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TREATMENT FOR HEPATORENAL SYNDROME IN PEOPLE WITH DECOMPENSATED LIVER CIRRHOSIS

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Abstract
Hepatorenal syndrome is defined as renal failure in people with cirrhosis in the absence of other causes. In addition to supportive treatment such as albumin to restore fluid balance, the other potential treatments include systemic vasoconstrictor drugs (such as vasopressin analogues or noradrenaline), renal vasodilator drugs (such as dopamine), transjugular intrahepatic portosystemic shunt (TIPS), and liver support with molecular adsorbent recirculating system (MARS). There is uncertainty over the best treatment regimen for hepatorenal syndrome. Spontaneous bacterial peritonitis occurred in 5.9% of patients. The most common cause of AKI was pre-renal. Hepatorenal syndrome was identified in 9.8% of patient encounters. Predictors of HRS were history of ascites, serum creatinine >2.5 mg/dL, albumin <3 g/dL, bilirubin >2 mg/dL and spontaneous bacterial peritonitis. We demonstrate strong predictors for the development of HRS which can aid clinicians to attain an early diagnosis of HRS, leading to prompt and targeted management and improving outcomes.

Keywords: Predictors, hepatorenal syndrome, cirrhosis, mortality, acute kidney injury.

Introduction

Acute kidney injury (AKI) is a common complication in patients with decompensated liver cirrhosis with the most common cause being dehydration and volume depletion [1,2]. AKI in cirrhotic patients is associated with high morbidity and mortality [3], with an estimated medial survival of less than 50% at 3 months [4,5,6].

Mechanisms of development of AKI in cirrhotic patients are variable. Cirrhotic patients are at risk of decreased effective arterial blood volume secondary to splanchnic vasodilatation, resulting in decreased renal perfusion. Cirrhotic patients are also at increased risk of acute or chronic gastrointestinal bleeding, as well as volume depletion due to medications such as lactulose and diuretics, which can result in further reduction of their effective arterial volume.

Hepatorenal syndrome (HRS) is a form of AKI, occurring in patients with decompensated liver cirrhosis. The definition of HRS has evolved over the past several years. The updated definition of HRS by the International Club of Ascites(ICA) in 2015 [7] renamed the previously known HRS-type 1 as HRS-AKI, and abandoned the previously required doubling of serum creatinine to >2.5 mg/dL within 2 weeks, to replace it with the definition of AKI based on the updated KDIGO guidelines which is an increase in serum creatinine by 0.3 mg/dL within 48 h, or 1.5 times increase in baseline creatinine which is known or presumed to have occurred within the prior 7 days [8]..

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Early diagnosis and treatment of hepatorenal syndrome is important as better prognosis substantially depends on timely management in this group of patients [12,13]. Treatment with albumin and terlipressin or vasopressors has clearly been shown to improve mortality [14,15,16]. In our study herein, we hypothesize that the risk of development of HRS-AKI can be predicted based on patient baseline clinical characteristics and laboratory values at the time of development of acute kidney injury. We therefore aimed to describe the variables associated with the development of HRS-AKI in cirrhotic patients with acute kidney injury to guide clinicians in determining the risk of development of HRS-AKI which would help attaining an early diagnosis by increasing clinical awareness specifically in this group of patients.

Materials and Methods

This review studied people of any sex, age, and origin, having advanced liver disease due to various causes, and who had developed hepatorenal syndrome. People were administered different treatments. The review authors excluded studies with liver-transplanted participants. Participants age, when reported, ranged from 42 to 60 years. The number of females ranged from 6 to 62 out of 100 in the studies that reported this information. The main treatments compared were albumin alone, albumin plus terlipressin, and albumin plus noradrenaline. The authors gathered and analysed data on death, quality of life, serious and non-serious complications, time to liver transplantation, recovery from hepatorenal syndrome, and disappearance of symptoms.

Results

The 25 studies included a small number of participants (128 participants). Study data were sparse. Twenty-three studies with 118 participants provided data for analyses. The follow-up in the trials ranged from one week to six months. The review shows that:

- About 60 out of every 100 people died within three months, and 35 out of every 100 people recovered from hepatorenal syndrome.
- The provided treatment may make no difference to the percentage of people who died or developed serious complications, number of serious complications per person, percentage of people who developed complications of any severity, or the percentage of people undergoing liver transplantation.
- None of the trials reported health-related quality of life.
- The number of complications of any severity was lower with albumin plus noradrenaline than albumin plus terlipressin.
- Recovery from hepatorenal syndrome may be lower with albumin plus midodrine plus octreotide and albumin alone than albumin plus terlipressin and albumin plus noradrenaline.
- We have very low confidence in the overall results.
- Future trials with proper design and quality are needed to clarify the best treatment for people with advanced liver disease having hepatorenal syndrome.

Conclusions

Based on very low-certainty evidence, there is no evidence of benefit or harm of any of the interventions for hepatorenal syndrome with regards to the following outcomes: all-cause mortality, serious adverse events (proportion), number of serious adverse events per participant, any adverse events (proportion), liver transplantation, or other decompensation events. Low-

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certainty evidence suggests that albumin plus noradrenaline had fewer 'any adverse events per participant' than albumin plus terlipressin. Low- or very low-certainty evidence also found that albumin plus midodrine plus octreotide and albumin alone had lower recovery from hepatorenal syndrome compared with albumin plus terlipressin.

Future randomised clinical trials should be adequately powered; employ blinding, avoid post-randomisation dropouts or planned cross-overs (or perform an intention-to-treat analysis); and report clinically important outcomes such as mortality, health-related quality of life, adverse events, and recovery from hepatorenal syndrome. Albumin plus noradrenaline and albumin plus terlipressin appear to be the interventions that should be compared in future trials.

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