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A Case Report of Neuroleptic Malignant Syndrome

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ABSTRACT

Neuroleptic malignant syndrome (NMS) is a life-threatening reaction to anti-psychotic medications that is characterised by fever, altered mental status, muscle rigidity and autonomic dysfunction. Not only hast it been associated with all anti-psychotic medications, but also with plenty other medications that affect the central dopaminergic neurotransmission like metoclopramide, promethazine, reserpine and many others. In our case, a 17 year old female who presented to the emergency room with multiple fractures was a known case of depression with bipolar affective disorder (BPAD) on irregular therapy. On admission sertraline mg, haloperidol mg and trihexyphenidyl mg per oral were started. Five days after, she developed fever, tachycardia, muscle rigidity, sialorrhea and drowsiness. Hence, was promptly diagnosed to have NMS with the help of history, clinical and laboratory findings. However uncommon, NMS must be a part of differential diagnosis in cases that present with fever and altered mental status. Ruling out NMS in the initial stages of management, reduces the chances of substantial morbidity and mortality.

Keywords: Altered mental status, Autonomic dysfunction, Hyperthermia, Muscle rigidity, Neuroleptics

INTRODUCTION

Neuroleptic Malignant Syndrome (NMS) is a rare but potentially life-threatening complication associated with the use of neuroleptics. Neuroleptics, otherwise known as anti-psychotic medications are used to treat a variety of psychiatric disorders. They are broadly classified into first generation or typical & second generation or atypical anti-psychotic drugs. The most life-threatening emergency associated with the use of neuroleptics is a neuroleptic malignant syndrome. The first reported case of NMS appeared in 1956, shortly after the introduction of the antipsychotic drug chlorpromazine (Thorazine).[1] This syndrome can occur from a single dose, increasing dose, or the same dose. It is mostly associated with the first-generation antipsychotics, but can also occur to a lesser degree with the second-generation antipsychotics, antiemetics (metoclopramide, promethazine), and from the withdrawal of anti-Parkinson medication.[2] NMS presents as a tetrad; that includes hyperthermia, muscle rigidity, altered mental status and autonomic dysfunction. Prompt recognition and correlation of symptoms remains the mainstay in the treatment of NMS.

CASE REPORT:

A 17-year-old female, known case of depression with bipolar affective disorder (BPAD) for 6 months, on irregular treatment for the same presented at the emergency room with multiple fractures. After evaluation she was prescribed sertraline 25 mg, haloperidol 5 mg and trihexyphenidyl 2 mg orally once a day and was posted for open reduction with internal fixation (ORIF) with dynamic hip screw (DHS) surgery electively. Five days after re-starting

neuroleptics, she developed fever spikes ranging from 104 -105 °F, tachycardia (heart rate >120 bpm), drowsiness, borderline raised blood pressure, diaphoresis, sialorrhea and dilated pupils. Infective work up including paired blood cultures were negative. Acute kidney injury and raised creatinine phosphokinase levels were observed (Table 1). Clinical scenario along with the laboratory reporting raised a strong suspicion NMS. Immediately the offending drugs were discontinued and the patient was given symptomatic treatment with anti-pyrectics, fluid therapy, tepid sponging and supportive care. Patient's symptoms and laboratory values resolved over a course of two weeks. After optimizing the patient, she was operated without any untoward complications.

DISCUSSION:

NMS is associated with use of anti-psychotic drugs also known as neuroleptics and drugs that affect the central dopaminergic neurotransmission. The abrupt cessation or reduction in dose of dopaminergic medications such as levodopa in Parkinson disease may also precipitate NMS.[3]

Patients typically develop NMS within hours or days after exposure to a causative drug, with most exhibiting symptoms within 2 weeks and nearly all within 30 days.[4] In our case, the patient developed symptoms five days after restarting the anti-psychotics for depression. The clinical course typically begins with muscle rigidity followed by a fever within several hours of onset and mental status changes that can range from mild drowsiness, agitation, or confusion to a severe delirium or coma.[5] NMS in hospitalised patients must be considered as an emergency as any delay in intervention may lead to significantly higher chances of morbidity or mortality. While the mainstay of treatment is to discontinue the offending drugs and start symptomatic management by adequate fluid therapy, especially if creatinine phosphokinase levels are high and possess a threat of kidney injury. Therapeutic measures like tepid sponging, cooling blankets etc must be advised to lower the temperature. Metabolic disturbances if any must be corrected, loading dose of sodium bicarbonate must be considered as it may help in lowering the chances of acute renal failure. Patients with NMS may be at increased risk of morbidity due to renal failure and disseminated intravascular coagulation (DIC) secondary to rhabdomyolysis.[6] If diagnosed early and managed aggressively, NMS is mostly not fatal and reduces scope of morbidity. Hence, early recognition and intervention is of utmost importance in reducing incidence of severe cases of NMS.

CONCLUSION:

Neuroleptics are the primary modalities of treatment in a variety of neuro-psychiatry and behavioural disorders. Therefore, awareness about clinical features of NMS and its management is crucial as early recognition and treatment are the main goals of management of patients with NMS.

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Day	*TLC*	*Creatinine*	*Urea*	*Uric Acid*	*Creatinine Phosphokinase*
01	7569/cm^3	1.53 mg/dl	102 mg/dl	56 mg/dl	584 U/L
02	6500/cm^3	2.36 mg/dl	126 mg/dl	64 mg/dl	625 U/L
03	6893/cm^3	3.80 mg/dl	132 mg/dl	79 mg/dl	745 U/L
07	6300/cm^3	1.60 mg/dl	45 mg/dl	10 mg/dl	227 U/L

Table 1: Day-wise laboratory readings of total leucocyte count, serum creatinine, serum urea, serum uric acid, serum creatinine phosphokinase levels.

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