

EPV0850

Weight gain as a secondary effect of Olanzapine, and its interference in treatment adherence: a case report.

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doi: 10.1192/j.eurpsy.2023.2153

Introduction: Olanzapine is a second generation antipsychotic that is approved for the treatment of schizophrenia, bipolar disorder type 1 as monotherapy, or as an add-on to lithium or valproate (manic or mixed episodes), and it is also used off label for acute anxiety, insomnia... It is one of the most effective antipsychotics but concerns remain due to its significant metabolic adverse effects. Olanzapine has one of the highest rates of weight gain among all antipsychotic drugs, which challenges patient's adherence to treatment.

Objectives: Review how much influence Olanzapine has on weight gain, its influence in treatment adherence and alternatives in clinical practice.

Methods: Presentation of a patient's case and review of existing literature, in regards to Olanzapine and its repercussions on weight gain and the old and new alternatives available right now.

Results: Olanzapine is an effective antipsychotic, however, it causes secondary effects that complicate treatment, especially weight gain. In the case presented, the patient does gain weight with Olanzapine and adherence is compromised. In these cases, professionals try to look for alternatives, either to try another drug or use adjunct treatment. In this patient, a change to lithium was made. In the last few years, adjunct treatment has gained traction, like for example: metformin and topiramate. The latest discovery in this matter are opioid antagonists: single dose oral tablet Olanzapine/samidorpham.

Conclusions: Even though Olanzapine is one of the most effective antipsychotics and medications in mental health, its impact on patients' weight hinders treatment continuity. The use of other already known medications and the appearance of new ones, that reduce weight gain probability, are the possible ways forward.

Disclosure of Interest: None Declared

EPV0851

Myocarditis induced by clozapine and COVID-19 infection: a case reportP. Veloso^{1*}, M. Gomes¹, R. Faria¹, D. Matos² and F. Pereira¹¹Psychiatry, Hospital de Braga, Braga and ²Neurology, Unidade Local de Saúde do Alto Minho, Viana do Castelo, Portugal

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doi: 10.1192/j.eurpsy.2023.2154

Introduction: Clozapine is the only available treatment for refractory schizophrenia and is rarely associated with the development of myocarditis. Usually, the onset of symptoms occurs within the first month of treatment. The symptoms of myocarditis include fever, flu-like symptoms, fatigue, and dyspnea, symptoms that overlap with the COVID-19 infection. Coronavirus has been associated

with cardiovascular complications, including myocarditis. It is not known whether clozapine increases the risk of developing viral myocarditis in patients with COVID-19 infection.

Objectives: Report a case of myocarditis in a patient treated with clozapine, who also had a history of COVID-19 infection.

Methods: Collection of clinical information and review of the literature.

Results: A 24-year-old man was admitted following severe psychotic symptoms that have been developing for the past several months. He presented with disorganized speech and behavior, paranoid delusions, thought alienation, auditory hallucinations, and blunted affect. He had no known medical co-morbidity, but he had tested positive for COVID-19 the month before admission. The lab and imaging tests and the electrocardiogram (EKG) were normal. He was diagnosed with schizophrenia and after treatment failure with three antipsychotics, the patient was started on clozapine, with symptom improvement. Two weeks after clozapine initiation, he started flu-like symptoms, fever, chest pain, and tachycardia. Lab tests showed leukocytosis (12 400 cells/uL), elevated inflammatory markers (C-reactive protein 143,30 mg/L) and cardiac biomarkers (troponin I 12.139 ng/mL, NT-proBNP 9321 pg/ml). The evaluation for viruses, including SARS-CoV-2, was negative. The EKG revealed ST-segment elevations and a trans-thoracic echocardiogram showed systolic dysfunction (left ventricular ejection fraction was 37%). Cardiac magnetic resonance confirmed severe left ventricular dysfunction and diffuse myocardial edema. The patient's symptoms resolved following the discontinuation of clozapine and supportive therapies. Troponin and EKG normalized over the following 7 days. By this time, the patient tested positive for COVID-19.

Conclusions: The temporal relationship with the initiation of clozapine supports the diagnosis of clozapine-associated myocarditis. However, the COVID-19 infection may have played a part in the emergence of cardiac alterations. We hypothesize that the co-occurrence of COVID-19 and clozapine treatment may act synergically as both factors increase the risk of developing myocarditis. However, further studies are needed to evaluate the relationship between these factors. While clinicians should stay alert for the risk of clozapine-associated myocarditis, the overall risk is low, and given the effectiveness of clozapine, as well as the absence of other evidence-based treatments, people with refractory schizophrenia should be given a monitored trial of clozapine, regardless of their COVID-19 status.

Disclosure of Interest: None Declared

EPV0853

Mirtazapine-induced psychosis on a young patient with severe malnutrition

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doi: 10.1192/j.eurpsy.2023.2155

Introduction: Mirtazapine is an antidepressant commonly prescribed to patients with depression and problems with weight and sleep. Case reports on Mirtazapine-induced psychosis either on initiation or increase in dosage in elderly patients and those with renal and liver impairment are found in the literature.

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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doi: 10.1192/j.eurpsy.2023.2156

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 which 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 received 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.

Disclosure of Interest: None Declared

EPV0855

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

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doi: 10.1192/j.eurpsy.2023.2157

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