Neuroleptic Malignant Syndrome (NMS) after Treatment with Metoclopramide - A Rare Case Report

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INTRODUCTION

Neuroleptic Malignant Syndrome (NMS) is a medical emergency. It presents with mental status change, rigidity, fever, and autonomic dysfunction. It is caused by antipsychotics especially neuroleptic agents and certain antiemetic drugs like metoclopramide that block central dopamine pathways. We present a case of a 32-year-old male chronic alcoholic who presented to us with alcohol withdrawal symptoms and was given injection metoclopramide and developed neuroleptic malignant syndrome (NMS).

Incidence rates for NMS range from 0.02 to 3 percent in patients treated with antipsychotic agents.[1,2] Though it is commonly encountered in young adults, any age group can be affected.[3,4] Males are two-fold more affected than females.[4]

Metoclopramide, a commonly used anti-emetic agent has anti-dopaminergic properties and can give rise to development of NMS. The signs and symptoms of NMS include hyperthermia, altered mental status, muscular rigidity resembling extrapyramidal rigidity, autonomic instability, diaphoresis, hyper salivation, dysphagia, tachycardia, hypertension. NMS if left untreated, is usually fatal. So, early recognition and treatment is of great importance.

PRESENTATION OF CASE

A 25-year-old male, labourer by occupation was admitted with complaints of fever, confusion, decrease in activity with stiffness of whole body and difficulty in deglutition since 4 days. On asking leading questions, the relative revealed that one week prior to onset of these features, he was admitted to a private nursing home for food poisoning and was treated with intravenous antibiotics and metoclopramide 10 mg 6 doses over 2 days and was discharged. He developed the above symptoms the next day after discharge. His past medical history was unremarkable. He was non-alcoholic. There was no history of hypertension and diabetes mellitus.

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On examination, pulse was 110 / min, regular, blood pressure was 148 / 88 mmHg. Other systemic examination was normal. On neurological examination, he was found to be confused. GCS: was 13 (E - 4, V - 4, M - 5). His speech was hypophonic and slurred. Face was mask like. Hyper salivation was present. Cranial nerve examination revealed normal pupillary reflex, no ophthalmoplegia and normal gag reflex. Motor system examination showed generalised lead- pipe type of rigidity in all four limbs. Power overall was grade 4. Tremors were present in lower extremities and cogwheeling was present in wrist joint. Deep tendon reflexes were brisk. Bilateral plantars showed withdrawal responses. Gait revealed flexion posture, no arm swing and turning en bloc because of rigidity.

Investigations: Hb - 10 gram %, TLC - 5600 / mm^3, Malaria antigen test, IgM Dengue, blood and urine culture was normal. CXR was normal. Urine was positive for myoglobin. Blood urea - 78 mg / dL, serum creatinine - 3.8 mg / dL, serum LDH - 188 U / L (normal - 140 - 280), serum total CPK - 1028 U / L (normal; 40 - 308), serum sodium was normal, potassium was 5.8 mg / dL. LFT was normal. CT brain was normal. After ruling out other possible infections and taking into consideration the extra pyramidal symptoms along with high CPK levels, and causality of patient condition with the metoclopramide administration, a clinical diagnosis of the neuroleptic malignant syndrome was made.

The patient was treated with IV fluids, IV calcium gluconate, glucose insulin drip, carbidopa 25 mg / levodopa 100 mg PO BID, baclofen 5 mg PO BID for rigidity. He became afebrile after 12 hours. Repeat CPK levels after 24 hours was 580 U / L and physical examination showed improvement in rigidity. Renal profile became normal by 4th day, repeat urine examination did not show myoglobinuria. Patient was discharged on the 5th day.

DISCUSSION

NMS was first described by Delay et al in 1968.[5] The classical features of NMS are fever, muscle rigidity, tachycardia, diaphoresis, hypertension and altered mental status.[6] Dopamine receptor blockade gives rise to NMS.[7,8]

The incident development of NMS after exposure to neuroleptic agents is approximately 0.5 % to 1.4 %. It can also occur as a side effect of non-neuroleptic agents like metoclopramide, that blocks the central dopaminergic pathways in brain.[9,10]

The common side effects of metoclopramide are akathisia, dystonia, oculogyric crisis but rarely it can cause serious effects like NMS. Dystonic reactions usually involve locally in face, neck or back, but it does not affect mentation like NMS.[11]

Metoclopramide induced dystonia usually involves the face, neck and back muscles sparing the higher functions of brain. Myoglobinuric renal failure secondary to rhabdomyolysis can occur like in our case. We presume that food poisoning induced vomiting and dehydration and administration of metoclopramide lead to NMS in our patient. The treatment of NMS includes stopping the offending agent, correction of dehydration and electrolyte imbalance, treating sepsis (if present) with antibiotics, DVT prophylaxis. The drugs used to treat NMS are IV dantrolene, benzodiazepines, dopamine agonists like bromocriptine, levodopa / carbidopa, amantadine, baclofen etc.[11] Few evidence based studies suggest that ECT is a reasonable treatment modality.[12,13] Most episodes resolve within two weeks.[3] Mortality rates vary between 5 to 20 percent.[1,3]

CONCLUSIONS

NMS is a rare fatal complication of metoclopramide therapy. Treatment usually is supportive in the form of stopping of the offending drug, correction of hydration, and electrolyte imbalance. Specific treatment is with drugs like dantrolene, dopamine agonists, and benzodiazepines.

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