Letters to the Editor

Reply to: “Noradrenaline in the treatment of patients with hepatorenal syndrome – Back to the roots?”

To the Editor:
We thank Dr. Lenz for his interest in our paper. We agree with his view on noradrenaline and terlipressin therapy in hepatorenal syndrome (HRS). It would have been ideal to compare the two vasopressors if given as infusion and dose monitored by mean arterial pressure. However, terlipressin has been used as bolus therapy in most of the studies [1]. Gerbes et al. [2] treated consecutive patients with HRS with continuous terlipressin infusion and reported reversal of HRS in 42% of patients in a retrospective study. They concluded that continuous terlipressin infusion may achieve response rates comparable with those of bolus administration, but possibly at a lower daily dose (3 mg) and with less severe complications (9%). In our study also, dose, adverse effects, and response rates with bolus administration were comparable. However, more studies focusing specifically on this aspect are needed to answer this question.

High urinary sodium was unusual in our patients. However, the following factors may explain this finding:

1. In patients with the hepatorenal syndrome, there is renal sodium retention, and urinary sodium is characteristically less than 10 mmol/L per liter throughout their clinical course [3]. However, occasional patients with the hepatorenal syndrome have been recognized in whom urinary sodium is consistently greater than 10 mmol/L per liter [3]. In some of these patients, urinary sodium is initially low but increases to levels of approximately 40 mmol/L per liter as renal impairment progresses, and it has been suggested that this late increase in urinary sodium concentration may represent the possible transition to acute tubular necrosis [3]. Trawale et al. [4], also demonstrated acute tubular necrosis in cirrhotic patients with HRS (high serum creatinine levels, proteinuria <0.5 g/day and no haematuria).

2. The current diagnostic criteria for HRS include presence of cirrhosis, ascites, serum creatinine >1.5 mg/dL after at least 48 h of diuretic withdrawal and volume expansion with albumin plus absence of shock, treatment with nephrotoxic drugs, and parenchymal renal disease [5]. The use of minor criteria (urine sodium <10 mEq/L) and exclusion of patients with infections is abandoned. The majority of the trials published showed low urinary sodium and used the previously established criteria [6], and might have excluded patients with urine sodium >10 mEq/L.

3. All our patients were admitted in emergency, hence were consuming salt without any restriction in their diet and some of the patients were on diuretics which were withdrawn only after admission.

4. A relatively high urinary sodium concentration has been observed in patients with HRS and alcoholic hepatitis [7] and more than 60% of our patients were alcoholic in etiology.

5. Relative adrenal dysfunction is a common problem in patients with cirrhosis with or without sepsis [8,9] and adrenal insufficiency is associated with high urinary sodium [10]. As pointed out by Dr. Lenz, in some of our patients ongoing infection cannot be excluded which might have contributed to the adrenal insufficiency and high urinary sodium, however, all our patients were covered with broad spectrum antibiotics.

Conflict of interest
The authors declared that they do not have anything to disclose regarding funding or conflict of interest with respect to this manuscript.

References

Virendra Singh*
Souvik Ghosh
Narendra S. Choudhary
Department of Hepatology, Postgraduate Institute of Medical Education and Research, Chandigarh, India
*Corresponding author.
E-mail address: virendrasingh100@hotmail.com