Hepatic Encephalopathy the Old and the New



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KEYWORDS

- Hepatic encephalopathy Acute liver failure Fulminant hepatic failure
- Chronic liver failure
 Acute-on-chronic liver failure

KEY POINTS

- An elevated plasma ammonia level (>150 µmol/L) in acute liver failure increases the risk of intracranial hypertension; however, a low level (<146 µmol/L) does not preclude it when associated with multiorgan failure.
- Acute-on-chronic liver failure patients admitted with overt hepatic encephalopathy have a significantly higher short-term mortality rate and small but devastating risk of brain herniation (4%) and are at an increased risk of intracranial hemorrhage (16%).
- Brain MRI pattern of restricted diffusion (cytotoxic edema) in hyperammonemia associated with urea cycle disorder or liver failure correlates in severity with plasma ammonia levels and clinical outcome.
- Therapeutic hypothermia is safe but does not confer a clear mortality benefit in acute liver failure.
- Invasive intracranial pressure monitoring used in an estimate of 20% to 30% of patients with acute liver failure in North America yields a 2.5% to 10% risk of intracranial hemorrhage with unproven benefit.
- Molecular adsorbent recirculating system and embolization of large spontaneous portosystemic shunting may facilitate improvement in grade of hepatic encephalopathy safely but without a proven mortality benefit.

INTRODUCTION

Hepatic encephalopathy (HE) represents brain dysfunction directly caused by liver insufficiency or portosystemic shunting (PSS) that manifests as a wide spectrum of neurologic and psychiatric deficits ranging from subclinical deficits to coma.

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CLASSIFICATION OF HEPATIC ENCEPHALOPATHY

To capture the complexity and breadth of HE, the recent 2014 combined European Association of the Study of the Liver and the American Association for the Study of Liver Diseases guidelines have integrated 4 characteristic factors into the classification of HE (Table 1): (1) underlying disease, (2) severity of manifestation, (3) time course, and (4) precipitating factors. Severity of manifestation was adapted from West Haven criteria¹ and merged with 3 newer definitions, minimal HE, covert HE, and overt HE. For the purpose of this critical care review, the focus is limited on overt HE (types A and C).

Classification of HE	Subclassification of HE	Defining Feature and Description	
1. Underlying disease ^a	Туре А	Acute Liver Failure	
	Туре В	Portal-systemic Bypass without intrinsic hepato-cellular damage	
	Туре С	Cirrhosis and portal hypertension with portal-systemic shunts	
2. Severity of Manifestation ^b	Grade 0	No HE	No HE
		Psychometric or neuropsychological alterations without clinical evidence of mental change	Minimal HE or covert
	Grade I	Trivial lack of awareness Euphoria or anxiety	Covert
		Shortened attention span Impairment of addition or subtraction	
	Grade II	Altered sleep rhythm Lethargy or apathy Disorientation for time Obvious personality change Inappropriate behavior	Overt
	Grade III	Dyspraxia Asterixis Somnolence to semistupor Responsive to stimuli Confused Gross disorientation Bizarre behavior	
	Grade IV	Coma	
3. Time course of presentation	Episodic Recurrent Persistent	Single or episodes occurring >6 mo Episodes occur <6 mo Behavioral alterations that are always present and interspersed with relapses of overt HE.	
4. Precipitating factors	None Precipitated	 Precipitating factors can be identified in nearly all bouts of episodic HE type C and should be actively sought and treated when found 	

 $^{\rm a}$ European Association of the Study of the Liver and the American Association for the Study of Liver Diseases Hepatic encephalopathy Guidelines. 2

^b Adapted from Ferenci P, Lockwood A, Mullen K, et al. Hepatic encephalopathy-definition, nomenclature, diagnosis, and quantification: final report of the working party at the 11th World Congresses of Gastroenterology, Vienna, 1998. Hepatology 2002;35(3):716–21.

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