Table 2. The severity of aggressiveness in unipolar and bipolar depression.

BDHI subscale	Bipolar Depression	Unipolar Depression	p (U-test)
Aggressiveness index	19 (13; 24)	18.5 (12; 24)	0.745
Hostility index	9 (7; 13.75)	9 (7; 11)	0.139
Assault Hostility	4 (2; 6)	4 (2; 6)	0.618
Indirect Hostility	5 (5; 6)	4 (4; 6)	**0.015**
Irritability	6 (4; 8)	5 (3; 7)	0.081
Negativism	2 (1; 4)	2 (1; 4)	0.262
Resentment	5 (4; 6)	5 (3; 6)	0.113
Verbal Hostility	7 (6; 8)	6 (5; 8)	**0.008**

As a result of the study, no statistically significant correlations were found (p>0.05, Spearman's test).

Conclusions: The conducted research did not yield convincing data that would allow us to make judgments about specific clinical patterns in the course of unipolar and bipolar depression. Thus, the problem of searching for unique biological markers of the courses of affective disorders remains relevant. Support by the Russian Science Foundation grant No. 23-75-00023.

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EPV0108

Neuropsychiatric symptoms in Multiple Sclerosis (MS): Case Report of a First Manic Episode in a Patient with Suspected MS

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Introduction: Multiple Sclerosis (MS) is an inflammatory disease affecting primarily the central nervous system, characterized by focal lesions of white-matter demyelination. It can present with a variety of neurological symptoms, including monocular vision loss, sensory loss, paresthesias, limb weakness, ataxia and bladder dysfunction, and has a typically chronic and progressive course. Neuropsychiatric manifestations including depressive or manic symptoms, anxiety disorders and psychosis, are also frequently observed, and are of particular importance to mental health practitioners.

Objectives: To describe a case of a 45-year-old female patient with a history of suspected MS presenting with manic symptoms, and to discuss the possible neuropsychiatric manifestations of Multiple Sclerosis.

Methods: Clinical case report and literature review.

Results: A 45-year-old woman was brought to the emergency department presenting with severe acute agitation, irritable mood, rapid speech and persecutory delusions. She had no prior history of neuropsychiatric symptoms, but her medical history was notable for a suspected diagnosis of MS, having suffered an episode of optic neuritis 16 years before the present episode. Magnetic Ressonance Imaging performed 3 months before emergency admission documented non-specific white-matter lesions presenting as hyper-intense in long TR sequences, as well as a cervical lesion of atypical characteristics, representing possible spondylotic myelopathy or demyelination. A head CT performed at emergency admission did not reveal relevant acute findings. The patient was hospitalized and initiated risperidone and valproic acid therapy. She responded favorably to medication, with progressive stabilization of mood and remission of delusional ideas over three weeks.

Conclusions: Neuropsychiatric symptoms are a common and concerning manifestation of Multiple Sclerosis. The present case illustrates that clinicians should be on alert for signs of mood and psychotic symptoms in patients with suspected or confirmed MS, as these can manifest at any point during the disease course.

Disclosure of Interest: None Declared

EPV0109

Our old friend lithium and encephalopathy: a case report

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Introduction: Lithium is a well-established mood stabilizer used in the management of bipolar disorder, that is generally well-tolerated; however, it is associated with rare but potentially severe neurological side effects. Lithium-induced encephalopathy is characterized by a spectrum of symptoms, ranging from subtle cognitive deficits to severe manifestations such as altered mental status to overt delirium, seizures and coma. Risk factors include advanced age, concomitant medication and underlying renal impairment. This symptoms do not consistentely correlate with lithium concentrations.

Objectives: This abstract aims to provide an overview of the clinical characteristics, underlying mechanisms, and management of lithium-induced encephalopathy.

Methods: We discuss a case of a 62-years-old woman diagnosed with bipolar disorder under treatment with lithium and olanzapine, without recent changes of posology. She presented to emergency department with subacute and fluctuating neuropsychiatric symptoms, including confusion, disorientation in time and space, complex visual hallucinations, delusional ideas, alteration in memory and logic thinking, dysarthria and dyspraxia. Neuroimaging showed no structural abnormalities, blood tests were normal and serum lithium levels were within the therapeutic range (0.8 mEq/L). Upon discontinuation of lithium, the patient exhibited a gradual resolution of symptoms. We conducted a comprehensive search of medical databases, including PubMed, to identify relevant articles related to lithium encephalopathy published up to September 2023.

