Proceedings of the 36th World Small Animal Veterinary Congress
WSAVA

Oct. 14 - 17, 2011
Jeju, Korea

Next Congress:

Reprinted in IVIS with the permission of WSAVA
http://www.ivis.org
FELINE HEPATIC LIPIDOSIS

P. Jane Armstrong, DVM, MS, MBA, Diplomate ACVIM (SAIM)

University of Minnesota, St. Paul, MN, USA

Hepatic lipidosis (HL) is a cholestatic syndrome that develops in cats in association with profound and protracted anorexia. It is the most common form of liver disease in cats in North America [1] and is seen in cats in other parts of the world, although anecdotally not as commonly in some regions [2]. Obese or overweight cats are at increased risk. Most affected cats are middle-aged adults (median age 7 years), overlapping the peak prevalence of obesity, but the condition has been reported in cats from 0.5-20 years [2,3]. There is no gender or breed bias.

Hepatic lipidosis can occur secondary to any disease process that results in marked decrease in appetite. The period of anorexia may be as short as 2-7 days [3,4]. Common co-morbidities are other hepatic disorders, small intestinal diseases, pancreatitis, neoplasia, kidney disease, and diabetes mellitus [3,4]. Hepatic lipidosis can occur in an otherwise healthy cat that voluntarily (new food refusal, for example) or involuntarily (such as accidental confinement away from food or overzealous caloric restriction for weight reduction) has severely restricted food intake.

In the absence of signs associated with an underlying disease, severe hypokalemia or hepatic encephalopathy, HL cats are commonly bright and alert despite profound anorexia, recent rapid weight loss and marked jaundice. Other typical clinical findings are non-painful hepatomegaly and hypechogenicity of the liver. Sonographic changes are not diagnostic [5]. Characteristically, ample falciform fat is retained. Hypokalemia can result in marked muscle weakness and coagulation abnormalities in easy bruising (venipuncture, cystocentesis or ultrasound probe site). The most consistent laboratory findings are poikilocytosis, Heinz bodies +/- mild anemia, hyperbilirubinemia, hypoalbuminemia, and increases in serum activity of ALP [3]. ALT activity is less consistently increased than ALP. Whereas increases in GGT tend to parallel increases in ALP in other forms of liver disease, a GGT activity within the reference range is common in HL [6]. Diagnosis is usually confirmed by fine needle aspiration cytology. Liver biopsy, while occasionally needed to confirm or exclude the presence of concurrent liver disease, is best avoided due to the risk of
hemorrhage from a friable, fatty liver [4,7].

**Therapy**

Successful recovery of cats with HL initially requires correction of fluid and electrolyte abnormalities but the cornerstone of therapy is enteral nutritional support concentrating on meeting protein and caloric needs. This is best achieved by inserting a nasoesophageal (NE) tube on the day of admission. Depending on patient tolerance and clinical progress, this may be replaced a few days later by either an esophagostomy or gastrostomy tube. An important component of treatment is recognition and concurrent management of any underlying process initially promoting the onset of HL.

**Fluid and electrolyte therapy**

Immediate attention must be given to rehydration therapy and correcting electrolyte imbalances primarily resulting from vomiting and lack of intake. Hypokalemia and hypophosphatemia are important causes of morbidity. Hypokalemia may persist in the face of appropriate supplementation if there is concurrent hypomagnesemia. Magnesium is present in enteral diets in quantities sufficient to normalize serum levels. This is the preferred route of supplementation. Fluid and enteral nutritional therapy should be accompanied by once or twice daily monitoring of serum electrolytes, especially potassium and phosphorus, for the first 3 days, as these may drop precipitously in re-feeding syndrome [2].

Fluid supplementation with dextrose is contraindicated as cats with HL are intolerant to glucose and such supplementation may exacerbate hyperglycemia [8]. Impaired lactate metabolism is suspected in some cats with HL and is the reason that some authors advise against using lactate-containing fluids, such as Ringer’s [4]. Although a theoretical concern, lactated Ringer’s solution is routinely used with success.

**Enteral feeding**

Enteral feeding must be initiated as early as possible in the course of HL and sustained until voluntary intake resumes. The most useful methods are feeding via NE, esophagostomy (E-tube) or gastrostomy tube (G-tube). Force-feeding is of limited benefit and should only be attempted for a short time in cats that appear mildly affected. Appetite stimulants are not recommended.

**Method** - An NE tube is inexpensive, does not require anesthesia for placement and uses readily available supplies. However, these tubes require absolute verification of correct placement before feeding and close observation of the cat (+/- use of an Elizabethan collar) to prevent premature removal. Feeding through an NE tube necessitates feeding a liquid diet. Placement of either an E- or a G-tube requires a short anesthesia, which is best delayed until NE feeding has been underway for several days, but either tube type allows the use of a blended solid (canned) food and is sufficiently durable for reliable use in a home environment. Propofol can be safely used in HL cats as an anesthetic agent, such as for placement of a feeding tube [9]. Liquid oral medications may also be administered through any type of feeding tube.

