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Evidence Based Case Report

Organic Catatonia Secondary to Central Nervous Infection: An Evidence Based Case Report

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Abstract

Background : Organic catatonia has been associated with various infectious etiologies, particularly central nervous system infections. While benzodiazepines and electroconvulsive therapy (ECT) are the most extensively studied treatments, accurate identification and targeted management of the underlying cause remain essential for effective treatment
Aims : evaluating evidence-based therapeutic strategies for the management of organic catatonia in patients with infections.
Methods : Electronic Data Based was conducted using PubMed, ScienceDirect, Google Scholar, Cochrane, and Proquest. Eligible studies published between 2015 and 2025 included randomized controlled trials, systematic reviews, and case reports or case series focusing on the treatment of organic catatonia associated with central nervous system infections. Literature reviews, abstracts, non-English publications, and studies not aligned with the predefined PICO were excluded. Selected articles were critically appraised using the Joanna Briggs Institute (JBI) Critical Appraisal tools.
Results : 458 articles was identified, two studies (case series and case report) met the inclusion criteria. Abhijita et al. emphasized evidence-based management of organic catatonia secondary to meningitis (treated with ceftriaxone, doxycycline, lorazepam, and olanzapine, in addition to electroconvulsive therapy), while Doval et al. highlighted key diagnostic and therapeutic considerations in organic catatonia following encephalitis (treated with haloperidol, promethazine, lorazepam, amantadine, and escitalopram). Overall, these case reports offer valuable clinical insights.
Conclusion : Alongside infection-directed therapy, early recognition and timely pharmacological management of organic catatonia are essential for achieving optimal clinical outcomes.

Keywords : organic catatonia, meningitis, encephalitis, infection, treatment.

INTRODUCTION

Catatonia is a complex neuropsychiatric syndrome that remains frequently underrecognized, particularly when it arises as a manifestation of underlying medical or neurological disorders. In organic catatonia, catatonic symptoms result from general medical conditions, such as neurological diseases, infections, metabolic disturbances, or autoimmune processes, rather than from primary psychiatric illness alone. This condition can occur across all age groups and presents with a broad range of clinical features, including motor, behavioral, and autonomic abnormalities. Without timely recognition and appropriate treatment, catatonia may rapidly progress to life-threatening states such as malignant catatonia, characterized by hyperthermia, autonomic instability, severe rigidity, and impaired consciousness. Therefore, a thorough understanding of the pathophysiological mechanisms underlying organic catatonia is essential to improve diagnostic accuracy and clinical outcomes.[1–3]

Organic catatonia has been reported in association with a wide spectrum of infectious diseases, including viral, bacterial, and parasitic etiologies, with the strongest evidence pointing toward infections involving the central nervous system. A systematic review found that approximately one-fifth of catatonia cases are attributable to underlying medical conditions, among which inflammatory disorders of the CNS–encompassing both infectious (encephalitis, meningitis, etc) and immune-mediated processes–constitute nearly one-third. In certain reports, resolution of catatonia followed successful antimicrobial treatment, whereas other cases necessitated targeted neuropsychiatric interventions such as benzodiazepines or electroconvulsive therapy. The pathophysiological mechanisms linking infection to catatonia remain incompletely understood. Proposed explanations include direct toxic effects on neural tissue, secondary psychological stress responses, or systemic inflammatory pathways associated with the acute-phase reaction. Numerous pathogens have also been implicated as potential triggers of catatonic states. Notably, the majority of reported viral-associated cases involved neurotropic viruses, supporting the hypothesis of direct central nervous system involvement. Similarly, several bacterial pathogens with the capacity to invade the CNS have also been associated with the development of organic catatonia.[4,5]

Benzodiazepines and electroconvulsive therapy (ECT) remain the most extensively investigated therapeutic options for organic catatonia. Nevertheless, a standardized treatment approach has not yet been established, and widely accepted clinical guidelines for the management of catatonia are still lacking. However, benzodiazepines are still considered as first line treatment. Accurate identification and appropriate treatment of the underlying condition are fundamental components of effective organic catatonia management.[6]

This evidence-based case report describes a 22-years-old woman diagnosed with organic catatonia in the context of a suspected central nervous system infection (toxoplasmosis or meningitis or encephalitis), with overlapping clinical and laboratory findings suggestive of both toxoplasmosis and bacterial infection. The patient presented with prominent catatonic features, necessitating urgent intervention, while the underlying etiology remained uncertain despite cerebrospinal fluid analysis revealing inflammatory changes, including the presence of diplococci. Given that delayed or inadequate management of organic catatonia may lead to rapid clinical deterioration and life-threatening complications, this EBCR focuses on evaluating evidence-based therapeutic strategies for the management of organic catatonia in patients with infections.

