SUMMER 2018

REFERRAL NEWS

IN THIS EDITION:

Bath Veterinary Referrals

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Summer update from BVR...

I'm sure like us you have all been sweltering in the heat, and many pets are struggling just as much as we humans. Nonetheless, work goes on, the last few months have been very busy for us, hence why this latest newsletter is a bit later than planned.

However, we are excited to have two new clinicians join us to help with the workload and bring new skills and interests to the team. Anna Ellams joins the medical referral team from a busy hospital where she has been managing the medical and critical cases. As well as the normal ranges of medicine cases, Anna has a keen interest in small animal emergency and critical care cases as well as veterinary anaesthesia and analgesia. Barbara Karolczak holds a certificate in surgery, and has joined us as a surgeon, working in both orthopaedic and soft tissue surgery fields. Our new team members have improved our ability to see cases at short notice, and we are always happy to be contacted for no-obligation advice.

Alex Gough
Head of Referrals

Patient Safety at Bath Veterinary Referrals

At Bath Veterinary Referrals, we have a keen interest in patient welfare and safety. We were an early adopter of the concept of pre-anaesthetic checklists, and have been successfully using these to reduce the risk of avoidable errors for some time. We closely monitor adverse reactions, and take action if a problem is identified. We also have a clinical care committee that oversees adverse reactions and discusses new protocols for optimal clinical care of our patients, such as monitoring, fluid therapy and analgesia.

You can be confident that patients referred to Bath Veterinary Referrals are treated with meticulous care.

A case of feline hyperadrenocortism

Lisa Gardbaum BVet Med CertSAM MRCVS

Case history

An eight year old male neutered DSH cat had initially been seen by the referring veterinary surgeon with a history of lethargy, weight loss, polyphagia, abnormal behaviour and diarrhoea. Blood tests at that time had shown raised levels of ALT at 97u/l (5-60), mildly raised cholesterol levels of 4.31mmol/l (2-2.4) and a mild hyperglycaemia of 14.6mmol/l (3.9-9). Total thyroxine levels had been in the low- normal range.

He had continued to lose weight and had remained lethargic and repeat examination had shown him to have a poor coat with skin excoriations of his flanks and a poor body condition with abdominal distension. As blood glucose remained high, he had been started on Caninsulin (insulin lente) at one unit twice daily. He had subsequently developed a large ulcerated and infected wound on his right flank, dyspnoea and tachypnoea and had developed further abdominal distension.

Presentation and clinical findings

At this stage, he had been referred to our medical referral team. On presentation he had marked dyspnoea and tachypnoea, a distended abdomen with a palpable fluid thrill, muscle atrophy and a very poor body condition of 2/9. There was severe generalised thinning of the skin with sloughing of skin on the right flank and severe skin ulceration.

Diagnostic tests

An abdominal ultrasound scan showed the presence of ascites, a large, slightly hyperechoic liver and bilaterally enlarged adrenal glands

Continued over...

A case of feline hyperadrenocortism continued.

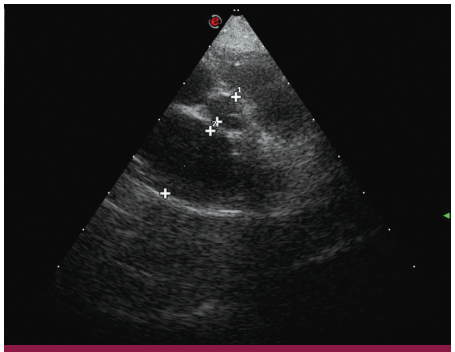


Figure 1: Right short axis view of the heart at the level of the aorta showing a large left atrium relative to the aorta. Ratio of 1:2.47 (normal <1.3).

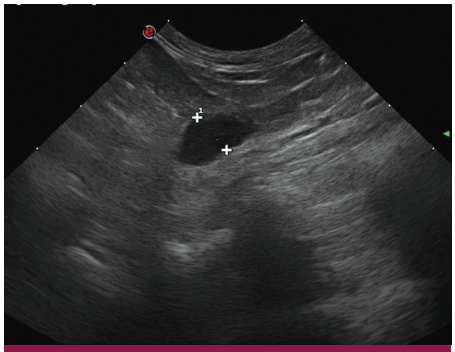


Figure 2: Large left adrenal gland suggestive of adrenal hyperplasia secondary to pituitary dependent hyperadrenocortism. Maximum width 0.72cm.

