SUMMER 2017

REFERRAL NEWS

In this edition:

Bath Veterinary Referrals

Rosemary Lodge
Veterinary Hospital
Wellsway
Bath
BA2 5RL

bathvetreferrals.co.uk
t: 01225 832521
f: 01225 835265
e: contact@bathvetreferrals.co.uk

FELINE HEPATIC LIPIDOSIS

Federica Manna
DVM MRCVS

Hepatic lipidosis (HL) is an acquired, multifactorial syndrome that develops in cats in association with profound and protracted anorexia. This syndrome is characterised by an excessive accumulation of lipids within hepatocytes and can lead to severe hepatic dysfunction or death.

Cats are unique in their tendency to develop this disorder. Peripheral fat mobilisation exceeding the hepatic capacity to either redistribute or use fat for β-oxidation (producing energy) leads to profound hepatocyte cytosolic expansion resulting in triglyceride (fat) accumulation. Damage to the liver is caused by swelling of liver cells filled with fatty deposits as well as additional processes.

A majority of cats that develop HL are historically overweight, and most have experienced a period of anorexia before the onset of lipidosis. Most affected cats are middle-aged adults (median age seven years) of either sex with no breed predilection.

HL can occur secondary to any disease process ("secondary HL") that results in marked decrease in appetite. Common co-morbidities include diabetes, pancreatitis, small intestinal disease, chronic renal disease, neoplasia and others. Hepatic lipidosis can occur in an otherwise healthy cat ("primary HL") due to inadequate intake during periods of forced overly rapid weight loss, unintentional food deprivation, change to a food unacceptable to the cat, sudden change in lifestyle, or stress (eg, boarding).

Anorexia is the primary, and sometimes the only, presenting complaint in cats with this syndrome. Other historical findings are vomiting, weight loss, pallor, diarrhoea or constipation and poor coat condition. Weakness, depression, and ptyalism may also indicate concurrent hepatic encephalopathy (HE). Common physical examination findings are jaundice, dehydration, loss of muscle mass, hepatomegaly and ventroflexion of the neck.

Laboratory results of HL patients may reflect a variety of abnormalities as they relate specifically to cholestasis or to other processes. The most consistent laboratory findings are mild to moderate normocytic, normochromic, non-regenerative anemia, poikilocytosis, increased Heinz bodies, variable WBC count, hyperbilirubinemia,

Summer update from BVR...

Welcome to the summer edition of our newsletter. I hope your year is running according to plan, including setting dates for a summer holiday!

As ever, we have several new developments at Rosemary Lodge. Most notably, we are excited to launch a new Oncology service. Robert (Bob) Matus is joining us this month. Bob is double boarded in Oncology and Internal Medicine and one of the most experienced veterinary oncologists worldwide. He has spent most of his career in the USA but is excited about coming to live and work in England. Bob will be joining us at our next CPD day and leading the day in the autumn.

We have also recently completed installation of a new mid-field MRI scanner which enhances our ability to scan larger dogs and to obtain more detailed scans in all cases.

In other staff changes, Ted Corfield is joining us as an assistant referral surgeon and we are saying good bye and good luck to Rhiannon Strickland as she leaves us to take up a residency at the RVC.

Finally and with regret, we are winding down the Exotics Referral service and Elisabetta has moved on to a position elsewhere. We will still try and accommodate outpatient CT scans on these patients if you contact us in the usual ways (phone or e-mail).

The lead article in this newsletter has been written by Federica and addresses hepatic lipidosis in cats, fitting into the theme of our next CPD day in June.

Feline Hepatic Lipidosis continued.

hypoalbuminemia, and increases in serum activity of ALP. ALT activity is less consistently increased than ALP. In contrast to other hepatobiliary diseases with a significant necroinflammatory component involving the pancreas, liver, bile ducts, or gallbladder, HL is not usually associated with elevated γ-glutamyltranspeptidase activity (GGT). The GGT:ALP relationship is useful in discerning underlying cholangitis/cholangiohepatitis and other diseases involving biliary structures (including pancreatitis).

Oesophagostomy tube placement for assisted feeding.

Hypokalemia, hypochloremia, hypophosphatemia, and hypomagnesemia may be present as the consequence of anorexia or vomiting or may develop during treatment as a result of volume expansion or refeeding. Blood urea nitrogen (BUN) concentration is low in more than half of the cats with lipidosis, reflecting generalised hepatocyte dysfunction. Depending on underlying disorders, hypoalbuminemia and hyperglobulinemia may be found. Prolonged PT or APTT may develop.

Ultrasonographic findings include hepatomegaly and diffuse hyperechogenicity of the liver. Hyperechogenicity, however, is not pathognomonic and may be seen in other feline hepatic disorders.

The diagnostic goal in cats with HL is two-fold: definitive diagnosis of the presence of lipidosis and simultaneous workup for the presence of an underlying disease process. A presumptive diagnosis of hepatic lipidosis may be reached by history, physical examination findings, clinical laboratory testing, and ultrasonographic findings. A liver fine-needle aspirate or, preferably a laparoscopic biopsy and histopathology along with the above supports the diagnosis and rules out other concurrent hepatic conditions. Cats should be closely monitored for significant post-biopsy bleeding due to the risk of haemorrhage from a friable, fatty liver.

The goals in treatment of the HL cat are to provide nutritional support, correct fluid and electrolyte imbalances, monitor for and manage complications of liver disease (coagulopathy, nausea, refeeding syndrome, HE) and identify and treat any underlying conditions.

Because cats with HL may have high lactate concentrations and may not be able to metabolise acetate, 0.9% NaCl is the fluid of choice. Fluids should be appropriately supplemented with potassium based on electrolyte status.

