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vet 360

Vol 10 | Issue 02 | May 2023

Medicine
Sialadenosis

CPD Article
Feline Hyperaldosteronism: A Mediator of Renal Disease?

Business
Succession Planning in a Veterinary Practice

Spinal Injury
Stabilization by SOP Plates

Also in this issue

Feline Hypertensive Retinopathy

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1. The BENCH Study Group. J Vet Cardiol 1999; 1: 7–18.
2. King JN et al. J Vet Intern Med 2006; 20: 1054–1064.
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Editor's Note

As winter draws near and the evenings get colder, I find myself looking at my senior cat carefully for signs of age-related discomfort.

Of course, ageing, particularly in cats, also carries an increased risk of kidney trouble, and that is what this edition of Vet360 focusses on.

This month we look at feline hypertensive retinopathy, often associated with chronic kidney disease, and at anaesthetic considerations for patients with impaired kidney function.

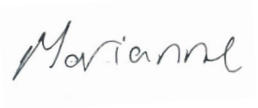
Our CPD article investigates a fascinating but hitherto under-diagnosed mediator of renal disease: feline hyperaldosteronism. And for a break from kidneys, an overview of Sialadenosis.

So another jam-packed issue to keep you company on Winter's nights, even if load-shedding has you reading by candlelight.

My sincere thanks to our world-class local contributors for this edition: Izak Venter, Dave Miller and Johan Schoeman. If you have a topic you'd like to read about in a future issue of Vet360, please let us know!

Stay warm and informed,

Marianne



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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Succession Planning in a Veterinary Practice

Andrew Christie
BComm (Business Management)

The veterinary profession has largely worked on a seller-financed model by which a younger vet works at the practice and gradually acquires a small amount of equity through a profit share. When the practice owner reaches a point where they want to retire, the younger vet takes over the practice and pays the practice owner out of future profits.

However, it has never been easy to find the right vet to buy a practice. And perhaps this is not surprising since the 'right vet' means someone you have worked with and found suitable, as well as someone who is neither too young nor too old.

Combined with the socio-economic conditions of South Africa (Eskom, inflation, rising CPI etc etc) and a lack of veterinarians, the traditional approach to succession planning has failed and a number of vets are confronted with practices that cannot be sold.

Understanding the roots of this problem (no, it's not only vets leaving the country) provides an opportunity to create a more meaningful succession strategy than the reactive approach that has befallen many practices.

The Basics of Succession Planning

Start ASAP

You should start planning your exit strategy as soon as the possibility of acquiring a practice first occurs – in fact, it should be part of your considerations when buying a practice.

Create the structure you want

Consider issues such as selling equity in parts over a period of time (eg 10% per annum over 10 years), or selling your practice only when you are ready to retire (100% at retirement) or a mixed approach (eg10% for 5 years, then 50% at retirement).

Integrate with your business strategy

Your succession plan must be part of your business strategy – no point in investing in a practice if you will get more money for selling the property for more than you could realise for the business.

Plan for your sudden death

At this point your family members cannot own your practice (unless, of course, they are also vets) - even if they could, it is unlikely that they will want to step into the chaos of a veterinary practice.

Update regularly

A succession plan should not be seen as a static document, stored on a piece of paper in your safe or, worse, in your head. It is a fluid strategy that should change in response to the economy, the profession, your needs and wants etc.

Generation Z vs the Millennials

We've spent so much time trying to figure out the millennials that we've forgotten that the newest graduates are actually 'Generation Z'. Why is this important? Millennials have certain traits that have changed the profession both locally and internationally.

For example, Millennials are 'global citizens' and like more freedom to move around than previous generations. The effect? An increasing number of locums and an increasing number of younger vets emigrating.

Broadly speaking, Generation Z are those people born after 1996. This means that they have grown up with 9/11, the 2008 economic collapse, not to mention massive unemployment and the deteriorating South African economy.

According to the US Census Bureau[1]:

- 77% of Gen Z expect to work harder than previous generations.
- 74% of GenZ prefer face-to-face interaction with colleagues.
- 69% of Gen Z would prefer their own workspace.
- 71% of Gen Z subscribe to "if you want it done right, then do it yourself".

These four areas where Generation Z widely differ to Millennials indicate that there is some movement back to the characteristics of earlier generations. While it is way to soon to be able to convert this information to meaningful succession planning, I do think that it could be an indication of more vets wanting to buy in to the practices they work at.

Changes to the profession

Changes to the veterinary and para-veterinary legislation has caused some unhappiness amongst veterinarians due to the perceived competition that could be created. However, I see the changes as a big opportunity for succession planning as it means for the first time some para-veterinary professionals can invest in vet practices – in short, your vet nurse is allowed to purchase equity in your practice.

This means that there is an increased pool from which buyers can emerge. However, it is a new road that has to be travelled, with the SAVC themselves acknowledging that the impact of the changes needs to be analysed.

Conclusion: Things to bear in mind

Succession planning does not end with the agreement to sell your practice to an interested party. The tax and legal implications form a maze of complications that you will need your accountant and lawyer to navigate.

Scenario 1:
You sell your practice for cash upon retirement.

You will have to pay Capital Gains Tax on the capital gain – very broadly, the capital gain is the difference between what you are selling the practice for less what you paid for the practice, less any costs for improvements / physical changes you made. This is particularly relevant to older practices where the practice was not purchased but started by the entrepreneurial veterinarian.

Scenario 2:
The buyer repays you the selling price over a period of time.

You will still have to pay CGT, and this could be difficult since you will have to pay it in the year of sale but will only be receiving funds monthly over a period of time.

The other tricky part here is that legally a company cannot pay for its own equity. So if your practice is a Pty, ownership will have to be transferred to the buyer, who can only pay in their personal capacity. This means that Operating Profit will have to have company tax deducted, then Dividends Tax, before you receive your repayment.

Once this happens, then you will need to pay tax on the interest component of the payment. For example, the buyer may pay you R100, R30 of which is interest. The R70 isn't taxable because technically it is a loan repayment. However, the R30 is because it is additional income which you earned from the loan.

By way of conclusion, I find vet practices to be significantly undervalued at the moment, some yielding up to a 45% return. I strongly encourage you to consider investing in a practice. For vets wanting to sell, it may be worth holding on another year or two until prices pick up again. For both, I have found that there seems to be a misconception amongst buyers that a practice is 'worth R3 million' and 'can be repaid in 5 years'.

Nether of these are true – the value of a practice depends on its profitability and net asset value, and the repayment period needs to be negotiated to suit both buyer and seller. After all, we buy houses over a 20-year period and make the financial sacrifice for this.

[1] As cited in "Gen Z @ Work: How the Next Generation Is Transforming the Workplace", 2017, Stillman D & Stillman, J.

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Introduction

The retina is an extremely complex structure, and the function and maintenance of the photo receptors within it requires huge amounts of energy. The photo receptors of the cat retina use three to four times more oxygen than other retinal and CNS neurons for the oxidative metabolism of glucose in the light adapted state. Any disruption in the supply of nutrients to the retina may lead to irreversible damage to the photo receptor cells and consequent loss of vision.

The possible risk of lack of nutrients to the photo receptor cells is further complicated by the inverted retina. (Figure 1.) Histologically, the photo receptor cells are not in close proximity to the retinal blood vessels. (Figure 2.) The function of this anatomical arrangement to exclude vessels from the outer half of the retina is to not compromise vision. This leads to the paradox that the most energy-demanding part of the central nervous system is the only part of the CNS region to lack intrinsic vessels.