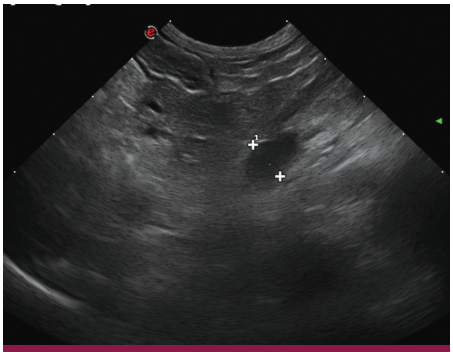


Figure 3: Large right adrenal gland suggestive of adrenal hyperplasia secondary to pituitary dependent hyperadrenocortism. Maximum width 0.77cm.

with a maximum diameter of 0.72cm of the left adrenal gland and 0.77cm of the right adrenal gland (see photos). Cardiac ultrasound examination showed the presence of a markedly dilated left atrium with an aortic to left atrium ratio of 1:2.47 (<1.3) but normal ventricular thickness suggestive of a restrictive cardiomyopathy (see figure 1).

Blood tests at this time showed a mild non-regenerative anaemia with a haematocrit of 26%, a marked neutrophilia, a mild lymphopaenia, a mild monocytosis and a mild hypophosphataemia of 0.8mmol/l (1.2-2.42). Urinalysis showed a urine specific gravity of >1.050 and a negative bacterial culture. The levels of B12 were low at 183ng/l (240-440) and folate levels were normal.

Treatment and further testing

Treatment was started on frusemide, benazepril, potassium phosphate, amoxycillin-clavulanate and clopidogrel and the insulin was changed to Prozinc (protamine zinc insulin) one unit twice daily as this is considered the insulin of choice in cats due to its longer duration of action than insulin lente. The large wound of the right flank was flushed with saline four times daily and kept covered to avoid contamination. Injections of B12 were started at 250ug subcutaneously once weekly for six weeks then monthly. The following day, the ascites had resolved and his breathing rate and effort had significantly improved. A low dose dexamethasone suppression test was carried out using 0.1mg/kg dexadreson intravenously and measuring cortisol levels pre- injection and four and eight hours later. Pre-injection cortisol levels were high at 359mmol/l (15-150) and four and eight hour post dexadreson cortisol levels were 248nmol/l and 312nmol/l respectively. As there had been no suppression in cortisol levels (normally <40nmol/l) and both adrenal glands had been markedly enlarged on ultrasound examination, a diagnosis of pituitary dependent hyperadrenocortism was made. Treatment was started with 10mg trilostane twice daily.

Follow- up

He continued to improve in terms of his demeanour and the wound on the right flank started to heal. He started to gain weight but new wounds on the right and left flanks started to form a few days later with sloughing, excoriation and a purulent discharge. Bacterial swabs from this grew a heavy growth of coagulase positive Staphylococcus sensitive to the amoxycillin-clavulanate that he had already been having and was negative for MRSA and fungus.

Two weeks after starting trilostane, he was well in himself although remained polyphagic and had not gained any weight. There was definite improvement of the skin thinning and the previous wounds had healed although other wounds were forming. A blood glucose curve showed a nadir of 14mmol/l, five hours post insulin and the levels then increasing to 24mmol/l, seven hours post-insulin. The dose of insulin was increased to 1.5 units Prozinc twice daily.

An ACTH stimulation test was carried out at this time to assess control of the hyperadrenocortism. Pre-ACTH cortisol levels were 99nmol/l (<125), one hour post ACTH levels were 178nmol/l (<500) and three hours post ACTH levels were 159nmol/l.

Although these results suggested reasonable control of the hyperadrenocortism, as new skin lesions involving sloughing were occurring, it was decided to increase the dose of trilostane to 30mg daily.

