

Piastrine: Alterazioni qualitative o quantitative

INVESTIGATING THE MEAN PLATELET VOLUME OF PATIENTS WITH LIVER CIRRHOSIS AND CHRONIC LIVER DISEASE: A CASE-CONTROL STUDY

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Background and Aims. Haemostasis is rebalanced in patients with liver cirrhosis, but few and contradictory data are available regarding the platelet size. We designed this study to investigate the mean platelet volume (MPV) in patients with liver cirrhosis.

Methods. All patients with liver cirrhosis and chronic liver disease were consecutively enrolled among the outpatients followed up at the Hepatology outpatient clinic of hospital San Paolo (Milan). Control subjects were enrolled among healthcare workers of hospital San Paolo. All subjects had at least one complete blood count (CBC) performed at the laboratory of hospital San Paolo between 2015 and 2020 (normal range for MPV: 9.3-12.6 fL). All CBCs were performed with the same instrument during this time. The primary outcome was the comparison of the MPV in the three groups. Additionally, the MPV was evaluated in cirrhotic patients classified according to their Child-Pugh score at the time of blood withdrawal and history of previous decompensation (which we defined as any among ascites, portosystemic encephalopathy, variceal bleeding, spontaneous bacterial peritonitis and portal vein thrombosis). Finally, in patients with liver cirrhosis, we evaluated the correlations between platelet count and the MELD-Na score at the time of blood withdrawal with the MPV.

Results. We enrolled 60 controls (32 females, median age 47 years, IQR 39-52), 120 patients with chronic liver disease (63 females, 63 years, IQR 53-73) and 120 patients with liver cirrhosis (52 females, 67 years, IQR 56-75). Cirrhotic patients had significantly lower median platelet counts ($122x10^9$/L, IQR 78-175) compared to chronic liver disease patients ($220x10^9$/L, IQR 185-260) and controls ($251x10^9$/L, IQR 226-277) (p<0,0001). Cirrhotic patients' Child-Pugh score was A for 59 subjects, B for 37 and C for 24. Sixty cirrhotic patients (50%) had a history of previous decompensation. The median MPV was significantly higher both in cirrhotic patients (11.3 fL, IQR 10.4-12.3) and chronic liver disease patients (11.0 fL, IQR 10.2-11.8) compared to controls (10.5, IQR 9.9-11.2; p<0.0001 and p=0.01, respectively). The proportion of cirrhotic patients with a MPV higher than the upper limit of the reference range was 13% (95% CI, 8-20), significantly higher compared to controls (0%, 95% CI 0-6, p=0.0072). Neither the median MPV nor the proportion of patients with a MPV higher than the reference range significantly differed across Child-Pugh A, B and C cirrhotic patients. The proportion of cirrhotic patients with a MPV higher than the reference range was non-significantly higher in patients with previous decompensation (20%, 95% CI 11-32 vs 7%, 95% CI 2-16; p=0.0575). There was an inverse correlation between the platelet count and the MPV (r=-0.36, p<0.0001) and a mild direct correlation between the MELD-Na score and the MPV (r=0.21, p=0.02) in cirrhotic patients.

Conclusions. Taken together, our results are in keeping with the notion that increased platelet size signals compensatory platelet production in response to consumption and/or sequestration, but in liver cirrhosis, contrarily to chronic liver disease, the MPV appears to plateau, with less pronounced variations that often fail to reach statistical significance as liver disease progresses. This may reflect the coexistence of pathomechanisms with opposite effects on platelet size, including inflammation, hypersplenism, decline in TPO synthesis, in patients with more advanced disease.