

CASE REPORT

Neuroleptic malignant syndrome complicated by complete left bundle branch block: Don't let your guard down

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ABSTRACT

Neuroleptic Malignant Syndrome is a potentially lethal condition due also to its life threatening complications. In particular, arrhythmias can be rare and severe early manifestations of this illness. They deserve a care approach because of their drug refractoriness. A fatal case of haloperidol induced neuroleptic malignant syndrome with concurrent complete LBBB and premature ventricular ectopics is presented, suggesting the existence of close relation between two.

Key words: Neuroleptic malignant syndrome, LBBB, haloperidol, arrhythmias

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INTRODUCTION

Neuroleptic malignant syndrome is a rare, but life threatening idiosyncratic reaction to neuroleptic medications that is characterized by hyperthermia, muscular rigidity, altered sensorium and autonomic dysfunction. Despite early recognition it is associated with high grade of mortality and morbidity.¹ Common complications of NMS include rhabdomyolysis, respiratory failure, sepsis, renal failure and cardiovascular collapse.² Few previous studies have reported myocardial infarction and arrhythmias.³ but till now no case of NMS with complete LBBB is reported. Here, we are reporting a case of neuroleptic malignant syndrome with complete LBBB which was successfully managed and discharged from the hospital.

CASE REPORT

A 45 year old male presented in the emergency department with high grade fever, rigidity all over the body and altered sensorium. Past history revealed that, three days before, patient had low grade fever with delirium for which he was admitted to the local hospital. Injection haloperidol intramuscular and paracetamol was given there. Next day patient develop high grade fever with altered sensorium

for which he was admitted to emergency department. At the time of admission, he was unresponsive with HR130/min, BP150/104 mmHg and respiratory rate30/min. Lungs were clear. ECG showed complete left bundle branch block with premature ventricular and atrial ectopics. Neurological examination showed GCS score 4(E1V2M1), lead pipe rigidity and brisk deep tendon reflexes. Pt was immediately intubated and put on mechanical ventilation. Patient was shifted to ICU for further management. Active cooling measures were started with intravenous cold saline and nasogastric cold saline lavage. Injection paracetamol was given for fever. Laboratory investigations showed hemoglobin10gm%, TLC15000, platlet1.5 lacs, normal serum electrolytes, normal KFT and LFT. Creatine phosphokinase level was 40000U/L. A diagnosis of haloperidol induced neuroleptic malignant syndrome was made after excluding intracranial pathology by normal MRI, normal CT, sterile blood culture and normal CSF examination. Bromocriptine tablet 2.5 mg TDS and clonidine tablet 0.1mg was given via nasogastric tube. Dantrolene was not available ever after our sincere efforts so it was not used. Patient condition gradually improved and he was extubated on third day after vital stabilization. Patient was discharged from the ICU on fifth day after complete recovery

DISCUSSION

Neuroleptic malignant syndrome is a life threatening emergency with an overall incidence of 0.5%-3% in patients taking neuroleptic drugs such as haloperidol and fluphenazine.⁴ Onset of syndrome is not related to the duration of neuroleptic, or the toxic doses. Even single dose can result in neuroleptic malignant syndrome as in our case.⁵ Pathophysiology of Syndrome is largely speculative. Neuroleptic drug blocks dopaminergic receptors in the substantia nigra causes muscle rigidity and alters thermoregulation in the hypothalamus. Increased heat production from muscle rigidity causes fever and possibly a higher core temperature set point.⁶ Tachycardia and raised blood pressures are common sign of autonomic dysfunction. Mortality is usually caused by cardiovascular collapse, respiratory failure and disseminated intravascular coagulation.

Treatment of Neuroleptic malignant syndrome is mainly supportive; it is directed towards controlling the rigidity, hyperthermia and preventing complications. Value of

other interventions, such as Dantrolene, amantidine, Bromocriptine and ECT is uncertain.⁷

In particular, hypo kinetic and hyperkinetic arrhythmias can be rare and severe early manifestations of this illness. They deserve a care approach because of their drug refractoriness. In our case, complete LBBB with premature ventricular and atrial ectopics can be explained by various mechanisms: neurotransmitter receptor blockade typical of neuroleptic drugs, clustered lipid droplets among the cardiac myofibrils and possible electrolyte disorders due to diaphoresis.³ If not diagnosed early, this syndrome may result into death.

Hence, awareness of the condition and consideration of early diagnosis is very important to reduce mortality. A vigilant approach to the clinical features remains the most important strategy by which clinicians can improve patient outcome.

Conflict of interest: None declared by the authors

Author contribution: S: Concept; VKS: Conduction of case; SD & VD: Manuscript preparation

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