

EPV0816

Sodium Oxybate-Induced secondary mania with psychotic symptoms: a case report and literature review

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Introduction: Sodium oxybate, an effective treatment for narcolepsy-associated daytime sleepiness and cataplexy, has been extensively. Despite its therapeutic benefits, sodium oxybate is not without its risks, and adverse psychiatric effects have been documented. This case report highlights a rare manifestation of sodium oxybate-related secondary mania with psychotic symptoms in a patient with narcolepsy, emphasizing the importance of recognizing and managing such adverse events. Additionally, we provide a brief review of similar cases reported in the literature.

Objectives: This report aims to describe the presentation, evaluation, and management of sodium oxybate-induced secondary mania with psychotic symptoms in a patient with narcolepsy. We also discuss the potential mechanisms underlying this adverse reaction and its clinical implications. Furthermore, we summarize findings from previous studies that have reported cases of secondary mania associated with sodium oxybate use.

Methods: We present the case of Mr. X, a 48-year-old male diagnosed with “Narcolepsy with cataplexy,” who had been receiving sodium oxybate treatment for 11 years. He was admitted to the hospital following a mild head injury and the emergence of a manic episode with psychotic features. Comprehensive clinical evaluation, including medical history, toxicology screening, and neuroimaging, was conducted.

Results: Upon evaluation, Mr. X exhibited hyperactivity, restlessness, grandiose delusions, paranoid delusions related to hospital staff, and decreased need for sleep. Notably, he had been consuming sodium oxybate excessively. Sodium oxybate was discontinued, and low-dose olanzapine was initiated. Within 24 hours, his manic and psychotic symptoms resolved. He admitted to overusing his medication, and his family reported a recent increase in his activity level. A review of the literature revealed similar cases of sodium oxybate-induced secondary mania with psychotic symptoms.

Conclusions: This case underscores the importance of vigilance for psychiatric side effects of sodium oxybate, particularly in patients with a history of substance abuse or potential overuse. Secondary mania associated with medications is a rare but significant clinical entity. Prompt recognition and intervention are crucial for patient safety and well-being. Further research is needed to elucidate the mechanisms underlying such reactions and to establish guidelines for their prevention and management.

Disclosure of Interest: None Declared

EPV0817

Brief psychotic disorder treatment with Olanzapine in a patient with Phelan-McDermid syndrome

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Introduction: The patient is a 50-year-old female, with multiple admissions in the PICU. At her first admission, at the age of 30 she presented the following main symptoms: mutism, negativism, crying and loss of bladder and bowel control. After collecting her complete family history, it was determined that her mother and one of her brothers were diagnosed with mild intellectual disability. Concerning her childhood history, she presented with late milestones as an infant and toddler and difficulties throughout primary education. Little information concerning her adult life was given, since the patient remained mute during the entirety of her first hospitalization.

Objectives: Determination of the efficacy of olanzapine in a patient with Phelan-McDermid syndrome with mild intellectual disability and psychotic symptoms such as auditory hallucinations, delusional ideas and disrupted behavior.

Methods: PANSS Test, intellectual capacity test, genetic testing.

Results: PANSS Scale Score at the 1st day of admission: 100

PANSS Scale Score at the last day: 79

Intellectual capacity test: mild intellectual disability

Genetic testing results: Phelan-McDermid syndrome

Conclusions: After 20 days, symptoms showed mild recession in response to 20mg of olanzapine. In a period of 12 months, the patient showed no signs of relapse and she was not readmitted in the PICU.

Disclosure of Interest: None Declared

EPV0818

Urinary retention induced by psychotropics: A case report

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Introduction: Neurological bladder is considered a functional disability that has a significant impact on the quality of life and psychological state of patients. Psychotropic drugs, in turn, can worsen the urinary dysfunction caused by this disease.

Objectives: Our objective is to illustrate, through the case of a patient suffering from a neurological bladder decompensated by the treatment of a characterized depressive episode, the link between these two pathologies.