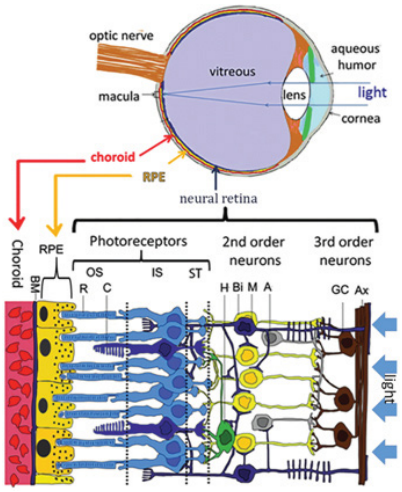

Figure 1: *Basic structure of retina's inverted architecture. Light travels through ganglion cells (GC) as well as second order neurons comprising bipolar (Bi), amacrine (A), horizontal (H), and Muller (M) cells to reach the photoreceptors. The retinal pigmentary epithelial cells [RPE] localizes between the choroid capillaries and the photoreceptors. Retinal blood vessels are mostly present in the nerve fibre layer. [Research gate]*

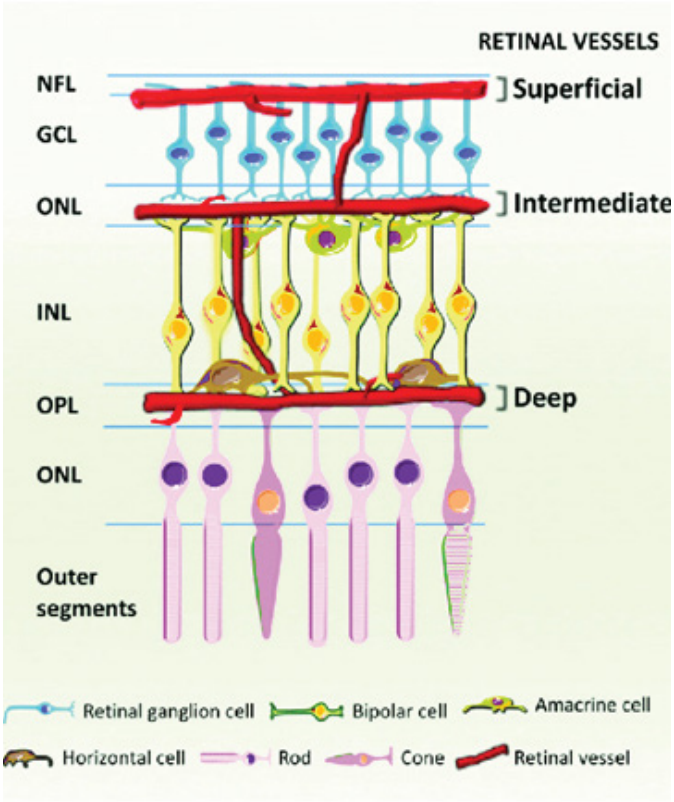

Figure 2: *Note the position of the retinal bloodvessels in comparison to the very metabolically active photoreceptor cells.*

The energy needs of the outer retina are met by diffusion of glucose and oxygen from the highly vascular choroid. The choroidal capillary vessels are highly fenestrated capillaries which are up to 30 times more permeable than those of skeletal muscle. This allows leakage of large amounts of plasma proteins and fluid into the choroidal interstitial space.

The choroidal throughput of blood is 40–50 times that of the retinal circulation. The end result of the highly fenestrated capillaries and high blood flow is the exposure of the photoreceptors to near-arterial levels of oxygen.

The choroidal vascular bed also differs from the retinal vascular bed by having an autonomic nerve supply and no blood–ocular barrier. Blood flow autoregulation offers protection against changes in perfusion pressure. This allows a tissue to maintain its blood flow during changes in perfusion pressure and it is a feature of the retinal, choroidal and optic nerve head vascular beds in a number of species, including the cat. Autoregulation probably operates by modifying blood flow through changes in the size of the lumen of the pre-capillary arterioles.

The blood–retinal barrier comprises of tight cell junctions between the endothelial cells of the retinal blood vessels and the tight cell junctions between the retinal pigment epithelial cells. The auto regulation mechanism may fail in cases with acute episodes of hypertension. With failure of auto regulation there is dilatation of the pre-capillary retinal arterioles followed by focal breakdown of the blood–retinal barrier. Leakage of plasma and red blood cells occurs when endothelial cells and vascular smooth muscle become damaged. Leakage of plasma within the retina leads to retinal oedema and foci of fluid accumulation within the neurosensory layer. Retinal detachment follows and is associated primarily with plasma effusion from diseased choroidal vasculature. The RPE undergoes ischaemic damage, contributing to retinal detachment.

Aetiology

Systemic hypertension is a relatively common disease of aged cats and is associated with chronic renal insufficiency and less frequently with hyperthyroidism. Primary hypertension in cats also exists.

Renal disease leads to increased sodium chloride retention, which leads to increased intracellular calcium. This in turn increases arteriolar tone and sensitivity to vasopressors such as angiotensin II. Renal disease also causes activation of the renin–angiotensin–aldosterone system, which raises blood pressure by increasing stroke volume and total peripheral resistance. Angiotensin II is a potent vasoconstrictor, while aldosterone causes renal sodium chloride retention.

Hyperthyroidism may contribute to systemic hypertension through its effects on the heart. It increases stroke volume and cardiac output, leading to systolic hypertension.

Clinical signs

Unfortunately,in most cases the disease is very advanced by the time the cats are presented. The reason is that the first indication of systemic hypertension is what appears to be an acutely blind animal. Even though cats may have an acute decompensation that leads to blindness, hypertensive retinopathy is usually one of gradual progression over several months and can be recognized before blindness occurs if fundic examination is performed.

Clinically, the ocular manifestations of systemic hypertension include:

- Retinal arterial tortuosity.
- Retinal haemorrhage [intra, pre-or sub retinal]. (Figure 3)
- Retinal oedema and focal bullae. (Figure 4)
- Retinal detachment and degeneration. (Figure 5)
- Hyphaema.
- Secondary glaucoma.

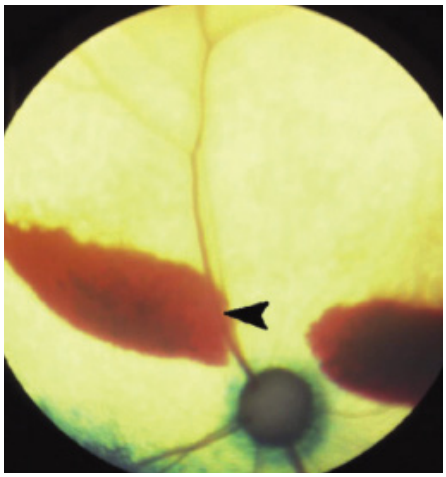

Figure 3: *Large areas of subretinal haemorrhage. The superior retinal vessel can be seen coursing over the area of haemorrhage (arrow). Atlas of Feline Ophthalmology Second Edition*

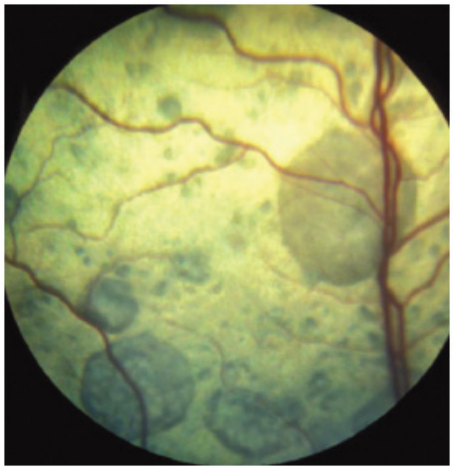

Figure 4: *Multiple rounded foci of oedema, seen as hypo-reflective areas in in the tapetum.*
Atlas of Feline Ophthalmology Second Edition

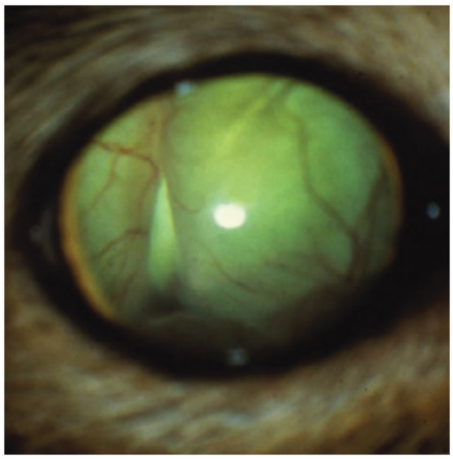

Figure 5: *Complete exudative retinal detachment.*
Atlas of Feline Ophthalmology Second Edition

The iris and ciliary body vessels may be compromised, leading to bleeding within the vitreous cavity and posterior or anterior chambers. A feature of hypertensive hyphaema is that unlike traumatic hyphaema it does not clear quickly and consequently may lead to synechiae and secondary glaucoma.

