

Hepatology Highlights

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Renal and circulatory effects of large volume plasma expansion in patients with hepatorenal syndrome type 1

Umgelter A, et al. Hepatorenal syndrome (HRS) type 1 is a rapidly progressive but potentially reversible form of renal failure that occurs in patients with cirrhosis and ascites and is associated with high mortality. The present uncontrolled interventional pilot study examines the hemodynamic and renal effects of large volume plasma expansion in HRS 1. Studied 14 patients who had persistently elevated serum creatinine above 221 $\mu\text{mol/L}$. He found that there were two complete responses after 48 h. At day 7, 2 patients had a partial and 4 a complete response. At day 12, 2 patients showed a partial response and 9 a

complete response. Of the non-responders, three were anuric and had to be treated by continuous veno-venous hemofiltration over 5, 7 and 24 days. Regarding to plasma expansion, hemodynamics and renal function. Author found that responders ($n = 9$) vs. non-responders ($n = 5$) had a significantly higher initial CrCreat [15 (10-24) mL/min vs. 8 (2-11) mL/min] ($p = 0.048$), but there were no significant differences between the two groups for any other hemodynamic or laboratory parameter at baseline.

The results of the present study suggest that HRS type 1 may revert after large volume plasma expansion with or without paracentesis in a proportion of patients.

However, the survival of cirrhotic patients with HRS type 1 remains poor, although it may be improved by this specific therapy.

Evidence for liver injury in the setting of obstructive sleep apnea

Byrne TJ, et al. The aim of this study was to assess the hypothesis that OSA is associated with liver injury independent of obesity. Authors reviewed the medical histories of 73 consecutive patients referred to a hospital-based sleep lab because of suspected OSA. 35/53 patients (66%) had OSA, 31/53 (58%) patients were obese, 15 (28%) and 12 (23%) patients had elevated AST and ALT, respectively. Elevated ALT was found in 11/35 (31%) patients with OSA, compared to 1/18 patients without OSA ($p = 0.041$).

Frequency of elevated AST [obese 11/31 (35%); non-obese 4/22 (18%)] or ALT [obese 10/31 (32%); non-obese 2/22 (9%)] was not significantly different in the obese and non-obese cohorts. OSA may be a risk factor for liver injury independent of obesity. Unfortunately, the study is done in a relatively small sample of subjects without histopathology. Also it is important to characterize whether or not the patients have underlying NASH and what the fibrosis stage is amongst those subjects. The prevalence and nature of liver disease in the setting of OSA should be determined with larger, prospective studies. The impact of OSA treatment, if any, on liver injury should be similarly evaluated.