PRESEPSIN LEVELS IN CIRRHOTIC PATIENTS WITH BACTERIAL INFECTIONS PRESENTED WITH OR WITHOUT ACUTE KIDNEY INJURY

Ioannis Elefsiniotis[1], Stefanos A. Tsakiris[1], Georgia Barla[1], Eftychia Evangelidou[1], Dimitrios Vrachatis[1], Christos Mavrogiannis[1]
[1]Academic Department of Internal Medicine-Hepatogastroenterology, General and Oncology Hospital of Kifisia "Agioi Anargyroi", National and Kapodistrian University of Athens, Greece

BACKGROUND & AIMS

Cirrhotic patients carry an increased risk to develop bacterial infections (BI), sepsis, severe sepsis and septic shock. Patients with liver cirrhosis are two times more likely to die from sepsis than patients without pre-existing liver disease and septic shock in this population is associated with a hospital mortality that may surpass 70% [1]. Serum presepsin has recently aroused as a potential biomarker for sepsis diagnosis. Presepsin is a direct witness of activated monocytes-macrophages in response to pathogens and is able not only to signal an earlier increase compared with CRP and PCT, but also to perform a unique capacity of distinguishing the severity of sepsis [2]. In this study we aimed to evaluate presepsin levels in a group of cirrhotic patients with compensated or decompensated liver disease, without documented BI and to identify potential correlations with the severity of liver disease, using well defined scores for patients with liver cirrhosis, such as Child-Pugh and MELD score. Moreover, we aimed to investigate the degree of change in presepsin levels with the presence of BI, portal hypertension related bleeding (PHRB) and acute kidney injury (AKI) [3].

METHODS

We prospectively evaluated presepsin levels (using PATHFAST chemiluminescent enzyme immunoassay) in 108 consecutive presenting uncomplicated outpatient patients with liver cirrhosis without documented BI. A subgroup of patients presented with documented BI and/or portal hypertension-related bleeding (PHRB) with or without acute kidney injury (AKI). These patients were admitted, hospitalized and re-evaluated at admission.

RESULTS

A total of 108 patients [gender, male/female: 78/30; age, mean (SD): 62.0 (10.6)] with liver cirrhosis were included in the study, 55/108 (50.9%) with compensated cirrhosis, 53/108 (49.1%) with decompensated cirrhosis. Regarding chronic liver disease severity, 71/108 (65.7%) patients presented with Child-Pugh score A, 37/108 (34.3%) with Child-Pugh score B or C, 58/108 (53.7%) with MELD score <10 and 50/108 (46.3%) with MELD score ≥10. The median (IQR) baseline presepsin levels of the entire study population (n=108) were 335.0 (273.8) pg/mL. Patients with decompensated cirrhosis (n=53) exhibited significantly higher baseline presepsin levels [median (IQR): 441.0 (422.5) pg/mL] compared with patients with compensated cirrhosis (n=55) [median (IQR): 262.0 (179.0) pg/mL], p <0.001. Significantly higher baseline presepsin levels were observed in the Child-Pugh B/C group (n=37) [median (IQR): 566.0 (356.0) pg/mL] compared with the Child-Pugh A group (n=71) [median (IQR): 263.0 (190.0) pg/mL)], p <0.001. Additionally, the baseline presepsin levels of cirrhotic patients with MELD score ≥10 (n=50) [median (IQR): 436.5 (365.3) pg/mL] were significantly higher than the corresponding ones of patients with MELD score <10 (n=58) [median (IQR): 262.5 (182.0) pg/mL], p <0.001. During the study period and follow-up a subgroup of patients (18.5%, 20/108) presented with documented BI with or without concomitant portal hypertension-related complication (PHRB, hepatic encephalopathy, AKI). These patients were hospitalized and re-evaluated at their admission. BI was documented in 18 patients (18/20), of whom 6 (6/18) suffered from concomitant PHRB, whereas 2 patients (2/20) were admitted because of PHRB without documented BI. Nine patients admitted with documented bacteremia (five of them with concomitant documented spontaneous bacterial peritonitis), 5 with culture positive urinary tract infection and 4 with documented lower respiratory tract infection. Presepsin levels at admission (n=20) [median (IQR): 943.0 (1593.0) pg/mL] were remarkably higher compared with their baseline levels [median (IQR): 528.0 (480.0) pg/mL], p <0.001. Ten of the 20 hospitalized patients (50%) developed AKI and all of them had a fatal outcome. The patients in the AKI group (n=10) exhibited significantly higher presepsin levels, both at baseline [median (IQR): 714.0 (637.0) pg/mL] and at hospital admission [median (IQR): 1616.0 (1470.0) pg/mL] compared with the cirrhotic patients in the non-AKI group (n=10) [baseline: median (IQR): 404.0 (419.0) pg/mL and at admission: median (IQR): 616.5 (664.0) pg/mL], p<0.05.

CONCLUSIONS

Plasma presepsin levels are elevated in uncomplicated cirrhotic patients, especially in those with decompensated liver cirrhosis and Child-Pugh stages B/C and/or high MELD scores. BI and/or PHRB further increase the presepsin levels especially in patients with advanced liver disease who developed AKI during their hospitalization.

REFERENCES

[1] Bacterial infections in cirrhosis: a position statement based on the EASL Special Conference 2013, Jalan et al., J Hepatol, 2014.
[2] Presepsin (sCD14-ST), an innate immune response marker in sepsis, Chenevier-Gobeaux et al., Clin Chim Acta, 2015.
[3] Diagnosis and management of acute kidney injury in patients with cirrhosis: revised consensus recommendations of the International Club of Ascites, Angeli et al., J Hepatol, 2015.

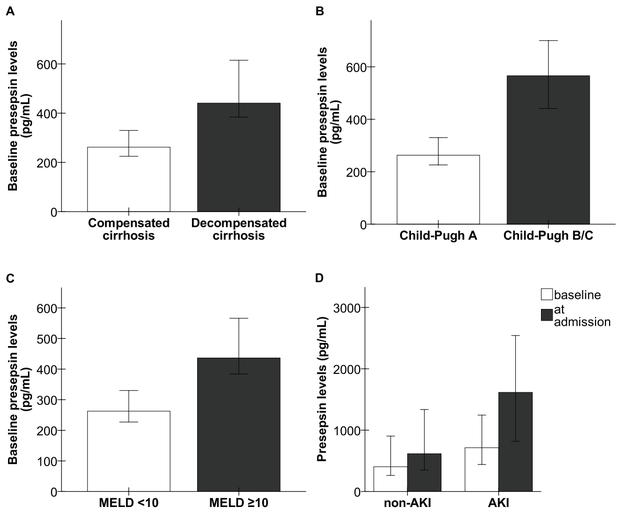

Figure 1. Graph of presepsin levels. (A) Patients with decompensated liver cirrhosis exhibited higher baseline presepsin levels. (B) Cirrhotic patients with Child-Pugh class B/C exhibited higher baseline presepsin levels compared with the Child-Pugh class A group. (C) Cirrhotic patients with MELD score ≥10 exhibited higher baseline presepsin levels compared with the MELD <10 group. (D) Cirrhotic patients who developed AKI exhibited higher presepsin levels both at baseline and at admission compared with the non-AKI group.