HEPATIC LIPIDOSIS
(ACCUMULATION OF FATS AND LIPIDS IN THE LIVER)

BASICS

OVERVIEW
- Disease in which fats and lipids (compounds that contain fats or oils) accumulate in the liver (condition known as "hepatic lipidosis")
- Possible complication of lack of appetite (known as "anorexia") in obese cats
- Feline hepatic lipidosis—more than 50% of liver cells (known as "hepatocytes") accumulate triglycerides, results in severe decrease or stoppage of the flow of bile (known as "cholestasis") and liver dysfunction in the cat
- Usually secondary to another underlying disease process or simply lack of food intake (such as a cat accidentally being locked in basement)
- The liver is the largest gland in the body; it has many functions, including production of bile (a fluid substance involved in digestion of fats); production of albumin (a protein in the plasma of the blood); and detoxification of drugs and other chemicals (such as ammonia) in the body
- Bile ducts begin within the liver itself as tiny channels to transport bile—the ducts join together to form larger bile ducts and finally enter the extrahepatic or common bile duct, which empties into the upper small intestine; the system of bile ducts is known as the "biliary tree"

SIGNALMENT/DESCRIPTION of ANIMAL

Species
- Cats primarily affected
- Dogs rarely affected (puppies, especially with storage disease as in the Maltese; "storage disease" is an inherited metabolic disease in which harmful levels of materials accumulate in the body's cells and tissues)

Breed Predilection
- None

Mean Age and Range
- Mean—8 years of age (range, 1 to 16 years of age)
- Primarily middle-aged adults

Predominant Sex
- Inconsistent susceptibility to develop hepatic lipidosis for obese females

SIGNS/OBSERVED CHANGES in the ANIMAL
- Lack of appetite (anorexia) and weight loss
- Yellowish discoloration to the gums and other tissues of the body (known as "jaundice" or "icterus")
- Sluggishness (lethargy)
- Weakness progressing to collapse
- Vomiting, diarrhea, or constipation
- Excessive salivation (known as "ptyalism"), may reflect hepatic encephalopathy (brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia) or food aversion
- Abnormal position of the head and neck, in which the chin is located near the chest (known as "neck ventroflexion")
- Enlargement of the liver (known as "hepatomegaly")
- Dehydration
- Abnormalities due to underlying diseases

CAUSES
- More than 85% cats with hepatic lipidosis have disorders causing lack of appetite (anorexia) or problems absorbing nutrients into the body following digestion (known as "malassimilation")
- Primary liver disease—portosystemic shunt (condition in which blood vessels allow blood to flow abnormally between the portal vein [vein that normally carries blood from the digestive organs to the liver] and the body circulation without first going through the liver); inflammation of the bile duct or biliary tree (known as "cholangitis") and inflammation of the bile ducts and liver (known as "cholangiohepatitis"); blockage of the extrahepatic or common bile duct (known as "extrahepatic bile duct obstruction"); presence of hard, solid material in the bile duct or gall bladder (known as "cholelithiasis"); cancer
- Gastrointestinal disease—blockage or obstruction of the gastrointestinal tract; cancer (such as lymphoma, a type of cancer that develops from lymphoid tissue, including lymphocytes, a type of white-blood cell formed in lymphatic tissues throughout the body); inflammatory bowel disease (IBD); inflammation of the pancreas (known as "pancreatitis")
- Urinary tract disease—long-term (chronic) inflammation of the tissue spaces in the kidneys (known as "chronic interstitial nephritis"); lower urinary tract infection; kidney failure
- Nervous system disorders

• Infectious diseases—toxoplasmosis; feline infectious peritonitis (FIP); feline immunodeficiency virus (FIV) or feline leukemia virus (FeLV)-related disease
• Excess levels of thyroid hormone (known as "hyperthyroidism")
• B_{12} deficiency (may increase susceptibility to develop hepatic lipidosis in cats)
• Many other generalized (systemic) conditions or toxins

RISK FACTORS
• Obesity
• Lack of appetite (anorexia)
• Breakdown of lean muscle mass and body tissues (known as "catabolism")
• Rapid weight loss
• B_{12} deficiency

TREATMENT

HEALTH CARE
• Inpatient—necessary for cats that have yellowish discoloration to the gums and other tissues of the body (jaundice or icterus); abnormal position of the head and neck, in which the chin is located near the chest (neck ventroflexion; caused by muscular weakness which can be due to severe electrolyte disturbance of potassium or phosphate, or to thiamine [a B vitamin] deficiency)
• Home care after stabilization and feeding tube has been placed and is functioning problem free
• Outpatient—reduces stress and thereby facilitates recovery in some cats
• Balanced fluids
• Potassium supplementation is important
• Phosphate supplements usually needed
• Magnesium supplements sometimes needed

ACTIVITY
• Cats recently diagnosed or early in recovery phase of hepatic lipidosis may be too weak for any activity
• Activity may help to improve motility of the stomach, when partial paralysis of the muscles of the stomach (known as "gastroparesis") complicates feeding

