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Vol 11 | Issue 02 | May 2024

Focus on Wellness

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Editor's Note

I hope everyone is well. I'm just back from the WVA Congress, I enjoyed meeting up with so many people I hadn't seen in a while. With all the ease and accessibility of webinars, I think we should see the benefits of an in-person conference now and again, especially for our mental health. I wasn't feeling particularly enthralled with and by my profession in the months before the conference. I was tired and procrastinating, hence the stress of the presentations, but chatting with all acquaintances and colleagues, some last seen 15 years ago, revitalised me and I felt more positive by Friday. It was a time to compare and share ups and downs, both personal and professional and realise we are all in the same boat, no matter if from a small or big practice; owner or employee; specialist or general practitioner. It was also exciting to see how so many people had made changes to mould their own way of being a vet and contributing to the field to suit their own circumstances. There are many ways to earn your living with our degree – we shouldn't need to feel trapped. Ok - so off my soapbox. A good edition this time I think. The wellness article is once again very relevant to our daily lives as vets. I have 2 CPD articles in this edition - my authors over-delivered and I'll not look a gift horse in the mouth – thanks Izak and Kelly. I also cornered some surgeons at the congress, so you can look forward to few local articles soon. I am starting a Practice Tip page, after I realised how people appreciated some of the tweaks I use on a day-to-day basis when I presented at the congress. We all have these little tweaks - so I am looking forward to your contributions.

Enjoy the read

Liesel

vet360

VET360 aims to be a leader in the field of continuing veterinary development in Southern Africa by providing veterinary professionals from diverse disciplines with tools to help them meet the challenges of private practice. The magazine aims to make information accessible, both paper and electronic, and provide clinical, business and other veterinary information in a concise form to enable the practitioner to rapidly acquire nuggets of essential knowledge

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Vet360 is a source of current, relevant educational material for practising veterinary professionals in Southern Africa. Published Bi-monthly in hard copy and electronically on www.vet360.vetlink.co.za.

Distribution: Southern Africa, 3 500 copies.

Editor: Liesel van der Merwe

Layout and design: Annalize de Klerk

Publisher and Owner: Vetlink Publications

Other Publications by Vetlink: Vet360 Mobile App, Livestock Health and Production Review, Hooo-Hooo, Equine Health Update

Publisher: Madaleen Schultheiss, madaleen@vetlink.co.za

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Striving towards Wellness:

Depression, Anxiety, Burnout and Compassion Fatigue

-The Tired That Does not go Away...

Brought to you by:

DIGITAIL

Retha Watson
(MA Industrial Psychology)

"I wanted to talk about it. Damn it. I wanted to scream. I wanted to yell. I wanted to shout about it. But all I could was whisper 'I am fine'." Unknown.

One of the most debilitating concepts that social media and some motivational speakers have tried to sell us, is that we have to be in a constant state of happiness and that everything must have meaning and purpose. Indeed, this is something to aim for, but sometimes, life is just tough. Sometimes we are worried, angry, or just numb and it is okay to experience various emotions and with varying intensity.

What we should strive for is the normalising of negative emotions: to name it, acknowledge it and have a healthy way of processing those emotions. We will deal with emotions in a following article.

We should be talking openly about mental illness and the negative impact it has on our work and home life; we need to create a safe space for ourselves and others to talk about mental illness. We still have pre-conceived judgments around mental illness. Surely, we will not tell a person with cancer that they should just find positive meaning

in life. To keep busy and get over it? There are specific medical interventions for cancer and there are also specific interventions and treatments for mental illness.

What a day it will be if you can walk into the workspace and say "I have been diagnosed with depression, so I will be out of sorts more often than not" or a sincere "Amanda, you have been diagnosed with anxiety, when things get too much, do what you need to do, so that you can get well and healthy again, we will support you all the way, we are here for you, talk if you need to."

Best, et al. (2020) wrote, "Veterinarian mental health is a reported area of concern in several countries worldwide with high levels of anxiety and depression and self-reported burnout and compassion fatigue".

Anxiety

Everybody is anxious from time to time: writing exams, doing a difficult surgery or driving in a dangerous area. Anxiety however becomes a disorder or causes dysfunction when it is intense, extreme, and results in a continual worry about everyday situations in such a manner that it interferes with normal life, social interactions and relationships.

Risk factors to anxiety may be medical conditions or previous traumatic experiences, it may even be a traumatic experience as an infant, stress due to illness or a long-term stress build-up. In the March edition, the propensity to anxiety due to personality was discussed.

High cognitive functioning individuals, leans towards anxiety as part of their personality traits. Genetics or other psychological illness, drugs or alcohol misuse may also lead to anxiety disorders.

Depression

As in anxiety, a depressive phase is normal, feeling down or sad as life happens to us. Short duration depression can occur during illness, after a traumatic event or as part of the grieving process. We may even experience mood changes from time to time.

Clinical depression is caused by a medical condition or simply an event so stressful or traumatic that it causes abnormalities in the neurotransmitters dopamine and serotonin. Depression may even be caused by specific medications or a genetic predisposition. Alcohol and drug abuse may also be a major cause of depression or exacerbate an underlying predisposition.

In sixty percent of cases, we find depression and anxiety in a co-morbid and complex relationship. The symptoms many times overlapping and looking much alike.

Within the working environment of the veterinary practice, along with possible depression and anxiety, the veterinarian is also confronted with traumatic incidents, euthanasia and animal neglect and abuse so that it is quite common that compassion fatigue and burnout will also present itself.

Compassion fatigue

Professionals who are working in an environment and being exposed to other people's trauma daily, is exceptionally vulnerable to compassion fatigue. Acute symptoms may lead to physical and psychological illness.

Empathy as a skill and part of one's character is valuable in medical professions, opening oneself up for the witnessing of others pain, sorrow, and trauma, will lead to vicarious traumatisation, making the professional vulnerable to compassion fatigue – the total exhaustion of one's physical and psychological being.

The causes may vary, and aggravated when professionals deny themselves self-care, volunteering for additional shifts, and don't take their days off.

Compassion fatigue can occur after a single trauma or years of accumulated exposure to emotions and traumatic events.

In a previous article it was highlighted that veterinarians are one of the professions who are passionately involved with the well-being of their patients, the enough is never enough. This is how this particular profession becomes vulnerable to compassion fatigue.

The "Superman" syndrome - trying to save the world and trying to be everything to everybody. This is quite unachievable and your mental and physical health may suffer due to this.

Burnout

The saying "I will rather burn out than fade away" is well known phrase, said by many, and meaning, giving themselves passionately for their cause, regardless of late nights and early mornings, living on heavy fuel consumption. On researching this, Neill Young wrote it as a tribute to the early deaths of young rock stars, dying at the peak of their careers, ironically Curt Cobain wrote this very same phrase in his suicide note.

Burnout is a state of psychological and physical exhaustion caused by extended and intense stress and is mostly work related. Where compassion fatigue is due to the witnessing of others trauma and emotions, burnout is caused by your own stress and exposure to traumatic incidents.

Should we then not reconsider changing the motto from "I will rather burn out than fade away" to "**He that fights and runs away, may turn and fight another day; but he that is in battle slain, will never ride to fight again" (Tacitus)**.

With "running" away, rest, self-care, healthy living is implied. Sometimes we have to save ourselves in order to be the best we can be for others.

With depression, anxiety, compassion fatigue and burnout, symptoms may appear quite similar. Let's have a look at the symptoms and how they overlap.

Symptoms of Depression, Anxiety, Compassion Fatigue and Burnout

	Symptoms specific	Corresponding/Overlapping Symptoms
Depression	• Thoughts of death and suicide • Constant sadness • Slowed movement and speech • Constant physical pain	• Lack of interest and enjoyment • Extreme fatigue • Digestive/gastrointestinal problems • Appetite and weight changes • Inability to concentrate • Inability to make decisions • Impaired rational thinking and problem solving • Sleeping pattern changes : either too much sleep or too little (insomnia) • Restlessness • Irritability • Difficulties with personal relationships • Self-contempt, feelings of guilt • Emotional disconnection • Decreased sense of purpose • Numbness • Nervous restlessness
Anxiety	• Sense of impending danger or doom • Hyperventilation • Sweating • Trembling • Avoidance of situations that may trigger anxiety • Excessive worry • Racing thoughts	
Compassion fatigue	• Headaches	
Burnout symptoms similar		

Recognising any of the above symptoms means that you should consult a medical doctor to rule out any underlying physical illness and then get a referral to a psychiatrist for consultation and appropriate medication.

Managing my Mental Health

If we are in a goal orientated space, it is easy to set objectives for our physical well-being, we should however prioritise our psychological well-being as well.

- ✔ Take your days off, take annual leave and ***REST.*** Take on mindless projects that does not cause stress during leave, relax with a puzzle or a good book, renovate an old piece of furniture.
- ✔ **Exercise** – endorphins are released during exercise that enhances a sense of well-being.
- ✔ **Healthy diet** – avoid sugars, caffeine. Research the concept of gut-health, we have more serotonin in our stomach than in our brain, that is the neurotransmitter we so desperately need for feeling mentally well.
- ✔ **Sleep hygiene:**
  - Be consistent with your sleep time, ideally at least two hours before midnight and 8 hours of uninterrupted sleep.
  - Quiet bedroom, dark and relaxing at a comfortable temperature.
  - Remove all electronics from your room, smart phones, laptops and other devices.
  - Avoid large meals, caffeine and alcohol before bedtime.
  - Gentle exercise before bedtime will enhance your sleep.
  - Put off or take away any heating devices as your body temperature needs to drop in order to go into a deep sleep.
- ✔ **Relaxation activities** – any activity where you consciously relax, which may include yoga, meditation, exploring nature and being wilfully present in the experience or listening to relaxing music.
- ✔ **Relaxation exercises** that is widely available on the internet as demonstration.
- ✔ **Socialisation** with undemanding, good for body and soul friends and family.
- ✔ Avoid **excessive use of alcohol and mood-altering drugs.**
- ✔ Practice **gratitude** and **mindfulness.**
- ✔ Set small and attainable **goals** and **objectives.**

- ✔ Give yourself a **spoil day** once every now and then – going for a massage, taking a road trip, watching a movie that you have wanted to see for ages, read the book that you never have time for.

- ✔ **Psychic numbing** – dialling down on your empathy when you are at work, being able to free up cognitive resources and find solutions to problems rather than being overwhelmed with the emotions and pain of others.

- ✔ The last, but definitely not the least – **when you feel that things are getting to be too much- get professional help,** as much as you may think that therapy or counselling cannot solve your problems. These interventions do not aim to solve your problems, it is a process of facilitating your own understanding and healing within a safe environment.

Retha is an Industrial Psychologist and Director of Watson Corporate Consulting since 2017, consulting companies on wellness, corporate social responsibility and human resources. She was a part time lecturer in counselling for the master's degree students in Industrial and Organisational Psychology for 10 years and the national trauma and suicide prevention manager for the South African Police Service for 18 years. She has consulted in Lesotho for three years and finds meaning and purpose in her work, walking the walk with people.

"Almost everything will work again if you unplug it for a few minutes, including you."

Anne Lamott

All the information has been factually and scientifically sourced and references available on request.

WCC has established a service of online counselling after hours in order to provide support with long working hours in mind.

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Diabetic Cataracts in Dogs and Cats

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Introduction

Diabetes mellitus (DM) is a common endocrinopathy in both dogs and cats, and the prevalence has reportedly increased during the last few decades comparable to the globally increasing prevalence described in humans. Prevalence in dogs is reported to be between 0.26 % to 1.33 % compared to cats with reported values of 0.76% to 2.24 %[1,2,3,4]

Most dogs suffer from insulin deficiency diabetes compared to cats where insulin resistant diabetes is more common.[2,4] Burmese cats are overrepresented in the cat population with up to a 3x more likelihood to develop diabetes.[3] Canine subjects with an elevated risk of developing diabetes mellitus (DM) were characterised by several factors. These included advanced age (over eight years of age), intact female status, neutered male status, belonging to the Border Terrier or West Highland White Terrier breed, prior administration of glucocorticoid therapy, and the presence of co-morbidities such as obesity, pancreatitis, or hyperadrenocorticism.[1]

Cataracts encompass a common group of ocular pathologies characterized by diminished transparency of the lens or its surrounding capsule. These opacities manifest with diverse characteristics, including extent, shape, localization within the lens, aetiology, age of onset, and progression rate.[5]

Canine diabetes mellitus frequently results in cataract formation, with dogs exhibiting a distinct predisposition to develop diabetic cataracts compared to other species.[6]

Studies indicate that 50% of canine diabetic patients exhibit cataract formation within 170 days of diagnosis, with that figure rising to 80% by day 470.[6] Astudy by Beam et al. demonstrated significant breed-dependent disparities in cataract formation, with a notably higher likelihood observed in non-sporting breeds, especially brachycephalic breeds and Shar-Peis, manifesting within 10 days of diagnosis.[6]

Diabetic cataracts have been reported as rare in cats in contrast to dogs.[8] Contradicting this Williams et al have reported that lens opacities are common in diabetic cats, occurring in the vast majority of animals. This is however not seen in clinical practice and the reason is that in diabetic cats most of these opacities are small and linear and do not progress to the typical mature intumescent cataract seen in dogs.[7] The possible reasons for this difference will be addressed in the pathophysiology section of this article.

