

loss and seizures followed by a movement disorder, loss of consciousness and changes in blood pressure, heart rate and temperature. Postpartum depression symptoms usually develop within the first few weeks after giving birth, but may begin earlier/during pregnancy / or later /up to a year after birth. They include: inability to sleep or sleeping too much, depressed mood or severe mood swing, difficulty bonding with your baby, withdrawing from family and friends, fatigue or loss of energy, feelings of shame, guilt or inadequacy, diminished ability to think clearly, concentrate or make decisions, anxiety and panic attacks, thoughts of harming yourself or your baby. Untreated may last for many months or longer.

**Objectives:** Recent studies have highlighted the possibility that a subset of patients with first-onset severe psychiatric episodes might suffer from undiagnosed autoimmune encephalitis. The acute onset of severe atypical psychiatric symptoms in young female patients should raise the index of suspicion for anti-NMDA receptor encephalitis, particularly in the setting of neurological symptoms, including side effects of antipsychotic treatment.

**Methods:** /

**Results:** /

**Conclusions:** Creating a therapeutic environment is an interdisciplinary clinical and theoretical approach to psychiatric treatment in hospital settings, the basic idea of which is that the entire environment has therapeutic potential. Psychodynamic knowledge and understanding of the process as well as principles of body-oriented psychotherapy may be of great importance in the treatment of these patients in addition to the use of pharmacotherapy.

**Disclosure of Interest:** None Declared

## EPV0812

### Differential diagnosis of cognitive dysfunction in a multi-morbid patient

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**Introduction:** Patients with systemic lupus erythematosus (SLE) have cognitive dysfunctions as a neuropsychiatric manifestation, associated with disabling symptoms. However, the presence of other medical or psychiatric comorbidities can delay or lead to a misdiagnosis.

**Objectives:** To present a case of a patient with diagnostic difficulty in the face of multiple medical and neurocognitive comorbidities.

**Methods:** Description of a case report.

**Results:** 19-year-old female, Mexican, unemployed, with incomplete high school, with medical history of preterm birth by cesarean at 30 weeks due to placenta previa, history of early puberty, 4 years evolution of focal epilepsy, 1 year evolution of hypothyroidism and mild depression.

She began her symptoms 4 years ago, characterized by an abrupt onset of memory disturbances, decreased concentration, poor academic performance, infantile behavior, need for affection, alternated with irritability periods, verbal and physical aggression,

repetitive and erratic behavior. She went to multiple specialists with different therapeutic approaches without clinical improvement. In 2020, she was referred to our service for evaluation, evidencing a mild depressive episode and psychotherapeutic treatment was started.

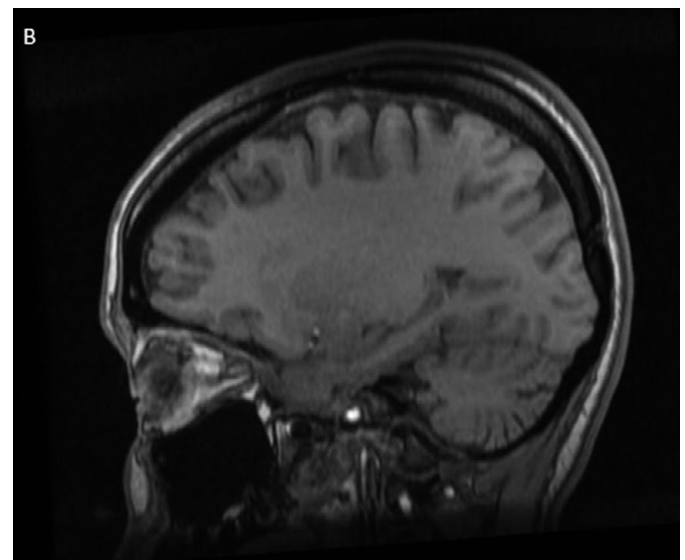
Mental and neurological examination without alterations, normal vital signs, at physical examination: malar rash, oral ulcers, alopecia. Labs: increased erythrocyte sedimentation rate, normocytic anemia, leukopenia, rest normal. An electroencephalogram was requested, without alterations. Simple brain MRI was performed (Figure 1).

Psychological (figure 2) and Neuropsychological tests (table 1) were performed, showing alterations in memory recall and inhibitory control.

Due to the symptoms presented by the patient, SLE was suspected, and rheumatology evaluation was requested, integrating a diagnosis of incomplete SLE, and started treatment. The patient presented symptomatic improvement in cognitive symptoms and systemic signs. Likewise, a genetic evaluation was requested, without meeting the criteria for a genetic syndrome. The patient continues with symptomatic improvement and multidisciplinary treatment.

