating the entire tracheal or bronchial lumen.

Reported foreign bodies in cats include mineralised material such as small pieces of bone (likely reflecting their nature as hunters), teeth and vegetal material such as grass, bark and grass seeds. Vegetal material such as grass seeds have the potential for migration and subsequent pyothorax development. The shape of the tooth in Smudge's case likely encouraged a deeper migration into the bronchial tree.

In 12 cases of cats with tracheal FB, 10/12 were successfully removed using forceps and fluoroscopic guidance (Tivers et al JSAP 2006). Two of the 12 cats had the foreign body removed via bronchoscopy. All of these cats had their foreign body cranial to the carina.

In the case series of 12 cats with tracheobronchial foreign bodies (Lea et al JFMS 2017) 83% of cats had the foreign body successfully removed via bronchoscopy and 2/12 required a surgical approach after bronchoscopic removal was unsuccessful, similar to Smudge.

Another technique that could have been considered with Smudge was the passage of an arterial embolectomy catheter (e.g. Fogarty Fortis) such as those used in interventional techniques in human medicine to remove emboli and thrombi from arteries. Barium can be added to the catheter tubing to provide radiopacity under fluoroscopic guidance. The advance of this type of catheter over a Foley catheter would be the lower profile of the balloon and the longer length that would allow passage alongside or through the bronchoscope.

Tracheobronchial foreign bodies in cats are uncommon and not all cats demonstrate respiratory distress. Various techniques can be attempted for foreign body retrieval and if these are unsuccessful then partial lung lobectomy can result in a good outcome.◆

Figure 4. The problematic tooth

Figure 5. Smudge recovers post-operatively. Note a soft Elizabethan collar was removed prior to photography.

Winner

Entitled to a CVE$100 voucher

Small

RECREATIONAL DRUG TOXICOSIS IN COMPANION ANIMALS

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C&T No. 5974

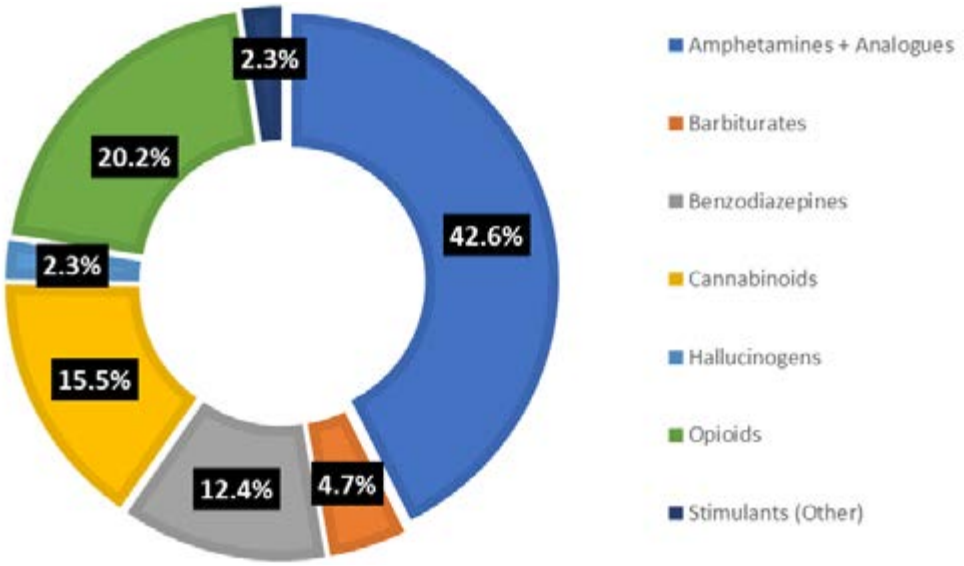
Management of companion animals that have been poisoned by recreational drugs can be challenging. Owners are often reluctant to admit possession of these substances or may be unaware that an exposure has occurred. Furthermore, as these substances are often unregulated they may contain undeclared pharmacologically active substances. Affected animals frequently exhibit moderate to severe signs of toxicosis, and optimal treatment of these patients requires a sound understanding of the mechanisms of toxicity and management options available.

The Australian Animal Poisons Centre is frequently contacted for assistance regarding animals that have been exposed to recreational drugs. In 2022, approximately 2.4% of all cases handled by the service involved these substances. Of these cases, 45.0% involved stimulants (including amphetamines and their analogues or non-amphetamine stimulants such as cocaine), 52.7% involved depressants (including opioids, cannabinoids, benzodiazepines and barbiturates) and 2.3% involved hallucinogens.

As a definitive diagnosis through a diagnostic laboratory cannot be made within a reasonable timeframe to facilitate clinical decision making, a presumptive diagnosis is often made based on clinical signs and a history of drug access.

Human urine drug screens (UDS) may be used by veterinarians to support the diagnosis of illicit drug intoxication; however, clinicians should be aware that these are not validated for use in small animals and both false positives and false negatives may occur.

This article discusses the mechanisms of toxicity, clinical presentations and management of common recreational drug intoxications.



Animal Poisons Centre Recreational Substance Cases (2022)

STIMULANTS

Amphetamines & Analogues

Amphetamines and their analogues are used both recreationally and therapeutically by humans. Commonly abused illicit forms include 3,4-methyl enedioxymethamphetamine (also known as MDMA, ecstasy and molly) and methamphetamine (meth, crystal meth, speed and ice to name a few). Legal forms include prescription medications used for the management of attention deficit disorders (dexamfetamine, lisdexamfetamine and methylphenidate), prescription medicines used for weight loss (phentermine and the now discontinued sibutramine) and over the counter products used as decongestants (pseudoephedrine and phenylephrine).

Mechanism of Toxicity

These substances cause sympathomimetic effects by stimulating α and β adrenergic receptors. They increase the release of catecholamines such as noradrenaline and dopamine as well as serotonin at peripheral and central neuronal synapses. **Signs of toxicity are predominantly a result of central and peripheral adrenergic stimulation which results in both cardiotoxicity and neurotoxicity.** An increase in the release of serotonin can also result in serotonin toxicity.

Clinical Presentation

Clinical signs are usually seen within 30 minutes to 2 hours of exposure, but may be more delayed when a modified release formulation is involved. Common clinical signs include hyperaesthesia, agitation, vocalisation, circling, head bobbing, mydriasis, tremors, hyperthermia, tachycardia, hypertension and potentially seizures.

Management

Gastrointestinal (GI) decontamination can be cautiously considered for animals without clinical signs that present soon after ingestion. Inducing emesis is not recommended in animals with established clinical signs, however gastric lavage under general anaesthesia may be warranted for animals that have recently ingested large doses and have established clinical signs. As seizures may occur, activated charcoal is not without risk; however, it could be offered to neurologically appropriate animals that present soon after ingestion. Repeat dosing of activated charcoal may be of benefit following the ingestion of modified release preparations.

Patients should be placed in a quiet, dimly lit area of the clinic to minimise excitation. Crystalloid intravenous fluid therapy should be considered and may provide thermoregulation, aid in renal drug clearance and protect the kidneys from myoglobin. Phenothiazines such as acepromazine are the preferred drug of choice for controlling central nervous system (CNS) signs and high doses may be required in some cases. Benzodiazepines have the potential to result in paradoxical CNS excitation and should therefore generally be avoided. Patients with persistent and significant tachycardia despite adequate sedation may benefit from the cautious administration of a beta blocker such as propranolol or atenolol. Tremors can be managed with methocarbamol, whilst seizures can be managed with phenobarbitone or levetiracetam if available.

As amphetamines also possess serotonergic activity, the addition of cyproheptadine could be considered as a specific anti-serotonergic therapy.

Close monitoring of blood pressure, heart rate and rhythm, body temperature and CNS status is required. Patients that experience severe tremors, seizures or prolonged hyperthermia should be monitored for the development of rhabdomyolysis and disseminated intravascular coagulopathy. Patients may require hospitalisation for up to 72 hours depending on the severity of toxicity and the formulation of the product ingested.

Cocaine

Cocaine is a non-amphetamine stimulant drug derived from the leaves of the coca bush (*Erythroxylum coca*). The extract is processed to form the three different forms of cocaine: cocaine hydrochloride (white powder), freebase (a powder form that is purer than cocaine hydrochloride), and crack cocaine (crystals). Cocaine is commonly also referred to as coke, crack, blow and snow. Small animal intoxications usually occur following accidental ingestion at home or by law enforcement dogs during search operations.

Mechanism of Toxicity

At lower doses, cocaine is a potent stimulant that inhibits catecholamine reuptake at sympathetic nerve terminals. It increases levels of adrenaline, noradrenaline, serotonin and dopamine, resulting in increased stimulation of the sympathetic nervous system. At higher doses, cocaine causes blockade of myocardial sodium channels which can be associated with significant cardiotoxicity. Cocaine is lipid-soluble and is well absorbed through all mucous membranes, including the nasal and oral cavity, digestive system and alveoli.

Clinical Presentation

Clinical signs develop very rapidly, usually within 10–15 minutes of exposure. Typically, clinical signs manifest as CNS stimulation which is usually followed by CNS depression that may result in coma. Catecholamine excess results in hyperactivity, hyperaesthesia, tachycardia, hypertension, hyperthermia, tremors and seizures. Other clinical signs that can occur include ataxia, mydriasis, vomiting and hypersalivation. Myocardial sodium channel blockade can delay ventricular depolarisation, resulting in malignant ventricular dysrhythmias.

Management

Due to the rapid onset of clinical signs, induction of emesis is rarely indicated. Gastric lavage under general anaesthesia is a safer method of GI decontamination for patients with recent ingestions of large quantities of cocaine.

Symptomatic and supportive care includes maintaining body temperature as hyperthermia is a factor most closely associated with poorer outcomes. CNS, cardiovascular and acid-base status should be monitored. Patients should be placed in a minimal stimuli environment where possible. Crystalloid intravenous fluid therapy will help maintain thermoregulation, aid in drug elimination, support perfusion and prevent dehydration. Seizures can be controlled with benzodiazepines or, in refractory cases, with phenobarbitone or levetiracetam. In some cases, induction of general anaesthesia will be required. Cyproheptadine may be used when severe serotonergic signs are present.

Intravenous sodium bicarbonate may be useful in treating cardiotoxicity secondary to sodium channel blockade. In case of life-threatening tachyarrhythmias, β-blockers can be considered although they should be used cautiously. Intubation and assisted ventilation may be required in cases with significant CNS depression. In cases with severe toxicity, intravenous infusion of lipid emulsion may decrease the bioavailability and clinical effects of cocaine, given it is lipophilic.

DEPRESSANTS

Opioids

Opioids are a group of natural and synthetic drugs that are commonly prescribed for the management of pain in both human and veterinary medicine. Common opioids include buprenorphine, codeine, fentanyl, methadone, morphine, oxycodone and tramadol. Exposures in small animals are commonly due to ingestions of human pharmaceuticals and of transdermal fentanyl and buprenorphine patches.

Mechanism of Toxicity

The toxic effects of an opioid are determined by its binding affinity to the central and peripheral opioid receptors and whether it acts as a full or partial agonist at the receptor sites. The major toxic effects (CNS and respiratory depression) are mediated via interactions with μ opioid receptors. Opioids act as agonists at these receptors reducing ventilation by blunting the responsiveness of the medullary chemoreceptors in the respiratory centre, resulting in profound respiratory depression. Opioids are well absorbed from the GI tract, but bioavailability is variable as some opioids have a significant first pass effect. Cats are deficient in glucuronyl transferase which accounts for their relative sensitivity to opioids.

Clinical Presentation

Exposure is associated with an opioid toxidrome where patients initially present with tachypnoea, hypersalivation, vomiting, defecation and urination. This may progress to ataxia, CNS depression, respiratory depression, hypotension and hypothermia. Cats may present with CNS excitation and hyperthermia. The duration of toxicity is dependent on the pharmacokinetics and formulation of the individual product. Toxicosis associated with modified release preparations or substances with long half-lives may be prolonged.

Management

Initial management of animals that present soon after ingestion and without clinical signs may include inducing emesis followed by the

administration of activated charcoal. In cases where respiratory depression is evident, administration of oxygen and establishing a patent airway may be necessary. Ventilatory support may also be needed. Monitoring for hypothermia and the provision of active warming if required is essential.

Naloxone is indicated in animals exhibiting CNS and/or respiratory depression. It is a pure μ antagonist which reverses the effects seen in toxicosis; however, it has a short half-life compared to most opioids. Its action will only last for about 45-60 minutes and multiple doses may be required during the course of the intoxication. Partial opioid agonists/antagonists, such as butorphanol, may be used to partially reverse pure agonists if naloxone is unavailable.

Cannabinoids

The main psychoactive compound found in cannabis is tetrahydrocannabinol (THC), which is found in dried flower buds, leaves and stems of the female *Cannabis sativa* plant. Illicitly obtained synthetic cannabis may contain other compounds. Most exposures occur when companion animals, most commonly dogs, have ingested some of the owner's supply or cannabis edibles. Some edibles (such as brownies and cookies) may pose an additional risk due to the additive effects from theobromine. THC intoxication has also been reported following the suspected ingestion of human faeces containing THC.

Mechanism of Action

THC acts as an agonist at the cannabinoid receptors CB_1 and CB_2. CB_1 receptors are located in the CNS and clinical effects of THC are believed to be the result of its action on these receptors. CB_2 receptors are primarily located within the immune system and serve a role in immune and inflammatory functions. Dogs have a higher concentration of CB_1 receptors in their brainstem and cerebellum compared to other species, which makes them more susceptible to the effects of THC.

After ingestion, THC is almost completely absorbed from the GI tract. As THC is an extreme lipophile, it has a large volume of distribution and readily distributes to the CNS and other fatty tissues. This contributes to a prolonged elimination half-life and duration of toxicosis.

Clinical Presentation

Clinical signs are usually seen within 30-60 minutes of ingestion and generally last for 24-72 hours (up to 5 days in severe cases). Exposure affects the CNS, cardiovascular system and the GI tract. Cannabis affected dogs are classically described as being depressed or ataxic and dribbling urine. Other signs such as disorientation, hyperaesthesia, tremors, mydriasis, bradycardia (but also tachycardia), hypotension, vomiting, hypersalivation and hypothermia are also commonly seen. In cases of significant toxicity seizures may occur.

Management

Without a witnessed exposure, clinical signs consistent with THC exposure and a positive UDS can support the diagnosis of THC intoxication. However, dogs excrete a large number of THC metabolites in their urine which can result in UDS false negatives.

Induction of emesis can be considered in animals without clinical signs that present soon after ingestion. However, as THC has antiemetic properties, attempts at inducing emesis may not be successful. Activated charcoal can be given to neurologically appropriate animals and multiple doses may interrupt the enterohepatic circulation of THC.

Management is primarily supportive, with provision of IV fluids if required to maintain hydration and manage hypotension if it occurs. Agitation can be managed with phenothiazines or benzodiazepines. As THC is an extreme lipophile (estimated to be ~870,000 more soluble in lipid phase over aqueous phase at physiological pH), IV lipid emulsion therapy ('lipid rescue') may be helpful in the treatment of severely affected patients.

Benzodiazepines

Benzodiazepines are pharmaceuticals usually prescribed for the management of insomnia and anxiety. Commonly abused benzodiazepines include diazepam, oxazepam, clonazepam and alprazolam. Australia is seeing an increase in the presence of novel benzodiazepines as

individuals look for new drug experiences or street alternatives to controlled prescription medicines. Unfortunately, very limited information is known about these novel drugs and their effects may be more severe and unpredictable than currently approved prescription products.

Mechanism of Toxicity

Benzodiazepines act by potentiating the effect of gamma-aminobutyric acid (GABA), a major inhibitory neurotransmitter in the CNS, by acting as positive modulators on $GABA_A$ receptors. Benzodiazepines bind to the BZD binding site on $GABA_A$ receptors, promoting the influx of chloride ions. This action blocks the ability of the neurons to generate a nerve impulse resulting in CNS depression.

Clinical Presentation

Ingestion of these substances is unlikely to result in life threatening effects in an otherwise healthy animal, and with good supportive care the prognosis is excellent. Common clinical signs seen include CNS depression, lethargy, ataxia and disorientation. In approximately 40-50% of exposures, paradoxical excitation, agitation and aggression occurs. Toxicity is dose dependent, and following large doses, there is a risk of hypotension, hypothermia and coma. In poisoned animals, clinical signs begin to develop within 30 minutes of ingestion, and these signs typically resolve within 12-24 hours, depending on the substance involved.

Management

Management is largely similar to previously mentioned depressant drugs and may include GI decontamination in selected cases and supportive care. Benzodiazepines are rapidly absorbed and GI decontamination should only take place with recent ingestions and if clinically safe to perform. Whilst a single dose of activated charcoal could be administered, often it is not required due to the limited severity of intoxication that typically occurs.

Patients should be placed in a quiet warm cage and monitored for hypothermia and respiratory depression. Intravenous fluids therapy can be used as clinically required to support blood pressure, maintain perfusion and correct dehydration. If paradoxical agitation occurs, sedation can be achieved with acepromazine or butorphanol. In rare cases of severe CNS or respiratory depression, flumazenil (a benzodiazepine antagonist) can be used to reverse signs if available.

Barbiturates

In human and veterinary medicine, barbiturates are used as sedatives, anticonvulsants, and in the past, were also used as anaesthetic induction agents. In recent years, barbiturates have been largely replaced by benzodiazepines. Small animal barbiturate intoxications usually occur following the accidental ingestion of human or veterinary prescription medications and less commonly from the ingestion of carcasses of animals that have been euthanised with pentobarbital.

Mechanism of Toxicity

Barbiturates are general depressants of nerve and muscle tissue. They bind to $GABA_A$ receptors, increasing the duration of chloride channel opening and thereby enhancing the inhibitory effects of GABA. Barbiturates also block the excitatory neurotransmitter glutamate.

Clinical Presentation

Barbiturate toxicosis can result in profound CNS and cardiorespiratory depression. High doses depress cardiac contractility, potentially resulting in end organ hypoperfusion. The most common clinical signs following exposure are weakness, ataxia, recumbency, hypothermia, hypotension, bradycardia, respiratory depression, and coma (which can be prolonged following exposures to long-acting barbiturates). The onset of clinical signs varies from 15 minutes to several hours, with a duration of up to several days.

