

## EV956

**Psychiatric symptoms as onset of anti-NMDAR encephalitis**

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**Introduction** Every more often, there is evidence that shows a relationship between psychiatric symptoms and autoimmune disorders. Such is the case of anti-NMDAR encephalitis, in which it has been recently described the development of psychotic symptoms. Anti-NMDAR encephalitis is an autoimmune disorder that involves IgG autoantibodies against the NMDA receptor subunit GluN1. This last fact could support the relationship with the glutamatergic model of schizophrenia.

**Objective** To conduct a current review to deepen the detection and management of anti-NMDAR encephalitis, due to the frequent existence of psychiatric symptoms at onset, which have contributed to the difficulty of diagnose.

**Method** Systematic review of the literature in English (PubMed), with the following keywords: “Autoimmune encephalitis”, “psychosis”, and “NMDA receptor”.

**Results** Autoimmune encephalitis appears more frequently in children and young adults and it is characterized by a prodromal period, in which there usually are non-specific symptoms of headaches or fever. Next, it could progress to cognitive deficits, seizures, catatonic symptoms and psychosis. However, sometimes in the rarest clinical presentations, there is nothing but psychiatric symptoms at the onset of encephalitis, which leads to misdiagnose and lack of proper treatment. This fact has stimulated the curiosity of the psychiatry scientific community, since the anti-NMDAR encephalitis may mimic the glutamatergic model of schizophrenia.

**Conclusions** To make an accurate and detailed diagnostic formulation in people with psychiatric symptoms as onset of any disorder is essential to determine whether it is a primary psychiatric disorder or symptoms associated to another disease.

**Disclosure of interest** The authors have not supplied their declaration of competing interest.

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## EV959

**Psychoneuroimmunology alternations as a comorbidity of post-traumatic stress disorder in veterans – case report**

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Post Traumatic Stress Disorder (PTSD) is defined as an extensive response to a major traumatic event. Psychoneuroimmunology represents an integrative approach in tackling and understanding various human diseases and disorders such as cardiovascular, autoimmune and physical complaints/chronic pain. Psychosocial context influences brain stress response pathways and modifies stress-related behavior. In this case report, we observed 5 patients, veterans from Croatian War of Independence (1990-1995), who suffer from PTSD. They have altered stress reactivity, as well as distinct expression for genes involved in immune activation. Those patients have been found to exhibit a number of immune changes including increased circulating inflammatory markers, increased

reactivity to antigen skin tests, lower natural killer cell activity, and lower total T lymphocyte counts. The traumatic event (Croatian War of Independence) generates downstream alterations in immune function. This case report imply that immune dysfunction caused by PTSD may mediate or facilitate somatic conditions.

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## EV960

**Possible anti-inflammatory role of perivascular macrophages in a model of depression induced by chronic mild stress in rats**

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Perivascular macrophages (PVM) are hematopoietic cells that migrate to the brain perivascular space modulating the interactions between the immune and central nervous systems (CNS). Previously, their depletion with the icv administration of the proapoptotic drug clodronate encapsulated in liposomes increased the vascular production of the proinflammatory prostaglandin E<sub>2</sub> (PGE<sub>2</sub>), the release of ACTH, corticosterone and fever, induced by the intravenous administration of Lipopolysaccharide (LPS). Further studies also demonstrated a decrease in the synthesis of the anti-inflammatory prostaglandin 15d-PGJ<sub>2</sub>.

With this background, we decide to deeper explore the mechanisms involved in the anti-inflammatory profile of PVM by depleting them in a model of depression induced by chronic mild stress (CMS) exposure in rats.

Our results showed an increase of the proinflammatory cytokines TNF $\alpha$ , IL-1 and IL-6 at mRNA levels in the prefrontal cortex of the groups of animals where the PVM were depleted, as well as in the protein levels of the pro-inflammatory nuclear factor NF- $\kappa$ B, the enzymatic pro-inflammatory enzymatic sources iNOS, COX-2 and m-PGES-1 and their product PGE<sub>2</sub>. A concomitant decrease of the 15d-PGJ<sub>2</sub> mediator was also observed. In addition, we also checked whether the depletion of PVMs could regulate the expression of molecules implicated in the leukocyte traffic and infiltration in the CNS in our CMS model. Thus, the mRNA levels of the chemokines MCP-1, fractalkine and the adhesion molecule VCAM appeared increased in the animals without PVMs.

In summary, our results could suggest a potential anti-inflammatory role for PVMs in a depression model chronic stress-induced as CMS.

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## EV961

**Management and psychiatric manifestations of anti-NMDA receptor encephalitis, a case report**

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**Introduction** Anti-NMDA receptor (NMDAR) encephalitis, formally recognized in 2007 by Dalmau et al, is an autoimmune disorder with a complex presentation that includes psychiatric symptoms, memory deficits, and autonomic instability. The exact incidence is unknown but age, gender, and ethnicity may all play a role. Presence of antibodies against the GluN1 subunit of the