

Table 2. The most common clusters of Neuropsychiatric features

NPI-Q	Positive Thyroid Autoantibodies (n=33)
Delusion	15 (45.4%)
Agitation/Aggression	14 (42.4%)
Irritability	14 (42.4%)
Motor abnormality	14 (42.4%)
Sleep disorder	15 (45.4%)
Appetite/Eating	14 (42.4%)

Conclusions: In particular, in a subset of schizophrenia spectrum disorder or affective disorder patients with positive anti-thyroid antibodies may indicate autoimmunity, especially in cases where catatonic symptoms dominate the clinical presentation.

Disclosure of Interest: None Declared

EPP0434

Features of the spectrum of immune markers in patients with juvenile depression with clinically high risk of psychosis

S. A. Zozulya*, M. A. Omelchenko, I. N. Otman, Z. V. Sarmanova, V. V. Migalina, V. G. Kaleda and T. P. Klyushnik

Mental Health Research Centre, Moscow, Russian Federation

*Corresponding author.

doi: 10.1192/j.eurpsy.2024.583

Introduction: Identification of biomarkers associated with the risk of psychosis manifestation in juvenile patients with depression may contribute to a better understanding of the pathogenesis of mental disorders and early diagnosis.

Objectives: To determine the level of pro-inflammatory and anti-inflammatory cytokines and other inflammatory indicators in the plasma of juvenile patients with depression and clinically high risk of psychosis, and to study the correlation of these markers with the severity of psychopathologic symptoms.

Methods: 80 young men aged 16-24 years with the first depressive episode (F32.1-2, F32.38, F32.8) were examined. Based on the severity of attenuated psychotic symptoms (APS) in the structure of depression according to the SOPS scale, all patients were divided into two groups - with clinically high risk of psychosis (n=58) and with depression without APS (n=22). The HDRS-21 and SANS scales were also used for psychometric assessment. Serum level of cytokines TNF- α , IL-6, IL-8, IL-10, TNF- α /IL-6 ratio, TNF- α /IL-10 ratio, leukocyte elastase (LE) and α 1-proteinase inhibitor (α 1-PI) activity, C-reactive protein (CRP) concentration, and the level of autoantibodies to S-100B protein were determined.

Results: Both groups of patients showed a high level of inflammation assessed by LE and α 1-PI activity ($p > 0.05$). Significantly higher level of IL-6 ($p = 0.03$), CRP concentration ($p = 0.026$) and TNF- α /IL-10 ratio ($p = 0.032$) were found in patients with clinically high risk of psychosis. This group was also characterised by high level of autoantibodies to the S-100B protein compared to patients with depression without APS ($p = 0.048$).

In the high clinical risk group, correlations were found between the SOPS positive subscale score and the level of TNF- α ($R = 0.32$, $p = 0.017$), IL-8 ($R = -0.3$, $p = 0.034$), TNF- α /IL-6 ratio ($R = 0.30$, $p = 0.021$) and TNF- α /IL-10 ratio ($R = 0.32$, $p = 0.014$). The SOPS negative subscale score correlated with CRP concentration ($R = 0.3$, $p = 0.043$). The SOPS total score correlated with TNF- α /IL-10 ratio ($R = 0.31$, $p = 0.021$). In this group of patients, the level of IL-10 was found to correlate with the duration of the disease ($R = 0.48$, $p < 0.001$). In patients with depression without APS, the level of IL-6 was correlated with the severity of depression according to the HDRS scale, and the level of TNF- α was associated with the duration of the depressive episode ($R = 0.51$, $p = 0.029$).

Conclusions: The obtained results confirm the involvement of inflammation in the development of juvenile depression. Qualitative and quantitative characteristics of the spectrum of immune markers and the cytokine profile, and correlations with the severity of psychopathologic symptoms were revealed in patients with clinically high risk of psychosis.

