



A TREATMENT PROTOCOL FOR IMPROVING SURVIVAL IN CATS WITH HEPATIC LIPIDOSIS

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*H*epatic lipidosis (HL) is the most common liver disease of cats. It is characterized by the accumulation of triglycerides within and around hepatocytes and severe intrahepatic cholestasis. It results in progressive liver failure that produces the clinical signs of anorexia, lethargy, vomiting, weight loss, and icterus.¹ The pathophysiology of hepatic lipidosis has been studied extensively. In other species, a lack of arginine or ornithine will disrupt the Krebs-Henseleit urea cycle resulting in a buildup of orotic acid. This knowledge serves as the basis for both understanding the pathophysiology and developing effective treatment protocols.² However, understanding the mechanism of feline hepatic lipidosis currently eludes veterinary researchers. Because of this, the formulation of specific treatment modalities has been greatly hindered.

It is well established that there are two predisposing factors for most cases of idiopathic lipidosis: obesity and anorexia.¹ Regardless of the cause of anorexia, an anorectic obese cat is likely to develop hepatic lipidosis. The process may begin after only a few days of anorexia, but it usually does not become clinically significant until 2 weeks or more.

The backbone of successful therapy of hepatic lipidosis is the combination of food and persistence. Although we do not know precisely which nutrients are essential for recovery, we know that a balanced feline diet fed in sufficient quantity for a sufficient length of time will result in recovery in at least 50% of affect-

ed cats. Some cats recover within 2 to 3 weeks and others take 8 weeks or more; the average recovery time is about 6 weeks regardless of the specific diet fed.² Therefore successful therapy requires appropriate nutrition and a food delivery system that will permit feeding an anorectic cat for several weeks.

MATERIALS AND METHODS

I have been treating hepatic lipidosis for several years in a private practice setting. The purpose of this study was to determine the survival rates of cats with hepatic lipidosis using my two-step treatment plan and to contrast that with the commonly reported survival rates currently found in the veterinary literature. To do so, records of 16 cases of cats with confirmed hepatic lipidosis were reviewed. Table 1 is a summary of these cases, presenting the method of diagnosis, treatment protocol, and time until recovery or death.

RESULTS

Clinical Signs

All of the cats in this study had a history of anorexia of variable length ranging from 1 week to 1 month. Many (10/16; 63%) were icteric, and some (7/16; 44%) had a history of vomiting. Lethargy and weight loss were common.

Diagnosis

The tentative diagnosis of hepatic lipidosis for the cats in this study was based on history, clinical signs, and serum biochemical values. Of particular merit was the relative elevation of the serum alkaline phosphatase (SAP) and the γ -glutamyltransferase (GGT). It has been established that most cats with hepatic lipidosis will have an elevation of the SAP that is equal to or greater than that of the GGT.³ In many cats the SAP will be substantially elevated, and the GGT will be normal. For example, the cat in Case 16 had a 21-fold increase in its SAP and a normal GGT. Five of the cases in the study have no reported GGT value because these cases predated the inclusion of that test in the chemistry profile offered by the veterinary reference laboratory.

Radiographs and ultrasound studies were used inconsistently because of cost constraints with some clients and because neither is specific, sensitive, nor confirmatory for hepatic lipidosis. Although many cats with hepatic lipidosis have an enlarged liver radiographically, others have a normal-sized liver. Although fatty livers are typically hyperechoic, livers affected by cirrhosis or lymphosarcoma may have the same echogenic pattern.

