



Hepatic Encephalopathy

(Brain Disorder Secondary to Liver Disease)

Basics

OVERVIEW

- “Hepatic” refers to the liver; “encephalopathy” is the medical term for any disorder of the brain
- Disorder affecting the central nervous system, developing secondary to liver disease (known as “hepatopathy”) or abnormal blood flow to the liver
- Primarily caused by accumulation of ammonia in the nervous system due to inability of the liver to rid the body of ammonia
- 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
- “Portosystemic shunt” or “portosystemic vascular anomaly” is a 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; it can be congenital (present at birth) or acquired (condition that develops sometime later in life/after birth)
- Hepatic encephalopathy is associated with (1) sudden (acute) liver failure, (2) portosystemic shunt without other liver disease, and (3) scarring of the liver (known as “hepatic fibrosis/cirrhosis”) with increased blood pressure in the portal vein (known as “portal hypertension”)

GENETICS

- Congenital (present at birth) portosystemic shunt or portosystemic vascular anomaly (in which blood flows 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)—inherited in some breeds (such as the Yorkshire terrier, Maltese, cairn terrier, Irish wolfhound, and miniature schnauzer)
- Certain long-term (chronic) diseases of the liver (hepatopathies) have increased likelihood in some breeds (such as the Bedlington terrier, Doberman pinscher, cocker spaniel, Labrador retriever)

SIGNALMENT/DESCRIPTION OF PET

Species

- Dogs
- Cats

Breed Predispositions

- Congenital (present at birth) portosystemic shunt or portosystemic vascular anomaly (in which blood flows 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)—usually in purebred dogs; increased occurrence in some breeds (such as the Yorkshire terrier, Maltese, cairn terrier, Irish wolfhound, and miniature schnauzer)
- Long-term (chronic) inflammation of the liver (known as “chronic hepatitis”) and condition characterized by abnormal accumulation of copper in the liver, causing liver disease (known as “copper-storage liver disease” or “copper-storage hepatopathy”) are more common in certain breeds (such as the Bedlington terrier, Doberman pinscher, cocker spaniel, Labrador retriever)

Mean Age and Range

- Congenital (present at birth) portosystemic shunt or portosystemic vascular anomaly (in which blood flows 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)—usually young pets
- Acquired (condition that develops sometime later in life/after birth) liver disease resulting in acquired portosystemic shunt—any age

SIGNS/OBSERVED CHANGES IN THE PET

- Nervous system signs—usually associated with meal ingestion, particularly a high-protein meal (such as red meat); signs may occur in pets with generalized infections; bleeding into the gastrointestinal tract; dehydration; excess levels of urea and other nitrogenous waste products in the blood (known as “uremia” or “azotemia”); constipation; or breakdown of lean muscle mass and body tissues (known as “catabolism”)
- Temporary resolution of signs may occur with dietary protein restriction and/or antibiotic or lactulose treatment
- Prolonged recovery from sedation or anesthesia
- Episodic abnormalities or signs may be seen
- Learning disabilities (difficult to train)
- Sluggishness (lethargy) and/or drowsiness or sleepiness (known as “somnolence”)
- Lack of appetite (known as “anorexia”)
- Vomiting
- Excessive salivation (known as “ptyalism”), especially in cats
- Disorientation—aimless wandering; compulsive pacing; head pressing
- Increased urination (known as “polyuria”) and increased thirst (known as “polydipsia”)
- Blindness related to brain abnormality
- Seizures
- Coma
- Inability to urinate in males that have developed ammonium biurate urinary tract stones (known as “ammonium biurate uroliths”) with subsequent blockage of the urethra (the tube from the bladder to the outside, through which urine flows out of the body; condition known as “obstructive urolithiasis”)
- Excessive salivation (ptyalism), seizures, aggression, disorientation—more frequent in cats than in dogs
- Compulsive behavior (such as head pressing, circling, aimless wandering, vocalizing); vomiting; diarrhea; increased urination (polyuria) and increased thirst (polydipsia); blood in the urine (known as “hematuria”), frequent voiding of small volumes (known as “pollakiuria”), and difficulty urinating (known as “dysuria”) associated with ammonium biurate urinary tract stones (uroliths)—more frequent in dogs than in cats
- Cats with congenital (present at birth) portosystemic shunt or portosystemic vascular anomaly (in which blood flows 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) may appear normal size, but most have stunted growth or stature; small liver (known as “microhepatica”); and a golden copper-colored iris (non-blue-eyed and non-Persian cats)
- Dogs with congenital (present at birth) portosystemic shunt or portosystemic vascular anomaly may appear normal size, but usually have stunted growth or stature; small liver (microhepatica)
- Acquired (condition that develops sometime later in life/after birth) liver disease—depends on long-term nature (chronicity) of underlying disorder and formation of acquired portosystemic shunt (in which blood flows 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); fluid buildup in the abdomen (known as “ascites”) is common in dogs with hepatic encephalopathy due to acquired liver disease and often may increase and decrease in severity over time (known as a “waxing and waning” course); variable bleeding