Anatomy and physiology of the lens

The normal lens is a transparent, avascular, biconvex structure within the eye. Histologically, the lens comprises three major components: the capsule, the anterior epithelium, and lens fibres. Throughout life, continuous fibre addition occurs in the equatorial region, depositing new layers on top of existing ones and consequently displacing older fibres towards the nucleus.[5,9]

The normal lenticular metabolism of glucose to carbon dioxide and water occurs predominantly through anaerobic metabolism, given both the avascular nature of the lens and

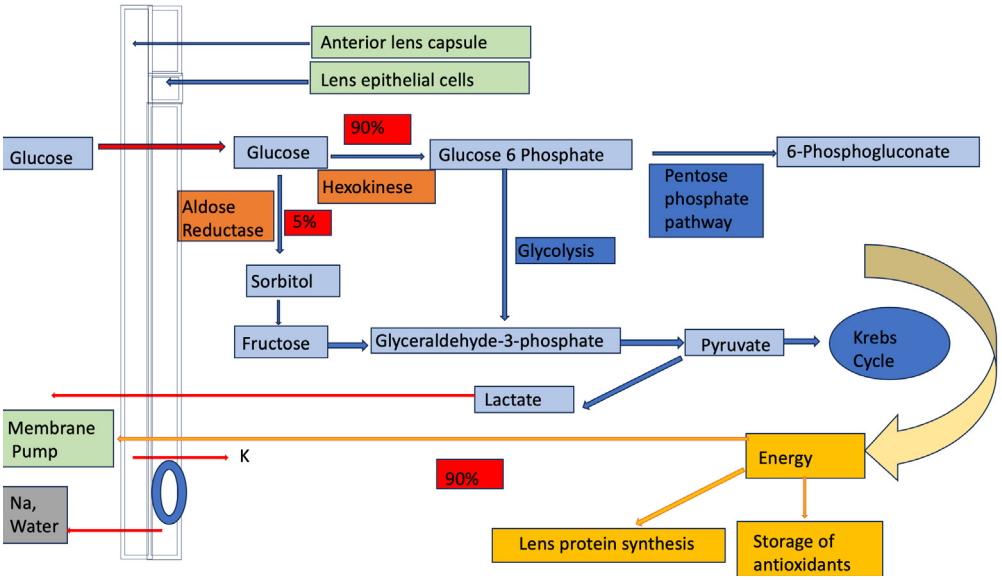

Figure 1 Overview of the major pathways of glucose metabolism and consumption of energy in the lens. Percentages represent the estimated amounts of glucose used and energy consumed in the different pathways.

its lack of mitochondria in all but the lens epithelium. The metabolic needs of the lens are met by the aqueous humor.[10] Glucose enters from the aqueous by both diffusion and active transport. Most of the glucose is broken down to lactic acid via the hexokinase pathway, although some aerobic glycolysis occurs via the citric acid cycle (see Fig. 1) .[5,9,10]

Pathophysiology of diabetic cataracts

In diabetic patients, the aqueous humor concentration of glucose increases leading to increased glucose in the lens. However, when glucose in the lens reaches a concentration where the enzyme hexokinase is saturated, aldose-reductase can convert the excess glucose into sorbitol which has a higher osmotic potential than glucose. This osmotic gradient draws water into the lens, resulting in the rapid development of cataracts. Therefore, the development of a diabetic cataract depends largely on the activity of aldose-reductase in lenticular cells.[5,11] The rate and severity of cataract development are directly proportional to the intracellular accumulation of sorbitol or galactitol.[12]

The relatively low incidence and lack of reaching maturity of feline diabetic cataracts compared to dogs suggests alternative mechanisms at play in cats. One potential explanation lies in species differences in aldose-reductase activity, the enzyme responsible for converting glucose to sorbitol. The aldose-reductase activity is significantly higher in cats younger than four years compared to older cats. Because diabetes mellitus occurs primarily in elderly cats, the relatively low aldose-reductase activity of older cats protects the lens from cataract formation.[5,7,8]

Beyond the mere osmotic effects of sorbitol, recent evidence suggests its direct interaction with lens proteins, leading to amino acid modifications and protein insolubilization may contribute to cataract formation.[7,8]

Protein glycation, a non-enzymatic reaction between sugars and proteins, emerges as another potential culprit. This process induces conformational changes in proteins, exposing previously buried sulfhydryl groups. Lysine, a susceptible amino acid, readily reacts with free carboxyl groups, which are abundant in cells, to form Schiff bases, initiating glycation. If this reaction progresses to form stable Amadori products, protein unfolding and cross-linking into aggregates occur. Glycation further makes the proteins more sensitive to oxidative stress caused by lowered pH and visible light.[8] The continuous exposure of the lens to light is compounded by oxygen immersion. Oxygen's reduction to water necessarily proceeds through the intermediary superoxide radical, O_2. Thus, the initially benign O_2 molecule transforms into a highly reactive free radical. Research utilizing laboratory rodent models has demonstrated that heightened oxidative stress associated with increased reactive oxygen species (ROS) accelerates cataract formation.[13]

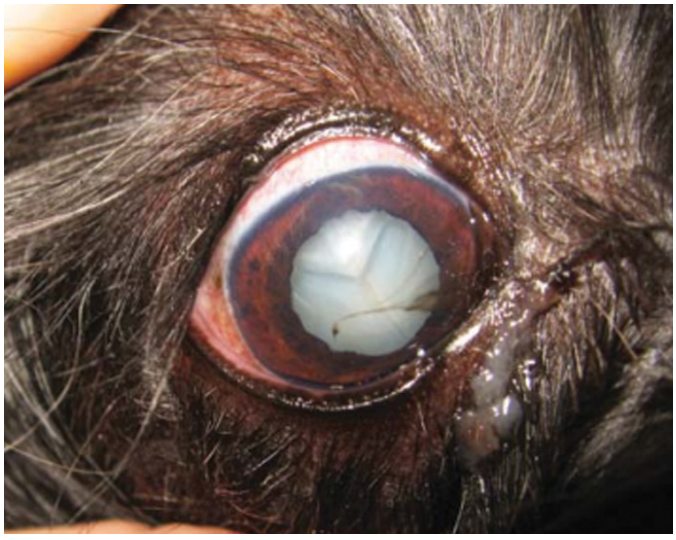

Figure 1 Mature diabetic cataract. Lens enlarged with "clefting" of lens material in association with intumescence is visible. Source: Clinical atlas of canine and feline ophthalmic disease. Douglas W Esson

Clinical signs and complications

In dogs, the classic presentation is one of very acute onset rapidly progressive, and bilaterally symmetrical cataracts.[14] Depending on the amount of ingressing fluid, a diabetic cataract may swell dramatically, a phenomenon known as an intumescent cataract.[5]

The intumescent (swollen) lens displaces the iris forward, thus increasing the posterior chamber pressure and causing the base of the iris to shift forward. This in turn narrows the iridocorneal angle and impinges on the ciliary cleft opening resulting in phacomorphic glaucoma in the dog.[9]

Lens induced uveitis (LIU) is another common complication seen in canine diabetic cataract patients. Traditionally LIU was considered a cell-mediated rejection of lens proteins resulting from the fact that the lens proteins are sequestered from the immune system by the lens capsule preventing these proteins from being recognized as "self".[15.]

In human ophthalmology, this theory is being questioned because lens crystallins have been detected in normal aqueous humor and are thought to be only weakly antigenic.[15] Whatever the exact pathogenesis lens proteins can elicit an inflammatory response in the uvea.[14,15,16] There are two forms of LIU in dogs namely phacolytic uveitis and phacoclastic uveitis. In cats only phacoclastic uveitis has been described and in cats it is usually the result of traumatic laceration of the lens capsule.[15]

Phacolytic uveitis

Phacolytic uveitis is associated with rapidly developing or hypermature cataracts and is characterized by leakage of soluble lens protein through an intact lens capsule. The inflammatory response is typically mild and composed primarily of lymphocytes and plasma cells.[9,15] A more severe form of phacolytic uveitis is granulomatous LIU: This form typically occurs in older dogs with rapidly progressing cataracts, particularly in the Miniature Schnauzer breed. Despite an intact lens capsule, these eyes exhibit significant uveitis, frequently accompanied by the presence of large keratitic precipitates. Unfortunately, this granulomatous form also demonstrates reduced responsiveness to therapeutic interventions.[9]

Diagnosis of both forms of phacolytic uveitis typically relies on a presumptive approach, with the definitive diagnosis hinging on the observation of cataracts in conjunction with the absence of other concurrent ocular or systemic pathologies.[9]

Phacoclastic uveitis

Phacoclastic uveitis ensues after the rupture of the lens capsule, resulting in abrupt exposure to a substantial quantity of lens protein. This exceeds the innate, low-level T-cell tolerance to lens proteins, triggering an inflammatory response.[16] Histologically it is characterized by a suppurative to lymphocytic perilenticular inflammation centred around the break in the lens capsule with intralenticular neutrophils.[15]

Lens capsule rupture may occur as a result of penetrating trauma, in relation to specific developmental disorders, an inadvertent complication of cataract surgery, or spontaneous in the canine lens as a result of lens intumescence secondary to acute onset of diabetic cataract.[14] Wilkie et al. reported that dogs with spontaneous lens capsule rupture represented 9% of all dogs with diabetes mellitus presented to Ohio State University for cataract surgery during the research period.[14] The dogs presented to them were on average diabetic for 123 days and had cataracts for 39 days.[14]

Spontaneous lens capsule ruptures typically occur equatorially (Fig 2) . The anterior chamber often appears asymmetrically shallow and, in most cases, free lens material can be visualized in the anterior chamber. Severe clinical signs of uveitis are always present.[14]

Lens luxation is another possible complication of diabetic cataracts. Lens shrinking occurs in long-standing cataracts and may lead to secondary zonular stretching and breakage. Additionally, the increased weight of the cataractous lens caused by a decrease in the amount of low-molecular-weight proteins of the lens and an increase in that of high-molecular-weight proteins leads to subsequent lens instability.[17]

Increased levels of matrix metalloproteinases and inflammatory mediators in chronic uveitis can also degrade the zonules.[17]Diabetic dogs are more likely to have vascular disruption and the severity of structural abnormalities within the iris vasculature is associated with serum fructosamine concentrations. The result is that poor glycaemic control is associated with an increased risk of complications.[17]

Lipid-laden aqueous humor (LLA) is a clinically recognized condition potentially associated with hyperlipidaemic disorders. These disorders include postprandial hyperlipidaemia, primary idiopathic hyperlipidaemia, and secondary hyperlipidaemia arising from underlying conditions like diabetes mellitus. Notably, diabetes mellitus can exacerbate primary hypertriglyceridemia through mechanisms involving reduced lipoprotein lipase production and subsequent impaired clearance of triglyceride-rich lipoproteins.[18]

The blood-aqueous barrier (BAB), comprised of the endothelium of iridal vessels and the non-pigmented ciliary epithelium, normally restricts the passage of large lipid-laden molecules into the aqueous humor. However, in cases with compromised BAB integrity and concurrent hyperlipidaemia, the likelihood of lipid leakage into the aqueous humor significantly increases. This phenomenon is particularly prevalent in Miniature Schnauzers experiencing diabetic cataracts and secondary LIU, with LLA constituting a frequent observation in this specific breed.[18]

Diabetic cataracts in cats develop slowly and do not typically lead to intumescent cataract seen in dogs. Complications associated with diabetic cataracts in dogs also occur infrequently in cats.[8]

Treatment

There are two main aims in the treatment of diabetic cataracts namely restoration of vision and prevention/ reduction of diabetic cataract-associated complications as discussed previously. Phacoemulsification surgery with or without intra-ocular lens replacement is the best way to restore vision in cataract patients.[9]Cataract surgery in diabetic dogs presents a more complex decision-making process for both veterinarians and dog owners compared to non-diabetic cases. Disruptions in glycaemic control can occur secondary to pre-operative fasting, anaesthetic effects, and postoperative topical corticosteroids.[17] Additionally, diabetic dogs exhibit an increased susceptibility to intraoperative hypotension during phacoemulsification and commonly have concurrent systemic disorders that further elevate anaesthetic risks.[19]

Gemensky-Metzler et al reported the incidence of keratoconjunctivitis sicca (KCS) in diabetic versus non-diabetic dogs prior to surgery to be almost similar namely 10% and 8% respectively.[20] However, within the post-operative time frame, KCS was diagnosed in nearly twice as many diabetics as nondiabetics (27.4% vs. 15.4%).[20] Based on the results of this study monitoring of tear production and the use of artificial tear supplements post-operatively are indicated in diabetic patients post phacoemulsification. These factors, coupled with the potential financial burden associated with surgery and subsequent management, may lead some owners to opt for medical therapy.

