Hepatic Disorders

PORTOSYSTEMIC SHUNTS AND HEPATIC ENCEPHALOPATHY

A portosystemic shunt allows portal blood to bypass the liver. The liver does not receive the nutrition it needs, resulting in liver atrophy. Nutrients and other compounds absorbed from the intestines do not undergo hepatic metabolism or detoxification and instead travel directly into the systemic circulation.

Compounds, e.g., ammonia derived from the nitrogen in protein and normally detoxified to urea in the liver, as well as bacteria, endotoxins, and aromatic amino acids, cause adverse effects on other systems in the body:[1,2]

- Due to effects of these compounds on the brain, portosystemic shunts are the main cause of hepatic encephalopathy (HE), causing signs such as lethargy, seizures, ataxia, and changes in behavior. In cats, ptyalism and copper-colored irises are common signs.
- Vague gastrointestinal signs, e.g., vomiting and diarrhea, may be seen.
- Elevated levels of ammonia and uric acid in the urine may result in urate urolithiasis.

Shunts may be either congenital or acquired. Congenital portosystemic shunts are more common in dogs than cats. Their prevalence has been reported at 0.02-0.6% in dogs and 0.02-0.1% in cats.[1] In dogs, congenital shunts are more common in purebreds, e.g., Irish Wolfhounds, Golden Retrievers, Yorkshire Terriers, and Maltese.[1,3] In cats, congenital shunts occur more often in domestic shorthairs.[2] Pets with congenital shunts are often small in stature with low body condition scores. Congenital shunts most commonly occur as one vessel in an individual pet, while acquired shunts are usually comprised of numerous vessels.[4] Acquired shunts may develop in any pet secondary to chronic liver disease or liver damage (e.g., aflatoxin poisoning) with fibrosis resulting in portal hypertension.[4,5]

Surgical ligation of a congenital shunt is generally the treatment of choice. However, some pets with congenital shunts are poor surgical candidates, the owners decline surgery, or surgery is not fully successful. In these cases, in pets prior to surgery, and in pets with acquired shunts, targeted nutrition may be utilized as part of management.[3,6]

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Key Messages

- Diet can be used to help manage associated clinical signs, especially of HE, and improve quality of life:[3]
  - A balanced protein intake is key to reduce the risk of hyperammonemia. A diet containing a moderate level of highly digestible, high biologic value protein is recommended.[7,8] Avoid excess restriction of protein to preserve lean body mass and to prevent cachexia,[9] which is associated with diminished immune function, increased morbidity, and shorter life span.[10]
    - Hepatic stores of glycogen are lower in pets with portosystemic shunts, leading to an increased utilization of amino acids for energy.[11,12] If protein intake is insufficient, muscle protein is catabolized at a high rate.[12] Muscle wasting in turn can potentiate hyperammonemia since muscle becomes the primary site of ammonia detoxification with a portosystemic shunt.[9]
    - Start with a level of 2.1–2.5 g protein/kg body weight/day for dogs and 4 g protein/kg body weight/day for cats in the diet. Provided that the pet shows no signs of HE, gradually increase the level of protein in 0.3–0.5 g/kg increments to the maximum level the pet will tolerate.[13,14]
    - In dogs, the protein source may be important. Non-meat protein sources, such as soy, are better tolerated in dogs with portosystemic shunts at risk for hepatic encephalopathy.[6,7]
    - Consult with a veterinary nutritionist if a homemade diet is elected to ensure individual amino acid requirements are met.[15]
  - Lactulose and prebiotics, e.g., pectin or chicory root, lower the intestinal pH (due to production of short chain fatty acids), which helps reduce ammonia absorption from the gastrointestinal tract. In an acidic environment, ammonia converts to ammonium, which is not absorbed and is excreted in the feces. An acidic environment also promotes the growth of non-urease producing bacteria, e.g., Lactobacillus, which decreases production of ammonia.[12]
  - Probiotics may also promote the growth of non-urease producing bacteria.[14,15]
  - Since zinc is a cofactor for several enzymes involved in detoxification of ammonia, increased dietary levels of zinc may help reduce the risk of hyperammonemia. Zinc is also an antioxidant.[14]
  - Supplementation of fish oil, a source of the long chain omega-3 fatty acids eicosapentaenoic acid and docosahexaenoic acid, may reduce inflammation which, in turn, may reduce the risk of HE.[8]
  - Ammonia is produced during digestion of food. Small, frequent meals should be fed to reduce the level of the post-prandial ammonia burden.[8]
  - To help prevent recurrence of urate urolithiasis, adding water to the diet may increase total water intake, increase urine volume, and decrease urine specific gravity. A more dilute urine contains a lower concentration of urolith precursors. A higher urine volume may also increase frequency of urination, helping eliminate precursors before they can form uroliths.[16]
- Regularly reassess weight, body condition, and muscle condition.

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