



ROLE OF MODIFIED ELECTROCONVULSIVE THERAPY AS ADJUNCTIVE TREATMENT IN REFRACTORY NEUROLEPTIC MALIGNANT SYNDROME: A CASE REPORT

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ABSTRACT

Neuroleptic Malignant Syndrome (NMS) is a rare, life-threatening idiosyncratic reaction to antipsychotic agents, characterised by hyperthermia, generalised muscular rigidity, altered sensorium, and autonomic instability. We report a 51-year-old male with a 19-year history of schizophrenia on long-term antipsychotic therapy who developed NMS amid acute psychiatric decompensation. Clinical features included high-grade fever, lead-pipe rigidity, acute confusional state, and markedly raised creatine phosphokinase (CPK: 9865 U/L). All antipsychotics were withdrawn immediately, and the patient was managed with bromocriptine, supportive care, and four sessions of Modified Electroconvulsive Therapy (MECT). Serial CPK values fell progressively from 9865 U/L to 112 U/L over the treatment period. Rigidity resolved, sensorium cleared, and haemodynamic parameters normalised. Concurrent right lower zone pneumonia caused by *Acinetobacter baumannii* was treated with culture-directed antibiotic therapy. This report illustrates the value of MECT as an adjunctive intervention in NMS, particularly in patients requiring long-term psychiatric pharmacotherapy. The patient was discharged after 20 days in a stable condition, with gradual reintroduction of atypical antipsychotics under close observation. Diligent serial monitoring of CPK, inflammatory markers, and clinical parameters is essential throughout NMS management.

KEYWORDS: Neuroleptic Malignant Syndrome (NMS), Modified Electroconvulsive Therapy (MECT), Schizophrenia, Bromocriptine, Antipsychotic, Creatinine Phosphokinase (CPK).

Introduction

Neuroleptic malignant syndrome (NMS), first described in the 1960s, is a rare and potentially fatal adverse drug reaction (ADR). The term neuroleptic historically referred to agents distinguishable from those with antidepressant or anxiolytic properties by their capacity to block dopamine-2 (D₂) receptors; over time it became interchangeable with antipsychotic. The syndrome retains this nomenclature

to encompass the broader range of causative agents now recognised[1].

Although NMS is individually uncommon, the widespread prescription of implicated agents means the condition is encountered with some regularity in clinical settings. Reported prevalence ranges from 0.167 to 32.6 cases per thousand patients. A meta-analysis of published epidemiological data derived an overall estimate of 0.991 cases per thousand individuals[2].

NMS was originally described as a severe complication of dopamine receptor antagonists, presenting with extrapyramidal disturbances, hyperthermia, and raised serum creatine kinase (CPK). Its clinical scope has since broadened; agents beyond conventional neuroleptics have been implicated, and analogous presentations have been documented in Parkinson's disease patients following abrupt dopaminergic withdrawal or during akinetic hyperthermic parkinsonian crises[3,4]. Prompt recognition of NMS is central to a favourable outcome; withdrawal of the offending drug is the single most important initial step. Patients who have had an episode of NMS should carry a written record identifying the causative agent and the key clinical features, so that appropriate caution can be exercised if psychotic symptoms recur[5,6].

We describe a male patient in his early fifties presenting with NMS in the context of chronic schizophrenia, Type 2 Diabetes Mellitus (T2DM), and right lower zone pneumonia, with a 15-day history of suspicious behaviour, self-directed muttering, poor self-care, and restlessness as chief complaints.

Patient Details

The index patient is a 51-year-old male, married, resident of Dehradun, Uttarakhand.

Clinical Findings

The patient was brought to the emergency department with a 15-day history of suspicious behaviour, self-directed muttering, self-neglect, and restlessness. Over the two days prior to admission, symptoms had worsened markedly, with onset of behavioural disorganisation and severely disrupted sleep. He carried a 19-year diagnosis of schizophrenia and was on Tab Chlorpromazine 50 mg, Tab Clozapine 200 mg at bedtime (HS), Tab Trihexyphenidyl 2 mg thrice daily, and Tab Memantine 5 mg twice daily. T2DM had been diagnosed one year earlier, and he reported a 40-year history of tobacco chewing.