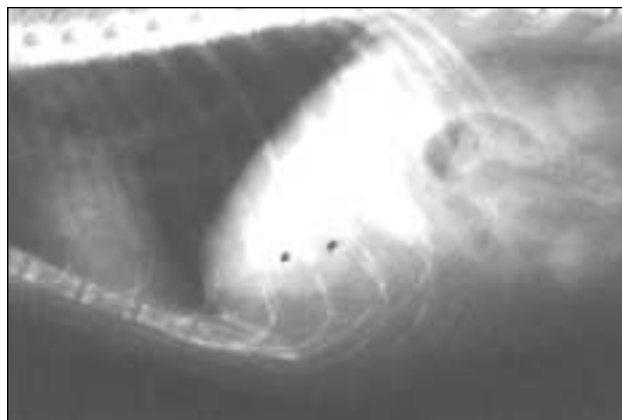


Figure 1. Landmarks for liver aspiration. The dots on this radiograph are in the eighth and ninth intercostal spaces at the costochondral junction. These are the locations that are usually appropriate for needle insertion using my liver aspiration technique. If the aspiration procedure is not successful at these locations, radiographs or ultrasound should be used to determine the proper location for needle insertion.

Confirmation of hepatic lipidosis in all of the cats in this study was based on hepatic cytology collected by fine-needle aspiration. Samples were collected via a percutaneous needle insertion made through the eighth or ninth intercostal space at approximately the costochondral junction (Figure 1; see also the box on p. 10). Aspirations were performed without local anesthesia or sedation. The samples were sprayed on a microscope slide, stained with Dip Quik™ (Jorgensen Laboratories) stain, and examined under 100x and 1000x magnification (Figure 2).

Treatment

Treatment consists of two distinct phases. The initial treatment phase occurs from the time of admission to the hospital until the cat receives a surgically implanted feeding tube. The long-term management phase begins at the time of feeding tube placement and continues until recovery or death.

Initial Treatment Phase

Cats in the initial treatment phase were usually critically ill. These cats were in a severe catabolic state as a result of starvation (often prolonged) and weight loss. Recognition of this fact was the basis for the treatment given and withheld.

If the cat was dehydrated, an intravenous catheter was placed and a balanced electrolyte fluid (lactated Ringer's solution) administered. Supplemental potassium chloride was added at the rate of 40 to 60 mEq per liter of fluids. In many cases, a nondehydrated cat was given subcutaneously administered fluids that were supplemented with about 30 mEq/L of potassi-

TABLE 1
Summary of Data from Cats in this Study

Cat Number	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Age (years)	4	5	8	13	12	11	6	12	9	6	11	8	9	14	2	4
Clinical signs	A, L	A, WL	A	A	A, V	A, WL, V	A, V	A, WL, V	A	A, L, WL	A, WL, V	A	PA, WL	A, WL, L	A, V	A, WL
Length of anorexia									1 month	3 weeks		1 week				1 week
Icterus	Y	Y	Y			Y	Y			Y			Y	Y	Y	Y
Liver aspiration	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL
Alkaline phosphorus (U/L)	509	226	559	145	361	1386	1409	708	341	804	169	125	488	1242	270	1658
Fold increase	6.3	2.8	7	1.8	4.5	17.3	17.6	8.6	4.3	10	2.1	1.6	6.1	15.5	3.4	21
GGT (U/L)	3	NA	NA	NA	NA	NA	1	0	4	5	1	1	10	5	4	7
Fold increase	0	NA	NA	NA	NA	NA	0	0	0	0	0	0	1.5	0	0	0
Radiograph (liver)				NS						Lg			NS	NS		
Ultrasound (liver)			HE, Lg		HE											Sli HE
Initial treatment																
OG tube	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
IV fluids								Y		Y		Y		Y		
SC fluids	Y	Y	Y	Y	Y	Y	Y	Y	Y		Y	Y	Y	Y	Y	Y
Antibiotics	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Vitamin B/K	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Antiemetic					Y	Y	Y	Y		Y	Y					Y
Esophagostomy tube placed	Day 4	Day 4	Day 5	Day 4	Day 9	Day 9	Day 4	Day 4	Day 6		Day 7	Day 4	Day 6	Day 5	Day 5	Day 5
Esophagostomy tube removed	Day 16	Day 43	Day 36	Day 57	Day 37	Day 30	Day 15	Day 31	Day 178			Day 49	Day 58		Day 16	Day 26
Form of HL	I	I	I	I	I	I	S	I	I	I	S	I	I	S	I	I
Outcome	Rec	Rec	Rec	Rec	Rec	Rec	Euth	Rec	Rec	Died	Euth	Rec	Rec	Euth	Rec	Rec
Comment							Pan			on Day 2	Day 26			Day 64		
Comment											HCM			BC		