Management

Decontamination for recent exposures, monitoring and symptomatic and supportive care form the basis of management. GI decontamination should only be initiated in patients without clinical signs and when the exposure is recent. For animals exhibiting severe depression, emesis is contraindicated due to the risk of aspiration. Gastric lavage under a general anaesthetic will often be required.

Monitoring of neurological status, body temperature and respiratory rate/effort are essential. Intubation and assisted ventilation may be required in cases where severe respiratory depression occurs. Intravenous fluid therapy will help maintain adequate blood pressure, support cardiac and renal function, and assist with renal excretion of the drug.

Barbiturates undergo enterohepatic circulation and therefore repeated doses of activated charcoal may result in a more rapid recovery, particularly when long-acting barbiturates are involved. Use of intravenous lipid emulsion in

severe intoxications may help by decreasing the bioavailability of the highly lipid soluble barbiturates. Urinary alkalinisation with sodium bicarbonate and haemodialysis have also been shown to be effective in enhancing the elimination of barbiturates in significant intoxications.

HALLUCINOGENS

LSD (Lysergic Acid Diethylamide) and psilocybin are both psychedelic drugs which are used recreationally for their hallucinogenic effects. LSD is a semi-synthetic drug synthesised from a substance found in ergot. LSD is most commonly found as a solution dried onto gelatine sheets, pieces of blotting paper and sugar cubes. Common street names for LSD include acid, blotter acid, dots, lucy and window pane.

Psilocybin is the main psychoactive substance in magic mushrooms. When ingested it is metabolised into psilocin by the liver, which exerts neurological effects similar to LSD. There are many different types of magic mushrooms. In Australia, they are often known by their common names which include golden tops, blue meanies and liberty caps.

Mechanism of Action

LSD and psilocybin exert their toxicological effects via increased serotonergic stimulation. LSD is an agonist at serotonin receptors. It is believed that its effects may be mediated primarily via activity at 5-HT_{2A} receptors in the cerebral cortex. LSD also causes an increase of glutamate release in the cerebral cortex, which may contribute to some of its effects. Both psilocybin and its active metabolite have a structure similar to serotonin. They bind with high affinity at 5-HT_{2A} receptors and to a lesser extent at 5-HT_{1A} receptors.

Clinical Presentation

Clinical signs for both LSD and psilocybin are secondary to serotonergic stimulation and include mydriasis, sedation or hyperesthesia, tachycardia, hyperthermia, aggression, vocalisation, ataxia, nystagmus, tremors and potentially seizures.

Trauma caused by altered behaviour is usually the greatest and most immediate risk. For LSD, clinical signs usually appear within 90 minutes of ingestion. For psilocybin, clinical signs usually occur within 30-60 minutes and rarely can be delayed up to 3 hours post ingestion. The effects of both LSD and psilocybin may last up to 12 hours.

Management

Treatment is symptomatic and supportive. Emesis is recommended only if the patient presents before clinical signs appear. Patients experiencing hallucinations may become extremely aggressive and care should be taken when handling them. Affected animals should be placed in a padded cage in a dark, quiet environment. Severe agitation can be managed with phenothiazines or benzodiazepines. Monitoring for hyperthermia and tachycardia is required. IV fluids may be considered as a supportive measure.

HELP

If you require advice for an animal that has been exposed to a recreational drug or other toxin, you can phone the **Animal Poisons Centre on 1300 869 738**. The Animal Poisons Centre provides 24/7, rapid, up-to-date and evidence-based advice to veterinary teams. For more information visit animalpoisonscentre.com.au

References

Alcohol, Tobacco & Other Drugs in Australia (2022) Australian Institute of Health and Welfare. Available at: https://www.aihw.gov.au/reports/alcohol/alcohol-tobacco-other-drugs-australia/contents/drug-type

Armstrong J. Pascu, O. (2022) *Toxicology Handbook* 4th Edition. Chatswood: Elsevier Australia.

Bischoff, K. (2018) "Toxicity of drugs of abuse.," in R.C. Gupta (ed.) *Veterinary toxicology: Basic and clinical principles.* 3rd edn. London, United Kingdom: Elsevier/Academic Press, pp. 385–406.

Brauer, C. ***et al.*** (2009) "Barbiturate intoxication in two dogs confirmed by toxicological urinalysis, *"Journal of Small Animal Practice*, 50(8), pp. 423–425. Available at: https://doi.org/10.1111/j.1748-5827.2009.00749.x.

Chemaxon Bioinformatics Software.

Crecraft, C., Prittie, J. and Mastrocco, A. (2021) "Hypoglycemia and presumptive rhabdomyolysis secondary to lisdexamfetamine toxicosis in 3 cats," *Journal of Veterinary Emergency and Critical Care*, 32(1), pp. 113–118. Available at: https://doi.org/10.1111/vec.13150.

De Briyne, N. ***et al.*** (2021) "Cannabis, cannabidiol oils and tetrahydrocannabinol—what do veterinarians need to know?," *Animals (Basel)*, 11(3), p. 892. Available at: https://doi.org/10.3390/ani11030892.

Drug facts. The Alcohol and Drug Foundation. Available at: https://adf.org.au/drug-facts/#list.

Freed, E. (2019) "Illicit drugs: what veterinary nurses need to know," *Today's Veterinary Nurse*, pp. 26–31.

Hovda, L.R. et al. (2016) *Small animal toxicology*. Ames, IA: Wiley Blackwell.

Kisseberth, W.C. and Trammel, H.L. (1990) "Illicit and abused drugs," *Veterinary Clinics of North America: Small Animal Practice*, 20(2), pp. 405–418. Available at: https://doi.org/10.1016/s0195-5616(90)50035-2.

Lauinger CA, Peacock R. Marijuana toxicosis in dogs in Melbourne, Australia, following suspected ingestion of human faeces: 15 cases (2011-2020). *Aust Vet J.* 2022 Mar;100(3):90-97.

Micromedex Healthcare Series. Electronic Dataset, Merative US L.P, USA.

Oster, E. *et al.* (2023) "Intoxication of dogs and cats with common stimulating, hallucinogenic and dissociative recreational drugs," *Veterinary and Animal Science*, 19, p. 100288. Available at: https://doi.org/10.1016/j.vas.2023.100288.

petTOX Animal Poisons Centre database case records.

Puschner, B. and Wegenast, C. (2018) "Mushroom poisoning cases in dogs and cats," *Veterinary Clinics of North America: Small Animal Practice*, 48(6), pp. 1053–1067. Available at: https://doi.org/10.1016/j.cvsm.2018.06.008.

Riahi-Zanjani, B. (2014) "False Positive and False Negative Results in Urine Drug Screening Tests: Tampering Methods and Specimen Integrity Tests," *Pharmacology Online*, 1, pp. 102–108.

Rossi, S. (ed.) (2023) *Australian Medical Handbook*. AMH Pty Ltd. Available at: amh.net.au

Royle, K. and Bandt, C. (2020) "Intravenous lipid emulsion to treat suspected cocaine toxicosis in a dog., "*Canadian Veterinary Journal*, 61(1), pp. 49–52.

Saitman, A., Park, H.-D. and Fitzgerald, R.L. (2014) "False-positive interferences of common urine drug screen immunoassays: A Review," Journal of Analytical Toxicology, 38(7), pp. 387–396. Available at: https://doi.org/10.1093/jat/bku075.

Smith, M.R. and Wurlod, V.A. (2020) "Severe rhabdomyolysis associated with acute amphetamine toxicosis in a dog," *Case Reports in Veterinary Medicine*, 2020, pp. 1–4. Available at: https://doi.org/10.1155/2020/2816373.

Stern, L. and Schell, M. (2018) "Management of attention-deficit disorder and attention-deficit/hyperactivity disorder drug intoxication in dogs and cats," *Veterinary Clinics of North America: Small Animal Practice*, 48(6), pp. 959–968. Available at: https://doi.org/10.1016/j.cvsm.2018.07.007.

Thomas, E.K., Drobatz, K.J. and Mandell, D.C. (2014) "Presumptive cocaine toxicosis in 19 dogs: 2004-2012, "*Journal of Veterinary Emergency and Critical Care*, 24(2), pp. 201–207. Available at: https://doi.org/10.1111/vec.12159.

Trinder, R. *et al.* (2022) "The use of haemodialysis for the treatment of phenobarbitone intoxication 30 hours after ingestion," *Veterinary Record Case Reports*, 10(3). Available at: doi.org/10.1002/vrc2.395.

Approaching the Acute Encephalopathic Patient

Read article outlining how to approach and treat a suspected toxin ingestion here:

veterinary-practice.com/article/approaching-the-acute-encephalopathic-patient

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ANXIETY IN A FRENCH BULLDOG

Kate Drew

Hurlstone Park Vet Hospital

C&T No. 5975

Lulu became a patient of the clinic in 2018. She had been adopted as an adult dog at approximately 3.5-years-old and had raised at least one litter. She had been desexed in the months prior to her first visit to us.

Initial Treatment

As part of a routine examination, a colleague prescribed gabapentin 100-200mg BID in December 2018 as the client was concerned about `anxious behaviours' (not specified). An Adaptil collar was also recommended.

By March 2019, Lulu's owner at that time reported that Lulu was barking at visitors and had become `protective' at home, even to the owner's housemates.

A colleague thus recommended a full behaviour consult and trainer for Lulu.

In February 2020, Lulu's previous owner requested behaviour medication after moving house and noticing an increase in Lulu's anxiety.

Lulu Was Diagnosed With Separation Anxiety.

Treatment recommended included:

- Restarting gabapentin (which the previous owner had ceased using);
- Crating to manage her destructive behaviour when she was left alone as she was already crate-trained;
- Monitoring with a webcam;
- Installing an Adaptil diffuser; and
- Keeping departures and homecomings low key

Also contemplated were:

- Referral for a behaviour consultation; and
- Trialing trazadone

After this visit, Sydney went into COVID-19 related lockdowns and Lulu's owner worked from home for several months. During this time, Lulu obviously did well and treatment with gabapentin continued.

In October 2020, the previous owner needed to return to the office, so a plan was implemented to practice mock departures, gradually increasing the amount of time that Lulu was left alone, whilst monitoring her with a webcam.

Lulu was then seen only for vaccination appointments until September 2022.

My Involvement

I first saw Lulu in September 2022. She had been recently rehomed into a household with two teenage children, one of whom has been diagnosed with ADHD.

Problems reported by her new owner were:

- Barking at visitors; and
- Barking/rushing at her teenage son when he bounced his basketball

The client noted that her son had ADHD, which sometimes made his behaviour explosive or erratic. The client was concerned as her son was becoming frustrated with Lulu's behaviour; it was affecting their relationship, such that he wanted Lulu to be rehomed.

Initial consultation

Lulu was no longer receiving gabapentin therapy when I first saw her.

She was fearful with handling by strangers (freeze, fight responses), so a full examination could not be performed at this first visit.

On distance examination, I noted that Lulu had bilateral nuclear sclerosis, along with an old corneal scar that may impact her vision. It was

Contents

noted that she had salivary staining of her feet, and that she had a history of allergic skin disease. She weighed 10.4kg. I understand that this is not a perfect approach, but I felt that it was best for Lulu to build confidence with me and in the clinic before proceeding with touching her. The client understood this approach and provided consent. I had the physical examination findings from vaccination consultations to rely upon in the interim.

After a long discussion about fear and anxiety in dogs, The Fear and Anxiety Scale (FAS), the 4 Fs (fiddle, freeze, flight and fight) and reading dog body language (which included using posters to demonstrate signs of stress in dogs), we formulated a phased plan to manage the home environment to reduce stress on Lulu and the family.

Phase 1 Was Initially Focused on Reducing Exposure to Triggers for Lulu, Including

1. Dealing with visitors:
    a. Lulu would be set up in a comfortable room behind a closed door before visitors arrived.
    b. The room was to have background music playing, such as classical or talkback radio.
    c. Visitors were to be given instructions that if Lulu was not able to be put in a room with a closed door before they arrived (as the teenage children often bring home friends unannounced), they were to ignore her and not attempt to interact with her. They could drop treats for her onto the floor if she was near them or throw them to get her to move away. No-one was allowed to try and touch her.
2. Basketball:
    a. As basketball could not be avoided as the client's son was initially unwilling to stop playing, Lulu would be set up in a comfortable room with company (the client), behind a closed door before the son went to play basketball.
    b. The room was to have background music playing, such as classical or talkback radio. Basketball was then able to be played.
    c. The client noted that Lulu would tremble when the son bounced the basketball after initiating this plan but was able to eventually settle down. She did not bark or rush at the doors or windows.
3. Improving the relationship with the teenage son:
    a. Make the son the most valuable person in the house by being the person responsible for doing all the 'fun stuff' with her like feeding, walking and games.

The 4Fs of Fear—Four Fear Responses

Flee	Freeze	Fidget	Flight
Creep away	Hold completely still so the scary thing might not see them	Hyperactivity	Bark
Move away	Freeze for a few seconds	Increased panting	Lunge
Walk away	Walk slowly like walking through Jello	Excessive jumping	Forward motions
Run away	Learned helplessness if staying still for long periods of time when afraid	Highly distracted	Growl
Find a way to escape		Goofy	Snarl
Trying to hide		Calming signals	Showing teeth
Try to avoid the potentially scary thing		*Sniffing*	Hackles raised
Cowering		*Looking away*	Body tense
Tail tucked		*Moving away*	Ears alert and forward
		Excessive blinking	Tail high and stiff
		Scratching	Biting
		Yawning	
		Lip licking	
		Overgrooming	
		Lifting a paw	
		Whale eye	

welfare4animals.org/blog/the-4fs-of-fear-fear-responses

b. Get the family to 'buy into' our plan, as it was not likely to work if all stakeholders, particularly her son, did not participate.

I recommended that the client book an appointment with a behaviour vet as Lulu's case was quite severe and likely outside of my skill set which the client subsequently did.

Whilst awaiting this appointment, the client had a trainer that was recommended by the behaviour vet come to the home and work with the family. .

Follow Up

Il next saw Lulu in November 2022. The client reported:

- Lulu tended to react fearfully whenever visitors arrived, irrespective of whether she had met the person before.
- The trainer had recommended throwing treats over Lulu's head when visitors arrive to try to stop her from barking, but it was not working. I reiterated that Lulu should not have the opportunity to interact with visitors at this stage of our treatment plan at all.
- Lulu and the client's son were still having difficulty. There had been a concerning incident where Lulu had chased her son around with an open mouth/teeth bared.
- Lulu was calm with her son when he was calm but would become reactive when he was heightened due to his ADHD (such as when he would come out of his room with a booming voice).
- Her son did not want to participate in feeding, walking or playing with Lulu.

I was able to perform a physical examination on Lulu at this visit with the assistance of the client, who placed the stethoscope onto the thoracic wall so that I could auscultate the heart and lungs from a distance. The client also lifted Lulu's lips so that the teeth and gums could be inspected and placed a thermometer into the inguinal fold so that an approximate temperature could be taken. I added 0.5°C to the reading on the thermometer. I learned this approach to temperature taking from a USYD nurse whilst on rotation in my final year of university. I use it daily for anxious or rambunctious patients when taking a rectal temperature is not possible or not good for the patient's wellbeing.

Updated Plan

Due to the difficulty in managing Lulu's environment (i.e. visitors arriving) and interactions with her son, a decision was made to include fluoxetine (10mg PO SID) and trazadone (3mg/kg) to the treatment regimen.

I start my fluoxetine patients on a slowly increasing dose starting with one quarter of the final dose in order to reduce side effects. I find that if patients experience side effects in those first few weeks of treatment, particularly inappetence and sedation, clients lose enthusiasm and confidence and may not continue with therapy. For Lulu, this meant a dose of 2.5mg PO SID for 4 days, increasing to 5mg for a few days after that and if no side effects were noted, and increasing to 10mg PO SID within 7-10 days. The dose for trazadone was chosen in order to reduce the chance of serotonin syndrome as it was being used in combination with fluoxetine. Gabapentin therapy continued whilst the fluoxetine took effect in the subsequent 4-6 weeks, and we planned to wean this off by reducing the dose by half every few days after trazadone treatment had started (as this is a compounded medication and needed to be ordered especially for the patient).

A Plan Was Made to Reduce Interactions Between Lulu and the Client's Son by:

- Having Lulu set up in a room with a closed door before her son arrived home from school. This gave her son time to decompress and use the house freely before any interaction.
- Once the son had retired to his bedroom, Lulu would be allowed to roam the house freely.
- If the son intended to come out of his room, he was to text or call out to the client so that Lulu could be set back up in her own space behind a closed door.
- Continue having Lulu set up in a room behind a closed door with background noise when the son intended to play basketball. The client noted that Lulu was coping better with this, especially if she sat with her at this time.

Week 6 Check-in

A medication review occurred in mid-December 2022. It had been 6 weeks since therapy with fluoxetine had started.

- The client reported an overall improvement in Lulu's behaviour as well as the relationship between Lulu and her son.
- Lulu was noted to only bark twice when the son played with the basketball. The client continues to sit with Lulu while he plays to help her cope.
- Lulu will now sit on the son's lap and their interactions are much more positive. The client's son is much happier.
- A plan was made to continue with management of Lulu's environment as described above.
- Gabapentin will be weaned after the Christmas and New Year period when there would likely be

changes to the family's routine or more visitors than usual.

- Lulu was more interactive and her body language was significantly more relaxed than on previous visits to the clinic. She approached me to interact and allowed me to pet her for the first time.
- Overall, the client reported being happy with the progress that Lulu has made.

Most Recent Visit

- I last saw Lulu in later January 2023 for her annual vaccinations and a behaviour check in.
- I was able to perform a full physical examination, noting that Lulu took treats throughout the examination and preferred to be examined on the client's lap rather than the treatment table.
- A plan was made to reduce the gabapentin to every other day for a few weeks and then cease.
- Management of Lulu's skin was noted to be an ongoing issue, and Cytopoint and Malaseb bathing were being used at this point in time.

THE CURRENT OWNER'S PERSPECTIVE

Update April 2023

Lulu's behaviour has improved noticeably in the last few months. I feel that fluoxetine has been key to her transition when paired with our efforts at behavioural modification. When she is home with just our four family members she is happy and relaxed at all times. She will still bark when a visitor comes over but if one of us holds her, she is quiet and complacent. In this situation, I will often put her in another part of the house and when the visitor leaves, she will bark again but not if we hold her.