Initial psychiatric assessment revealed a markedly altered sensorium with a depressed Glasgow Coma Scale (GCS) score, miosis, and florid delirium. The patient was admitted under psychiatry and subsequently transferred to the Medicine Intensive Care Unit (MICU-1) owing to clinical deterioration.

Physical examination revealed hyperthermia (temperature 101.7°F), tachycardia (pulse rate 130 beats per minute), tachypnoea (respiratory rate 24 breaths per minute), and oxygen saturation of 88–98%. Neurological assessment demonstrated generalised lead-pipe rigidity, coarse tremors, profuse diaphoresis, and a fluctuating level of consciousness ranging from drowsiness to unresponsiveness. The patient was disoriented in time, place, and person, with intermittent agitation. Cardiovascular examination showed tachycardia with a regular rhythm; respiratory examination revealed

bilateral air entry with crepitations in the right lower zone. Abdominal examination was unremarkable.

Physical restraints were applied during episodes of agitation to ensure patient and staff safety. Enteral nutrition was delivered via Ryle's tube given the swallowing impairment associated with altered sensorium, and a Foley catheter was inserted for strict fluid balance monitoring. An ophthalmology review was arranged in view of conjunctival congestion, and appropriate topical treatment was instituted. Sequential neurological assessments documented steady improvement in orientation and progressive resolution of rigidity over the course of treatment.

Timeline

The patient presented to Shri Mahant Indires Hospital on 29/09/2025 and was initially admitted to the Emergency Department. Due to worsening clinical condition with features suggestive of Neuroleptic Malignant Syndrome, he was transferred to the Medicine Intensive Care Unit (MICU-1).

The inpatient course extended over 20 days (29/09/2025 to 18/10/2025). During the initial phase (Days 1–7), the patient remained critically unwell; CPK peaked at 9865 U/L on Day 2 and C-reactive protein (CRP) reached 179.71 mg/L on Day 3. All antipsychotic agents were discontinued at once, and supportive therapy incorporating bromocriptine and lorazepam was started.

Right lower zone pneumonia was initially treated with empirical broad-spectrum antibiotics; therapy was narrowed to Colistin following blood culture identification of *Acinetobacter baumannii* on Day 9. Serial chest radiographs confirmed progressive clearance of pulmonary infiltrates.

The first MECT session was conducted on Day 10 of the medicine admission, with three further sessions thereafter. Each session was associated with measurable clinical improvement: rigidity diminished progressively, vital signs stabilised, and consciousness level improved. The patient was stepped down from MICU to the Medicine High Dependency Unit (HDU) and then to the general ward.

Antipsychotic therapy was reintroduced cautiously under close clinical monitoring with Tab Clozapine. The patient was discharged on 18/10/2025 in satisfactory condition, with complete resolution of NMS features.

Diagnosis

The patient was diagnosed with a case of Neuroleptic Malignant Syndrome (Primary diagnosis).

The diagnosis of NMS was established based on the presence of major criteria, including recent exposure to dopamine antagonists (chlorpromazine, clozapine, haloperidol), hyperthermia (101.7°F), and severe generalised lead-pipe rigidity. Minor criteria fulfilled included tachycardia (130 bpm), altered mental status with confusion and disorientation,

diaphoresis, leukocytosis, and markedly elevated CPK levels (peak 9865 U/L, normal range 55-170 U/L).

Therapeutic Intervention

Upon diagnosis of Neuroleptic Malignant Syndrome, all

antipsychotic medications, including chlorpromazine and clozapine, were immediately discontinued. The patient was managed in the Medicine Intensive Care Unit with comprehensive supportive care and specific pharmacological interventions.