Results: This case challenges the conventionally established threshold of elevated serum lithium levels in the development of encephalopathy. The underlying pathophysiology is complex and multifactorial, with proposed mechanisms including alterations

in neurotransmitter balance, oxidative stress, mitochondrial dysfunction and individual susceptibility to idiosyncratic reactions. Early diagnosis is challenging, necessitating a high clinical suspicion, neuroimaging and exclusion of other etiologies. Management strategies involve discontinuation of lithium, even when serum lithium levels are within the therapeutic range, supportive care, and, in severe cases, hemodialysis to reduce lithium levels rapidly.
Conclusions: Clinicians should maintain a high index of suspicion of lithium-induced encephalopathy, especially in patients presenting with neurological symptoms while on lithium treatment. Early recognition and intervention are essential for minimizing morbidity and preventing potentially irreversible neurological damage. Further research is needed to better understand the precise mechanisms underlying it, risk factors and to refine treatment strategies.

Disclosure of Interest: None Declared

EPV0110

Multifactorial etiology of manic episodes. About a case

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Introduction: Manic episodes have a multifactorial etiology, with frequent association with genetic factors, comorbidities such as systemic diseases or secondary to infectious diseases, and environmental exposure factors. The prevalence of bipolar disorder is markedly higher in patients with autoimmune disease. The risk of developing bipolar disorder in some studies has been seen to be higher among patients with rheumatoid arthritis, therefore chronic inflammation would be a potential mechanism and could be a modifiable risk factor for bipolar disorder. Growing evidence indicates that Sars-CoV-2 may also trigger the acute onset of mood disorders or psychotic symptoms.
Objectives: We present the case of a patient who presents symptoms compatible with an acute manic episode after an outbreak of rheumatoid arthritis and comorbid COVID infection.
Methods: 52-year-old patient. She went to the hospital emergency room presenting affective symptoms compatible with a manic episode and psychomotor agitation. Personal medical history: rheumatoid arthritis, antiphospholipid syndrome. Psychiatric personal history: Depressive disorder under follow-up by a private psychiatrist under treatment with antidepressants. During the interview, the patient presented accelerated speech, with great emotional incontinence. Saltigrade thought and tachypsychia. She verbalizes delusional ideas of megalomaniacal and mystical and religious characteristics. She verbalizes that she is the reincarnation of the holy spirit, that God has taken her body and speaks through her. In the emergency room, a Sars-CoV-2 infection that the patient was unaware of was diagnosed. She is admitted to the hospital in the mental health unit, in the first interviews the patient maintains speech with delusional ideas "I notice the stigmata of Christ on my body".
Results: The patient recovers after treatment for the COVID infection, remaining asymptomatic. It was decided to start lithium to stabilize mood and the patient presented good tolerance and treatment with antipsychotics. The patient presented a favorable response, remitting the psychotic symptoms of which she was critical and stabilizing the affective symptoms. The patient is diagnosed with Severe Manic Episode with Psychotic Symptoms, as the main diagnosis and we could conclude the diagnosis of Bipolar Disorder since she has presented 2 depressive episodes in the past that have required treatment and follow-up by psychiatry.
Conclusions: Manic episodes have a multifactorial etiology and require an individualized approach, and comorbid medical conditions must always be assessed in order to establish a therapeutic plan with patients.

Disclosure of Interest: None Declared

EPV0111

Carbamazepine-induced toxidermia: Case report and a literature review

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Introduction: Carbamazepine is effectively used in treatment of bipolar disorder for its thymoregulatory virtues, but it can induce numerous side effects, including skin eruptions that can be severe sometimes.
Objectives: To study the relationship between toxidermia and treatment with carbamazepine.
Methods: We report the case of a patient who developed a toxidermia following the intake of carbamazepine.
Results: Mr. AD, 19 years old, with medical history of diabetes, has been diagnosed with bipolar disorder since the age of 17. He was initially treated with risperidone with an irregular follow-up.
He was hospitalized in our department for a manic episode with psychotic features with agitation and refusal of treatment.
The patient was put on injectable treatment 15 mg/day of Haloperidol and 20 mg/day of diazepam.
After 5 days in hospital, we switched to the oral route, gradually increasing haloperidol doses to 30mg, reducing diazepam doses and introducing carbamazepine for thymoregulatory purposes.
Carbamazepine was progressively increased up to a dose of 800mg per day.
Fourteen days after the introduction of carbamazepine, the patient presented a generalized rash requiring the discontinuation of this medication. He was treated with an anti-histamine and local corticosteroids, on the advice of dermatologists.
In the days following discontinuation of carbamazepine, skin lesions regress and then disappear.
Biologically, we observed a rise in eosinophilic polynuclear cells to 580, followed by a gradual decrease after stopping the treatment.
A pharmacovigilance opinion was sought, concluding that carbamazepine was responsible for the toxidermia, given the delay in