Diagnosis

Clinical signs and signalment are very suggestive of hypertensive retinopathy. Measurement of blood pressure confirms the diagnosis. Normal blood pressure for cats is usually defined as 120/80mmHg. The Doppler is unreliable in obtaining a diastolic value, so the systolic value is typically used to assess blood pressure. Typically, systolic values greater than 160mmHg are considered hypertensive.

Treatment

The treatment of hypertensive retinopathy includes controlling the underlying disease processes and treating the systemic hypertension. There is the potential in some animals for blindness to be reversible if systemic hypertension can be quickly controlled.

Bullous retinal detachments can resolve if the underlying effusion is controlled. Depending on the length of time the retina has been detached, vision may be regained. Retinal degeneration begins during the first week of detachment but becomes progressive the longer it remains detached.

There are numerous anti-hypertensive treatment options, but the calcium channel blocker amlodipine is considered the drug of choice. A dose of 0.625mg to 1.25mg orally once daily is recommended.

Conclusion

Early recognition of animals at risk for systemic hypertension is an important factor in preventing blindness. Any cat over 10 years of age should have a fundic examination performed as well as blood pressure measurement at least once per year. Ideally, hypertension should be recognized and treated before severe ocular disease occurs.

Even if vision is not restored, treating hypertension is important to prevent other serious effects such as neurological disease.

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Introduction

Sialoadenosis - A SYNDROME with many know triggers that causes painful enlargement of especially the mandibular salivary glands, causing pain on opening the mouth, excess saliva production, pain on salivary gland palpation or apparent cervical pain when turning the patients neck.

Often presented to the veterinarian with very non-specific signs but hyporexia and a "Huff" like cough are often part of the history. Clinical examination and the finding of mandibular salivary gland enlargement and pain is not pathognomonic but is very helpful in deciding if your patient falls into this syndrome.

Once this syndrome is identified, the attending veterinarian should then perform a "trigger search" to decide if the sialoadenosis is idiopathic/primary or secondary to a treatable trigger or a form of epilepsy [possibly limbic in origin?]. Terrier dogs may be predisposed. *Also known as – Hypersialorrhea, Phenobarbital-responsive sialadenosis.*

Clinical Signs

Clinical signs and examination abnormalities may include regurgitation of balls of white saliva that looks like whipped egg white, vomiting saliva balls, retching, gagging, choking, dysphagia (especially pain on swallowing) excessive head movement with telescoping of head into neck when swallowing (esp saliva), ptyalism, salivary gland enlargement, pain when opening the mouth, weight loss, apparent cervical pain, anorexia, hyporexia, inspiratory sounds as if the dog is breathing through water/liquid, ropey nasal discharge, exophthalmos, epiphora, strabismus, and chemosis.

Oedema of head or face or neck.

Known Triggers

A mass in the chest – lungs, mediastinal, oesophageal

- Megaoesophagus
- *Spirocerca lupi*
- Pulmonary cancer
- Pulmonary abscess

- Hiatal hernia
- Diaphragmatic hernia – congenital or traumatic

Epilepsy
Oesophagitis
Gastritis
Gastroesophageal motility disorders and megaoesophagus
Gastroesophageal reflux
Oesophageal strictures
Gastro-oesophageal hiatal hernias
Para-oesophageal hiatal hernias
Idiopathic

Immune mediated
- IMA
- MUO

Any mass in the abdomen
- Liver
- Adrenal
- Pancreatic
- Splenic

The author has seen cases where UTI's have been the trigger

Breed Predilection
Terriers

Diagnostic Procedures

Diagnostic Procedures:	Diagnostic Results:
Clinical exam	Enlarged, hard sore salivary glands. Mild pressure causes salivation, choking, pain and "fight and flight"
Ultrasonography of eye/ orbit	Orbital, retrobulbar mass
Ultrasonography of soft tissue involved	Soft tissue mass observed
Biopsy and histopathology of salivary gland	Salivary gland lobular necrosis Squamous metaplasia
Chest Radiography/CT	Mass in chest/ /mass in abdomen
Oesophageal Endoscopy	*S lupi*, Reflux, stricture, hernia
Abdominal U/S	Mass in Abdomen/thick bladder wall

THERAPY
Sialadenosis is usually very responsive to phenobarbital therapy. Reported effective dosage is 1 -4 mg/kg PO BD. Medication is started at higher doses at the same time as therapy for predisposing cause (if found) and then once control of the salivary gland pain is achieved meds are tapered and either stopped after 3-4-5-6 weeks or tapered to lowest effective dose and used life-long. Other anticonvulsants may also be effective, with the author having had success with KBr and Zonisamide and partial benefit with Keppra.

SUPPORTIVE THERAPY
Spirocerca lupi
Remove all faeces daily.

Dectomax has a tissue half-life of over 10 days and thus is the therapy of choice. In non MDR mutation breeds, many doses have been shown to be effective and the injectable product can be used orally.

The author has cured cases using doses ranging from 1ml/25 kg body weight to 1ml/600 kg body weight.
- The author mainly treats at 1 ml/20-25 kg body weight per dose
- Every 10-14 days S/C or per os.
- Used 1 to 6 to 10 times [mostly 4 to 6 times]
- Scope is advised to prove resolution but very few dogs are not cured with 4 to 6 doses

As the worm migrates from the gastro-intestinal tract to the oesophagus via the arteries, it can cause pain and aneurism. In endemic areas the author advises preventative dosing with Dectomax.
- Prevention – 1ml/50 to 1ml/200kg of body weight monthly Milbemycin = *Milpro/Milbemax etc* – at package insert dose
- Treat with - weekly for 4 to 6 times then
- Prevention - monthly for prevention

Advantix and other Milbemycin products can also be used.

Gastroesophageal diseases:
- PPI - omeprazole at 0.75-1 mg/kg 2X/DAY 1 h before food.
- If courses over 2 weeks, should taper medication
- Usually life-long in brachycephalic dogs and hiatal hernia cases

Antinausea
- Ondasetron 3x/day or Cerenia daily
- If needed use in combination

Pro-kinetics
- Metoclopramide (works best as a CRI) or Cisapride - often life long in Hiatal hernia cases

Tube-Feeding
- Many cases with chronic sialoadenosis need feeding for up to a week before the pain is well controlled by the phenobarbitone shrinking the glands for them to start eating.

MONITORING
Monitor patients on long-term phenobarbital therapy similarly to cases of epilepsy. Doses under 7 mg/kg 2x/day often induced Alkp levels but very, very, very rarely cause liver disease. Also monitor for recurrence of salivary gland swelling and/or signs of gastroesophageal disease (e.g. regurgitation, vomiting, excessive swallowing or pain on swallowing).

This talk *(www.savaevents.co.za - replay of webinar)* is illustrated with case studies to show the variability of presentation of this syndrome. The author strongly recommends that readers watch multiple videos, of dogs with sialoadenosis having a clinical exam to see the level of discomfort, even the slightest pressure on the glands causes and how the dogs react to this pressure.

Spinal Injury Stabilization by SOP Plates

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Iowa State University College of Veterinary Medicine

Reasons for performing the study/Background

Spinal fractures or luxations in small animals are most commonly caused by trauma and the thoracic and lumbar spinal segments are the most commonly affected regions. Multiple methods of fixation have been described.[1] The application of String of Pearls (SOP) plates in spinal trauma has gained attention due to its locking characteristics, while still allowing for flexibility in bending and twisting to assist with the safe positioning of screws in vertebral bones.[1,2]

Most reports focus on the use of SOP plates in stabilizing the cervical or lumbosacral regions of the spine, but no comprehensive review is available regarding the application of SOP plates for thoracic or lumbar vertebral fractures or luxations in dogs and cats.

Hypothesis/Objective(s)

The objective is to determine if using SOP plates for stabilization of traumatic thoracic or lumbar fractures or luxations would lead to an overall good outcome and prognosis.

