VOLUME - 10, ISSUE - 12, DECEMBER - 2021 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper

General Medicine

NEUROLEPTIC MALIGNANT SYNDROME: A RARE CASE REPORT

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ABSTRACT The neuroleptic malignant syndrome is idiosyncratic reaction to antipsychotic medication. It is an iatrogenic unpredictable neurologic emergency that to be diagnosed early to prevent morbidity and mortality. The distinctive clinical features are fluctuating mental status, hyperpyrexia, dystonia, and generalised rigidity. If not diagnosed early, it can be lethal and even may lead to death of the patient. The greater awareness, early diagnosis and advanced critical care had decreased the morbidity and mortality of the syndrome recently. Using this case study as an example this article discusses the prevalence of NMS even in a small dose of Haloperidol in the elderly.

KEYWORDS : Anti-psychotic, Early diagnosis, Neuroleptic malignant syndrome

INTRODUCTION:

The neuroleptic malignant syndrome is caused due to the use of neuroleptics. It's rare but potentially life-threatening neurologic emergency associated with significant mortality and morbidity. The incidence rate is 0.02% to 3% among patients taking neuroleptics. Mostly it is associated with high potency neuroleptics (e.g., haloperidol, fluphenazine). It can also be associated with low-potency neuroleptics (e.g., chlorpromazine), atypical antipsychotics (e.g., clozapine, risperidone, olanzapine) and antiemetic drugs (e.g. metoclopramide, promethazine). It is characterized by fever, muscular rigidity, altered mental status, autonomic dysfunction, and elevated creatine phosphokinase.

CASE REPORT:

This 70-year Diabetic and hypertensive female presented with fever, breathlessness, and cough with expectoration. At presentation, her vitals were stable (PR 82/min, Temp 98.7 F, BP 134/86 mm of Hg RR 20/min), Systemic examination revealed B/L diffuse wheeze. Rest of the examination was unremarkable (GCS was 15/15 no rigidity tremor and anisocoria). She had hyponatremia (112 Meq/l), while other investigations were normal. With a provisional diagnosis of Hyponatremia, SHTN, Type 2 Diabetes mellitus and OAD. She was treated with antibiotics, Bronchodilators, insulin, anti-hypertensives, and Na supplementation. Her sodium was corrected wheeze resolved and no fever further. During her 7 the day of stay in the hospital, she developed delirium for which Inj Haloperidol 2.5 mg intramuscularly was given. The next day she had fluctuating consciousness with fever refractory to medical measures, profuse sweating, and dystonic movements.

On examination Vitals (HR 121 beats/min BP 142/88 mm Hg, RR 26 breaths /min, SpO2 86% @room air and 92% with 60%Fio2 on venturi mask), GCS: - responding to pain full stimuli. In view of impending respiratory failure, she was mechanically ventilated. Her repeat investigations showed an increasing trend of CPK (initially was 667 IU/L and 997 IU/L) and had urinary myoglobinuria, RFT (urea 76 mg/dl and creatinine was 2.6 mg/dl), hyperkalaemia (6.2 Meq/dl). She was provisionally diagnosed with Neuroleptic malignant syndrome and treated with Bromocriptine, Hydration and her potassium was corrected. after 2 days her CPK level, potassium and RFT have come to normal. She made full recovery and was discharged in a hemodynamically stable state.

DISCUSSION:

Delirium is a well-known complication of patients in critical care. It is associated with increased morbidity, mortality, prolonged hospital stays and long-term cognitive impairment. Majority of NMS cases were noticed after burns, trauma, and perioperatively.

The Society of Critical Care of Medicine sedation guidelines recommends haloperidol as the preferred agent for the management of delirium in the critically ill patients (grade C recommendation).[1] The optimal dose, regimen, efficacy and safety of haloperidol have not been well defined for the management of ICU delirium.[1] Fewer cases of Neuroleptic induced NMS were reported. All neuroleptics have been implicated in the genesis of NMS, although, high-potency agents like haloperidol are reported most often with NMS.[2,3]NMS is dose-independent and may occur at any time during treatment. Risk factors are higher doses of neuroleptics, greater dose increments over a short period, simultaneous use of two or more neuroleptics.[4.] Other risk factors are increased ambient temperature, dehydration, organic brain syndrome or affective disorder, genetics, young age and male gender, history of NMS, trauma, infection, malnutrition, alcoholism, premenstrual phase in females and thyrotoxicosis. About 16% develop within 24 h after initiation of antipsychotic therapy, 66% within the first week and nearly all cases within 30 days[5].NMS is secondary to decreased dopamine activity in CNS (blockade of D2 receptors or decreased availability of DA). Acute blockage of nigrostriatal and hypothalamic DA pathways results s in signs and symptoms of NMS.[6]Diagnostic is based on DSM IV and Levenson's criteria. Recommendations for specific medical treatments in NMS are based upon case reports and clinical experience and their efficacy. Commonly used agents are dantrolene, dopaminergic agents (Bromocriptine) and benzodiazepines. [7]

CONCLUSION:

Even a single minimal dose of antipsychotics can cause NMS, clinicians must be aware of the clinical features of NMS and vigilant eye in detecting early signs changes the prognosis and morbidity of the patient.

Declarations:

Funding: None

Conflict of interest: None declared

Ethical approval: Not required

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