He started to gain weight and three weeks later the skin lesions were healing well although his respiratory effort had increased. Repeat cardiac ultrasound examination showed the presence of pleural fluid which was drained by thoracocentesis under sedation. Pimobendan was started at 0.625mg twice daily in addition to the benazepril and after two weeks, the benazepril was stopped and the pimobendan continued as his breathing had remained stable. The dose of insulin was further increased to two units Prozinc twice daily as blood glucose levels failed to reduce to below 17mmol/l with a repeat glucose curve. As there were still some new skin lesion developing the dose of trilostane was increased further to 20mg twice daily. Finances prevented further ACTH stimulation tests being carried for monitoring purposes. A further two weeks later, the dose of insulin was increased again to 2.5 units after another glucose curve showed blood glucose levels remained high.

Three months after diagnosis, he remains well with no new skin lesions and a normal respiratory rate and effort, although he has still failed to gain any further weight. Blood glucose levels on a recent blood glucose curve showed levels of between six and 15mmol/l so the dose of insulin was reduced back to two units twice daily and a repeat glucose curve is planned one week later to assess whether further reductions in insulin dose are required.

Discussion

This case showed a classic presentation of hyperadrenocortism in cats. It is a rare condition in cats but veterinarians should still be aware of its clinical signs in cats and how it differs from the condition in dogs as, if left untreated, prognosis is extremely poor to grave, whereas treated cats can often have a good quality of life with a median survival of 20 months with a range of three months to three years.

Approximately 80-85% of cats with hyperadrenocortism, have pituitary dependent form of the disorder as in this case. It is a disease of middle aged to older cats with a median age of 11 years.

In contrast to dogs with hyperadrenocortism, cats are quite resistant to the effects of excess glucocorticoids and the onset of polyuria and polydipsia is often delayed and often only become polyuric and polydipsic once they develop diabetes mellitus. Cushingoid cats that do develop PU/PD, tend to have a urine specific gravity of >1.020 compared to dogs who tend to have very poorly concentrated urine.

Approximately 85-90% cases have concurrent diabetes mellitus compared to only 5% of dogs and resistant diabetes is the most common presentation. The dose of insulin often needs to be reduced after starting medical treatment for hyperadenocortism as reduced cortisol levels will reduce insulin antagonism and enhance insulin action. Most cats, however, remain diabetic after treatment with trilostane.

Up to half of all cats with hyperadrenocortism develop fragility of skin which very rarely develops in dogs. This causes tearing of skin with routine handling leaving large denuded areas. As with dogs there is often skin thinning, an unkept hair coat and bilateral symmetrical alopecia.

A major difference between cats and dogs with hyperadrenocortism is that cats tend to get marked weight loss rather than weight gain. The weight loss in most of these cases is due to the concurrent poorly controlled diabetes. Muscle weakness, lethargy and a pot-bellied appearance are similar in both species.

Biochemistry differences between the two species with hyperadrenocortism are mainly that ALKP is only elevated in 10-20% of cats compared to 90% of dogs due to lack of steroid induction of a specific isoenzyme of ALKP in cats and the elevation of ALKP in cats, is most likely due to the poorly controlled diabetes rather than the glucocorticoid excess. Cholesterol levels are increased in only 25% of cats compared to 90% of affected dogs.

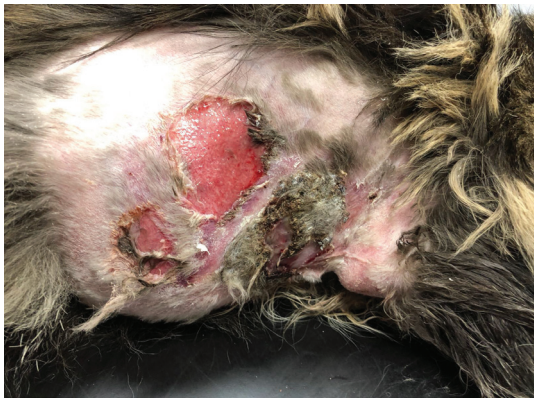
The low dose dexamethasone suppression test is considered the test of choice for diagnosing hyperadrenocortism in cats as the ACTH stimulation test is poorly sensitive with only 35% to 55% of cats compared to about 85% sensitivity in dogs. Up to two thirds of cats with the disease have normal results.

The low dose dexamethasone suppression test in cats uses a ten fold higher dose of dexamethasone (0.1mg/kg) than is used in dogs (0.01mg/kg). In healthy cats and cats with non-adrenal diseases, cortisol concentrations should suppress to <40nmol/l. It has an almost 100% sensitivity and a reasonably high specificity and so is the best screening test for hyperadrenocortism in cats. The urine cortisol/creatinine ratio is also a very sensitive test with a sensitivity of 70-90% but specificity is much lower than the LDDST so is best for ruling out a diagnosis of hyperadrenocortism rather than confirming a diagnosis.

