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Neuroleptic Malignant Syndrome: A Case Report and Literature Review

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Abstract:

Neuroleptic malignant syndrome is a rare but potentially life-threatening complication of antipsychotic medication use, characterized by dopamine receptor blockade in the central nervous system. First described in the 1960s following the introduction of neuroleptics, its reported incidence ranges from 0.02% to 3.3% of cases, typically appearing within the first weeks of treatment. We report the case of a 46-year-old male patient who presented with marked deterioration of general condition shortly after initiating high-dose levomepromazine for a depressive episode. He was receiving the oral solution at a concentration of 40 mg/mL (1 drop = 1 mg), with a total daily dose of 600 mg. He was emergently hospitalized in the medical intensive care unit following the onset of neuropsychiatric symptoms including anorexia, altered consciousness, rigidity, dyskinetic movements, and sialorrhea, associated with digestive symptoms such as nausea and vomiting, all occurring in a febrile context. On admission, clinical examination revealed a patient in shock with hypotension at 80/40 mmHg, anuria, and profuse sweating. The patient was unconscious with a Glasgow Coma Scale score of 9/15 and bilateral mydriasis. Laboratory findings revealed bicytopenia with anemia at 8 g/dL and thrombocytopenia at 33,000/mm³, severe acute kidney injury with creatinine clearance of 10 mL/min, hepatic cytolysis with a prothrombin time of 50%, and rhabdomyolysis with markedly elevated muscle enzymes including CPK at 60,000 IU/L and LDH at 1,500 IU/L. Fundoscopic examination showed stage I changes. Lumbar puncture was unremarkable, and all cytobacteriological examinations of urine, blood, and stool for infectious agents were negative. The diagnosis of neuroleptic malignant syndrome was established based on Levenson's criteria with 3 major and 6 minor criteria, although likely delayed, and no specific treatment was administered. The acute kidney injury was secondary to rhabdomyolysis in a patient with multiorgan failure including respiratory distress and hepatic failure, associated with disseminated intravascular coagulation. Continuous renal replacement therapies would have been of great benefit for this patient, but were not available at the time. Despite initial hemodialysis sessions, the patient's condition continued to deteriorate, and he died 15 days after admission following seven daily hemodialysis sessions. The pathophysiology and specific treatment of neuroleptic malignant syndrome remain poorly understood. General supportive measures, discontinuation of the offending neuroleptic agent, and management of complications remain the mainstay of treatment.

Key words: neuroleptic malignant syndrome; antipsychotics; rhabdomyolysis; acute kidney injury; dopamine antagonists; levomepromazine

Introduction:

Neuroleptic malignant syndrome is a rare but potentially fatal iatrogenic disorder first described in the late 1950s following the introduction of neuroleptic drugs for the treatment of psychotic disorders [1, 2]. It is characterized by a spectrum of clinical manifestations resulting from central dopamine receptor blockade, primarily affecting the hypothalamic and brainstem regulatory systems [3, 4]. The reported incidence of neuroleptic malignant syndrome ranges from 0.02% to 3.3% of patients exposed to antipsychotic medications, with more recent estimates suggesting an incidence of approximately 0.01% to 0.02% among individuals treated with these agents [1, 5, 6].

Neuroleptic malignant syndrome typically manifests within the first weeks of treatment, although it can occur at any time during therapy [1, 2]. The

classic presentation includes four cardinal features: muscular rigidity, hyperthermia, altered consciousness, and autonomic dysfunction [1, 2, 7]. However, there is considerable heterogeneity in the onset, progression, and outcome of neuroleptic malignant syndrome, and no universally agreed-upon diagnostic criteria exist, which complicates both diagnosis and research efforts [8].

The pathophysiology of neuroleptic malignant syndrome is thought to involve neuroleptic blockade of D2 dopamine receptors in the hypothalamus and brainstem, leading to a generalized systemic hypermetabolic syndrome [2, 9] [3,9]. This dopamine antagonism results in dysregulation of the sympathetic nervous system, which may explain many of the clinical features of neuroleptic malignant syndrome, including hyperthermia, autonomic instability, and altered consciousness [9, 10].

Virtually all antipsychotic medications have been associated with neuroleptic malignant syndrome, including both first-generation and second-generation antipsychotics [5, 11, 12]. High-potency first-generation antipsychotics have traditionally been considered to carry a greater risk, although this assumption remains unproven [5, 11, 13]. In a recent analysis, 51% of neuroleptic malignant syndrome cases were associated with first-generation antipsychotics and 45% with second-generation agents, with haloperidol identified as the etiologic agent in 44% of all cases [5].