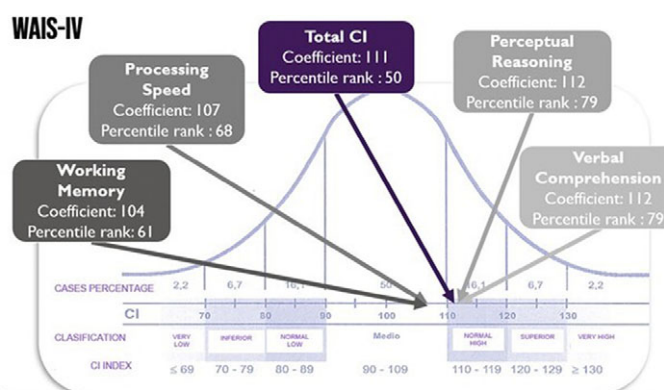
Total scores	Natural	Normalized	Diagnosis
<i>Orbitomedial</i>	180	83	Mild alteration
<i>Pref-Anterior</i>	22	106	Normal
<i>Dorsolateral</i>	207	88	Normal
<i>BANFE total</i>	409	104	Normal

**Image:**



**Figure 1.** Simple sagittal MRI of the brain showing a generalized decrease in cortical and subcortical cerebral and cerebellar parenchyma.

Image 2:



**Conclusions:** Neurocognitive alterations are one of the most frequent manifestations of neurolupus, although its diagnosis and treatment may be delayed in the absence of clinical suspicion, mainly in multi-comorbid patients.

In the case, the patient presented multiple diseases that can explain a picture of neurocognitive impairment, such as epilepsy, depression, hypothyroidism. However, in these cases, a multidisciplinary approach is imperative, requiring to rule out the different causes of the patient's symptoms.

**Disclosure of Interest:** None Declared

## EPV0813

### Subpopulation composition of monocytes and inflammation markers in schizophrenia

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**Introduction:** Taking into account the role of the immune system in the pathogenesis of schizophrenia, it is important to study the peculiarities of innate immunity in the development of the disease. A special role in these processes belongs to monocytes, which play an integral role in the inflammatory reactions and perform regulatory and effector functions to other immunocytes.

**Objectives:** To analyze the subpopulation composition of monocytes and other inflammatory markers in patients with schizophrenia

**Methods:** The study included 36 women with schizophrenia (F20, ICD-10) (30±12 years) in the acute stage of the disease and 20 healthy donors. Flow cytometry was used to determine the relative number of monocyte subpopulations. The activity of leukocyte elastase (LE) and α1-proteinase inhibitor (α1-PI) in blood was determined by spectrophotometric method. The level

of autoantibodies to S100b and CRP concentration were assessed by ELISA.

**Results:** A decrease in “classical” monocyte subpopulation (p=0.02) was accompanied by an increase in cells of the pro-inflammatory phenotype (p=0.03) “Transitional” and “non-classical” subpopulations did not differ from controls. A negative correlation was found between the proportion of “classical” monocytes with “transitional” and “intermediate” cells (r=-0.66 and r=-0.54, p=0.01). All inflammatory and autoimmune blood markers in patients were significantly elevated compared to controls (p<0.05).  
**Conclusions:** The redistribution of the subpopulation composition of monocytes with an increase in “intermediate” subpopulation and an increase in other immune markers in the acute stage of schizophrenia serve as an additional link confirming the involvement of cellular immunity in the pathogenesis of the disease.

**Disclosure of Interest:** None Declared

## EPV0814

### Haloperidol-induced facial and upper limbs oedema: a case report study

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**Introduction:** Haloperidol is one of the first-generation anti-psychotics which is used widely around the world and still has its place in psychotic disorders along with his documented antimanic properties.

It acts by blocking essentially dopaminergic receptors but also serotonergic and alpha-adrenergic receptors.

Haloperidol has many side effects, especially extrapyramidal symptoms. Oedema associated with haloperidol is a rare side effect.

**Objectives:** The aim of this presentation is to describe the case of a young female who developed a rare side effect of haloperidol.

**Methods:** we conducted a review of literature on different data base about this side effect in order to discuss its potential underlying mechanisms.

**Results:** Here we report a case of a 24-year-old female with the history of bipolar disorder type 1, and an allergy to chlorpromazine. She was admitted to our psychiatric department for a manic episode with psychotic features and was initially treated with 50 mg of Haloperidol in addition to 30 mg of valium per day. She developed pronounced oedema of the face and upper limbs after two weeks of treatment. All of the paraclinical examinations including blood cell count, liver function tests, renal function tests, serum electrolytes, ECG, urine test didn't show any abnormalities. We solicited the opinion of both nephrology and pharmacovigilance and concluded to an allergic oedema.

The symptoms disappeared 10 days after the discontinuation of haloperidol which suggested a potential incrimination of this drug. As far as we know, there are very few reports of allergic oedema with haloperidol in the literature. Potential underlying mechanisms will be discussed.

**Conclusions:** Through this case study, we aimed to focus attention on this very rare but still possible side effect.

**Disclosure of Interest:** None Declared