Our son played basketball yesterday for the first time in a long time. I put Lulu and our other dog in their sleeping space where the noise of the basketball can still be heard. She did not bark but she did scratch at the door to be let out. I did not respond and she quietened down. When my son finished his basketball playing, he returned inside the house and Lulu did not bark at him. She seems to have accepted the noise as non-threatening.

I am so happy that we persevered with making adaptations to our home life to make room for Lulu. Although I will be honest, the levels of stress in our household were very heightened by her arrival and the first few months were very hard at times, as it completely change our family dynamic.

Although it was only temporary, my relationship with my son was adversely affected by my decision to adopt Lulu. Also, my relationship with my husband was strained at times as he felt I was favouring Lulu over my son.

There were times when it was so bad that I almost entertained the idea of giving her up but I feared that would just add to her lifelong history of trauma and I could see that we were her last resort.

It is now 10 months since we adopted Lulu. She has formed a very strong bond with my husband who works from home. In hindsight, he now agrees that the adjustment period was worthwhile. My son also now has a strong bond with Lulu, he will often pick her up and hold her and he happily sits with both dogs in front of the TV.

Although it has been a bumpy ride over the last 10 months, we are all happy to have Lulu as a family member. Our original dog is also very happy to have a constant companion and his life is greatly improved because of her.

They spend all day everyday together and gets lots of attention and walks around the neighbourhood which also contributes to my husband's wellbeing and my own.

COMMENT COURTESY OF

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She has worked in Canberra and Sydney since graduation, owning a large multi vet practice for almost 20 years. She currently works in a behaviour practice, Canberra Animal Behaviour Solutions.

Isabelle has been actively involved in further veterinary behaviour education. She has been a tutor for the CVE Distance Education Veterinary Behaviour course for the last 4 years and is the current president of the Australian Veterinary Behaviour Medicine group (formerly known as AVBIG). She is a certified Fear Free and Stress Free veterinary practitioner and is passionate about helping our pets and their owners create a stress-free home, as well as educating vets about behavioural medicine.

Lulu, the French bulldog's case, highlights how complex behavioural cases can be, and how there can be multiple behavioural and medical diagnoses that all need attention and management. It can be a difficult journey diagnosing the medical and behavioural problems; navigating the owners' ability and desire to change environmental triggers; finding medications that are effective and ensuring the clients are on board; practicing calming, relaxation exercises and ensuring that we look for and recognise the small wins. Behavioural change can take months to years, and client communication is vital throughout this journey. This can be particularly difficult within the confines of a routine 20-minute general practice consultation.

Lulu appears to have multiple anxiety-based issues, as well as potential medical ones. French bull dogs have frequent spinal/orthopaedic and gastro-intestinal issues that may contribute to reactivity and aggressive responses. When considering any sudden change in behaviour, particularly aggressive responses, especially in an older dog, we need to consider a medical condition that may be causing pain, such as arthritis, spinal disease, dental disease, skin allergies, ear infections etc.[1,2,3] Behavioural issues, like any medical disease, requires a comprehensive history, an examination, assessment, diagnosis and plan. Behaviour questionnaires are a vital tool when working through the often long and complex history. Video footage is also essential, especially when dealing with separation related issues. It is difficult to know what the animal is doing or feeling when the owner is absent. Diagnoses in veterinary behavioural medicine are also essential. However, due to different terminology used in different countries, this can be a difficult and complex area. As the field of veterinary behavioural medicine is growing, we are seeing more scientific research emerging all the time that is helping to grow our knowledge and at times challenging previous diagnostic terms. Without a diagnosis, it is difficult to effectively manage and treat behavioural disease.

A great example of the evolution of terminology is the term 'separation anxiety'. Separation anxiety is currently more typically called separation related behaviour disorders or problems due to complexity of this syndrome.[4] Separation associated distress usually manifests as destructive behaviour, usually occurring near the site of the owner's departure; vocalisations such as barking; and/or toileting/elimination accidents. There can be many causes for these behaviours, and we need to have a better understanding of the emotional state of the pet when left alone. Anxiety disorders are associated with emotions such as fear, frustration and panic.[4]

For a diagnosis of separation related anxiety to be made, other causes of these behavioural signs, such as inadequate house training, puppy chewing, playing, or barking at specific stimuli, must be ruled out.[5]

Dogs behave the way they do due to a complex interplay of genetics; learning and experiences; and the environment (both internal and external) they live in. When wanting to change a dog's behaviour, we also need to use a multi-factorial approach. In behavioural medicine, we use **the 4M approach**:

- Environmental **M**anagement—providing predictability and avoiding triggers for anxiety
- Behaviour **M**odification—reward spontaneous relaxed and calm behaviour and put on cue; desensitisation and counterconditioning
- Using **M**edications to reduce anxiety
- **M**onitoring progress.

There are no magic pills in veterinary behavioural medicine. Medication can help provide a better platform for the brain to think, focus and learn new behaviours.

Veterinary behavioural medications can be roughly grouped into 4 groups.

- **Non-drugs** such as Adaptil® pheromones, Zylkene®, diets such as Royal Canin Calm®, other food supplements etc.
- **Long term 'background' medications** to help mood stabilise and provide a brain that has 'space' to learn. They help provide a platform where the dog can focus better. This group includes the anti-depressants such as selective serotonin reuptake inhibitors (SSRI's) such as fluoxetine (Prozac®, Zactin®), sertraline, paroxetine for example, and tricyclic antidepressants (TCA's) such as clomipramine (Clomicalm®, Clomav®). It can take 4-6+ weeks to see the full effects of these medications.
- **Immediate acting and adjunctive anti-anxiety medications.** These may be used as an adjunct to the background medications, situationally (e.g. vet visit) or as a 'rescue' medication with unexpected events. They help to reduce the immediate anxiety.
- **Treatment of underlying medical issues** such as orthopaedic issues, skin disease, gastro-intestinal disease. Disease is usually associated with behavioural change, and we need to ensure that we look for underlying medical issues that may be contributing to behavioural change rather than just assuming that it is a primary psychological issue.

There is a common misconception that immediate acting/situational medications will smooth the transition when starting as SSRI, while the background medications are taking effect. They do not target the same receptors as the long-term background medications such as the SSRI's and have different effects. Commonly used immediate acting, situational medications include benzodiazepines such as diazepam (Valium®) and clonazepam (Paxam®); clonidine (Catapres®), gabapentin, pregabalin (Lyrica®) etc. We are fortunate that Sileo®, an oromucosal dexmedetomidine gel, registered for noise sensitivities, is being launched in Australia this year, adding another fast-acting drug to the toolbox. These medications work in a number of different ways, targeting different receptors.

Trazodone is a commonly used medication in Australia, despite its cost, lack of registration (human or veterinary) and the need to be compounded (a legal grey zone). Trazodone is an atypical antidepressant used to treat anxiety and depression by acting as a serotonin antagonist/reuptake inhibitor (SARI). It increases serotonin levels by blocking presynaptic reuptake in humans and presumably in dogs and cats. It can also have a sedative/hypnotic effect at higher doses. It is most effective as a hypnotic, often used as a sleeping medication for humans. It needs to be used with some caution, particularly at higher doses, when used with an SSRI or TCA, due to the potential to cause serotonin syndrome.[6] Often, it is used for the sedative effect rather than being the most effective anti-anxiety medication. Although trazodone can be a useful medication, there are many cheaper and more effective immediate acting anxiolytic medications.

Medications certainly can help, but as this case highlights, ongoing appropriate environmental management and behaviour modification is vital. We need to reduce the triggers that the dog is exposed to, or at least try to reduce the intensity of the triggers. Routine, calm predictable interactions are also important. Avoid any form of punishment to reduce the dog's anxiety.

Another vital component of behavioural change is behavioural modification. We want to change the emotional response that the dog has when encountering a situation that makes her anxious, and when she is over-aroused. We do this by teaching her to be relaxed more often, and ultimately, we want to try to train her to relax on cue. We are teaching her to calm down and giving her an opportunity to think before acting. These exercises help the brain to 'retrain' or 're-wire' into a more relaxed place more of the time. We are using the principle of neuroplasticity—we can

change the wiring of the brain by practicing using the pathways that we want to fire more frequently i.e., practicing calm behaviour. This means that the part of the brain wiring becomes stronger and more likely to be the 'go to' wiring, just like going to the gym to improve muscle bulk and strength.

Behavioural change is a long-term work in progress. As this case highlights all the components of the behaviour plan need continuous updating.

References

1. Mills, Daniel S., et al. "Pain and problem behavior in cats and dogs." *Animals* 10.2 (2020): 318.
1. Bécuwe-Bonnet, Véronique, et al. "Gastrointestinal disorders in dogs with excessive licking of surfaces." *Journal of Veterinary Behavior* 7.4 (2012): 194-204.
1. McAuliffe, Lindsay R., et al. "Associations Between Atopic Dermatitis and Anxiety, Aggression, and Fear-Based Behaviors in Dogs." *Journal of the American Animal Hospital Association* 58.4 (2022): 161-167.
1. De Assis, Luciana S., et al. "Developing diagnostic frameworks in veterinary behavioral medicine: disambiguating separation related problems in dogs." *Frontiers in Veterinary Science* 6 (2020): 499.
1. Blackwell, Emily Jayne, Rachel A. Casey, and John WS Bradshaw. "Efficacy of written behavioral advice for separation-related behavior problems in dogs newly adopted from a rehoming center." *Journal of Veterinary Behavior* 12 (2016): 13-19.
1. Sueda, Karen Lynn C., and Julie Cho. "Trazodone can reliably and safely induce sedation and anxiolysis in dogs and cats, making it a useful pharmacologic agent beyond the treatment of behavior problems." *anxiety* 1: 3.

◆

2 COMMENTS

Aine Seavers

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C&T No. 5976

C&T No. 5966 Feline Leprosy

Could the change in the cat's ears be a fixed drug eruption as a result of the use of Clarithromycin?

1. Clarithromycin has a 1% incidence of alopecia - did the ears go bald then the pigment appear or did the hair fall out and the pigment found?[1]

2. Re the origin of the black lesion? Why not the Clarithromycin? In humans, this drug does cause FDE - fixed drug eruptions - the lesions fade away like a charcoal sketching of black pigment.[2] I suspect cats are no different given that black area on this cat's nose. This wouldn't mean one would not use the drug in this presentation, just means the origin of the said lesion would be understood.

1. ehealthme.com/ds/clarithromycin/hair-loss/
2. Malkarnekar SB, Naveen L. Fixed drug eruption due to clarithromycin. *J Res Pharm Pract.* 2013 Oct;2(4):169-71. doi: 10.4103/2279-042X.128152. PMID: 24991627; PMCID: PMC4076927.

Editor's comment: Any drug can cause a fixed drug eruption. You can diagnose this by withdrawing the drug and seeing if the lesion is clear, then giving the drug again. In vet circles, Dr Ken Mason is an expert and has published about this entity. Certain drugs have a reputation for doing this — and often there is a fixed anatomical area involved. I can remember for example that diethyl carbamazine (DEC; used as a daily drug to prevent heartworm) caused scrotal lesions. Ouch! In this case, the cat had feline leprosy, which in Victoria is usually caused by *M. lepraemurium* or *M. tarwinense*. Untreated, this can be a terminal disease. Five drugs are known to have some efficacy: rifampicin, clarithromycin, pradofloxacin, clofazimine and ethambutol. Of these – clarithromycin is the safest and has the best toxicity profile. Cats often develop side effects during therapy. Some are bad enough to merit discontinuation of therapy. The side effect in this cat is trivial. The changes observed were not sufficient to merit swapping to a different drug e.g. azithromycin.

C&T No. 5968 Cutaneous Xanthomatosis

Xanthomata often disappear by irritation to the area i.e. Mechanical irritation to trigger a foreign body reaction, such as by cautery on the edges of the lesions, microneedle roller scarification, electrolysis or by linear incision with extraction of yellowed fat underneath. I am aware of several that persisted for up to 8 years on parts of the body that didn't have trauma so it can be a very long wait for them to disappear. As such, in this case, I suspect the biopsy mechanics may have triggered the self-cure, rather than the 4 days of hyporexia. Many sufferers of xanthomata have zero lipid serum abnormalities, so a diet change will not benefit those patients. However, in this case it would be interesting to know if that Black Hawke cat food contained a very high level of fat or fat from a source the cat would not normally be used to digesting. In such a case, then changing the diet to a normal or lower fat diet would be beneficial in this patient when it might not always be so.

Editor's note: The best treatment of cutaneous xanthomas is to make sure serum levels of cholesterol and triglycerides are not elevated, and let the body deal with the lesions by normal tissue remodelling by macrophages. In this instance, it seems plausible that the biopsy somehow sped up lesion resolution. It is also possible the lesions were secondarily infected and the marbofloxacin was helpful. In this case, follow-up testing of blood for triglyceride levels was not done, but the diet was changed, and the cat has had no recurrence of lesions.◆

Read C&T No. 5966 & 5968 in the eBook.

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Large

ANAEMIA, WEAKNESS & DEATH IN SOME LAMBS IN THE MALLEE

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C&T No. 5977

Introduction

Some young stud sheep from the Mallee area died having been seen breathless and weak. The concerned owner contacted local vets and investigations concluded that a blood parasite was most likely responsible.

Experienced producers often accept small numbers of deaths in their livestock without investigation and common explanations include 'pulpy kidney', 'grain poisoning' and so on.

In this case, the producer requested and obtained assistance immediately, and having a diagnosis, can now affect remedies. Diagnosis is not always straightforward and is usually achieved via a combination of the skills of producers, veterinarians and laboratories.

History

A stud producer in the SA Mallee noticed 6, 6 to 8-week-old lambs weak, lagging behind and then found dead over a period of 2 to 3 weeks in July 2022. The lambs were observed with extreme pallor and breathing difficulties prior to death. They had been treated with 'weaner guard' (containing moxidectin and clostridial vaccines), a few weeks previously.

A freshly dead lamb was presented for necropsy at a local vet clinic and a freshly dead lamb was autopsied by the owner and photographs sent to the consulting vet, and a further lamb was examined and blood tested on farm.

Before results were fully available at the lab, an initial diagnosis (based on the vet post-mortem examination) was pneumonia, and a recommendation made that further cases should be treated with antibiotics (Oxytetracycline 300mg/mL; long acting; Alamycin LA) and treated for internal parasites. One affected lamb was treated this way and appeared to recover after about 3 days.

Results from samples summary below

Two lambs were tested, and the results from necropsy indicated that pneumonia was a possible diagnosis in lamb 1. Unfortunately, the spleen was not collected for examination, although it was noted as slightly enlarged at post-mortem examination.

A second lamb was sampled on farm and displayed a profound anaemia, with sheet-white mucus membranes:

Red cell count—1.67,(range 9—1 5)

HCT—0.09 (range 0.27—0.45)

Plasma pepsinogen 38.9U/L (5-10 normal)

PCR positive for *Mycoplasma ovis*

Zero FEC (faecal egg count)

Discussion

This case demonstrates the benefit of repeated and persistent investigations in sheep cases where a diagnosis is not obvious. Although in the first case the pathologist interpretation was pneumonia—based on observed changes in the lungs—this did not fit the clinical picture, history or observations.

The second case showing profound anaemia (blood loss) and very high pepsinogen indicated that abomasal parasites were possibly contributing. This lamb seemed to take a while to recover, and the assumption was that this could be related to profound anaemia or abomasal damage, or both. *Mycoplasma ovis* is the diagnosis here, possibly complicated by internal parasites.

In early August more weaner lambs were observed breathless, pale and struggling to keep up with the mob—all symptoms of *M. ovis* infection, and possibly internal parasites also. One lamb also had 'dark coloured urine'.

The previously affected lambs treated with a long acting oxytetracycline injection and *Ivomec* drench all appeared to have responded well to this treatment, so this treatment was used with these lambs, which responded similarly.

Since *M. ovis* has not been previously diagnosed on this property, it is possible that the 2021 summer, where a much larger population of mosquitos was recorded in SA and other States, allowed the introduction and spread into this flock. Mosquitos are recorded as vectors, although it is suggested that 90% of flocks in southern Australia are infected.[1]

Contents

Mycoplasma ovis (previously *Eperythrozoonosis ovis*) is described in texts as a widespread infection from surveys done in Southern Australia, but no cases have been reported in recent years in SA. The disease causes profound anaemia (as in this case), sometimes icterus (jaundice) and haemoglobinuria (dark urine) as in this case. Transmission is described as through biting insects and mechanical, so the use of vaccination syringes could allow spread through a flock, since infections can be transmitted by reusing needles during herd immunization and as a very small number of infected erythrocytes is sufficient to transmit disease. It has also been suggested that blood on tools during shearing or ear tagging may contribute to spread.[2]

Oxytetracycline is a recommended treatment for these infections but additional recommendations are to avoid stress in affected mobs and only treat severely affected lambs. Merino sheep are reported as being more severely affected than other breeds or crossbreds.[3]

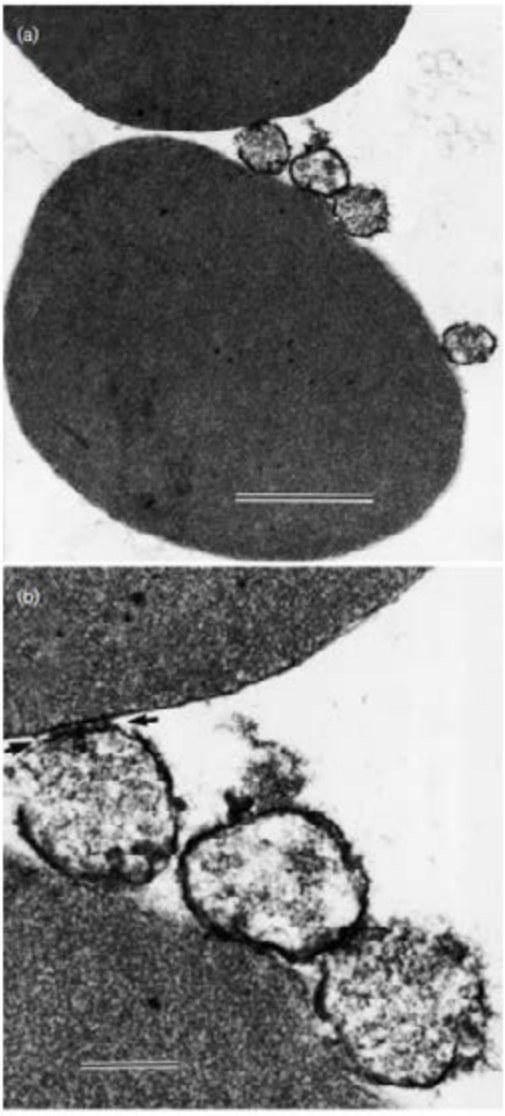

Figure 1. Electron micrographs of a thin section of infected sheep erythroctes. (a)Wall-less bacteria are located on the surface of erythrocytes. The surface of some organisms is partly surrounded by an electron dense material. (b) Enlarged view. In some instances, fine fibrils can be seen to bridge between the bacterium and the erythrocyte surface (arrows). Bars, 0.1µm (a), 0.2µm (b).

