Case Report

Low serum iron as a possible risk factor for neuroleptic malignant syndrome

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Abstract

Neuroleptic malignant syndrome (NMS) is a rare, but fatal condition characterized by rigidity, fever, dysautonomia and altered consciousness along with elevated serum creatinine phosphokinase levels and leukocytosis. Treatment of NMS includes symptomatic and specific treatment with drugs like bromocriptine may be given. Risk factors for NMS include dehydration, parenteral antipsychotics and high potency antipsychotics. One of the important, but lesser known risk factors for NMS is low serum iron. Pronounced reduction in serum iron suggests that acute phase reactants do have a role in NMS. The present case report focuses on the importance of low serum iron as a risk factor for NMS.

Key words: Low serum iron, neuroleptic malignant syndrome, risk factor

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Introduction

Neuroleptic malignant syndrome (NMS) is a fatal reaction to neuroleptics[1] which occurs in 0.02-3% of patients on neuroleptics and has mortality risk of 10-20% respectively.[2,3] Bromocriptine, dantrolene and benzodiazepines are drugs that may be tried in the treatment of NMS.[4] Risk factors for NMS include dehydration, psychomotor agitation, parenteral antipsychotics and low serum iron.[5,6] The effects of anti-psychotics in a patient with low serum iron can be avoided by early detection, adequate monitoring and specific treatment.

Case Report

Mrs.A, 30-year-old woman was admitted for the first episode of suspiciousness, irrelevant speech and very violent behavior for 1 month. There was no history of any medical or neurological illness in the patient. Her physical examination was normal and her mental status examination revealed a fearful mood, formal thought disorder loosening of association and tangentialityand auditory hallucinations (second person). She was diagnosed as brief psychotic disorder and put on injection haloperidol 5 mg i.m. every 12 h to control her violent behavior. 1 day later the patient became febrile and developed minimal rigidity and diaphoresis. Haloperidol was discontinued and the patient was closely monitored. Her vital signs showed significant fluctuations-temperature: 100-102 F, pulse rate: 80-120 beats/min, blood pressure: 130/80-150/110 mm of Hg. Her blood investigations revealed white blood cell (WBC) count of 12,000/mm^3 (normal range: 4000-11,000/mm^3) and her hemoglobin (Hb) was 8 g/dl (normal range: 12-16 g/dl). Her serum creatinine phosphokinase (CPK) levels were 2500 IU/L (normal range: 40-150 IU/L). Her X-ray chest, electrocardiography, liver and renal function tests, serum electrolytes were within the normal limits. On detailed hematological investigations we found her serum iron low i.e., her serum ferritin was 20 ug/dl (normal: 30 ug/dl), her total iron-binding capacity was 380 ug/dl (normal: 300-360 ug/dl) and transferrin saturation was 18% (normal: 25-50%).

She was diagnosed as a case of NMS and started orally on bromocriptine 2.5 mg t.d.s. and lorazepam 2 mg t.d.s. along with nutritional support and serial monitoring of serum CPK. Over the next 2 days, the patient became alert and afebrile

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and her vital signs stabilized. The serum CPK levels (80 IU/L) and WBC count (8000/mm^3) fell concomitant with clinical recovery. The recovery was complete at the end of 2 weeks. Bromocriptine was tapered off and the patient was maintained on lorazepam 2 mg b.d. Patient was started on ECTs for behavioral control.[7,8]

Patient was discharged after a month's stay in the hospital and at the time of discharge patient started on olanzapine 5 mg a day. In addition, hematinics started to correct underlying iron deficiency. During a 3 months follow-up period, the patient maintained her recovery and she is asymptomatic at present.

Discussion

To diagnose NMS Levenson's criteria have been used which are widely accepted. The major criteria include fever, rigidity and raised levels of CPK while the minor criteria include tachycardia, abnormal blood pressure, tachypnea, altered consciousness, diaphoresis and leukocytosis. The presence of all the three major or two major and four minor criteria is essential for the diagnosis of NMS.[3] Our patient satisfied all the major and minor criteria thereby substantiating the diagnosis of NMS.

A rapid loading with potent neuroleptics like haloperidol is considered to be the major contributing factor in the development of NMS, by causing a sudden hypodopaminergic state.[9] Usually this alone is attributed as the singular cause for development of NMS. What is interesting in this case was the rapidity of development of NMS with just 10 mg of parenteral Haloperidol. This led us to investigate other associated etiology and what we found was that serum iron may reduce the number of functional dopamine receptors, thereby making the patient more vulnerable to an even seemingly low dose of anti-psychotics.[6] In a population where we often deal with individuals (particularly women) having anemia, diagnosed by low Hb and low serum iron levels, this becomes a vital clinical experience.

In patients with suspected anemia with the requirement of parenteral anti-psychotics, it would be wise to not only do a complete hemogram, but also to initiate correction of the anemia early on and give a lower dose of parenterals (if really required); whilst being vigilant of the potential chance of early development of NMS.

Iron deficiency anemia is an easily reversible condition, treatment for which is cheap and can be initiated in our psychiatric clinics itself; and does not require a specialist referral unless very severe (Hb < 7 g/dl).

Nevertheless, NMS can be a fatal condition if not detected early and if intervention is not done rapidly. Complications may arise mainly secondary to hyperthermia and metabolic abnormalities and patients require an intensive care setting due to the rapidity of complications. It is thus imperative to assess any correctible factor which may help in decreasing the morbidity.

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