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SEROTONIN SYNDROME DUE TO MULTIPLE PSYCHIATRIC DRUGS INCLUDING CHLORPROMAZINE: A CASE REPORT AND

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ABSTRACT

Background: Serotonin syndrome is a potentially life-threatening adverse drug reaction resulting from excessive serotonergic activity in the central and peripheral nervous systems. It most commonly occurs following the use of multiple serotonergic medications, drug interactions, or recent dose escalation of psychiatric drugs. Early recognition is essential because the condition can rapidly progress and mimic several neurological and infectious disorders. **Case Presentation:** A 38-year-old male with a history of schizophrenia and depressive symptoms presented with agitation, confusion, excessive sweating, tremors, muscle rigidity, hyperreflexia, inducible clonus, tachycardia, hypertension, and hyperthermia following recent exposure to multiple psychotropic medications, including sertraline, chlorpromazine, and tramadol. Laboratory investigations revealed elevated creatine kinase levels, while infectious, metabolic, and structural neurological causes were excluded through appropriate diagnostic workup. The diagnosis of serotonin syndrome was established based on clinical findings and fulfillment of the Hunter Serotonin Toxicity Criteria. All suspected medications were discontinued immediately, and the patient was treated with intravenous fluids, lorazepam, cyproheptadine, external cooling measures, and close monitoring. **Conclusion:** The patient showed significant clinical improvement within 48 hours and achieved complete recovery without complications. This case highlights the importance of early recognition of serotonin syndrome, careful medication review, and prompt initiation of appropriate treatment. Increased awareness of potential drug interactions among psychiatric medications is essential to prevent serious morbidity and ensure favorable patient outcomes.

KEYWORDS: Serotonin Syndrome, Sertraline, Chlorpromazine, Tramadol, Psychotropic Drugs, Drug Interaction, Cyproheptadine, Adverse Drug Reaction.

INTRODUCTION

Serotonin syndrome is a serious drug-related condition that occurs when there is an excessive accumulation of serotonin in the body. Serotonin is a naturally occurring chemical messenger that helps regulate mood, sleep, appetite, body temperature, and several other important functions. While medications that increase serotonin levels are commonly used to treat depression, anxiety, and other psychiatric disorders, excessive serotonergic activity can lead to harmful effects. The syndrome is most often associated with the use of antidepressants, particularly selective serotonin reuptake inhibitors (SSRIs), either alone in high doses or in combination with other medications. Although serotonin syndrome is considered uncommon, its incidence has increased over recent years because of the growing use of psychiatric

medications and the frequent occurrence of polypharmacy in clinical practice.[1,2]

The clinical presentation of serotonin syndrome can vary from mild symptoms to severe life-threatening manifestations. Patients may initially experience restlessness, anxiety, sweating, tremors, diarrhea, and insomnia. As serotonin toxicity progresses, more severe symptoms such as confusion, hyperthermia, muscle rigidity, hyperreflexia, clonus, seizures, and cardiovascular instability may develop. Because these manifestations overlap with several other medical conditions, including neuroleptic malignant syndrome, sepsis, and anticholinergic toxicity, diagnosis can be challenging. In many cases, the condition is overlooked during the early stages, leading to delays in treatment. Therefore, awareness of the characteristic symptom pattern and careful review of medication history are essential for timely recognition and management of serotonin syndrome.[3,4]

Psychiatric patients are particularly vulnerable to serotonin syndrome because they often require multiple medications to control symptoms of depression, anxiety, psychosis, and behavioral disturbances. Drug interactions involving antidepressants, opioids, antipsychotics, and certain over-the-counter medications can significantly increase serotonergic activity. Tramadol is a commonly prescribed analgesic that possesses serotonin reuptake inhibition properties and has been frequently implicated in cases of serotonin toxicity when combined with antidepressants. Similarly, changes in medication dosage, addition of new psychotropic agents, or accidental overdosing may further increase the risk. Healthcare professionals must remain vigilant when prescribing combinations of medications that may influence serotonin pathways to minimize the likelihood of adverse drug reactions.[5,6]