References

1. *The Practice of Sheep Veterinary Medicine* Kym Abbott University of Adelaide Press 2005
2. Harold Neimark, Brent Hoff , and Martin Ganter *Mycoplasma ovis* comb. nov. (formerly *Eperythrozoon ovis*), an epierythrocytic agent of haemolytic anaemia in sheep and goats. *International Journal of Systemic and Evolutionary Microbiology* (2004), 54, 365-371
3. Dr Colin Trengove, University of Adelaide, SA – pers comm

Editor's Note

For some reason, it is often the largest, fastest growing lambs which are affected. This is an example of a haemotropic mycoplasma infection, very similar conceptually to feline infectious anaemia due to Mycoplasma haemofelis based on the pathophysiology in cats and other species. Mosquitoes are actually considered an unlikely vector, as are fleas and other biting parasites, and it is instruments used for lamb marking which are more likely to be the culprit.

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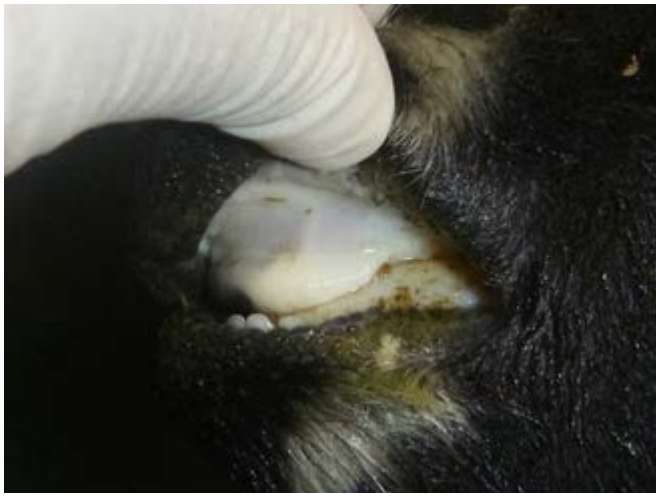

Figure 2. Anaemic lamb with pale mucus membranes

Haemotropic *Mycoplasma ovis* is recognised as an emerging zoonotic pathogen with human infection been reported in pregnant women, immunocompromised patients, people working with animals and in those exposed to arthropods.

From a production perspective, jaundice in affected lambs leads to condemnation of carcasses at abattoirs.◆

Contents

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Reference

8698.0 - Waste Management Services, Australia, 2009-10

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Small

WHAT'S YOUR DIAGNOSIS?

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C&T No. 5978

Opal is a 7-year-old domestic short hair that has been coming to our clinic since she was 2-years-old. She was adopted from the RSPCA as a kitten by her current owners.

When Opal was 3-years-old her owners commented that she wanted to lie around a lot more and had stopped chasing the birds. Opal was an inside/outside cat but only had access to a secure back yard. Her owners were retirees who spent a lot of time in the garden with her.

At her 2021 check-up, her owners mentioned again that she was laying around more than normal and she had gained 1.5 kg in the last couple of years. We discussed weight loss. Her owners were not concerned enough to investigate further.

August 2022 Opal presented for Hyporexia and 'just not being right'. It was noted that Opal was particularly sore on gentle lumbar spine palpation and that she was slow to walk around the consult room and sit down. Generally she just looked uncomfortable. She was treated conservatively as an out-patient with methadone, a fentanyl patch, oral maropitant and mirtazapine. At her revisit 2 days later, she was considerably better and her owners thought pain was the main culprit. She was prescribed oral meloxicam with a plan to see her back in a couple of weeks.

Opal was seen again 4 months later. Her owners thought her mobility was good; she was back jumping up on things and doing really well. They had been giving her metacam intermittently and I discussed changing to a lower daily dose over every other day (or less) dosing which would be better for Opal.

In March 2023 Opal presented because she could not stand on her back legs as she would normally do each night to get a treat, followed by dragging her hind left leg while she followed her owners to bed. She was eating and drinking normally at home. In the consultation, Opal was weak in her back legs and would take a couple of steps and then lie down on her side which I found very unusual. Opal looked like she was considerably older than her 7 years but her owners assured me they had got her has a kitten.

Opal was booked in for routine bloods and radiographs.

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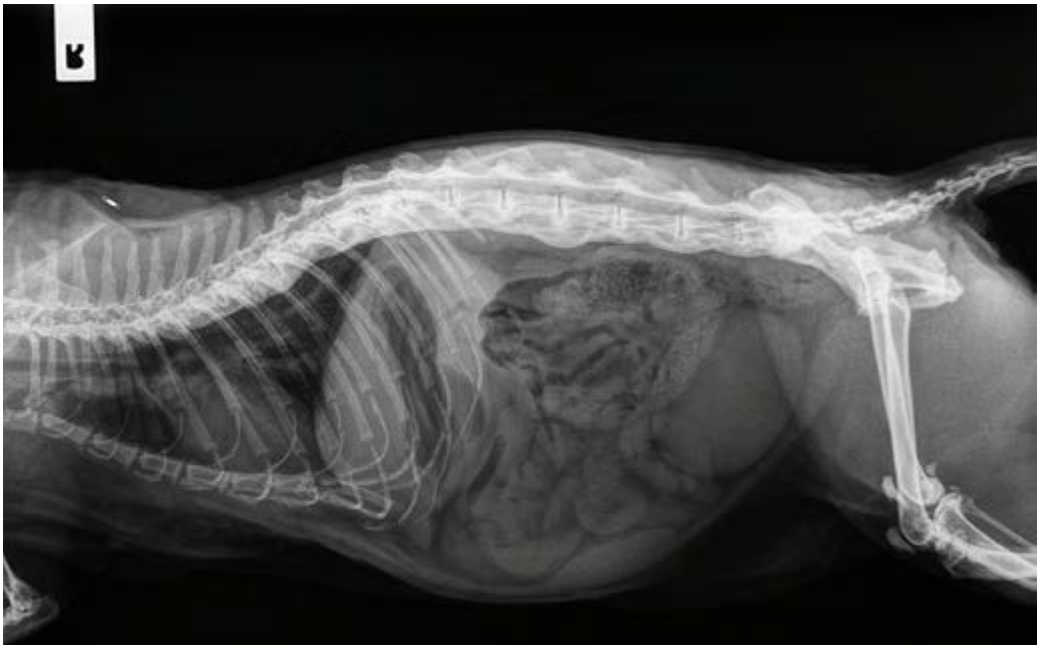

Small

USE OF A CONTINUOUS GLUCOSE MONITORING DEVICE IN FELINE DIABETES MANAGEMENT

Samantha co-tutors the CVE's Feline Medicine Distance Education course.

C&T No. 5979

Continuous glucose monitors are used with increasing frequency in small animal practice to monitor canine and feline diabetic patients, seeming to both offer a solution to the problem of interpreting single blood glucose curves and avoiding serial venepuncture. The devices are generally well tolerated and easy to place, and recent research has shown that they can play an important role in feline diabetic management. This article will discuss this evidence, describe the procedure of placement and complications, as well as explain when such a device can be most useful for the diabetic feline patient.

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Samantha Taylor graduated as a veterinary surgeon in 2002 from the Royal Veterinary College, UK, and spent time in practice before becoming the International Cat Care Resident in Internal Medicine in 2006. She became a European Specialist in Internal Medicine in 2009 and a Royal College of Veterinary Surgeons Recognised Specialist in Feline Medicine in 2010. She currently works at a referral practice in Hampshire and is ISFM Academy Lead and Specialist Veterinary Advisor to ISFM.

Feline diabetics can be challenging to manage. We know that monitoring response to insulin and detecting hyper- and hypoglycaemia are vital to successful control of clinical signs. However, blood glucose curves can be influenced by stress in the veterinary clinic.[1]

To overcome this complication, blood glucose can be measured at home by willing owners,[2] yet even if this is successful, there can be day-to-day variation in the curves that would alter treatment decisions.[3] Continuous glucose monitors (CGMs) offer a solution to the problem of interpreting single blood glucose curves and avoiding serial venepuncture.

About the System

The most commonly used CGM system currently is the 'FreeStyle Libre' (Abbott), which consists of a small sensor disc placed on the animal (Figure 1a), which measures interstitial glucose, and either a mobile phone application or a reader device (Figure 1b) that stores the data from the sensor disc. The device, unlike previous systems, does not require calibration with blood glucose and can take readings for 14 days, although it may not function for this long in cats. The sensor disc can store up to 8 h of data, which is then easily transferred to the phone or reader device.

Accuracy of the FreeStyle Libre has been assessed in humans comparing favourably with capillary blood glucose, and three recent studies in cats have shown good agreement between interstitial and blood glucose readings.[4–6] However, the time lag for equilibration between blood and interstitium must be considered when there are discrepancies between the results.

Del Baldo *et al* showed that during rapid changes in blood glucose, there can be marked differences between the two compartments, and this should be considered when interpreting results.[5] Deiting and Mischke also comment on certain readings that differed greatly between interstitial and blood glucose, and particularly in certain patients.[6] All non-plausible results should be checked by measuring blood glucose, and trends analysed, taking into account the limitations of the device.

The system is designed for human diabetics, so has preset parameters for that species. The 'Libre 2' system allows for an alarm to be set when interstitial glucose falls above or below certain parameters.

Application of the Device

The vast majority of sensors can be placed in conscious patients, but pushing the sensor on requires a little pressure and for some feline patients, and when starting to place sensors, sedation/anxiolysis with gabapentin or butorphanol may be helpful. If diabetic patients are sedated for other procedures (eg, imaging), the opportunity should be taken to place a sensor. The sensor comes with an applicator device (Figure 2) that is used to place the sensor.

The device can be placed in any location, and studies have used different sites. The dorsal neck can be used cats,[4] or the dorsal thorax caudal to the scapula (eg, see Figure 3).[6,7] It is important to clip the area well, clean to remove dirt (wipes are supplied with the sensor) and allow to dry

Key point

Continuous glucose monitors can provide a more detailed picture of blood glucose levels, making treatment decisions easier.

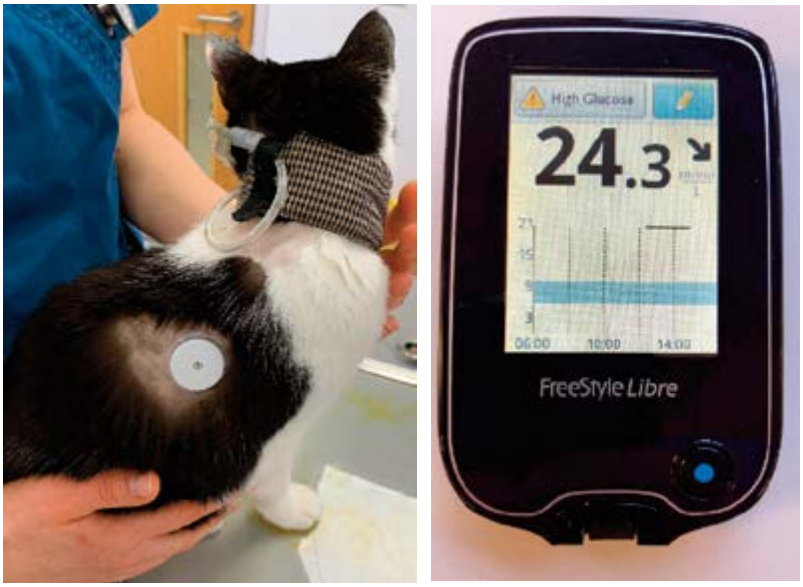

Figure 1: (a) A FreeStyle Libre sensor device on the lateral thorax of a cat. (b) The reader device; an application for the phone can be used instead or in addition. The arrow on the screen indicates if the blood glucose is going up or down, or is stable. Once a sensor has been scanned it can only be read by that reader device and not others

Figure 2: The sensor disc in the blue applicator. The thin catheter in the centre is flexible and sits under the skin (arrow)

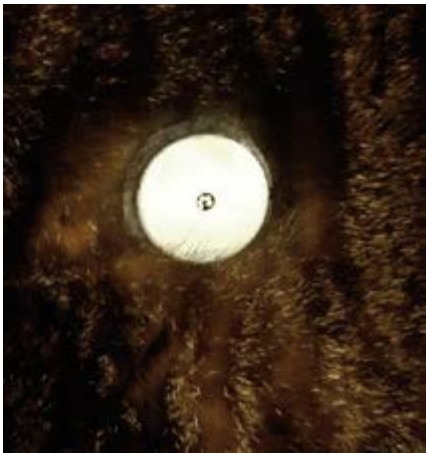

Figure 3: The sensor placed on the cat's lateral thorax caudal to the scapula

Figure 4: Adding additional tissue glue to the sensor disc in small 'dots' around the edge of the adhesive disc34 icatcare.org/felinefocus

completely before application. Although the sensor disc has adhesive, the author adds 'dots' of tissue (cyanoacrylate) glue around the edge of the adhesive area or the edges can 'lift' with time (Figure 4); the same strategy was adopted in the study by Shea and Hess.[4] In one study of 34 diabetic cats, the authors did not use additional adhesive, but secured the edges with sutures, which they report was well tolerated.[6] In the author's experience this is not required.

Patient interference is also uncommon in the author's experience and in general the sensor does not need to be covered. In cats, dressings and T-shirts cause stress and may limit normal behaviour and affect readings. A soft Buster collar, if anything, is used in cats in the author's clinic, although some clinicians cover a sensor placed on the neck, for example, with a dressing.[4]

FreeStyle Libre Sensor Application Tips

- Consider mild sedation if the patient is anxious or you are unfamiliar with sensor placement. Pre-application gabapentin or butorphanol may be adequate.
- Ensure skin is completely clipped, clean and dry before placement of the sensor.
- Apply a few drops of 'tissue' glue around the edge of the sensor adhesive surface, but avoid excessive amounts that could irritate the skin.
- Once the sensor is placed, do not immediately remove the applicator as you may inadvertently remove the sensor too. Keep in position for a few seconds and check the edges of the sensor's adhesive face are stuck to the patient before removing the applicator.
- Try to select an area that has less motion (eg, lateral thorax behind the scapula, lumbar area).
- Avoid 'pet shirts' and adhesive dressings, if possible, as they can cause stress/anxiety and limit normal mobility in cats.
- If using a collar to avoid patient interference, choose a more 'cat friendly' soft collar (Figure 5).

Interpretation of Data

If at home or in the veterinary clinic, the sensor should be scanned every 8 h to upload the data to a phone or reader device. Results can be sent from clients to the clinic using the application, or the reader connected to a computer to upload the data once the required software is installed. Figure 6 shows an example of a report that has various features including daily graphs, average daily glucose and the proportion of time interstitial glucose is within certain parameters (again preset for humans).

Indications for Use of a CGM

The system can be used in clinic on newly diagnosed diabetics looking at the response to insulin and duration of effect (patients can then be discharged with the sensor), cats with diabetic ketoacidosis, unstable diabetics (eg, persistent clinical signs, persistent hyperglycaemia or

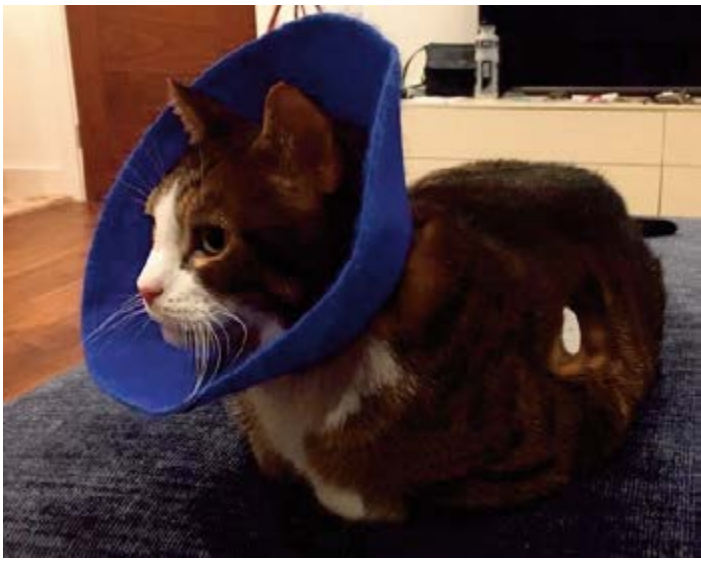

Figure 5: The author tries not to cover the sensors, or use dressings or shirts as they are poorly tolerated in cats. A soft Buster collar can be used to prevent patient interference

hypoglycaemic episodes, suspicion of Somogyi overswing) or those feline patients approaching remission. It could also be used in septic or critical patients where hypoglycaemia is a concern, or patients on insulin or glucose continuous rate infusions, but these indications have not been studied in cats at this time, and the time lag between blood and interstitial glucose must be taken into account.[5] Validation of results with measurement of blood glucose is recommended in such patients before adjusting treatment protocols. The author has found it most useful:

- in cats approaching remission;
- in patients with diabetic ketoacidosis; and
- in animals where stress in the clinic is highly likely to influence glucose curve interpretation and decisions on insulin dose.

Figure 6 shows data from a FreeStyle Libre device on a cat entering diabetic remission.

