Introduction: Major depressive disorder (MDD) is a prevalent neuropsychiatric condition influenced by genetic, environmental, and inflammatory factors. Alterations in T helper (Th) cell subsets, including Th1, Th2, Th17, and regulatory T cells (Tregs), have been implicated in the immune dysregulation observed in MDD, linking the adaptive immune system to depression. However, the link between these T cell subsets, MDD severity and their connection with childhood trauma (CT), a major risk factor for depression, remain incompletely understood.
Objectives: In this project we characterized peripheral blood T cell subsets and their association with CT and MDD.
Methods: In this study, we performed multiparameter flow cytometry analysis on peripheral blood immune cells from a subgroup of the FOR2107 cohort. T cell differentiation is characterized by their phenotypic markers. Age- and gender- matched groups of 46 individuals with depression and 55 healthy controls (HC) were included, both with and without a history of childhood trauma. Depression severity was assessed using the Hamilton Rating Scale for Depression), (HAM-D21), and CT was evaluated via the Childhood Trauma Questionnaire (CTQ). Correlational analyses examined relationships between T cell subtype frequencies, depression severity, and CT subtypes.
Results: The analysis revealed an increased frequency of circulating Th17 cells in patients with MDD compared to healthy controls. In participants with a history of CT, the overall frequency of $CD3^+$ T cells was decreased, while Th2 cells and Treg cell frequencies were reduced when compared to individuals without CT. Frequencies of specific T cell types correlate with CT subtypes, especially in depressive patients. Th1, effector memory T cells (Tem) and central memory T cells (Tcm) showed a positive correlation with physical abuse, while Treg cells correlated with the overall CTQ score and emotional neglect.
Conclusions: Our findings indicate dysregulations of the adaptive immune system in CT and MDD, characterized by alterations in peripheral blood Th17, Th2, and Treg cells. These data highlight the influence of early life adversity on immune function and its potential contribution to the pathophysiology of depression.

Disclosure of Interest: None Declared

EPV1523

Strike three, you're out - when a primary psychosis just doesn't cut it

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Introduction: Autoimmune psychosis has gained increased recognition as a distinct entity and is known to mimic variants of primary psychosis, typically presenting with an acute onset of polymorphic psychotic symptoms. We describe a case of probable autoimmune psychosis in a young patient who experienced a severe first psychotic episode.
Objectives: Reflection over the diagnostic challenges of autoimmune psychosis.
Methods: Clinical case report.
Results: A 19-year-old male patient with no relevant medical history was admitted to the psychiatric ward due to a first psychotic episode with a peracute onset. The episode peaked with severe confusion, disorientation, and disorganized behavior, leading to his referral to the emergency room. This episode was characterised by delusional ideation with mystical, self-referential, and persecutory themes, complex auditory-verbal hallucinations, and marked negative and cognitive symptoms (including affective blunting, social withdrawal, apathy, alogia, impaired attention, decreased social cognition, and reduced speed of cognitive processing). The analytical study, substance screening, and brain CT upon admission were normal, leading to the assumption of a primary psychotic disorder. Antipsychotic therapy was initiated with progressive titration (risperidone, cariprazine, and clozapine), yet there was no significant improvement. Given the severe presentation and treatment resistance, a neurological examination was requested, which revealed no focal signs. A comprehensive laboratory workup showed positive ANAs, anti-recoverin antibodies, and hypocomplementaemia (C3 and C4). No significant abnormalities were observed in the brain MRI. CSF analysis revealed slight protein elevation (55 mg/dL) without pleocytosis, oligoclonal bands, or antibodies. EEG indicated mild to moderate encephalopathy with FIRDA bursts and focal paroxysmal activity in the left temporo-parieto-occipital region. Brain PET-FDG showed no significant abnormalities. Serum and CSF neurofilament levels were normal. Full-body CT and PET-FDG scans were also unremarkable. Given the findings, autoimmune psychosis was assumed. Treatment with IV immunoglobulin (30 g) and methylprednisolone (1 g for 5 days) was administered. The case was discussed in a multidisciplinary meeting, and a regimen of daily prednisolone (10 mg) was chosen. At follow-up, the patient showed slight improvement, with mitigation of the positive symptoms.
Conclusions: Psychosis that does not respond to antipsychotic treatment and presents with atypical signs should raise suspicion of secondary immune-mediated schizophreniform psychosis. However, the challenge lies in identifying these patients, selecting appropriate diagnostic tests, and establishing criteria for implementing treatment.

Disclosure of Interest: None Declared

EPV1524

Heterogeneity of juvenile depression with different risks of developing psychosis according to immunological blood parameters

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Introduction: One of the mechanisms underlying the pathogenesis of mental disorders, including endogenous depression, is systemic inflammation. It is of interest to study the immunological aspects of the early stages of endogenous disorders and identify subgroups of patients with immunotypes that characterize a high risk of developing the first psychotic episode.
Objectives: Comparative analysis of the spectrum of inflammatory markers in patients with a juvenile depression with high and low risk of developing psychosis.

