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Atypical Neuroleptic Malignant Syndrome: a Case Presentation

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Atypical Neuroleptic Malignant Syndrome: A Case Report

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Introduction

Neuroleptic malignant syndrome (NMS) is an emergent, often fatal, medical condition associated with the use of dopamine receptor antagonist medications.[1,5] Hyperthermia, muscle rigidity, autonomic instability, evidence of muscle injury, and altered mental status are known to be the hallmarks of the disease[2]. In contrast with the typical presentation, atypical NMS presents with a different set of symptoms, making diagnosis difficult.[2] It is important to use clinical acumen to make a timely and efficient diagnosis of NMS. We present a case of Atypical NMS in a 21 year old male.

Neuropsychiatric	Lethal/malignant catatonia Delerium	Nonconvulsive status epilepticus
Infectious	Rabies Septic shock Tetanus	Brain abscess Encephalitis Meningitis
Environmental	Heatstroke	Spider envenomation
Pharmacological	Anticholinergic delirium Drug-drug interaction Drug withdrawal	Extrapyramidal side effects Malignant hyperthermia Serotonin syndrome
Toxic	Heavy metals (lead, arsenic) Lithium	Salicylates Substances of abuse
Endocrine	Pheochromocytoma	Thyrotoxicosis

Table 1: Differential diagnosis for NMS[3,6]

References:
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Clinical Case

A 21-year-old African American male with previous psychiatric history of "schizophrenia, bipolar disorder, acute dystonia, and tardive dyskinesia" was transferred to the emergency department from county jail, where he was being held for disturbing the peace, and assault. He was brought to the ED with a history of 2 to 3 days of decreased activity, not eating, drooling, "howling", and altered mental status. During this time patient was in isolation for previously erratic behavior. Conversations with staff at jail state that he was tachycardic and hypertensive at times during this episode yet when asked for full records they declined and explained they "don't keep" charts or records. Jail nurses were able to tell us his medications include haloperidole decanoate, oral haloperidole and trihexylphenidyl yet he had been refusing medications in recent days to weeks. They were unable to provide information regarding PRN medications given during this period of time. On admission vital signs were as follows: BP 128/80, HR 113, Temp. 97.6, RR 20, O2 Sat. 99%. He expressed much pain and immobility. He was able to communicate with small head movements, grunts, and finger movements. Exam showed posturing, bradykinesia, trismus, sialorrhea, diaphoresis, and urinary incontinence. Lab findings indicated elevated creatinine kinase of 18,963 IU/L, WBC of 14.2 x10^9/L, AST 330 IU/L, and ALT 98 IU/L. Iron studies showed the following: iron 42 mcg/dL, ferritin 293 ng/mL, iron sat 20%, transferrin 168 mg/dL, TIBC 213 mcg/dL. CT of head was unremarkable. Throughout the hospitalization he displayed no cogwheeling, no muscle rigidity, no hyperthermia, and no obvious cognitive dulling. He did not respond to 2mg lorazepam IV nor 10mg diazepam q8hrs IV for 1 ½ days. Due to components of the history and physical exam, laboratory and imaging studies, it was determined that this patient was presenting with atypical NMS and bromocriptine therapy was initiated. The patient steadily improved back to baseline over a 22 day admission.

Discussion

It is essential to recognize NMS to prevent associated mortality. Mortality rates of unrecognized NMS are estimated to be 10%-20%.[3] The presentation of NMS may vary significantly, making the diagnosis difficult to ascertain; however, key lab values are of diagnostic value. Initially, the differential included NMS, catatonia, and anticholinergic delirium. Catatonia was a less likely diagnosis as lorazepam 2mg IV push demonstrated no improvement of symptoms. Anticholinergic delirium usually presents with altered mental status rather than movement disorder. CK and AST were elevated due to sustained muscle rigidity and rhabdomyolysis, this supports the diagnosis of NMS.[7] Serum iron levels below 55.8 mcg/dL is also associated with NMS.[4] Low serum iron concentration is an acute phase reactant in NMS and acts as a helpful adjunctive biochemical marker.[4] The presentation of NMS may be insidious over days. Most cases present within 3 days, though some may be up to 30 days. The patient was treated with bromocriptine and there was significant improvement. Once diagnosis is made, antipsychotic medications should be held and re-challenging the patient with an antipsychotic can occur after a 5-14 day washout period from symptoms. Our patient was started on aripiprazole and titrated up to 15mg TID.

In conclusion, NMS can present with a variety of symptoms. Recognizing atypical presentations of NMS is very important. Several sources propose various characteristic symptoms of atypical NMS including tremor, sialorrhea, akinesia, dystonia, trismus, myoclonus, dysarthria, dysphagia, diaphoresis, urinary incontinence, pallor, elevated CPK, leukocytosis, low serum iron, and elevated liver enzymes, often but not always associated with the classic features.[1-4] Our patient exhibited many of the proposed characteristics of NMS (posturing, bradykinesia, trismus, sialorrhea, diaphoresis, and urinary incontinence) yet he did not present with any of the "hallmark" features. The elevated creatine kinase, leukocytosis, transaminitis, and iron studies were helpful in making the diagnosis.