



**Veterinary Emergency
+ Referral Center**
of Hawaii

**Ettinger: Textbook of Veterinary
Internal Medicine, 7th Edition
Hepatic Lipidosis in Cats
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What is hepatic lipidosis?

Hepatic lipidosis, or fatty liver, is a disease process characterized by excess fat accumulation in the liver. This is the most common form of liver disease seen in cats in North America, yet it is far from being completely understood. This disease can occur in cats of any age or breed and may affect females slightly more than males. The common finding in almost all cats with this syndrome is that it occurs following 2 or more weeks of anorexia (not eating). This disease condition may occur regardless of the cause of the anorexia. When an additional disease state causes loss of appetite and precedes hepatic lipidosis, the hepatic lipidosis is defined as "secondary." The terms "primary" or "idiopathic" hepatic lipidosis are used when another disease state cannot be identified, as is the case in approximately 50% of cats so diagnosed. A state of obesity before the period of anorexia increases the risk of a cat developing clinical hepatic lipidosis.

Numerous causes of anorexia can result in this condition, some of which include predisposing diseases and behavioral or "stress-related" changes.

Some examples of possible contributions to the disease include the following:

- Diabetes mellitus
- Pancreatitis (inflammation of the pancreas)
- Cancer
- Other liver diseases
- Owners going away on vacation
- Family members leaving or being introduced to the household
- Spending time in a kennel
- Changes in the pet population in the home
- Diet changes

Unfortunately, once this disease develops, cats feel ill and will not eat even if the initial cause for their loss of appetite has been resolved. A vicious cycle develops because the longer the cat refrains from eating, the worse the hepatic lipidosis becomes, and vice versa. Without aggressive medical intervention this cycle will lead to death in as many as 90% of affected cats.

The exact mechanism by which fasting in a previously healthy cat causes hepatic lipidosis is not clear. The process is unique to cats in both severity and rate of occurrence. It is understood as the anorexia continues more and more fat is broken down throughout the body and that this fat is then transported to the liver. The liver should then process this fat and export it back to the rest of the body in a new form. In cats developing hepatic lipidosis this process is impaired and fat accumulates in the liver. Damage to the liver occurs as a result of liver cells being too swollen with fat.

What are the symptoms of hepatic lipidosis?

The symptoms most commonly associated with this syndrome are the following:

- Loss of appetite
- Weight loss
- Lethargy
- Jaundice (yellow tinge to the skin, inside of ears, and gums)

Owners occasionally notice behavioral or neurologic signs such as excessive drooling, blindness, severe obtundation, or seizures. These neurologic signs are caused by a condition called *hepatic encephalopathy*, or brain dysfunction as a result of liver disease. This condition is caused by toxins circulating to the brain that come from abnormally functioning liver or by bacteria in the gut that are normally detoxified in the liver. One important such toxin is ammonia. Ammonia is produced by bacteria in the gut from proteins eaten. This substance is detoxified in the normal liver. When the liver fails to function normally, ammonia builds up in the blood stream and causes the brain to dysfunction. If treated early, the changes in the brain may be reversible.

What tests are needed?

Your veterinarian confirmed the suspicion of liver disease by performing a physical examination and feeling an enlarged liver. Your cat's blood work revealed tell-tale changes.

Imaging techniques such as x-rays or ultrasound of the abdomen are helpful in demonstrating liver enlargement. The typical appearance of a fatty liver is usually evident on ultrasound. These changes, together with ruling out other disease states that could have triggered the lipidosis, help in making a diagnosis.

Definitive diagnosis of hepatic lipidosis requires visualization of fat globules in the liver cells from a sample under a microscope. The sample needed is from the liver itself. This liver piece, called a *biopsy*, can be obtained using several methods:

- **Surgically**, using laparoscopy (special tools and a camera inserted through small holes in the skin under general anesthesia)
- **Aspiration**, placing a hypodermic needle through the skin directly into the liver, usually under ultrasound guidance. When the syringe plunger is pulled back cells are aspirated into the needle. The needle and syringe are then removed from the pet and the cells are squirted out onto a glass slide.

The advantages of an aspirate are that general anesthesia is not usually required and there are fewer complications. The disadvantages of an aspirate are that it only allows visualization of a group of liver cells and not an actual piece of liver tissue, thus being less accurate than a biopsy.

What treatment is needed?

The treatment of hepatic lipidosis varies depending on severity of illness and existence of other diseases. Some possible treatments may include the following:

- Hospitalization
- Fluid therapy
- Supportive care
- Antibiotics
- Vitamin K (to prevent clotting problems)
- Specific treatment of hepatic encephalopathy

The cornerstone of therapy and the only way to reverse the process of fat accumulation in the liver is feeding to supply your cat with his/her full caloric requirement. A number of ways exist to attempt to achieve this goal but only a few are consistently successful. Trying different diets may induce a cat to eat in the initial phases of anorexia and is worth trying, but feeding will most likely not induce a cat to eat once clinical signs of hepatic lipidosis develop. Force-feeding is usually not a good idea. Even with the most cooperative cat it is virtually impossible to feed adequate amounts in this fashion. Cats also seem to rapidly develop aversions, and the association between food and the unpleasant forced experience might delay the return of normal eating habits. Appetite stimulate medications may also work in the initial phase of anorexia but are unlikely to consistently cause a cat to eat once symptoms develop.

When cats stop eating completely the only viable option to reverse the disease process is tube feeding. The use of long-term tube feedings has changed the outcome in this disease from more than 90% mortality to less than 30%. There are three commonly used types of feeding tubes in this disease. A tube placed through the nose into the stomach or esophagus can be used temporarily. Alternatively, a tube can be surgically inserted into the esophagus or a surgically or endoscopically placed tube through the body wall directly into the stomach are used for long-term feeding. Most cats require between 1 to 3 months of feeding through the tube. A commercially available, calorie-rich diet is used in most cats. Your veterinarian will provide you with the exact amount and feeding schedule. Cats with hepatic encephalopathy may require a protein-restricted diet initially. Your veterinarian may also include the addition of amino acids and vitamin supplements for the diet.

Frequent rechecks will be required with your veterinarian to assess the tube, your cat's health, and blood work. Liver parameters found in the blood usually improve within 2 to 8 weeks after initiating feeding. Oral food is usually not offered until the liver parameters normalize. At that point food can be offered. If the cat eats, one daily tube feeding can be discontinued. This schedule should be maintained for a week, after which an additional daily tube feeding can be dropped. This routine can be followed until the cat is completely off tube feedings. The tube should only be removed after the cat has been off tube feedings, eating the full caloric requirement and healthy in every other way for a duration of at least 2 weeks. If the cat refuses to eat when offered food initially or at any point in the process of decreasing the tube feedings then a return to full tube feedings is warranted and the cat should not be offered food again for a week. This is to avoid food aversions, which is critically important at this stage. When this regime is followed most cats' tubes can be removed within 3 months after placement. Occasionally cats will require tube feeding for longer periods, but even they have a good chance for full recovery. In cats with idiopathic hepatic lipidosis recurrence is rare and most cats that recover go on to live normal lives.