CASE ILLUSTRATIONS

A 22-year-old woman, was brought to a hospital, with chief complaints of prolonged immobility and restlessness. Approximately three months prior to admission (August 2025), the patient developed fever and cough. During this period, she frequently accompanied and cared for her mother, who had end-stage renal disease requiring hemodialysis three times weekly for the past two years and a history of stroke seven years earlier. Following one of these hospital visits, the patient experienced fever that was managed at home with paracetamol without temperature measurement. She subsequently complained of headache and insomnia. The family initially attributed these symptoms to fatigue, as similar complaints had previously resolved spontaneously. After two days of fever, the patient began exhibiting abnormal behavior, including frequent daydreaming, bizarre actions, incoherent and tangential speech, and inappropriate urges to leave the house late at night. She expressed fear of being followed and reported hearing whispering voices. Over time, she became increasingly withdrawn, frequently remaining silent and failing to respond when spoken to by family members. Due to lack of improvement, she was evaluated by a general practitioner and diagnosed with fever and vertigo; her disorganized speech was attributed to sleep deprivation.

The patient's condition continued to deteriorate. She became unable to care for her mother or perform household tasks and ceased interacting with her family and surroundings. Most of her time was spent sitting silently. Activities of daily living such as eating, drinking, and bathing required step-by-step instructions and assistance from her sister. She was subsequently hospitalized for approximately one week under the care

TABLE 1
Case Illustration

Category	Details
Patient	22-year-old woman
Chief Complaint	Progressive mutism, immobility, withdrawal, and behavioral changes for 3 months
History of Present Illness	Symptoms began with fever, cough, headache, and insomnia, followed by psychotic symptoms (paranoia, auditory hallucinations, disorganized behavior and speech). The patient gradually became withdrawn, mute, unable to perform daily activities, and dependent on family for self-care.
Mental Status Examination	Confused consciousness, poor eye contact, hypoactive psychomotor activity, mutism, dysphoric mood, constricted affect, loose associations, and psychomotor retardation.
Neurological Findings	Neck stiffness and positive Babinski sign.
Investigations	CT scan suggestive of meningitis; MRI normal. CSF showed elevated protein, mild pleocytosis, positive Pandy test, and Gram-positive diplococci. Serology revealed elevated *Toxoplasma gondii* IgM/IgG and positive HSV-1 IgG. HIV test was negative.
Diagnosis	Organic catatonic disorder secondary to suspected central nervous system infection (encephalitis/meningitis).
Treatment	Risperidone 1 mg twice daily, lorazepam 1 mg nightly, diazepam as needed, intravenous dexamethasone, ceftriaxone, metronidazole, fluid therapy, and electrolyte correction.
Hospital Course	Hospitalized for 12 days. Catatonic symptoms persisted with mutism, withdrawal, and psychomotor slowing, resulting in significant impairment in self-care and social functioning.

of a neurologist and was reportedly diagnosed with encephalitis (brain inflammation). However, after discharge, the patient did not attend follow-up visits. Two weeks prior to the current admission, her symptoms worsened significantly. She became increasingly withdrawn, spoke only one to two words at a time, and often remained motionless without purposeful activity. She occasionally screamed or spoke incoherently, rarely slept at night, appeared frightened, and was no longer capable of caring for her mother. The family sought psychiatric consultation at Hospital due to persistent symptoms.. The patient was prescribed risperidone 1 mg twice daily; however, there was no clinical improvement. She became increasingly distressed, cried frequently, and was unable to sleep for three consecutive days, prompting the family to bring her to the Emergency Department of the hospital, where she was admitted. Initial asesment it was found the patient was largely mute, failed to respond verbally to questions, and only stared at the examiner. She frequently looked at her sister and tugged at her clothing without speaking. She was hospitalized for 12 days in the psychiatric ward. Throughout hospitalization, the patient remained mostly silent, responded slowly with brief answers, and repeatedly attempted to leave the ward when not accompanied by her sister, expressing a desire to go home. She was diagnosed with organic catatonic disorder and treated with risperidone 1 mg twice daily and lorazepam 1 mg nightly. There was no prior history of psychiatric illness. According to the family, the patient had never experienced similar symptoms, episodes of elevated mood, excessive energy, or prolonged depressive symptoms. She denied substance use. A history of headache was noted.

