

EPV1107

Dynamics of Psychopathological Disorders and Changes in the Functional Activity of Neutrophils in Mercury Intoxication

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Introduction: The overall goal was to determine the degree of mental, neuromuscular and statodynamic functions disorders resulting from toxic effect of mercury and its compounds, to study relationship between the degree of loss of professional ability to work as a result of occupational disease with the presence of complications due to concomitant diseases.

Objectives: The relevance of studying the degree of violations of the main indicators of vital activity due to the toxic effect of mercury and its compounds is due to the persisting high level of loss of professional ability to work, and in some cases, and disability of this contingent. Analysis of indicators of primary and repeated disability due to occupational diseases showed that a sufficiently high number of persons recognized as disabled.

Methods: We studied the dynamics of psychopathological disorders: violation of the emotional sphere, thinking, perception, attention, volitional activity and cognitive functions. The functional and metabolic activity of segmented neutrophils in the peripheral blood of 42 patients, men and women aged 43 to 64 years, and 22 healthy donors were studied.

Results: Long-term exposure to chronic industrial mercury intoxication led to persistent disorders of mental functions, suppression of phagocytic activity of neutrophils up to 42.3+/-4.7 per cent and inhibition of the reaction with nitro blue cytosol 4.2+/-0.1 per cent..

Conclusions: The revealed violations of the emotional-volitional sphere and cognitive mental functions are possibly associated with the suppression of nonspecific cellular reactions of microphages. Violations of neuroimmune interactions in mercury intoxication require further study.

Disclosure: No significant relationships.

EPV1106

“I want to kill my mother”

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Introduction: The incidence of Anti-NMDA receptor encephalitis is 1.5 per million population per year but it has only been described sporadically in the paediatric population. The median age of onset is 21 years old. In children, seizures, abnormal movements, insomnia, and irritability are more frequently identified than psychosis or abnormal behavior. Early detection and treatment can improve treatment outcomes.

Objectives: Presentation of a case of a paediatric patient with Anti-NMDA receptor encephalitis displaying homicidal behavior towards her family members

Methods: Case report

Results: A 12 year old with no significant past psychiatric and medical history, was brought in by police to the hospital after she attempted to stab her mother with a knife. She had low grade fever, headaches and lethargy prior to presentation. Organic workup revealed serum and CSF anti-nmda receptor to be positive. She received early treatment with steroids and intravenous immunoglobulins and no longer harboured further homicidal ideations.

Conclusions: Initial suspicion of anti-NDMA receptor encephalitis should be raised and early auto-immune workup performed in paediatric patients presenting with acute change in behavior especially in young females with no past medical or psychiatric history

Disclosure: No significant relationships.

Keywords: Anti-NDMA receptor encephalitis; paediatric auto immune encephalitis; homicidal ideations

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Association between markers of inflammation and indicators of systemic endotoxemia in endogenous psychosis

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Introduction: The clinical and biological studies indicate the involvement of inflammation in the pathogenesis of endogenous mental disorders. The inflammation markers leukocyte elastase, α 1-proteinase inhibitor, and autoantibodies to neuroantigens reflect the severity of the pathological process in the brain. Systemic endotoxemia is a pathological process caused by an excess of endotoxins in the systemic circulation, can be considered as one of the components of the inflammatory process in endogenous psychosis.

Objectives: To evaluate the association between systemic inflammation markers and indicators of systemic endotoxemia in patients with endogenous psychosis.

Methods: The study included 25 patients aged 23-49 with endogenous psychoses (F20, F25) and 25 healthy people. The severity of symptoms was assessed using PANSS. We detected the activity of leukocyte elastase and α 1-proteinase inhibitor, antibodies to neuroantigens, endotoxin (ET) concentration, and antibodies to endotoxin (aET) in serum.

Results: In 24% of cases, an increase of inflammation markers activity, ET concentration, and aET deficiency were observed ($p < 0.05$), which is an unfavorable factor that aggravates the clinical course of the disease. In 76% of cases, ET concentration remained within control values ($p > 0.05$) but associated with different levels of

aET, which is likely to be a consequence of previous endotoxin aggression. There were correlations between ET concentration and antibodies to neuroantigens S-100B and MBP. We also revealed the association between the activity of the inflammatory marker with the severity of clinical symptoms in patients.

