Table 2: Pathophysiology of etiology-specific metabolic encephalopathies[2]	
Septic encephalopathy	• multiple contributing factors including microcirculatory abnormalities, altered blood-brain barrier permeability, and inflammatory cytokines.
Hepatic encephalopathy	• multifactorial etiology with ammonia playing a central role. Ammonia is cytotoxic, depleting intermediates of cell-energy metabolism, and most likely acts as osmolyte causing astrocyte swelling. Metabolic products formed from ammonia (i.e., glutamine and alpha ketoglutramic acid) are elevated in cerebrospinal fluid (CSF) in patients with liver disease. • Other theories have been proposed, including production of false neurotransmitters (out of favor), abnormal fatty acid metabolism, free radical damage, cerebral edema and increased mercaptans.
Electrolyte abnormalities	• Altered membrane excitability
Wernicke's encephalopathy	• Due to diencephalic and mesencephalic dysfunction of central gray structures surrounding the third and fourth ventricles
Hypoxic encephalopathy	• Hypoxemia leads to hypotension and ischemia which then triggers an apoptosis pathway resulting in brain damage