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

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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), α1-proteinase inhibitor (α1-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 α1-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 α1-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

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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 α1-proteinase inhibitor (α1-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 α1-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 α1-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