- Lower urinary tract signs (such as straining to urinate; difficulty urinating); orange/brown color to urine due to presence of ammonium biurate crystals in the urine; inability to urinate in males that have developed ammonium biurate urinary tract stones (ammonium biurate uroliths) with subsequent blockage of the urethra (the tube from the bladder to the outside, through which urine flows out of the body; condition known as obstructive urolithiasis)

CAUSES

- Congenital (present at birth) portosystemic shunt or portosystemic vascular anomaly (in which blood flows 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)—malformations of blood vessels during development of circulation
- Acquired (condition that develops sometime later in life/after birth) portosystemic shunt—occurs with diseases that lead to high blood pressure in the portal vein (the vein carrying blood from the digestive organs to the liver; high blood pressure in the portal vein is known as “portal hypertension”), such as progressive damage and scarring of the liver (known as “cirrhosis”)
- Sudden (acute) liver failure—induced by drugs, toxins, or infection

RISK FACTORS

- Alkalosis (condition in which the pH of the body is too high)
- Decreased levels of potassium in the blood (known as “hypokalemia”)
- Low levels of glucose or sugar in the blood (known as “hypoglycemia”)
- Certain anesthetics and sedatives
- Certain medications (such as methionine, tetracycline, antihistamines)
- Bleeding into the intestine—most common cause that leads to signs of hepatic encephalopathy (blood acts as a source of protein, which when broken down forms ammonia)
- Transfusion—stored blood products containing high concentrations of ammonia; incompatible blood transfusions
- Infections
- Constipation
- Breakdown of lean muscle mass and body tissues (catabolism)—disorders causing muscle wasting; normally large amounts of ammonia are detoxified temporarily by storage in muscle

Treatment

HEALTH CARE

- Depends on underlying condition; eliminate factors promoting hepatic encephalopathy
- Improve dietary protein tolerance and alter protein (type and quantity) in diet
- Discontinue medications administered by mouth, if pet is comatose (known as a “hepatic coma”)
- Avoid risk factors
- Fluids—if pet has low blood glucose or sugar (hypoglycemia), supplement fluids with 2.5–5% dextrose; provide potassium chloride, according to needs; restrict sodium in fluids for pets with acquired (condition that develops sometime later in life/after birth) liver disease, fluid buildup in the abdomen (ascites), and/or marked low levels of albumin (a protein) in the body (condition known as “hypoalbuminemia”)
- B vitamins

ACTIVITY

- Keep the pet warm, inactive, and hydrated

DIET

- Adequate calories—avoid breakdown of lean muscle mass and body tissues (catabolism) and maintain muscle mass (as a site for temporary ammonia detoxification/storage)

- Dietary protein restriction—cornerstone of medical management; use commercially formulated diet specific for liver disease or kidney disease, as directed by your pet's veterinarian; dogs: dairy and soy protein best sources; cats: as pure carnivores, they must have meat-derived protein
- Good-quality vitamin supplements—vitamin usage in the body is changed with liver disease and vitamins are lost in urine
- S-adenosylmethionine (SAME) is preferred to methionine supplementation
- Ensure adequate thiamine (a B vitamin)
- Nutrition through intravenous route (known as “parenteral nutrition”)—may be used as a partial means of providing nutrition (recommended for short-term lack of appetite) to minimize breakdown of muscle (catabolism) or as a total means of providing nutrition (known as “total parenteral nutrition” or TPN), which is recommended if the pet has lack of appetite of more than 5 days and providing nutrition via the gastrointestinal tract is not possible

