

EVALUATION OF DIFFERENT SYSTEMIC INFLAMMATORY MARKERS, CRP AND TROPONIN LEVELS IN PATIENTS INITIATED ON CLOZAPINE TREATMENT IN A TRAINING AND RESEARCH HOSPITAL

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BACKGROUND AND AIM: One of the serious adverse effects of clozapine use is acute myocarditis. In our study, we aim to compare different systemic inflammatory markers, and CRP and Troponin levels, which are important markers for the risk of acute myocarditis, in patients who started clozapine treatment over a two-year period at a training and research hospital. Based on the comparison, we aim to identify statistically significant markers for risk assessment in clinical practice.

METHODS: A retrospective analysis was conducted on 92 patients who started clozapine treatment. Data on CRP (C-reactive protein), troponin, HDL (high-density lipoprotein), albumin, lymphocytes, neutrophils, platelets, monocytes, CALLY (Superiority of CRP Albumin Lymphocyte Index) (Albumin* $Lymphocyte/CRP$), SIRS (Systemic Inflammatory Response Syndrome) (Platelet* $Neutrophil/Lymphocyte$), monocyte/lymphocyte ratio, neutrophil/lymphocyte ratio, platelet/lymphocyte ratio and monocyte/HDL ratio (MHR), along with sociodemographic characteristics, were extracted from the hospital system. Inflammatory markers were assessed at weeks 0, 1, 2, and 4 after initiating clozapine treatment, and statistical analyses were performed to determine significant changes. The study was approved by the ethics committee (Protocol Code: 2024-TBEK 2024/09-08).

RESULTS: Among the 92 participants, 71.7% were male and 28.3% were female, with a mean age of 38.32 (SD = 10.60) years. A history of chronic disease was present in 26.1% of participants while 73.9% had no chronic illness. A significant increase was observed in troponin ($p < 0.001$), platelet ($p = 0.003$) and albumin ($p = 0.015$) levels. However, no statistically significant changes were detected in CRP, HDL, neutrophils, lymphocytes or monocytes. According to Friedman test results, the monocyte/HDL ratio was 0.01 (SD=0.01) at baseline and increased to 0.03 (SD=0.01) by week 4, with this increase being statistically significant ($p = 0.024$). Other changes, apart from MHR, were not statistically significant. None of the recorded inflammatory indexes was found to be a predictor of troponin increase.

CONCLUSIONS: Recent studies suggest that MHR, a non-invasive inflammatory marker, is a novel prognostic factor for cardiovascular diseases. Clozapine use for 12-14 days increases pro-inflammatory cytokines, boosting inflammation. Inflammation and oxidative stress are key in myocarditis development. While MHR may aid in assessing acute myocarditis, the impact of clozapine on the immune system requires further investigation.

Keywords: Clozapine, inflammatory markers, monosit/HDL, myocarditis