The present case describes a patient who developed serotonin syndrome following exposure to multiple psychiatric medications, including sertraline, chlorpromazine, and tramadol. The patient presented with classical manifestations such as agitation, confusion, hyperthermia, diaphoresis, tremors, hyperreflexia, and inducible clonus shortly after recent medication adjustments. Prompt recognition of the syndrome, discontinuation of the offending agents, and initiation of supportive care along with cyproheptadine therapy resulted in complete recovery without complications. This case highlights the importance of thorough medication review, early diagnosis, and multidisciplinary management in preventing serious outcomes. It also emphasizes the need for increased awareness among clinicians regarding potential drug interactions and the risks associated with psychotropic polypharmacy.[7,8]

CASE PRESENTATION

Patient Demographics

A 38-year-old male was brought to the emergency department by his family with complaints of altered behavior, excessive sweating, tremors, muscle stiffness, and fever. The patient weighed 74 kg and had a known history of schizophrenia with depressive symptoms for the past 8 years. He had been receiving regular psychiatric treatment and was compliant with prescribed medications. There was no history of recent travel, exposure to infectious diseases, or substance abuse. The patient was living with his family and was independent in activities of daily living before the onset of the current illness.

Chief Complaints

The patient presented with the following complaints:

- Agitation and restlessness for 2 days
- Excessive sweating for 2 days
- Tremors involving both upper and lower limbs for 1 day
- Fever for 1 day
- Confusion and altered mental status for 12 hours
- Muscle rigidity and involuntary jerking movements for 12 hours
- Difficulty sleeping and increased anxiety for 3 days

History of Present Illness

The patient was apparently stable until five days before admission when he developed worsening depressive symptoms, insomnia, and increased anxiety. During a psychiatric follow-up visit, the dosage of sertraline was increased from 100 mg/day to 150 mg/day, and chlorpromazine was added for behavioral symptoms and sleep disturbances. Three days later, the patient developed severe lower back pain following strenuous physical activity. He consulted a local practitioner and was prescribed tramadol 50 mg three times daily. Within 48 hours of initiating tramadol, he began experiencing restlessness, anxiety, excessive sweating, and fine tremors of the hands. The symptoms progressively worsened over the next day and were accompanied by confusion, fever, muscle stiffness, involuntary limb jerking, and abnormal behavior. Family members reported that the patient became increasingly agitated, was unable to recognize relatives, and demonstrated purposeless movements. Due to the rapid progression of symptoms, he was brought to the emergency department for further evaluation and management.

Past Medical History

- Schizophrenia diagnosed 8 years previously
- Major depressive disorder with psychotic symptoms
- Essential hypertension for 5 years
- No history of diabetes mellitus
- No history of epilepsy or seizure disorders
- No previous episodes of serotonin syndrome
- No previous hospitalizations for drug toxicity
- No significant surgical history

Medication History

Medication	Dose	Frequency	Duration
Sertraline	150 mg	Once daily	5 years (dose increased recently)
Chlorpromazine	100 mg	Twice daily	5 days
Clonazepam	0.5 mg	Twice daily	2 years
Amlodipine	5 mg	Once daily	5 years
Tramadol	50 mg	Three times daily	3 days

No history of herbal supplements, over-the-counter serotonergic medications, or recreational drug use was reported.

Family History

- No family history of psychiatric disorders.
- No family history of neurological diseases.
- No hereditary metabolic disorders reported.
- No family history of adverse drug reactions.

Social History

The patient was married and lived with his wife and two children. He was a non-smoker and denied the use of tobacco products. Alcohol consumption was occasional, limited to social gatherings. There was no history of illicit drug use, substance abuse, or dependence. The patient maintained good family support and adherence to medical follow-up appointments.

Occupational History

The patient worked as an accountant in a private financial firm for the past 12 years. His work primarily involved desk-based administrative responsibilities. There was no occupational exposure to industrial chemicals, heavy metals, pesticides, solvents, or environmental toxins. No recent workplace stressors or occupational injuries were reported.

Allergic History

- No known drug allergies.
- No known food allergies.
- No history of allergic rhinitis.
- No history of bronchial asthma.
- No previous episodes of anaphylaxis or hypersensitivity reactions.

