Decompensated liver cirrhosis

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Abstract

The incidence of liver disease continues to increase and is now one of the leading causes of death in the United Kingdom. The increasing prevalence of viral hepatitis combined with a surge in the incidence of both alcohol and obesity related liver disease mean that critical care units are increasingly being called upon to assist in managing those with life-threatening complications of end-stage liver disease. Decompensated cirrhosis is not a single organ illness but a complex multisystem disorder typified by impaired immunity, malnutrition and multiorgan failure and presents a significant challenge to the critical care physician. In this article we describe the epidemiology, aetiology, and pathophysiology of decompensated liver disease and describe the management strategies of a range of resulting clinical complications.

Keywords Decompensated liver disease; encephalopathy; liver cirrhosis; liver transplant

Royal College of Anaesthetists CPD matrix: 1A01, 1A02, 2C04, 3A03, 3C00

Learning objectives

After reading this article, you should be able to:

- appreciate the frequency of critical illness relating to liver disease
- recognize and investigate the causes of chronic liver disease
- understand the pathophysiology of complications of decompensated cirrhosis
- formulate a management plan for patients with decompensated cirrhosis
- understand specific considerations for anaesthesia in patients with decompensated cirrhosis

Incidence, aetiology and pathophysiology

In the UK 10–20% of the population are potentially at risk of liver disease and over half a million adults have cirrhosis. Admissions to intensive care of cirrhotic patients continue to increase and are associated with mortality rates approaching 50%.

Cirrhosis occurs as the end stage of a chronic fibrotic process within the liver. Fibrosis occurs following protracted exposure of hepatocytes to a wide variety of insults (Table 1). Cirrhosis results in the gradual decline of the normal metabolic and synthetic function of the liver. Additionally, the development of fibrosis progressively destroys normal hepatic histological and vascular architecture resulting in a progressive increase in hepatic blood flow resistance and portal hypertension. Decompensated liver cirrhosis is defined by the development of clinically evident complications of portal hypertension (ascites, variceal haemorrhage) or liver insufficiency (jaundice, hepatic encephalopathy). Pathophysiological mechanisms are outlined in Figure 1.

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Patients with decompensated cirrhosis must be distinguished from patients with acute liver failure. A short history of liver function abnormality (<24 weeks) and an absence of clinical signs and imaging features of chronic liver disease support the diagnosis of an acute disease process. In selected cases histological confirmation of cirrhosis may be necessary via biopsy. Liver biopsy is performed by a transjugular approach in the presence of ascites or coagulopathy.

Patients with suspected cirrhosis should be investigated with a screening panel of tests to identify aetiology and allow treatment of reversible causes. Additionally, staging tests are used to identify patients with cirrhosis at risk of varices and hepatocellular carcinoma and to provide prognostic information (Table 2). A variety of scoring systems exist (MELD – model for end-stage liver disease, UKELD – United Kingdom model for end-stage liver disease, Child–Pugh score) that provide prognostic information in chronic liver disease, but in the setting of critical care the sequential organ failure assessment (SOFA) score is a more useful prognostic tool (Table 3).

It is important to remember that cirrhosis is not necessarily an inexorably progressive process. Successful treatment of the underlying cause can potentially partially reverse the disease course. Even patients critically ill with decompensated disease can potentially re-compensate and hence a trial of full critical care support is often appropriate.

Common aetiologies of chronic liver disease

Drug induced	Alcohol
Infectious	Hepatitis B and C
Obesity	Non-alcoholic fatty liver disease
Genetic	Hereditary haemochromatosis
	Wilson's disease
	α_1 anti-trypsin deficiency
Autoimmune	Autoimmune hepatitis
	Primary biliary cirrhosis
	Primary sclerosing cholangitis
Vascular	Budd–Chiari syndrome
Biliary	Secondary biliary cirrhosis