On examination, the patient opened her eyes spontaneously, demonstrated poor eye contact, was able to follow simple commands, but had significant difficulty communicating. She was predominantly mute and intermittently tearful. Vital signs were within normal limits. Neurological examination revealed neck stiffness and a positive Babinski sign. Mental status examination showed confused consciousness, poorly sustained and inappropriate psychic contact, hypoactive psychomotor behavior, and uncooperative attitude. Her mood was dysphoric with constricted and incongruent affect. Speech quantity and quality were markedly reduced. Thought processes were non-realistic with loose associations and psychomotor retardation. Thought content and perceptual disturbances could not be adequately assessed. Overall, these symptoms had persisted for approximately three months, resulting in significant functional impairment in role functioning, use

of leisure time, and self-care.

A contrast-enhanced head CT scan performed previously (September 1, 2025) showed features consistent with meningitis. Current laboratory investigations included TORCH and HSV panels, which revealed elevated Toxoplasma gondii IgM (1.03) and markedly elevated IgG (67.6), indicating prior infection with possible recent activity, and positive HSV 1 IgG. HIV screening was non-reactive. Chest radiography was unremarkable. Lumbar puncture demonstrated slightly turbid cerebrospinal fluid with positive Pandy test (6%), elevated protein (85 mg/dL), normal glucose ratio, mild pleocytosis (MN 8 cells/mm^3, PMN 6 cells/mm^3), and Gram-positive diplococci on Gram staining. Brain MRI was within normal limits. Although serological findings suggested toxoplasmosis, cerebrospinal fluid analysis, PCR studies, and neuroimaging did not demonstrate active infection, raising the possibility of partial resolution or an alternative infectious etiology, such as meningitis or encephalitis.

From a psychiatric division, supportive care was initiated with intravenous Ringer's lactate infusion at a rate of 20 drops per minute to maintain hydration. For acute agitation, diazepam 10 mg was administered intravenously as a slow bolus when required. Pharmacological management of catatonic symptoms included risperidone 1 mg orally every 12 hours and lorazepam 1 mg orally once daily at night. Neurological and medical management focused on treating the suspected central nervous system infection and associated inflammation. The patient was administered intravenous dexamethasone 10 mg every 8 hours to reduce cerebral inflammation. Empirical broad-spectrum antimicrobial therapy consisted of ceftriaxone 2 g intravenously every 12 hours and metronidazole 500 mg intravenously every 8 hours. Gastric protection was provided with ranitidine 50 mg intravenously every 8 hours. Electrolyte imbalance was corrected with intravenous potassium chloride supplementation. The patient received 25 mEq of potassium chloride administered at a controlled infusion rate of 2 mEq per hour.

METHODS

The clinical question was developed based on the population, intervention, comparison, and outcome (PICO) of this topic. The population (P) was determined to be organic catatonia patients with CNS system infection; the intervention (I) was any antibiotic, antiviral or standard treatment for organic catatonia with CNS infection (meningitis, encephalitis); the comparison (C) was -; and the outcome (O) was recovery or clinical improvement.

Literature search was done using online databases from PubMed, Science direct, Cochrane, Proquest and Google Scholar using keywords such as 'organic catatonia', 'meningitis', 'encephalitis', 'postencephalitic', and 'treatment'. The articles were then screened using inclusion criteria. The article selection was limited to randomized controlled trials (RCTs), systematic review, and case reports or case series studying treatment in organic catatonia with CNS infection (meningitis, encephalitis), published in the last 10 years (2015 – 2025). The exclusion criteria used in this study were literature review articles, abstracts, journals that were not in English, and studies that were not relevant to the PICO mentioned above. Filtered articles were then critically reviewed for their validity, importance and applicability using The Joanna Briggs Institute (JBI) Critical Appraisal tools.