Complications and Limitations

A recent study,[6] examined the complications of the FreeStyle Libre device in 20 cats (33 sensors placed). Most sensors were placed over the dorsolateral thorax in this study. The most common complications were early sensor detachment (15%) and other complications related to the skin site where the sensor attached, mainly minor

Figure 6: Example of a report provided by the FreeStyle Libre system for a diabetic cat. The grey area reflects ideal blood glucose readings in humans, although these can be adjusted on the reader. This cat is approaching/in remission and hypoglycaemia is noted on day 1 (blue arrow), but also note the period of hyperglycaemia on day 2 (green arrow) that coincided with a trip to the clinic, illustrating the effect of stress on blood glucose, and how decisions made based on those readings could result in increasing the insulin dose.

skin irritation, although two cats had erosion or abscess. The authors of this study felt that additional cyanoacrylate glue applied to the skin-facing surface of the sensor could have contributed to the skin trauma, particularly if the sensor is then prematurely removed. It seems sensible to be cautious with the amount of glue used. Shea and Hess used cyanoacrylate glue in small amounts and found minimal skin irritation.[4]

Hair may be slow to regrow in the area of sensor placement and in pointed cats the fur may grow back a different colour (Figure 7).

Deiting and Mischke noted that a shortened duration of sensor function was common in their patients with premature sensor detachment occurring, leading to an average runtime of 8.3 days.[6] Additionally, Shea and Hess found in their study of cats discharged with sensors, the median time of sensor activity was 7 days. In 80% of placements sensor failure or displacement was reported before the end of the 13-day study period.[4] This high number of sensor failures could be related to the positioning (all on the dorsal neck), suggesting the lower detachment rate in the Shoelson *et al* study (15%) could relate to the placement on the lateral thorax.[7] The sensors are designed for human skin, and in human patients various dressings and bands are available to secure the sensors. Cats are highly mobile, with thin skin, and in debilitated patients the small catheter can easily become dislodged from its position. During the period of sensor placement, hair may regrow and loosen the adhesive. Owners should be warned that premature sensor detachment may occur, and the costs of such a complication and sensor replacement taken into account.

The FreeStyle Libre CGM system has an upper interstitial glucose measurement limit of 27.8 mmol/l and readings above this level are recorded as 'Hi'. This is potentially a limitation in cats with diabetic ketoacidosis, for example, that can be extremely hyperglycaemic, and in poorly controlled cats where that additional information might be useful.

Conclusions

CGM can be a useful tool in the management of diabetic cats. However, data must be interpreted along with clinical parameters indicative of a response (eg, weight, water intake) and limitations of the monitoring system acknowleged.

There may be a discrepancy between interstitial and blood glucose, and particularly during rapid changes in blood glucose when a time lag between the two compartments may occur. The FreeStyle

Libre sensor has been studied in feline diabetics, is well tolerated and has good accuracy. However, premature sensor dislodgement is common and the duration of sensor function is likely to be less than the full 14 days that the sensor functions for in humans.

References

1. Sparkes AH, Cannon M, Church D, *et al.* ISFM consensus guidelines on the practical management of diabetes mellitus in cats. *J Feline Med Surg* 2015; 17: 235–250.
2. Hazuchova K, Gostelow R, Scudder C, *et al.* Acceptance of home blood glucose monitoring by owners of recently diagnosed diabetic cats and impact on quality of life changes in cat and owner. *J Feline Med Surg* 2017; 20: 711–720.
3. Alt N, Kley S, Haessig M, *et al*. Day-to-day variability of blood glucose concentration curves generated at home in cats with diabetes mellitus. *J Am Vet Med Assoc* 2007; 230: 1011–1017.
4. Shea EK and Hess RS. Validation of a flash glucose monitoring system in outpatient diabetic cats. *J Vet Intern Med* 2021; 35: 1703–1712.
5. Del Baldo, F, Fracassi F, Pires J, *et al.* Accuracy of a flash glucose monitoring system in cats and determination of the time lag between blood glucose and interstitial glcuose concentrations. *J Vet Intern Med* 2021; 35: 1279–1287.
6. Deiting V and Mischke R. Use of the 'FreeStyle Libre' glucose monitoring system in diabetic cats. *Res Vet Sci* 2021; 135: 253–259.
7. Shoelson AM, Mahony OM and Pavlick M. Complications associated with a flash glucose monitoring system in diabetic cats. *J Feline Med Surg* 2021; 23: 557–562.

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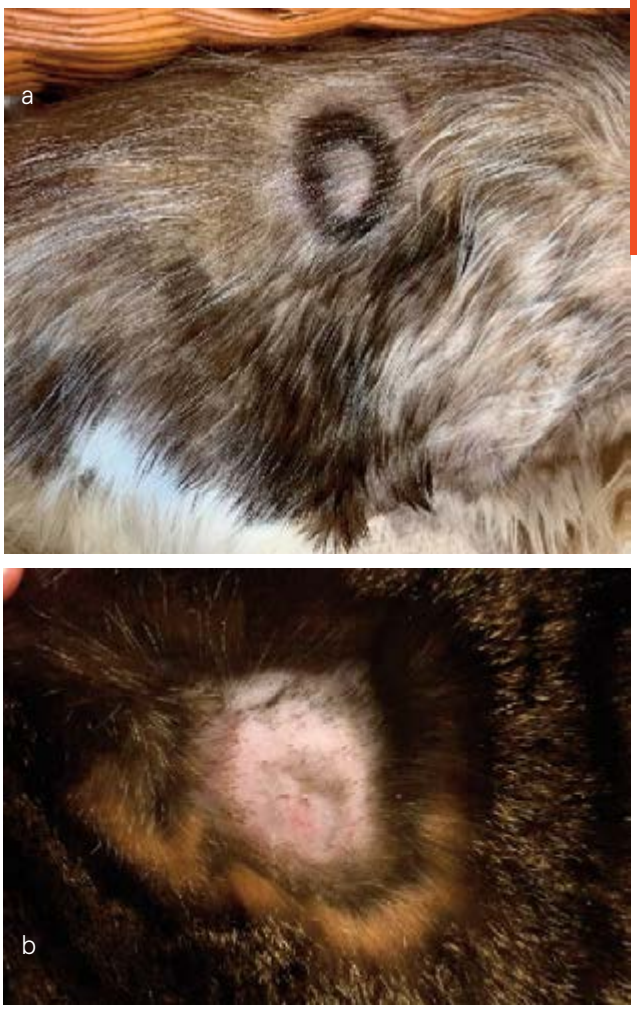

Figure 7: (a) Regrowth of different coloured fur at the sensor site in a cat. (b) An area of alopecia persisting where a sensor was placed a few weeks previously.

RESEARCH ROUNDUP COURTESY OF ISFM

Our Feline Medicine DE Education Partners

C&T No. 5980

Welcome to Research Roundup, where we bring you summaries of the latest feline research. Following this month's Clinical Spotlight review, we look at a large study of common disorders affecting cats in the UK with interesting findings, and follow with the non-medical but important topic of caregiver burden (the strain of caring for sick pets or people). Finally, we have two urinary papers, which answer some questions about upper urinary tract urolithiasis, and urethral obstruction and the COVID-19 pandemic. We hope you enjoy reading our summaries and catching up with the feline literature.

The International Society of Feline Medicine is the veterinary division of the pioneering cat welfare charity International Cat Care. Trusted by vets and nurses, it provides a worldwide resource on feline health and wellbeing, via the Journal of Feline Medicine of Surgery, by fostering an international community of veterinary professionals with a shared vision of feline welfare, and supporting professional development with practical CPD. Additionally, International Cat Care's website provides a valuable resource of accurate information delivering what both vets and cats would want owners to know.

Read more here: cve.edu.au/Common/Uploaded%20files/CT/Research-Roundup-courtesy-of-ISFM.pdf

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Perspective No. 158

THE RECALCITRANT GALLBLADDER

Perspective & Critical Literature Review

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For a trivial little organ that appears to be unnecessary and dispensable, the Gallbladder (GB) often provides a conundrum in management, and disease can be insidious, sometimes providing lethal consequences.

Cholecystitis, Cholelithiasis, Gallbladder Mucoceles, Extrahepatic Biliary Obstruction (EHBO), and Biliary Neoplasia are the main biliary diseases associated with high morbidity and historically high mortality rates. Clinical signs of biliary disorders are often non-specific as well as physical examination parameters; laboratory abnormalities can be confusing – all can have overlapping similarities to other GIT and systemic disorders. Ultrasonography has markedly increased identification of biliary disease, along with cytological sampling. However, we still often are faced with the clinical quandary of when medical management is appropriate or when surgical intervention (cholecystectomy) is necessary.

Gallbladder Function

The GB is essentially a storage vessel for bile during periods of fasting. The canine liver produces bile continuously at a rate of approximately 4-10 mLs/kg/day and the GB's capacity is 15-30 mLs (20kg dog). Therefore, part of the GB's ability to store bile resides in its capacity to concentrate bile by removing water, ions, phospholipids, bile acids and bilirubin in varying amounts. Bile can undergo a 5-to-20-fold concentration within the GB.

Bile is directed into the GB as a result of increased pressure within the common bile duct secondary to contraction of the bile duct and the hepatopancreatic sphincters (formerly the Sphincter of Oddi) during periods of fasting. While there is autonomic innervation, the majority of the stimulus to contract and empty the GB comes from the release of Cholecystokinin (CCK) produced within the duodenal mucosa in response to the presence of ingesta.

Therefore, In addition to storage, the GB is also responsible for determining the constituents of the bile being expressed into the proximal duodenum. Following a meal and the release of CCK, the GB will empty and refill several times, altering the makeup of the bile, especially the composition and concentration of the bile acids and cholesterol.

Composition of Bile

Bile essentially has two main functions; to expel waste products from haemoglobin metabolism and excrete bile acids to aid in the digestion and processing of fats and fat-soluble vitamins. To a lesser extent, bile acids also play a role in protein and carbohydrate digestion. Primary bile acids (chenodeoxycholic acid and cholic acid) are produced within the liver; within the lumen of the gastrointestinal tract these primary bile acids are converted into secondary bile acids by the resident microbiota—the principal role of a bile acid is to act as a detergent and as such, they are toxic to epithelial cells.

The biliary tree protects itself from the toxic components by secreting mucins—large molecules that contain proteins within a nucleus and multiple branching carbohydrate side chains. This structure gives them hydrophilic and hydrophobic zones, allowing them to bind to each other and to bile acids, cholesterol, bilirubin, etc. In large concentrations, the mucins convert from a sol (soluble) to a gel phase which binds to bile, preventing it from damaging the biliary epithelium.

Gallbladder Mucocele

Gallbladder Mucocele (GBM) is an accumulation of semisolid mucins and condensed bile in the GB, producing an ultrasonic image of intraluminal hyperechoic biliary ridges and stripes of inspissated bile in hypoechoic mucous often referred to as a stellate, kiwifruit or starfish pattern; histologically the gallbladder (GB) wall shows cystic mucinous hyperplasia.

Curiously, there is a famine of literature identifying this disease in cats, whereas GBM is the most common reason for biliary surgery in the canine patient.

Recognised risk factors include age (mostly geriatrics), breed (Miniature Schnauzer, Shetland Sheepdog, Scottish Terriers and others), endocrinopathies (hyperadrenocorticism, hypothyroidism) while perhaps significantly, hyperlipidaemia and gallbladder hypomotility have likely received inadequate prior attention in veterinary medicine.

Animal models show there are a number of elements that contribute to the formation of

Julian Lunn BVSc MANZCVS (Small Animal Surgery and Small Animal Medicine) MVetClinStud (Neurology)

Dr Julian Lunn graduated from the University of Sydney in 1996. He spent the first four years in mixed practice in South-eastern NSW (Braidwood) and Bath in the UK.

On returning to Australia, Julian took up a position at the University of Sydney's Veterinary Teaching Hospital working in the Primary Accession and Medicine referral services before commencing a residency in Small Animal Surgery in 2003. He successfully gained membership in Small Animal Medicine in 2003 and in Small Animal Surgery in 2006.

Julian completed his surgical residency in 2007, having spent time at Murdoch University and Colorado State University as part of his training. He also graduated with a Masters of Veterinary Clinical Studies after completing a thesis on Canine Neural Angiostrongyliasis.

In January 2008, Julian joined Veterinary Specialist Services, Australia's largest and most comprehensive veterinary referral service offering Surgery, Medicine, Emergency, Oncology, Cardiology, Dermatology, Ophthalmology and Dentistry. VSS sees over 5000 new patients every year.

In 2018, Julian and his family moved down to Newcastle in NSW to start a new referral practice at the Animal Referral and Emergency Center. As well as a seachange, Julian was able to build up a busy referral surgery practice alongside the Emergency service, continuing to train interns as part of the practice.

As the children approached high school age, Julian and his family elected to move back North of the Border and settle the kids into new schools in October 2021. Julian is looking forward to the challenge of building up a new referral practice for the Darling Downs UQ's Veterinary Hospital in Gatton. Julian is one of the senior clinicians in Small Animal Surgery at UQ Gatton campus and is involved in teaching of undergraduates and supervision of the interns at the Veterinary Teaching Hospital

Julian is the author of several peer-reviewed articles published in multiple journals. He has lectured in several countries including the US, UK and Thailand. He has presented at numerous conferences including WSAVA, Australian College Science Week and the AVA annual conference as well as regularly speaking at online conferences in recent years for Vet Education and Vet Prac.

Through his time at the University of Sydney, Veterinary Specialist Services, and AREC, Julian has gained a wide experience in all areas of surgery from cardiothoracic, oncology and vascular to orthopaedic, neurological and trauma procedures.

Outside of work hours, Julian is kept busy running around after his three young children, two energetic ex-RSPCA dogs and four guinea pigs.

Terry King BVSc MACVSc

Terry, born and bred in Townsville, North Queensland, received his BVSc from University of Queensland in 1975, and Membership of the Australian College of Veterinary Scientists (now ANZCVSc) in 1996 in Emergency and Critical Care.

Terry believes he has been blessed with three major career moves, all in small animal practice—19 fantastic years in general practice on Brisbane's northside, then 7 years as a medical clinician in UQ's Veterinary Teaching Hospital (a time he wouldn't swap for anything) before being extremely fortunate to join Veterinary Specialist Services in 2002, and this has `seen him out'. Terry is not a specialist, but spent the last 30 years as an internal medicine referral clinician with a special interest in the emergency and critical care patient, working alongside bona fide specialists and specialists-in-waiting in all the traditional and emerging veterinary disciplines.

Devoted to the veterinary profession, Terry has strived to repay it and his colleagues for the enjoyment and friendships he's received by serving on various committees and sometimes office bearer on Brisbane Veterinary Practitioner Branch of AVA, AVA Qld, ASAVA, and the ANZCVSc. He has been external examiner, guest lecturer and tutor at UQ's School of Veterinary Science as well membership examiner for ANZCVSc. Terry has been an active participant in scores of meetings and conferences around the country, lecturing in many of these, and has accepted the honour of `getting a guernsey' to speak at WSAVA and FASAVA conferences in many parts of Asia, and one cameo in the USA in a toxicology conference with the topic `Deadly Australians', being able to warn our American cousins on the dangers of drop bears!

Awards Terry treasures (straight to the Pool Room) are the AVA Meritorious Service Award (1991), Life Member of the UQ Veterinary Student's Association (2002), ASAVA Practitioner of the Year (2005), CVE's Tom Hungerford Award for excellence in continuing veterinary education (2016) and Honorary Fellowship of ANZCVS (2018).

Blessed with being able to work and learn with veterinarians all over this country and the World, Terry is adamant that Australian veterinary care is second to none. Terry is a committed family pet practitioner, loves the animal-person bond, and undertakes to prolong it. While all facets of small animal medicine and surgery have progressed enormously over Terry's nearly 50 years of involvement, he believes care of the critical patient as well as palliation (a multidisciplinary approach and involving multi-model therapy) of the chronically (and terminally) ill lead our advancement in family pet medicine.

Terry loves reading, writing and sharing good beer and wine with family and friends, or anyone he can find to fill a glass with. He considers himself a champion of the bar-b-que; loves walking his dogs, swimming (slowly), anything to do with horses having grown up with them, and mowing the lawn with his trusty Victa (his favourite pastime). Did you know that it has been clinically proven that cut grass (freshly mown lawn) releases a chemical called Serenascent that makes people happy and relaxed and prevents mental decline in old age; it works directly on the brain, in particular the amygdala (emotions) and the hippocampus (memory)—Lavidis and Einstein 2006; Spiers *et al*, 2016. As well as the above, on his days off work, Terry cooks dinner, takes out the rubbish and does the household chores, trying to make up for the last 45 years of marital neglect.

mucoceles and choleliths—clearly, hypomotility and hypersecretion of mucins play significant roles. In susceptible individuals there appears to be an excessive absorption of cholesterol through the GB mucosa—this may be a primary event or secondary to stasis. The GB lacks a submucosa and therefore cannot synthesise the lipoproteins necessary for transporting the cholesterol into the venous circulation; it therefore has to be esterified within the muscularis. This process is proinflammatory leading to a degree of muscular toxicity and a decreased sensitivity to CCK. The end result is stasis of the GB. As mucin concentration increases, there is a phase change to the gel form and the bile becomes significantly more viscous which then adds a mechanical element to the stasis and any contraction by the remaining functional muscularis will be less effective in emptying the GB organ.

As the biliary sludge builds, it begins to obstruct the flow of bile through the cystic duct and potentially the common bile duct leading to EHBO. As the gallbladder becomes inflated with the gelatinous material, the resultant GB distension predisposes to necrosis of the GB wall and ultimately rupture with peritonitis the sequelae. While biliary rupture demands immediate surgery, and GB obstructive disease is largely a surgical condition, and it is commonly accepted that surgery is preferred over medical management where there is clinical and biochemical evidence of biliary disease, there remains controversy as to the best approach to treating the more 'silent' or 'immature' GBM.

History & Physical Examination – Hippocrates Revisited

"A thorough and comprehensive patient history often guides the physical examination to the appropriate diagnosis and helps in identifying the appropriate care" - old veterinary saying.

While we reluctantly accept that, in contemporary veterinary medicine, history and physical examination have been largely displaced by the allure of diagnostic testing, a comprehensive history along with a thorough physical examination can prove invaluable, despite the fact that many signs and symptoms are non-specific and occur with numerous abdominal disorders.