Conclusions: Results suggest the relationship between systemic inflammation markers and indicators of systemic endotoxemia and their involvement in the pathogenesis of endogenous psychosis.

Disclosure: No significant relationships.

Keywords: endogenous psychosis; systemic inflammation; systemic endotoxemia; antiendotoxin antibodies

EPV1108

Immunoregulatory and neuroprotective activity of ovocystatin

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Introduction: Ovocystatin has beneficial properties for cognitive function in young rats and might prevent aging-related cognitive impairment in older animals, as well as reduce memory decline in APP/PS1 mice model.

Objectives: Our study aimed at assessing the impact of ovocystatin on microglia activation and neurogenesis.

Methods: Immunoactivation: Mouse wild type microglia were stimulated with ovocystatin at dose of 100 micrograms/ml. The effect of ovocystatin on nitric oxide production and interleukin 1 beta secretion were determined. Neurogenesis: Primary rat hippocampal neurons of H19-7 cell line was used. The impact of ovocystatin on proliferation, nitric oxide production, and expression of markers of neurogenesis: microtubule-associated protein 2 (MAP2, isoforms A/B and C/D) and Synapsin 1, were determined.

Results: It was shown that ovocystatin does not stimulate microglial cells to produce inflammatory mediators. Whereas, no toxic effect of ovocystatin (1-100 ug/ml) on H19-7 cells viability, and dose-dependent down-regulation of proliferation were demonstrated. It was also shown that in primary hippocampal neurons of H19-7 cells incubated with ovocystatin (100 micrograms/ml), the expression level of MAP2 C/D (75kDa) - characteristic form of immature neurons is unchanged. However, the increased expression of MAP2 A/B protein (280 kDa) - characteristic for mature neurons was observed after 6 and 24h incubation with ovocystatin. Relatively to MAP2 A/B, increased expression of synapsin 1 was observed.

Conclusions: The ovocystatin might be a potential activator of molecular mechanisms in primary hippocampal neurons, participating in regulation of neurogenesis. Nevertheless, further studies are needed.

Disclosure: No significant relationships.

Keywords: ovocystatin; Immunoactivation; Neurogenesis

EPV1109

Immunology and psychosis: 22q11.2 syndrome as a model of study

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Introduction: The 22q11.2 deletion syndrome (22q11DS) defines a set of developmental abnormalities due to a loss of genetic material. Phenotypic expression is highly varied. The immunological alterations that present as a severe combined immunodeficiency and the neurodevelopmental alterations stand out, especially the psychotic symptoms. It is the best described genetic alteration for the development of psychosis, presenting up to 30% of patients with compatible symptoms, with various hypotheses that justify it.

Objectives: General objective:

1. Justify that SD2q11 can be used as a model for human research on the origin of psychosis.

Specific objectives: 1. Describe the pro-inflammatory state present in SD2q11.

2. Describe differences at the immunological level in SD2q11 among those patients with presence of psychotic symptoms of those who do not present them.

3. Describe possible biomarkers.

Methods: A systematic review of the literature in the last 5 years using electronic resources (PubMed and WOS) until June 2021 following the PRISMA recommendations.

Results: Three original articles were reviewed. There is a very marked pro-inflammatory state in 22q11DS patients with psychotic symptoms. They present an increase in subpopulations of CD4. The IL-17 is important in the formation in primitive stages of the hippocampus, its ease of breaking the blood-brain barrier (BBB). The neutrophil / lymphocyte ratio is presented as a possible biomarker to predict patients at high risk of developing psychosis.

Conclusions: There is sufficient evidence that patients with 22q11DS can be used as a research model regarding possible hypotheses about the genesis of psychosis.

Disclosure: No significant relationships.

Keywords: Digeorge syndrome; psychosis; schizophrenia; inflammation

EPV1110

Leukocyte elastase, a1-proteinase inhibitor, and autoantibodies to neuroantigens in diagnostics of endogenous depressive disorders

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