RESULTS

A total of 598 articles were obtained from searches on PubMed, Science Direct, Google scholar, Cochrane and Proquest. The selection process is shown in Figure 1. At the end of the selection process, 2 articles were included in this study. The summary of these 2 studies is shown in Table 2. The two studies were then reviewed with The JBI Critical Appraisal tools. The critical appraisal results are shown in Table 3.

Based on the JBI critical appraisal checklist for case reports, both studies demonstrate overall good methodological quality.[7,8] In both reports, patient demographic characteristics, including age and sex, were clearly described at the beginning of the case presentation. The patients' clinical histories were presented in a chronological timeline, detailing symptom onset, disease progression, prior diagnostic evaluations, and therapeutic interventions. In one study, the patient's clinical condition at presentation was clearly described, beginning with hospitalization in the internal medicine ward for suspected meningitis, followed by organic catatonia treatment.[7] Similarly, the other study clearly reported the patient's initial presentation with altered sensorium and seizures due to meningoencephalitis, followed by partial neurological recovery and subsequent development of catatonic features, including mutism, negativism, and generalized rigidity.[8] In both reports, diagnostic investigations and assessment methods were adequately described, and the results were appropriately reported. Interventions were clearly detailed in both studies. One report described the use of antimicrobial therapy, benzodiazepines, antipsychotics, and electroconvulsive therapy,[7] whereas the other reported treatment with antipsychotics, benzodiazepines, antidepressants, and dopaminergic agents.[8] Post-intervention clinical outcomes were clearly described, with both cases demonstrating clinical improvement following treatment. However, neither report explicitly described adverse or unanticipated

