Hepatic Encephalopathy – Feline

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Definition

Hepatic encephalopathy (HE) is a disturbance in function of the central nervous system resulting from nitrogenous substances derived from the gastrointestinal tract that gain access to the brain from decreased hepatic function or portal-systemic shunting of blood. HE is uncommon in cats but can occur as a result of congenital portal systemic shunts (PSS), idiopathic hepatic lipidosis (IHL), acute liver failure, and rarely from chronic inflammatory liver disease.

Key Diagnostic Tools and Measures

Diagnosis of HE is based on history, clinical signs, and elevated blood ammonia concentrations. Hypersalivation and seizures are common signs of HE in cats. Laboratory testing and imaging is required to determine if HE is the result of liver failure or PSS. Precipitating factors that can promote HE (see below) should also be investigated. The body condition score (BCS) and body weight (BW) should be noted and the energy requirements for the patient should be calculated based on ideal BW.

Pathophysiology

HE results from nitrogenous substances absorbed from the intestine that without adequate liver metabolism enter the brain and produce alterations of neurotransmission affecting consciousness and behavior. Ammonia is a key factor but other gut-derived toxins include benzodiazepine-like substances, short- and medium-chain fatty acids, phenols, and mercaptans. Alterations in the ratios of aromatic amino acids (AAA) to branched-chain amino acids (BCAA) in the brain may also contribute to HE as increased concentrations of AAA are thought to promote the formation of false neurotransmitters.

Signalment

Congenital PSS are usually identified in young cats. Some are reported to have unusual copper-colored irises. Acute liver disease can occur at any age or in any breed. Idiopathic hepatic lipidosis occurs in middle-aged obese cats. Chronic liver disease occurs in older cats with no breed predilection.

Key Nutrient Modifications

Diet for the management of HE should be selected to reduce the nitrogenous load in the gut. In most cases protein restriction should be avoided when feeding cats with HE because of their high protein requirements. A high-quality, highly digestible moderate dietary protein source contributing 25 to 30% on a dry matter basis (DM) is recommended instead. Vegetable and dairy sources of protein are preferable to animal protein as they provide a higher calorie to nitrogen ratio and tend to be higher in BCAA than AAA.

Cats with IHL often require B vitamin (including cobalamin) supplementation. Other supplements suggested include carnitine, arginine, thiamine, and taurine although their importance in various types of liver disease is not well documented. Carnitine functions to transport fatty acids into hepatic mitochondria for energy production. Arginine is an essential amino acid that must be derived from the diet and plays an important role in the urea cycle of cats. Taurine is also an essential nutrient for cats and is involved in CNS function and taurine deficiency could be confused with signs associated with HE.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Protein#	25–30	5–8	28	6.5
Fat	20–40	4–7	9	2.3
Carbohydrate	30–50	4–12	n/a	n/a

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary DM and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.
*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials
#Meat-based diets should be avoided. Highly digestible milk and plant based proteins are preferred when managing hepatic encephalopathy.

Therapeutic Feeding Principles

Nutrition is the key to the management of cats suffering from IHL as well as other causes of HE. A catabolic state develops quickly in the anorexic cat and prompt measures should be taken to correct this condition. Generally placement of a gastrointestinal (GI) feeding tube is indicated for management of the anorexic cat to meet nutritional requirements. Nasoesophageal, esophageal, or gastrostomy tubes are used for this purpose. Next the caloric needs should be calculated for the patient based on optimal BW; generally feeding 50 to 55 kcal/kg BW is adequate for most cats. Feeding the calculated caloric requirements will promote recovery of hepatic function, regeneration, and adequate protein synthesis.

The amount and protein content of the diet should be considered when HE is present. Unlike dogs, however, protein restriction is rarely initiated in cats with clinical evidence of HE because of the cat's innate protein requirements. The protein fed should be highly digestible and make up 25% to 30% DM. Meat-based proteins are higher in AAA content and should be avoided while dairy- and vegetable-based proteins are higher sources of branched-chain amino acids (BCAA) and can lessen HE. Diets high in fiber generally should be avoided for cats because they decrease the nutrient density of the diet.

Adequate vitamin supplementation, given as a B-complex product, is suggested due to the important metabolic roles vitamins play in the liver. Many types of liver disease may benefit from support in the form of nutritional antioxidants. Nutritional supplements given for antioxidant function including vitamin E and glutathione precursors such as S-adenosylmethionine (SAMe) may be beneficial. Feeding multiple small frequent meals a day may help maintain glucose concentrations and lessen the metabolic impact on the liver at one time.

