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Hepatic Failure: An Evidence-Based Approach In The Emergency Department

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This issue of Emergency Medicine Practice focuses on the management of acute liver failure and the acutely symptomatic cirrhotic patient. *For a more detailed discussion of this topic, including figures and tables, clinical pathways, and other considerations not noted here, please see the complete issue on the EB Medicine website at www.ebmedicine.net/topics.*

Key Points	Comments
Acute liver failure (ALF) is a rapidly progressive serious illness; prognosis varies with etiology. Acetaminophen intoxication and viral hepatitis are common causes.	Acetaminophen overdose, hepatitis A, "shock liver," and pregnancy-related ALF have the best spontaneous (non-transplanted) survival; ALF due to Wilson disease, hepatitis B, autoimmune hepatitis, Budd-Chiari syndrome, and malignancy fare worse.[8,12]
Liver failure is manifested by characteristic laboratory tests including evidence of hepatocellular injury, cholestasis, coagulopathy, and hyperammonemia.	While the correlation of ammonia with degree of encephalopathy is controversial, it may be useful in the workup of undifferentiated encephalopathy. The AASLD position paper on the management of acute liver failure recommends its inclusion in the routine laboratory analysis of these patients.[46]
Acute liver failure patients may develop hemodynamic instability, cerebral edema, seizures, and hepatorenal syndrome. Reassess vital signs and mental status frequently.	Intracranial hypertension (IH) (as appreciated via neurological signs) should be managed with head-of-bed elevation (30˚) and mannitol bolus (0.5-1g/kg); its use has been shown to decrease intracranial pressure and improve survival in ALF patients.[64] Hypernatremia (Na 145-155), produced via administration of 30% hypertonic saline, may prevent development of IH.[67]
Patients with refractory ascites are susceptible to spontaneous bacterial peritonitis (SBP); ascitic fluid should be sent for analysis during therapeutic paracentesis.	While recent analyses of asymptomatic patients undergoing outpatient routine paracentesis have shown a low rate (0-3%) of occult SBP,[97,98] analysis of serial samples from inpatients (who may be more similar to a symptomatic emergency department population) demonstrated a much higher rate (21%).[99]
Patients with cirrhosis and ascites with clinical SBP (fever, leukocytosis, abdominal pain) but < 250 cells/mm^3 should be treated until culture results are known. Antibiotic regimens include cefotaxime (2g IV q8h), ampicillin 2g IV q4h combined with tobramycin 1.75 mg/kg q8 hr, or oral ofloxacin (400 mg bid) for less severe illness in patients who can take PO.	While 1 older study suggests that cefotaxime is superior to ampicillin/tobramycin,[105] a 2001 Cochrane Review concluded that there was insufficient evidence to support a particular antibiotic regimen, based on 9 available randomized controlled trials comparing antibiotic regimens (no placebo trials) for SBP.[106]
Variceal hemorrhage should be managed with hemodynamic support, vasoactive agents, and endoscopy. Medical therapy regimens in the US include octreotide (50 µg bolus then 60 µg/hr) and vasopression (0.2-0.4 units/min) accompanied by IV nitroglycerin (starting dose 40 µg/min) to counteract ischemic effects.	A Cochrane meta-analysis of the effects of somatostatin analogues did not demonstrate a mortality benefit, but there was a reduced failure of initial hemostasis and a slightly decreased amount of blood transfused.[113] A Cochrane meta-analysis of 15 RCTs comparing all types of medical therapy with endoscopic sclerotherapy demonstrated that sclerotherapy was not superior to medical therapy on a variety of outcomes including mortality for initial treatment of variceal hemorrhage.[114]

See reverse side for reference citations.

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These references are excerpted from the original manuscript. For additional references and information on this topic, see the full text article at ebmedicine.net.

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