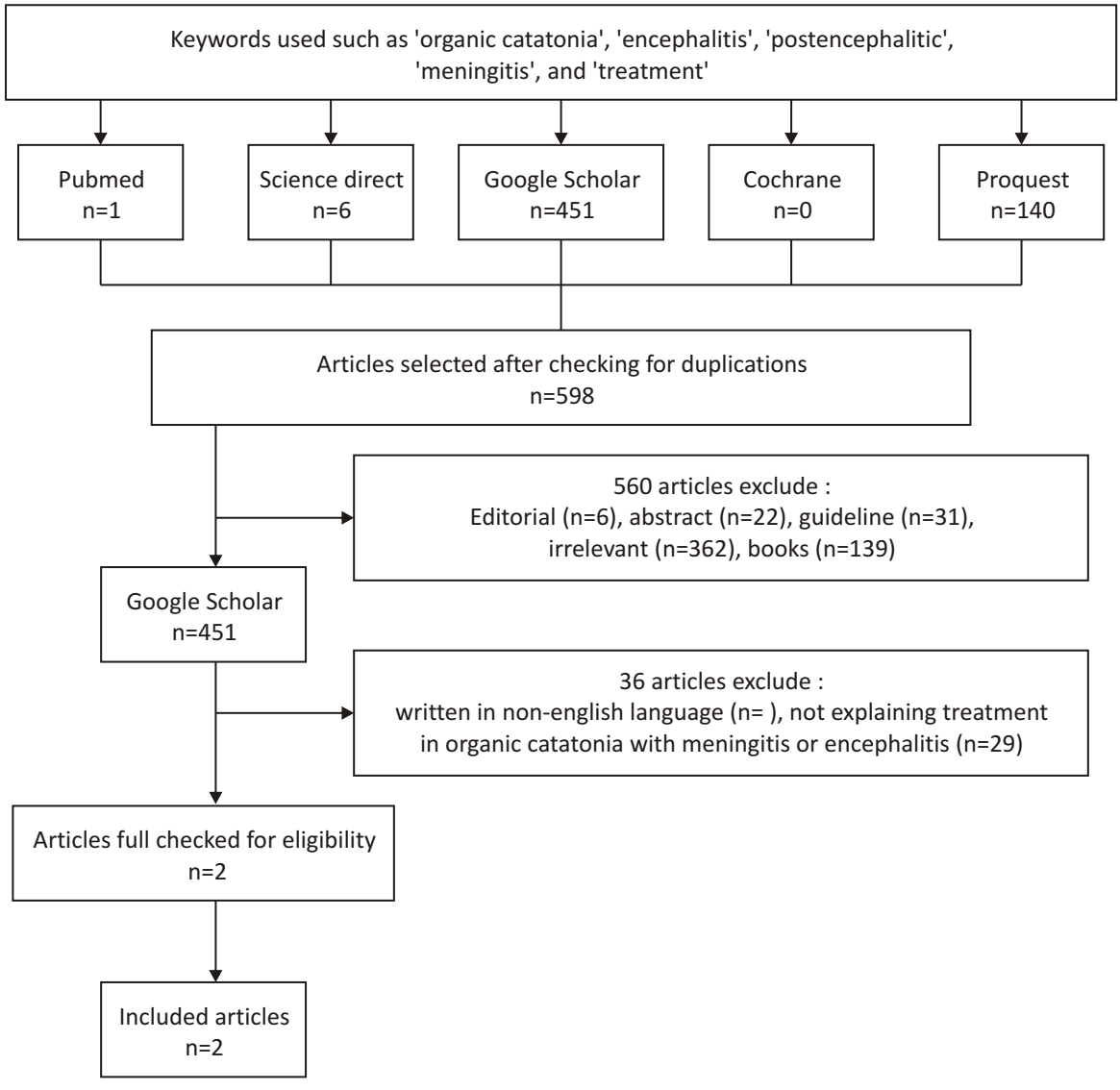


Figure 1. Articles selection process

events, which represents a limitation according to JBI appraisal criteria. Despite this limitation, both case reports provide meaningful clinical takeaways. One Study emhasized the importance of evidence-based management of organic catatonia secondary to meningitis,[7] whereas the other highlighted key considerations in the diagnosis and treatment of organic catatonia following encephalitis.[8] Overall, these case reports offer valuable clinical insights, although their inherent descriptive design limits generalizability.

DISCUSSION

Catatonia is a neuropsychiatric syndrome that has traditionally been associated with severe mental illnesses but is also increasingly recognized in the context of various general medical conditions. It is defined by a constellation of symptoms involving marked disturbances in motor activity and communication, often presenting as immobility, mutism, or reduced responsiveness, and may be accompanied by agitation, confusion, or restlessness. Historically, catatonia was regarded as a subtype of schizophrenia; however, revisions in the DSM-5 have redefined it as a distinct clinical syndrome that may occur in association with multiple psychiatric disorders. Furthermore, catatonia may also be diagnosed independently of any primary psychiatric or medical disorder.[9–11]

The conceptualization of catatonia has evolved significantly since it was first described as a separate entity by Karl Kahlbaum in 1874. Clinically, catatonia presents with a diverse array of motor, behavioral, and

TABLE 2
Summary of the studies

Author	Study design	Subjects	Summary of results	Level of Evidence
Abhijita *et al,* 2025[7]	Case series	1 case out of 4 cases (27 year old male with organic catatonia and meningitis)	The patient was initially hospitalized in the internal medicine ward with a working diagnosis of meningitis and was empirically treated with ceftriaxone and doxycycline. Two weeks after symptom onset, due to the persistence of symptoms, he was referred for psychiatric evaluation. A lorazepam challenge test resulted in a notable but incomplete improvement; despite dose escalation up to 24 mg, the patient continued to exhibit poor oral intake and impaired self-care. Consequently, electroconvulsive therapy was initiated, leading to complete remission of catatonic symptoms after six sessions, as reflected by a BFCRS score of zero. Given the presence of catatonia associated with another mental disorder, olanzapine was introduced and titrated to 15 mg daily. The patient subsequently received maintenance ECT totaling 12 sessions, while lorazepam was gradually tapered to 10 mg daily without relapse of symptoms.	4
Doval *et al*, 2015[8]	Case report	32-year-old female with Japanese encephalitis and organic catatonia	After childbirth, the patient developed altered sensorium and seizures and was treated for meningoencephalitis, with partial neurological recovery. Persistent behavioral symptoms progressed to aggression and hyperactivity; initial oral antipsychotics were ineffective, and parenteral haloperidol 10 mg plus promethazine 50 mg twice daily for 5 days controlled agitation but were followed by reduced oral intake, mutism, negativism, and generalized rigidity. She was hospitalized with a provisional diagnosis of organic catatonia as a postencephalitic sequel. Intravenous lorazepam 4 mg led to initial improvement in speech and feeding; subsequently, lorazepam was increased to 6 mg/day in three divided doses for 2 days, but the response diminished. MRI showed bilateral basal ganglia, thalamic, and temporal involvement consistent with encephalitis, and CSF was positive for Japanese encephalitis IgM. The patient was transferred to neurology care and started on amantadine 200 mg/day in divided doses; lorazepam was gradually tapered, and escitalopram 10 mg/day was added. Over the next 2 weeks, oral intake, verbal output, rigidity, and emotional dysregulation improved, with full independence in activities of daily living achieved by 3 months.	4