The aforementioned risk factors of age, breed, endocrinopathies, and even hyperlipidaemia could be signalling gall bladder disease and non-specific clinical signs such as lethargy, inappetence, hiding, adopting the 'praying position', pacing, ptyalism at mealtimes, vomiting, diarrhoea, whimpering / vocalisation, showing signs of pain on being picked up, etc behest the clinician to at least consider gall bladder disease in the list of differentials.

Intermittent unexplained lethargy and / or sporadic signs of cranial abdominal pain, and diurnal inappetence, especially morning meal refusal with a more normal nocturnal appetite may be signals of evolving GB disease. The hypothesis is that the GB fills overnight with lessened GIT and hence GB activity resulting in a distended organ in the morning; this, plus increased GB pressure especially with attempts to contract against a closed sphincter combine to induce discomfort, nausea, inappetence and sometimes more. Two papers by A.D.Viljoen *et al* in JSAP 2021 explain this supposition more eloquently, albeit describing only 16 & 14 dogs respectively – on the table of literature following. While this is not at pathognomonic for GB disease, it does provide food for thought.

Laboratory Diagnostics

Haematology and Biochemistry do form essential databases in the investigation of most abdominal diseases, but again, often show anomalies which are therapeutically important, but for the most part non-specific for biliary disease.

CBC may be normal or show a neutrophilic leucocytosis or even neutropaenia in the very sick; it is of little help in the sub-clinical and 'silent' GBM situations. Non-regenerative anaemia of chronic inflammation is an uncommon and non-specific finding.

Raised hepatic enzymology is common in biliary disease, with increased ALP in particular, but also possibly ALT, GGT +/- AST. Hypercholesterolaemia is frequently seen and hyperglobulinaemia may occur. Hyperbilirubinaemia and/or hypoalbuminaemia in a clinically ill patient would portend substantial concern. Hyperlipidaemia is sometimes seen concurrently or solely. Specific pancreatic lipase levels complement the diagnostic trail.

Imaging in GB Disease

Radiography, the veteran of veterinary imaging, while being an effective war horse for the diagnosis of abdominal disease, has major limitations in detecting GB disease, being bounded by the presence of air (emphysematous cholecystitis) or radio-impermeable structures (in this case, cholecystoliths). Ultrasound, for now, remains the gold standard imaging technique, the various categorised stages of development from sludge to mucocele are described according to their ultrasonographic images; however, it lacks acceptable sensitivity for recognising GB rupture, at least in the early stages.

The 'new kid on the block', Computer Tomography (CT) is reputed by some to be destined to become the zenith in abdominal imaging, with early case studies claiming similar accuracy in identifying GB disease as Ultrasound, and better accuracy in cholelithiasis cases. It has shown early promise in differential diagnosis of canine patients presenting for acute abdominal signs, including those of biliary origin.

Watch this space!

Medical Therapy

Ultra-low-fat diet, choleretics such as Ursodeoxycholic Acid (UDCA), SAMe +/- antimicrobials along with treatment of associated comorbidities (hyperadrenocorticism, hypothyroidism, hyperlipidaemia) has had few reported resolutions once the biliary mucocele has formed, with numerous reports of non-progressive disease, and just as many recordings of progressive disease requiring cholecystectomy—some clinicians are recommending elective cholecystectomy if medical management hasn't resolved the problem within six months.

URSODEOXYCHOLIC ACID (URSODIOL, UDCA) is a naturally recurring hydrophilic bile acid used in human eastern medicine as a hepatoprotectant for >100 years, and the last ~50 years for dissolution of gallstones (bile from bears). In western human medicine, it has been advocated in the management of various types of chronic inflammatory hepatic disease and this is where veterinary medicine took it on, in the adjunctive management of chronic inflammatory hepatic disease in dogs and cats; the tempering of bile toxicity believed to be its most important role. There is a paucity of evidence of its efficacy in human medicine, even more so in veterinary medicine.

UDCA's Mechanism of Action Reportedly Includes:

a. reduction of intestinal absorption and suppression of hepatic synthesis and storage of cholesterol which is believed to decrease the cholesterol saturation of bile, thereby allowing solubilisation of cholesterol-containing gallstones (not calcified gallstones or bile-pigment gallstones)—this requires a functioning GB to be successful
b. hepatoprotection against toxicity of hydrophobic bile acids in cholestasis via its hydrophilicity, choleresis (increasing bile flow), and immunomodulation
c. inhibition of ileal (distal GI) uptake of the toxic secondary bile acids formed by bacterial modification of primary bile acids in the GIT lumen; this probably has less value in dogs and cats compared to humans due to the higher hydrophilicity of the major circulating bile acids in these animal species
d. protonation of UDCA occurs in secreted bile generating a bicarbonate ion which is absorbed passively by biliary epithelial cells which serves as an osmotic draw for biliary water secretion; the induced choleresis may protect the hepatocytes from copper, leukotrienes, cholesterol, and bilirubin which are normally secreted in the bile
e. immunomodulatory effects are believed to involve reduction in Ig production by B-lymphocytes, reduction in interleukin-1 and -2 production by T-lymphocytes and possibly stimulation of the hepatocytes glucocorticoid receptors

Pertinent UDCA Pharmacokinetics:

a. >90% absorption from SI into the portal circulation and excreted into the bile after conjugation with taurine and glycine; it dissolves rapidly in bile and pancreatic juice hence the administration with food enhances its absorption and is recommended
b. Minimal amounts enter the systemic circulation and is minimally detected in urine
c. Undergoes enterohepatic circulation and ultimately is faecally excreted after GIT bacterial degradation and reduction to less soluble compounds

Adverse Effects Seem Few:

a. Generally well tolerated with anorexia, vomiting +/- diarrhoea only rarely reported
b. Warning about potential taurine depletion in cats (obligate taurine conjugators) with chronic administration
c. Warning about/perhaps contra-indicated in EHBO, cholecystitis, pancreatitis—arguably an important unfavourable consequence of its use
d. Reduced efficacy if used with aluminium-containing antacids which bind to UDCA

S-ADENOSYL METHIONINE (SAMe) sees its most common use in vet medicine adjunctively in a variety of liver disorders including acute hepatic toxicities (e.g. paracetamol, cycad intoxication, xylitol, mycotoxins, mushroom intoxication), chronic hepatopathies (e.g. chronic hepatobiliary disease, hepatic lipidosis, feline triaditis) and patients at risk of hepatotoxicity from medications with a reputation for potential liver toxicity (e.g. the chemotherapy drug Lomustine).

There is minimal robust evidence of its benefits in these situations, especially in veterinary medicine, one small study espousing its use in combination with Silybin to minimise the induction of liver enzymology in dogs receiving CCNU (Lomustine) chemotherapy.

SAMe is an essential factor in 3 major biochemical pathways of most importance in the liver—transmethylation, transsulphuration, aminopropylation—and is essential for the activation and elimination of many substances.

It is important in the synthesis of glutathione (GSH) which is essential for many metabolic processes and detoxification reactions. Folate (Vitamin B9), Cobalamin (Vitamin B12), Pyridoxine (Vitamin B6) are required for the conversion of SAMe to GSH. Exogenous SAMe administration increases hepatic and red blood cell GSH concentrations which in turn inhibits apoptosis secondary to the presence of alcohol or bile salts in hepatocytes. It has anti-depressant activity (hence the name 'Mood Lift' in human nutraceutical medicine) due to increased serotonin turnover and increased dopamine and noradrenaline release.

SAMe is poorly absorbed from the GIT and food reduces this absorption further. Once absorbed, it enters the portal circulation and is metabolised in the liver.

It appears safe in animals with few adverse effects—vomiting, diarrhoea, flatulence, constipation.

Even though drug interactions aren't readily listed, in theory at least, the use of SAMe would add to the serotonergic effects of MAO inhibitors (e.g. selegeline), serotonin reuptake inhibitors (e.g. fluoxetine) and other anti-depressants (amitriptyline, clomipramine) as well as Tramadol.

Surgical Therapy

Several recent studies demonstrate a significantly lower mortality rate with elective cholecystectomies compared to those performed due to primary hepatobiliary disease. Further, a cohort of dogs that failed medical management and required surgery also had a lower survival rate, suggesting more aggressive intervention may have led to a better outcome.

Therefore, surgical intervention has its best success rate in the 'well' patient. Biliary rupture, especially when it is associated with infection, and bile peritonitis worsen the prognosis for short-term survival. While there are compelling reports of long-term survival in uncomplicated cholecystectomies, those more complicated cases who survive the peri-operative period have a good to excellent prognosis. Surgery is not without consequence; there is a tabulated 25% perioperative mortality rate, GB rupture cases looms large here, however mucocele rupture does not preclude a successful outcome. Reported long-term postoperative survival rates from cholecystectomy range from 65-91% with elective surgery survival rates the higher; the presence of intraoperative GB rupture at the time of surgery rendering the dog 2.5 times more likely to die than those without GB rupture or bile peritonitis.

While these numbers suggest that 'elective' procedures are recommended, it is important to remember that cholecystectomy carries a significant morbidity, even in a subclinical patient. Recent literature suggests the mortality rates of elective procedures range from 2% to 7%.

Cholecystectomy can be performed via a number of techniques, but the basics remain the same:

- Mobilise the gall bladder using a stay suture within the apex of the bladder and gentle blunt dissection of the liver parenchyma away from the gall bladder wall.
- Expose the cystic duct and artery to achieve the 'Critical View of Safety (CVS)'—the cystic duct with its associated artery free of other tissues suspended in space.
- Ligate the cystic duct and artery together using sutures or staples.

Mobilisation is aided by the use of stay sutures. Monofilament is ideal and considering the GB is thin and friable, particularly in diseased states, a cruciate pattern is recommended to avoid tearing through the wall. Some leakage around the suture is expected. The liver lobes can be retracted away from the GB fossa using ribbon (malleable) retractors and the parenchyma is bluntly dissected away using either the inner cannula of a Poole suction catheter or a sterile Q tip. Occasionally large vessels can be identified draining the gall bladder wall and these can be cauterised or clamped prior to avulsion. Cats tend to have more obvious venous drainage and can bleed profusely. In most cases, this haemorrhage can be controlled with pressure or the placement of haemostatic agents like gelatine sponges. This dissection should continue to the cystic duct where the hilar fat is gently freed from the duct and artery to give the critical view (CVS)—the cystic duct and artery suspended in space, clearly free of any other hilar structures. This is the essential part of the surgery and accurate dissection reduces the risk of accidental lobar duct occlusion and post-op bile leakage.

Ligation of the cystic duct and artery can be accomplished using monofilament ligatures although staples, such as Hemoclips, are significantly easier to place. The duct is ligated at either end and divided. The site should be checked for leakage. It may be advisable to pack the hilus and GB fossa with omentum prior to closure.

Unresolved Issues in Gallbladder Mucocele Disease

To sum up questions clinicians have been asking:

- Do asymptomatic GBMs progress and if so, how fast, and are these changes reversible?
- Is there any benefit to medically managing asymptomatic GBMs?
- Does medical management delay GBM progression?
- Does the risk of perioperative mortality render surgery inadvisable where there are no clinical or clinicopathological abnormalities?

1. What is the association between biliary sludge (somewhat defined as gravity dependent/ still liquidy hyperechoic biliary contents) and mucocele (more solid but malleable/squishy +/- lumps of organised mucous) formation?

    - GB sludge, and to a lesser degree GBM, is a common ultrasound finding, incidentally and as a response to investigating a clinical complaint usually involving inappetence, lethargy +/- vomiting and less frequently diarrhoea, diarrhoea, weight loss and/or ptyalism especially at mealtimes
    - This may represent a syndrome akin to biliary dyskinesia in people. There are several mechanisms at play here including primary biliary hypomotility, hepatopancreatic and bile duct sphincter dysfunction, altered gall bladder selective resorption and decreased CCK sensitivity.
    - Increases in serum liver enzymology (ALP, GGT—the cholestatic hepatic enzymes; ALT, +/- AST—the hepatocellular enzymes) is supportive of hepatobiliary disease, as is an associated increased Cholesterol level; Hyperbilirubinaemia is concerning for more severe disease (and is a negative prognostic sign) along with hypoalbuminaemia. Haematological changes can indicate inflammatory disease and may worsen with more severe disease.
    - While most dogs clinical for GBM have relatively acute onset of illness (days but up to some months), some dogs can have vague irregular symptoms for many months or longer; decision-making in treatment is compounded by the difficulty in imaging definitively confirming whether surgery is necessary or whether medical management is appropriate

2. Is it the physical properties of the gel-forming mucins produced by the GB epithelium that changes/slows the function of the GB, resulting in further accumulation of mucous and inspissated bile, or is it the GB dysmotility/dysfunction that predisposes to the accumulation and potentially obstruction, or are both scenarios possible?
    - Certainly, GB structure anomalies (e.g. Septate GBs) have proven predisposition to GBM
    - Ultrasound changes in the GB are often found when investigating patients showing acute abdominal signs, both mild symptoms of cranial abdominal pain right up to severe and fulminating abdominal disease where the pancreas, liver, +/- GB are the chief suspects
3. Is it pressure necrosis of the GB wall that results in rupture—GB distension causing mural ischaemia and subsequently necrosis, and is the associated EHBO (cystic and common bile ducts) secondary to extension or migration of mucin—the main cause of bile peritonitis/ secondary septic peritonitis?
   Is bacterial cholecystitis a relevant co-morbidity?

    - 13.5% of GBM have concurrent bacterial colonisation in several studies; however, there are at least two studies indicating survival rate from surgery for cholecystectomy is not affected by concomitant bacterial migration of bile or indeed bile peritonitis
4. What is the role of hyperlipidaemia, both primary and secondary?

    - Breeds such as Miniature Schnauzers have a higher incidence of primary hyperlipaemia and can this be a cause or just an association with their higher incidence of GBM
    - Secondary hyperlipidaemia from endocrine disorders (e.g. HyperA, HypoT), cholestasis, pancreatitis, obesity, etc
    - Although largely hypothetical, hyperlipidaemia is considered by some to be an important cause of lethargy, abdominal pain +/- vomiting and diarrhoea not necessarily attributable to pancreatitis or other abdominal disorders

- High-fat diets, as well as hyperlipidaemia (both hypertriglyceridemia and hypercholesterolaemia) have resulted in GB hypomotility with a reduction in GB smooth muscle contractility, and, in people at least, this mechanism is via decreased GB sensitivity to cholecystokinin
- Hypertriglyceridemia has been shown experimentally and clinically to respond to very low-fat diets, and there is some weak evidence that Omega-3 fatty acids, fibrates (e.g. Gemfibrozil) and even Niacin may be of benefit, but is there any resolution in any associated biliary disease?

5. In medical management, are choleretics (UDCA, SAMe) safely administered where there may be significant EHBO or GB wall compromise because of their promotion of smooth muscle contraction of the GB wall and elevation of intraluminal pressure?

   - Theoretically, if we are looking to improve the flow of bile through the hepatic and common bile ducts, it shouldn't matter too much if the GB is partially obstructed. The bile duct sphincter and hepatopancreatic (Oddi) sphincter are resistive (oblique muscles fibres rather than concentric) so it should be relatively easy to overcome them assuming there is no blockage here.

A Short Cook's Tour of the Critical Literature

M.J. Thomson *et al*

Cholecystitis and Gall Bladder Rupture in a dog

AVP 1996; 26: 114-117

Perhaps the first reported case in the veterinary literature of long-term survival after surgery for septic bile peritonitis.

Key point

Although prognosis in the 2020's remains guarded, there has been marked improvement in confidence levels for surgical and intensive care success in GB rupture & septic situations.

J.A.Jaffey *et al*

Ultrasonographic Patterns, Clinical Findings, and Prognostic Variables in Dogs From Asia with Gallbladder Mucocele

JVIM 2022; 36: 565-575

Key points

Six GBM types are described by their ultrasound images:

i. Organised echogenic debris occupying >30% of the lumen
ii. Combination of organised echogenic debris with partial stellate strands adhered to the GB wall
iii. Stellate pattern
iv. Combination of stellate and kiwi pattern
v. Kiwi pattern with echogenic debris
vi. Kiwi pattern

J.A. Jaffey *et al*

Gallbladder Mucocele: Variables Associated with Outcome and the Utility of Ultrasonography to Identify Gallbladder Rupture in 219 Dogs (2007-2016)

JVIM 2018; 32:195-200

219 dogs

Abdominal ultrasonography had low sensitivity 56.1% for identification of GB rupture, with specificity 91.7%.

Significantly higher risk of death with intraoperative evidence of GB rupture and bile peritonitis.

Key points

Abdominal ultrasonography has low sensitivity for identification of GB rupture.

GB rupture and bile peritonitis are negative prognostic factors for perioperative survival.

J.A. Jaffey *et al*

Effect of Clinical Signs, Endocrinopathies, timing of surgery, hyperlipidaemia, and Hyperbilirubinemia on Outcome in Dogs with Gallbladder Mucocele.

Vet Journal 2019; 251

Retrospectively

1194 dogs form 41 referral hospitals.

Key points

Dogs with GBM and abnormal clinical signs were more likely to die than subclinical dogs as were those with increased total serum/ plasma bilirubin concentrations.

Concurrent hyperadrenocorticism, but not hypothyroidism or diabetes mellitus, and the Pomeranian breed were also negative prognostic indicators.

Advancing age was also associated with increased odds of death in-hospital.

Dogs with GBM that demonstrated clinical signs attributable to biliary tract disease at the time of cholecystectomy had 4.2 times the odds of death than dogs who were subclinical.

S.M. DeMonaco *et al*

Spontaneous Course of Biliary Sludge Over 12 Months in Dogs with Ultrasonographically Identified Biliary Sludge

JVIM 2016; 30:771-778

45/77 healthy dogs >4 years old found with biliary sludge on ultrasound and monitored over 12 months with 2% resolved, 19% decreased amounts of sludge, 40% static, 29% increased and 10% recurrent.

Key point

GB sludge has significant presence and affected dogs remained asymptomatic over one year with very rare resolution and 30% changed to non-gravity dependent sludge indicative of changes in consistency of sludge with time.

A.K. Cook *et al*

Gallbladder Sludge in Dogs: Ultrasonographic and Clinical Findings in 200 Patients

JAAHA 2016; 52 (3):125-131

200 GB contents were evaluated and sludge quantified; dogs with >25% sludge were significantly older than those with minimal sludge (8 years versus 11 years); liver enzymology increases didn't correlate with the presence of biliary sludge; dogs with hyperA or HypoT were more likely to have >25% GB sludge.