The high incidence of spontaneous lens capsule rupture and severe phacoclastic uveitis in dogs with diabetic cataracts is the main reason why referral and surgical intervention should be discussed with the owners of diabetic dogs with cataracts. Should lens capsule rupture occur preoperatively prompt removal of the lens is associated with a favourable outcome.[14]

Surgical removal of lenses does not eliminate the need for future/lifelong topical treatment to control uveitis. According to a study by Lee et al, 94.0% of patients received at least one ophthalmic solution at three months following treatment, 84.1% of patients required treatment for the first six months post operatively and 62.5% at three years following treatment.[17] Post operative medications included one or more of the following: corticosteroids to control uveitis, immunomodulating lacrimo-stimulants (cyclosporine and tacrolimus) for KCS, hyperosmotic agents (5% sodium chloride) for corneal endothelial decompensation secondary to the surgery, ocular hypotensive agents for ocular hypertension and ethylenediaminetetraacetic acid for corneal degeneration.

Medical treatment of diabetic cataracts is essential to prevent possible serious complications that may ultimately require an enucleation. Should a conservative approach be followed patients must be started on topical anti-inflammatory medications soon as cataracts are visible and patients must be regularly monitored. These patients are also at risk for the development of KCS and glaucoma. Should these occur treatment must be started promptly.[17,20]

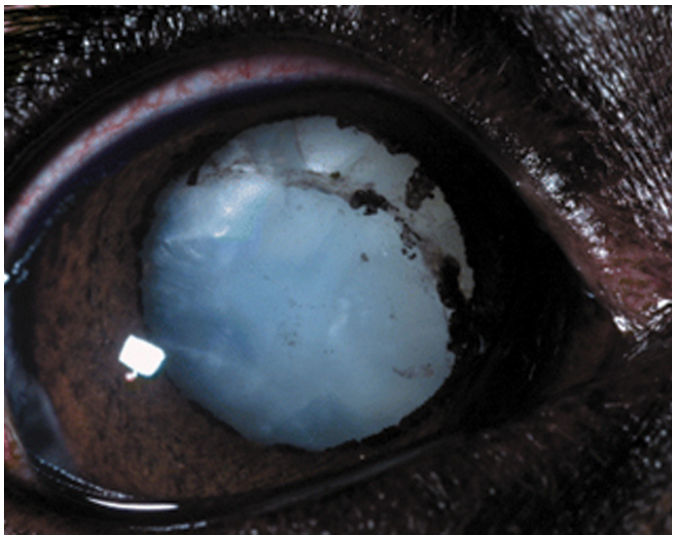

Figure 2 An equatorial lens capsule rupture in a patient with a mature diabetic cataract. The rupture extends from 11 to 4 o'clock. Pigment is present on the lens capsule and within the lens cortex.

Source: Wilkie DA, Gemensky-Metzler AJ, Colitz CMH, Bras ID, Kuonen VJ, Norris KN, Basham CR. 2006. Canine cataracts, diabetes mellitus and spontaneous lens capsule rupture: a retrospective study of 18 dogs. Veterinary Ophthalmology. 9(5):328–334.

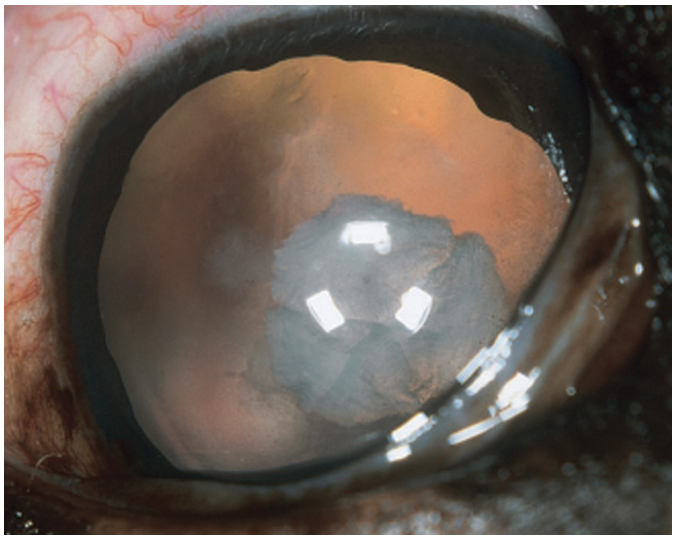

Figure 3. Extensive resorption of a hypermature cataract. Cataract material only remains in the nucleus and has sunk ventrally in the capsular bag due to liquefaction of the cortex. The tapetal reflex is partially visible.
Source: Gonzalez-Alonso-Alegre E, Rodriguez-Alvaro A. 2005. Spontaneous resorption of a diabetic cataract in a geriatric dog. Journal of Small Animal Practice. 46(8):406–408

Lee et al. compared the outcome of surgical and non-surgical treated dogs with diabetic cataracts. In the surgical group, 94.8% of eyes had restored vision at the end of the study period compared to 7.6% of eyes in the non-surgical treatment group. The outcome for the surgical treatment is similar to the 96 % success rate reported Brikshavana[20] but

higher than the 86 % reported by Edelmann ML et al. in a large retrospective study in 182 eyes.[21]

The resorption of cataracts originates in hypermature cases, where degenerative enzymes released from disintegrating lens fibres break down the cataractous material. These enzymes penetrate the intact lens capsule, leading to a reduction in lens size and a characteristically wrinkled or irregular anterior lens capsule.[9,23] As resorption progresses, the tapetal reflection becomes partially visible, particularly in cortical areas (Fig 3). Hypermature cataracts invariably exhibit some degree of liquefaction of lens proteins, with microscopic evidence of resorption in most cases. Lens-induced uveitis (LIU) is a common complication in eyes experiencing spontaneous cataract resorption, caused by the leakage of soluble lens proteins through the intact capsule.[9,23] This process is invariably associated with iridocyclitis, which can be challenging to manage medically. Unfortunately, there is no reliable method to predict whether a cataract will undergo resorption, nor the extent of potential vision restoration in affected animals.[23]

Prevention

As discussed earlier, lens opacification in diabetic canine patients occurs primarily through the generation of sorbitol, through the action of aldose-reductase (AR). Inhibition of AR should thus prevent the generation of cataracts.[24,25]

Kador et al. have shown that topical administration of an aldose-reductase inhibitor Kinostat™ can reduce the incidence of cataract formation in dogs.[25] Unfortunately this product is currently not commercially available. Williams et al have shown that alpha lipoic acid, given orally at a dose of 2mg/kg daily potentially acts as both an antioxidant and aldose-reductase inhibitor, delays and possibly prevents the onset of cataracts in diabetic dogs.[24] As seen earlier AR reductase does not play an important role in the development of diabetic cataracts in cats and therefore AR inhibitors are not indicated in this species.

Conclusion

Diabetic cataracts remain a serious sight threatening as well as potentially painful ocular condition in dogs. Most diabetic dogs will develop cataracts despite proper control of the underlying diabetes. Preventative treatment with aldose-reductase inhibitors may prevent the onset of diabetic cataracts, but as soon as signs of cataract development is visible immediate treatment with topical anti-inflammatory products must be started. If cataract surgery is an option early referral should be recommended. Patients must be monitored regularly for possible development of KCS and glaucoma and treatment initiated as soon as possible.

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CPD Questions

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1. Which one of the following statements regarding diabetes mellitus is CORRECT?

a. The prevalence of diabetes in dogs is two times higher than cats.
b. Both dogs and cats are more likely to suffer from insulin deficiency diabetes compared to insulin resistance diabetes.
c. Burmese cats are a 3x more likely to develop diabetes compared to other breeds.
d. Unneutered male dogs are more likely to develop diabetes mellitus.
e. Border collies are predisposed to diabetes mellitus.

2. What percentage of diabetic dogs develop diabetic cataracts within the first 6 months after diagnosis?

a 25 %
b. 35 %
c. 50 %
d. 60 %
e. 80 %

3. Which one of the statements below is most CORRECT to answer the question, What is most of the energy produced in the lens used for?

a. Supplying energy to a membrane Na-K pump maintaining the dehydrated state of the lens.
b. Lens protein synthesis.
c. Storage of anti-oxidants.
d. Maintenance of the anterior lens epithelial cells.
e. Maintaining mitotic activity of the lens equatorial cells

4. Which one of the statements below is the most likely reason why intumescent cataracts are more likely to form in dogs compared to cats?

a. Higher concentrations of aldose-reductase in dogs compared to cats.
b. Higher concentrations of Lysine in the canine lens.
c. The increased chance of the formation of Schiff bases and Amadori products on dogs.
d. The increased incidence of lens-induced uveitis in dogs alters the hydration state in the canine lens.
e. Lower concentrations of hexokinase in the canine lens.

5. Which one of the following statements is not a possible complication of diabetic cataracts?

a. Phacolytic lens induced uveitis.
b. Keratoconjunctivitis sicca.
c. Phacoclastic lens induced uveitis.
d. Phacomorhic glaucoma.
e. Lens luxation.

6. Which one of the following five listed substances have shown promise in reducing the incidence of diabetic cataract formation in dogs ?

a. Topical corticosteroids.
b. Topical non-steroidal anti-inflammatories.
c. Alpha lipoic acid.
d. Geen tea extract.
e. Lutein.

7. Which one of the following statements regarding the treatment of diabetic cataracts is INCORRECT ?

a. Phaco emulsification leads to an increased incidence of keratoconjunctivitis sicca.
b. Up to 7 % of patients may regain vision with medical treatment alone.
c. Phaco emulsification of lenses that had spontaneous capsular rupture has a very poor prognosis.
d. Surgical treatment of diabetic cataracts eliminate the need for long term treatment.
e. Successful outcome of phacoemulsification in diabetic cataracts can be high as 95 %.

8. Which one of the following statements regarding glucose metabolism in the lens is true ?

a. Hexokinase is responsible for converting glucose to glucose-6-phosphate.
b. The normal lenticular metabolism of glucose is predominantly through aerobic metabolism.
c. The pentose phosphate pathway does not play a role in lenticular metabolism.
d. Aldose-reductase play a major role in the normal metabolism of the lens.
e. Glucose only enters the lens by active transport.

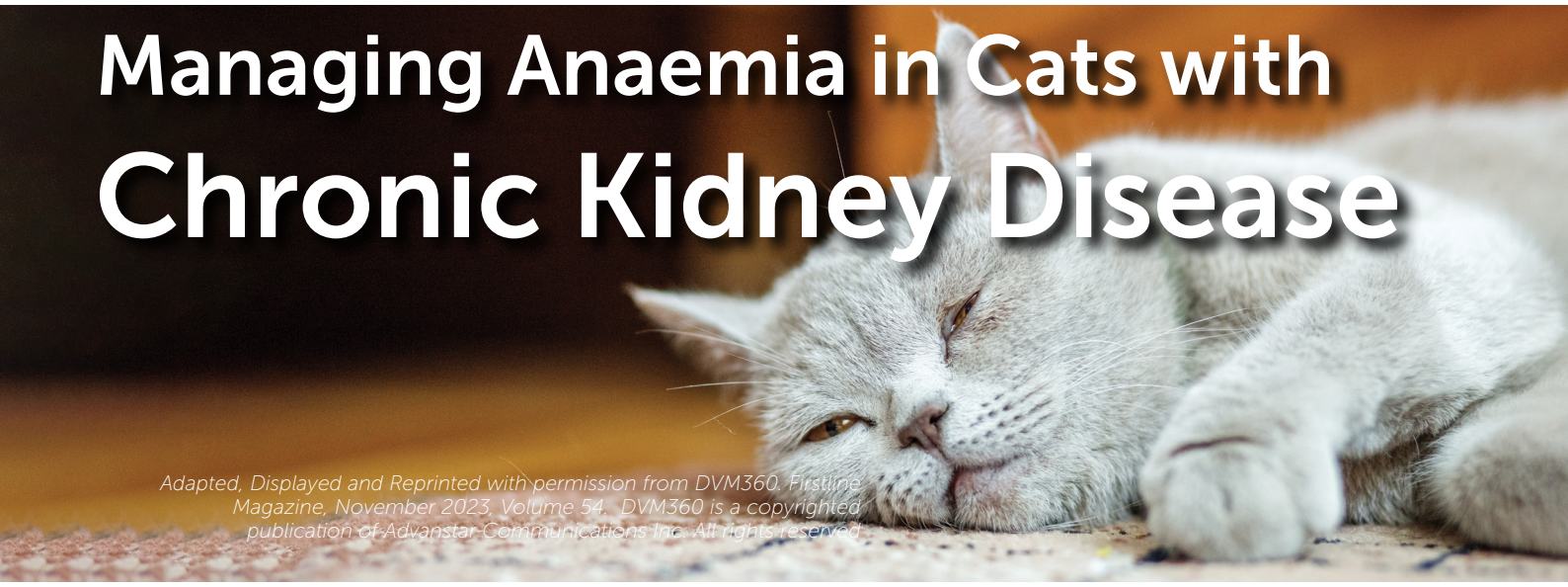
9. Regarding diabetic cataracts in cats which one of the following statements is CORRECT?

a. In cats most cataracts small and linear and do not progress to the typical mature intumescent cataracts.
b. Phacolytic uveitis is common is diabetic cats.
c. Spontaneous lens capsule rupture in cats leads to phacoclastic uveitis.
d. The antioxidants lutein and zeaxanthins may prevent the development of diabetic cataracts in cats.
e. Phacomorphic glaucoma is a serious and common complication of diabetic cataracts in cats.

