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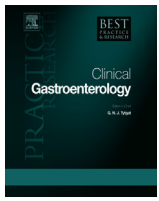

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Non-hemorrhagic acute complications associated with cirrhosis and portal hypertension

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A B S T R A C T

Timely recognition and management of acute complications of cirrhosis is of significant importance in order to reduce morbidity and mortality, especially in the hospitalized patient. In this review, we present a practical approach to the identification and management of non-hemorrhagic acute complications of cirrhosis, specifically bacterial infections, acute kidney injury, and acute exacerbation of hepatic encephalopathy, focusing on patient stratification.

Introduction

Cirrhosis represents the final stage of chronic liver disease due to any cause. In the natural history of cirrhosis, there is a compensated phase (i.e. asymptomatic phase) followed by a decompensated phase defined by the development of complications of portal hypertension and/or liver insufficiency, specifically ascites, variceal haemorrhage, and/or encephalopathy [1]. The decompensated patient can become 'further' decompensated by the development of refractory ascites, hepatorenal syndrome, recurrent variceal haemorrhage, recurrent/persistent hepatic encephalopathy, and/or jaundice. Of

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these, the onset of acute kidney injury (AKI) and/or an acute exacerbation of hepatic encephalopathy (HE) motivate admission to the emergency room. Common precipitants of AKI and/or HE are bacterial infections (Fig. 1). The timely recognition and treatment of infection and other precipitants of further decompensation are essential in order to improve survival and/or to allow the patient to successfully be transplanted. This review will present a practical approach to the identification and management of non-hemorrhagic acute complications of cirrhosis, specifically bacterial infections, acute kidney injury, and acute exacerbation of hepatic encephalopathy.

Bacterial infections in cirrhosis

Bacterial infections are a common complication in hospitalized patients with cirrhosis. It is estimated that approximately a third of patients are admitted with an infection or develop it during admission. This is in contrast to the rate of infections in the hospitalized non-cirrhotic population which is 5–7% [2].

In a recent prospective study of consecutively hospitalized patients, the most common bacterial infections were the so-called 'spontaneous' infections (accounting for a third), that is, spontaneous bacterial peritonitis (SBP), spontaneous bacterial empyema (SBE), and spontaneous bacteraemia (SB) followed by urinary tract infection (UTI) and pneumonia [3]. Any of these infections can lead to further decompensation with development of acute kidney injury, jaundice, coagulopathy, and/or encephalopathy (Fig. 1). The development of this multi-organ failure in a patient with cirrhosis has been recently referred to as acute-on-chronic liver failure [4]. Early recognition and initiation of antibiotic therapy have led to decreases in the mortality of infections in general [5] and in mortality due to SBP [2,6].

Spontaneous infections (SBP, SBE, and SB) are unique to patients with cirrhosis and occur in the absence of a contiguous (or remote) identifiable source of infection (e.g. in the case of SBP there is no intestinal perforation or intra-abdominal abscess and in the case of SBE there is no pneumonia). The main mechanism appears to be translocation of bacteria from the gut lumen to mesenteric lymph nodes, to the systemic circulation (bacteraemia), and then to existing fluids (ascites and/or hydrothorax) [7].

As would be expected given its pathophysiology, SBP (and other spontaneous infections) are mostly due to enteric organisms (mostly gram-negative). However, because of the widespread use of antibiotic prophylaxis, a greater incidence of infections due to antibiotic-resistant organisms has been reported in the literature [3,8].

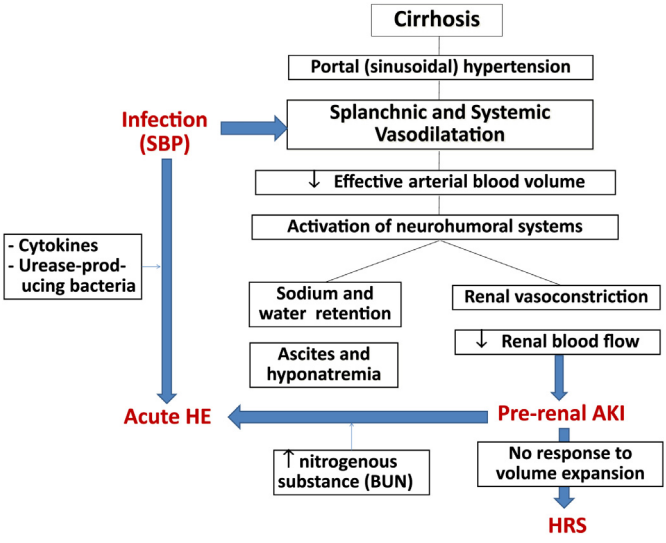

Fig. 1. Overview of the pathophysiology of the non-hemorrhagic acute complications associated with cirrhosis and portal hypertension. Infection, acute kidney injury, and hepatic encephalopathy are closely interrelated. (SBP = spontaneous bacterial peritonitis; HE = hepatic encephalopathy; AKI = acute kidney injury; HRS = hepatorenal syndrome; BUN = blood urea nitrogen).