

NEUROLEPTIC MALIGNANT SYNDROME AND ITS SUCCESSFUL TREATMENT WITH BROMOCRIPTINE - A CASE REPORT

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The Neuroleptic Malignant Syndrome (NMS) is a rare complication of neuroleptic use. This complication may be fatal and is characterized by hyperpyrexia, rigidity, tachycardia, hypertension, tremors, diaphoresis, autonomic dysfunctions and altered states of consciousness¹⁻³. Although agreement about the incidence as well as natural history remains controversial the cases have been reported from almost all countries of the world with of course many atypical forms.⁴⁻⁸ Here we describe a typical case of NMS and its successful treatment.

CASE REPORT

A.S. aged 34 years, married farmer presented in October 1989 with two years history suggestive of Schizophrenia. His diagnosis was made fourteen months ago and the treatment was started with 30 mg of haloperidol, 200 mg of chlorpromazine and 10 mg of procyclidine per day. The doses of haloperidol and chlorpromazine were increased upto 50 mg and 400 mg respectively during the treatment. The present complaints were rigidity, tremors, restlessness, high fever and abnormal movements. There was no history suggestive of any medical illness. On examination, patient was restless with marked stiffness of the body, accompanied by tremors and sialorrhoea. He was febrile (axillary temperature 101°F), pulse 100/mm, and blood pressure was 130/75 mmHg. There was no cyanosis, jaundice or oedema. Auscultation of chest and heart did not reveal any abnormality. Nervous system examination showed an increase tone in both limbs. Tendon reflexes were poorly elicited. Planters were down going bilaterally and there were no signs of meningeal irritation. During first two days of admission clinical signs fluctuated with great rapidity. For brief periods he became confused but generally consciousness level remained intact. Laboratory investigations showed a haemoglobin level of 8.2 G%, total leucocyte count 18000/ cmm with polymorphonuclear leucocytosis. Erythrocyte sedimentation rate was 50 mm/1st hour and random blood urea, blood sugar, liver function tests and sodium and potassium levels were within normal limits. Serum creatinine phosphokinase (CPK) was markedly raised (1235 u/ml). A working diagnosis of NMS was made. Neuroleptics were stopped and bromocriptine 2.5 mg three times a day (TDS) was started. The dose of bromocriptine was increased to 5 mg TDS during next few days with an addition of diazepam 5mg at bedtime. Patient's clinical state started to improve with resolution of the autonomic instability and CPK serum level and other laboratory investigations returned to normal within next two weeks.. He was discharged after three weeks. His physical and mental state continued to improve and there was no objective evidence of any sequelae related to NMS.

DISCUSSION

Despite controversies about incidence, aetiology, pathogenesis and diagnostic issues, the subject of NMS has continued to cause considerable interest in recent years. The treatment aspect of this disorder becomes more relevant and important as certain drugs have made fatal outcome of this disorder unusual in many cases.⁹ Bromocriptine, a dopamine agonist, has shown a considerable reduction in

mortality and marked improvement in morbidity in many studies¹⁰. The present case which showed many typical symptoms of NMS like high fever, muscle rigidity, alteration in the level of consciousness, dysphonia, difficulty in swallowing, history of use of neuroleptics and laboratory findings especially raised CPK and improvement with bromocriptine confirms the efficacy of this drug when given in recommended doses (5 - 10mg TDS) in addition to cessation of neuroleptics. Although the full pathophysiology of this syndrome remains obscure, possible aetiological factors include use of neuroleptics¹¹, lethal catatonia¹² old age, organic brain syndrome¹³ and high environmental temperatures¹⁴. It is, however, difficult to evaluate whether these factors are the only primary triggering factors. The casual relationship between NMS and neuroleptic treatment is explained on the basis of a sudden and massive blockade of the dopaminergic system. The recovery from NMS, therefore basically involves changes in the neurotransmitter system with a tendency to decrease the dopaminergic blockade. Successful treatment with bromocriptine, in our case supports this hypothesis. Many aspects of this syndrome still remain to be elucidated. One of the main issues, which has great clinical interest, is the long term outcome and the risk of recurrence on rechallenge with a neuroleptic,^{15,16}. Fortunately the incidence of NMS is very low despite the frequent use of neuroleptics. Increased awareness on the part of clinicians has made the diagnosis important in differentiating this fatal condition from other syndromes like lethal catatonia and malignant hyperthermia. NMS remains a dangerous condition. With an increase in the use of neuroleptics it becomes very important for the clinicians to keep this rare entity in mind while treating patients with these drugs. The need of discontinuing neuroleptics is not only stressed in such cases but early use of appropriate therapeutic agent like bromocriptine is strongly recommended.

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