

Serum glial fibrillary acidic protein (GFAP), SERPINA3, IL-33 levels in patients with schizophrenia and their relationship with treatment resistance

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T. Özdemir Tekeli^I, S. Elgün Ülkar^I, H. Devrimci Özgüven^{II}, M.Ç. Aydemir^{II}, B. Yıldırım^{II}, A.H. Bayraktar^{II}

^IAnkara University Faculty of Medicine, Department of Medical Biochemistry, Ankara, Türkiye, ^{II}Ankara University, Faculty of Medicine, Department of Psychiatry, Ankara, Türkiye

Recent studies have highlighted the inflammation theory of schizophrenia, suggesting a potential association between the disorder and elevated inflammatory markers. However the relationship between inflammatory markers and treatment-resistant schizophrenia (TRS) is unclear. Neuroinflammation is characterized by activation of glial cells and GFAP is a marker of reactive astrocytes. Reactive astrocytes, the primary source of SERPINA3, also contain abundant IL-33, which mediates CNS inflammation and may play a role in schizophrenia pathophysiology. This study aims to investigate the relationship between the treatment response status of schizophrenia patients and the underlying inflammatory condition. Our study includes TRS patients, schizophrenia patients in the remission phase, and healthy control groups. Serum GFAP, SERPINA3, IL-33 levels were analyzed using the ELISA method, and NLR, MLR, PLR values obtained from the complete blood count were calculated. Correlations between PANSS total/subscale scores and these markers were analyzed. In our study, GFAP concentrations were significantly higher in the TRS group than in the control group. SERPINA3 concentrations, neutrophil levels and NLR were significantly higher in both TRS and remission groups compared to controls. Platelet levels were significantly higher in the TRS group compared to the control group and remission group. In the TRS group, MLR showed a positive correlation with PANSS positive score, while NLR correlated negatively with PANSS negative score. ROC analysis was performed to evaluate GFAP's ability to discriminate between TRS and remission schizophrenia patients, yielding an AUC of 0.676 ($p < 0.05$). Our study suggests that an increased inflammatory response may play a role in the pathogenesis of schizophrenia. The higher levels of inflammatory markers observed in treatment-resistant patients suggest a more severe inflammatory response, indicating that the degree of inflammation may be associated with treatment response status.