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The Neutrophil-To-Lymphocyte Ratio (NLR) and the Platelet-To-Lymphocyte Ratio (PLR) Prognostic Values in Decompensated Cirrhosis

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Introduction:

Patients with decompensated cirrhosis present a state of chronic inflammation that results from an abnormal bacterial translocation and contributes to the development of circulatory dysfunction and failure.

Scoring systems such CHILD PUGH, chronic liver failure-sequential organ failure assessment (CLIF-SOFA) can help predict prognosis and consequently select patients requiring admission to intensive care unit.

Note: Decompensated cirrhosis may be a term that doctors use to explain the complications of advanced disease. People with compensated cirrhosis often do not have any symptoms because their liver remains properly functioning. Later liver function reduces, it can become decompensated cirrhosis. Decompensated cirrhosis is defined as an acute deterioration in liver function in a patient with cirrhosis and is characterized by jaundice, ascites, hepatic encephalopathy, hepatorenal syndrome or variceal haemorrhage.

Clinical trial data demonstrate that within the population of persons with decompensated cirrhosis, most patients receiving direct-acting antiviral (DAA) therapy experience improvement in clinical and biochemical indicators of disease between baseline and post treatment week twelve, includes patients with CTP a class C cirrhosis. Real-world data comparing DAA response rates demonstrate that patients with cirrhosis and hepatoma (HCC) have lower SVR rates than cirrhotics without HCC (Prenner, 2017); (Beste, 2017). In a large VA study including sofosbuvir, ledipasvir/sofosbuvir, and paritaprevir/ritonavir/ombitasvir plus dasabuvir regimens (with and without ribavirin), overall SVR rates were 91% in patients without HCC versus 74% in those with HCC (Beste, 2017). After adjusting for confounders, the presence of HCC was associated with a lower likelihood of SVR (AOR=0.38). Whether this lower SVR are often overcome with an extended duration of therapy is unknown.

NLR is measured by dividing the amount of neutrophils by the amount of lymphocytes. NLR could also be an indicator of systemic inflammation, as neutrophils and lymphocytes are thought to be significant in tumour immunology and inflammation. Inflammation plays a significant role in the proliferation, angiogenesis, and metastasis of cancer cells and is important in the development and progression of the disease.[2,3] Even when white blood cell count is in normal range, NLR has been demonstrated to play a predictive role in the prognosis of chronic and acute inflammatory processes. The mean NLR of the prostate cancer group was significantly higher than that of the benign prostatic hypertrophy (BPH) group (p=0.002). The mean NLR of the prostatitis group was higher than that of both the prostate cancer and BPH groups (p=0.0001). The mean NLR of the Gleason score (GS) 8-10 group was higher than that of the GS 7 and GS 5-6 groups. The authors conclude that NLR was found to vary with regard to histology of prostate biopsy and higher GS was associated with higher NLR in patients with prostate cancer}

This study aimed to determine whether or not the neutrophil-to-lymphocyte ratio (In medicine neutrophil to lymphocyte ratio (NLR) is used as a marker of subclinical inflammation. It is calculated by dividing the number of neutrophils by number of lymphocytes, usually from peripheral blood sample, but sometimes also from cells that infiltrate tissue, such as tumor.) and the platelet-to-lymphocyte ratio (PLR) have a prognostic value in patients with decompensated cirrhosis.

Methods: We conducted a descriptive and analytic retrospective study at the gastro intestinal department at Charles Nicolle hospital, Tunis, Tunisia. We have included all the patients' admitted for decompensated cirrhosis between September 2015 and October 2016. The baseline characteristics, complications of portal hypertension, laboratory values, Child–Pugh class, Model for End-stage Liver Disease (MELD), NLR and PLR were assessed. The primary outcomes were 3 month mortality and prognostic factors.

Results: In total, 92 patients met our inclusion criteria. Overall, the 3 month mortality rate was 15% (14 patients). Hepatic encephalopathy, documented bacterial infection, white blood cell count, MELD score, CLIF–SOFA score and NLR and PLR were significantly associated with mortality; the respective P value: P value: (P= 0.021, P=0.001, P=0.018, P=0.026, P=0.013, P= 0.001, P=0.001). The area under the ROC curves of respectively NLR and PLR were 0.633 (CI 95%;0.459-0.808) and 0.600 (CI 95%; 0.423-0.777). The respective thresholds were 4.96 (sensitivity: 57%; specificity 61%) and 85 (sensitivity 64% specificity 53%). However, the analysis of ROC curves of the other prognostic factors showed that only the MELD score was more discriminative.

Conclusions: The NLR and PLR in decompensated cirrhosis are independent factors for 3month mortality.

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