General Physical Examination

- Profuse sweating present
- Agitated behavior
- Tremulous movements observed
- No pallor
- No cyanosis
- No clubbing
- No pedal edema

Vital Signs

Parameter	Value
Temperature	39.2°C
Pulse Rate	128 beats/minute
Blood Pressure	168/96 mmHg
Respiratory Rate	24 breaths/minute
Oxygen Saturation	98% on room air

Systemic Examination

Mental Status Examination

- Conscious but confused
- Disoriented to time and place
- Reduced attention span
- Agitated and restless behavior
- Impaired short-term memory

Neuromuscular Findings

- Generalized hyperreflexia
- Bilateral inducible ankle clonus
- Ocular clonus present
- Increased muscle tone
- Lower limb rigidity
- Fine tremors involving upper extremities
- Intermittent myoclonic jerks

Cranial Nerve Examination

- Normal findings
- Pupils equal and reactive to light
- No focal neurological deficits

Cardiovascular Examination

- Tachycardia present
- Normal heart sounds
- No murmurs detected
- Peripheral pulses palpable and symmetrical

Respiratory Examination

- Bilateral air entry equal
- No wheeze or crepitations
- Normal respiratory effort

Abdominal Examination

- Abdomen soft and non-tender
- No hepatosplenomegaly
- Normal bowel sounds

Laboratory Investigations

Hematological Investigations

Investigation	Result	Reference Range
Hemoglobin	13.9 g/dL	13–17 g/dL
Total Leukocyte Count	13,200/mm³	4,000–11,000/mm³
Neutrophils	78%	40–75%
Lymphocytes	18%	20–45%
Platelet Count	2.68×10^5/mm³	$1.5–4 \times 10^5$/mm³
ESR	16 mm/hr	<20 mm/hr

Biochemical Investigations

Investigation	Result	Reference Range
Blood Glucose	102 mg/dL	70–140 mg/dL
Blood Urea Nitrogen	22 mg/dL	7–20 mg/dL
Serum Creatinine	0.9 mg/dL	0.6–1.2 mg/dL
Sodium	138 mmol/L	135–145 mmol/L
Potassium	4.2 mmol/L	3.5–5.0 mmol/L
Chloride	102 mmol/L	98–107 mmol/L
Calcium	9.0 mg/dL	8.5–10.5 mg/dL

Liver Function Tests

Investigation	Result	Reference Range
AST	38 U/L	<40 U/L
ALT	35 U/L	<45 U/L
ALP	98 U/L	44–147 U/L
Total Bilirubin	0.8 mg/dL	0.2–1.2 mg/dL

Muscle Injury Markers

Investigation	Result	Reference Range
Creatine Kinase (CK)	968 U/L	30–200 U/L
LDH	295 U/L	140–280 U/L

Inflammatory Markers

Investigation	Result	Reference Range
C-Reactive Protein	4.5 mg/L	<10 mg/L
Procalcitonin	0.08 ng/mL	<0.5 ng/mL

Toxicology Screening

Investigation	Result
Alcohol Screen	Negative
Urine Drug Screen	Negative
Toxicology Screen	Negative

Microbiological Investigations

Investigation	Result
Blood Culture	No Growth
Urine Culture	No Growth

Radiological Investigations

Computed Tomography (CT) Brain

- No acute intracranial pathology.
- No hemorrhage or infarction.

Magnetic Resonance Imaging (MRI) Brain

- Normal study.
- No evidence of encephalitis or structural lesions.

Chest X-ray

- Normal lung fields.
- No evidence of infection.

Electrocardiogram (ECG)

- Sinus tachycardia (128 beats/minute)
- Normal PR interval
- Normal QT interval

Final Diagnosis

The final diagnosis of serotonin syndrome was established based on the patient's clinical presentation and recent exposure to multiple serotonergic and psychotropic medications, including sertraline, chlorpromazine, and tramadol. The patient exhibited the characteristic triad of serotonin toxicity consisting of altered mental status (confusion, agitation, disorientation), autonomic dysfunction (hyperthermia, tachycardia, hypertension, diaphoresis), and neuromuscular hyperactivity (tremors, hyperreflexia, inducible clonus, myoclonus, and muscle rigidity). The rapid onset of symptoms following medication adjustments, together with the fulfillment of the Hunter Serotonin Toxicity Criteria and exclusion of alternative diagnoses, confirmed serotonin syndrome as the definitive diagnosis.

Differential Diagnosis

Several conditions were considered before confirming serotonin syndrome. Neuroleptic malignant syndrome (NMS) was suspected because of fever, rigidity, and altered mental status; however, the presence of hyperreflexia and inducible clonus favored serotonin syndrome. Sepsis was excluded due to negative cultures and absence of an infectious source. Anticholinergic toxicity was considered but was unlikely because the patient had diaphoresis rather than dry skin and mucous membranes. Drug-induced delirium could explain confusion but not the prominent neuromuscular findings.