10. Which one of the following statements regarding lens-induced uveitis (LIU) is CORRECT?

a. Phacolytic uveitis is associated with immature diabetic cataracts in dogs.
b. Granulomatous LIU is a very mild form of uveitis and seldom causes visual deficits.
c. A breed predisposition exists in miniature Schnauzers for the development of granulomatous uveitis.
d. Unlike infectious diseases leading to keratitic precipitates [KP's] in uveitis patients KP's are never seen in lens-induced uveitis patients.
e. None of the above.

Managing Anaemia in Cats with Chronic Kidney Disease



Veterinary professionals gain a better understanding of the condition's pathogenesis and new treatment strategies

Up to 50% of cats will be diagnosed with chronic kidney disease (CKD) in their lifetime, and for 17% of geriatric cats, this will be the cause of death.[1,2] A common comorbidity seen in 65% of these patients is anaemia,[3] which becomes more severe as CKD progresses and can have a significant impact on quality of life.

Shelly Vaden, DVM, PhD, DACVIM, a founding member of the American College of Veterinary Nephrology-Urology, reviewed the current understanding of the pathogenesis of this anaemia and available treatment options, during a session sponsored by Elanco at the 2023 Fetch Coastal conference in Atlantic City, New Jersey.[4]

Anaemia of CKD affects patient survival and quality of life

The presence of anaemia in cats with chronic kidney disease is associated with lower quality of life, more rapid progression of CKD, and shorter survival times. Clinical signs of cats with anaemia of CKD include lethargy and social isolation (hiding and decreased owner interaction). Vaden has seen cats who receive treatment for their anaemia have increased energy, owner interaction, and overall quality of life.[4]

According to Vaden, historically, "we [have delayed] treatment for a disorder that makes animals feel better once treated." In a retrospective study of 211 cats with chronic kidney disease (serum creatinine >175 umol/L) median survival from the time of diagnosis was 771 days.[5] However, those cats with a packed cell volume (PCV) less than 25% had a median survival of 100 days, and for the 42 cats that received treatment for their anaemia, median survival was only 25 days.[5] Vaden concluded that "we are intervening late with anaemia of CKD. Probably because we haven't had great drug options."

Pathophysiology: More complex than a lack of erythropoietin

The pathophysiology of anaemia of CKD is multifactorial and more complex than once thought. Traditionally, the lack of erythropoietin (EPO) production by the kidneys was assumed to be the primary mechanism. Although this is a contributing factor, proposed additional mechanisms include[4]:

1. Iron deficiency because of changes in iron regulation and transportation
2. Increased fragility of red blood cells in a uraemic state
3. Nutritional deficiencies including copper, zinc, cobalamin, and folate
4. Secondary bone and mineral disorders
5. Treatment factors including aluminium overload and use of RAAS inhibitors.

"We can't talk about anaemia without talking about iron," said Vaden. The iron deficiency that occurs in CKD is often thought to be a functional deficiency, meaning that iron is present in the body but is not accessible for use. Iron has many functions in the body, including red blood cell production. Iron levels are tightly regulated. One major regulator is *hepcidin*, a protein, which inhibits iron absorption and transport and promotes sequestration of iron in storage.[4] (see info box)

Another consideration in the pathophysiology is worsening anaemia through the use of other medications used for CKD, in particular aluminium hydroxide as a phosphate binder. "Once you get to the point of an anaemic patient, I'm not sure that aluminium hydroxide is the best choice," said Vaden. Aluminium and iron both bind to transferrin for transport, so increased aluminium in the body can block iron transport, thus further exacerbating functional

iron deficiency and anaemia. The use of ferric citrate, which serves as both a source of iron supplementation and phosphate binder, should be considered as an alternative therapy.[4]

Treatment with erythropoietin stimulating agents

Historically, erythropoietin stimulating agents (ESA) have been used to manage anaemia of CKD. Cats treated with recombinant human EPO have shown increases in haematocrit and improved quality of life, including better appetite, energy, body condition, strength, and activity levels.[6] However, Vaden noted that the risk of antibody formation and subsequent anaemia is high and, therefore, using human EPO is not recommended.

Darbepoetin, a second-generation ESA, has been the preferred treatment because of its longer half-life and decreased risk of pure red cell aplasia. However, not all patients will respond, and non-responders are more likely to have concurrent diseases.[7] Adverse events in cats include vomiting, fever, and exacerbation of hypertension.[7] Some seizures in cats can be caused by this hypertension. Pure red cell aplasia is rarely seen with darbepoetin use.

Arecombinant feline EPO product is being studied but is unlikely to become commercially available. New research into an adeno-associated virus vector containing the gene for feline EPO is currently under investigation and may offer a one-time treatment option.[8] However, it too is associated with hypertension exacerbation and seizures.

A new medication for treating anaemia of CKD in cats

A new class of drugs, hypoxia-inducible factor-prolyl hydrolase (HIF-PH) inhibitors, may offer better outcomes for anaemia of CKD in cats. HIF stimulates endogenous EPO production and mobilizes iron by blocking hepcidin and increasing transferrin, a protein necessary for iron transport. The drug prevents the degradation of HIF, prolonging its effects in the body.[4]

Molidustat oral suspension (Varenzin-CA1; Elanco) has received conditional approval from the FDA for control of nonregenerative anaemia associated with CKD in cats. It is administered by mouth once daily for up to 28 days followed by a 7-day treatment break to avoid development of polycythemia. Weekly monitoring of PCV is recommended starting 14 days after the beginning of treatment. Because the drug is conditionally approved, it may not be used off-label. The most common adverse effect is vomiting. Additionally, changes in serum potassium and exacerbation of hypertension can be seen.[4]

Iron supplementation

Iron supplementation is recommended for any patients who are being treated for anaemia. While it is likely that most cats have a functional iron deficiency, this could become an absolute deficiency once iron is mobilized and utilized with treatment for anaemia. It is currently difficult to accurately measure iron levels with available laboratory testing. "Right now, we treat them as if they are iron deficient," said Vaden.

What Is Hepcidin?

Hepcidin, is a peptide produced by the hepatocytes, which the ds emerged as the key regulator of uptake and release of iron in the tissues to maintain a steady supply of iron to erythron and other tissues while avoiding higher levels of iron that could be detrimental to the organs. Hepcidin itself is regulated by the supply of iron, the need for erythropoiesis, and the state of inflammation. Alterations in hepcidin levels are associated with restricted erythropoiesis, anaemia, and iron overload.

Discovery of hepcidin and understanding of its mechanism of action and the consequences of its upregulation and suppression have given valuable insight into many haematologic disorders including the anaemia of CKD.

This knowledge has also unlocked unique opportunities to modulate hepcidin via agonists and antagonists, and its feedback pathways to treat clinical conditions.

Source: K. Agarwal, Jerry Yee . Advances in Chronic Kidney Disease, Volume 26, Issue 4, July 2019, Pages 298-305

Supplementation can be provided parentally or orally.[4]

Take home points

- Anaemia of chronic kidney disease is multifactorial. While treatment with erythropoietin stimulating agents has historically been used, newer medications are now available.
- Molidustat oral suspension is conditionally approved (in the USA) to control non-regenerative anaemia associated with CKD in cats. Its novel mechanism promotes stimulation of endogenous erythropoietin and mobilizes iron from storage sites in the body.
- Regardless of how anaemia of CKD is treated, Vaden encouraged veterinarians to discuss treatment with their clients as cats who are treated for their anaemia have a better quality of life.
- Iron supplementation is encouraged as part of the treatment plan to prevent iron deficiencies from developing.[4]

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References available on request

Stepwise Approach
to Diagnosing Immune-Mediated Haemolytic Anaemia

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There is no one gold standard test that has a high enough sensitivity and specificity to diagnose IMHA. Instead, a combination of clinical signs and clinicopathological abnormalities is needed to recognise this condition

Immune-mediated haemolytic anaemia (IMHA) is a type of anaemia that is caused by the formation of antibodies against erythrocyte auto-antigens. This condition most commonly affects dogs with cats, horses, and cattle affected to a lesser degree.

Most IMHA-related deaths occur within in the first two weeks, usually due to complications such as thrombosis due to hypercoagulability, systemic inflammatory response syndrome(SIRS) and liver or kidney failure. Thus early recognition of IMHA is important in order to mitigate these complications.

In 2019, the American College of Veterinary Internal Medicine (ACVIM) published a consensus statement on the diagnosis of IMHA in dogs and cats. This was based on published studies and case reports with input from a panel of experts. Overall, the panellists agreed that there is no one gold standard test that has a high enough sensitivity and specificity to diagnose IMHA. Instead, a combination of clinical signs and clinicopathological abnormalities is needed to recognise this condition. Using the step-wise approach detailed in this consensus statement, clinicians can apply a degree of certainty to an IMHA diagnosis.

Step 1 – Is there anaemia?

This may seem obvious, but anaemia must first be demonstrated for a diagnosis of IMHA. Anaemia may be recognised by a decrease in haematocrit, measured by an in-clinic or reference laboratory haematology analyser, and/or a decreased packed cell volume (PCV) measured from a centrifuged microhematocrit tube.

Marked erythrocyte agglutination may affect the red cell indices measured by haematology analysers, especially the mean cell volume (MCV) which can be spuriously increased.

If the haematology analyser flags any of the red cell results or cytograms, then the PCV must be measured to confirm the automated haematocrit.

These two results should be within 3% of one another and,

if not, a properly spun PCV will be the more accurate of the two results. Measuring the PCV will also have a secondary benefit, which is the opportunity to visualise the plasma colour. This will be necessary to identify any signs of haemolysis (see step 2).

Regeneration is not necessary for a diagnosis of IMHA; only 62 – 82% of dogs will have a regenerative response at presentation. A non-regenerative IMHA may be due to one of two reasons. Firstly, and most commonly, the bone marrow needs some time to respond to an acute anaemia. This "pre-regenerative" anaemia may last a few days with a peak regenerative response by 4 – 7 days after the onset of anaemia.

Secondly, the auto-antibodies may target reticulocytes or earlier erythrocyte precursors in the bone marrow in a condition called precursor-targeted immune-mediated anaemia (PIMA).

As a result, the anaemia will remain non-regenerative or poorly regenerative, even if the bone marrow has had adequate time for a regenerative response. Bone marrow cytology and/or histopathology is required for a definitive diagnosis of PIMA.

An inappropriate regenerative response may also be due to iron deficient or restricted erythropoiesis (IDE). In some dogs with IMHA, the stimulus for accelerated erythropoiesis is so strong that the body is not able to mobilise iron stores in the bone marrow, spleen, and liver fast enough to keep up with the demand.

This transient phenomenon results in a functional iron deficiency, which limits the rate of erythropoiesis. This should be remembered when interpreting a decreased reticulocyte haemoglobin content (RET-He or CHr) in dogs with IMHA.

Once you have established that your canine or feline patient has regenerative anaemia or an anaemia that is rapidly progressive (which is not expected WITH decreased production alone), then you can conclude that the erythrocytes are being lost in whole blood (i.e., haemorrhage) or being destroyed by a haemolytic process, which may be immune-mediated or non-immune-mediated.

Step 2 – Is there evidence of haemolysis?

There must be evidence of haemolysis for a diagnosis of IMHA. This will also help to rule out regenerative anaemia caused by haemorrhage. With haemorrhage, there may be a history of bleeding or obvious bleeding at presentation but occult bleeding may also cause anaemia.

Clues indicating whole blood loss are hypoproteinaemia, hypoalbuminemia, and evidence of IDE in the mature and immature erythrocytes (e.g. microcytosis, hypochromasia, and leptocytosis). Haemolysis may be extravascular or intravascular, and these two pathomechanisms will result in different abnormalities. Extravascular haemolysis is due to macrophage phagocytosis, usually in the spleen, whereas intravascular haemolysis refers to erythrocyte lysis in circulation. Signs of extravascular haemolysis include hyperbilirubinaemia, bilirubinuria, and clinical icterus. Hyperbilirubinaemia has been reported in 60 – 100% of dogs presenting with IMHA.

Bilirubinuria is a cost-effective and sensitive way of screening for hyperbilirubinaemia, as bilirubin may be present in the urine before clinical icterus is present. That said, male dogs have a low renal threshold for bilirubin and healthy dogs may have mild bilirubinuria (1+), especially in concentrated urine.

When signs of extravascular haemolysis are identified it is important to rule out sepsis (which may cause functional hyperbilirubinaemia) and hepatobiliary disease, which may be indicated by increased ALP, GGT, and/or ALT activities, signs of hepatic dysfunction, and/or an abnormal ultrasonographic appearance of the liver and gall bladder.

Signs of intravascular haemolysis include ghost erythrocytes, haemoglobinaemia, and haemoglobinuria. Ghost erythrocytes are very pale staining erythrocytes that have lysed.

These erythrocyte membranes are rapidly cleared from circulation and thus, their presence indicates recent or ongoing intravascular haemolysis. Ghost erythrocytes are best identified on a freshly prepared blood smear using a 100x oil objective.

Haemoglobinaemia should also be interpreted in light of the sample collection method and storage. If there is mild haemolysis and platelet clumping and/or the blood collection was difficult, then haemolysis may be iatrogenic. Iatrogenic haemolysis may also occur with delayed sample processing, blood collection via intravenous catheters, and sample freezing (e.g., tube was in direct contact with an ice pack).