A = anorexia; L = lethargy; WL = weight loss; V = vomiting; PA = Partial anorexia; wk = week; Y = yes; HL = hepatic lipidosis; NA = not applicable (usually not performed); NS = normal size; Lg = large in size; HE = hyperechoic; Sli = slight; OG tube = orogastric tube feeding; I = idiopathic; S = secondary; Rec = recovered; Euth = euthanized; Pan = pancreatitis; HCM = hypertrophic cardiomyopathy; BC = biliary carcinoma.

PROCEDURE FOR LIVER ASPIRATION

Position the cat standing or in ventral recumbency
To minimize liver trauma, use a 25 (or 22) gauge needle, 5/8 (or 1 inch) long, with a 3 cc syringe
Generally, no sedation or local anesthetic is needed.
However, mild sedation using ketamine (2 mg/kg IV) can be used if the cat is resistant to the procedure. It is important not to lacerate the liver
Prep the skin in the appropriate area
The location is determined by two radiographic views, or the needle is inserted between ribs 8 and 9 or 9 and 10 at the costochondral junction. The latter is about one third the distance from the sternum to the dorsal processes of the thoracic vertebrae (see Figure 1)
Insert the needle, aspirate rapidly, release the suction on the syringe, and withdraw in one motion
Spray the aspirate on a microscope slide and make a smear
Grossly, a liver aspirate looks like a small drop of blood
Repeat the procedure on the other side
Look for clusters of hepatocytes to confirm that the liver has been aspirated

um chloride. Injectable B vitamins, especially thiamine, and vitamin K1 were given because of presumed depletion associated with prolonged anorexia and because many cats with hepatic lipidosis have abnormal clotting profiles.¹ Amoxicillin (10 mg/kg SC bid) and enrofloxacin (2 mg/kg SC or PO bid) were given because of the recognition that some of these cats were likely to have had concurrent suppurative cholangiohepatitis or other underlying bacterial infections.

Orogastric tube feeding using a 16 Fr feeding tube (Robinson/Nélaton Catheter, #220500-160, Rusch) commenced on either Day 1 or Day 2 of treatment. Day 1 was the day of admission to the hospital. The goal of therapy was to feed calories at the rate of 60 to 80 kcal/kg/day and protein at the rate of approximately 3.5 g/kg/day. To meet these nutritional goals, a 4 kg cat should receive about 180 to 200 ml per 24 hours of the food mixture in Table 2. In most instances 25 to 40 ml of a warm, blenderized diet was fed via an orogastric tube once or twice on Day 1. Although this was a suboptimal level, full feeding at this point usually results in vomiting and diarrhea because of stomach contracture and villus atrophy. Feeding continued each day, and the amount was