Disclosure of Interest: None Declared

EPP0435

Immunological predictors of rhythmic transcranial magnetic stimulation (rTMS) efficiency in patients with treatment-resistant schizophrenia

S. A. Zozulya*, A. N. Pomytkin, D. V. Tikhonov, V. G. Kaleda and T. P. Klyushnik

Mental Health Research Centre, Moscow, Russian Federation

*Corresponding author.

doi: 10.1192/j.eurpsy.2024.584

Introduction: Model and clinical studies demonstrate the efficiency of rhythmic transcranial magnetic stimulation (rTMS) in diseases associated with neuroinflammation. The therapeutic potential of rTMS is related to modulation of neuroplasticity in the CNS, activation of neurogenesis and reduction of neuroinflammatory processes. Presumably, one of the factors that determines the efficiency of rTMS can be the features of the immune status of patients.

Objectives: To reveal the features of the spectrum of inflammatory markers in patients with treatment-resistant schizophrenia with different efficiency of rTMS.

Methods: 31 male patients aged 16 to 47 years (mean age 29.9 ± 8.4 years) with treatment-resistant schizophrenia who developed a first psychotic episode in adolescence (19-25 years) were examined. The course of rTMS was conducted for 3 weeks (15 sessions). Depending on the dynamics of clinical and psychometric parameters after the course of rTMS, the patients were divided into three groups: group 1 - with worsening of clinical condition (n=8); group 2 - without therapeutic effect (n=12); group 3 - with good therapeutic response (n=11). Before rTMS, leukocyte elastase (LE) and α 1-proteinase inhibitor (α 1-PI) activity, and the levels of autoantibodies to S-100B protein and myelin basic protein (MBP) in the plasma of patients were determined. The parameters of 18 healthy male donors without clinical signs of psychiatric and somatic pathology were used as controls.

Results: All groups of patients were characterised by moderate and high levels of immune system activation, determined by a complex of inflammatory and autoimmune markers. At the same time, the high level of immune system activation in patients with low MTR efficiency was associated with low LE activity in plasma (within the reference range or below the lower limit - 200.6 (168.5- 220.3) nmol/min·mL), which was not consistent with the overall level of inflammation. This group of patients also showed high levels of antibodies to MBP compared to control values ($p < 0.05$). The low LE activity can be explained by the transmigration of neutrophils from the blood to the brain due to a critical increase in the permeability of the blood-brain barrier, which is largely controlled by LE.

Conclusions: The study confirmed the participation of immune mechanisms in the formation of therapeutic resistance in schizophrenia and revealed the characteristics of the spectrum of immune markers in patients with low efficiency of rTMS.

Disclosure of Interest: None Declared

EPP0436

Connection of molecular and cellular components of the immune system in endogenous psychoses with depressive-delusional symptoms

S. A. Zozulya*, Z. V. Sarmanova, I. N. Otman, I. V. Oleichik, E. G. Cheremnykh, T. A. Prokhorova, O. K. Savushkina, I. S. Boksha, E. B. Tereshkina and T. P. Klyushnik

Mental Health Research Centre, Moscow, Russian Federation

*Corresponding author.

doi: 10.1192/j.eurpsy.2024.585

Introduction: The data of current research indicate the participation of systemic inflammation in the pathogenesis of endogenous psychoses. Changes in the level of peripheral immune markers are associated with the development of neuroinflammation and correlate with the severity of psychopathological symptoms detected in patients. However, the association between individual components of the immune system involved in the development of endogenous psychosis remains poorly understood.

Objectives: To study the connection between molecular and cellular components of the immune system in women with endogenous psychoses with depressive-delusional symptoms.