Methods

Medical records for twenty-six canine and two feline cases diagnosed with traumatic thoracic or lumbar fractures or luxations and stabilized with SOP plates between 2010 and 2022 were reviewed at 4 surgical facilities. Data was collected regarding age, sex, weight, type of trauma, radiographic assessment, neurological assessment, and complications. The neurological status was graded using a 5-point Modified Matthiesen neurological assessment score (Table 1). Statistical analyses were performed by GraphPad Prism 9 Software. Kruskal-Wallis test was used for neurological status comparison. XY data correlation was to analyze between trauma causes (motor vehicle or non-motor vehicle accident) and injury types (fracture or luxation or both).

Complications were reported according to their time frame (perioperative at 0-3 months, short-term at 3 to 6 months, mid-term at 6-12 months, and long-term above 12 months) and severity: catastrophic complication (associated morbidity that causes permanent unacceptable function, related to death, or the cause for euthanasia), major complication (requires surgical or medical treatment), and minor complication (no additional surgical or medical intervention).[3]

Table 1: *Modified Matthiesen neurological assessment score*

Grade	Neurological status
0	No spinal hyperesthesia and no neurological deficits
1	Hyperesthesia, no neurological deficits
2	Proprioceptive deficits and/or ataxia
3	Non-ambulatory with purposeful movement
4	No purposeful movement with deep pain sensation
5	Loss of deep pain sensation

Results

Of all cases, 46.4% (13/28) were male and 53.6% (15/28) were female. Mean body weight of all patients was 14.4kg (median: 10.9kg,range: 2.7-37.2kg), and mean body weight of all dogs was 15.1kg (median: 12.1kg,range: 2.7-37.2kg). Trauma occurred mostly at younger ages (mean: 3.9 years, median: 2.3 years, range: 5 months-14 years). Motor vehicle accidents accounted for 67.9% (19/28) of injuries, while animal-related attacks were 21.4% (6/28), and falling from height was 10.7% (3/28). Motor vehicle or non-motor vehicle accidents were not significant predictors for causing fracture or luxation or a combination of both.

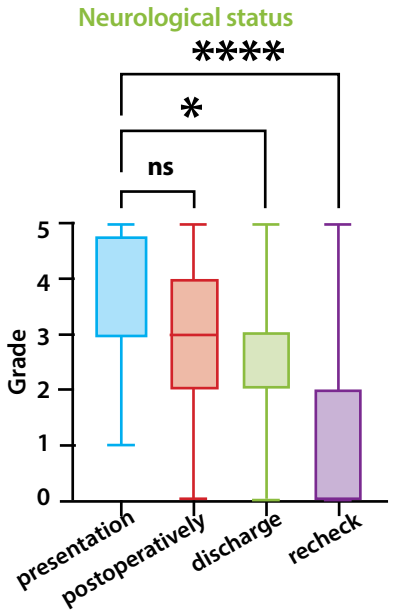
Of the patients, 82.1% (23/28) underwent bilateral plating while 17.9% (5/28) had unilateral plating. In cases with bilateral spinal plating, the median number of screws cranial to the lesion was 4 (range: 2-8), and caudal to the lesion was 4 (range: 2-7) (Table 2).

Table 2: *The SOP locking plates application detail in 26 dogs and 2 cats*

Size of cases	Plate numbers	Size of plates
Small dogs 10<kg or cats	Bilateral (10)	2.0 (6), 2.7 (4)
	Unilateral (4)	2.0 (4)
Medium dogs 11-26kg	Bilateral (11)	2.7 (7), 3.5 (4)
Large dogs 27-45kg	Bilateral (2)	3.5 (2)
	Unilateral (1)	3.5 (1)

At presentation, 25% (7/28) were deep pain negative. Deep pain perception was regained in 85.7% (6/7) of the deep pain negative cases at discharge. The neurological status at discharge ($p<0.05$) and at recheck ($P<0.0001$) showed significant improvement compared to the neurological status at presentation (Table 3).

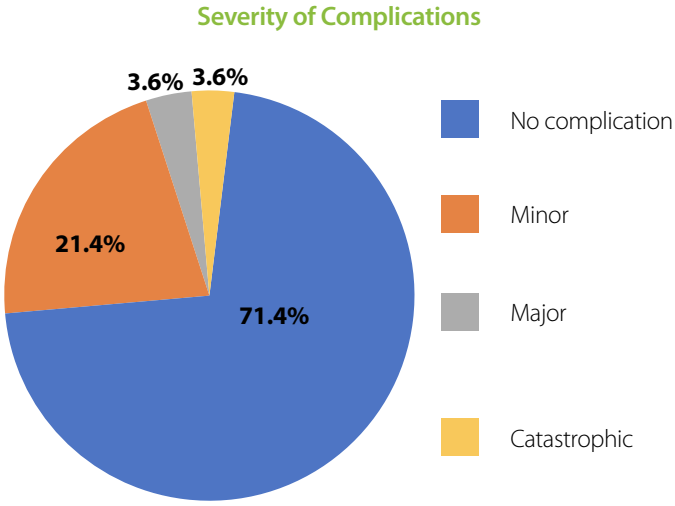
Table 3: *Neurological status assessment*



* significantly different at p value < 0.05
** significantly different at p value < 0.01
*** significantly different at p value < 0.001
**** significantly different at p value < 0.0001
ns no significant difference.

The mean duration of hospitalization was 5.9 days (range: 3-11 days). Complications were observed in 28.6% (8/28) of cases. According to the time frame for complications, 87.5% (7/8) were perioperative and 12.5% (1/8) were long-term complications. The severity of complications showed 3.6% (1/28) were catastrophic, 3.6% (1/28) were major and 21.4% (6/28) were minor complications (Table 4).

Table 4: *Severity of Complications*



Discussion

The unique feature of SOP plates is that they can be contoured in three planes with 6 degrees of freedom, allowing the natural kyphotic curvature to be mimicked. Bilateral placement of SOP plates on each dorsolateral aspect of the spine has been recommended with minimum of 3 screws cranial and caudal to the fracture based on the principle of bone plate fixation and clinical experience.[1,2]

Although no randomized prospective studies have compared clinical outcomes between different fixation techniques in spinal fracture animals, one biomechanical study compared pin and PMMA implants with bilateral SOP plates in lumbosacral fractures and showed that while both groups provided similar stability, the SOP group had better range of motion of L6-L7 in flexion.[2]

Surgical treatment, coupled with intact nociception, has resulted in good outcomes for 80-90% of dogs and cats with spinal fractures while the prognosis for animals that lack deep pain perception is less certain. The importance of nociception as a prognostic factor dictates its careful assessment in spinal fracture patients. Once it is present, the prognosis for recovery of ambulation is excellent.

Complications of spinal surgery include hematoma, surgical site infections, nerve root injury, implant failure, and progressive myelomalacia. Of our study, 8 animals had complications including myelomalacia(1), surgical site edema(1), surgical site seroma(1), surgical site infection(1), screw loosening(3), and surgical site tissue fibrosis causing spinal cord compression(1). One study reported the postoperative complication rate of overall spinal surgery was 13% of 220 dogs.

The overall complication rate for spinal surgery in dogs and cats may vary depending on the specific procedure performed, the individual patient's health status, and the neurological status at presentation. The rate of complications reported herein may not reflect the overall complication rate for all types of spinal surgeries.

Study limitations

The limitations of the study include its retrospective nature, small sample size, lack of direct objective outcome measures, and varying follow-up time frames for individual cases.

Scientific or Clinical Relevance

Our findings indicate that the use of SOP plates in dogs and cats with traumatic thoracic or lumbar fractures or luxations can lead to an overall good prognosis and outcome, characterized by an improvement in neurological status and an acceptable incidence of complications.

