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Review Article

Malignant Catatonia – A Rare Presentation

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ABSTRACT

Malignant catatonia (MC) - a lethal catatonia, is extremely rare, most severe, and a life-threatening manifestation which occurs either because of an underlying neuropsychiatric or general medical illness. We report a case of excited catatonia with fever, autonomic instability, and delirium-a picture of MC.

KEYWORDS: Catatonia; Psychosis; Antipsychotics; Fever; Rigidity; Malignant Catatonia.

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INTRODUCTION

Malignant catatonia (MC) - a lethal catatonia, isextremely rare, most severe, and a life-threatening manifestation which occurs either because of an underlying neuropsychiatric or general medical illness. [1-6] we report a case of excited catatonia with fever, autonomic instability, and delirium-a picture of MC.

A sixty-four-year-old African American male with past medical history of schizophrenia, diabetes mellitus and hypertension presented to the medical emergency department (ED) approximately forty minutes after being found unconscious in the stairwell of his apartment building during the midst of a heat wave warning that day. In the ED,

he appeared to have incoherent speech, psychomotor agitation, and altered mental status (AMS). His last psychiatric hospitalization was in 2008 and has since been following up regularly with outpatient psychiatric services, been stable on the same psychiatry medication regimen (Seroquel, Haldol, Paxil, Gabapentin, and Cogentin).

The patient lived with one roommate and had been in constant touch with his sister who lives in a different state. His sister mentioned that he has been diagnosed with schizoaffective disorder and has been managing his medications and doctor's appointments for the past 20 years all on his own. She stated that they talk every night and is very compliant with his treatment. However, she mentioned that she couldn't get hold of him from past two days. She appeared genuinely concerned and worried for his overall health and felt that something was medically wrong with him. She reported "this is not his baseline."

ASSESMENT

The patient weighed 306 pounds, with a body mass index of 43.9, blood pressure of 89/65 mmHg, pulse 107/min, and temperature of 103.7° Fahrenheit. Physical examination showed normal findings with some rigidity in his extremities. EKG, liver function tests, complete blood count, glucose, urine analysis, RPR, and HIV tests were within normal limits. The urine drug screen and alcohol test were negative. Abdominal ultrasound, Chest X-ray, CT scan of the head, neck without contrast and MRI of the head showed no acute findings. However, labs showed lactic acid of 2.8 (reference range: 0.5-1 mmol/L), BUN/Creatinine of 34/5.1 (reference range: 10/1), Creatine phosphokinase (CPK) of 721 (reference range: 5-25 IU/L), and Anion Gap of 21 (reference range: 8-16 mEq/L). He was admitted to medical floor. The diagnosis of stroke was ruled out and sepsis protocol was initiated. He was started on several antibiotics which normalized his temperature and started showing some improvement as he became afebrile, saturating well on 50% ventimask, and his lactic acid trended down over night from 2.8 to 0.7. His blood pressure improved from 89/65 to 110/70. LP resulted in a failed attempt, however 2 weeks later LP was obtained and was negative for CNS infection including HSV. Later, the blood and urine culture results were negative. Antibiotics were discontinued as there were no signs and symptoms of infection.

However, after 2 days of his stay in the hospital, psychiatry was consulted. The patient was agitated, hallucinating, and started pulling out intravenous lines, blood pressure cuffs, and heart monitors. He attempted to get out of his bed to hit and push nurses. He appeared restless, diaphoretic, tachypnea, constantly moving his arms and legs, however movement appeared to be in slow motion consistent with psychomotor retardation. He developed autonomic instability as his blood pressure fluctuated from 190/110's to 150/90's, developed tachycardia (pulse 120's), tachypnea, and diaphoresis. For his agitation, he was placed in mechanical restrains and started on

haloperidol, diphenhydramine, and lorazepam, neither of which produced any improvement. The patient was noted to have signs of catatonia. These signs included confusion, excitement, posturing, and possible psychotic features; later they included pyrexia, autonomic instability, and over the subsequent days, the CPK levels began trending up to maximum 1353.

