# Decompensated liver cirrhosis

Jonathan Pearson

Euan Thomson

## 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 or end-stage liver disease. Decompensated cirrhosis is not a single organ illness but a complex multi-system disorder typified by impaired immunity, malnutrition and multi-organ 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. Fibrosis progressively destroys normal hepatic cytological 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.

***Jonathan Pearson*** *RAMC FRCA is a Military Specialty Trainee and Transplant Fellow at the Scottish Liver Transplant Unit, Royal Infirmary of Edinburgh, UK. Conflicts of interest: none declared.*

***Euan Thomson*** *MRCP FRCA is a Consultant in Liver Transplant Anaesthesia at the Scottish Liver Transplant Unit, Royal Infirmary of Edinburgh, UK. Conflicts of Interest: None declared.*

Patients with decompensated cirrhosis must be distinguished from patients with acute liver failure. A short history of liver function abnormality (<26 weeks) and an absence of clinical signs and imaging features of chronic liver disease support the diagnosis of an acute disease process.

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-Turcotte-Pugh score) that are used to prognosticate short and long term mortality in those with chronic liver disease. The MELD score is calculated using a complex formula based on the patient's bilirubin, creatinine and INR (international normalized ratio). The UKELD score is based on a similar complex formula but includes patient's sodium in addition to the above values. MELD scores predict 3-month survival in patients with liver cirrhosis (Table 3), whereas UKELD scores indicate 1-year mortality risk. UKELD score of more than 49 indicates a 1-year mortality risk of 9%. MELD and UKELD have generally superseded the older Child-Turcotte-Pugh Score, although this is still used occasionally in clinical practice to determine survival in cirrhosis (Table 4).

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.

## Specific 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. Variceal haemorrhage is present in 25–40% of cirrhotic patients and each bleeding episode has a 10–30% mortality rate.





Please cite this article in press as: Pearson J, Thomson E, Decompensated liver cirrhosis, Anaesthesia and intensive care medicine (2017), https://doi.org/10.1016/j.mpaic.2017.12.010

**Common aetiologies of chronic liver disease**

| | |
|---|---|
| Drug induced | Alcohol |
| Infectious | Hepatitis B & C |
| Obesity | Non-alcoholic fatty liver disease |
| Genetic | Hereditary haemochromatosis |
| | Wilson's disease |
| | $\alpha_1$-Antitrypsin deficiency |
| Autoimmune | Autoimmune hepatitis |
| | Primary biliary cirrhosis |
| | Primary sclerosing cholangitis |
| Vascular | Budd-Chiari syndrome |
| Biliary | Secondary biliary cirrhosis |