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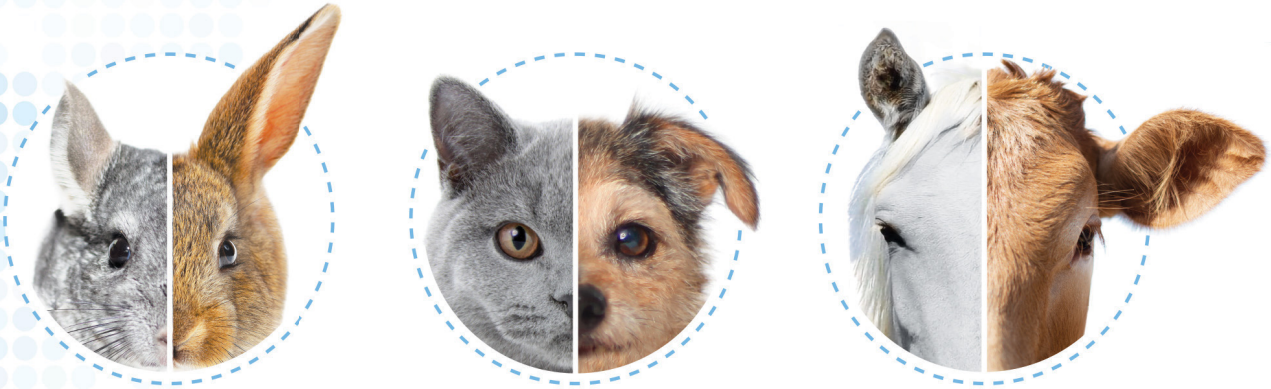

- Non-cytotoxic – shown to have beneficial effects on fibroblast and keratinocyte migration at low concentrations;[1,2]
- Kills bacteria, fungi, viruses, spores and amoeba – killed 99.99% of test pathogens within 2 minutes of exposure;[4]
- And improves wound healing, reduces pain, exudate, odour and scar formation.[3,5]

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Feline Hyperaldosteronism: A Mediator of Renal Disease?

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Background and epidemiology

Primary hyperaldosteronism, also known as Conn's syndrome, is an adrenocortical disorder characterized by excessive, autonomous secretion of mineralocorticoids, mainly aldosterone, leading to systemic arterial hypertension and/or hypokalaemia. The condition was first described in man in 1955 by Dr Jeremy Conn. Later he suggested that primary hyperaldosteronism is the underlying cause in up to 20% of people with arterial hypertension. With improved screening methods, the disorder is now diagnosed more often in humans and the prevalence is indeed much higher than previously thought.

Primary hyperaldosteronism is the most common adrenocortical disorder in cats and, like in humans, it is associated with arterial hypertension. The cat is the domestic animal in which the condition is most prevalent, yet under diagnosed, consequently excluding a potentially large number of cats from appropriate therapy and a possible cure. This unfortunate situation may be due to the frequent attribution of arterial hypertension and/or hypokalaemia to chronic kidney disease, thereby dissuading further diagnostic efforts.

Following on from the above, it is not commonly known that chronic kidney disease may, in and of itself, be a consequence of primary hyperaldosteronism. Therefore, arterial hypertension and hypokalaemia are often treated symptomatically, without a thorough search for the underlying cause. Moreover, arterial blood pressure is not measured routinely in many veterinary practices. This short communications seeks to highlight the link between hyperaldosteronism and chronic kidney disease and to sensitize practitioners as to the iceberg upon which they are likely finding themselves.

Synthesis, metabolism and physiology of aldosterone

The adrenal cortex consists of 3 layers: the outer zona glomerulosa, the middle zona fasciculata, and the inner zona reticularis (See Figure 1). The difference in hormone production between the 3 adrenocortical zones is primarily due to differences in the expression of cytochrome P-450 enzymes. These cytochrome P-450 enzymes are responsible for most of the sequential enzymatic conversions of cholesterol to steroid hormones.

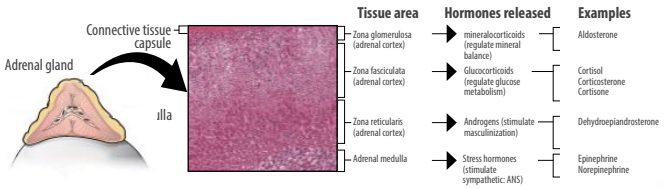

Figure 1. *Basic anatomy and secretory functions of the adrenal gland*

The pertinent enzyme in the zona fasciculata and the zona reticularis is 17a-hydroxylase (CYP 17), which catalyses the 17a-hydroxylation of pregnenolone and progesterone as well as the side-chain cleavage at C17 of 17-a-hydroxy C21 steroids, leading to the formation of cortisol and androgens in these zonal layers. Conversely, the low expression of 17a-hydroxylase (CYP 17) in the zona glomerulosa is the main reason why cholesterol is converted into aldosterone, instead of into cortisol or androgens, in this layer. The other steroidogenic enzymes areexpressed in all 3 zones.

Aldosterone was historically considered to be a hormone produced exclusively in the adrenal cortex. However, it has recently been revealed that aldosterone is also produced in tissues other than the adrenal cortex, including the heart, brain, and blood vessels. In these extra-adrenal tissues, aldosterone is thought to act in a paracrine or autocrine fashion. These new insights may contribute to the understanding of a number of long-term complications of primary hyperaldosteronism.

Unfortunately, little is known about the metabolism of aldosterone in cats. The liver is considered to be the most important site for inactivation and conjugation of steroid hormones. In cats, cortisol, oestradiol, and progesterone are excreted mainly or almost exclusively via the bile into the faeces, and, considering the structural similarities, this is likely also the main excretion route for aldosterone.

The primary function of aldosterone is to regulate plasma sodium and potassium concentrations as well as intravascular fluid volume. Notably, increases in potassium directly stimulates aldosterone secretion. However, Contrarily, a decreased blood pressure, which is sensed by the kidneys, upregulates plasma renin activity and consequent renin-angiotensin-aldosterone-system (RAAS) stimulation, which then results in aldosterone secretion. Subsequently, aldosterone will act on the nephrons and cause sodium reabsorption as well as potassium and hydrogen excretion. This all results in increased blood volume by conserving water and thereby increasing blood pressure.

Pathophysiology of hyperaldosteronism

Hyperaldosteronism can be primary or secondary. Primary hyperaldosteronism (PHA) is due to an autonomous aldosterone secretion by the adrenal cortical cells of the zona glomerulosa as mentioned previously. This is characterized by higher circulating aldosterone, as well as renin suppression due to negative feedback. Secondary hyperaldosteronism (SHA), on the other hand, is the result of a comorbidity such as heart failure or chronic kidney disease, in which renin secretion is increased, which then leads to hyperaldosteronism. Hence, in SHA renin is increased and not decreased, as is the case with PHA.

In other words, two pathophysiologic mechanisms may lead to hypersecretion of aldosterone. Firstly, a decrease in the effective arterial blood volume (e.g. owing to heart failure or oedema caused by hypoproteinaemia), which then activates the renin–angiotensin system, which in turn stimulates aldosterone synthesis. This pathophysiologic response to hypovolemia is called secondary hyperaldosteronism or high renin (hyperreninemic) hyperaldosteronism. In contrast, the autonomous and excessive aldosterone secretion in primary hyperaldosteronism is associated with suppressed plasma renin (activity) and is thus low renin (hyporeninemic) hyperaldosteronism.

Following on from the above, primary hyperaldosteronism can be due to autonomous hypersecretion of aldosterone (and/or deoxycorticosterone) by an adrenocortical tumour or by nontumorous, nodular hyperplastic zona glomerulosa tissue. In the majority of the reported cases, feline primary hyperaldosteronism is caused by a unilateral adrenocortical tumour of varying degrees of malignancy, ranging from well-capsulated adenomas to carcinomas with growth into the caudal vena cava and even distant metastasis.

These reported figures seem to differ markedly from those in humans, where bilateral hyperplasia of the zona glomerulosa accounts for 60% to 65% of cases and aldosterone-producing adenomas for 30% to 35%. In addition, cats with hyperplasia of the zona glomerulosa are often treated medically, meaning that adrenal tissue is not examined histologically except in post-mortem examinations.

This practice probably means that idiopathic bilateral nodular hyperplasia of the zona glomerulosa occurs more often in cats than suggested by data based on histopathologic findings. In line with this hypothesis, Javadi *et al* reported 11 cats with "idiopathic" primary hyperaldosteronism in which diagnostic imaging failed to demonstrate an adrenal tumour, suggesting that the cause was adrenocortical hyperplasia, as is the case for the human condition.