Key point

>25% sludge was associated with increased volume of GB, indicating that changes in GB function or contractility may impact the formation of biliary sludge in dogs.

Mehmet Kesimer *et al*

Excess Secretion of Gel-Forming Mucins and Associated Innate Defense Proteins with Defective Mucin Un-Packaging Underpin Gallbladder Mucocele Formation in Dogs

PloS one, 2015

Key points

GBM formation involves an adoptive excess secretion of gel-forming mucins with abnormal properties by the gallbladder epithelium. The mucus is characterized by a disproportionally significant increase in mucin type, defective mucin un-packaging, and mucin-interacting innate defence proteins that are capable of dramatically altering the physical and functional properties of mucus. These findings suggest that abnormal mechanisms for maintenance of gallbladder epithelial hydration may be an instigating factor for mucocele formation in dogs.

R. Glauert & P. Watson

A Retrospective Study Examining the Relationship Between Biliary Sludge and Gall Bladder Mucoceles

BSAVA Congress Proceedings 2015; P457

30 dogs

One dog had documented progression in ultrasound findings from biliary sludge to GB mucocele over a 10-month period (first reported case).

Key points

GBM was as common as biliary sludge.

Consideration should be given to treating biliary sludge in at-risk breeds to try to prevent progression to GBM.

P. Secchi *et al*

Prevalence, Risk Factors, and Biochemical Markers in Dogs with Ultrasound-diagnosed Biliary Sludge

Res Vet Sci 2012 (93) 1185-1189

100 dogs.

The incidence of biliary sludge is high especially in older dogs.

Biochemical markers didn't have a significant correlation with biliary sludge.

The type of diet was not considered a major risk factor.

Key point

Hepatomegaly, often associated with cardiopathies, was frequently observed, and considered a risk group for the development of inspissated bile.

S. Mizutani *et al*

Retrospective Analysis of Canine Gallbladder Contents in Biliary Sludge and Gallbladder Mucoceles

J Vet Med Sci 2016

43 samples of GB contents (21/42 biliary sludge, 22/43 GBMs) were analysed and cultured.

The principal components of GB contents in both GBMs and biliary sludge are mucins, and both exhibit low rates of bacterial infection of the GB (14.3% and 10% respectively), nearly all intestinal flora.

Key points

It is possible that GBMs and biliary sludge have the same pathophysiology and possibly represent a continuous disease.

Hence, biliary sludge could be considered as the stage preceding the appearance of GBMs.

F.I. Hill *et al*

High Frequency of Cholecystitis in Dogs with Gallbladder Mucocele in Hong Kong

The Vet Journal 2022 Vol 287

56 surgical cases.

84% had histopathological evidence of cholecystitis.

Positive bile culture in 29.6% (E. coli and Enterobacter spp the most common).

89.3% (50 dogs) survived to discharge including 5/5 dogs with ruptured GBs.

Key points

GBMs were frequently associated with both acute and chronic inflammation.

High survival rates were achieved with surgery.

S.L. Friesen *et al*

Clinical Findings for Dogs Undergoing elective and Nonelective Cholecystectomies for Gallbladder Mucoceles

JSAP 2021 (62) 547-553

Mortality rate of 2/31 (6%) undergoing elective cholecystectomy and 21/90 (21%) for dogs undergoing non-elective cholecystectomy.

Complication rates were similar (~50%) for both groups; however, mild complications were seen in the elective group.

Key point

Elective cholecystectomy in dogs with GBM carried a low mortality rate and a relatively high frequency of minor complications.

G. Youn *et al*

Outcome of elective cholecystectomy for the treatment of gallbladder disease in dogs

JAVMA 2018 Vol 252 No 8

Mortality rate 2% (1/45) in elective surgery and 20% (5/25) in non-elective group → overall mortality 9% (6/70).

Dogs with clinical signs of lethargy, anorexia, vomiting, icterus or azotaemia less likely to survive as were dogs with low serum albumin and high serum ALT and total Bilirubin concentrations.

Key points

Elective cholecystectomy in dogs with early signs of biliary disease may be appropriate to avoid the higher mortality rate associated with more advanced disease and non-elective cholecystectomy.

J.G. Besso *et al*

Ultrasonographic Appearance and Clinical Findings in 14 Dogs with Gallbladder Mucocele

Vet Radiol Ultrasound 2000, Vol 41, No 3, 261-271

14 dogs with GBM studied histological diagnosis, about half of which had signs of EHBO at surgery and half had loss of GB wall integrity and/or GB rupture.

Positive aerobic bacterial culture was obtained from bile in 6/9 dogs.

Key points

Cholecystectomy appears to be an appropriate treatment for mucoceles, if not to treat a GB rupture, at least in most dogs to prevent it since GB necrosis was identified in 9/10 dogs.

GB infection was not present in all the mucoceles suggesting that biliary stasis and mucosal hyperplasia may be the primary factors involved in mucocele formation.

J. Choi *et al*

Comparison Between Ultrasonographic and Clinical Findings in 43 Dogs with Gallbladder Mucoceles

Vet Radiol Ultrasound 2014; Vol 55, No 2, 202-207

43 dogs with GBM, 24/43 were symptomatic, including 11 with GB rupture.

5 patterns were categorised (type 1—echogenic immobile bile; type 2—incomplete stellate pattern; type 3—typical stellate pattern; type 4—kiwifruit-like pattern and stellate combination; type 5—kiwifruit-like pattern with residual central echogenic bile) in which there were no significant correlations found between ultrasonographic patterns of GBMs and clinical disease status or GB rupture

Key point

Ultrasonographic patterns of GBMs may not be valid bases for treatment recommendations in dogs.

T. Tsukagoshi *et al*

Decreased Gallbladder Emptying in Dogs with Biliary Sludge or Gallbladder Mucocele

Vet Radiol Ultrasound 2012; Vol 53, No 1, 84-91

60 dogs including 24/60 normal controls, 24/60 with mobile sludge, 5/60 with immobile sludge, 7/60 with GBM showed GB ejection fraction was significantly decreased in all 3 abnormal groups and all abnormal dogs had GB distension.

Key points

Biliary stasis occurs in dogs with biliary sludge as well as those with GBM.

Cholestasis may play a role in the pathogenesis or progression of these diseases in dogs.

A.D. Viljoen *et al*

Clinical Characteristics and Histology of Cholecystectomised Dogs with Non-gravity-dependent Biliary Sludge: 16 Cases (2014-2019)

JSAP 2021 (62) 478-488

16 cholecystectomised dogs with non-gravity-dependent biliary sludge; 0/16 had histological evidence of GBM, 11/16 normal biochemistry, 12/16 had cholecystitis, 9/12 had biliary mucosal hyperplasia, 13/16 had lymphoplasmacytic enteritis, 9/16 had reactive hepatitis and all 6/16 with non-gravity dependant biliary sludge filling <1/2 GB volume has sub-optimal GB function.

Presenting clinical signs including diurnal inappetence in the morning and exercise intolerance resolved in 12/14 (86%) dogs after cholecystectomy and clinical improvement was noted in 13/16 (81%) overall.

Key points

Duodenal inflammation could potentially impact GB dysmotility in dogs with non-gravity-dependant biliary sludge.

Diurnal inappetence in the morning and exercise intolerance could indicate symptomatic GB disease in dogs with non-gravity-dependant biliary sludge and can precede more obvious systemic clinical signs associated with GBMs.

A.D. Viljoen *et al*

Gall Bladder Ejection Fractions in Dogs Investigated for Chronic Altered Appetite: 14 Cases (2015-2017)

JSAP 2021 (62) 1101-1107

14 dogs with chronic GI disease and altered appetite (9/12 had suboptimal GB function with or without gravity-dependent biliary sludge; there was observable difference in GB ejection fractions between groups defined by appetite (no statistically significant difference).

Key points

GB dysmotility and distension can occur in the absence of GB sludge and GBMs in younger dogs.

GB dysmotility requires further investigation as a potential contributing factor to altered appetite in dogs.

M. Kutsunai *et al*

The Association Between Gall Bladder Mucoceles and Hyperlipidaemia in Dogs: a Retrospective Case Control Study

The Vet J 2014; 199 (1) 76-79

58 dogs with GBM; 15/37 had hypercholesterolaemia, 13/24 had hypertriglyceridemia showing significant association with GBM. American Cockers, Miniature Schnauzers and Chihuahuas were significantly predisposed to GBM.

Key point

A significant association between GBM and hyperlipidaemia was confirmed, suggesting that hyperlipidaemia may play a role in the pathogenesis of GBM.

P.G. Xenoulis & J.M Steiner

Canine Hyperlipidaemia (Review)

JSAP (2015) 56, 595-605

Hyperlipidaemia can be primary (breed related) or secondary to endocrinopathies, pancreatitis, cholestasis, PLN, and obesity.

Hypertriglyceridemia of Miniature Schnauzers is the most common primary hyperlipidaemia.

Key point

Management of primary hyperlipidaemia is achieved by administration of ultra-low-fat diets with or without lipid lowering drugs such as omega-3 fatty acids, fibrates, niacin, and statins.

J.A. Jaffey

Canine Extrahepatic Biliary Disease (Review)

JSAP (2022) 63, 247-264

Review of extrahepatic biliary disease in the dog, including Gall Bladder Mucocele

F.S. Pike *et al*

Gallbladder Mucocele in Dogs: 30 Cases (2000-2002)

JAVMA 224 (10) 1615-1622

Cholecystectomy in 23/30 dogs & 7/30 medically managed

Perioperative mortality in 5/23 (21.7%).

18/23 survivors perioperatively had complete resolution of signs and alive at follow-up ~13.9 months (range 1-34 months).

Retrospective study.

No separation of uncomplicated BM versus complicated cases (GB rupture, peritonitis).

Key point

Cholecystectomy for dogs with GBM is associated with substantial perioperative mortality of 21.7% (5/23).

M. Parkansky *et al*

Long-term survival of dogs treated for gallbladder mucocele by cholecystectomy, medical management, or both

JVIM 2019; 33 (5) 2057-2066

89 dogs.

46/89 had surgery, 33/89 medical management, 10/89 medical followed by surgery.

Retrospective study.

No separation of uncomplicated BM versus complicated cases (GB rupture, peritonitis).

Key points

89% (79/89) dogs who survived >14 days, the medically treated group had significantly shorter mean survival time (MST 1340 days) than the surgically treated group (MST 1802 days).

MST of the 10/89 who had both medical then surgical treatment (MST 203 days) was significantly shorter than both the medical and surgical groups.

Emily M. Brand *et al*

Computed tomographic features of confirmed gallbladder pathology in 34 dogs

Vet Radiol & Ultrasound 2020; Vol 61, Issue 6

15/34 had cystic mucosal hyperplasia, 9/34 with GB oedema, 8/34 had GBM, 6/34 with cholecystitis

Key points

CT is accurate for identifying GB thickening, hyperattenuating material, mineral attenuating material and intraluminal nodules but there is overlap with a variety of GB pathologies such as cystic mucosal hyperplasia, cholecystitis, GBM, GB wall oedema, cholelithiasis.

Jason A. Fuerst & Eric T. Hostnik

CT attenuation values and mineral distribution can be used to differentiate dogs with and without gallbladder mucoceles

Vet Radiol & Ultrasound 2019; Vol 60, Issue 6

Prospective study where 20 normal dogs were compared with 13 dogs with >25% sludge in GB lumen and 18 with confirmed GBM on histopathology.

Key points

CT can differentiate a subset of GBMs from dogs with and without GB sludge, especially precontrast.

S.C. Marroquin

Comparison of abdominal computed tomography to ultrasound in the diagnosis of canine biliary disease manifesting as acute abdominal signs

Master's Thesis, College of Vet Med, Mississippi State University 13th May 2022

28/35 dogs with biliary pathology compared with 7/35 normals.

Key points

CT seems as accurate as Ultrasound in identification of GB disease.

CT is more accurate in identification cholelithiasis than ultrasound.

CT may be used in place of Ultrasound in canine patients presenting for acute abdominal signs of biliary origin.

Case Studies

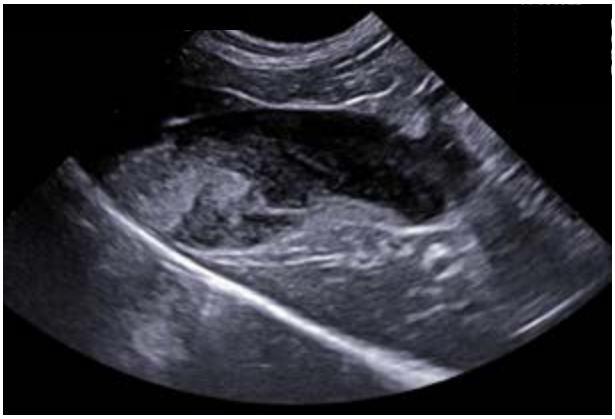
'ROBERTA' 4 years FS JRT X 8.6kg

Presenting Signs: Uncomfortable, stretching/praying positioning recurrent usually after eating with sometimes vomiting and inappetence; episodes at least weekly over a 6-month period variously responsive to IVFs and GI support therapy.

Physical Examination: Cranial abdominal discomfort on palpation the only anomaly.

Diagnostics: Haematology and Biochemistry essentially normal.

Ultrasound:



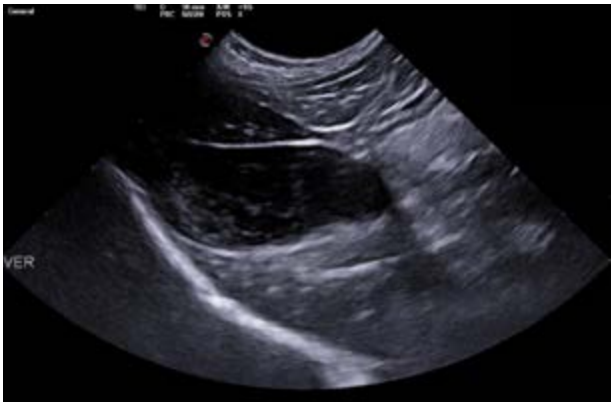


Figure 1: Look at all that sludge. But the histology was unremarkable. What happens when the GB does not empty properly for other reasons!

Surgery: Cholecystectomy, GIT and Liver and Pancreatic Biopsies.

Pathological Diagnosis: Mild lymphoplasmacytic gastritis, histologically normal duodenum, jejunum. Histologically normal liver—Mild diffuse hepatocellular glycogen vacuolation.

Pancreas: No significant lesion.

Gallbladder: No significant lesions.

Pathologist's Comments: The gastric inflammation is very mild. Lymphoplasmacytic inflammation is relatively commonly seen in gastric biopsies from vomiting dogs but is not aetiologically specific. It may be a manifestation of immune mediated gastritis, analogous to inflammatory bowel disease (IBD), though in such cases the inflammation is usually much more prominent than seen here. IBD cannot be diagnosed on the basis of histologic findings alone but must be based on all available information including clinical and imaging findings.

The mild hepatocellular glycogen vacuolation may reflect stress induced hypercortisolaemia. The reported clinical signs and/or imaging appearance of the gastrointestinal tract and gall bladder may be related to issues of gastrointestinal or gall bladder function including tone and motility, which do not have a histologic correlate. Histologic changes restricted to the caudal small intestine should also be considered.

Outcome: Episodes very scant now (1-2 minor episodes over 6-month period) after 12 months on low-fat novel protein diet fed three times daily.

'RASTUS' 12 years Dachshund FS

Presenting Signs on initial visit: Acute onset vomiting, inappetence.

Physical Examination: Essentially normal.

Diagnostics: Haematology all within normal limits; Biochemical abnormalities—ALP 276 U/L (20-150), ALT 340 U/L (10-118), Tbilirubin 5 µmol/L (2-10).

Treatment: IVFs and anti-emetics; Symptoms resolved and no further diagnostics or treatment.

Presenting Signs on second visit 12 months later: Acute onset vomiting, inappetence.

Physical Examination: Mostly normal with mild cranial abdominal discomfort on palpation.

Diagnostics: Haematology all within normal limits; Biochemical abnormalities—ALP 478 U/L, ALT 434 U/L, Tbilirubin 17 µmol/L.

Ultrasound:

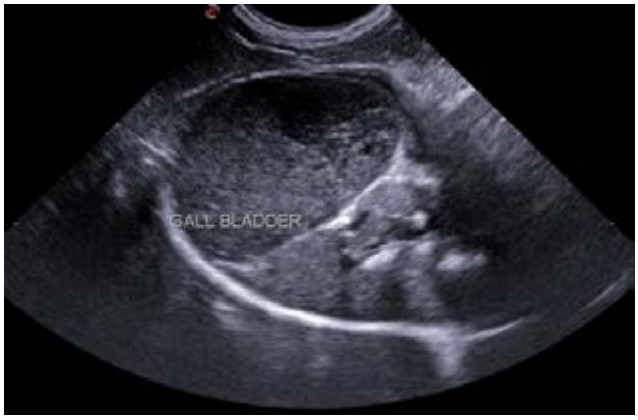


Figure 2: Histologically confirmed GB mucocele

Treatment: IVFs and analgesics anti-emetics.

Follow-up 5 days later: ALP 2176 U/L, ALT 841 U/L, TBil 11 µmol/L.

Surgery: Cholecystectomy

Pathology: The specimen consists of an opened gallbladder measuring 50 mm in length and 30 mm in diameter. The serosal surface is smooth and haemorrhagically stained.

Upon sectioning the wall measures up to 2 mm maximum thickness. The lumen is filled with a haemorrhagic semisolid material. The lumen of the gallbladder contains dense mucinous material and blood. The wall exhibits papillary epithelial hyperplasia. There is an adjacent intra-mural focus of haemorrhage surrounded by fibrosis.

Diagnosis: Gall bladder mucocele with subacute mural haematoma.

Pathologist's Comments: Gall bladder mucocele is characterised by accumulation of dense mucus in the gall bladder, which may become inspissated and lead to ischaemic pressure necrosis of the gall bladder wall and bile peritonitis. The aetiology is not known. The mural haematoma presumably reflects infarction at one point in the compromised mucosa.

'HARRY' 5 Years MN French Bulldog

Presenting Signs: Inappetence, Weight loss over 1-2 weeks.