Table 1

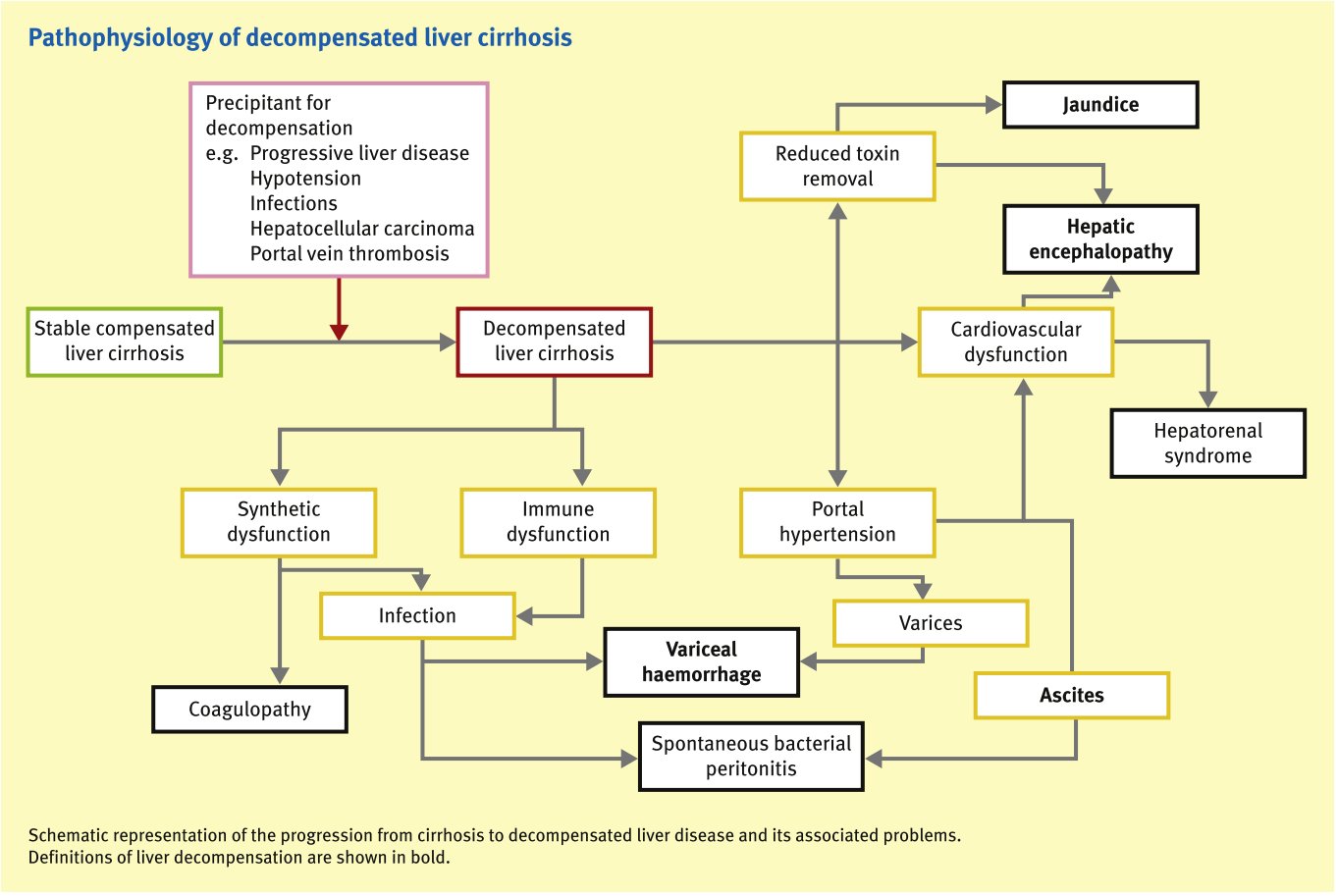

Figure 1

Complications of chronic liver disease

Variceal haemorrhage

As portal blood pressure increases, blood is forced through anatomical venous anastomoses between portal and systemic circulations. These dilated varices are thin walled and prone to spontaneous bleeding. The majority of varices occur within the lower oesophagus (90%) but can also occur in the stomach or elsewhere in the GI tract. Despite the improvements in management, the mortality from variceal haemorrhage remains high. In those with advanced cirrhosis and active variceal bleeding the mortality is 30%. For many patients variceal haemorrhage may be the initial presentation of their chronic liver disease.

Initial management of variceal bleeding is with volume resuscitation, aiming for a target mean arterial pressure (MAP) >65 mmHg. The administration of fresh frozen plasma and platelets to correct coagulopathy is widely undertaken, but it should be borne in mind that a prolonged prothrombin time/INR in cirrhotic patients does not necessarily reflect bleeding risk. Blood product administration should be undertaken judiciously[1] and, if possible, be guided by thromboelastometry. Vitamin K 10 mg IV is often appropriate. Current evidence indicates that a restrictive blood transfusion policy may reduce rebleeding risk and improve survival.[2] A transfusion threshold of 7–8 g/dl has been suggested.[3]

Vasoactive drugs (terlipressin or somatostatin/somatostatin analogues), which selectively reduce portal blood pressure, have been shown to reduce rebleeding risk and mortality. Appropriate treatment (e.g. terlipressin 2 mg IV) should be commenced as soon as variceal bleeding is suspected and continued (e.g. terlipressin 1–2 mg q.d.s. IV) for up to 5 days following endoscopic therapy.[3] Terlipressin should be avoided in patients with significant cardiovascular disease. Empirical broad-spectrum antibiotic therapy (e.g. ceftriaxone 2 g o.d. IV) has been shown to reduce bacterial infection, rebleeding and mortality and should be commenced at time of presentation and continued for 5–10 days.

Upper GI endoscopy should be performed as soon as possible after adequate resuscitation. These patients are at risk of aspiration, especially those with massive haemorrhage or hepatic encephalopathy, and endotracheal intubation and mechanical ventilation should always be considered. These patients are best managed in the critical care or theatre environment. Endoscopic management of bleeding oesophageal varices is with band ligation, whereas gastric or ectopic varices are treated with injection therapy using cyanoacrylate (glue) or thrombin.

Balloon tamponade using a Sengstaken-Blakemore tube can be used for up to 24 hours in uncontrolled haemorrhage as a 'bridge' to more definitive therapy. Patients requiring this should always be intubated for airway protection.

Transjugular intrahepatic portosystemic shunt (TIPSS) insertion is usually considered in patients with uncontrolled or recurrent variceal bleeding. Recent data suggests early TIPSS (within 72 hours) may improve mortality in patients with a high risk of rebleeding.[4]