Clinical findings in hyperaldosteronism

Clinical signs mainly relate to hypokalemia and systemic hypertension. Persistent and progressive weakness is noted due to a hypokalemic polymyopathy, characterized by cervical ventroflexion of the neck, hind limb weakness which leads to a plantigrade stance, difficulty in jumping, listlessness and ataxia. Some cats even exhibit limb rigidity, as well as dysphagia and eventually flaccid paralysis and collapse. Acute blindness or sudden change in eye color is often noted by owners and is generally due to intra-ocular haemorrhage or retinal detachment, both of which are a consequence of systemic hypertension.

Systemic hypertension can also cause seizures, ataxia and behavior change due to CNS oedema, haemorrhage, or ischemia. PU/PD is noted in less than 20% of cats, however hypokalemia can result in reversible, secondary nephrogenic diabetes insipidus, which will then exhibit overt PU/PD. Appetite is variable, some patients display polyphagia, but this is usually a sign of the concurrent excessive production of other hormones, such as progesterone or cortisol.

A physical examination will reveal clinical signs related to the hypokalemia/hypertension. Patients will often present with physical weakness. The eyes can show tortuous retinal vessels, retinal detachment as well as haemorrhage or oedema. The patient may have alopecia, a pot-bellied appearance and fragile skin, as a result of concomitant glucocorticoid or progesterone

excess. The heart could exhibit left ventricular hypertrophy due to the sustained systemic hypertension, which will likely be noted as a heart murmur, a gallop rhythm, or an arrhythmia.

Diagnosis of hyperaldosteronism

Diagnosis can be challenging (see Figure2). Hypokalemia is the typical abnormality seen (yet it can be absent in a proportion of, especially early, cases) and is persistent despite potassium supplementation. There will be increased urinary fractional excretion of potassium and this confirms that the potassium loss is renal. However, hypernatremia is uncommon due to the aldosterone escape phenomenon: hypertension and volume expansion overcome the sodium sparing effects of aldosterone, causing natriuresis. Creatinine kinase (CK) may also be increased due to hypokalemic myopathy, but this is variable and metabolic alkalosis is a common finding.

Since diagnosis is challenging, clinicians should have an increased index of suspicion for hyperaldosteronism in any cat with unexplained hypertension, especially if it is refractory to therapy. Imaging, specifically radiography is only helpful if a large adrenal tumour is evident. Importantly, adrenal calcification is present in 33% of older healthy cats, therefore avoid the over-interpretation of this finding. Pulmonary metastasis is an uncommon finding.

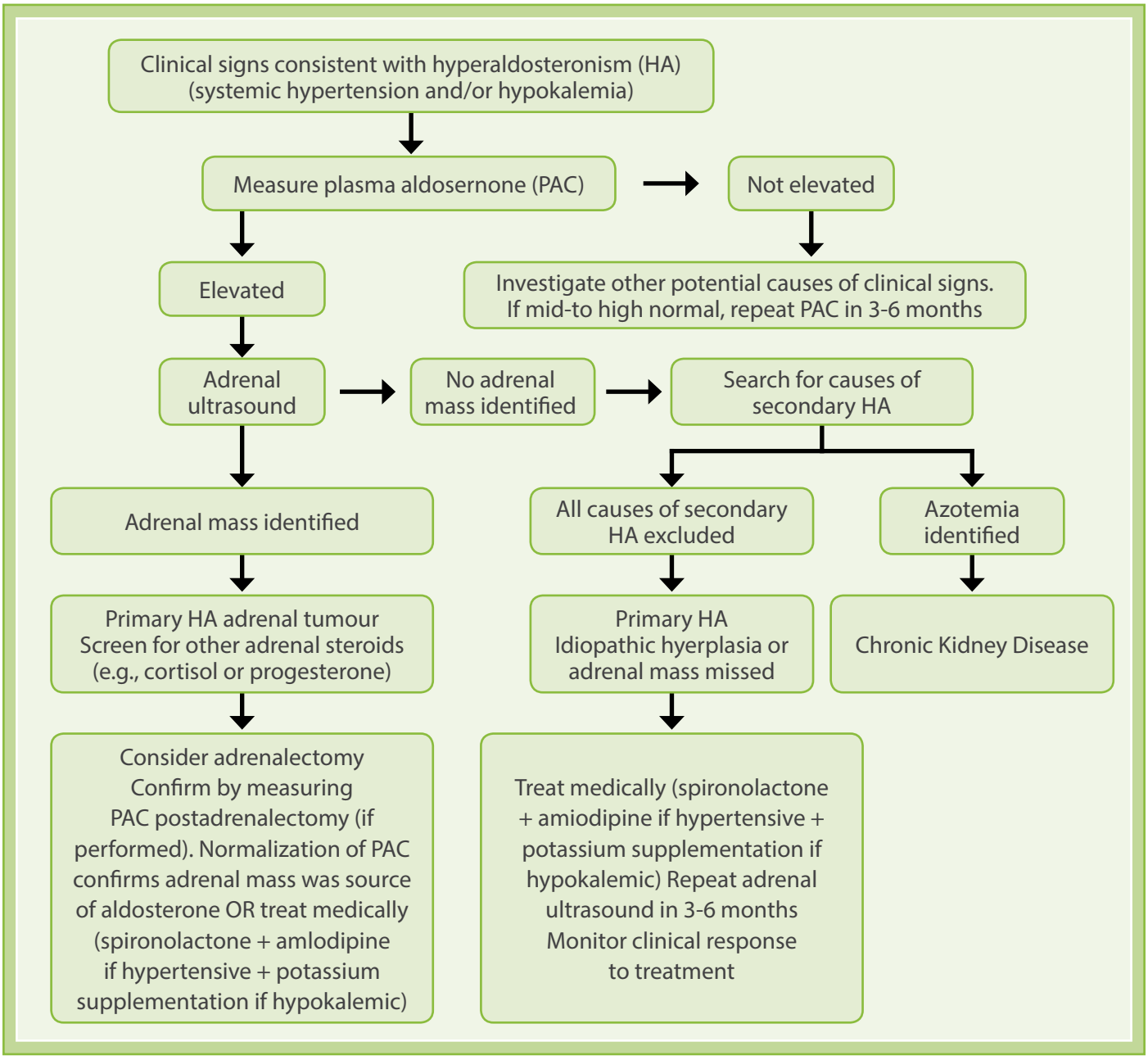

Figure 2. *Clinical approach to a cat with suspected hyperaldosteronism*

Abdominal ultrasound, on the other hand, is an especially useful tool in diagnosing these tumours as well as cases with bilateral hyperplasia of the zona glomerulosa. Most reported cases of cats with advanced hyper-aldosteronism have adrenal tumours on ultrasound, some even bilaterally, yet there is increasing evidence of cases with bilateral adrenal hyperplasia, commensurate with an increase in our diagnostic capabilities.

It is important to always interrogate the contralateral adrenal gland, because a decreased size could indicate additional glucocorticoid or progesterone secreting capacity in the adrenal tumour, which leads to negative feedback inhibition of ACTH secretion and subsequent contralateral adrenal atrophy.

Plasma aldosterone concentration (PAC) is used for confirmation of hyper-aldosteronism. Values in primary or secondary aldosterone concentrations will overlap, and early in the disease process some cats will have serum aldosterone concentrations in the mid to upper end of the reference range. Therefore, a high normal PAC in a cats with concomitant clinical signs raises the index of suspicion and should be repeated in 3-6 months.

Plasma potassium concentrations are an important determinant of PAC concentrations. Importantly, low potassium will necessarily lower PAC and therefore a mid-range to top normal aldosterone concentration in the face of hypokalaemia should raise the suspicion of hyperaldosteronism. The currently available human aldosterone assay through Ampath seems to work well. However, the human reference range of up to 1780 pmol/l that they provide is much higher than the high-normal cut-off for cats which is in the region of 540 pmol/l.