■ **Treats** – Treats are generally not recommended or given to cats with liver disease.

■ **Tips for Increasing Palatability** – Palatability of the diet is extremely important. First, investigate for liver-associated conditions that could be contributing to anorexia such as GI ulceration or electrolyte abnormalities and correct as needed. Dietary fat improves palatability and there is no need to restrict fat content in the cat with IHL or other causes of HE. Fat also is an important source of energy density. Warming the food or adding flavorings may be helpful. Force-feeding anorexic cats with IHL should be avoided as it may cause food aversions.

■ **Diet Recommendations** – Recommended diets for the patient suffering from HE include the specialty liver or renal diets, both of which contain highly digestible moderate proteins. If tube feeding is required, use

specialty high-density diets formulated for such use. Nasoesophageal feeding tubes require a liquefied feline formula. The caloric requirements should be calculated to determine the patient's needs and the amount divided into four to six small meals a day. Animals exhibiting protein intolerance and worsening clinical signs will require lower-protein diets and additional therapy for HE. Home-cooked diets for cats generally are not recommended because of the cat's unique nutrient requirements.

Client Education Points

- For cats with congenital PSS that have clinical signs of HE, the general recommendation is surgery to correct the anomaly. Cats that do not have surgery must be treated medically, which includes diet, oral lactulose, and if necessary intestinal antibiotics to control the signs. The long-term prognosis is quite variable.
- Acute liver failure causing HE is generally severe and has a guarded prognosis. Acute liver disease has the potential to be reversible, however, if vital metabolic functions can be maintained until the liver has time to regenerate.
- Cats having IHL require aggressive nutritional management and the owner must be involved in tube feeding at home until the cat begins eating on its own. The prognosis generally is good for these cases. If hepatic lipidosis occurs secondary to other conditions causing anorexia, the prognosis generally is not as good and is based on the underlying condition.
- The prognosis for chronic liver disease causing HE is grave. When HE occurs in chronic liver disease, cirrhosis generally is present. In this situation therapy is only supportive and involves treating complications of chronic liver disease. With appropriate HE therapy the patient may be improved in the short term.

Common Comorbidities

Cats with congenital PSS may have seizures associated with elevated blood ammonia concentrations. Occasionally seizures occur following surgical correction of the shunt and may require anticonvulsant therapy.

Cats with IHL occasionally may experience a refeeding syndrome, a condition that results in metabolic electrolyte disturbances. With the introduction of food insulin secretion increases and causes intracellular uptake of phosphorus, potassium, and magnesium. Hypophosphatemia can result in muscle weakness and hemolytic anemia. Slow introduction of food and correction of electrolytes prevents the refeeding syndrome. Hyperglycemia and glucose intolerance are common in cats having IHL or other liver disorders and can be lessened by decreasing the carbohydrate content of the diet.

Chronic inflammatory liver disease (cholangitis) is often associated with concurrent chronic pancreatitis and or inflammatory bowel disease. On rare occasion chronic pancreatitis can result in exocrine pancreatitic insufficiency requiring pancreatic enzyme supplementation.

Interacting Medical Management Strategies

The medical management of HE usually involves additional therapy beyond dietary manipulation. If the initial diet results in continued signs of HE then further protein restriction should be instituted. With acute HE bowel cleansing is a mainstay of therapy because colonic evacuation enemas remove intestinal nitrogenous substrates. Chronic management of HE usually requires the use of oral lactulose and intestinal antibiotics. Lactulose can also be used in the final evacuation enema. Precipitating factors in HE including hypoglycemia, hypokalemia, alkalosis, and GI ulceration should be avoided or prevented. Chronic inflammatory liver disease is usually treated with corticosteroids and may result in glucose intolerance.

Monitoring

The patient should be consuming adequate calories and specific therapies for the primary disease condition should be instituted. The clinical and neurologic status should be improved with appropriate diet and lactulose therapy. If these measures fail to improve the patient status then intestinal antibiotics should be initiated. Precipitating factors in HE should be excluded with a biochemical profile including electrolytes and with a fecal occult blood analysis. If abnormalities are identified they should be corrected.

Algorithm – Nutritional Management of Feline Hepatic Encephalopathy (HE)