

Feline Hepatic Lipidosis

- by Mark Robson

Not long ago an experienced veterinarian said to me "a yellow cat is a dead cat". Happily this is definitely not the case, but he was almost certainly scarred by dealing with hepatic lipidosis (HL). If undetected, HL is almost always fatal as the treatment involves careful but sustained nutrition for weeks to months, and most cats with undiagnosed HL will be euthanased due to lack of response. Cats are unusually vulnerable to the accumulation of lipid in hepatocytes. HL is usually a syndrome of multifactorial origin, but the end result is that triglyceride accumulates in >50-80% of hepatocytes. This leads to liver failure and the myriad systemic effects that such failure always precipitates. A principle source of the lipid is thought to be adipose stores, which are mobilised in response to even short periods of anorexia. Hepatocyte uptake of the mobilised fatty acids exceeds the ability of the liver to convert them into triglyceride then re-excrete it as lipoprotein and the cells "fill up" with fat.

Dr Sharon Center, a respected worker in this field, states that "the term idiopathic HL is obsolete". This strong statement results from her experience that over 80% of cats with HL can be proved to have one or more underlying diseases. These precipitate metabolic changes that result in lipid accumulation in hepatocytes. As in most idiopathic syndromes, the more thorough the diagnostic evaluation the less likely that the condition will remain idiopathic!

Some authors use the terms primary and secondary HL. Primary HL refers to a "typical" obese cat going off food for an unknown reason with no detectable underlying disease. Within days they go into the full-blown syndrome featuring icterus, anorexia, depression etc. Secondary HL describes those cats with an underlying disease, e.g. cholangiohepatitis, neoplasia and infection who then develop lipid accumulation that progresses to full blown HL. They may well be recovering from the primary disease while slipping into liver failure from HL. Diagnosis is best achieved using history, blood work and ultrasound, preferably with guided aspiration of the liver. Consideration of clotting ability is needed. Biopsy can be used, with impression smears taken from samples to speed interpretation, but an increased (though still small) risk of bleeding is incurred. Attention must be paid to electrolyte disorders and the diagnosis and treatment of underlying conditions. Nutritional support is the core treatment for HL. Force-feeding is rarely sufficient or desirable. Many of these cats are too ill for invasive procedures under anaesthetic, so careful nasogastric tube feeding along with IV fluid and electrolyte support may be needed for a few days before placing a more permanent feeding tube. Most internists prefer a percutaneous endoscopically placed gastrostomy (PEG) tube but an oesophagostomy or pharyngostomy tube can also be used.

Long term feeding at home may be needed. My "record" patient was fed at home via PEG tube for 8 months before suddenly recovering her appetite and ripping the tube out, all in the same day! Physical exams and monitoring of liver enzymes and bilirubin will help to plot progress. Space precludes discussion of supplementation with Vitamin K, B12 and carnitine, and the interested reader is referred to the articles listed below: • Hepatic Lipidosis In Cats - Sharon Center - Proceedings of Western Veterinary Conference 2002

- Common Feline Liver Diseases - Keith Richter - Proceedings of Western Veterinary Conference 2004

Both articles above are available online at www.vin.com to members. A free one-month trial membership is available to all veterinarians. Gypsy Collard, our feline HL example was presented after a week of illness centred around her liver. I believe that she was suffering from cholangiohepatitis that then progressed to full blown HL. Her owner was extremely motivated and conscientious and allowed us to treat her comprehensively. PEG tube placement was essential to get food into her in a reliable and stress free way until her liver recovered. She is now well in all respects after looking to be "on death's door" for quite a few days.

Hepatic lipidosis is a major differential diagnosis in all cats with evidence of liver disease, and prompt aggressive treatment is needed to give a good chance of recovery. Recovery rates varying from 30-90% appear in the literature, with the higher figure or better being the norm in our practice.