Plasma renin activity (PRA) can be used to rule out causes of secondary hyperaldosteronism. Hypertensive cats can have increased serum aldosterone concentrations, and it can be difficult to distinguish PHA from CKD, as they often co-exist. It is best to assess PRA and aldosterone concentration in one sample. With PHA, PRA will be low or within the reference range due to negative feedback, as explained above. In the case of secondary hyperaldosteronism, as is the case with CKD, PRA will be increased.

Other tests

Urinary aldosterone to creatinine ratio (UACR), has been used, but seems to have limited sensitivity.

Fludrocortisone administration, a synthetic corticosteroid with mineralocorticoid activity will suppress aldosterone secretion in healthy cats. Conversely, 6 out of 9 cases op PHA did not suppress, making non-suppression a very specific, yet insensitive test for PHA. Suspicion of PHA is based on persistent hypokalaemia and hypertension, despite treatment. Elevated plasma aldosterone concentration and the identification of an adrenal tumour or adrenal hyperplasia are consistent with the diagnosis. Histopathology will confirm tumour origin and resolution of clinical signs post adrenalectomy will confirm diagnosis.

Treatment

Adrenalectomy is the treatment of choice. Post-surgical treatment with oral or parenteral potassium is often needed. It is important to assess the patient for tumour thrombi and/or metastasis, as these could increase surgical and post-surgical complications, as well as survival rate. If the tumour is secreting other hormones such as glucocorticoids or progesterone, adrenalectomy could result in a transient cortisol deficiency, which needs to be closely monitored and treated, if necessary.

Cats that are not surgical candidates can be treated medically to control the hypokalaemia and hypertension. Potassium supplementation is key, as well as drugs to lower blood pressure, such as the calcium-channel blocker, amlodipine. Hypertension however may become refractory to medication. Spironolactone is a competitive aldosterone receptor antagonist, which helps to lower blood pressure and increase potassium and acts as a reno-protective agent (see discussion).

Discussion

Glomerular- and tubular-interstitial injury initiate a cascade of pathogenic events that lead to chronic renal insufficiency. More specifically, excessive accumulation of extracellular matrix (ECM) plays a central role in this progressive loss of renal function. In this regard, several mediators promote ECM accumulation, including transforming- and connective tissue growth factor as well as RAAS activation. Angiotensin II is a potent peripheral vasoconstrictor, thereby regulating glomerular filtration, whilst also acting as a growth factor and a true cytokine.

Lately, its true proinflammatory mediating effects has been found to contribute to the progression of renal damage. Angiotensin II, in addition to low salt intake, are potent secretagogues for aldosterone, which, of its own accord, has been identified as instrumental in promoting thrombosis and fibrosis in the kidneys. Moreover, bilateral adrenal hyperplasia causes lower aldosterone levels than is the case with tumours.

These relatively low aldosterone levels fail to fully suppress plasma renin concentrations and this apparently contributes to progressive renal vascular damage.

Once this reno-vascular damage is established, it leads to more renin release even in the presence of elevated aldosterone levels. Thus, in relatively mild hyperaldosteronism, the kidneys are persistently exposed to all these mediators of vascular change and fibroproliferative destruction, viz. renin, angiotensin II and aldosterone.

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1. Which ONE of the following statements regarding the epidemiology of hyperaldosteronism is WRONG?

a. Hyperaldosteronism is the most common adrenal disorder in the cat.
b. Hyperaldosteronism is more common in cats than in any other domestic animal
c. Hyperaldosteronism is the cause in at least 20% of humans with hypertension.
d. Arterial hypertension and hypokalaemia are frequently attributed to chronic kidney disease, halting further diagnostics.
e. Primary hyperaldosteronism, also known as Conn's syndrome, is an adrenocortical disorder characterized by excessive, autonomous secretion of glucocorticoids, mainly aldosterone.

2. In which layer of the adrenal gland is aldosterone synthesised?

a. Z. fasciculata
b. Z. glomerulosa
c. Z. reticularis
d. Adrenal medulla
e. a) and c)

3. Which ONE of the following hormonal/metabolic changes will stimulate the release of aldosterone?

a. High salt diet
b. Hypotension
c. Hypertension
d. High serum potassium
e. Low plasma renin

4. Which ONE of the following is an additional clinical sign in cats with hyperaldosteronism and excessive cortisol production?

a. Flaccid paralysis
b. Neck ventroflexion
c. PU/PD
d. Thin abdominal skin
e. Dermal hyperpigmentation

5. Which ONE of the following is NOT a likely finding in cats with primary hyperaldosteronism?

a. Acute onset blindness
b. Limb rigidity
c. Hyperkalaemia
d. Normal to low plasma renin
e. Ataxia

6. Which ONE of the following is the optimal test for the diagnosis of early primary hyperaldosteronism in cats?

a. PAC
b. PRA
c. PAC/PRA ratio
d. Urinary aldosterone to creatinine ratio
e. Serum potassium

7. Which ONE of the following drugs is indicated for the treatment of systemic hypertension in cats with hyperaldosteronism?

a. Pimobendan
b. Amlodipine
c. Fludrocortisone
d. Phenoxybenzamine
e. Mifepristone

8. Which ONE of the following is the treatment modality of choice in cases with unilateral aldosterone-producing adrenal mass?

a. Surgical adrenalectomy
b. Chemical adrenalectomy
c. Spironolactone
d. Fludrocortisone
e. Monitoring to see if the other adrenal becomes affected

9. Which ONE of the following drugs is NOT used in the treatment of cats with non-tumourous hyperplastic adrenal tissue; i.e. bilateral adrenocortical hyperplasia or a non-resectable unilateral adrenocortical neoplasm?

a. Mineralocorticoid receptor blocker
b. Calcium channel blocker
c. Oral potassium supplementation
d. Fludrocortisone
e. All are used

10. Which ONE of the following metabolites/hormones is UNLIKELY to play a role in the progression of renal disease in cats

a. Aldosterone
b. Renin
c. Angiotensin II
d. Thyroxine
e. Cortisol

Neuronal Ceroid Lipofuscinosis (NCL) in Dogs

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NCL are a group of progressive inherited lysosomal storage disorders of the central nervous system. Lysosomes are structures in cells that can be thought of as the garbage disposal unit of the cell.

These organelles are responsible for breaking down waste and other by-products that accumulate in the cells. Dogs affected with NCL lack one of several enzymes which are necessary for the correct form and transport of the lysosomes.

Without the correct receptor sites, the lysosomes cannot be transported to the areas of the cell where they are needed to perform the normal breakdown these products. As this "debris" accumulates in neuronal cells (and to a lesser extent in other cells), the animal's mental and motor functions start to deteriorate.[3]

Different causative mutations of NCL have been described in at least 13 genes. As many of the clinical signs for the different types of NCL can overlap significantly, genetic testing is the only definitive way of establishing which type of NCL the dog is suffering from. Cases of NCL have been documented in over 20 canine breeds and mixed breed dogs [2], with known causative variants tabulated on the right:

Type	Breed	Mutation
CLN1	Cane Corso	G > A splice donor mutation
CLN1	Dachshund	Small insertion
CLN2	Dachshund	Small insertion
CLN5	Golden Retriever	Small deletion
CLN5	Border Collie, Australian Shepherd	C > T missense mutation
CLN6	Australian Shepherd	T > C missense mutation
CLN7	Chihuahua, Chinese crested	Small deletion
CLN8	Saluki	Small duplication
CLN8	Alpenländische Dachsbracke	Large deletion
CLN8	Australian Shepherd	G > A nonsense mutation
CLN8	English Setter	T > C missense mutation
CLN10	American Bulldog	G > A missense mutation
CLN12	Cattle Dog	C > T missense mutation
CLN12	Tibetan terrier	Small deletion

Dogs born with NCL will develop normally and appear to be fully functional. Depending on the type of NCL they have and the severity of the dysfunctional protein, they will begin to develop clinical signs between 6 months and 4-6 years (adult-onset types may vary in progression). The rate of progression will be linked to the type of NCL the dog presents with.