speech abnormalities, with a diagnosis requiring the presence of at least three characteristic signs. While it is commonly observed in patients with schizophrenia or mood disorders, catatonia may also develop in individuals with underlying medical conditions. Various general medical illnesses can either mimic catatonia or predispose patients to its development, leading to potential diagnostic delays. Case reports have

TABLE 3
Critical Appraisal of the Studies

Checklist	Abhijita *et al*, 2025[7]	Doval *et al*, 2015[8]
Were patient's demographic characteristics clearly described?	Yes. The patient's demographic characteristics, including age, sex, and socioeconomic status were clearly stated at the beginning of the case description.	Yes. The patient's demographic characteristics, including age, and sex, were clearly stated at the beginning of the case description.
Was the patient's history clearly described and presented as a timeline?	Yes. The patient's history was clearly described in chronological order, outlining symptom onset, progression, prior evaluations, and treatments over time.	Yes. The patient's history was clearly described in chronological order, outlining symptom onset, progression, prior evaluations, and treatments over time.
Was the current clinical condition of the patient on presentation clearly described?	Yes. the patient's clinical condition at presentation was clearly described, the patient was initially hospitalized in the internal medicine ward with a working diagnosis of meningitis. Two weeks after symptom onset the patient continued to exhibit poor oral intake and impaired self-care.	Yes. the patient's clinical condition at presentation was clearly described, the patient developed altered sensorium and seizures and was treated for meningoencephalitis, with partial neurological recovery, but were followed by reduced oral intake, mutism, negativism, and generalized rigidity.
Were diagnostic tests or assessment methods and the results clearly described?	Yes. The diagnostic tests, assessment methods, and their results were clearly described and appropriately reported.	Yes. The diagnostic tests, assessment methods, and their results were clearly described and appropriately reported.
Was the intervention(s) or treatment procedure(s) clearly described?	Yes. The patient received pharmacological treatment including ceftriaxone, doxycycline, lorazepam, and olanzapine, in addition to electroconvulsive therapy.	Yes. The patient was treated with haloperidol, promethazine, lorazepam, amantadine, and escitalopram.
Was the post-intervention clinical condition clearly described?	Yes. Clinical improvement achieved post treatment.	Yes. Clinical improvement achieved post treatment.
Were adverse events (harms) or unanticipated events identified and described?	Not reported	Not reported
Does the case report provide takeaway lessons?	Yes. The case report highlights important clinical lessons regarding evidence-based management of organic catatonia secondary to meningitis causes.	Yes. The case report highlights important clinical lessons regarding evidence-based management of organic catatonia secondary to encephalitis causes.

documented catatonia in association with conditions such as encephalitis, meningitis, etc. The diagnostic criteria for catatonia in the DSM-5 require the presence of three or more features, including stupor, waxy flexibility, catalepsy, mutism, posturing, negativism, stereotypy, mannerisms, grimacing, agitation, echopraxia, or echolalia. These criteria apply to both adult and pediatric populations, although in children, catatonia more frequently arises secondary to somatic illness or substance use.[9,12]