DIET
• Nutritional support—cornerstone of recovery
• High-protein, high-calorie diet is essential
• Energy—60 to 90 kcal/kg ideal body weight/day
• Forced feeding of some type usually is required; however, forced feeding by mouth may lead to food aversion
• Tube feeding; initially by tube placed into the nose, down the esophagus (the tube from the mouth to the stomach) and into the stomach (known as a "nasogastric tube") and then an esophageal tube after corrected hydration and electrolyte status, and administration of vitamin K_1
• Avoid surgery to place a stomach feeding tube, as cats with hepatic lipidosis have high mortality
• Cautiously offer food daily to assess interest in food and appetite
• Human "stress-formula" intestinal diets generally are not recommended—require supplemental amino acids (arginine [or citrulline], and taurine)—amino acids are the smallest components of protein; taurine is an amino acid that is an important component of the diet of cats; cats cannot produce enough taurine in their bodies and so, must obtain taurine from their food to maintain the health of several organs, including the retina (back of the eye) and heart
• Supplements—improve survival in severely affected cats: L-carnitine; taurine; thiamine (a B vitamin); vitamin B_{12}; water-soluble vitamins (vitamin B, vitamin C); vitamin E; thiol donors (such as *S*-adenosyl-L-methionine [SAMe]); potassium gluconate (for low levels of potassium in the blood [known as "hypokalemia"]), reduces fluid potassium supplements; marine oil in food
• Carnitine supplements have wide variability in bioavailability; Carnitor™ (liquid medical grade carnitine) is recommended

SURGERY
• Exploratory surgery and liver biopsy (if indicated)—inspect for underlying disorders; possibly biopsy the pancreas, stomach, and/or small bowel
• Avoid surgical interventions until hydration, electrolyte depletions, and any blood abnormalities are corrected

MEDICATIONS
Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

• Vitamin K_1—recommended for all cats suspected of having hepatic lipidosis

• Drugs to decrease signs of hepatic encephalopathy (such as drooling, seizures, aggression, and disorientation) usually are not needed; hepatic encephalopathy is a brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia
• Metoclopramide—for vomiting, nausea, and partial paralysis of the muscles of the stomach (gastroparesis)
• Systemic antibiotics—as appropriate for coexistent infections
• *S*-adenosyl-L-methionine (SAMe; Denosyl-SD4®)

FOLLOW-UP CARE

PATIENT MONITORING

• Frequent reevaluations—imperative
• Body weight, condition, hydration, electrolytes
• Judicious adjustments of energy, fluids, and electrolyte provisions—important
• Serum bilirubin—"bilirubin" is a normal bile pigment formed from the breakdown of hemoglobin; "hemoglobin" is the compound in red-blood cells that carries oxygen to the tissues of the body; the liver takes up the hemoglobin following normal or abnormal breakdown of red-blood cells and processes it to form bile (a fluid substance involved in digestion of fats), which provides a means of eliminating bilirubin from the body; bilirubin levels in the blood can increase if the flow of bile is blocked—in the case of hepatic lipidosis, bilirubin is not eliminated from the body at a normal rate, so they increase—serum bilirubin levels decline within 2 weeks of adequate medical management of hepatic lipidosis and predict recovery
• Liver enzyme activity levels are slow to return to normal; do not predict recovery
• Discharge for home care—when vomiting is controlled, partial paralysis of the muscles of the stomach (gastroparesis) has resolved, serum bilirubin levels are declining, patient is able to walk around (known as being "ambulatory"), and tube-feeding apparatus has been problem-free
• Tube feeding—discontinued only after confirmed voluntary food consumption for 2 weeks

PREVENTIONS AND AVOIDANCE

• Obesity—prevent; weight reduction must not exceed 2% body weight per week
• Caution owner to verify food intake during weight loss regimens and during at-home stress

POSSIBLE COMPLICATIONS

• Feeding tube malfunction or obstruction—tube blockage or obstructions may be relieved by papaya juice, carbonated soft drink, or pancreatic enzyme slurry, as directed by your pet's veterinarian
• Rare to have hepatic encephalopathy (brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia) develop after dietary support introduced
• Liver failure, leading to death
• Untreatable underlying cause

EXPECTED COURSE AND PROGNOSIS

• Untreated—progressive disease and death
• Optimal response to tube feeding and nutritional supplements—recovery in 3 to 6 weeks
• Treatment as described— 85% recovery of severely affected animals
• Underlying disease influences outcome
• Hepatic lipidosis rarely recurs
• Hepatic lipidosis does not cause long-term (chronic) liver dysfunction

KEY POINTS

• Sequential blood work (serum biochemical assays) needed to assess recovery
• Learn use and care of feeding tube
• Feeding tubes may be retained for 4 to 6 months
• Underlying disease influences outcome
• Hepatic lipidosis rarely recurs
• Hepatic lipidosis does not cause long-term (chronic) liver dysfunction