Meningoencephalitis was ruled out by normal neuroimaging and lack of meningeal signs. Heat stroke and malignant hyperthermia were also considered but were clinically less consistent.

TREATMENT

Upon admission, all suspected offending medications, including sertraline 150 mg once daily, tramadol 50 mg three times daily, and chlorpromazine 100 mg twice daily, were immediately discontinued. The patient was shifted to a high-dependency unit for continuous monitoring. Supportive management was initiated with 0.9% normal saline infusion at 100 mL/hour intravenously to maintain hydration and prevent renal complications. Oxygen was administered through a nasal cannula at 2 L/min to maintain oxygen saturation above 94%. For agitation, tremors, and neuromuscular hyperactivity, lorazepam 2 mg intravenously every 4–6 hours as needed was administered. Specific therapy was initiated with cyproheptadine 12 mg orally as a loading dose, followed by 2 mg orally every 2 hours until clinical improvement was observed. Hyperthermia was managed using cooling blankets, ice packs, and tepid sponging. Continuous cardiac monitoring and hourly vital sign assessments were performed.

On the second day, the patient showed partial improvement in agitation and autonomic instability. Intravenous hydration with 0.9% normal saline at 100 mL/hour was continued. Following initial symptom control, cyproheptadine therapy was transitioned to a maintenance regimen of 8 mg orally every 6 hours. Lorazepam requirements decreased, and 1–2 mg intravenously every 6 hours as needed was administered for residual agitation and tremors. The patient's blood pressure remained elevated intermittently, reaching 170/100 mmHg; therefore, labetalol 10 mg intravenously over 2 minutes as needed was administered on one occasion. Serial monitoring of serum electrolytes, renal function, liver function, and creatine kinase levels was continued.

By the third day, the patient's body temperature had normalized, and mental status had significantly improved. Cyproheptadine was continued at 8 mg orally every 6 hours. Intravenous fluids were reduced to 75 mL/hour because oral intake had improved. Lorazepam was switched to 1 mg orally every 8 hours as needed for mild residual anxiety and restlessness. Creatine kinase levels showed a downward trend, indicating improvement in muscle injury secondary to excessive neuromuscular activity. No further antihypertensive intervention was required. Physical therapy and gradual mobilization were initiated to prevent deconditioning.

On the fourth hospital day, hyperreflexia, tremors, and inducible clonus had completely resolved. Cyproheptadine therapy was continued at 8 mg orally every 6 hours for an additional 24 hours to ensure sustained symptom resolution. Intravenous fluids were discontinued, and the patient was encouraged to maintain oral hydration of approximately 2–2.5 liters/day. Lorazepam was required only once at 1 mg orally for mild anxiety. Repeat laboratory investigations showed near-normal creatine kinase levels and stable renal and hepatic function. No complications such as rhabdomyolysis, acute kidney injury, seizures, or respiratory compromise were observed.

The patient remained asymptomatic on day five. Cyproheptadine was tapered to 4 mg orally every 6 hours and subsequently discontinued after 24 hours. No recurrence of serotonin toxicity symptoms was noted. Daily psychiatric review was performed to evaluate future psychotropic therapy and to avoid medications with significant serotonergic interactions. Nutritional support, hydration, and routine nursing care were continued. Vital signs remained stable, and neurological examination was normal.

On day six, the patient continued to remain clinically stable without evidence of recurrent autonomic or neuromuscular abnormalities. No further cyproheptadine or benzodiazepine therapy was required. Psychiatric consultation recommended initiation of a revised treatment regimen after complete recovery. The patient and family received counseling regarding avoidance of unsupervised medication use, particularly serotonergic antidepressants and opioid analgesics such as tramadol. All laboratory parameters had returned to normal ranges.

The patient was discharged on the seventh hospital day in a stable condition. At discharge, vital signs were normal, mental status was fully restored, and neurological examination revealed no residual abnormalities. Laboratory investigations including complete blood count, renal function tests, liver function tests, electrolytes, and creatine kinase were within normal limits. The patient was advised to follow up with the psychiatry department after one week for reassessment and initiation of an alternative long-term psychiatric treatment plan. Written instructions were provided regarding recognition of early symptoms of serotonin syndrome and the importance of informing healthcare providers about previous serotonin toxicity before receiving new medications.

