Management of hyponatraemia in cirrhotic ascites

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Citation

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Abstract

Hyponatremia is a commonly encountered problem in patients with ascites and its incidence increase with the use of diuretics. Management of such patients often poses problems and since symptomatic hyponatremia is unusual in cirrhosis, and hence close electrolyte monitoring is essential. Here ways of managing this condition with evidence supporting these methods is analysed.

INTRODUCTION

Hyponatraemia is a common clinical problem faced during treatment of cirrhotic patients.

The prevalence of this condition has increased due to diuretic usage, which has become a standard part of the treatment. Management of these patients is often complex due to problems with salt restriction and worsening ascites and the need for recurrent paracentesis and more complex procedures in patients with resistant ascites.

PATHOPHYSIOLOGY

The primary mechanism of development of hyponatremia is different to development of ascites in itself. Patients with cirrhosis and ascites usually have a marked reduction in systemic vascular resistance (SVR)- mostly in the splanchnic circulation and in mean arterial pressure (MAP) plus an increase in cardiac output.($_1$).Ability to excrete water is reduced with the advancement of cirrhosis($_2$) combined with increased ADH-antidiuretic hormone release as shown in studies by Arroyo et al.($_3$). Hyponaatremia acts as a prognostic marker of worsening liver disease, especially with values below 130meq/L, as shown by Papadakis et al. Degree of hyponatraemia parallels severity of liver disease and hyponatraemia <120mmols is seen in <1-2% patients.($_4$).

MANAGEMENT

Diagnosis is simple with testing of serum biochemistry. There is no firm evidence regarding the frequency of estimation of serum electrolytes but common clinical practice is to test every week after a dose change and once stable clinical status is achieved, frequency of testing can be reduced to once in two weeks, should suffice.

Management of such patients often poses problems and since symptomatic hyponatremia is unusual in cirrhosis, only modest water restriction that is tolerated by the patient is followed in our clinical setting as first step.

Withholding diuretic doses, especially furosemide when plasma sodium drops below 126meq/L along with fluid restriction should be the next step, with monitoring of urine output as a measure of fluid restriction. This can be followed by cessation of spironolactone if sodium levels do not respond to the initial measures.

Overly rapid drop in sodium levels should be avoided since it can precipitate hepatic encephalopathy. No studies or controlled trials have done in this specific condition, and use of normotonic or hypertonic saline to raise plasma sodium has been attempted, but being cautious not to increase it by more than 12meq/l per day is necessary to avoid central pontine myelinolysis. Alcoholic patients are more prone to neurological complications on rapid correction in plasma sodium levels ($_5$).

Other treatments including vasopressin receptor analogues, demeclocycline etc have been tried, but no definite benefit ($_6$). Paracentesis is an option independent of the fluid restriction, since indications are only tense ascites with respiratory splinting, peripheral pedal edema or failed diet and diuretics. But if fluid has to be lost urgently, paracentesis is preferred over persistence with diuretics. Monitoring renal functions very closely during this period helps to prevent/treat hepatorenal syndrome early and improve outcomes.

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