Methods: The study involved 98 women 16 to 25 years (20.9 ± 5.14 years) with depression within the framework of various nosologies (F31.3-4; F33.0-1; F60.0-9; F21.3-4; F20.01-2; F25.1). Two groups of patients without a history of psychosis were identified: group 1 (n = 47) - without symptoms of psychosis risk, group 2 (n = 51) - with depression associated with psychopathological symptoms of psychosis risk. The control group consisted of 42 healthy women of the corresponding age. The severity of depressive symptoms was assessed using the HDRS-21, the severity of negative and positive symptoms was determined using the SANS and SAPS. In group 2, the severity of attenuated positive symptoms was determined using the SOPS. The activity of the leukocyte elastase (LE) and α1-proteinase inhibitor (α1-PI), as well as the level of autoantibodies (AB) to S100B and MBP, were determined in plasma.
Results: The groups were characterized by a statistically significant increase in both LE and α1-PI ($p<0.05$), and the level of AB compared to the control ($p<0.05$), but no significant differences were found. In group 1, clinical and biological correlations were found between LE activity and the total score on the SANS ($r=0,44$, $p=0.002$). In group 2, a negative correlation was found between LE activity and the age of onset of the disease ($r=-0.3$, $p=0.046$).
The clustering of patients by LE activity and their distribution by immunological groups showed that 29.4% and 27.5% of patients in groups 1 and 2, respectively, were characterized by a high level of inflammatory markers and the absence of an autoimmune component to neuroantigens, which is a sign of a more favorable course of the pathological process. On the contrary, 70.6% and 72.5% of patients in groups 1 and 2, respectively, were characterized by the type of inflammatory response associated with an increase in the level of AB and varying degrees of insufficiency of the functional activity of neutrophils, which is considered an unfavorable factor that aggravates the course of the disease.
Conclusions: Comparison of the spectrum of inflammatory markers in juvenile depression with different risk of developing psychosis indicate their significant immunological heterogeneity. The immunotype characterized by a high level of AB and insufficient LE activity can presumably be considered as a predictor of the risk of developing psychosis.

Disclosure of Interest: None Declared

EPV1525

Quantitative and functional characteristics of monocytes and neutrophils in patients with treatment-resistant schizophrenia

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Introduction: Despite significant progress in the treatment of schizophrenia, the number of patients with schizophrenia who do not respond to treatment remains constant. Identification of biomarkers of therapeutic resistance in the schizophrenia can help in early prediction of these conditions, as well as in the development of new approaches to treatment. Inflammation is considered as one of the possible mechanisms involved in the development of the pathological process in the formation of resistance to therapy in schizophrenia. The main cells of innate immunity, neutrophils and monocytes, are involved in the implementation of the inflammatory response.
Objectives: To compare the subpopulation composition of monocytes and the level of other inflammatory markers in patients with treatment-resistant schizophrenia and in the control group.
Methods: The study included 17 men with treatment-resistant schizophrenia (TRS) (27.0±8.0 years) and 15 healthy individuals without signs of mental and inflammatory diseases. The relative content of neutrophils and monocytes in the blood, as well as the ratio of monocyte subpopulations, estimated by the expression level of CD14 and CD16 receptors, were determined by flow cytofluorometry. The functional activity of neutrophils was determined spectrophotometrically by the activity of leukocyte elastase in plasma. The level of autoantibodies to S100B in plasma was estimated by ELISA.
Results: A significant increase in the relative content of monocytes ($U=28.0$, $p<0.01$) and a decrease in neutrophils ($U=35.0$, $p=0.036$) were found in TRS patients compared to the controls. An increase in a proportion of the "transitional" CD14+CD16− subpopulation ($U=61.5$, $p=0.04$) and a decrease in the "classical" CD14++CD16− subpopulation ($U=60.5$, $p=0.036$) were accompanied by the proportion of "intermediate" inflammatory CD14++CD16+ and "non-classical" CD14+CD16+ subpopulations that did not differ from controls. A moderate increase in leukocyte elastase activity ($U=34.0$, $p=0.001$) and a high level of S100B autoantibodies ($U = 55.0$, $p = 0.02$) were found in blood plasma of patients. The proportion of "intermediate" CD14++CD16+ monocytes was negatively correlated with the level of autoantibodies to S100B ($r= -0.55$, $p=0.021$). It should be noted that this spectrum of immune parameters differs from the corresponding profile that we identified in patients with schizophrenia who responded to treatment. The main differences concern the proportion of "intermediate" monocytes, the relative content and functional activity of neutrophils.
Conclusions: The identified quantitative and functional characteristics of monocytes and neutrophils in patients with TRS indicate the possible involvement of the cellular component of immunity in the development of resistance to treatment and may be associated with the severity of the disease in a long-term pathological process in the brain.

Disclosure of Interest: None Declared

Psychopathology

EPV1526

Distinguishing Obsessive-Compulsive Symptoms in Schizophrenia-Spectrum Disorders and Obsessive-Compulsive Disorder: The Role of Basic Self Disturbances

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Introduction: Obsessive-compulsive symptoms (OCS) are frequently observed in both obsessive-compulsive disorder (OCD) and schizophrenia-spectrum disorders (SSD), creating significant diagnostic challenges. Historically, Karl Jaspers defined "true obsessions" as a struggle against intrusive ideas that appear nonsensical and "alien" to the personality, demarcating this concept from delusions and overvalued ideas, in which cases the person