For monitoring purposes, the ACTH is the most useful test and should ideally be carried out two weeks, one month, and 2-3 months after starting treatment and then every 2-4 month. The ideal post-ACTH cortisol concentration should be 50-150nmol/l. Trilostane is the medical treatment of choice with recommended starting doses of 20-30mg/cat per day, divided at the time of feeding. Dose adjustments are commonly needed and should be made based on post ACTH cortisol levels and more importantly, on clinical signs. The dose can be increased to 30-60mg/cat/per day and some cats need doses as high as 90-120mg to control clinical signs and lower ACTH-stimulated cortisol concentrations into the ideal range.

In this case, it is likely there was concurrent restrictive cardiomyopathy which had been exacerbated by the chronic excessive glucocorticoids and control of the hyperadrenocortism led to stabilization of the disease.

The low B12 levels likely represented a concurrent inflammatory bowel disease which was the likely cause of the previous diarrhoea and current lack of weight gain. He is now being trialled on a single protein prescription diet for two months in addition to monthly B12 injections to see if he gains weight. As diabetes is currently very well controlled, it is considered an unlikely cause of the failure to gain weight.



Figures 4 and 5: Denuded areas of skin due to fragile skin syndrome secondary to hyperadrenocortism on presentation to the referral team

Figure 6: Healing wounds two weeks after starting treatment with trilostane

Figure 7: Recent photo showing complete healing of skin wounds

Cases recently seen

Immune mediated neutropenia, secondary to a benign splenic mass in a ten year old Giant Schnauzer; Concurrent immune-mediated neutropenia and immune-mediated destruction of red cell precursors in a Cocker Spaniel; relapsing acute polyradiculoneuritis in a young collie; medial subluxation of the bicipital tendon in a seven year old cross breed; primary pulmonary carcinoma; recurrent diabetic ketoacidosis in a cat; brachial plexus tumour in a dog; cervical ischaemic myelopathy in a German Shepherd; hippurate/adipic aciduria associated with seizure-like episodes in a seven month old French Bulldog.

Why choose Bath Veterinary Referrals?

- We pride ourselves on giving you the highest level of service
- We strive to enhance your reputation, looking after your clients and their pets in a way you would be proud of
- We offer a caring, friendly and personalised service. We keep clients and referring vets informed at all times
- We have a superb team of night nurses and night vets, a flagship hospital and the very latest equipment

Our clinicians

Alex Gough MA VetMB CertSAM CertVC PGCert (Neuroimaging) MRCVS – Head of Referrals

Jon Shippam BVSc CertSAS MRCVS - Orthopaedic Surgeon

Jenny Lambert BVM&S CertVOphthal MRCVS - Ophthalmology

Lisa Gardbaum BVetMed CertSAM MRCVS - Internal Medicine

Samantha Lane BVSc BSAVAPGCertSAS MRCVS – Soft Tissue Surgeon

Federica Manna DVM CertAVP MRCVS - Assistant to Internal Medicine

Edward Corfield BVSc CertAVP MRCVS - Assistant Referral Surgeon

Barbara Karolczak MSc CertSAS MRCVS – Soft Tissue Surgeon

Anna Ellams BVMS CertAVP MRCVS - Internal Medicine

Organising a referral is simple

To make a non-urgent referral please email **contact@bathvetreferrals.co.uk** or call the team on **01225 832 521**, **option 3**.

To make an urgent referral please call one of our Referral Administrators who will be happy to take down the case details and speak with the team regarding an appointment. Where possible we will see emergency cases on the same day they are referred to us. Tel: **01225 832 521**, **option 3**.

To request advice on a case from one of our clinicians, please email or call the team using the details above.

Once you have contacted us, with your permission we will speak with the client directly to book a convenient appointment.

We ask that you forward any client history to us as soon as possible using the email address above.

Free Radiograph Reading

To receive a free radiograph interpretation please email your images to **contact@bathvetreferrals.co.uk**.

One of our experienced clinicians will email in response at their earliest convenience.