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Pathophysiology of hepatic encephalopathy in canines

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Abstract
Hepatic Encephalopathy (HE) is a significant neuropsychiatric complication arising from impaired hepatic function or the diversion of portal blood away from the liver. In veterinary medicine, HE is most frequently associated with congenital or acquired portosystemic shunts (CPSS, APSS), acute hepatic failure, and chronic liver disease in dogs, while in cats it is additionally linked to hepatic lipidosis and dietary arginine deficiency. Although well documented in human medicine, HE in companion animals remains comparatively underreported, contributing to ongoing gaps in understanding pathophysiology and optimal diagnostics. The primary objective of this review is to summarize current knowledge on the classification, mechanisms, and clinical aspects of HE in dogs and cats, and to highlight parallels with human HE research.

HE is classified into three types: Type A (acute liver failure), Type B (portosystemic bypass without intrinsic liver disease), and Type C (cirrhosis or chronic hepatopathy), with severity ranging from minimal cognitive deficits to seizures and coma. The pathogenesis is multifactorial and centers on the accumulation of neurotoxins particularly ammonia resulting from impaired hepatic detoxification or shunting of portal blood. Additional contributors include altered neurotransmission, inflammatory cytokine activity, oxidative stress, electrolyte imbalances, and the accumulation of other gut-derived toxins. Ammonia-induced astrocyte swelling represents a major mechanism underlying neurologic dysfunction.

Diagnostic evaluation incorporates clinical history, neurological assessment, laboratory analyses, imaging, and, when indicated, cytology or histopathology. Treatment depends on the underlying etiology but often includes medical stabilization, ammonia-reducing strategies, nutritional modification, hepatoprotective agents, antimicrobial therapy, or surgical correction of portosystemic shunts. Early recognition and targeted therapy are critical for improving outcomes. Continued investigation is needed to refine diagnostic criteria and optimize management strategies for HE in veterinary species.

Keywords: Hepatic encephalopathy, portosystemic shunt, liver failure, ammonia metabolism, veterinary neurology, hepatopathy, dogs, cats

Introduction

Hepatic Encephalopathy (HE) refers to a spectrum of neurological and behavioural abnormalities that arise secondary to liver dysfunction or abnormal blood flow bypassing the liver. The condition has been recognized for more than a century early descriptions date back to experimental dogs with surgically created portocaval shunts who developed neurological signs after eating high-protein meals, historically termed “meat encephalopathy”. In modern veterinary medicine, HE is most commonly associated with: Congenital portosystemic shunts (CPSS) Acquired portosystemic shunts (APSS) secondary to chronic liver disease or portal hypertension Acute or chronic hepatic failure Cats may also experience HE, particularly in association with: Feline hepatic lipidosis Arginine deficiency Congenital portosystemic shunts Studies show that a significant proportion of dogs with CPSS exhibit neurological abnormalities before surgical intervention.

Cats with CPSS frequently show ptyalism and episodic neurological signs. The severity of HE ranges from mild behavioural changes such as disorientation or depression to life-threatening complications including seizures, stupor, or coma. Veterinary literature on HE in companion animals remains limited compared to human medicine, so much of our understanding draws from human studies and experimental models.

Classification of Hepatic Encephalopathy
HE is categorized into three major types based on the underlying cause:

Type A (Acute)
Acute Liver Failure occurs when massive hepatocellular necrosis suddenly impairs liver function. Neurological signs arise rapidly.

Type B (Bypass)
Portosystemic Shunting Results from abnormal blood flow bypassing the liver without primary liver disease. Common in: Congenital portosystemic shunts (CPSS), Acquired shunts caused by vascular abnormalities

Type C (Cirrhotic)
Associated with chronic liver disease occurs in animals with chronic hepatitis, cirrhosis, or severe portal hypertension. This type is further subdivided based on: Persistence (episodic or chronic), Severity of neurological signs.
Minimal HE (MHE) refers to animals with no outward neurological signs but abnormal performance on specialized cognitive tests.