SURGERY

- Congenital (present at birth) portosystemic shunt or portosystemic vascular anomaly (in which blood flows 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)—surgical correction in which abnormal blood vessels are “tied off” or “ligated” is a possibility in many cases (potential exists that only some of the blood vessels can be ligated)
- Acquired (condition that develops sometime later in life/after birth) portosystemic shunt—abnormal blood vessels should not be “tied off” or “ligated”

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

- Medications that increase dietary protein tolerance, change bacteria or conditions in the intestines, or reduce production or availability of substances provoking hepatic encephalopathy
- Antibiotics—antibiotic selection based on ability to change the bacteria in the intestines or their products; first choice antimicrobial selections: administered by injection (known as “systemic administration”), such as metronidazole or amoxicillin; combine use with lactulose
- Non-absorbable-fermented carbohydrates—lactulose, lactitol, or lactose (if lactase deficient); decrease production or absorption of ammonia; increase rate of stool transit; trap nitrogen in bacteria; lactulose most commonly used; therapeutic goal is passage of two to three soft stools daily; also may be administered as an enema for sudden (acute) hepatic encephalopathy and coma *after* cleansing enemas have removed debris
- Probiotics with non-absorbable-fermented carbohydrates—may be helpful in changing the intestinal bacteria to decrease production of toxins that contribute to hepatic encephalopathy
- Enemas—*cleansing enemas* (warmed polyionic fluids) mechanically clean colon; *retention enemas* directly deliver fermentable substrates or directly alter colonic pH and organisms: diluted lactulose, lactitol, or lactose; neomycin in water; diluted Betadine
- Zinc supplementation, as directed by your pet's veterinarian
- Fluid buildup in the brain (known as “cerebral edema”)—complicates sudden (acute) hepatic encephalopathy; administer medication (mannitol) to decrease fluid buildup; administer nasal oxygen and *N*-acetylcysteine; use of steroids to decrease fluid buildup (edema) is controversial as steroids may promote bleeding in the intestinal tract (which is a risk factor)
- If epileptic seizure activity—levetiracetam is the preferred medication to control seizures (known as “anticonvulsants”); other medications include zonisamide and potassium bromide

Follow-Up Care

PATIENT MONITORING

- Reevaluate the pet's at-home behavior, demeanor, body condition, and weight
- Monitor albumin and glucose—in pets with non-correctable disorders; adjust nutrition

- Monitor electrolytes (chemical compounds, such as sodium, potassium, chloride)—especially potassium; avoid low levels of potassium in the blood (hypokalemia) as it aggravates increased levels of ammonia in the blood (known as “hyperammonemia”)

PREVENTIONS AND AVOIDANCE

- Avoid dehydration; excess levels of urea and other nitrogenous waste products in the blood (known as “uremia” or “azotemia”); breakdown of red-blood cells (known as “hemolysis”); constipation; bleeding into the intestines; internal parasites; transfusion of stored blood; ammonium challenge; urinary tract infections; low levels of potassium in the blood (hypokalemia); low levels of magnesium in the blood (known as “hypomagnesemia”); and high pH in the blood (known as “alkalemia”)

POSSIBLE COMPLICATIONS

- Permanent nervous system damage (rare)
- Complications from surgical ligation of abnormal blood vessels in cases with congenital (present at birth) portosystemic shunt or portosystemic vascular anomaly (in which blood flows 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)
- Liver failure
- Death

EXPECTED COURSE AND PROGNOSIS

- Depend on underlying disorder
- Sudden (acute) or long-term (chronic) liver failure—may be fully or partially reversible, or the pet may die

Key Points

- Hepatic encephalopathy—often episodic and relapsing, if underlying disorder cannot be cured
- Learn to administer enemas and to adjust the dose of medications carefully, as directed by your pet's veterinarian
- Congenital (present at birth) portosystemic shunt or portosystemic vascular anomaly (in which blood flows 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)—surgical ligation may be curative, but ligation can cause complications in some dogs; post-operative clinical signs may persist requiring long-term (chronic) nutritional and medical management

Notes

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