Table 1: Pharmacological Management

Category	Drug Name	Dosage	Route / Frequency	Indication/Use
Medications	Bromocriptine	2.5 mg	Oral, thrice daily (8-hourly)	Counteract dopamine blockade
	Lorazepam	1 mg	IV, twice daily & SOS	Muscle relaxation, sedation
	Propranolol	40 mg, then 20 mg	Oral, twice daily	Autonomic support, tachycardia management
	Trihexyphenidyl	2 mg	Oral, Twice daily	Manage extrapyramidal symptoms
	Pantoprazole	40 mg	IV, Once daily (24-hourly)	Gastric protection
	Ondansetron	4 mg	IV, As needed (SOS)	Antiemetic - nausea/vomiting
Antimicrobial	Tazobactam (Piperacillin-Tazobactam)	4.5 g	IV, thrice daily	Empiric & targeted pneumonia treatment
	Clindamycin	600 mg	IV, thrice daily	Cover Gram-positive/anaerobic organisms
	Polymyxin B (Colistin)	7.5 mg	IV, 12-hourly	For Acinetobacter baumannii (post sensitivity)
Supportive Care	Normal Saline	120-150 ml/hour	IV, continuous	Aggressive hydration
	Paracetamol	650 mg	Oral/IV, as antipyretic	Fever management
	Cold sponging		Topical, as needed	Fever management
	Ryle's tube feeding	750 ml every 3 hours	Enteral	Nutritional support
	Foley's catheter		Urethral	Strict input-output monitoring
	Oxygen supplementation	Maintain SpO ₂ > 95%	As needed	Prevent/treat hypoxia
	Calcium gluconate	10 ml	IV, as needed	Electrolyte management
	Eye preparations (Sodium hyaluronate, Moxifloxacin, Carbomer gel)	As per schedule	Topical	Infection prevention, lubrication
MECT Anesthetic Protocol	Glycopyrrolate	0.2 mg	IV, before ECT	Anticholinergic reduce secretions
	Fentanyl	50 mcg	IV, before ECT	Analgesic pain relief
	Succinylcholine	50 mg	IV, before ECT	Muscle relaxant
	Propofol	100 mg	IV, before ECT	Induction/Anaesthesia
Antipsychotic Reintroduction	Clozapine	12.5 mg BD to titrate	Oral: 12.5 mg BD 25 mg BD titrate to 50 mg BD	Antipsychotic (after clinical improvement)
	Lorazepam	2 mg	Oral, at night	Sedation/anxiolytic
Supportive Oral Medications	Esomeprazole	40 mg	Oral, once daily (before breakfast)	Gastric protection
	L-Carnitine	1 tab	Oral, twice daily	Mitochondrial support/nutrition

Additional Interventions

1. Physiotherapy for passive and active range of motion exercises
2. Chest physiotherapy for pneumonia management
3. Restraints when necessary for patient and staff safety during agitated episodes
4. Serial monitoring of CPK (daily initially, then alternate days), CRP, complete blood count, renal function tests, liver function tests, and electrolytes

Follow-up and outcomes

The patient showed steady clinical improvement throughout the admission, with a sustained response to the multimodal treatment approach. Discharge Medications:

1. Tab Esomeprazole 40mg once daily before breakfast
2. Tab L-Carnitine 1 tab twice daily
3. Tab Multivitamin 1 tab twice daily
4. Tab Lorazepam 2mg at night
5. Tab Clozapine with a gradual titration schedule: 25mg BD × 4 days → 25mg morning + 50mg night × 4 days → 50mg BD

Follow-up Plan: Psychiatry OPD review after 2 weeks for assessment of psychotic symptoms and medication adjustment. Medicine OPD review on Tuesday and Friday after 7 days for monitoring of general medical status, diabetes management, and monitoring for any recurrence of NMS features.

