High serum human beta-defensin-1 is associated with increased short-term mortality in patients with decompensated cirrhosis

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INTRODUCTION
Defensins are natural antimicrobial peptides, produced by epithelial cells and involved in the defense mechanisms of the mucous membranes, protecting against bacterial translocation (BT). Specifically, human beta-defensin-1 (hBD-1) is expressed in the epithelia of multiple tissues including the digestive tract.

AIM
hBD-1 levels were determined in different subsets of patients with decompensated cirrhosis (DC). In addition, the association of hBD-1 with 30-day mortality was assessed.

METHODS
Eighty-eight patients with DC were divided into 3 groups: 18 with acute-on-chronic liver failure (ACLF) (group 1), 33 with acute decompensation (AD) without ACLF (group 2) and 37 without an acute event (group 3). The hBD-1 was evaluated in serum (N=88) and ascites (N=49) with an enzyme immunoassay (Human BD-1 Mini EIA, Peprotech, London, UK). A control group of 15 healthy controls was also included. There was no statistically significant difference in age and gender between patients and controls.

RESULTS
Liver disease was more severe in patients with ACLF and AD-non ACLF compared to those without an acute event (Table 1). Patients with ACLF had higher white blood cell count, procalcitonin, INR, creatinine, total bilirubin, ALT and AST values compared to those without an acute event (Table 2).

CONCLUSIONS
The serum hBD-1 was associated with systemic inflammatory response and increased in acute-on-chronic liver failure and acute decompensation of liver cirrhosis. Serum hBD-1 was an accurate predictor of short-term mortality in acute-on-chronic liver failure indicating that it may be correlated with organ failure. The serum hBD-1, ACLF and presence of infection were independently associated with mortality.

REFERENCES

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