Methods: 32 female patients aged 23 [17; 36] years with endogenous psychoses within different nosologies (F20, F21, F31, depressive-delusional conditions) and 17 women without clinical signs of psychiatric pathology were examined. The activity of leukocyte elastase (LE), $\alpha 1$ -proteinase inhibitor ($\alpha 1$ -PI), the proportion of four subpopulations of monocytes (classical CD14+CD16-, intermediate CD14++CD16+, nonclassical CD14+CD16+ and transitional CD14+CD16-) in plasma, activity of cytochrome-c oxidase (COX), glutamate dehydrogenase (GDH), glutathione s-transferase (GST) and glutathione reductase (GR) in platelets and functional activity of complement system (faCS) in serum were determined. The PANSS scale was used to assess the severity of psychopathological symptoms.

Results: Increased activity of the inflammatory markers LE ($p = 0.033$) and $\alpha 1$ -PI ($p = 0.02$) was found in the plasma of the patients. Increased percentage of pro-inflammatory monocytes

(intermediate and transient) in plasma ($p = 0.003$) was confirmed by negative correlations between CD14++CD16- and CD14++CD16+ ($R = -0.685$, $p = 0.00002$), CD14++CD16- and CD14+CD16- ($R = -0.608$, $p = 0.0002$), CD14++CD16- and CD14+CD16+ ($R = -0.424$, $p = 0.002$). A decrease in GDH activity ($p = 0.0079$), GST activity ($p = 0.002$) and GR activity ($p = 0.0006$) was observed in patient platelets, which can reflect changes in the activity of intracellular metabolic pathways. A positive correlation was found between COX activity and $\alpha 1$ -PI ($R = 0.51$, $p = 0.025$). A significant decrease in faCS compared to control ($p = 0.0003$) and a negative correlation between faCS and GST activity ($R = -0.496$, $p = 0.011$) were observed. faCS was positively correlated with the degree of reduction in the PANSS score ($R = 0.416$, $p = 0.038$).

Conclusions: The revealed connection between molecular and cellular components of the immune system in patients with endogenous psychoses reflect activation of the systemic inflammatory response accompanied by changes in the ratio of monocyte subpopulations and impaired regulation of the complement system. The data obtained can be used to develop methods of monitoring patients taking into account their immunological features.

Disclosure of Interest: None Declared

EPP0437

Logistic regression model for the prediction of asthenia development in schizophrenia based on inflammatory blood markers

S. A. Zozulya*, A. N. Simonov and T. P. Klyushnik

Mental Health Research Centre, Moscow, Russian Federation

*Corresponding author.

doi: 10.1192/j.eurpsy.2024.586

Introduction: According to a number of authors, inflammation is involved in the development of asthenic syndrome in different diseases. The results of our own studies indicate that the main feature of the spectrum of inflammatory markers in patients with asthenic syndrome in schizophrenia is low enzymatic activity of leukocyte elastase against the background of high levels of other inflammatory markers. Presumably, the decrease in LE activity may be associated with functional exhaustion of neutrophils and/or their transmigration to the brain through the disrupted blood-brain barrier due to a long-term chronic inflammatory process.

Objectives: To create a logistic regression model for predicting the development of asthenia in schizophrenia based on the analysis of the association between leukocyte elastase (LE) and $\alpha 1$ -proteinase inhibitor ($\alpha 1$ -PI) activity in blood plasma.

Methods: A database including clinical and demographic parameters (ICD-10 diagnosis, duration of the disease, psychometric evaluation according to the PANSS and MFI-20 scales, sex and age) and immunological parameters (enzymatic activity of LE and functional activity of $\alpha 1$ -PI in blood plasma) of 95 patients from 22 to 55 years old with paroxysmal-progressive (F20.x1) and paranoid (F20.00) schizophrenia was used to construct the model. An asthenic symptom complex was diagnosed in 61 patients.

Results: A binary logistic regression model linking the probability of developing asthenia to LE and $\alpha 1$ -PI activity was constructed by analyzing a database of patients with schizophrenia.

$P = 1 / (1 + \exp [-(11.71 - 0.057 \cdot LE + 0.027 \cdot \alpha 1\text{-PI})])$ (2), where