Finding haemoglobinuria on urinalysis of a fresh urine sample (i.e., the dipstick is positive for haemoglobinaemia without intact erythrocytes in the urine sediment) it will be useful to confirm that any haemoglobinaemia is a pathological change. If there are other erythrocyte abnormalities indicating haemolysis due to oxidative injury (apart from spherocytes; e.g., Heinz bodies, eccentrocytes) or erythrocyte fragmentation (e.g., keratocytes, schistocytes, acanthocytes), then haemolysis due to a non-mediated mechanism should be considered rather than IMHA.

Icterus and haemoglobinaemia may affect the haematology analyser results; they most notably cause a spurious increase in the MCHC. Ghost erythrocytes and Heinz bodies may be mis-identified as platelets by the analyser, which will spuriously increase the platelet concentration.

Step 3 – Is there evidence of immune-mediated erythrocyte destruction?

The next diagnostic step is to demonstrate an immune-

mediated mechanism to the anaemia. This can be done in three ways: a high number of spherocytes, positive ISA test, and positive Coomb's test.

(i) Spherocytes:

This characteristic is only applicable to dogs and may be present in 61 – 95% of cases. Canine erythrocytes typically have central pallor, making it easy to identify smaller dark-staining spherocytes with no central pallor. Normal feline erythrocytes do not have obvious central pallor, so spherocytes are not easily recognised in cats.

A well-made blood smear with a red cell monolayer is needed to assess the presence of spherocytes in dogs. This monolayer is behind the feathered edge, where the red cells have an obvious central pallor and are spread out without touching. The presence and number of spherocytes may be over- or underestimated without this monolayer.

Short smears (e.g., peripheral smears using a small drop of blood) often lack such an area so it is worthwhile to make a blood smear using whole blood from an EDTA tube.

Once the blood smear is dried, the red cell monolayer has an iridescent quality when holding the slide up to the light.

An average of ≥5 spherocytes in each high-power field (HPF; using the 100x oil objective) is supportive for IMHA. An average of 3 – 4 spherocytes/HPF may also be supportive, if no other cause of spherocytosis is present.

These causes include blood transfusions with stored blood, oxidative damage (e.g., zinc toxicity), bee or snake envenomation, increased erythrocyte phagocytosis by macrophages (e.g., haemophagocytic histiocytic sarcoma), congenital haemolytic diseases, erythrocyte fragmentation (e.g., haemangiosarcoma), and iron deficiency.

Usually, there is relevant history or other erythrocyte morphological abnormalities (e.g., eccentrocytes, Heinz bodies, microcytosis) that may point to these conditions. These conditions should also be considered if both the ISA and Coomb's tests are negative.

(ii) Positive in-saline agglutination test (ISA) test:

Agglutination may be obvious on a blood smear (microagglutination) or within the EDTA tube (macroagglutination); 42 – 86% of dogs with IMHA may show microagglutination. However, an ISA test is still advised to rule out rouleaux.

The key to a good ISA test is being precise with your saline-to-blood ratios, and for this reason, it is best to use a known volume of room temperature (not refrigerated) blood from an EDTA tube.

Drops of blood and saline can also be used, but then the dropper must be the same; for example, using a drop of blood from a needle or syringe and dropping saline from a dropper bottle is not going to be accurate!

The ISA test is a great tool to screen for agglutination, especially when the test is standardised. The premise of this test is that the bonds between the anti-erythrocyte antibodies (IgG or IgM) and the erythrocytes themselves should not be broken with saline.

Conversely, the weak bonds with rouleaux will be dispersed with saline; these bonds form between erythrocytes due to changes in membrane charge (e.g., if they are coated with globulins).

Using a saline-to-blood ratio of 4:1 (e.g., 1 mL of blood in 4 mL saline) has a high sensitivity (88%) but a variable specificity depending on the study (49 – 100%). In other words, the likelihood of a dog or cat still having IMHA with a negative 4:1 ISA test is low (~12% of cases).

False negatives may be due to excessive agitation breaking up antibody bonds, complement-mediated haemolysis (see "Positive Coomb's test" for more information), and very high numbers of antibodies (especially IgG) resulting in less cross-linking between erythrocytes. If a Coomb's test cannot be performed (e.g., financial constraints), then an ISA test using a larger volume of saline may be used as a confirmatory test.

A saline-to-blood ratio of 49:1 (e.g., 0.1 mL of blood in 4.9 mL saline) has high specificity (97%), meaning very few false positives are expected. A washed ISA test may also be performed if a Coomb's test cannot be used. For more on a washed ISA tests, please refer to the ACVIM consensus statement listed in the references.

(iii) Positive Coomb's tests:

A positive Coomb's test, also known as the direct antiglobulin test (DAT), will also be supportive of IMHA. Ideally, this test should not be used for screening; i.e. it should not be the first test you run when working up a patient with anaemia.

The lower sensitivity of the Coomb's test (61 – 82%) means that a significant number of IMHA patients (18 – 39%) may be missed with a negative Coomb's test. Rather, a Coomb's test should be used to confirm a positive 4:1 ISA test or to demonstrate an immune-mediated mechanism when IMHA is still suspected after a negative 4:1 ISA test.

The advantage of the Coomb's test is that it also detects complement proteins attached to erythrocytes and not just antibodies linking erythrocytes, as with the ISA test. Complement proteins are pro-inflammatory proteins that, when adhered to the erythrocytes, will interact with antibodies, enhance macrophage phagocytosis, or cause intravascular haemolysis.

There are also disadvantages to the Coomb's test. Firstly, this test is species-specific (e.g., a human Coomb's test cannot be performed with canine blood). Another disadvantage is apparent when this test is used as a screening test before ensuring that the patient has a regenerative anaemia due to haemolysis.

In this case, the performance of the Coomb's test is reduced. In one study, the number of false positives was greater than true positives when a Coomb's test was performed regardless of whether the patient had signs of haemolysis, resulting in a low positive predictive value. Coomb's tests are also only positive in a small number of PIMA cases.

How do I combine steps 1 – 3 for a diagnosis of IMHA?

Using the steps listed above, the combination of findings can be used to support, confirm, or exclude a diagnosis of IMHA. A definitive diagnosis requires ≥2 signs of immune-mediated destruction (spherocytes, positive Coomb's test, and positive ISA test) and ≥1 sign of haemolysis (evidence of hyperbilirubinaemia, haemoglobinuria, haemoglobinaemia, and ghost erythrocytes). If there are no signs of immune-mediated destruction or haemolysis, then IMHA is excluded as a cause of the anaemia.

If some, but not all, of the criteria for a definitive diagnosis are met, then the findings are only supportive or suspicious for a diagnosis of IMHA, provided another cause for the anaemia is not found.

Other common clinicopathological findings

Other common findings in IMHA include leukocytosis (43 – 99% of dogs) and a left shift with circulating band and immature neutrophils (80% of dogs). A band neutrophil concentration of >3 x 109/L is a negative prognostic indicator in dogs. Care should be taken not to confuse a rubricytosis (or increased circulating nucleated erythrocytes) with a leukocytosis.

Most in-clinic and reference laboratory haematology analysers will mistakenly classify nucleated erythrocytes as leukocytes (usually lymphocytes), falsely increasing the leukocyte concentration. This is another important reason to examine a blood smear in addition to running a CBC.

Thrombocytopenia may also be seen (29 – 70% of dogs) and is associated with decreased survival in dogs when the platelet concentration is <150 x 109/L. This may be due to thrombosis (IMHA may result in hypercoagulability) or concurrent immune-mediated thrombocytopenia. Prolonged prothrombin time (PT) and/or activated partial thromboplastin times (aPTT) are also indicative of a coagulopathy and decreased survival in dogs with IMHA.

I have diagnosed IMHA, now what?

A diagnosis of non-associative (formerly known as primary) IMHA is one of exclusion and cannot be made until a full investigation to find any potential triggers or causes is complete.

Possible triggers causing associative (formerly known as secondary) IMHA include canine and feline babesiosis, certain antibiotics (e.g., cefazedone in dogs), feline haemotrophic mycoplasmosis, feline leukaemia viral infection, and neoplasia. In dogs and cats, there is only a low level of evidence to support an association between IMHA and neoplasia. In contrast, the incidence of neoplasia was significantly higher in horses with IMHA compared to those without and investigation for neoplasia is warranted in all horses with IMHA.

The recommended minimum database for any patient diagnosed with IMHA includes:

- History, including vaccination and travel history, and flea and tick prevention
- Physical examination
- Laboratory testing, including a CBC, blood smear examination, clinical chemistry, urinalysis, and faecal flotation.
- Abdominal radiographs and other imaging to screen for neoplasia
- Testing for pancreatitis, if the clinical presentation is suggestive.

Summary

A diagnosis of IMHA is made using of combination of findings including the presence of anaemia, signs of haemolysis, and demonstration of a immune-mediated mechanism.

When more findings are present, a diagnosis of IMHA can be made with more confidence. Once a diagnosis is made, it is important to determine if there are any potential triggers.

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1. Demonstration of which one of the following five findings is necessary for the veterinarian to make a definitive diagnosis of non-associative IMHA?

a. Presence of anaemia.
b. Signs of extravascular and/or intravascular haemolysis.
c. An immune-mediated mechanism.
d. Absence of a potential trigger or cause.
e. All of the above.

2. In which of the scenarios listed below is a Coomb's test indicated?

a. In all anaemic patients.
b. In patients with haemoglobinuria.
c. With a positive 49:1 saline-to-blood ratio ISA test.
d. With a positive 4:1 saline-to-blood ratio ISA test.
e. With a positive washed ISA.

3. Which one of the five abnormalities listed below is not associated with feline IMHA?

a. Spherocytosis
b. Hyperbilirubinaemia
c. Anaemia
d. Regenerative response
e. Haemoglobinuria

4. Which one of the CBC results listed below may be inaccurate when analysing blood from a dog with IMHA?

a. Platelet concentration
b. MCV
c. MCHC
d. Leukocyte concentration
e. All of the above

5. Which one of the scenarios listed below best describes the situation where a 4:1 saline-to-blood ratio ISA test is indicated?

a. To confirm a diagnosis of IMHA.
b. In all anaemic patients.
c. To differentiate iatrogenic and true haemolysis.
d. To differentiate rouleaux and microagglutination.
e. When a Coomb's test cannot be done.

6. Which one of the statements below is INCORRECT when completing the following sentence: The Coomb's test:

a. is species-specific
b. is a good screening test.
c. is a confirmatory test.
d. detects complement adhered to the erythrocytes.
e. detects antibodies bound to the erythrocytes.

7. Which one of the statements listed below is not a valid reason for measuring the PCV in an IMHA patient?

a. To check the automated haematocrit measurement is correct.
b. To examine the plasma for icterus.
c. To identify macroagglutination.
d. To examine the plasma for haemolysis.
e. To identify the presence of anaemia.

8. Which one of the abnormalities listed below is a negative prognostic indicator in dogs with IMHA?

a. Band neutrophilia of >3 x 109/L
b. Thrombocytopenia of <150 x 109/L
c. Prolonged PT and aPTT
d. Clinical signs of a coagulopathy
e. All of the above

9. Which one of the abnormalities/findings listed below is the reason a veterinarian should examine a blood smear in all canine and felline IMHA patients?

a. A left shift
b. Rubricytosis
c. Microagglutination
d. Any infectious agents
e. All of the above

10. Which one of the statements listed below regarding spherocytes in dogs is INCORRECT?

a. The absence of spherocytes rules out IMHA.
b. Spherocytes are best identified in the red cell monolayer.
c. ≥5 spherocytes/HPF is supportive for IMHA.
d. Spherocytes appear smaller and darker without central pallor.
e. Low numbers of spherocytes may be noted with iron deficiency.