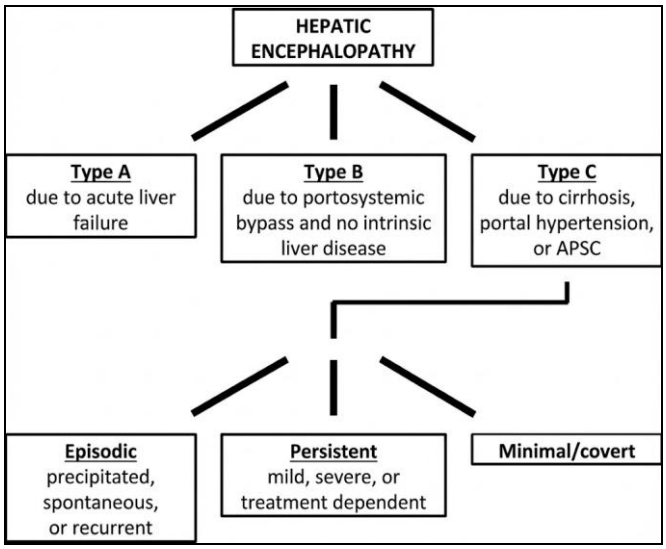

Fig 1: Classification of Hepatic Encephalopathy

Pathogenesis of Hepatic Encephalopathy
It is multifactorial, involving a complex interaction between toxins, neurotransmitter alterations, inflammation, and impaired liver detoxification. One of the most important contributors is ammonia.

- **Ammonia Metabolism Ammonia (NH_3) exists in equilibrium with ammonium ions (NH_4^+):** At physiologic blood pH (≈7.4), NH_4^+ predominates. NH_3 is lipid-soluble and crosses the blood-brain barrier easily. NH_4^+ requires carrier-mediated transport. Abnormal ammonia metabolism is central to HE pathophysiology.
- **Sources of Ammonia The gastrointestinal tract is the body's major site of ammonia production:** Colonic bacteria break down nitrogenous substances and urea Gastric bacteria (e.g., Helicobacter pylori) also produce ammonia Intestinal breakdown of glutamine releases additional ammonia Dietary protein contributes to increased ammonia load Studies in animals with portocaval shunts demonstrate that even germ-free dogs can develop HE, indicating both bacterial and metabolic origins.
- **Impaired Detoxification:** Healthy livers convert ammonia into urea via the urea cycle. In HE, Liver failure reduces conversion efficiency. Portosystemic shunts divert ammonia-rich blood away from the liver. Ammonia accumulates in systemic circulation. High ammonia levels affect astrocytes in the brain, causing swelling and dysfunction.
- **Additional mechanisms other known contributors include:** Altered neurotransmission (e.g., GAB Aergic signaling), Increased brain inflammatory cytokines, Oxidative stress, Accumulation of other neurotoxins (mercaptans, short-chain fatty acids), Electrolyte disturbances, and alkalosis, enhancing ammonia uptake by the brain.

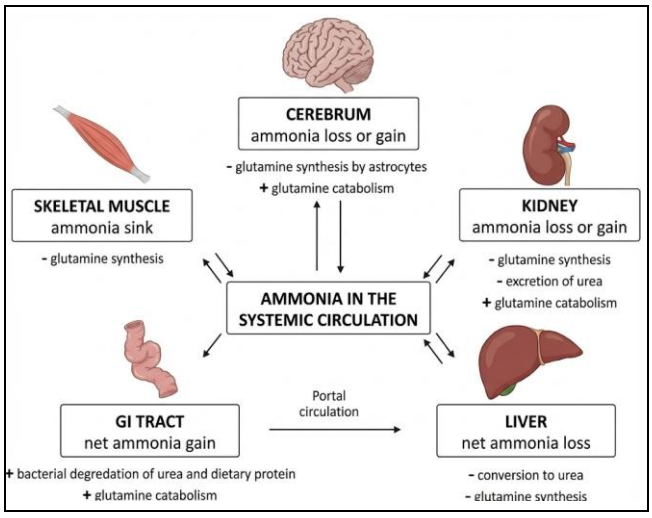

Fig 2: Pathogenesis of Hepatic Encephalopathy

Conclusion
Hepatic Encephalopathy (HE) is a shared complication in both species, reflecting impaired liver detoxification or the diversion of blood away from hepatic metabolism. HE involves neurological dysfunction ranging from subtle cognitive changes to seizures or coma, often requiring urgent intervention. Effective diagnosis of liver disease relies on: Comprehensive history Laboratory evaluation (CBC, chemistry, coagulation tests) Imaging Cytology and histopathology Culture or molecular diagnostics when infectious causes are suspected Treatment varies widely depending on the cause but may include: Supportive care (fluids, nutrition, hepatoprotective medications) Antimicrobials or antiparasitic agents Immunomodulatory therapy Surgical correction of shunts Management of complications such as coagulopathy, neurologic signs, and bile duct obstruction Prevention focuses heavily on vaccination, environmental management, and early identification of at-risk animals

Conflict of Interest
Not available

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Not available

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