Objectives: To present a case of Mirtazapine-induced psychosis in a patient with severe malnutrition, and with no history of psychosis and despite on sedating antipsychotic.
Methods: This is a case report.
Results: Ms. NC, a 40-year-old female with major depressive disorder, anorexia nervosa, stimulant use disorder, and sedative, anxiolytic, hypnotic use disorder with no history of psychosis even when intoxicated or during withdrawal, was admitted for involuntary inpatient psychiatric care for detoxification and management of severe malnutrition. Ms. NC has always been conscious with her weight growing up but it was only during the COVID-19 pandemic that excessive preoccupation with weight and symptoms of clinical depression were noted. Ms. NC restricted her diet and engaged in excessive exercise resulting to BMI of 16.1. She started use cocaine and diazepam daily to address the weight and mood, and sleep and anxiety, respectively. Due to a suicidal attempt, consult was done with a psychiatrist, and patient was eventually maintained on Mirtazapine 30mg and Gabapentin 100mg which addressed the mood and sleep. Despite improvement in mood and decrease in use of cocaine and diazepam, patient started to use methamphetamine around once a week. Despite with euthymic mood, preoccupation with weight resurfaced. After a few months, she restricted her food intake to only four times a week with no binge-eating or purging resulting to BMI to 13.8. Upon admission, Mirtazapine 30mg was continued and Gabapentin was increased to 300mg. Special care in her food intake was done to prevent refeeding syndrome. Benzodiazepine withdrawals symptoms were minimal. She has normal values for electrolytes, liver function tests and creatinine. On the first days of admission, she was noted to be irritable and was mostly asleep. On the fifth hospital day, she started to have difficulty sleeping and was placed on Olanzapine up to 10mg and Gabapentin 600mg but no improvement in sleep. On the tenth hospital day, Mirtazapine was increased to 45mg and later in the night, had visual and auditory hallucinations and paranoia. Upon discontinuation of Mirtazapine and initiation with Clozapine up to 75mg, the psychosis resolved after five days.
Conclusions: Mirtazapine-induced psychosis may be seen in patients with severe malnutrition. Despite its advantages in terms of weight gain and sleep, psychiatrists should be wary of this possible side effect when initiating or increasing Mirtazapine for patients with severe malnutrition.

Disclosure of Interest: None Declared

EPV0854

BILATERAL TEMPOROMANDIBULAR JOINT DISLOCATION AND ANTIPSYCHOTIC TREATMENT: A CASE REPORT

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Introduction: Acute dystonia is a type of extrapyramidal effect that is produced by the blockade of dopaminergic D2 receptors typical of antipsychotics. There is a subtype acute dystonia called oromandibular, which produces perioral manifestations. In extreme cases it can even produce temporomandibular joint dislocation, bilateral being more frequent than unilateral. In this abstract it is presented the clinical case of a 22-year-old female who attended to the Emergency Department due to a bilateral temporomandibular joint dislocation that was finally attributed to antipsychotic treatment.
Objectives: The objective of the clinical case is to point out the importance of examination and clinical history for psychiatric diagnosis.
Methods: Review of various scientific articles related to acute dystonia.
Results: It is a report of a 22-year-old female with no medical-surgical or psychiatric history who was imprisoned for legal conflicts. During her stay in prison, she presented reactive depressive and anxiety symptoms, receiving antidepressant and anxiolytic treatment. After two months in prison, she was released and, two days after her release, she attended to the Emergency Department due to rigid akinetic symptoms, drowsiness, mutism and urination difficulties. Complementary tests revealed bilateral temporomandibular joint dislocation, with no other organicity wich could justify the rest of the symptoms, so she was admitted to the Acute Psychiatry Unit for study.
During her admission, the physical examination (akinetic rigid picture, muscle contraction and galactorrhea) raised the possibility that it was extrapyramidal symptomatology secondary to antipsychotic treatment. Given that suspicion, intramuscular biperiden 5 mg/ml was administered, improving the condition in two hours. In a second time, the initial anamnesis was redone; the patient added that during her stay in prison she had presented psychomotor agitation for which she had recieved an intramuscular treatment that she was not able to specify. All this information confirmed the initial suspicion; it was extrapyramidal symptomatology induced by antipsychotic treatment. Thus, treatment with oral biperiden 4 mg/12 hours was continued and the condition completely remitted in five days.
Conclusions: In this abstract it is presented the case of a bilateral temporomandibular joint dislocation induced by antipsychotic treatment. Although it is a rare presentation, other cases like that have been described in the literature, specifically with the use of haloperidol, risperidone, amisulpride and aripiprazole. Given the high frequency of adverse effects of antipsychotics, it is essential that psychiatrists remain trained in their prediction and management.

