Background and aims: Presence of hyponatremia is associated with development of hepatic encephalopathy (HE), but the dynamic relationship between S-sodium and rate of HE is unknown. We examined the association between S-sodium and changes in it, and HE occurrence in a large dataset from three randomised trials of satavaptan in cirrhosis patients with ascites. Methods: During follow-up regular clinical examinations for HE and repeated measures of S-sodium were performed. We used fractional polynomials to estimate the association between S-sodium and the hazard rate of HE. We used Cox regression to adjust for confounding by patient gender, age, cirrhosis aetiology, MELD score, albumin, total bilirubin, platelets, creatinine, hospitalization, use of lactulose, previous HE, and diabetes. We also analysed the effect of current S-sodium and the effect of change in sodium since inclusion on the hazard rate of HE. Results: We included 1116 patients of whom 302 developed HE. Median S-sodium at inclusion was 137 (IQR, 134-139). The association between S-sodium and the logarithm of the HE hazard rate was linear over the range of measured S-sodium values (109-168 mmol/L), so that the confounder-adjusted HE hazard rate increased by 8% (adjusted HR = 1.08, 95% CI 1.06-1.10) for every mmol/L decrease in S-sodium. Current S-sodium had a much stronger effect on the HE rate than the change in S-sodium since inclusion. Conclusion: The rate of HE development increased by 8% per mmol/L decrease in serum sodium. Current S-sodium was more important than the change in S-sodium since inclusion.

Figure: Association between serum sodium and the HE rate
Association between serum sodium and the HE rate

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