Discussion

This case documents the management of a severe NMS episode through prompt antipsychotic withdrawal, targeted pharmacotherapy, adjunctive MECT, and structured supportive care. The sustained fall in CPK from 9865 U/L to 112 U/L across 20 days of treatment, alongside full clinical recovery, reflects the benefit of early, coordinated intervention. In this patient, prolonged concurrent exposure to a typical antipsychotic (chlorpromazine) and an atypical agent (clozapine), in the context of probable dehydration and intercurrent illness, likely produced the degree of central dopaminergic blockade sufficient to precipitate NMS. The emergence of the syndrome despite continued medication adherence underscores its well-recognised idiosyncratic and unpredictable character. A CPK concentration approaching one hundred times the upper reference limit signalled substantial rhabdomyolysis risk, necessitating aggressive intravenous hydration and vigilant renal function monitoring. MECT was introduced from Day 10 as an adjunct when pharmacological measures alone showed limited effect. Its therapeutic benefit in NMS is attributed to enhanced dopaminergic and serotonergic neurotransmission, reduction of hypothalamic dysregulation, and anti-excitotoxic properties, though the precise mechanisms remain under investigation. The use of MECT in NMS is supported by several published reports. Trollor and Sachdev (1999) reviewed 55 cases of NMS treated with ECT and observed clinical improvement in the majority, with a mean resolution time shorter than

that seen with pharmacotherapy alone[7]. Davis et al. similarly reported successful NMS reversal with ECT in patients refractory to bromocriptine and dantrolene[8]. In the present case, four MECT sessions administered over approximately ten days were each followed by measurable gains in rigidity, sensorium, and haemodynamic stability, consistent with outcomes described in the published literature. Superimposed right lower zone pneumonia caused by *Acinetobacter baumannii* added considerably to the clinical complexity. Culture-directed therapy with Colistin achieved satisfactory resolution, and the concurrent decline in CRP from its peak of 179.71 mg/L mirrored the fall in CPK, indicating parallel control of both infective and NMS-related systemic inflammation. Bromocriptine 2.5 mg thrice daily was maintained throughout hospitalisation to restore dopaminergic tone and was well tolerated, with no clinically significant hypotension or nausea recorded. Deciding when and how to restart antipsychotic therapy in a patient who has just recovered from NMS poses a significant clinical dilemma. In this patient, gradual reintroduction of low-dose clozapine (Skizoril-ER) was begun on Day 15, after haemodynamic stability was achieved and CPK showed a consistent downward trend. This stepwise approach aligns with current guidance, which recommends waiting at least two weeks after full NMS resolution, selecting an agent from a different class where possible, and beginning at the lowest effective dose under close monitoring.

A Japanese report of three patients who had experienced neuroleptic malignant syndrome and a separate report of a single, more severe case both show that using several treatments together stopping antipsychotics, providing strong supportive care, and adding other therapies can lead to recovery, but the details of each case change how treatment is given and how quickly patients improve. In the three-patient series, dantrolene and fluids slowly eased fever, disturbed consciousness, muscle stiffness, tremor, sweating, and fast heart rate, but psychotic symptoms worsened, so modified ECT was given safely after inducing anaesthesia with thiopental and using vecuronium, which made the anaesthesia time slightly longer than when suxamethonium is used[9]. The single severe case, however, showed much higher laboratory disturbance with a CPK of 9865 U/L and also had *Acinetobacter baumannii* pneumonia with a very high CRP; that patient received bromocriptine, intensive supportive care, and four MECT sessions, after which CPK dropped to 112 U/L and the patient recovered fully. These reports together emphasize that dantrolene, hydration, and ECT are important core treatments, but the amount of muscle breakdown, presence of infection, choice of dopamine-stimulating drug, and anaesthesia approach all affect management and outcomes, so care should be tailored by a multidisciplinary team.

This report is limited because it describes only one patient, so we cannot determine how much each treatment; bromocriptine, MECT, or supportive care—contributed

to recovery on its own. Future studies should look for dependable early biomarkers for NMS, clarify the best timing and number of MECT sessions, and identify which factors raise the risk of NMS coming back when antipsychotics are restarted. Even cases of NMS complicated by bacteremia and organ stress can fully recover if they are recognized early and treated by a coordinated, multidisciplinary team. The smooth reintroduction of antipsychotic medication in this patient shows that effective psychiatric treatment does not have to be withheld permanently after NMS. Ongoing communication between specialties and individualized treatment planning are essential for good outcomes.

Patient Consent Statement

Written informed consent was obtained from the patient/legal guardian for publication of this case report and accompanying clinical details.

Conflict of Interest

None

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