**Diet selection** - Dietary protein is the nutrient that is most efficient at reducing hepatic lipid accumulation in cats in negative energy balance [10]. Protein restriction is contraindicated unless needed in the <5% of cases with hepatic encephalopathy (HE) [3]. Carbohydrates are less well tolerated than lipids as a source of calories.
Diets that are too high in carbohydrates may cause diarrhea, abdominal cramping, borborygmus, and hyperglycemia [2,11].

The diet selected to feed a cat with HL should be rich in protein (30 to 40% of metabolizable energy), moderate in lipids (about 50%) and relatively poor in carbohydrate (<20%) [2]. Suitable commercial liquid diets are Clinicare, EnteralCare™ HLP and FORTOL C+. Recovery formula commercial canned diets are suitable for feeding through E- or G-tubes and have high caloric density, which aids in combating volume intolerance.

**Refeeding plan** - Nutritional support should aim to deliver 50-60 kcal/kg of body weight/day in most cats. Calculate RER for overweight using estimated optimal weight to prevent overfeeding. The feeding schedule is determined by the patient’s volume tolerance and the logistics of feeding. Gastric volume in a cat with HL may be dramatically reduced to as little as 10% of its original volume [2]. To minimize vomiting, use a continuous rate infusion or provide small meals with an interval of about 3 hours between meals for the first few days. Decrease to 3-4 meals per day as volume tolerance improves. Each meal must be followed by a low volume water flush. It is often necessary to feed approximately 20% of resting energy requirement (RER) on day 1 (in divided feedings) and then increase the amount by 10-20% every 24 hours until full feeding (RER) is reaching. Fortunately, even cats that require more time to become volume-tolerant can be expected to show clinical improvement in the first week of therapy. Feeding should be stopped if there is gulping or retching, the meal size reduced by 50% for 12 hours and then increased gradually. It is critical for recovery, however, to continue to feed some food even if vomiting occurs.

**Other therapeutic considerations**

**Antiemetic therapy** - Antiemetics often facilitate reintroduction of food. Vomiting can sometimes be reduced by minimizing handling of the cat at the time of feeding and immediately afterwards. Metoclopramide (CRI of 1-2 mg/kg/day or 0.2-0.5 mg/kg q8h SC 30 minutes before feeding) is often a first choice drug because of availability, low cost and prokinetic effects. It is a weak antiemetic in cats, however, and better control of emesis may be obtained by use of maropitant (1 mg/kg IV or SQ q24h) [12], dolasetron (0.5 mg/kg q24h IV or SQ) or ondansetron (0.1-0.3 mg/kg q8-12h IV). An H2 receptor antagonist is often used to protect the lower esophagus from acid damage and to help alleviate possible gastritis.

**Cobalamin therapy** - Evaluation of plasma B12 (cobalamin) concentrations in cats with lipidosis revealed that 40% had subnormal values [13]. Cobalamin deficiency may be severe enough to produce signs such as neck ventroflexion, and anisocoria. The route of choice for supplementation is subcutaneous injection (250 µg/injection once weekly initially for six weeks).

**Treatment of coagulation disorders** - Vitamin K deficiency is frequently suspected in cats with HL [3,4,14]. Response to vitamin K suggests that prolongation of coagulation tests is more often the result of impaired vitamin K absorption than decreased factor production [3]. This makes parenteral, rather than oral, supplementation important. If coagulation abnormalities are suspected (or possibly in all cases [4]), administer 3 doses of vitamin K1 (0.5-1.5 mg/kg SQ or IM at 12-hour intervals using a 25 g needle). This treatment is particularly important if a biopsy is to be obtained.
Other nutrients - Supplementation with other nutrients has been suggested by some authors but beyond meeting nutritional requirements, benefits to supplement use are poorly documented in the cat. To date, no prospective clinical trials have been conducted to evaluate specific nutrients or supplements in cats with spontaneous HL. Prescribing multiple supplements and medications risks decreasing client compliance with feeding instructions.

Some clinicians provide L-Carnitine to HL cats (250 mg PO/day) to promote fatty acid oxidation and retention of lean body mass [3,4], but evidence is lacking that it provides any benefit in recovery from HL, and a much lower dose (7-14 mg/kg) was used for a protective effect in experimental weight loss studies [15]. Foods for cats with HL should provide L-carnitine at least 0.02 DM (dry matter).

Low hepatic glutathione concentrations in the liver of cats with HL compared to healthy feline liver is consistent with reduction in tissue antioxidant availability and provides the rationale for some authors to recommend SAMe (40 mg/kg PO q24 hours) for treating HL cats [4, 5].

Prognosis
Cats making a successful clinical recovery from HL demonstrate a gradual reduction in laboratory abnormalities over time. Expect the total bilirubin concentration to decline by > 50% within 7-10 days, even though serum liver enzyme activities may remain close to values documented at the time of case admission [3]. Two important factors affecting the outcome in HL are the presence of a serious, irreversible concurrent disease and how early enteral nutritional support is begun. Absent diagnosis of a fatal underlying condition, recovery rates of 80% or higher can be expected if enteral feeding is initiated early in the course of the disease and sustained until voluntary intake resumes. Cats may need tube feeding for several (3-6) weeks, requiring that the owner is an active participant in their cat’s recovery. Once a cat recovers from HL, recurrence is unlikely.

References