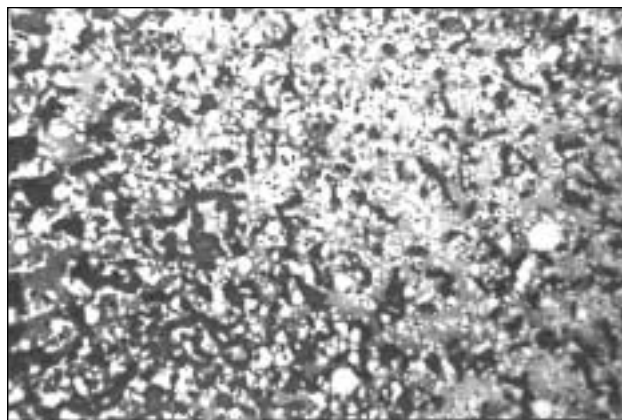


Figure 2A

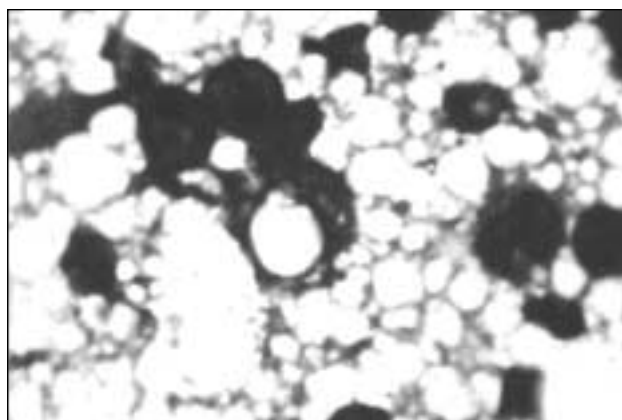


Figure 2B

Figure 2. Photomicrograph of hepatic lipidosis aspirates. **(A)** Low power (100 \times) of liver aspirate. Note the scattered hepatocytes and copious amount of fat. **(B)** High power (1000 \times) of liver aspirate. The hepatocyte in the center of the field is laden with intracellular fat and is surrounded by extracellular fat.

gradually increased until most cats were receiving 75 to 100 ml twice daily by Day 4 or 5. The diet should be as follows:

- A commercial feline diet to provide the balance and nutrients (i.e., taurine, etc.) needed by the cat.
- Protein levels sufficiently high so that adequate protein would be available to form the lipoprotein complexes necessary to remove excess triglycerides from the liver.
- An adequate caloric density (1.6 kcal/ml) to supply 60 kcal/kg/day (i.e., about 300 kcal for a 5 kg cat) in a volume that would be practical for hospital and home feeding.

Vomiting was part of the history of 7/16 (44%) of the cats in this study. Vomiting is generally considered a negative prognostic sign because it often rep-

DIET USED FOR FEEDING CATS WITH HEPATIC LIPIDOSIS

Ingredients

3 cans (5.5 oz each) of commercial wet cat food
8 oz tap water
2 oz vegetable oil
16 mEq potassium gluconate

Nutrient Composition

<i>Ingredient</i>	<i>As Fed (%)</i>	<i>Dry Matter (%)</i>
Moisture	72.60	0.00
Protein	7.80	28.47
Fat	13.78	50.29
Fiber	0.36	1.31
Ash	1.50	5.47
Potassium	0.32	1.17
Carbohydrates	3.96	14.45
Taurine	0.08	0.31
Phosphorus	0.25	0.91

Preparation

Put all of the ingredients in a blender and run at the fastest cycle
Pour the food through a kitchen strainer to prevent plugging of the 12 Fr esophagostomy tube
Store the food in a refrigerator in a microwave-safe container

Feeding Instructions

Heat the food in a microwave to approximately body temperature
Infuse the food slowly using 12 cc syringes. The small syringes prevent overfilling of the esophagus, regurgitation, and aspiration of food. Elevating the cat's front legs during feeding ensures flow into the stomach
Flush the tube with 3 to 5 ml of water to prevent food drying within the tube and causing tube obstruction
Feed three to six small meals each day
Feed a 4 kg cat 180 to 200 ml per 24 hours
If an antiemetic is needed, flush it through the esophagostomy tube at least 20 minutes before feeding

resents advanced liver disease or concurrent pancreatitis⁴ and because it prohibits proper feeding. Vomiting was treated either with metoclopramide or famotidine; the latter was generally more effective at a dose of 0.1 mg/kg bid. Because of the expense of the prescription suspension, three over-the-counter 10 mg Pepcid-AC™ tablets (Merck & Co.) were crushed



Figure 3A



Figure 3B

Figure 3. Esophagostomy tube placement. **(A)** The esophagostomy tube is inserted percutaneously into the esophagus in the left cervical region. A tape butterfly is used to anchor the tube to the skin at the ostomy site. **(B)** The tube is routed to the dorsal midline and anchored with tape. Further taping covers all except the tip of the tube. The tube is capped when not in use.

and placed in a palatable liquid vitamin. This created a suspension with a concentration of 1 mg/ml. It was administered at 0.4 ml bid for a 4 kg cat, and the cat was fed at least 20 minutes after administration.

