EPV1520

Assessment of inflammatory markers in late-onset schizophrenia

V. Pochueva[1]*, I. Kolykhalov[1] and L. Androsova[1]

[1]FSBSI Mental Health Research Center, Moscow, Russian Federation
*Corresponding author.
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Introduction: Previous studies have shown that neuroinflammation can play a significant role in the pathogenesis of schizophrenia. The search for inflammatory markers in late-onset schizophrenia are very important for diagnostics.
Objectives: Determination of inflammatory markers in the peripheral blood plasma of patients with late-onset schizophrenia in relation to the clinical characteristics of patients.
Methods: The study included 46 patients with schizophrenia aged 61 [56; 69] years (2 men and 44 women); the age of disease onset was 51 [45; 60] years. The severity of mental disorders was assessed using the PANSS scale, and cognitive impairment was assessed using the MMSE and MoCA scales. The control group consisted of 77 people comparable in age with the patients (p=0.16). A spectrophotometric method was used to determine inflammatory markers (enzymatic activity of leukocyte elastase (LE), functional activity of α1-proteinase inhibitor (α1-PI)). The protease inhibitory index (PII) was calculated - the ratio of LE and α1-PI activity, indicating the direction of the inflammatory process. Comparative data analysis was performed using the Statistica 10.
Results: The 1st cluster (22 patients (47.8%)) was characterized by a significantly increased α1-PI activity (p=0.000), decreased LE activity (p=0.000) compared to the control values, and, accordingly, a low PII value (p=0.000), which is an unfavorable prognostic factor for further development of the disease and response to therapy. A 28-day course of therapy with 1st and 2nd generation neuroleptics didn't change the immunological parameters in patients of this cluster. In this cluster, a positive correlation was found between LE activity and scores on the MMSE (r=0.512, p<0.05) and MoCA (r=507, p<0.05) scales at the start of treatment, i.e. the lower score on the cognitive functioning scales and the more severe the disease correlate with lower the LE activity.
The 2nd cluster (24 patients (52.2%)) was characterized by a significant increase in inflammatory markers, LE and α1-PI activity (p=0.000, p=0.000, respectively) in relation to the control parameters, while the PII value didn't differ from the control. In this group, paranoid and schizotypal personality were significantly less common in the premorbid period (41.6% of cases), and formal thinking disorders were expressed to a lesser extent. No clinical and immunological relationships were revealed. A 28-day course of therapy influenced the change in immunological parameters towards their relative normalization.
Conclusions: The obtained results confirm the involvement of the inflammatory link in the development of late-onset schizophrenia, as well as the heterogeneity of the patient group in terms of clinical and immunological parameters. Evaluation of the spectrum of inflammatory markers allows us to identify patients with an unfavorable course of the disease and resistance to therapy.

Disclosure of Interest: None Declared

EPV1521

Psychotic episode associated with autoimmune limbic encephalitis in a patient treated with checkpoint inhibitors: a case report

P. Sánchez Díez[1]*, C. Blanes Morell[1] and J. Torres Cortés[1]

[1]Psychiatry, Ramón y Cajal Hospital, Madrid, Spain
*Corresponding author.
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Introduction: Immune checkpoint inhibitors are being used in patients with advanced malignancies. Although it can effectively treat tumors, 30–60% of patients could experience immune-related adverse events such as encephalitis with antibodies against the NMDA receptor. We present a case of a 57-years-old man with no prior mental health history who was diagnosed of kidney cancer and received treatment with checkpoint inhibitors. He developed incoherent speech, visual hallucinations, delusional megalomania, disorientation, sleepiness and a low-grade fever of 37.7ºC. He was admitted in Neurology unit and diagnosed of autoimmune limbic encephalitis in a patient treated with checkpoint inhibitors.
Objectives: To describe a case of a psychotic episode associated with autoimmune limbic encephalitis in a patient treated with checkpoint inhibitors.
Methods: Clinical assesment and bibliographic review of pertinent literature.
Results: During his admission in Neurology ward, the patient was suspicious, inattentive, aggressive with healthcare staff and he developed incoherent speech with visual hallucinations.
MRI suggested bilateral limbic encephalitis and the antibody test in cerebrospinal fluid were positive for NMDA receptor.
The psychotic episode was treated with olanzapine up to 20 milligrams and the limbic encephalitis with rituximab with a good response.
Conclusions: The case presented is consistent with other reports of psychotic symptoms and development of encephalitis associated with antibodies against the NMDA receptor.
The diagnosis of anti-NMDAR encephalitis is usually delayed.
The differential diagnosis should be established with primary psychiatric disorders.

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EPV1522

Blood Circulating T Cell Subsets in Relation to Childhood Trauma and Major Depressive Disorder

Z. Susam[1,2]*, A.-L. Boller[1,3], J. Freff[1,3], T. Kircher[4,5], U. Dannlowski[6], B. T. Baune[1,7,8] and J. Alferink[1,3]

[1]Department of Mental Health, University of Münster, Münster; [2]Department of Psychiatry and Psychotherapy, Heinrich-Heine University, LVR Düsseldorf, Düsseldorf; [3]Cells-in-Motion Interfaculty Centre (CiMIC), University of Münster, Münster; [4]Department of Psychiatry and Psychotherapy, University of Marburg, Marburg; [5]Center for Mind, Brain and Behavior (CMBB), University of Marburg and Justus Liebig University Giessen, Giessen; [6]Institute for Translational Psychiatry, University of Münster, Münster, Germany; [7]The Florey Institute of Neuroscience and Mental Health and [8]Department of Psychiatry, The University of Melbourne, Melbourne, Australia
*Corresponding author.
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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.

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EPV1523

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

M. Vasconcelos[1]*, C. Silva[2], M. Bajouco[1], D. Mota[1], S. Batista[2] and A. Mendes[1]
[1]Psychiatry and [2]Neurology, Coimbra Hospital and University Centre, Coimbra, Portugal
*Corresponding author.
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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.

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EPV1524

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

S. A. Zozulya[1]*, T. I. Shishkovskaya[1], I. N. Otman[1], I. V. Oleichik[1], Z. V. Sarmanova[1], P. A. Baranov[1] and L. V. Androsova[1]
[1]FSBSI "Mental Health Research Centre", Moscow, Russian Federation
*Corresponding author.
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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.