Methods: We report the case of Ms. M.W., aged 51, with a history of high blood pressure stabilized under nebivolol and a neurological bladder diagnosed 10 years ago with episodic pollakiuria, admitted to the psychiatric department for repeated suicide attempts. She had never used psychoactive substances and had no family psychiatric history. The patient presented depressive symptoms evolving for 5 months. The diagnosis of a characterized depressive episode with melancholic features was made and the patient was treated with sertraline. From the first intake of the drug, the patient presented acute urinary retention (UR) requiring the placement of a permanent bladder catheter. The urinary symptoms improved upon stopping the treatment. Sertraline was changed to olanzapine and escitalopram. The patient stopped the treatment after one month because of the worsening of urinary symptoms requiring the installation of a suprapubic catheter. The urinary problem, together with the cessation of treatment, were responsible for a worsening of psychiatric symptoms leading to multiple suicide attempts. Given the advanced stage of the neurological bladder demonstrated by the urodynamic tests, our patient was treated with paroxetine, quetiapine and oxazepam along with psychotherapeutic education. The evolution was characterized by improvement in psychiatric symptoms and the urinary symptoms were stable.

Results: The lack of improvement after treatment discontinuation could be explained by an underlying neurological bladder manifesting with pollakiuria. The current literature on UR induced by psychotropic treatments is quite rare limited in case reports. This effect occurs especially when selective serotonin reuptake inhibitors (SSRIs) are prescribed in combination with other antipsychotics. Unlike first generation antipsychotics, atypical antipsychotics have muscarinic receptor antagonist properties which can induce UR. Among atypical antipsychotics, olanzapine has been shown to have the greatest antimuscarinic effects. Regarding SSRIs, they are associated with a lower risk of UR than other antidepressants and sertraline had the highest risk of UR.

Conclusions: SSRIs can induce UR particularly in combination with atypical antipsychotics. Coordination of care across multiple specialties and understanding the side effects of psychotropic medications can enable faster diagnoses and adequate management.

Disclosure of Interest: None Declared

EPV0819

False-Positive Urine Drug Screening in a Patient on Quetiapine

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Introduction: Urine drug tests are commonly used in psychiatry settings, mainly for the purpose of screening for substance abuse and excluding drug-induced psychiatric disorders. When carefully interpreted, these tests offer critical information for clinical judgement. However, certain psychotropic medications can trigger false-positive results in common urine drug screenings. For example, aripiprazole has been reported to cause false-positive urine amphetamine test results, and haloperidol has been associated with false-

positive urine drug tests for lysergic acid diethylamide (LSD). It is clinically significant to recognize some false-positive urine drug results and interpret certain results cautiously in clinical settings.

Objectives: We present a case of false-positive urine drug screening for tricyclic antidepressant (TCA) in a patient on quetiapine and aim to highlight the importance of accurate result interpretation in urine drug tests.

Methods: Details of the case were described. Information was gathered based on medical records.

Results: Mr. A, a 25-year-old construction worker, first presented at our hospital's emergency room on a Saturday in January 2023. He was brought by the police because he was aggressive and mentioned his colleagues were monitoring him. Being a foreigner, he did not have any prior medical records in our hospital. Urgent blood tests were performed, and organic causes were ruled out. He was started on quetiapine and lorazepam in the emergency room and was then admitted to our hospital.

A urine drug test was ordered on the following Monday, the third day of his admission. Surprisingly his urine drug screening revealed positive results for TCA and benzodiazepines. Initially as the patient was psychotic and could not give reliable history, we considered a few differential diagnoses, such as schizoaffective disorder and major depressive disorder with psychotic features, based on the presumption that TCA had been prescribed by the psychiatrist in Mr. A's home country. After further treatment, Mr. A became less psychotic and was able to share that he had a past psychiatric history of schizophrenia, but he had stopped antipsychotic medications four months ago.

Conclusions: This case report described a false-positive urine drug test for TCA while the patient was taking quetiapine. In this case, initially other diagnoses, such as schizoaffective disorder, were considered based on the incorrect assumption that patient was taking TCA.

False positive urine drug results can be confusing and misleading for clinicians. This report underscores the possibility of such false positives arising from quetiapine and emphasizes the critical importance of careful result interpretation.

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EPV0820

Syndrome of Irreversible Lithium-Effectuated Neurotoxicity: Silent, but not innocent

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Introduction: Lithium is one of the main drugs used in Bipolar Affective Disorder. However, it has a narrow therapeutic window, which requires close monitoring and progressive dose adjustment, according to serum levels, clinical response and the appearance of side effects. The term 'SILENT' explains descriptively persistent neurological sequelae related to lithium salt intoxication when symptoms persist for more than 2 months after stopping treatment. SILENT Syndrome is more common in females, at ages ranging