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EPV0855

A case of phentermine-induced psychosis: the need for caution for drug-drug interactions

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Introduction: Phentermine is a sympathomimetic amine that the U.S Food and Drug Administration has approved for short-term use in the treatment of obesity. However, there have been case reports of phentermine being associated with neuropsychiatric symptoms, and thus caution is needed to avoid drug-drug interactions when prescribing phentermine (Nathan PJ, *et al.* CNS

Neurosci Ther 2011;17:490-505) We present a case of phentermine-induced psychosis that could have been precipitated after being co-prescribed with fluoxetine.
Objectives: To discuss a case of phentermine-induced psychosis that could have been precipitated by CYP3A4 inhibition of phentermine by fluoxetine.
Methods: Miss X is a 61-year-old female with a history of major depressive disorder, generalized anxiety disorder, obesity, and rheumatoid arthritis. Her psychiatric symptoms were stable with oral fluoxetine 60 mg daily, oral aripiprazole 2mg daily, oral amitriptyline 100mg at night, and oral lorazepam 1mg daily. Miss X was prescribed oral phentermine 15mg daily for appetite suppression for weight loss. Subsequently, she started developing paranoid delusions against her family members, generalized anxiety, increased psychomotor activity, decreased appetite, and decreased sleep. Her symptoms continued to worsen even after discontinuing her medications on the 7th day. Miss X was eventually brought to the emergency room on the 14th day as her symptoms continued to deteriorate and she could not take care of herself.
Results: Miss X's symptoms resolved after a dose of Intramuscular injection of 2mg of lorazepam. No signs of serotonin syndrome were present during the examination. Drug-drug interaction between phentermine and fluoxetine is suspected to be a causative factor in the precipitation of psychosis as fluoxetine can inhibit the CYP3A4 metabolism of phentermine. Her electrocardiogram also demonstrated prolonged QTc (470ms), which could have been precipitated by co-prescribing phentermine and amitriptyline. Miss X was admitted to the inpatient psychiatric unit, and oral fluoxetine 60mg daily, oral aripiprazole 2mg daily, and oral lorazepam 1mg daily were restarted. Due to QTc prolongation oral trazodone 50mg daily was started instead of amitriptyline. After her psychiatric symptoms were stable on the medication regimen, Miss X was discharged on the third day of admission to the inpatient psychiatric unit.
Conclusions: Our case demonstrates the caution needs to be taken when prescribing phentermine not only for its neuropsychiatric side-effects but also for drug-drug interactions.

Disclosure of Interest: None Declared

Psychophysiology

EPV0856

Glutamatergic dysfunction, neuroplasticity, and redox status in patients with functional movement disorders

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Introduction: Functional Movement Disorders (FMD) are characterized by the presence of neurological symptoms that cannot be explained by typical neurological diseases or other medical conditions. First evidence showed that, compared to healthy controls (CTR), FMD patients presented increased levels of glutamate+glutamine in the anterior cingulate cortex/medial prefrontal cortex, and decreased levels of glutamate in the cerebrospinal fluid, suggesting that a glutamatergic dysfunction might play a role in FMD pathophysiology.
Objectives: According to the evidence of these abnormalities in many neuropsychiatric disorders at level of brain network activity, connectivity, and specific anatomic areas of altered metabolic, and given the evidence of a potential role of glutamate and BDNF in the pathophysiology of FND, in this study we aimed to assess circulating levels of glutamate, BDNF, dopamine, oxidative stress biomarkers, creatinine, neopterin and uric acid in patients with FMD and in a control group of healthy subjects.
Methods: 12 FMD patients (4 males, 8 females) and 20 CTR (4 males, 16 females) were recruited and underwent venous blood sampling and urine collection: levels of glutamate, BDNF, dopamine, oxidative stress, creatine, neopterin, and uric acid were analysed. Participants also underwent a psychometric assessment investigating depression, anxiety, and alexithymia.
Results: Levels of glutamate, BDNF and dopamine were significantly lower in the blood of FMD patients than CTR. Glutamate and dopamine levels were positively associated with levels of alexithymia.
Conclusions: Our findings give further evidence that glutamatergic dysfunction might be involved in the pathophysiology of FMD, possibly representing a biomarker of disease; moreover, since glutamatergic and dopaminergic system are closely interconnected, our results might have a relevance in terms of treatment options for FMD patients.

Disclosure of Interest: None Declared

EPV0857

Locus of control as a personal coping resource of a sportsman

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Introduction: In sports psychology, the issue of finding resources to overcome stress remains relevant at present. Currently, the priority is the search for personal resources that can help overcome difficult life situations. Currently, the priority is the search for personal resources that can help overcome difficult life situations. Research by many psychologists (Folkman S., Hobfoll S., Haan N.A., Heim E., Lazarus R., Moos R.N., Schaefer C., Grin O.R., Dementiy L.I., Kalnysh V.V., Tukaiev S.V., Khazova S.A. et al) is devoted to this topic. Among the coping resources, the authors single out motivation, locus of control, resilience, self-control, purposefulness, outlook, intelligence, etc.
Objectives: The purpose of the study is the analysis of literary sources regarding the study of the locus of control as a personal coping resource of an athlete.