

# Adrenocortical Insufficiency in a Patient with Stable Liver Cirrhosis

Kirresh, A; Htay, T; Muralidhara, K

Department of Diabetes and Endocrinology,

Northwick Park Hospital, London Northwest Healthcare NHS Trust



London North West Healthcare NHS Trust

**Case History:** A seventy-six year old Caucasian man was admitted with confusion, irritability and lethargy for one week duration. He has stable Child-Pugh Class A chronic liver disease. On Examination, he was mildly confused (AMTS 8/10), and euvoletic with a blood pressure 130/78 mmHg and no postural hypotension. Respiratory cardiovascular and abdominal examinations were unremarkable. He was taking spironolactone 150 mg and furosemide 40 mg daily for recurrent ascites.

## Investigations:

Haemoglobin 122g/L  
 White cell  $9.8 \times 10^9/L$   
 Platelet  $118 \times 10^9/L$   
 Urea 8.7 mmol/L  
 Creatinine 85  $\mu\text{mol/L}$   
 Sodium 111 mmol/L  
 Potassium 5.66 mmol/L  
 Albumin 33 g/L  
 ALT 108 IU/L  
 Alkaline phosphatase 116 IU/L

Random Cortisol 68 nmol/L  
 Plasma Osmolarity 242 mOsm/kg  
 Urine Osmolarity 361 mOsm/kg  
 Spot urine sodium 39 mosm/L  
 TSH 2.94 mIU/L, fT4 17.1 pmol/L  
 FSH 6.0 IU/L  
 LH 4.5 IU/L  
 Prolactin 480 mIU/L  
 IGF1 3.0 mmol/L

**CXR:** Normal appearances

**CT Abdomen:** Normal adrenal glands

**Although Spironolactone was discontinued the sodium level remained unchanged therefore Standard Short Synacthen test with 250 microgram was performed**

	ACTH ng/L	Cortisol nmol/L
Basal	9	65
30 minutes		204
60 minutes		252

Table 1. Short Synacthen test values

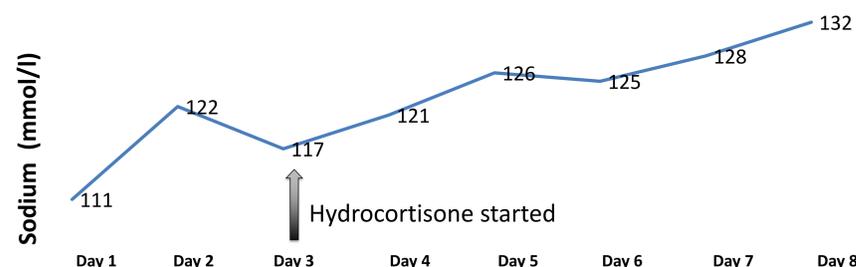


Figure 1. Sodium levels before and after hydrocortisone

**Management:** As he had a decreased response to Synacthen (Table 1), hydrocortisone 10mg, 5mg, 5mg was commenced which improved his confusion and sodium to 132 mmol/L (Fig 1). Spironolactone was restarted and he was discharged home on hydrocortisone. At two week follow-up, he was well and his sodium was 136 mmol/L.

## Discussion and Learning Points

- Up to 72% of patients with chronic liver disease have adrenal insufficiency.<sup>(1,2)</sup>
- Free cortisol is a better biomarker but is not available for standard clinical use.<sup>(1)</sup>
- In CLD, there is reduced synthesis of cortisol binding globulin, which overestimates the incidence of AI.<sup>(2)</sup>
- There is no consensus treatment guideline, however in critically-ill patients with AI (also known as hepatoadrenal syndrome), glucocorticoid replacement reduces mortality and vasopressor dependence.<sup>(1,3)</sup>
- 1-year survival rates in non-critically ill patients with cirrhosis with and without AI were 69% and 95% respectively. Therefore, although there is controversy, treatment in this group should be considered.<sup>(1, 3)</sup>

## References:

- (1) Marik, P; Gayowski, T; Starzl, T et al. The hepatoadrenal syndrome: A common yet unrecognized clinical condition (2005). *Crit Care Med*; 33 (6), pp. 1254- 1259.
- (2) Fede, G; Spadaro, L; Tomaselli, T et al. Adrenocortical dysfunction in liver disease: a systematic review (2012). *Hepatology*; 55 (4).
- (3) Relative adrenal insufficiency in chronic liver disease: its prevalence and effects on long-term mortality. *Aliment Pharmacol Ther*. 2014 Oct ;40(7):819-26. doi: 10.1111/apt.12891. Epub 2014 Jul 30.