If the cat is very ill at presentation a naso-oesophageal tube can be placed for short-term nutritional support while the cat is stabilised. Depending on patient tolerance and clinical progress, this may be replaced a few days later by either an oesophagostomy or gastrostomy tube. Most cats need four to six weeks of tube feeding.

Dietary protein is the nutrient that is most efficient at reducing hepatic lipid accumulation in cats in negative energy balance. Protein restriction is contraindicated unless needed in the < 5% of cases with hepatic encephalopathy (HE). Carbohydrates are less well tolerated than lipids as a source of calories. Diets that are too high in carbohydrates may cause diarrhoea, abdominal cramping, borborygmi and hyperglycemia.

Occasionally, cats with lipidosis may develop re-feeding syndrome when oral nutrition is introduced, with a marked hypokalaemia and hypophosphataemia which, in turn, can lead to haemolysis. Phosphate can be supplemented by administering potassium phosphate and the food should be introduced more gradually.

Many cats require vitamin K therapy for the management of coagulopathies.

Vomiting is common in HL cats that are being re-fed and can result from severe hepatic dysfunction and reduced stomach volume. Metoclopramide (CRI of 1–2 mg/kg/d) is often a first-choice drug because of availability and low cost, and because it has both antiemetic and prokinetic effects. Additional antiemetics may need to be added if nausea and/or vomiting persist (e.g, maropitant 1 mg/kg IV, SC q24h or ondansetron 0.22 mg/kg q8–12h IV). Nevertheless, antiemetics often facilitate reintroduction of food.

An H2 receptor antagonist (famotidine 0.5–1.0 mg/kg q12–24h IV or orally) is often used if gastritis is suspected.

Because cats with HL are in metabolic liver failure, appetite stimulants are inappropriate; diazepam, oxazepam, cyproheptidine, and mirtazepine should not be used and will not recover an affected cat. Occasionally, an appetite stimulant may help initiate feeding early in syndrome development.

Hepatic encephalopathy (HE) is managed with antibiotics and lactulose to limit ammonia production and NH_3 diffusion across the blood brain barrier.

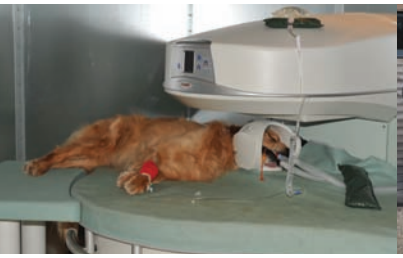
Anorectic cats are susceptible to depletion of B vitamins, especially thiamine (B1) and cobalamin (B12) and supplementations have been recommended on an empirical basis in cats with hepatic lipidosis. Cats with underlying intestinal disease are especially prone to cobalamin deficiency.

Low tissue glutathione concentration has been detected in some cats with hepatic lipidosis, suggesting a high risk for oxidant damage. For this reason, dietary supplementation with s-adenosylmethionine or vitamin E is recommended.

A number of metabolic supplements have improved recovery of affected cats: taurine (250–500 mg/cat/day), medical grade liquid oral l-carnitine (250–500 mg/cat/day), vitamin E (10 IU/kg/day), and potassium gluconate (if hypokalemia is persistent).

Prognosis for recovery in cats with hepatic lipidosis is reasonably good as long as feeding is rapidly and effectively instituted. Recurrence of HL is rare in recovered cats.

Hepatic lipidosis complicated by other disease (renal disease, IBD) has a more guarded prognosis and requires us to also manage the primary disease. Concurrent pancreatitis is a poor prognostic indicator.





Cases recently seen

Pyothorax caused by actinomyces, syringomyelia causing monoparesis in a yorkie cross, otitis media with intracranial extension and secondary hydrocephalus in a cat, SRMA in a bulldog, necrotising encephalitis in a pug, intestinal perforation from a jejunal mass in a young labrador, ruptured bladder following minor abdominal trauma in a labrador.

Types of referral seen

- Internal medicine
- Soft tissue surgery
- Endoscopy/laparoscopy
- Medical and surgical oncology
- Ophthalmology
- Rabbits, small mammals and exotics
- Neurology
- Cardiology
- Orthopaedic and fracture repair
- Onsite MRI/CT scanning
- Hydro/physiotherapy

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

Organising a referral is simple

Just phone Rosemary Lodge Veterinary Hospital on **01225 832521** and book in with one of our receptionists.

One of our clinicians will be very happy to discuss the case details prior to arranging the referral. Once you have made contact we will normally ask to speak directly to the pet's owner to swiftly arrange an appointment that fits in to their timetable. We do ask you email, fax or post us any relevant history with a supporting referral letter.

We will always do our best to fit in any emergency cases immediately and see them on the day you call us.

Now Available: Free Film Reading

Post your X-Rays to Rosemary Lodge or email them to **contact@bathvetreferrals.co.uk** to receive a free verbal report from one of our clinicians

Our clinicians

Alex Gough MA VetMB CertSAM CertVC PGCert (Neuroimaging) MRCVS - Head of Internal Medicine

Alasdair Hotston Moore MA VetMB CertSAC CertVR CertSAS CertMEd MRCVS - Head of Surgery

Jon Shippam BVSc CertSAS MRCVS - Orthopedic Surgeon

Jenny Lambert BVM&S CertVOphthal MRCVS - Ophthalmology

Lisa Gardbaum BVetMed CertSAM MRCVS - Internal Medicine

Robert E Matus DVM MS Diplomate ACVIM (Oncology and SAIM) MRCVS - Oncology and Internal Medicine

Rhiannon Strickland BVetMed MRCVS - Assistant Surgeon

Samantha Lane BVSc PGCertSAS MRCVS - Surgeon

Federica Manna DVM MRCVS - Assistant to Internal Medicine

J. Andrew Jagoe MVB PhD CertSAM MRCVS - Internal Medicine