



Case Report

Neuroleptic Malignant Syndrome Associated with Haloperidol: A Case Report

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Abstract

A 42-year-old adult schizophrenic patient developed high fever (106 degree Fahrenheit), mental state changes and muscle rigidity after treatment with parenteral haloperidol. Neuroleptic malignant syndrome (NMS) associated with haloperidol was suspected. Other differentials were excluded. Haloperidol was discontinued and prompt treatment with bromocriptine and lorazepam was started resulting in a good recovery. Because of the temporal relationship between the patient's improvement with the discontinuation of haloperidol and treatment with lorazepam and bromocriptine, the diagnosis was believed to be haloperidol-induced NMS.

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Introduction:

Neuroleptic malignant syndrome (NMS) is a life threatening idiosyncratic reaction to antipsychotic drugs characterized by fever, altered mental status, muscle rigidity and autonomic dysfunction. The first reported case of NMS appeared in 1956, shortly after the introduction of the antipsychotic drug chlorpromazine.¹ The primary trigger of NMS is dopamine receptor blockade and the standard causative agent is an antipsychotic. Potent typical neuroleptics such as haloperidol, trifluoperazine, fluphenazine, and chlorpromazine have been most frequently associated with NMS and thought to confer the greatest risk. Although atypical neuroleptics appear to have reduced the risk of developing NMS compared to typical neuroleptics.²

Case presentation

A 42 year-old man was admitted for the first episode of suspiciousness, irrelevant speech and aggressive behavior for three month. There was no history of any organic illness in the patient. His

physical examination was normal and his psychiatric examination revealed a fearful mood, thought disturbances and auditory hallucinations. He was diagnosed as acute schizophrenic episode and was put on injection haloperidol 5 mg IM every 8 hours. The dose was increased after two days. Three days later the patient became confused and febrile. Haloperidol was discontinued and the patient was closely monitored. After the next two days he was noted to have alteration of consciousness, marked rigidity and diaphoresis.

His vital signs showed significant fluctuations - temperature: 103-105⁰ F, pulse rate: 100-110 beats/minute, blood pressure: 120-150/90-110 mm of Hg and respiratory rate: 18- 22/minute. His investigations revealed WBC count of 24,100/cmm and serum CPK levels of 2000 IU/L. His X-ray skull and chest, ECG, liver and renal function tests, serum electrolytes were within normal limits.

He was diagnosed as a case of NMS and started orally on bromocriptine and lorazepam. Nutritional

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support was given and serial monitoring of serum CPK urine was done. Over the next five days, the patient became alert and afebrile and his vital signs were stabilized. The serum CPK levels and WBC count decreased with clinical recovery.

Discussion

All neuroleptics have been implicated in the genesis of NMS, although, high-potency agents like haloperidol is reported most often with NMS.^{3,4} NMS is an idiosyncratic reaction independent of the dose and may occur at any time during treatment. Predisposing risk factors include dehydration, agitation, substance abuse, parenteral administration, high doses or rapid upward titration of neuroleptics.^{3,4,5} NMS is characterized by the development of hyperthermia, muscle rigidity, change in mental status and autonomic dysfunction. Approximately 16% of cases of NMS develop within 24 h after initiation of antipsychotic therapy, 66% within the first week and nearly all cases within 30 days. NMS usually lasts for 7–10 days; however, duration may be longer when depot injections of the drug is implicated.⁵

The predisposing factors that probably led to the development of NMS in our patient included high dose, rapid titration of initial haloperidol dose. Our patient exhibited classic symptoms of NMS with development of hyperthermia and muscular rigidity. This patient's creatine kinase (CPK) level was elevated. The dramatic response to dantrolene with improvement in temperature was consistent with the diagnosis of NMS. The patient made a remarkable recovery from NMS and was discharged. As demonstrated by our case, high dose and quick up-titration of the haloperidol dose

can lead to the development of NMS. Bromocriptine, dantrolene sodium and benzodiazepines are shown to be effective in the treatment of NMS.^{6,7,8} A need for awareness of the syndrome in view of the widespread use of neuroleptics and its potential lethality which can be averted by early detection and specific treatment have prompted the present report.

References

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