Physical examination: Jaundice the only anomaly.

Diagnostics: Haematology all within normal limits; Biochemistry abnormalities—ALP 1800 U/L (20-150), ALT 303 U/L (10-118), Tbil 305 µmol/L (2-10) and after 4 days of IVFs and support therapy ALP 1753 U/L, ALT 717 U/L, TBil 105 µmol/L

Ultrasound: Markedly distended GB with >50% contents hyperechoic immobile material; CBD dilation and obstruction with similar hyperechoic echogenic sludge.

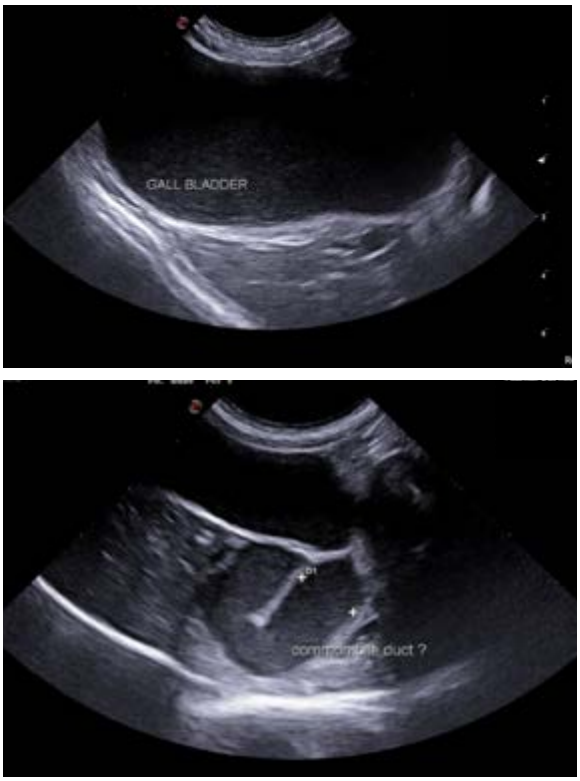


Figure 3: A GB mucocele, but maybe less obvious sonographically

Surgery: Cholecystectomy.

Pathology:

Gallbladder: The specimen consists of an intact and distended bile-stained gallbladder measuring 65 x 33 x 30 mm. The wall measures 2-3 mm in thickness and the lumen contains semisolid bile. The gallbladder lumen contains extensive, often grey/blue mucoid (inspissated) bile. The mucosa shows cystic mucosal hyperplasia, forming thin and often anastomosing finger-like projections that project into or are separated by the inspissated bile. The submucosa contains scattered lymphocytes and plasma cells, and occasional nodular aggregates of lymphocytes.

Diagnosis:
Gallbladder mucocele. Mild lymphoplasmacytic cholecystitis.

Pathologist's Comments:

Histopathology is consistent with a gallbladder mucocele, which is accompanied by mild chronic lymphoplasmacytic cholecystitis. The latter is not an uncommon concurrent finding in canine gallbladder mucoceles, and at least in some cases may be associated with ascending infection via the bile ducts from the intestine, often facilitated by bile stasis. Some affected patients may have concurrent cholangitis or cholangiohepatitis and/or cholelithiasis.

Gall Bladder wall, Biliary contents and adnexal liver tissue

Culture:

NEGATIVE

'WILLIAM' 14 years MN Shih-Tzu

Presenting Signs: Vomiting, Anorexia, Weak, Jaundice.

Prior history: Intermittent mild GIT upsets over some years. Mild increase in ALP enzyme on screening bloods (prior to dental surgery) 6 months prior. Has spent the previous 7 days in hospital on IVFs, antibiotics, anti-emetics, analgesics. Bloodwork over this time showed ALP consistently >2000 U/L (20-150), ALT levels improving from 1959 U/L to 1387 U/L (10-118), TBilirubin 200 µmol/L reducing to 148 µmol/L.

Diagnostics: Haematology showed leucocytosis WCC 33.9 x10*9/L (6.0-17.0) with Neutrophilia (N's 30.35 x10*9/L—NR: 3.0-12.0); Biochemical anomalies of ALP >2000 U/L (20-150), ALT 680 U/L (10-118), TBilirubin 85 µmol/L (2-10)

Pathology: The specimen consists of an opened gallbladder measuring 55 x 35 x 25 mm. There are possible pinpoint perforations in the mid-section. The wall measures 2 mm in thickness and shows multifocal cystic structures. Residual contents are semisolid mucinous green bile.

Ultrasound:

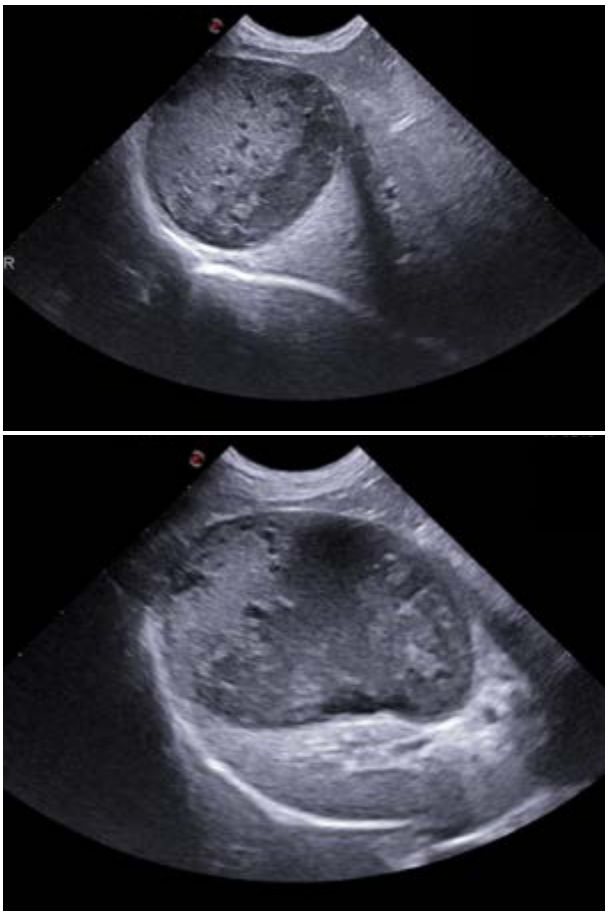


Figure 4: An open misère GB mucocele. This screams—please cut me out!

Five sections of gallbladder are evaluated. Within the biopsy samples evaluated, the gallbladder wall is lined by hyperplastic mucosal epithelium which frequently forms papillary cystic projections into the lumen associated with extensive amounts of inspissated amphophilic congealed material. Scattered throughout the inspissated material are brown, orange choleliths. The mucosal lamina propria contains a mild inflammatory infiltrate consisting predominantly of lymphocytes and plasma cells with fewer histiocytes and rare neutrophils. Multifocally in a few sections, the mucosal epithelium is ulcerated and replaced by a mat of fibrin, and degenerate granulocytes. Directly underlying the ulcer are regions of fibroplasia and neovascularisation.

Diagnosis:

Gallbladder mucocele with multifocal transmural rupture and cholelith formation.

Mild chronic lymphoplasmacytic cholecystitis and cystic mucosal hyperplasia.

Pathologist's Comments: Histologically, the biopsy samples evaluated contain evidence of a gallbladder mucocele with associated choleliths and multifocal small regions of rupture. Although the wall contains evidence of pinpoint ruptures, the peripancreatic adipose tissue and serosa contains no evidence of serositis concerning for bile peritonitis. The gallbladder wall contains evidence of cystic mucosal hyperplasia which is considered a predisposing lesion to the development of gallbladder mucocele formation. There is no evidence of neoplasia or infectious organisms within the gallbladder.

'GEORGE' 10-month MN Poodle X 3.2kg

Presenting Signs: Intermittent vomiting, inappetence.

Physical Examination: Normal.

Diagnostics Day 1: Haematology all within normal limits; Biochemical anomalies ALP 443 U/L (20-150), ALT 176 U/L (10-118), Tbilirubin 11 µmol/L (2-10)

Treatment: IVFs, anti-emetics.

Diagnostics Day 3: Haematology all within normal limits; Biochemical anomalies ALP 578 U/L (20-150), ALT 219 U/L (10-118), Tbilirubin 9 µmol/L (2-10).

CT Scan: In the gallbladder there are numerous mineral bodies [LIME GREEN circle]. The gallbladder lining enhances irregularly, suggesting mild thickening. The common bile duct is normal. The liver has a normal volume. Intrahepatic portal branches are challenging to identify, however the diameter of the portal vein [LIME GREEN arrow] at the porta hepatis is normal relative to that of the aorta. Right- and left-divisional portal branches are definitively visualized. Along the cranial contour of the liver on the left there is an ill-defined linear area of contrast uptake [PINK arrow]; a link to the caudal vena cava is not definitively established. The

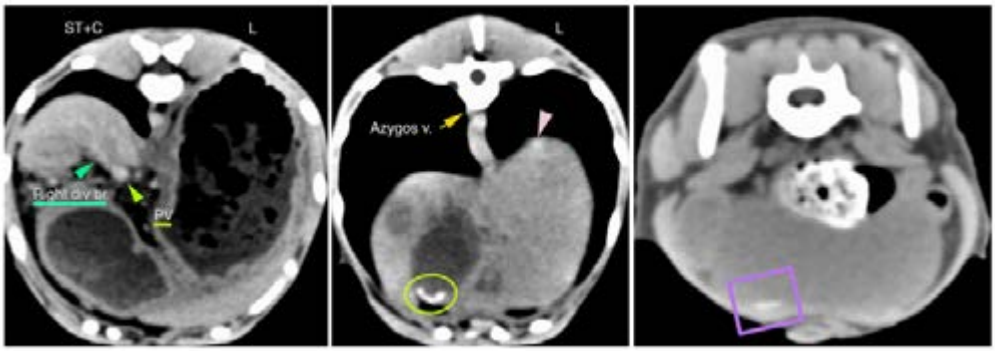


Figure 5: George's CT scans

azygos vein [YELLOW arrow] has a normal diameter. The left hepatic lymph node is mildly enlarged, 6.5mm.

CT Diagnosis: Cholecystolithiasis, moderate

- Urinary bladder sediment, non-obstructive—urinalysis is recommended to identify mineral type and rule out infection
- Grossly normal hepatic vasculature—no portosystemic shunt is definitively identified; however, depending on the results of bile acid testing a triple-phase CT angiography performed with breath hold may be indicated to rule out a small epiphrenic abnormal vessel

Radiologist's Comments: The presence of cholecystolithiasis in a puppy this age is concerning for the presence of hepatobiliary disease/cholecystitis. This finding will need to be evaluated further by means of blood chemistry and abdominal ultrasound. CT has a limited ability to identify diffuse hepatic parenchymal changes—with ultrasound a preliminary differential list can be reached based on echogenicity alone.

'MITZI' 13 years FS Toy Poodle 2.3kg

Presenting Signs: Vomiting, inappetent.

Physical Examination: Cranial abdominal pain on palpation.

Diagnostics Day 1: Haematology all within normal limits; Biochemistry ALP 253 U/L (20-150) ALT >2000 U/L (10-118) Tbil 16 µmol/L (2-10)

Ultrasound day 1:

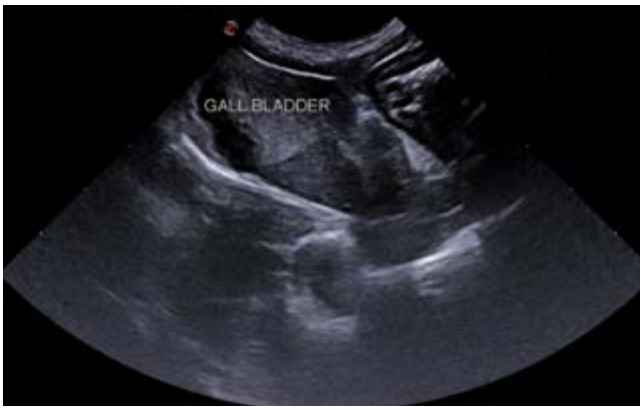


Figure 6A: Mitzi's Ultrasound on day 1

Treatment: IVFs, anti-emetics, analgesics.

Diagnostics Day 2: Haematology all within normal limits; Biochemistry ALP 301 U/L (20-150) ALT 1827 U/L (10-118) Tbil 6 µmol/L (2-10).

Diagnostics Day 5: Haematology all within normal limits; Biochemistry ALP 126 U/L (20-150) ALT 467 U/L (10-118) Tbil 6 µmol/L (2-10).

Ultrasound Day 5:

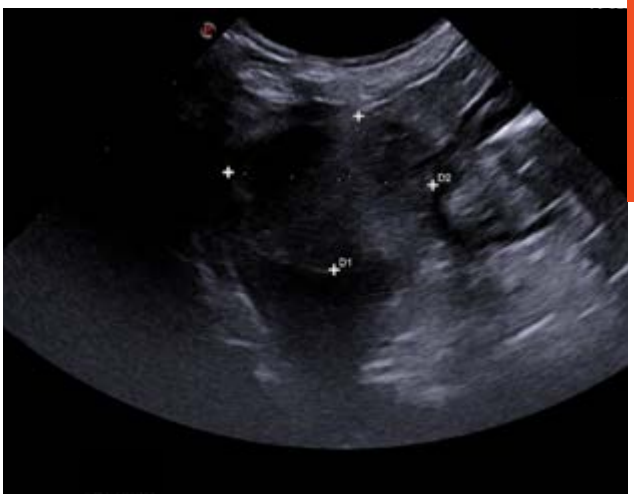


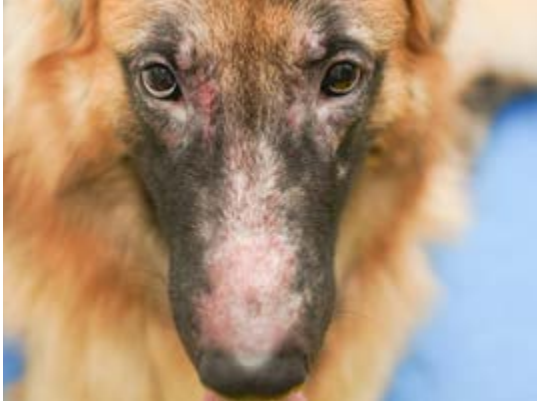
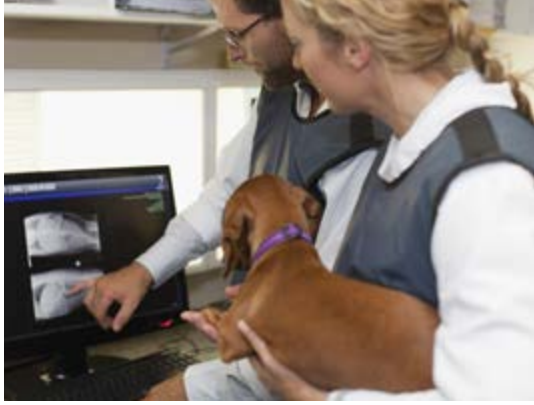
Figure 6B: Ultrasound a few days later.

Treatment: Surgery Cholecystectomy.

Gallbladder Pathology: The specimen consists of an intact and distended gallbladder measuring 32 x 25 x 24 mm. The wall measures 1 mm in thickness and the contents are consistent of granular to semisolid bile. Frequently adhered to the mucosal surface is a thin layer of grey/blue mucoid bile that sometimes separates the mucosal folds. The mucosa often exhibits cystic to papillary hyperplasia with areas of irregular papillary and sometimes arborising projections in the mucosal epithelium. The lamina propria often contains low to moderate numbers of widely scattered lymphocytes and plasma cells, sometimes accompanied by fewer pigment-laden macrophages. Also evident in the lumen are numerous oval to irregular gold-brown laminated concretions (choleliths), the largest measuring less than 2 mm in diameter.

Diagnosis: Early Gallbladder mucocele; Cholecystitis, Mild chronic lymphoplasmacytic cholecystitis.

Pathologist's Comments: The presence of extensive inspissated bile on the mucosal surface supports early gallbladder mucocele formation. There are concurrent proliferative mucosal changes that in some regions are quite prominent and resemble a papillary adenoma; however, given the distribution, this change is considered most consistent with cystic to papilliferous mucosal hyperplasia. There is no evidence of malignancy. There is also evidence of mild chronic cholecystitis and cholelithiasis.◆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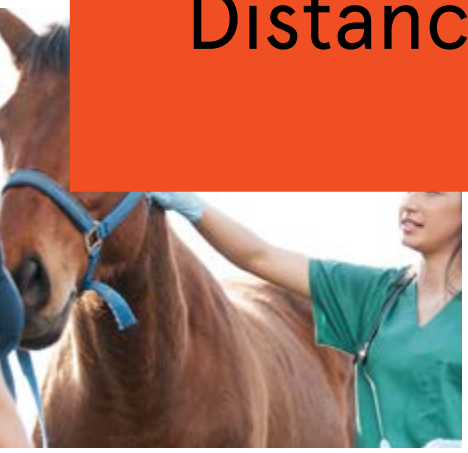
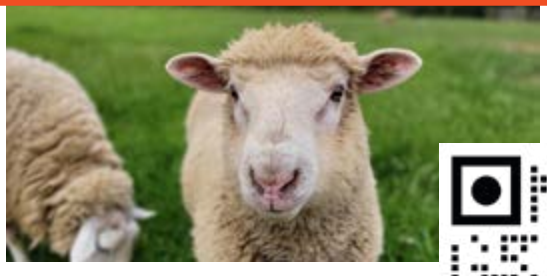



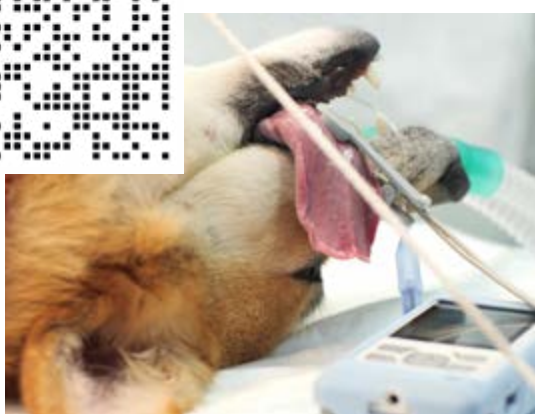

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