The cats were deemed clinically stable when they were no longer dehydrated, their general demeanor was improved, and they were retaining at least 90% of the tube-administered food. This occurred as early as Day 4 and as late as Day 9. At that time, they were anesthetized with a combination of ketamine and diazepam. These drugs were mixed in the same syringe and given intravenously to effect at approximate doses of 1 mg/kg and 0.5 mg/kg, respectively. An esophagostomy tube was placed according to published protocol⁵ (Figure 3; see also the box on p. 12). If the surgery was performed in the morning, these cats received their first feeding that evening.

ESOPHAGOSTOMY TUBE PLACEMENT

Equipment

12 Fr Sovereign Feeding Tube and Urethral Catheter
(Sherwood Medical)
5" curved hemostat
Scalpel
Needle holders
Thumb forceps
Soft nonabsorbable suture material
1" adhesive tape

Placement

Use a short-acting, intravenous general anesthetic
(i.e., ketamine + diazepam)
Pass the curved 5" hemostat into the esophagus in the
left cervical area. It should be dorsal to the jugular
vein
Cut down over the tip of the hemostat with the scalpel
Pull the tube through the incision into the esophagus
and out the mouth
Cut off the tip of the tube, then feed it down the esoph-
agus
Suture, tape, and bandage the tube to the head. A
tape butterfly is placed around the tube at the ostomy
site and sutured to the skin to prevent tube
movement. The tube should go under the chin and
behind the right ear. The tube should be premea-
sured so its distal end will be placed in the mid-
esophagus, not in the stomach

Many were discharged from the hospital on the day of esophagostomy tube placement. This concluded the initial treatment phase and signaled the beginning of the long-term management phase.

Long-Term Management Phase

The long-term management phase began with placement of the esophagostomy tubes. The purpose of the feeding tubes was to permit the cats to return home in a state that allowed the owners to perform proper feeding. The esophagostomy tubes were found to be comfortable for the cats and permitted one-person feeding. The owners were instructed to feed the prescribed amount of food in three to six feedings per day. It was requested that the cats be returned every 14 days for a recheck. The purposes of the rechecks were to examine and repair, if needed, the tape securing the tube to the cat and to examine and weigh the cat. In addition, the owners were queried about their success in the feeding process and about any interest their cats had in eating.

The owners were told to return the cats for esophagostomy tube removal after the cats had been eating well for at least 3 days. Some cats ate for 1 or 2 days and then did not do so again for several days. After 3 days of eating, however, it was safe to remove the feeding tubes because the cats' appetites remained normal.

Of the cats that recovered, the esophagostomy tube remained in place for an average of 40 days for all cases and 29 days if Case 9 is excluded. The owner of the cat in Case 9 was advised to allow tube removal on this cat on Day 90 even though it was not eating. She refused this advice until Day 178. Within 36 hours of tube removal, the cat began eating. It is presumed that the cat's refusal to eat was psychological. Of the cats that recovered, the overall time from hospital admission until recovery was 36 days, if Case 9 is excluded.

Survival Rates

Of the 16 cases in the study, 12 recovered for a survival rate of 75%. Three of the four that did not survive had severe underlying disease and secondary hepatic lipidosis. Thirteen cases were diagnosed as idiopathic hepatic lipidosis; 12 of these recovered for a survival rate of 92%.

Appetite Stimulants

Appetite stimulants were not administered. These drugs produce inconsistent results and often only have effects for a few days. In addition, food intake that occurs is usually not sufficient to meet the cat's caloric requirements. Diazepam, a commonly used appetite stimulant, is contraindicated for repeated use in cats with hepatic failure because it is metabolized by the liver.¹ (Its use as an intravenous anesthetic for esophagostomy tube placement occurred once per cat at the end of the initial treatment phase; all of the cats in this study recovered quickly from it.)