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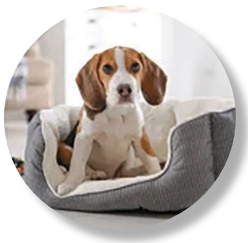

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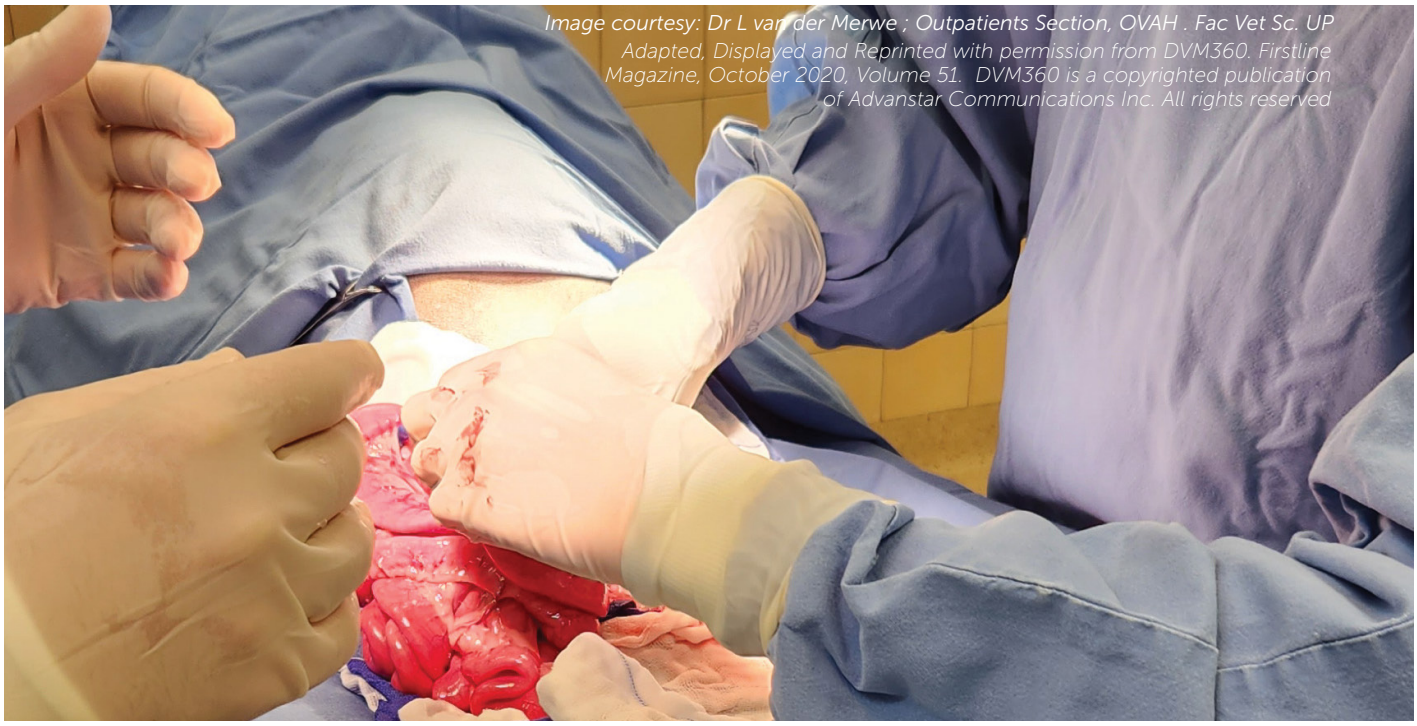

Removing Linear Foreign Bodies in Dogs: Surgical Pearls

Use these tips to make removing linear foreign bodies a little easier.

As anyone who routinely performs gastrointestinal (GI) surgery will attest, all foreign bodies are not created equal. While gastric, and some small intestinal, foreign bodies can be treated successfully in primary care practice, linear foreign bodies can create a much more challenging scenario for the surgeon, and can be potentially life-threatening for our patients. During a recent Fetch® dvm360 conference, Bronwyn Fullagar, BVSc, MS, DACVS, a specialist small animal surgeon based in Canmore, Alberta, offered some great advice for detecting and treating linear foreign bodies in dogs.

"It's important to understand some key techniques for treating them that will greatly improve your patient outcomes," Fullagar said. "In addition to preoperative patient stabilisation, gentle tissue handling, and meticulous suture placement, it is really helpful to have had lots of prior experience performing GI surgery on non-linear foreign body cases before taking on your first linear case. If in doubt, it's never wrong to refer these more challenging cases to a specialist."

1. Look for gas patterns on radiographs

Fullagar began by sharing a few diagnostic tips she learned from a veterinary radiologist. "One feature of linear foreign bodies that can make them trickier than other foreign bodies to diagnose on radiographs is that about 50% don't cause any small intestinal dilation that's visible on radiographs," she explained. But that doesn't mean you'll always need to resort to ultrasonography straight away. You just have to know what to look for, and in the case of linear foreign bodies, it's characteristic comma- or paisley-shaped gas patterns, often combined with evidence of small intestinal plication and/or loss of serosal detail (Figure 1-4). If you're still unsure, abdominal ultrasound is usually the next logical diagnostic step.

2. Take left lateral radiographs

Another radiography tip Fullagar passed along was to always obtain 3 views of the abdomen, including a left lateral radiograph, on any dog you think might have a GI foreign body. "The left lateral shows what's left in the stomach," she explained. "When a dog is in left lateral recumbency, the pylorus, on the dog's right side, will be uppermost. The gas will rise into it and nicely highlight whatever is in the pylorus." In dogs, Fullagar noted, most linear foreign bodies will be anchored in the pylorus.

3. Be prepared for enterectomy and anastomosis

If you suspect a linear foreign body in your patient based on

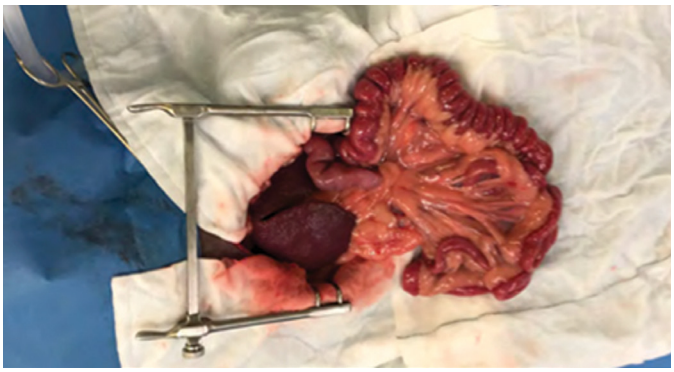

Figure 5. Intraoperative photograph of the dog in Figure 4. There is small intestinal plication. The foreign body is anchored in the stomach and extends to the mid-jejunum.

radiographs or ultrasound, Fullagar stressed that you need to be prepared to do an enterectomy and anastomosis. "About 40% of dogs with a linear GI foreign body will have septic peritonitis at the time of surgery," she said. Enterectomies – anastomoses usually require a surgical assistant and are more technically difficult than an enterotomy. "So, if you're not experienced in assessing the viability of small intestine, or if you haven't done a small enterectomy before, make sure you have either a mentor to help or consider referring the case," she said. "You don't want to get halfway through surgery and think, 'I need to resect some of this intestine, but I've never done it before.'"

4. Make an appropriate surgical approach

Visualisation is key. Start with a coeliotomy incision that's large enough for you to perform a complete exploratory laparotomy. "This isn't an elective procedure that you're doing," Fullagar said. "This is an emergency surgery and good visualisation is very important. So, the incision should usually start at the xiphoid and go to the fourth mammary gland of a female dog, or to the tip of the prepuce of a male dog."

Fullagar also stressed the importance of keeping the abdominal tissues moist throughout the procedure, to avoid postoperative adhesions. "Using moistened abdominal swabs to isolate the part of the abdomen that you're doing surgery on will prevent other structures from drying and will also avoid contamination of the abdomen from GI contents," she said.

5. Release the anchor point first.

In dogs, this usually means starting with a gastrotomy. Although most abdominal surgery begins with exploration of the abdomen, in cases where there is obvious small intestinal plication from a linear foreign body, Fullagar recommends very gentle handling of the intestine initially, and waiting until the foreign body has been removed before completing full abdominal exploration. "What you don't want to do is manipulate this very plicated intestine excessively, because you risk causing more damage to the mesenteric border, which may be compromised by the foreign body," she explained.

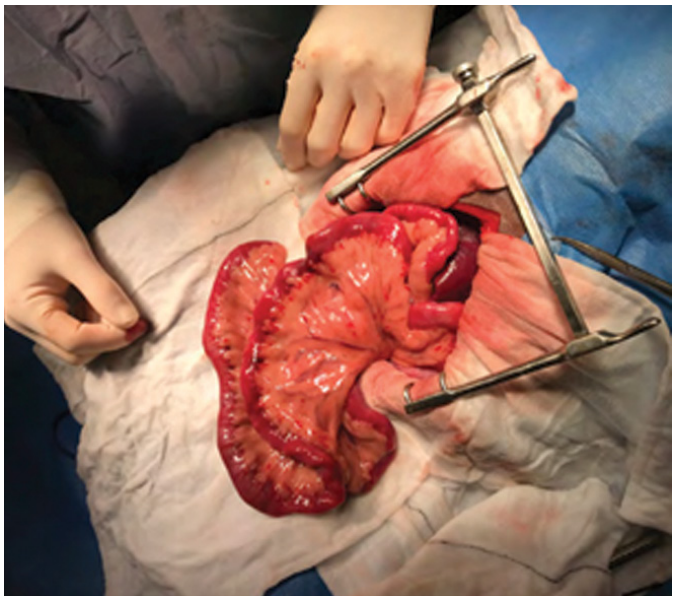

Figure 6. Following gastrotomy to relieve the anchor point of the foreign material, the material has been gently milked into the mid-jejunum to relieve the plication. Note that the small intestine is erythematous and oedematous, and there is bruising along the mesenteric border.

Instead, Fullagar gently protects the plicated portion with moistened abdominal swabs and follows the foreign body to its proximal anchor point, which in dogs is usually the pylorus. "Usually you'll palpate something in a dog's stomach," she continued. That's because, according to Fullagar, dogs tend to eat more "robust" items like toys, pieces of carpet, socks, and dish- rags. "Cats are a little different, as they are more prone to eating tiny piece of thread, and the surgical technique is slightly different," she added.

After confirming the material is anchored in the pylorus, Fullagar performs a gastrotomy. "Don't forget to use stay sutures to exteriorise the stomach, and isolate the stomach with moistened abdominal swabs to prevent spillage of stomach contents," she added. "The stomach of a patient with a linear foreign body is often full of fluid, so if this is the case, an orogastric tube can passed intraoperatively to drain some of this fluid prior to gastrotomy."

The gastrotomy should be performed close to the pylorus, over the region where the foreign material is palpable (Fig 5). "Then what I'll do at this stage is exteriorise the foreign body just far enough from the pylorus that I can get underneath the bulk of it and cut it with scissors," she explained. Referring to a 10-year-old dachshund patient named Ollie who'd eaten a handkerchief, Fullagar noted, "It's important not to pull too hard on the foreign body, as this can cause further damage to the small intestine. It's very rare that a linear foreign body can be completely removed safely via a gastrotomy incision alone. You're just going to cut underneath it and then release it," she continued. "You should feel the remainder of the material move through the pylorus and into the duodenum."

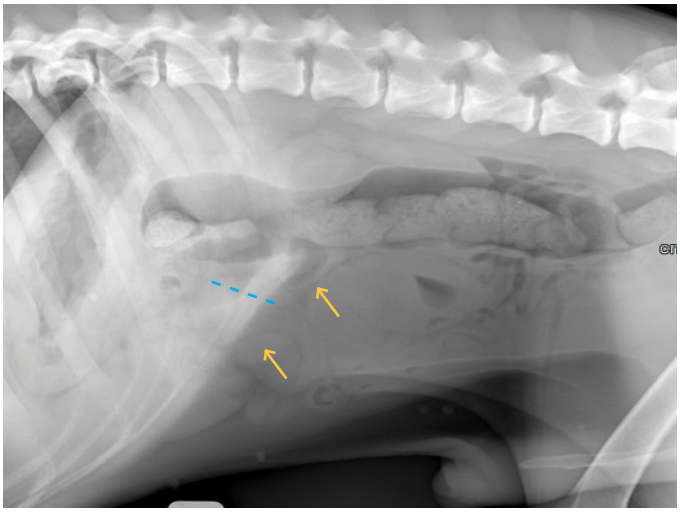

Figure 1: Right lateral recumbent view: The arrows indicate some of the abnormally shaped or segmental gas opacities associated with the small intestine. In this case a portion small intestine is distended ***(dashed line).*** *In this region (mid to caudoventral abdomen) there is loss of serosal detail.*

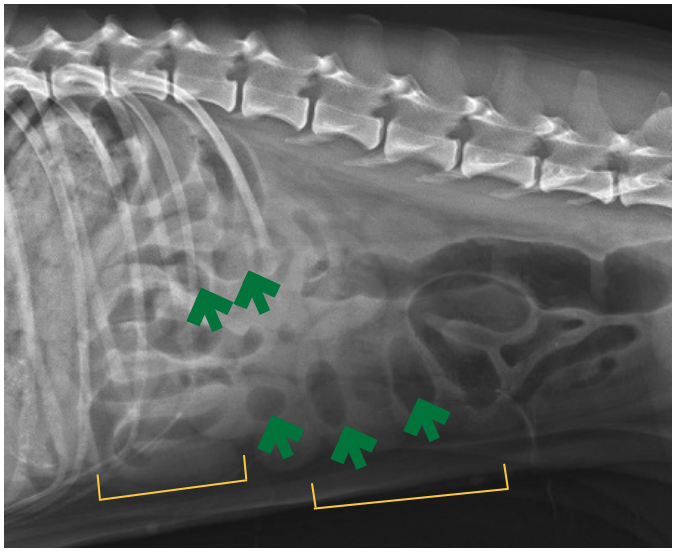

Figure 2: Right lateral recumbent view: In the cranial abdomen indicated by the yellow bracket there is dorsoventral stacking of small intestinal loops. The blue bracket indicates small intestinal stacking in a craniocaudal direction in the ventral abdomen. The green arrows indicate abnormally shaped/segmental gas opacities associated with both.

