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HEPATIC ENCEPHALOPATHY IN DOGS AND CATS

Shidow Torisu, DVM, PhD

Laboratory of Teaching Hospital Faculty of Agriculture
University of Miyazaki, Miyazaki, Japan

Introduction

Hepatic encephalopathy is a type of neurological disorder that occurs as a result of liver dysfunction or portosystemic shunt (PSS). In small animals, PSS is a major cause of hepatic encephalopathy. The approach to diagnosis of hepatic encephalopathy is similar to that of PSS. It is thought that several chemical factors are responsible for hepatic encephalopathy. In this lecture, important chemical factors in clinical assessments and their pathophysiological roles will be discussed. The approach of diagnosis and management of hepatic encephalopathy will also be discussed.

Pathophysiology

The pathophysiology of hepatic encephalopathy is often divided into three different phases: 1. Ammonia and other waste products are generated from protein in the process of food digestion and absorption in the digestive tracts. The blood concentration of these toxic metabolites increases due to impaired liver function or the direct entry into the systematic circulation in PSS. 2. Amino acid imbalance subsequently occurs and causes the changes in the serum. 3. Brain cells are damaged by these toxic metabolites. Impaired cognition is also caused by the changes of neurotransmitters including monoamine and their receptors. Thus, such conditions can rapidly lead to impaired cognition in chronic liver dysfunction or congenital PSS. The following are factors responsible for hepatic encephalopathy; 1. ammonia, 2. mercaptan, 3. amino acid imbalance, 4. abnormalities of the GABA—benzodiazepine receptor.

Diagnosis

Diagnosis of hepatic encephalopathy is made by identifying several non-specific signs and symptoms. Depression and impaired cognition are typical symptoms. Seizure or collapse of the rear legs may be seen. Hepatic encephalopathy should be differentiated from other neural disorders.
Laboratory tests

Blood tests such as total protein, albumin, blood urea nitrogen (BUN), total cholesterol, glucose, total bilirubin, ammonium and total bile acids (TBA) are useful to determine liver function. It is recommended that ammonium and TBA be tested before and after meals.

Amino acid imbalance can be evaluated by measuring branched-chain amino acids (BCAA) and tyrosine (TYR). Liver dysfunction results in a lower level of BCAA and a higher level of TYR. A lower level of BCAA and a higher level of aromatic amino acids (AAA) are also seen by the blood amino acids analysis.

Images

Ultrasound findings are the unsharpness of intrahepatic portal vein branches. Portosystemic shunt vessel can be detected by ultrasound, but is often not convincing. X-ray is useful to determine if it is accompanied by a hepatic enlargement or a small liver.

CT

CT-angiography portography examination is performed under general anesthesia. It is a non-invasive examination that is particularly useful for congenital PSS. However, it may not be useful for multiple PSS. Multiple PSS should be determined by laparoscopic observation and liver biopsy.

MRI

It has been reported the deposition of manganese in the basal ganglia is detected in patients with hepatic encephalopathy. The accumulation of manganese can be detected in dogs and cats with PSS, but it is unclear the MRI finding is associated with neurologic symptoms. It seems to me that there is no direct correlation between the accumulation of manganese and neurologic symptoms in dogs with PSS. However, cerebral atrophy and hyper intensity in the basal ganglia on T1 weighed images are seen in many cases of PSS. These brain MRI findings may be helpful in distinguishing neurological disorders associated with liver diseases (including PSS) from other neurological disorders. Brain edema is also observed in severe hepatic encephalopathy.

Treatments

There are four main types of treatment: 1. To lower ammonia levels, 2. To improve amino acid imbalance, 3. benzodiazepine antagonists, 4. To lower brain pressure.

In acute hepatic encephalopathy with status epilepticus, the first approach is to lower brain pressure. Then, in order to lower ammonia levels, infusion therapy or intravenous arginine is conducted. Lactulose may be administered as an enema when accompanied by constipation. In order to improve amino acid imbalance, BCAA infusion or a diet rich in BCAA is administered. Benzodiazepine antagonists are administered if neurologic symptoms still exist.

In chronic hepatic encephalopathy, the first approach is to lower ammonia levels and improve amino acid imbalance at the same time. A low-protein diet and lactulose are administered. BCAA, dietary fibers and zinc may be administered. Antibiotics are usually not used.

In this seminar, hepatic encephalopathy will be discussed from a clinical point of view. It will be more focused on the practical issues of management.