



*The Medical **Bulletin***

Epetology

Differentials include subdural hematomas, wernicke's encephalopathy, sedating drugs , withdrawal delirium from alcohol or benzodiazepines, hyponatremia, hypoglycemia, SBP, Co2 narcosis, precipitating factors, Medication nonadherence, Infection Increased generation of ammonia, such as during a GI bleed or increased azotemia from overdiuresis, volume depletion or acute renal insufficiency Decreased liver clearance of ammonia, such with alcoholic hepatitis, portal vein thrombosis, HCC or increased shunting after a TIPS Electrolytes disturbances Asterixis reflects an abnormal function in brain structures responsible for alertness and posture. also seen in any form of toxic-metabolic encephalopathy, thalamic/BG NOT specific to cirrhosis. Asterixis is only one symptom of hepatic encephalopathy; cognition, affect, personality, and arousal are equally important.

The goal in treatment is to reduce ammonia absorption from the intestinal lumen. In addition to treating reversible causes, there are two major categories of treatments: antibiotics and disaccharides Lactulose is first-line for both treatment of HE and for prevention of recurrence. AASLD's recommendation is to add rifaximin to lactulose after the second episode of HE (grade 1 A recommendation). The kidney also generates ammonia. Hypokalemia increases renal ammoniogenesis and can precipitate or worsen HE. Correcting hypokalemia in HE is essential. There is no role for NH3 measurements in diagnosis or management of HE.

Dr. Sangram S. Biradar
MD, FICP, FACP, FRCP