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METABOLIC ENCEPHALOPATHIES IN CATS: A LITERATURE REVIEW

INTRODUCTION

Metabolic encephalopathies in cats are a group of neurological disorders resulting from systemic metabolic imbalances that impair brain function. Their diagnosis and management present significant challenges in veterinary medicine, requiring a multidisciplinary approach that often combines clinical neurology and internal medicine. Increased clinical awareness is essential when assessing cats with neurological signs, particularly in the context of systemic illness.

OBJECTIVE

The aim of this literature review is to summarise current knowledge, identify clinical and diagnostic patterns, and highlight gaps in research. It focuses on explaining the pathophysiology of the main metabolic encephalopathies in cats, particularly how imbalances in systemic molecules or toxins can lead to central nervous system dysfunction. The goal is to support veterinarians in recognising and diagnosing these complex disorders more effectively.

METABOLIC ENCEPHALOPATHIES

Table 1. Summary of the pathophysiology, clinical signs, diagnosis and prognosis of the main metabolic encephalopathies documented in cats

	Hepatic Encephalopathy	Uraemic Encephalopathy	Hypoglycaemic Encephalopathy	Thiamine Deficiency Encephalopathy
Primary Cause	Portosystemic shunts, liver dysfunction	Acute or chronic kidney disease	Insulin overdose primarily, rarely other causes of hypoglycaemia	Inadequate thiamine intake or absorption
Main Toxin/Deficit	Ammonia, manganese	Uraemic toxins, electrolyte and acid-base imbalances	Glucose deficiency	Thiamine (vitamin B1) deficiency
Patho-physiology	Astrocyte dysfunction, ↑ glutamine, GABAergic tone ↑	BBB disruption, astrocyte dysfunction, neuroinflammation	Energy failure, ischaemic-like neuronal cell death	↓ ATP, ↑ lactate, oxidative stress, neurotransmitter dysfunction
Common Clinical Signs	Tremors, altered mentation, lethargy, blindness, seizures	Altered mentation, lethargy, muscle tremors, irregular breathing	Ataxia, visual deficits, weakness, seizures, stupor, coma	Cervical ventroflexion, vestibular signs, ataxia, seizures
Diagnostic Clues	↑ serum ammonia or bile acids, characteristic MRI changes	Azotaemia + other causes ruled out	Blood glucose <50 mg/dl, clinical history	Diet history, characteristic MRI changes
Prognosis	Variable, can resolve with treatment of underlying cause.	Can resolve with treatment of underlying cause, but poor for end-stage CKD	Usually good with rapid treatment, can cause brain damage if left too late	Good if treated early, but can be fatal if left too late

METHODOLOGY

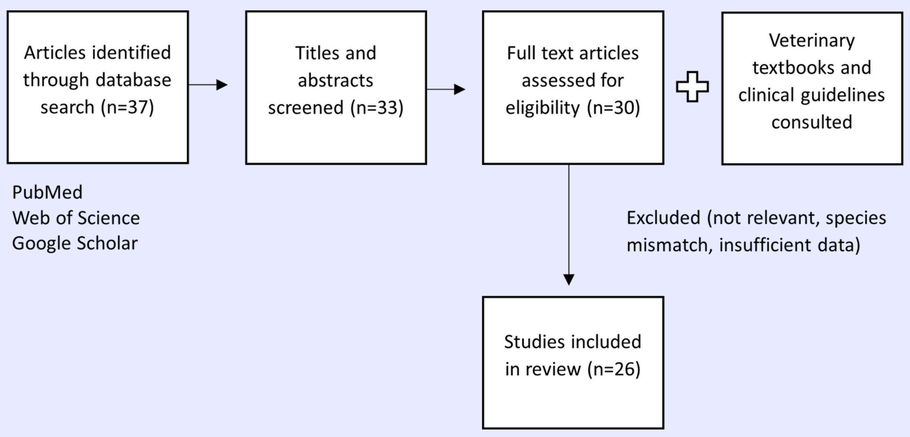
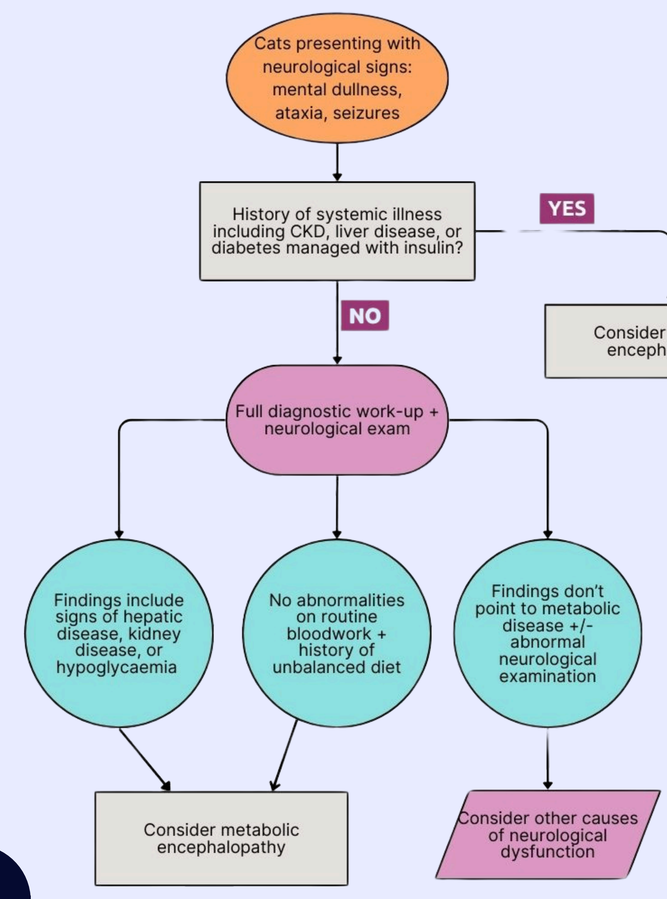


Chart 1: Flowchart outlining a systematic clinical approach to recognising metabolic encephalopathies in cats



CONCLUSIONS

- Despite having different underlying mechanisms, these conditions share some common features such as central nervous system affectation (mainly due to astrocyte dysfunction) and reversible neurological signs when diagnosed and treated early in some instances.
- Hepatic encephalopathy and uraemic encephalopathy are complex disorders with several systems, compounds and mechanisms potentially implicated.
- Hypoglycaemia and thiamine deficiency have a better prognosis if diagnosed early, but demand urgent recognition to prevent irreversible damage.
- The clinical signs of these conditions can be subtle, overlapping, and easily missed — emphasizing the need for high clinical suspicion and a systematic diagnostic approach.
- Early recognition, supportive care, and addressing the underlying cause are essential to improve outcomes in affected cats.
- Further research, especially in feline-specific pathophysiology, is needed to clarify mechanisms and guide treatment strategies.