

INCREASED URINARY LEVEL OF NEUTROPHIL GELATINASE–ASSOCIATED LIPOCALIN (NGAL) IN PATIENTS WITH CIRRHOSIS AND TYPE 1 HRS

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Background and aims: Hepatorenal syndrome (HRS) in patients with advanced cirrhosis has been defined as a functional renal failure. Nevertheless there are some new evidence of the presence of renal parenchymal lesions in these patients. Neutrophil gelatinase–associated lipocalin (NGAL) is secreted into the urine by the thick ascending limb of Henle and collecting ducts of the kidney. Thus, urinary excretion of NGAL is increased in parenchymal acute injury (AKI) but not in functional AKI. The aim of the study was to evaluate baseline urinary excretion of NGAL in patients with cirrhosis and type 1 HRS and in patients with cirrhosis and normal renal function.

Methods: We tested the baseline urinary excretion of NGAL in 28 patients with cirrhosis and type 1 HRS and in 15 patients with cirrhosis and normal renal function. We defined type 1 HRS according to the International Club of Ascites criteria. We estimated also renal plasma flow (RPF) and glomerular filtration rate by using p-aminohippurate clearance and inulin clearance, respectively. The urine NGAL was measured by ELISA kit (BioPorto Diagnostics, Gentofte, Denmark).

Results: Urine NGAL was significantly different in the two groups of patients, with higher levels noted in the type 1 HRS group (175.23 ± 58.62 vs 24.86 ± 6.11 ng/ml, $p < 0.05$). Patients with type 1 HRS were then divided into two subgroups according to the response to treatment with terlipressin and albumin. Complete responders ($n = 13$) showed a lower value of urinary NGAL (59.72 ± 16.60 vs 275.34 ± 103.08 ng/ml, $p < 0.05$), and of serum creatinine (269.33 ± 18.71 vs 353.50 ± 37.67 $\mu\text{mol/l}$, $p < 0.05$) and an higher level of RPF (214.33 ± 47.45 vs 65.66 ± 11.94 ml/min, $p < 0.0025$) when compared to partial responders and non responders considered as a whole ($n = 15$).

Conclusions: Our results show that type 1 HRS could not be entirely functional in nature but may be associated to tubular damage. This fact may affect to some extent the response of this complication to treatment with terlipressin and albumin in patients with advanced cirrhosis.