While mortality rates have declined significantly over the past decades due to increased awareness and earlier recognition, neuroleptic malignant syndrome remains a potentially lethal condition with reported fatality rates of up to 30% in historical series and approximately 10 to 15% in more recent reports [1, 2, 14]. Myoglobinuria and renal failure are strong predictors of mortality, with a risk of approximately 50% [14].

We report a case of severe neuroleptic malignant syndrome complicated by rhabdomyolysis, acute kidney injury, and multiorgan failure in a 46-year-old patient treated with high-dose levomepromazine for severe depression. This case highlights the importance of early recognition, prompt discontinuation of the offending agent, and aggressive supportive care in improving outcomes.

Case Presentation

Patient History and Presenting Complaints:

A 46-year-old male patient, with no significant prior medical history, presented with a marked deterioration of general condition shortly after initiating treatment with levomepromazine oral solution (40 mg/mL, 1 drop = 1 mg) at 600 mg/day for a depressive episode. He was emergently hospitalized in the medical intensive care unit following the onset of neuropsychiatric symptoms including anorexia, altered consciousness, rigidity, dyskinetic movements, and sialorrhea, associated with digestive symptoms such as nausea and vomiting, all occurring in a febrile context.

Physical Examination:

On admission to the intensive care unit, clinical examination revealed a patient in shock with hypotension at 80/40 mmHg, anuria, and profuse sweating. The patient was unconscious with a Glasgow Coma Scale score of 9/15 and bilateral mydriasis. Fever was present, and generalized rigidity with dyskinetic movements was noted.

Laboratory Findings:

The initial laboratory workup revealed multiple abnormalities indicative of severe systemic involvement. Hematological abnormalities included anemia with hemoglobin at 8 g/dL and thrombocytopenia with platelet count at 33,000/mm³, suggesting a bicytopenia consistent with disseminated intravascular coagulation or bone marrow suppression. Renal function assessment showed severe acute kidney injury with creatinine clearance of 10 mL/min. Hepatic function evaluation revealed hepatocellular injury with elevated transaminases and coagulopathy with prothrombin time at 50%. Muscle enzyme analysis demonstrated marked rhabdomyolysis with creatine phosphokinase at 60,000 IU/L and lactate dehydrogenase at 1,500 IU/L. Fundoscopic examination showed stage I changes. Lumbar puncture was unremarkable with no evidence of central nervous system infection. All cytobacteriological examinations of urine, blood, and stool were negative for infectious agents.

Diagnostic Considerations:

The diagnosis of neuroleptic malignant syndrome was established based on Levenson's diagnostic criteria. The major criteria include fever, rigidity, and elevated creatine phosphokinase, all of which were present in our patient. The minor criteria include tachycardia, abnormal blood pressure, tachypnea, altered consciousness, diaphoresis, and leukocytosis, all of which were also present. Our patient met the criteria for neuroleptic malignant syndrome with 3 major and 6 minor criteria. However, the diagnosis was likely made with some delay, and no specific treatment such as dantrolene, bromocriptine, or amantadine was administered.

Clinical Course and Complications:

The offending medication (levomepromazine) was immediately discontinued upon admission to the intensive care unit. The patient's clinical course was complicated by multiorgan failure including respiratory distress requiring ventilatory support, hepatic failure with coagulopathy, and acute kidney injury due to rhabdomyolysis. Disseminated intravascular coagulation was suggested by laboratory findings with thrombocytopenia and coagulopathy. The acute kidney injury was primarily attributed to rhabdomyolysis in a patient with multiorgan failure, with creatinine clearance of 10 mL/min.

Hemodialysis was initiated on the second day of hospitalization and continued daily. Despite seven hemodialysis sessions, the patient's multiorgan failure proved refractory to maximal supportive care. He died 15 days after admission.

Discussion:

Pathophysiology and Clinical Features

Neuroleptic malignant syndrome is generally conceptualized as a disorder triggered by neuroleptic blockade of D2 dopamine receptors located in the hypothalamus and brainstem regulatory systems, which in turn culminates in a generalized systemic hypermetabolic syndrome [3, 9]. Dopamine receptor antagonism leads to hypothalamic dysfunction with disruption of thermoregulation resulting in hyperthermia, striatal dopamine blockade leading to

extrapyramidal symptoms including rigidity and bradykinesia, and autonomic nervous system dysregulation resulting in tachycardia, hypertension or hypotension, diaphoresis, and other autonomic manifestations [1-3].