DISCUSSION

The purpose of this study was to determine the survival rates of cats with hepatic lipidosis when a two-step treatment plan was used and to contrast that with the commonly reported survival rates currently found in the veterinary literature. Of the 16 cases in the study, 12 recovered for an overall survival rate of 75%. This is considerably higher than the commonly reported recovery rates of 50% to 65%.^{2,3} Of the 16 cases, 13 (81%) had either idiopathic hepatic lipidosis or hepatic lipidosis resulting from a primary disease that responded to the treatment ren-



Figure 4. Gross view of primary liver cancer as a cause of hepatic lipidosis. This is a surgical view of the liver of the cat in Case 14. Note the presence of multiple masses. The histopathologic diagnosis was biliary carcinoma.

dered. Of these 13 cases, 12 recovered for a survival rate of 92%. The cats with hepatic lipidosis secondary to an identified primary disease (Cases 7, 11, 14) all died because the primary disease was fatal. The cat with idiopathic hepatic lipidosis that died did so on Day 2 of hospitalization. It was very dehydrated and hypothermic on presentation.

Case 14 is notable. This cat was presented for weight loss, anorexia, and icterus. The biochemical profile was typical for hepatic lipidosis (SAP = 1242; GGT = 5), and a liver aspirate confirmed the disease. The cat was treated in the hospital with the normal protocol for feline hepatic lipidosis. An esophagostomy tube was placed on Day 5, and the cat was discharged on Day 6. On the recheck at 2 weeks post-discharge, the cat was feeling better but its weight was not increasing as was typical of the other cats in the study. The cat was receiving an average of 160 ml of food each day; vomiting was occurring at least four times per week. On Day 64 the cat was returned for a scheduled recheck. At that time it had lost weight, and the liver had enlarged compared with other examinations. A biochemical profile was performed which revealed normal SAP and GGT, 78 and 0, respectively. The next day a laparotomy was performed, and the liver was found to be grossly infiltrated with multiple masses (Figure 4). The cat was euthanized. Histopathology revealed biliary carcinoma with no evidence of hepatic lipidosis.

The composition of the diet is included in Table 2. I do not claim to be a nutritionist and do not intend to infer that this is the only formula that is successful in feeding cats with hepatic lipidosis. Indeed, there is no consensus in the literature on what the proper diet is. It is notable, however, that this very high success rate

is achieved without the addition of carnitine, arginine, citrulline, or taurine, which are micronutrients thought by some to be important in recovery from this disease. This may mean that these are not as important as thought. On the other hand, these cats might have recovered faster if these nutrients had been added. Future controlled studies are needed to better understand the appropriateness of this diet.

The high-fat diet used may alarm some because the liver is already infiltrated with triglycerides. One study, however, of three cats with hepatic lipidosis that all recovered concluded that the high-fat diet used likely indicates that high-fat diets may be beneficial to cats with hepatic lipidosis.⁶ Consistent with that conclusion are current recommendations for cats with liver disease⁷:

- Caloric requirements should be met with carbohydrates and fat to minimize protein catabolism for energy. Energy-depleted cats catabolize protein (i.e., muscle) quickly. This accounts for the weight loss commonly seen in cats with hepatic lipidosis.
- The glucose intolerance observed in some cats with liver disease can be avoided by decreasing the carbohydrate content and increasing the fat content.

Case 9 is notable because of the length of time the cat depended on esophagostomy tube feeding. This case was referred to me for esophagostomy tube placement and returned to the referring veterinarian for completion of the case. After 3 months the referring veterinarian told me that the tube was still in place even though the cat appeared normal except for anorexia. The former was instructed to perform more diagnostics or consider tube removal because this was an unusually long period. The owner declined diagnostics and tube removal. After 6 months of esophagostomy tube feeding, the owner consented to tube removal. Within 36 hours the cat began eating normally. I have placed over 50 esophagostomy tubes for hepatic lipidosis and other conditions. This is the second case in which a cat appeared content to be fed through the esophagostomy tube. Both of these cats would come to the kitchen when they heard the microwave running and waited patiently for feeding. In both instances, normal eating returned after the tube was removed.