Deterioration coincided and soon after the patient was transferred to the intensive care unit (ICU), his psychiatry home medications (quetiapine, paroxetine, benztropine, and haloperidol) were stopped. Patient started showing some improvement in his movements and his CPK levels started trending down. Heat Stroke, anti-NMDA encephalitis, neuroleptic malignant syndrome (NMS), and MC were considered in differential diagnosis. The patient was started on Lisinopril for high blood pressure, dantrolene sodium for catatonic symptoms and the electroconvulsive therapy (ECT) was scheduled to perform after his blood pressure is normal. Patient was given Ativan, Benadryl and Haldol for sedation. During this time, patient remained afebrile with autonomic instability, developed lead pipe rigidity in all four extremities. Blood work showed magnesium level of 1.3, serum iron of 43, and transferrin level of 168. Urine metanephrines and norepinephrines were negative.

After two weeks of stay, patient was able to respond to verbal commands (stick out your tongue, lift your arm), however still presented with echolalia, catalepsy, posturing and continued lead pipe rigidity. Later over subsequent days, he was able to recognize objects, remember familiar faces, recall names, observed laughing, smiling, and registration became more intact with slow recovery on recall. Restraints were removed.He explained why he was in the hospital, how long he had been in the hospital and that he finally was showing positive signs of improvement. His appetite improved, his vital signs remained stable and he continued to be less and less rigid with no more posturing or catalepsy. He adapted well to the rules of the inpatient unit, was sociable with peers, and showed no behavioral disturbances. His mood and affect improved. He was compliant with his medications with no reported side effects. He denied suicidal or homicidal ideations and auditory or visual hallucinations during his stay in the inpatient unit. After 8 days, the patient showed significant improvement in mood and social interaction and was discharged to a skilled nursing facility.

DISCUSSION

In this case, MC, NMS, heat stroke and anti-NMDA encephalitis were considered in differential diagnosis once stroke and sepsis were ruled out. The lumbar puncture was never performed on the patient and therefore, anti-NMDA encephalitis was neither diagnosed nor treated. Moreover, treatment with dantrolene was proven effective to reduce muscle rigidity in this patient and among thepatients diagnosed with MC, NMS, and the heat stroke. [7-10]

However, treatment with ECT changed the trajectory in this case. This patient was taking neuroleptics for over 10 years and was on the same dosage. Majority cases of NMS occur within 30 days of neuroleptic initiation or because of change in dosage. [11,12] NMS is often considered a variable form of malignant catatonia (medication induced) and the typical presentation of NMS usually present with very high levels of white blood cell count, fever, and CPK. [11, 12] However, in this case, patient's WBC count was within normal range, and therefore NMS was ruled out. On the other hand, the heat stroke usually present with more electrolyte imbalance on admission, which was not seen in the laboratory workup throughout his hospitalization. [7] Moreover, possible altered mental status rather than catatonic symptoms would be appreciated in patients with heat stroke. However, CPK level was high so heat stroke was not completely ruled out.We suspected dehydration could have raised the levels of his medications since the patient was admitted on a very hot summer day and was taking several psychiatric medications for the past 20 years. Thus, MC was considered as our final diagnosis and was confirmed once ECT was found to be life-saving. [13] in this case.

CONCLUSION

MC is extremely rare and has been reported in only less than 2% of cases of catatonic patients. [14] The case presented herein illustrates that MC is an emergent and life-threatening illness that can be accompanied by multiorgan dysfunction, including cardiopulmonary and metabolic defects requiring concurrent ICU and psychiatric care. [1] The exact pathophysiological mechanism of malignant catatonia is unknown.The motor symptoms are presumed to be related to a deficient GABA-ergic state in the orbito-frontal cortex that disturbs the modulation to the basal ganglia 'top-down'. [15] The GABA-ergic cortical dysfunction may explain the affective and behavioral disturbances in catatonia, which are not seen in NMS. [16] Postulating different mechanisms for catatonia, ECT would be more effective in 'top-down' variants of the syndrome. [17] Unfortunately in clinical practice these variants are indistinguishable.Both MC and NMS may lead to muscle injury, aspiration, and metabolic disturbance due to hyperthermia and altered mental status. However, prodromes of MC have psychiatric undertones of psychosis, agitation, stupor, mutism, or anxiety. Currently, no biomarkers exist for MC to assist in diagnosis, although there appears to be an association of elevated d-dimer in the disease. [18] Use of this marker may be helpful in expediting detection of this disease. Bo-mocriptine, dantrolene, and benzodiazepines have been used with success but ECT has been found to be life-saving. [17] Benzodiazepines are theorized to improve MC and NMS by increasing $GABA_A$ activity. Likewise, the use of ECT may involve GABA activity by inducing a neural storm with increased GABA transmission and clinical improvement. [19,20]

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