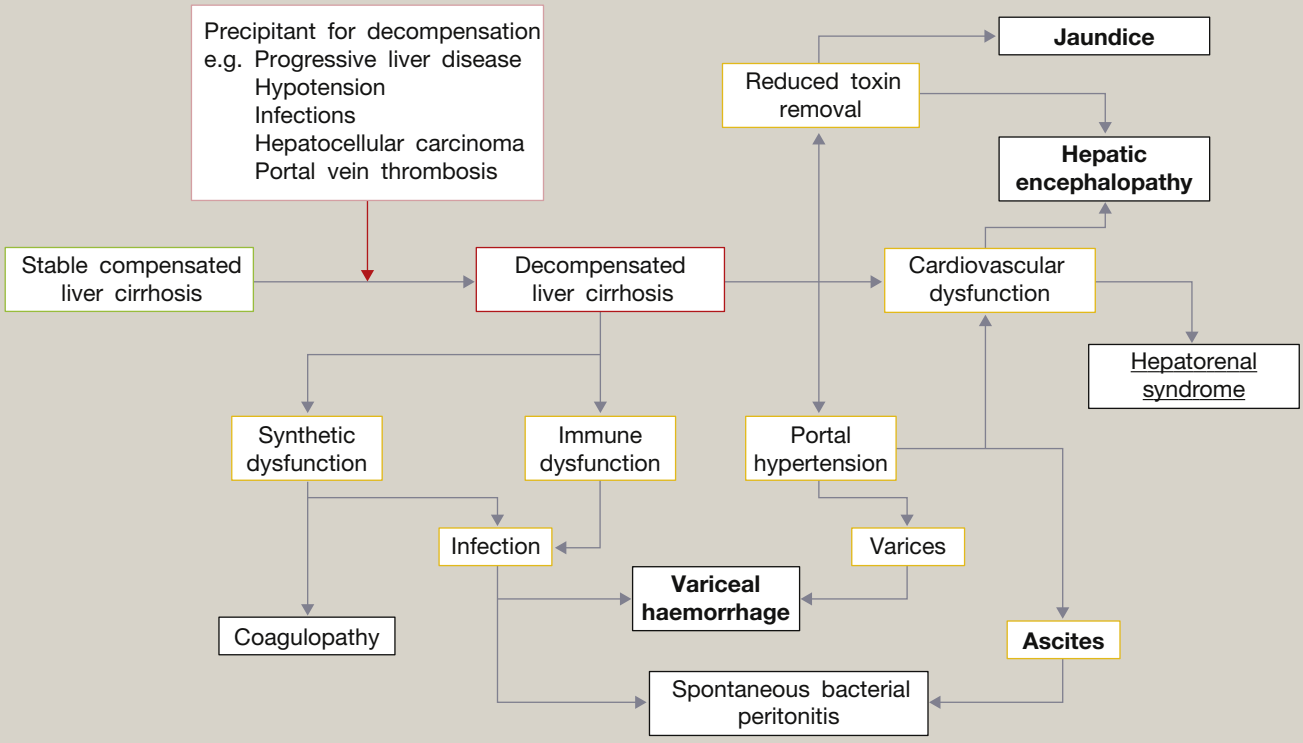
**Table 1**

Initial management of variceal bleeding is with volume resuscitation, aiming for a target MAP >65 mmHg. Current guidelines recommend transfusion of blood, platelets and clotting factors in line with local transfusion policy in patients with massive bleeding.[1] Prolonged prothrombin time/INR in cirrhotic patients does not necessarily reflect bleeding risk. Blood product administration should be undertaken judiciously[2] and, if possible, be guided by thromboelastometry. Fresh frozen plasma should be considered where fibrinogen levels are <1 g/L or prothrombin time/activated partial thromboplastin times are >1.5 times normal. Platelet transfusion should be considered if platelet count $<50 \times 10^9$/L.[1] Vitamin K 10mg IV is often appropriate. Current evidence indicates that a restrictive blood transfusion policy may reduce rebleeding risk and improve survival.[3] A transfusion threshold of 7–8 g/dl has been suggested.[4]

Vasoactive drugs (terlipressin or somatostatin/somatostatin analogues, e.g. octreotide), which selectively reduce portal blood pressure, have been shown to reduce rebleeding risk and mortality. Appropriate treatment (e.g. terlipressin 2 mg IV q.d.s.) should be commenced as soon as variceal bleeding is suspected and continued for up to 5 days following endoscopic therapy.[4] Empirical broad spectrum antibiotic therapy (e.g. ceftriaxone 1 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. Early airway protection with tracheal intubation should be considered in these patients who are at risk of aspiration, especially those with massive haemorrhage or hepatic encephalopathy. Endoscopic management of

**Pathophysiology of decompensated liver cirrhosis**




(Schematic representation of the progression from cirrhosis to decompensated liver disease and its associated problems. Definitions of liver decompensation are shown in bold.)

**Figure 1**




Please cite this article in press as: Pearson J, Thomson E, Decompensated liver cirrhosis, Anaesthesia and intensive care medicine (2017), https://doi.org/10.1016/j.mpaic.2017.12.010