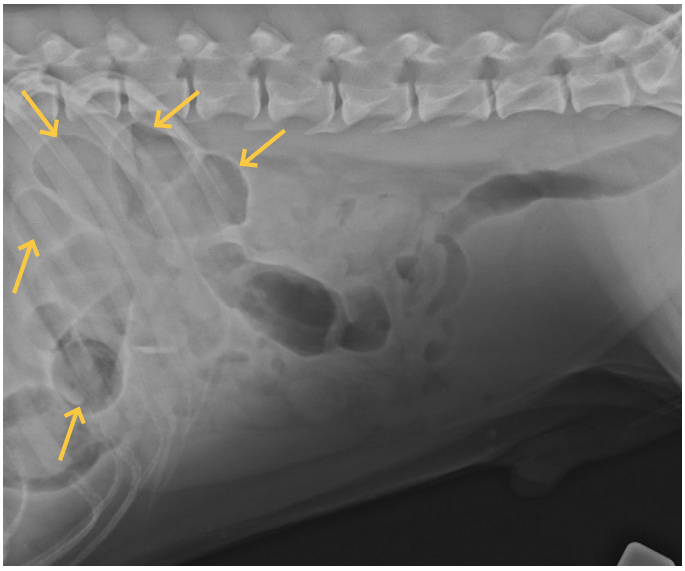

Figure 3: Left lateral recumbent view: arrows indicated bunched up distended small intestinal loops with an almost circular arrangement of abnormally shaped or segmental gas opacities resulting in focal loss of serosal detail. The small intestinal loops in the mid-abdomen is located in close proximity to each other with smaller segmental gas opacities.

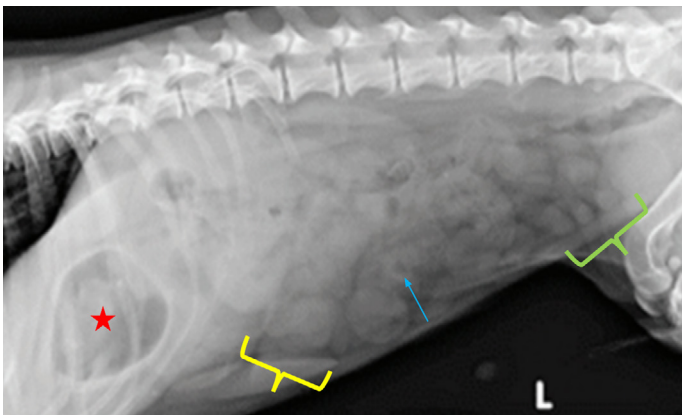

Figure 4: Characteristic left lateral abdominal radiograph of a dog with a linear gastrointestinal foreign body.

Note the foreign material present in the pylorus (indicated by star), plicated small intestine (indicated by yellow bracket), and comma-shaped gas patterns (indicated by blue arrow). The small intestine also shows stacking(indicated by the green bracket).

Images: Figure 1 - Figure 3. Courtesy: Drs Hoepner and Sandra de Sousa Jesus, Section DIM, OVAH, Fac VetSc, UP

6. "Milk" the foreign body to relieve intestinal plication before enterotomy

Once the gastrotomy is complete, Fullagar gently "milks" the rest of the foreign body aborally (Fig 6). "You're getting the part of the foreign body in the stomach to catch up with the part that's farther down," she said. "You're also moving it into a healthier region of the intestine that's safer for an enterotomy, because now it's not attached anywhere. Usually the material ends up in the jejunum, which is the easiest part of the intestine to exteriorise for surgery. It's more difficult to do an enterotomy in the duodenum, because this section is harder to exteriorise and closer to the pancreas."

And if you've started with the gastrotomy, Fullagar explained that in the vast majority of cases, you should be able to remove the entire foreign body through a single enterotomy site. Minimising the number of GI incisions will reduce the risk for post-operative dehiscence.

Evaluate the entire mesenteric border of the small intestine According to Fullagar, even if you know your patient is going to need resection and anastomosis because you've already identified a small intestinal perforation, in most cases it is still helpful to remove the foreign body so you're able to relieve the intestinal plication and fully assess the mesenteric border.

"In linear foreign body cases, the mesenteric border is the place to really check very, very closely along the entire length for perforation—even if the overall colour of the intestine is healthy," she explained. "The mesenteric fat is often oedematous, and this can make small perforations quite difficult to visualize. Surgical experience is really important when it comes to assessing intestinal viability."

7. Don't forget post-operative care

Fullagar ended by reminding the audience of the importance of post-operative care for patients who have undergone GI surgery for linear foreign bodies. "These patients may not have eaten for several days prior to surgery, and their intestinal tract is usually significantly more inflamed and oedematous than that in patients that have had a solitary intestinal foreign body," she explained.

"The trauma and inflammation caused by the foreign body and plication, combined with general anaesthesia and surgical manipulation, can lead to quite significant ileus, and it's important to pre-emptively treat this to give our patients the best chances of successful intestinal healing." In addition to rehydration with intravenous fluids and an opioid for pain relief, Fullagar recommends post-operative treatment with prokinetics and anti-emetics.

Enteral feeding, starting within 12 hours of surgery, is also key to intestinal motility and healing, so a nasogastric feeding tube should be considered at the time of surgery for patients who have been anorexic. Referral to a facility with 24-hour care and monitoring can also be beneficial to maximize the chances of a successful outcome.

Take home points

- Many linear FB don't cause any small intestinal dilation visible on radiographs. Look for characteristic comma- or paisley-shaped gas patterns, often combined with evidence of small intestinal plication and/or loss of serosal detail. If you're still unsure - perform abdominal ultrasound.
- Visualisation is key - make a large incision
- Keep abdominal tissues moist with soaked abdominal swabs
- Avoid excessive intestinal manipulation and do not pull too hard on the FB - this can cause laceration to intestine wall
- Release the anchored material in the pylorus first via gastrotomy
- Meticulously evaluate the mesenteric border of the intestin

Sarah Mouton Dowdy, a former associate content specialist for dvm360.com, is a freelance writer and editor in Kansas City, Missouri.

Journal Scan

Summarised by: Dr L.L. van der Merwe BVSc MMedVet(Med)

Evaluation of a urine dipstick protein to urine specific gravity ratio for the detection of proteinuria in dogs and cats.

Barchilon M, Perez-Nieves N and Palerme JS. Journal of Veterinary Internal Medicine (2024) 38: 1060 – 1067.

Why they did it: Urine specific gravity influences the quantification of proteinuria from urinary dipsticks.

A calculated dipstick urine protein to urine SG ratio has been suggested to circumvent the need for performing a UPC ratio. the objective was to evaluate the correlation between the dipstick protein to USG ratio (DUR) and UPC ratio and the performance of the DUR, as well as evaluating

the effect of urine characteristics on the DUR.

How they did it: Urine samples from 308 dogs and 70 cats were tested. Physical and chemical analysis was performed. Dipstick analyses was performed as per manufacturer guidelines and protein read as + = 30mg/dL; ++ = 100mg/dL, +++ = 300mg/dL and ++++ = 2000mg/dL. Samples with trace protein were assigned a value of 15mg/dL. Urine was then centrifuged and a sediment examination performed. A sample was reserved and stored for UPC evaluation.
DUR ratios were calculated using the formula:

Dipstick protein result in mg/dL divided by ([sample USG – 1] x 1000).
Example: patient with 1+ protein with SG 1.015 calculates as 30mg/dL / (1.015 – 1)x1000 = 30 / 0.015 x1000 = 30 / 15 = 2.

What they found:
For canine urine samples a significant moderate positive correlation existed between UPC and DUR, irrespective of the presence of sediment or range of USG(p=<0.01). the same applied to cat an was also not affected by the presence or absence of glucosuria.

The ROC analysis was used to assess performance of DUR.

Cutoff levels will affect sensitivity and specificity of the result.

Take home message: The DUR cannot replace the UPCR for quantification of proteinuria. Furthermore the high PPV of the DUR for the lower 0.2, 0.4 and 0.5 UPC rations in dogs and cats makes a positive result highly suggestive of proteinuria and supports further testing by UPC ratio

However, the NPV in these cases was low and thus the test cannot be used to exclude non-proteinuric patients, based on the DUR, from having clinical disease i.e it will give false negative results. This makes it a poor screening test.

In dogs the optimal cutoff DUR was determines was:

UPCR	Optimal DUR	Sensitivity	Specificity	PPV	NPV
>0.2	1.4	83%	91%	99%	29%
>	1.4	89%	83%	96%	63%
>2.0	5.7	83%	83%	84%	83%

In cats the optimal DUR to obtain the best results was:

UPCR	Optimal DUR	Sensitivity	Specificity	PPV	NPV
>0.2	1.2	73%	90%	95%	59%
>0.4	2.8	70%	100%	100%	75%
>2.0	2.8	91%	78%	43%	43%

Journal Scan

Summarised by: Dr L.L van der Merwe BVSC MMedVet(Med) Small Animals

Association between cigarette smoke exposure and urinary bladder cancer in Scottish terriers in a cohort study.

Knapp DW, Dhawan D, Ruple A, et al. The Veterinary Journal. 2024;303

Why they did it:
There is a 20x higher risk for canine urothelial carcinoma (transitional cell carcinoma) in Scottish terriers than other dog breeds.

The objective of the study was to identify environmental and host risk factors in a cohort of 120 Scottish terriers participating in a cancer screening study using owner questionnaires. Cigarette smoking is the most consistent risk factor for human UC.

How they did it:
A screening study for 120 Scottish terriers was run over a 3 year period where 32 of 120 dogs developed UC. a questionnaire was filled in by these owners. The owners were asked to give information related to the dogs home and other environments, health, urination habits, types of food and water, supplements and chemical exposures during their lifetime. The information and patient bodyweight and condition score was updated every 6 months.

TO compare the level of cigarette exposure between dogs

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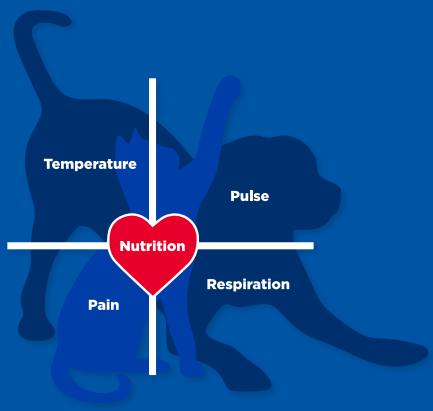

SCIENCE DID THAT.

What is the BRAF mutation?

It is a somatic mutation in the BRAF gene which only occurs in the tumour cells of urothelial or prostatic carcinoma. It is assumed that this mutation leads to tumour development through permanent activation of the MAP kinase pathway.

In 2018, a method for detecting BRAF mutation in TCC was established. Specificity was 100% as the BRAF mutation could not be detected in any dogs with cystitis, urinary bladder polyp or in those without any clinical and pathological signs. Sensitivity of BRAF mutation detection in TCC was 71%.

Possible sample materials include tissue (e.g. biopsies), cytological smears (e.g. FNA) and urine rich in cells (early morning urine). Invasive sampling can thus be avoided by testing spontaneous urine, or there is no repeated invasive sampling necessary in cases of questionable histopathological and cytological diagnoses (poor sample quality, overlapping images of inflammation and neoplasia).

the pack-years smoked by the owner in the house was calculated by multiplying the number of packs smoked per day times the number of years the dog was exposed to that level of smoke. The pack years of each smoker in the house was combined for each year of the dogs life.

NO owners reported long term use of COS inhibitors for any reason. Cotinine, a nicotine metabolite, was measures in the urine and normalised to creatinine concentrations. the highest level was used for statistical analyses.

What they found:

Risk factors which were statistically significantly associated with the develeopment of UC in these dogs were living in households with cigarette smokers (OR, 6.34, p=0.033) living within a mile of a marsh or wetland (OR, 21.23, p = 0.001), a history of prior urinary tract infection (OR, 3.87, p = 0.050), and older age at enrollment (OR, 1.38;p=0.024). Among dogs living with smokers, the dogs with UC were exposed to a median of 10 pack-years (range, 0.75–36.0 pack-years) compared to a median of 1.5 pack-years (range, 0.25–7.0 pack-years) in dogs without UC which was significant (p= 0.052).

(OR = odds ratio = An OR of 1.2 means there is a 20% increase in the odds of an outcome with a given exposure. An OR of 2 means *there is a 100% increase in the odds of an outcome with a given exposure*. Or this could be stated that there is a doubling of the odds of the outcome.) UC developed in 35% of dogs with quantifiable cotinine levels in the urine compared to 15% of dogs without. The concentration of BRAF mutation was not correleated to detectable urine cotinine or any other environmental variable.

Take home points:

Scottish terriers living in households with smokers were 6x more likely to develop UC and the amount of exposre increased the risk. The increased risk in marshy wetlands is proposed to be water pollutants.

Increased risk of UC in Scottish Terriers Exposed to Herbicides

A previous study published in 2004 In JAVMA demonstrated, using a questionnaire-based study, on 83 Scottish Terriers diagnosed with UC, compared to a control group of 83 Scottish Terriers with health conditions excluding urothelial carcinoma (UC) - The new terminology for transitional cell carcinoma.

The results showed that the risk of UC was significantly increased among dogs exposed to lawns or gardens treated with both herbicides and insecticides (odds ratio [OR], 7.19) or with herbicides alone (OR, 3.62), but only very mildly with dogs exposed to lawns or gardens treated with insecticides alone (OR, 1.62), compared with dogs exposeda to untreated lawns. Exposure to lawns or gardens treated with phenoxy based herbicides (OR, 4.42) was associated with an increased risk of UC, compared with exposure to untreated lawns or gardens, but exposure to lawns or gardens treated with non-phenoxy based herbicides (OR, 3.49) was not significantly associated with risk of UC.