Some clinicians prefer other feeding tubes. The nasoesophageal tube is commonly used for initial feeding, and the gastrostomy tube is the preference of some for long-term feeding. I find it difficult and time-consuming to administer the correct amount of

nutrients through a nasoesophageal tube. Orogastric tube feeding has been neglected in the training program of many veterinary colleges, and new graduates are often unfamiliar with its use. If the proper equipment and techniques are used, it exerts only minimal stress on the cat and permits delivery of adequate quantities of food in just a few seconds. I have had very good success with the esophagostomy tube and thus have not used the gastrostomy tube. Placement of the gastrostomy tube has a comparatively long learning curve and, if performed improperly, can result in peritonitis and death.⁸ Surgeons adept at placing it, however, especially when using techniques that do not require gastrostomy, find it very satisfactory for long-term feeding of cats with hepatic lipidosis.

It has been my experience that the initial treatment phase is the time when most of these cats die; this observation led to development of this protocol. This is the time when they have very serious liver disease induced by anorexia and that produces anorexia. Anorexia of as few as 3 to 5 days' duration can produce immunoincompetence, depletion of energy stores, weakness, and organ failure.⁹ Prolonged anorexia creates a catabolic state that predisposes the cat to very serious consequences, including death, if further stress is induced. It is common practice to make a tentative diagnosis of hepatic lipidosis based on history, clinical signs, and serum biochemical values. These cats are then sent to surgery on Day 2 for a liver biopsy and placement of a gastrostomy tube. Death often follows a few hours or a day or two after surgery.

The approach discussed in this paper minimizes stress in the following ways:

- Medical management begins on Day 1. This includes small amounts of food administered by an orogastric tube. Lipoprotein complexes are necessary for mobilization of fat from the liver. Formation of lipoprotein complexes necessitates protein intake to prevent further protein catabolism. Although the amount of protein given on Days 1 to 3 is usually suboptimal, any protein intake is better than none.
- Confirmation of hepatic lipidosis is made by a liver aspirate. In a sick cat, this procedure does not require sedation or general anesthesia and is minimally invasive.
- Medical management, including orogastric tube feeding, continues until the cat is clinically improved and stable.

- When the cat is improved and stable, an esophagostomy tube is placed. At this point, the cat is significantly stronger than on Day 1 or 2. The esophagostomy tube requires short, intravenous anesthesia, no special equipment, and no major surgery.

There is one significant disadvantage to this approach as demonstrated by Case 14. A liver aspirate samples only a small number of cells compared with a core needle biopsy or a wedge biopsy. Liver architecture cannot be determined, which means that underlying liver disease may go undetected. Even though the cat in Case 14 improved clinically for about 6 weeks and its hepatic lipidosis resolved within 4 weeks, its neoplastic disease progressed and proved fatal. Suppurative cholangiohepatitis is a common underlying disease in cats with hepatic lipidosis. It is not reliably diagnosed with a fine-needle aspirate. My approach of using a combination of amoxicillin and enrofloxacin is an attempt to treat this disease even though it is not diagnosed. These drugs, especially amoxicillin, are usually effective in this disease.

Although some feel that a liver biopsy is necessary to properly diagnose hepatic lipidosis and to detect underlying disease, the outcome of this study indicates that the significantly increased survival rate justifies this omission. However, if clinical improvement does not occur within the first 1 to 2 weeks of treatment, a liver biopsy is indicated and encouraged.

CONCLUSION

Over 90% of cats with idiopathic hepatic lipidosis can be expected to survive if they are treated with the two-step protocol described. This protocol recognizes the need for minimal stress during the initial treatment phase. The cat is not anesthetized for diagnostic procedures, and anesthesia for feeding tube placement does not occur until the cat has been treated for several days and is stable. At that time an esophagostomy tube is placed, and the cat is discharged for treatment at home.

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