Gurrera hypothesized that dysregulated sympathetic nervous system hyperactivity explains many of the features of neuroleptic malignant syndrome [9]. An exaggerated or extreme sympathetic nervous system dysregulation in response to emotional or psychological stress may constitute a trait vulnerability for neuroleptic malignant syndrome, which may be precipitated by dopamine receptor antagonism [9]. Mann and colleagues suggested that there is compelling evidence that disruption of dopaminergic neurotransmission by antipsychotic drug antagonism at the D2 receptor results in neuroleptic malignant syndrome [10]. They proposed that preexisting central dopaminergic hypoactivity when coupled with adjustments in the dopamine system from acute and repeated exposures to stress could precipitate neuroleptic malignant syndrome [10].

In the classic form, patients present with four major symptoms: muscular rigidity, hyperthermia, altered consciousness, and autonomic dysfunction [1, 2, 7]. However, there is heterogeneity in the onset, progression, and outcome of neuroleptic malignant syndrome [1, 8]. Regarding onset and progression, 16% of patients develop signs of neuroleptic malignant syndrome within 24 hours of initiating a neuroleptic agent, 66% develop signs within 1 week, 96% develop signs within 30 days, and neuroleptic malignant syndrome is unlikely to occur after 30 days of instituting a neuroleptic but has been reported in 4% of patients [1, 2, 5]. Velamoor and colleagues found a predominance of 4 signs in most cases, with the following sequence in over 70% of cases: mental status changes, then muscular rigidity, followed by hyperthermia and autonomic dysfunction [3]. This order sequence may aid clinicians in early detection and intervention [3].

Risk Factors and Implicated Medications

Virtually all dopamine antagonists have been associated with neuroleptic malignant syndrome [1, 2, 5]. Patient-related risk factors include young adult males who predominate, exhaustion, dehydration, malnutrition, organic brain syndromes, intellectual disability, and prior episode of neuroleptic malignant syndrome with a 17% to 30% recurrence rate with rechallenge [1, 2, 5, 6, 15, 16]. Medication-related risk factors include high-potency first-generation antipsychotics which carry a greater risk, parenteral administration routes, rapid dose titration, and higher total daily drug doses [1, 2, 5, 13].

Medications associated with neuroleptic malignant syndrome include first-generation antipsychotics such as haloperidol, chlorpromazine, and fluphenazine [11, 14]; second-generation antipsychotics including clozapine, olanzapine, quetiapine, risperidone, aripiprazole, and ziprasidone[5, 11, 12, 17] ; antiemetics such as prochlorperazine, promethazine, and metoclopramide [1, 5, 18]; and other agents including droperidol and amoxapine [1, 5, 19]. In our patient, the offending agent was levomepromazine, a phenothiazine antipsychotic with neuroleptic properties. The patient was receiving 600 mg/day orally, a very high dose significantly exceeding the maximum recommended dose of 400 mg/day, which was initiated rapidly without gradual titration. The high dose, rapid initiation, and the use of a phenothiazine antipsychotic all likely contributed to the development of neuroleptic malignant syndrome in this patient.

Diagnostic Criteria and Differential Diagnosis

No universally agreed-upon criteria exist for neuroleptic malignant syndrome [2, 8]. Using the Delphi method, an international multispecialty panel proposed diagnostic criteria that included exposure to a dopamine antagonist or withdrawal of a dopamine agonist within the 72 hours preceding onset of symptoms, hyperthermia $\geq 38°C$ on at least 2 occasions, rigidity, mental status alteration, creatine kinase ≥ 4 times the upper limit of normal, sympathetic system lability, hypermetabolism, and absence of other etiologies [8]. Levenson's criteria, which were used in our case, include 3 major criteria comprising fever, rigidity, and elevated CPK, and 6 minor criteria including tachycardia, abnormal blood pressure, tachypnea, altered consciousness, diaphoresis, and leukocytosis.

