EPP0982

Correlation of clinical and biological parameters in endogenous psychoses developed after coronavirus infection (COVID-19)

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Introduction: The importance of inflammation as a common pathophysiological mechanism underlying infectious and non-infectious diseases determines the relevance of studying the effect of coronavirus infection (COVID-19) on the course of endogenous psychoses.
Objectives: Clinical and immunological study of the potential impact of coronavirus infection on the course of endogenous psychoses.
Methods: Two groups of female patients aged 16 to 48 years with depressive-delusional conditions (F20.01, F21, F31) developed after a coronavirus infection were examined. In 15 patients, psychosis developed 1-2 months after COVID-19, and in 18 patients it occured within 2-6 months after the disease. The severity of psychopathological symptoms was assessed using PANSS and HDRS-21 scales. The activity of inflammatory markers leukocyte elastase (LE) and α1-proteinase inhibitor (α1-PI) in the blood of patients was determined. Neutrophil count, lymphocyte count and neutrophil/lymphocyte ratio were calculated. The standard values of indicators of healthy donors corresponding to patients by age and sex were used as controls.
Results: Endogenous psychoses that developed later after coronavirus infection were associated with "typical" inflammation, increased α1-PI activity and increased neutrophil degranulation (by LE activity) compared to normal values. Patients with this immune profile were observed to develop depressive-delusional states with a prevalence of positive affectivity (anxiety, melancholy) and an expanded nature of delusional disorders that were predominantly non-congruent with affect. Endogenous psychoses that developed within 2-6 months after COVID-19 were characterized by decreased neutrophil count, decreased LE activity, prevalence of negative affectivity (apathy, asthenia, adynamia), and a relatively undeveloped nature of delusional disorders that were predominantly congruent with affect.
Conclusions: Clinical and biological correlates presumably indicate the modulating effect of the coronavirus infection on (neuro) inflammation and the structure of endogenous psychoses.

Disclosure of Interest: None Declared

Depressive Disorders 05

EPP0983

Telomere length from peripheral blood DNA in major depressive disorder: a case/control comparison and association with antidepressant treatment response

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Introduction: Major depressive disorder (MDD)is one of the most common psychiatric disorders and a large number of patients present a poor response to treatment. Shortening of telomeres physiologically occurs after each cell division and their shortening is also associated with cell ageing. Studies are affirming that patients who suffer from mental illnesses have shorter telomeres in comparison with patients without such affections. [4] The effect of antidepressant medication on the biology of the telomeres was studied very little in humans and the current data suggest that the length of telomeres is a promising target regarding the prognosis and the response to psychotropic treatment.
Objectives: To analyze the telomere length (TL) from peripheral blood DNA of patients with major depressive disorder (MDD) compared to healthy controls. A second objective was to compare the TL of patients in relation to antidepressant treatment.
Methods: We analysed the clinical data from 16 patients admitted to the Psychiatry Clinic of Timisoara with the diagnosis of MDD and 10 healthy controls. The collection of clinical data was carried out in a structured and standardized manner both on paper and electronically, and the Hamilton Depression Rating Scale (HDRS) was applied to assess the severity of depression. Also, blood samples were collected, and plasma and white blood cells (WBC) were separated by centrifugation. Patients' samples were collected before and after 12 weeks of escitalopram antidepressant treatment, and a structured diagnostic interview and a standardized depression rating scale were done. DNA was extracted from WBC using the QIAamp DNA Mini Kit (Qiagen), and TL was determined by real time PCR using the Absolute Human Telomere Length Quantification qPCR Assay Kit (Sciencell Research Laboratories) according to the manufacturers' specifications. The TL expressed as megabases/diploid cell (Mb/DC) were compared between cases and controls using a Mann-Whitney test, and between patients before and after treatment using a Wilcoxon matched-pairs signed rank statistical test.
Results: The mean±SD telomere length for healthy controls was 0.3614±0.082Mb/DC, for treatment naïve patients was 0.4513±0.199Mb/DC, and for patients after treatment was 0.3476±0.070Mb/DC. There was no statistical significant difference in TL between patients and controls (p=0.266), nor between patients before and after treatment (p=0.055).
Conclusions: In this pilot project of limited sample size there was no difference in TL between MDD patients and healthy controls. Moreover, the TL of patients did not significantly change after 12 weeks of escitalopram antidepressant treatment.

Disclosure of Interest: None Declared