Source : J Am Vet Med Assoc, . 2004 Apr 15;224(8):1290-7. Herbicide exposure and the risk of transitional cell carcinoma of the urinary bladder in Scottish Terriers. Lawrence T Glickman 1, Malathi Raghavan, Deborah W Knapp, Patty L Bonney, Marcia H Dawson

#PRACTICE TIPS

Preventing Phlebitis in Puppies With Parvoviral Enteritis

Contributed by: Dr Liesel L van der Merwe, OVAH, Outpatient

One of the complications which occurs reasonably frequently in puppies affected with canine parvoviral enteritis is the development and phlebitis and infection at the site of catheter insertion. This is often regardless of excellent sterile placement technique.

Additional delayed complications as a result of bacterial translocation from the GIT the translocation of bacteria from the GIT and bacteraemia is bacterial arthritis and discospondylitis, both thankfully much rarer than IV catheter infection. We cannot do much to limit these systemic complications be we can adapt our IV catheter placement technique to reduce phlebitis and infection. Although specifically used in the isolation ward at the OVAH for puppies with CPV enteritis, this method is also applicable to any immunocompromised patient requiring long term (days) IV catheter placement.

Figure 1: IV catheter placed in a puppy with parvoviral enteritis. Note the Ioban® extending right over the Elastoplast and also not that the dripline is not doubled back - as the dripline itself is also secured to the patient with a butterfly.

What we need to consider in puppies with CPV enteritis is that no matter how aseptically we place these catheters as soon as saliva, vomitus or diarrhoea gets onto the elasoplast, it will soak down to the skin and can then easily infect at the point of intravenous insertion. Our solution at the Isolation ward is to place an impermeable dressing over the Elastoplast, in our situation we use Ioban®, to stop any fluid from wicking.

Additionally, after clipping, we will wash the skin with a wet gauze swab with hibitane soap. This cleans off oil and dirt from the skin. We dry the area and only then swab with alcohol. D-germ is also a massive help as it is simple to quickly disinfect your hands after prepping the patient.

Since implementing the above process and adding the Ioban® over the top, we have reduced our catheter-site infections to almost none, and this regardless of inexperienced students placing the catheters in most cases.

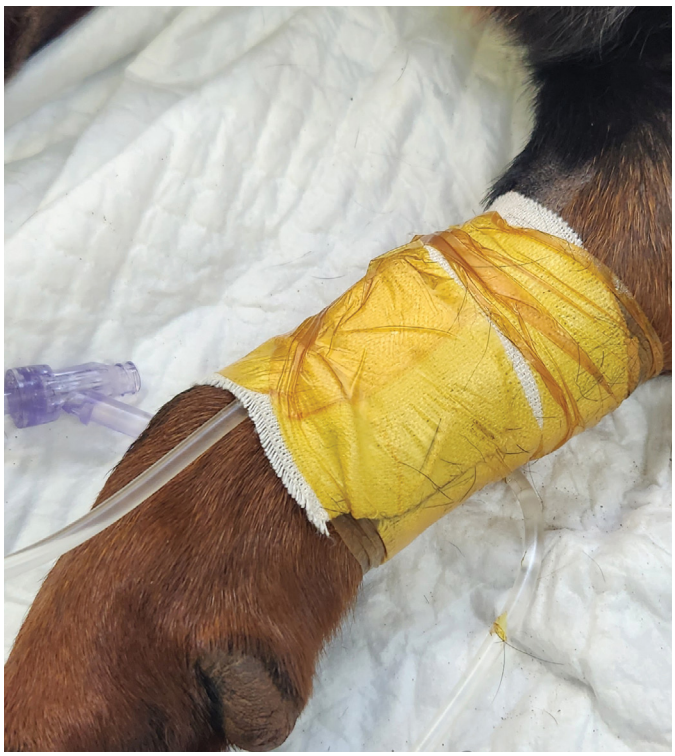

Figure 2: Improper placement of the Ioban® layer as fluid will still wick into the elastoplast.

Procedure:

- Clip the skin
- Quick wash with wet hibiscub soaked gauze pad, is not a surgical prep.
- Dry the area with a dry gauze / swab.
- Swab with alcohol
- Disinfect hands - D-Germ
- Place and secure IV catheter
- Wrap all exposed Elastoplast with Ioban®. Any cloth sticking out from edges will wick fluid.
  o If you are going to use the Ioban® sheet - we cut then to size - slowly and deliberately - using a guillotine. Cutting with a pair of scissors doesn't work.

Please submit any good ideas or tips you have to publications@vetlink.co.za
Please note I will not publish controversial procedures - I am looking for the quick helpful practical tip or method to improve an existing method.

Another tip is to place an Elastoplast butterfly around the actual drip line just before it connects to the IV catheter - this means you do not need to bend the dripline, and this reduces the development of kinking and blockage, especially when not using a fluid pump. This method of securing the drip is very robust. (Fig 3)

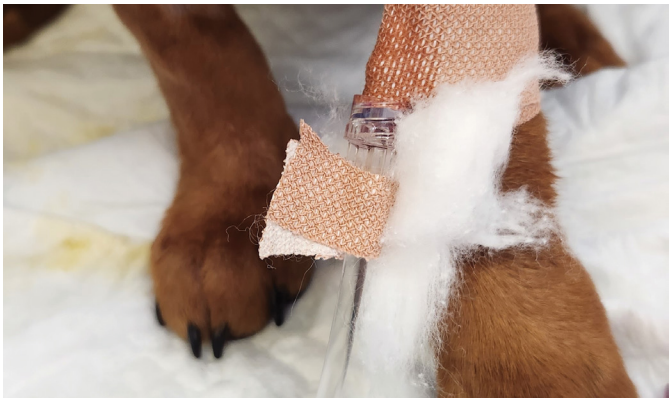

Figure 3. A simple butterfly on the dripline really secures the line and helps the iv catheter stay in place.

Journal Scan

Summarised by: Dr L.L van der Merwe BVSC MMedVet(Med) Small Animals

Use of molidustat, a hypoxia-indicible factor prolyl hydrolylase inhibitor, in CKD associated anaemia in cats.

Charles S, Sussenberger R et al, Journal of Veterinary Internal Medicine 2024:38 197 -204

Why they did it: Investigating if molidustat will safely stimulate erythropoiesis in the failing kidneys of cats with chronic kidney disease (CKD).

The production of erythropoietin (EPO) is controlled by hypoxia-inducible factor (HIF) found in specialised renal EPO producing cells. In normal oxygen conditions EPO is degraded by HIF prolyl-hydroxylase (HIF-PH) enzymes.

Mammals thus have the ability to adjust to environmental changes in oxygen levels. In CKD in cats, there is decreasing metabolic activity, which leads to a relative renal hyerpoxia, leading to decreased EPO production due to increased degradation of HIF by HIF-PH. Molidustat is a HIF-PH inhibitor.

What they did: A multicentre, blinded, placebo controlled study was performed. Cats diagnosed with anaemia (PCV <27%) and a creatinine ≥ 160ug/mmol (unfortunately IRIS staging was not performed) with a systemic blood pressure < 165 mmHg were included. Molidustat was administered per os once daily at 5mg/kg. Control patients received the carrier only.

At the end of the trial patient numbers were n=14 for the treatment group and n =5 for the control group. Blood was collected D0, D7, D14, D21 and D28. A clinically relevant increase was defined as a 25% increase in PVC or an absolute 4% increase from baseline.

What they found: The baseline HCT was 23.2% in the control group and weekly responses remained low (20 .1 – 24.3%) at all assessed points. The baseline PCV in the treated group was 23.6% and on D21 was 27.3% (versus 20.1% in the control group) and on D28 was 27.8% (versus 23.4% in the control group). the difference was significant on D21 (p=<0.001). vomiting was the most frequently reported side effect occurring in 40% of cats.

Take home points: Despite the limitations of the study (low power due to low case numbers, no IRIS staging, unknown impact of CKD stage on results, short duration of study) there was sufficient evidence to show a positive effect of molidustat in stimulating eythropoeisis.

An additional finding in those patients which elected to continue treatment for an additions 60d was an ongoing increase in PCV (28%) and an increase in body weight.

(Launched & September 2023, by Elanco, Varenzin™-CA1,(molidustat oral suspension, is the FIRST and ONLY FDA conditionally approved option for the treatment of non-regenerative anemia in cats with chronic kidney disease (CKD).) Not available in South Africa as yet.

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References: 1. Miarikova, M., 2017. The capacity and effectiveness of diosmectite and charcoal in trapping the compounds causing the most frequent intoxications in acute medicine: A comparative study. *Environmental Toxicology and Pharmacology*, 52:214-220. 2. 2009. As many as 1 000 dogs poisoned per week in SA. *Infurmation*. Available at: https://www.infurmation.co.za/home-page/1000-dogs-poisoned-per-week/.

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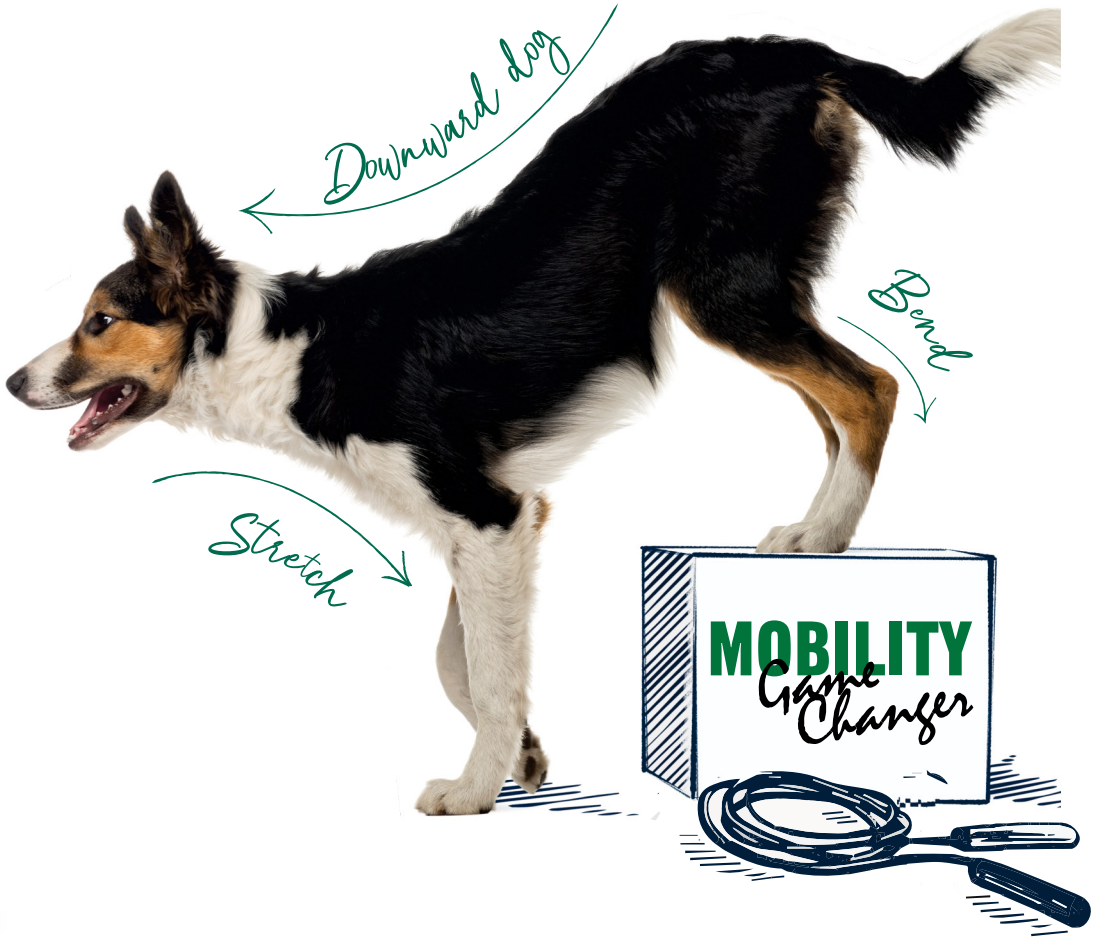

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Reference 1 GlycOmega-PLUS® Greenshell Mussel COA - Available from Kyron Laboratories office, on request.
Reference 2 A pilot Trial Assessing the Efficacy of GlycOmega-PLUS® (Green-Lipped Mussel Powder) for Osteoarthritis of the Knee, S. Coulson et al, University of Queensland, Brisbane, June 2011.
Reference 3 Collagen Supplementation for Joint Health. Martinez-Puig et al. Nutrients 2023,15,1332: https://doi.org/10.3390/nu15061332
Reference 4 RevitaCOL® Collagen COA - Available from Kyron Laboratories office, on request.
Reference 7 The Story of New Zealand's Green Lipped Mussels - An Excellent Superfood. https://www.einnews.com/pr_news/572137849/the-story-of-new-zealand-s-green-lipped-mussels-an-excellent-superfood
Reference 8 Data on file - available from Kyron Laboratories office, on request.

KL-M-MBCVET360-01