Management of catatonia typically involves benzodiazepines, particularly lorazepam, which may be used alone or in combination with antipsychotic medications to alleviate symptoms. Electroconvulsive therapy represents a highly effective definitive treatment, especially when initiated early or in cases refractory to pharmacotherapy. Given the heterogeneity of its presentation and etiologies, catatonia remains a

diagnostic challenge and is frequently underrecognized in both psychiatric and medical settings. Despite advancements in diagnostic classification with the DSM-5, clinicians must maintain a high index of suspicion and consider catatonia in the differential diagnosis of patients presenting with compatible clinical features to ensure timely and appropriate treatment.[9]

Catatonia due to a medical condition is less likely to respond to benzodiazepine therapy if the primary causes were not treated; therefore identification and treatment of the underlying cause is crucial in the treatment of organic catatonia.[6,13,14] For example, in patients with bacterial meningitis and organic catatonia, immediate hospitalization with broad-spectrum antibiotics (like Ceftriaxone, metronidazole) and possibly corticosteroids for swelling was given, while encephalitis (often viral) needs antivirals (like acyclovir for herpes), seizure control (anticonvulsants), and inflammation reduction (corticosteroids) with supportive care like IV fluids, often requiring inpatient care.[15,16] The recommended empiric ceftriaxone dosing regimen for acute bacterial meningitis in adults is 2 g every 12 h. After penicillin-susceptible *Streptococcus pneumoniae* is isolated as a causative microorganism, the ceftriaxone dose may be continued or reduced to a single dose of 2 g every 24 h, per institutional preference.[17] In patients with a suspected *Toxoplasma gondii* infection, treatment is typically initiated empirically based on clinical suspicion without waiting for confirmatory test results. The recommended first-line regimen consists of pyrimethamine, administered as a 200 mg loading dose followed by 50 mg daily in patients weighing less than 60 kg or 75 mg daily in those weighing more than 60 kg, in combination with sulfadiazine at a dose of 1000 mg four times daily for patients under 60 kg and 1500 mg four times daily for patients over 60 kg. Trimethoprim–sulfamethoxazole may be used as an alternative therapy. The induction phase of treatment should be continued for six weeks, followed by long-term maintenance therapy. Folic acid supplementation is routinely included to prevent folate deficiency, as sulfadiazine interferes with folic acid synthesis.[18]

One case report described a patient who initially presented with neuropsychiatric symptoms in the context of meningitis, was treated with empirical antimicrobial therapy (ceftriaxone and doxycycline). Despite their meningitis condition, catatonic features exist, prompting psychiatric referral. Although the patient showed partial improvement following a lorazepam challenge, high-dose benzodiazepine therapy alone was insufficient to restore basic functioning. The subsequent initiation of electroconvulsive therapy resulted in complete remission of catatonic symptoms, highlighting the effectiveness of ECT in benzodiazepine-refractory catatonia. Maintenance ECT combined with antipsychotic therapy allowed successful tapering of benzodiazepines without symptom recurrence, underscoring the role of multimodal treatment in sustaining remission.[7]

Another case report described a case of catatonia emerging as a postencephalitic sequel following meningoencephalitis in the postpartum period. Initial treatment with antipsychotics controlled aggressive behavior but was followed by worsening catatonic features, including mutism, negativism, and rigidity, illustrating the potential limitations and risks of antipsychotic monotherapy in catatonia. Benzodiazepine therapy led to initial symptomatic improvement; however, the response was not sustained. Neuroimaging and cerebrospinal fluid findings confirmed an underlying encephalitic process, and the introduction of amantadine as a glutamatergic modulator resulted in gradual and sustained clinical improvement. This case highlights the importance of addressing both residual neurological dysfunction and catatonic symptoms through targeted pharmacological strategies beyond benzodiazepines alone.[8]