DISCUSSION

Serotonin syndrome is a potentially life-threatening adverse drug reaction caused by excessive serotonin activity within the central and peripheral nervous systems. In the present case, the patient developed agitation, confusion, diaphoresis, tremors, hyperreflexia, inducible clonus, tachycardia, and hyperthermia shortly after exposure to multiple psychotropic medications. The temporal relationship between medication changes and symptom onset strongly supports drug-induced serotonin toxicity. Similar findings were reported by Mason and Blackburn, who described serotonin syndrome following concomitant administration of sertraline and tramadol,

where patients developed confusion, agitation, diaphoresis, tachycardia, and tremors shortly after dose escalation. Likewise, Chan reported serotonin syndrome even with low-dose sertraline exposure, emphasizing that serotonin toxicity can occur unexpectedly and requires early recognition by clinicians. These reports support the clinical presentation observed in this patient.[9,10]

A notable aspect of this case was the combination of an SSRI with tramadol and concurrent psychiatric medications. Tramadol possesses serotonin reuptake inhibition properties in addition to its opioid activity, making it a recognized precipitating factor for serotonin syndrome when combined with antidepressants. The patient's symptoms emerged rapidly after tramadol initiation, which is consistent with previously published literature. Nayyar described a patient receiving sertraline and trazodone who developed serotonin syndrome after tramadol exposure, presenting with delirium, agitation, hyperreflexia, diaphoresis, and fever. Similarly, Ferreira de Lemos reported serotonin syndrome in a patient taking antidepressants and tramadol, highlighting the importance of recognizing high-risk drug combinations. These studies reinforce the role of polypharmacy and medication interactions as major contributors to serotonin toxicity.[11,12]

The diagnosis of serotonin syndrome remains primarily clinical because no laboratory test can definitively confirm the condition. In this patient, the presence of inducible clonus, hyperreflexia, tremors, autonomic instability, and altered mental status fulfilled the Hunter Serotonin Toxicity Criteria. Dunkley and colleagues demonstrated that the Hunter Criteria provide superior sensitivity and specificity compared with earlier diagnostic systems and remain the preferred diagnostic tool worldwide. Furthermore, Caamano et al. reported a patient with serotonin syndrome induced by tramadol and escitalopram who presented with agitation, tachycardia, fever, and altered mental status, closely resembling the manifestations observed in the current case. These studies highlight the importance of recognizing characteristic neuromuscular signs, particularly clonus and hyperreflexia, which help distinguish serotonin syndrome from other neurological emergencies.[13,14]

The favorable outcome in this case was largely attributable to prompt discontinuation of the offending medications and initiation of appropriate supportive therapy. Management included withdrawal of serotonergic agents, intravenous fluids, benzodiazepines for agitation, external cooling measures, and cyproheptadine administration. Clinical improvement became evident within 24–48 hours, with complete resolution of symptoms during hospitalization. Similar successful outcomes have been reported in recent literature. Liu et al. described a patient who developed serotonin syndrome associated with psychiatric medications and improved after early recognition and cessation of causative drugs. Likewise, Duignan and colleagues reported sertraline-induced serotonin syndrome that resolved rapidly following withdrawal of the medication and supportive treatment. These observations emphasize that early diagnosis and intervention remain the most important determinants of patient prognosis and recovery.[15,16]

CONCLUSION

Serotonin syndrome is a potentially life-threatening but largely preventable adverse drug reaction that can occur following the use of multiple serotonergic medications or drug interactions involving psychotropic agents. The present case highlights the importance of maintaining a high index of suspicion in patients presenting with the characteristic triad of altered mental status, autonomic instability, and neuromuscular hyperactivity after recent medication initiation or dose escalation. Early recognition of clinical features such as agitation, diaphoresis, tremors, hyperreflexia, and inducible clonus is crucial for timely diagnosis. Prompt discontinuation of the offending medications, aggressive supportive care, and administration of cyproheptadine resulted in complete recovery without long-term complications. This case emphasizes the need for careful medication reconciliation, avoidance of high-risk drug combinations, and close monitoring of patients receiving multiple psychiatric medications. Increased awareness among healthcare professionals regarding serotonin syndrome can facilitate early intervention, reduce morbidity, and improve patient outcomes. Furthermore, multidisciplinary involvement, including psychiatrists, physicians, and clinical pharmacists, plays a vital role in preventing recurrence and ensuring safe long-term pharmacotherapy.

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