Neuroleptic Malignant Syndrome with Acute Renal Failure Associated with Rhabdomyolysis: A Case Report

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ABSTRACT

Neuroleptic malignant syndrome is a rare, life threatening neurologic emergency characterized by fever, rigidity, autonomic instability, mental status changes and an elevated creatine kinase level. It often occurs shortly after the initiation of neuroleptic treatment, or after dose increase. The management of patients with NMS is based upon clinical severity and includes supportive care, withdrawal of antipsychotic agent and agents like bromocriptine and dantrolene. Complications include acute renal failure associated with rhabdomyolysis, respiratory failure, electrolyte imbalance, hepatic failure, seizures from hyperthermia and metabolic derangements, etc.

KEYWORDS
Neuroleptic malignant syndrome, Creatine kinase level, Fever, Rigidity, Autonomic instability, Hyperthermia, Antipsychotic agent

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INTRODUCTION

NMS is a life threatening neurologic emergency associated with use of dopamine antagonists, and less commonly with dopamine agonist withdrawal. First generation antipsychotic agents (e.g., haloperidol, fluphenazine) are most commonly implicated but NMS can occur with any antipsychotic agent including second generation drugs (e.g., clozapine, risperidone, olanzapine) and also with antiemetic drugs (eg, metoclopramide, promethazine and levosulpride). Incidence rates for NMS range from 0.02%–3% among patients taking antipsychotics case control studies implicate recent or rapid dose escalation, a switch from one agent to another, and parenteral administration as risk factors. Clinical features include tetrad of typical symptoms including: Fever (typically above 38 degree celcius), rigidity (generalized, lead pipe rigidity), mental status changes and dysautonomia manifesting as tachycardia, labile blood pressure, tachypnea, arrhythmias.[1] Elevated creatine kinase, typically more than 1000 international units/L. An international multispeciality consensus group published diagnostic criteria for NMS in 2011.[2] Differential diagnosis include meningitis, encephalitis, serotonin syndrome, malignant hyperthermia, malignant catatonia, other drug related syndromes (withdrawl of intrathecal baclofen therapy, acute intoxication with cocaine, MDMA, etc.) A case is described that highlights the challenges met in making the diagnosis, treatment and final outcome of the patient.[3]

CASE PRESENTATION

A 67 year old female with history of BPAD for the last 30 years, Parkinson's disease? Drug induced, hypothyroidism sought psychiatry consultation for altered behavior in the form of restlessness, decrease response to conversation and pacing around purposelessly which was noticed by her family. A provisional diagnosis of exacerbation of psychosis was made and she received Inj Haloperidol, Inj Phenergan (promethazine), Inj Lorazepam, Tab Olanzapine, Tab Venlafaxine (75 mg OD), Tab Divalproex-500 mg, Tab Lamotrigene (100), Tab Paroxetine, Tab Cariprazine, Tab Trihexyphenidyl, Tab Syndopa Plus, Tab Donepezil, Tab Thyroxine (25 mcg). 2 days after admission to psychiatric ward, the patient developed agitation, decrease responsiveness and anuria with high colored urine for which she presented to our hospital for further treatment. On examination her urine output was 100 ml since 2 days, although normotensive (BP 110/70 mmHg) she had tachycardia (heart rate 110 beats/min), and exhibited parkinsonian features including rigidity (lead pipe rigidity).[4] An electrocardiogram revealed no acute ischemic changes. Cardiovascular, respiratory and abdominal examinations were unremarkable. 6 hrs after admission she developed fever (temperature 102 degree Fahrenheit) and diaphoresis. Laboratory investigations revealed markedly elevated CK levels -168593 U/L, raised LDH levels -3243 U/L, elevated SGOT/PT levels -1836/128 U/L, alkaline phosphatase -82 U/L, raised neutrophil count -90%, urine R/E showed albuminuria (++++), elevated serum creatinine level 8.4 mg/dl, raised urea level -16 mg/dl. Serum electrolytes showed potassium -4.4 meq/L, sodium -136 meq/L, calcium -8.4 mg/dl, serum uric acid -8.8 mg/dl. NCCT head revealed cerebral atrophy. CSF study was normal (Table 1).[5]

Investigation	On admission	Day 3	Day 10
S-CK U/L	168593	50797	568
S-LDH U/L	3243	1508	352
S-Creatinine mg/dl	8.4	5.4	7.7
S-Sodium meq/l	136	138	131
S-Potassium meq/l	4.4	3.9	3.5
S-Calcium mg/dl	8.4	8.5	9.3
S-GOT U/L	1836	938	28
S-GPT U/L	128	224	36
TLC/cu mm	8500	5700	12000

Table 1. Revealed CK Levels After Admission.

A firm diagnosis of NMS with AKI with rhabdomyolysis was made. The patient was taken for hemodialysis on the same day of admission in view of AKI due to rhabdomyolysis. The patient was kept in intensive care unit. Tablet Bromocriptine was started (2.5 mg TDS) which was tapered over the course of 14 days and then stopped. For her agitation Inj. LORAZEPAM in the dose 1 mg iv/im every 6 hrly was given Inj. DIAZEPAM (10 mg i/v slowly) single dose was also helpful. The patient underwent regular hemodialysis for her ATN due to rhabdomyolysis, however her other lab parameters showed decreasing trend. After a few days she developed focal seizure one episode. NCCT Head (Image 3) was repeated and subsequently MRI brain (Image 4) was done which revealed increased hypodense areas in B/L cerebral and cerebellar hemispheres. MRI brain was suggestive of encephalopathy. Her GCS dropped in view of the same we continued the supportive therapy and she was started steroids. The patient's clinical status and GCS improved significantly afterwards and she was discharged in hemodynamically stable state after 20 days.[6]

RESULTS AND DISCUSSION

This case illustrates one of the many clinical presentations possible with the depot use of potent antipsychotic drugs and the many difficulties faced during her treatment due to complications of NMS. The diagnosis of NMS was established based on typical history of depot drug administration and other characteristic features including markedly raised serum CPK levels, worsening mental status, muscle rigidity and diaphoresis.[7] Our patient developed rhabdomyolysis and landed into acute kidney injury. Even with more sensitive criteria, a high index of suspicion is still necessary for clinicians to make a prompt diagnosis based on clinical history.

CONCLUSION

Neuroleptic Malignant Syndrome (NMS) is a life threatening neurologic emergency associated with use of antipsychotic (neuroleptic) agents and characterized by a distinctive clinical syndrome of mental status change, rigidity, fever, and dysautonomia. Incidence rates for NMS range from 0.02%–3% among patients taking antipsychotic agents. While most patients with NMS are young adults, the syndrome has been

described in all age groups from 9-78 yrs. In most studies males outnumber females twofold. This was a rare case where a female was affected with many complications of NMS. A rapid loading schedule, especially with potent neuroleptics like haloperidol is considered to be the principal contributing factor in the development of NMS, by causing a sudden and massive down regulation of dopaminergic transmission. This could have occurred in our patient.

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