Early initiation of treatment in organic catatonia is essential to reduce the risk of serious complications.[19] Prolonged immobility significantly increases the risk of deep venous thrombosis and pulmonary embolism, while reduced mobility and refusal of oral intake may lead to malnutrition, infections, and musculoskeletal complications. Despite these risks, most patients experience symptom resolution with timely and appropriate management. The presence of autonomic instability should prompt urgent evaluation for an underlying life-threatening medical condition, as treating the primary cause is crucial for clinical recovery. Benzodiazepines are still considered first-line therapy for catatonia or organic catatonia, except in cases of malignant catatonia. They act by enhancing GABA-A receptor activity and correcting inhibitory neurotransmission deficits. Lorazepam is most commonly used, although other benzodiazepines may be considered depending on clinical context. A lorazepam challenge test, administered intravenously or intramuscularly, can aid diagnosis and predict treatment response. Initial dosing typically ranges from 2–6 mg/day and may be titrated up to 12–16 mg/day, with clinical improvement usually observed within several days. Treatment duration remains variable, as premature tapering may lead to symptom recurrence.[9]

Benzodiazepines and ECT are the first treatments for which there is clinical evidence of catatonia. However, identifying and treating the underlying disorder is essential in the treatment of organic catatonia. Moreover, the role of antipsychotics is not yet entirely clear. However, there does not seem to be evidence for the use of antipsychotics as therapy for catatonic patients without any underlying psychotic disorder. Typical antipsychotics can worsen the clinical picture.[6,20]

Effective management of organic catatonia depends on accurate identification and treatment of the underlying etiology, alongside supportive care and appropriate pharmacological intervention. Therapeutic options primarily include agents that enhance γ-aminobutyric acid (GABA) activity, such as benzodiazepines, medications that augment dopaminergic transmission, including amantadine, and agents that modulate glutamatergic activity, such as memantine. In addition to reducing catatonic symptoms, benzodiazepines facilitate more thorough assessment of underlying psychopathology, thereby guiding targeted treatment of the primary condition.[21–23]

Since the 1980s, benzodiazepines have replaced short-acting barbiturates as the preferred agents for diagnostic challenge testing in catatonia. Lorazepam, administered at doses of 1–2 mg via oral, intramuscular, or intravenous routes, is the most commonly used agent. A positive response is characterized by rapid and often marked improvement in catatonic features following a single dose, enabling improved communication and clinical evaluation. Although initially considered a short-term or diagnostic intervention, benzodiazepines are now regarded as first-line therapy, with approximately 70% of patients demonstrating a rapid clinical response. Lorazepam, at daily doses ranging from 2 to 16 mg administered orally or parenterally, or diazepam, is most frequently prescribed, although other benzodiazepines such as oxazepam, clonazepam, and midazolam have also shown efficacy. The therapeutic effects of benzodiazepines are mediated through GABAergic mechanisms and may indirectly enhance dopaminergic function within the basal ganglia. In cases of malignant catatonia, higher doses of lorazepam, up to 8–24 mg per day, have been recommended. Despite their effectiveness in alleviating catatonic symptoms, benzodiazepines do not address the underlying cause, which typically requires specific treatment. While antipsychotic medications may exacerbate catatonia, they can be beneficial in managing concurrent psychotic disorders when present; however, their routine use in catatonia is generally not recommended. Catatonic symptoms are relatively common and should be actively assessed in patients presenting with psychomotor retardation or agitation. When identified early, treatment response is often favorable, with benzodiazepines serving as the treatment of choice during ongoing clinical assessment and diagnostic evaluation.[21,24,25]

CONCLUSION

This case underscores the importance of maintaining a high index of clinical suspicion for organic catatonia secondary to infectious etiologies, particularly central nervous system infections such as meningitis or encephalitis. Early identification of the underlying infection and prompt initiation of appropriate antimicrobial therapy are essential to prevent catatonic symptoms, systemic complications, and prolonged morbidity. Failure to address the primary infectious process may result in delayed recovery and results in other comorbidity such as organic catatonia. Therefore, in addition to infection-directed treatment, early recognition and appropriate pharmacological management of organic catatonia, particularly with benzodiazepines, are crucial to achieving clinical improvement. A multidisciplinary approach involving psychiatry, neurology, and internal medicine is fundamental to ensure optimal management of both the infectious process and its neuropsychiatric manifestations, thereby improving overall clinical outcomes in patients with organic catatonia.

CONFLICT OF INTEREST

The authors declare that they have no competing interests or conflicts of interest related to the publication of this article.

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