Clinical signs of NCL may include the following [2,3,4]:

- Intellectual decline
- Motor disturbance progressing to seizures, tremors
- Respiratory impairment
- Lack of muscle coordination, difficulty swallowing
- Visual disturbances that may progress to blindness
- Difficulty balancing, abnormal gait
- Behavioural changes such as aggressiveness
- Loss of learned skills
- Anxiety, Confusion, depression, dementia
- Aimless wandering or circling
- Eventual death

The progressive nature of this disease often results in euthanasia of the affected dog by 3 years, due to the poor quality of life. [3]

Diagnosis of NCL can be difficult as many unrelated diseases can share overlapping symptoms. Should the affected dog pass before testing is done, the nervous system tissue can be examined upon necropsy. Although cartinene levels in the blood may be lower in affected dogs, these levels alone do not provide a definitive diagnosis. The only way to accurately test for NCL, is through genetic testing. Laboratories will be able to test for known NCL causal variants, although there could still be undiscovered mutations causing similar symptoms. [4]

It is important for breeders to remember that NCL is a recessively inherited disorder, which means that the affected puppy requires 2 copies of the mutated gene in order to develop symptoms. Should only 1 copy be inherited from a parent pair, the puppy will not develop NCL, but will be a carrier of the mutation. This in turn means that the dog will be able to pass on this mutation to offspring, should it be used for mating. If paired with another carrier, each puppy born will have a 25% chance of developing NCL. Because of the severity of this disease and the current lack of any treatment or cure, breeders should seriously consider knowing the NCL status of their animals and avoid breeding carrier to carrier to prevent the spread of this fatal disease [1].

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No More Kidney Around: Anaesthesia and the Renally Impaired Cat

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Pre-planning and basic diagnostics can take the stress out of anaesthetising cats with kidney problems in your veterinary practice.

The kidney is a complex organ with three primary functions: filtration, reabsorption and secretion. The renal system, which the kidney is part of, filters blood by removing nitrogenous waste while preventing various solutes, proteins and blood cells from being excreted. The renal system also maintains the appropriate balance of sodium and water; regulates acid-base balance; upholds and regulates electrolyte homeostasis, bone metabolism and erythropoiesis; participates in controlling blood pressure and is involved in drug metabolism and excretion.[1] As such, the kidney is an important organ to manage, preserve and maintain during anesthesia.

The kidney receives approximately 25 percent of cardiac output[2,3] and consumes a large quantity of oxygen. Blood flow to the kidney (renal blood flow; RBF) is reflected in the glomerular filtration rate (GFR; the amount of blood passing through the glomerulus each minute).

Renal autoregulation stabilises the GFR despite large fluctuations in systemic blood pressure. Maintaining constant perfusion pressure preserves and protects the kidney during bouts of hyper- or hypotension. The range at which autoregulation occurs is approximately 80-180 mm Hg.[4]

Due to their inherently unique vasculature, the kidneys may experience local tissue ischaemia and hypoxia regardless of normal organ blood flow. Blood pressure support by way of fluid therapy is recommended in the renal patient.

There is no single test that is an ideal way of assessing renal function. Instead, performing a variety of tests can offer the most complete picture. When running traditional tests, a 75 percent decrease in renal function must be present before abnormalities are noticed on a blood chemistry profile.[5] Even mild increases in serum blood urea nitrogen (BUN) and creatinine concentrations may indicate severe disease. The biomarker test, symmetric dimethylarginine (SDMA-Idexx) can detect as little as a 25 percent decrease in GFR, alerting clinicians to early renal disease. Evaluation of all middle-aged and older patients and those with suspected renal disease is recommended and test trends are more useful than singular measurements.[6]

Renal patients may present without clinical signs or they may be dehydrated, anaemic, azotaemic, anorectic or have acid-base abnormalities. A thorough physical exam and detailed history should precede a complete blood count (CBC), serum chemistry profile and urinalysis (at minimum, a urine specific gravity) plus the SDMA biomarker test for all suspected renally impaired patients.

These tests provide vital information that can reduce patient morbidity and mortality. For example, azotaemia increases the sensitivity to anaesthetic drugs by affecting the permeability of the blood brain barrier.[7] Having this type of data prior to choosing and delivering drug dosages can help to avoid overdosing a patient and increasing morbidity.

Some anaesthetic drugs can have deleterious effects on renal physiology but for the most part tend to be well tolerated at appropriate doses. Phenothiazines (e.g. acepromazine) can cause vasodilation and subsequent hypotension that may dip below the range of autoregulation.[8,9] Appropriate doses along with careful monitoring of blood pressure and treatment of hypotension is essential.

Alpha-2 adrenergic agonists (e.g. medetomidine, dexmedetomidine) are used cautiously in cats with renal disease because this class of drug has the potential to cause up to a 60 percent decrease in cardiac output.[10] In addition, a 50 percent decrease in RBF is seen in dogs alongside an increase in GFR but no definitive evidence exists to support this in cats; however, it would be prudent to assume these mechanisms may be similar in cats as well. Alpha-2 agonists should be avoided in obstructed cats due to their diuretic effect and should be used with caution in all other cases of renal impairment if possible.

Benzodiazepines (e.g. diazepam, midazolam) are well tolerated in renally impaired cats; nevertheless, this class of drugs can cause paradoxical excitement and should be used alone with caution in cats or young, healthy animals. Co-administration with an opioid is recommended. Opioids (e.g. morphine, hydromorphone, fentanyl, buprenorphine, methadone, butorphanol) are generally safe for renally impaired cats[11] and their use is often beneficial.

Opioid administration may help decrease the sympathetic response associated with pain and surgery, thereby minimizing renal vasoconstriction. The addition of opioids can also decrease the amount of inhalant necessary to keep the patient immobilized, avoiding unnecessary hypotension.[12]

Nonsteroidal anti-inflammatory drugs (NSAIDs) have the potential to cause ill effects in renally impaired patients but multiple studies evaluating COX-2-specific NSAIDs in healthy patients with moderate hypotension has not been established.[13-16] Some evidence exists that supports the careful use of NSAIDs in renally impaired cats.

Induction agents like propofol and alfaxalone are good choices for anaesthetic induction since the negative effects associated with these drugs are transient and generally well-tolerated.[17-19]

The use of premedication is recommended to reduce the dosage necessary to induce anaesthesia. Ketamine as an induction agent is often avoided in the renally impaired cat mainly because this drug partially relies on renal excretion. Compromised renal function may lead to impaired drug elimination.[20] Subanaesthetic doses of ketamine as an adjunctive medication are considered safe.

Inhalants should always be used sparingly, but especially in renally impaired patients. Inhalants are potent vasodilators which may cause hypotension and a reduction in RBF. The effects of inhalants are usually rapidly reversed once discontinued, helping its safety profile.

When anaesthetising a renally impaired cat, it becomes more important to focus on the management of the individual and the sequelae of their disease in order to avoid exacerbation of their illness. The general rule of thumb is to stabilise before you anaesthetise. It is also understood that this is not always possible since renally impaired cats do sometimes present an emergency. Weigh the value of the procedure against the risks of the event.

Begin improving hydration by replacing fluid loss with a balanced electrolyte solution, keeping a close eye on electrolytes and blood pH. Intraoperatively, monitoring and supporting blood pressure is essential. Clinicians need to be prepared and efficient to complete any procedure in a timely manner. Positive ionotropic support may be indicated to maintain arterial blood pressure and subsequent renal blood flow well within the range of autoregulation.[21]

As is true in all surgical procedures, pain should be adequately treated. Untreated or undertreated pain may cause renal vasoconstriction and a reduction in renal blood flow. Sufficiently treating pain using a balanced approach is best practice and can be achieved by utilising locoregional techniques and adjunctive medications to keep inhalant settings low whilst maintaining adequate blood pressure. In the recovery period, renal patients should be maintained on intravenous fluids to ensure adequate fluid volume and diuresis. Body temperature should be supported and oxygen supplemented when deemed necessary.

A renally impaired cat doesn't need to be a stress-inducing challenge when anaesthesia is needed. Basic diagnostics and pre-planning can prove beneficial. Proper dosages, dilligent monitoring and fluid support can mean the difference between an animal that just survives the procedure and one that thrives.

References www.vet360.vetlink.co.za

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