Neuroleptic malignant syndrome is a diagnosis of exclusion[1, 2, 5] . The differential diagnosis includes central nervous system infections such as acute viral encephalitis, tetanus, and bacterial, fungal, and parasitic infections [1, 2]; central nervous system mass lesions including tumors, abscesses, stroke, and trauma [1]; endocrinopathies such as pheochromocytoma and thyrotoxicosis [1]; autoimmune disorders including systemic lupus erythematosus and mixed connective tissue disease [1]; other hypermetabolic states such as heatstroke [1, 20], serotonin syndrome [16], malignant hyperthermia [1, 2, 16, 20], and parkinsonian hyperthermia syndrome[1, 2, 6, 19] ; drug-related conditions including toxin exposure, stimulant abuse, hallucinogen abuse, and atropine poisoning [1, 2, 5, 19]; and withdrawal states including alcohol or sedative withdrawal [1]. Considerable controversy remains regarding the distinction between malignant catatonia and neuroleptic malignant syndrome [5, 11, 19, 21, 22]. Both conditions share common symptoms including fever, rigidity, autonomic instability, and stupor, as well as laboratory abnormalities such as elevated CPK, leukocytosis, and low serum iron [5, 11, 21-23]. Catatonia may be diagnosed prior to the advent of antipsychotics and may be attributed to psychiatric disorders or medical conditions[24, 25] . Lorazepam at 1 to 2 mg intravenously is the initial treatment, with electroconvulsive therapy for refractory cases [24-26].

Laboratory Findings

No single laboratory abnormality is specific to neuroleptic malignant syndrome [1, 5]. Typical findings include leukocytosis with or without a left shift[1, 2, 5] , elevated serum creatine phosphokinase in 95% of cases [1, 2, 5], myoglobinuria in 67% of cases [1, 2, 5], metabolic acidosis or hypoxia in about 75% of cases [1, 2, 14], electrolyte abnormalities including hyponatremia and hypernatremia [1, 5], decreased serum iron levels [[1, 5], elevated serum catecholamines [1, 9], and coagulopathies [1]. In our patient, the laboratory findings were particularly severe with CPK at 60,000 IU/L indicative of massive rhabdomyolysis, creatinine clearance at 10 mL/min indicating severe acute kidney injury, bicytopenia with anemia and thrombocytopenia, coagulopathy with PT at 50%, and elevated LDH at 1,500 IU/L.

Complications: Rhabdomyolysis and Acute Kidney Injury

Rhabdomyolysis is a frequent and potentially life-threatening complication of neuroleptic malignant syndrome [1, 2, 5]. The pathogenesis involves muscle necrosis from severe rigidity, hyperthermia, and ischemia, release of muscle contents including myoglobin, creatine kinase, electrolytes, and other intracellular contents into the circulation, and myoglobinuria where myoglobin is filtered by the glomerulus and can precipitate in the renal tubules, causing tubular obstruction and direct tubular toxicity [1, 2, 5]. Acute kidney injury in neuroleptic malignant syndrome can result

from prerenal azotemia due to dehydration and hypotension, intrinsic renal injury due to myoglobinuria, and acute tubular necrosis secondary to ischemia or direct toxicity [1, 2, 5]. Risk factors for acute kidney injury in neuroleptic malignant syndrome include severe rhabdomyolysis with high CPK levels, hypotension and hypovolemia, dehydration, and myoglobinuria [1, 2, 5]. In our patient, the acute kidney injury was severe with creatinine clearance of 10 mL/min and likely resulted from a combination of rhabdomyolysis, hypotension, and dehydration. The massive CPK elevation at 60,000 IU/L indicates extensive muscle necrosis.

Management and Treatment:

Treatment of neuroleptic malignant syndrome requires a multifaceted approach [1, 5, 6, 27]. Immediate measures include discontinuation of the offending neuroleptic agent as the most critical step, supportive medical care with aggressive volume replacement and correction of electrolytes, physical cooling for extreme hyperthermia, and intensive monitoring for complications [1, 5, 6, 27].

Pharmacologic treatment options include benzodiazepines such as lorazepam at 1 to 2 mg intravenously every 4 to 6 hours as a first-line treatment that reduces rigidity and fever within 24 to 48 hours [27, 28]; bromocriptine, a dopamine agonist started at 2.5 mg 2 to 3 times daily and increased to a total daily dose of 45 mg if required, which may reduce mortality by half but can worsen psychosis [5, 27, 29]; amantadine, another dopamine agonist at 200 to 400 mg daily in divided doses, which may reverse parkinsonism [5, 27]; and dantrolene, a skeletal muscle relaxant initiated at 1 to 2.5 mg/kg intravenously, then 1 mg/kg every 6 hours, which is useful in cases with extreme hyperthermia and rigidity [5, 27, 29].

Electroconvulsive therapy has been determined to be effective in treating neuroleptic malignant syndrome, particularly in cases refractory to pharmacotherapy or when lethal catatonia cannot be ruled out [30]. A total of 6 to 10 treatment sessions are recommended, with onset of response after a mean of 4.1 treatments [30].

