

Introduction: It has been suggested that the activation of systemic inflammatory response in depression is associated with inflammatory changes in the brain (neuroinflammation) and may reflect the severity of the clinical symptoms in patients.

Objectives: To study the relationship between clinical and immune parameters in patients with endogenous depressive disorders for the possible use of these indicators for diagnostics of these conditions.

Methods: Patients with bipolar affective disorder (group 1) and recurrent depressive disorder (group 2) (F31, F32, F33) were examined before the therapy. Mentally healthy age- and gender-matched persons were investigated as controls. The severity of depressive symptoms was assessed by HDRS. The activity of inflammatory indicators (leukocyte elastase (LE) and 1-proteinase inhibitor (α 1-PI)), as well as the level of autoantibodies (AB) to S-100B and MBP, were measured in plasma.

Results: Group 1 was characterized by an increase of LE and α 1-PI activity in comparison with the control group ($p < 0.001$; $p = 0.002$) and group 2 ($p < 0.05$). No significant difference in AB to neuroantigens was found. Group 2 was distinguished by the increase in activity of the inflammatory indicators ($p < 0.01$; $p < 0.05$) as well as the autoimmune reactions to neuroantigens compared with control one ($p = 0.03$). The correlations between complex assessment of the immune system and the severity of depressive symptoms in both groups were revealed ($\chi^2 = 6.1$; $p = 0.013$; $\chi^2 = 4.8$; $p = 0.05$).

Conclusions: Revealed correlations suggest that inflammatory markers are involved in the pathogenesis of endogenous depressive disorders and can be used as an additional differential diagnostics criterion for the assessment of the clinical state of patients.

Disclosure: No significant relationships.

Keywords: endogenous depressive disorders; leukocyte elastase; α 1-proteinase inhibitor; autoimmune reactions

EPV1111

Psychiatric symptoms in autoimmune encephalitis. A case report.

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doi: 10.1192/j.eurpsy.2022.1805

Introduction: Early stages of autoimmune encephalitis (AE) often present cognitive and neuropsychiatric symptoms such as personality change, irritability, anxiety, depression, behavioral disorders, hallucinations, disorientation, sleep-wake cycle reversals, ...). Thus often these cases are first treated as psychiatric disorders.

Objectives: A literature review throughout a case report presentation.

Methods: We present the case of a 25-year old female with a medical history of iron-deficiency anemia who arrives at the emergency service. She presents the following one week of evolution clinical picture: complex auditory hallucinations, behavioral disturbances, sleep disorder and short term memory impairment. Neurological examination, LP and cranial CT are all normal. CSF analysis has no abnormalities. Thus she entered the psychiatric ward. There she was

treated with neuroleptics with no improvement of symptoms presenting a severe psychomotor agitation and language impairment. After neurology interconsultation AE is suspected.

Results: She was performed an EEG (left temporal epileptiform activity), CSF (inflammatory pattern), MRI (bilateral temporal lobe hyperintensity). Suspecting limbic encephalitis the presence of anti-NMDAR antibodies was tested, which turned out to be positive. First she was treated with corticotherapy with mild results. Then she was treated with intravenous immunoglobulin improving significantly.

Conclusions: Anti-NMDAR encephalitis is usually a multistage illness. Early in the course of disease psychiatric manifestations are not rare. Therefore the proper diagnosis and approach of AE may require a highly organized assessment, starting with detailed history and physical examination and an appropriate testing to exclude other possible relevant pathologies.

Disclosure: No significant relationships.

Keywords: autoimmune encephalitis; neuroleptics; auditory hallucinations; immunoglobulin

EPV1113

Immune system and schizophrenia

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doi: 10.1192/j.eurpsy.2022.1806

Introduction: Schizophrenia affects approximately 1% of the world population, having a devastating impact not only in patients but in all society. As a result, it has been subject of extensive investigation and the presence of certain genes was associated with an increased risk of developing schizophrenia. However, the presence of these genes is not sufficient, therefore, other factors are necessarily involved. Observation of the association between schizophrenia and inflammatory states of the Central Nervous System led to the hypothesis that a dysfunction of the immune system may play a central role in this process.

Objectives: In this work we intend to make a brief review of the existing literature related to the immunological theory of schizophrenia.

Methods: A bibliographic research was conducted in Medline library using the following terms: "schizophrenia and immune system"; "schizophrenia and inflammation" and "schizophrenia and neuroinflammation".

Results: The survey results reveal increasing evidence of the key role of the immune system in schizophrenia. Several studies show benefits of treatment with anti-inflammatory drugs in patients at an early stage of the disease. In the same way, it was verified that pro and anti-inflammatory cytokines influence glutamatergic transmission and tryptophan metabolism. Furthermore, the decrease in microglial activity appears to have a beneficial effect on schizophrenia.

Conclusions: Future will say if neuroimmunology mechanisms are primary or a secondary consequence in Schizophrenia. Recent discoveries in this area are encouraging and open the possibility of new therapeutic targets and new therapeutic approaches to this disease.