Supportive care in specific complications includes aggressive fluid resuscitation, alkalinization of urine with bicarbonate, and monitoring for compartment syndrome in cases of rhabdomyolysis; renal replacement therapy including hemodialysis or continuous renal replacement therapy for acute kidney injury; supportive care and correction of coagulopathy for disseminated intravascular coagulation; and mechanical ventilation for respiratory distress [1, 5]. Continuous renal replacement therapy would have been particularly beneficial for our patient as it allows for slow and gentle fluid removal, correction of metabolic acidosis, removal of myoglobin and inflammatory mediators, and management of hemodynamic instability.

Prognosis and Outcome:

Mortality rates in neuroleptic malignant syndrome have declined substantially over the past decades [1, 14, 31]. Historical mortality prior to 1984 was up to 30%, while more recent mortality is estimated at 10% to 15% [27, 32]. Predictors of mortality include myoglobinuria and renal failure with a risk of approximately 50% [14], aspiration pneumonia, cardiorespiratory failure, pulmonary embolism, and disseminated intravascular coagulation [1, 5]. Mean recovery time is 7 to 10 days after drug discontinuation [1, 2, 5], and virtually all patients recover within 30 days [1, 2]. However, long-acting depot antipsychotics may prolong episodes [1, 5]. In some patients, catatonic and parkinsonian symptoms may persist for weeks to months [7]. Regarding rechallenge after neuroleptic malignant syndrome, the recurrence rate is up to 30%[1, 2, 5] . It is recommended to wait at least 2 weeks after neuroleptic malignant syndrome resolves, to use low doses of low-potency agents, and to carefully monitor for early signs [1, 6, 11, 31].

Comparison with the Literature:

Our case presents several features consistent with typical neuroleptic malignant syndrome. The patient's age of 46 years is consistent with the literature which shows that young adult males predominate [1, 2, 5]. The offending agent was levomepromazine, a high-potency antipsychotic, and virtually all antipsychotics have been implicated [1, 2, 11]. The time to onset was days, which is consistent with the finding that 66% develop signs within 1 week [2, 5]. The cardinal features including rigidity, fever, and altered consciousness were present [1, 2, 7]. CPK elevation at 60,000 IU/L was massive, which is consistent with elevation in 95% of cases [1, 2, 5]. Rhabdomyolysis was present as a frequent complication [1, 2, 5]. Acute kidney injury was severe with creatinine clearance of 10 mL/min, which occurs in severe cases [1, 2]. Multiorgan failure was present as a severe complication [1]. Despite seven hemodialysis sessions, the patient's condition continued to deteriorate, and he died 15 days after admission. This fatal outcome is consistent with the literature reporting that myoglobinuria and renal failure are strong predictors of mortality, with a risk of approximately 50% [14]. The severity of our case with CPK at 60,000 IU/L, creatinine clearance of 10 mL/min, disseminated intravascular coagulation, and multiorgan failure represents a particularly severe presentation of neuroleptic malignant syndrome, highlighting the potentially fatal nature of this syndrome.

Conclusion:

Neuroleptic malignant syndrome is a rare but potentially lethal complication of antipsychotic use. Our case illustrates the severe consequences of this syndrome, including rhabdomyolysis, acute kidney injury, and multiorgan failure. The diagnosis was established based on Levenson's criteria, although delayed, and no specific treatment was administered. Despite seven hemodialysis sessions, the patient died 15 days after admission, illustrating the potentially fatal outcome of this syndrome when diagnosis is delayed.

The pathophysiology and specific treatment of neuroleptic malignant syndrome remain incompletely understood. General supportive measures, discontinuation of the offending agent, and aggressive management of complications remain the cornerstone of treatment. Early recognition and prompt intervention are essential to reduce morbidity and mortality. Continuous renal replacement therapy should be considered early in patients with rhabdomyolysis-associated acute kidney injury. Increased awareness among clinicians prescribing antipsychotics is essential for early recognition and timely intervention.

Lessons learned from this case:

- Early recognition is critical, as diagnostic and therapeutic delay significantly worsens prognosis.
- High-dose levomepromazine (600 mg/day), exceeding the maximum recommended dose of 400 mg/day, can precipitate severe neuroleptic malignant syndrome when rapidly titrated.
- Continuous renal replacement therapy should be considered early in patients with rhabdomyolysis-associated acute kidney injury.

- Specific treatments (dantrolene, bromocriptine) should be initiated promptly when neuroleptic malignant syndrome is suspected, even before definitive diagnosis.

Conflict of Interest:

The authors declare no conflicts of interest.

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