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Review

Catatonia: A Narrative Review for Hospitalists

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ABSTRACT

Background: Catatonia is a complex psychomotor syndrome commonly associated with psychiatric disorders. However, hospitalists encounter this condition on medical floors, where it is typically due to an underlying medical, especially neurological, etiology. Delays in the diagnosis of catatonia are common and lead to worsened outcomes for patients, including a multitude of medical complications, such as venous thromboembolism and stasis ulcers. Catatonia due to a medical condition is less likely to respond to benzodiazepine therapy; identification and treatment of the underlying cause is crucial.

Methods: This article provides a practical review of the catatonia literature, with a focus on diagnosis, workup, and management of catatonia for patients admitted to medical hospitals.

Conclusions: With greater knowledge about catatonia, internists are uniquely positioned to recognize and initiate treatment.

Introduction

Catatonia is a complex syndrome, characterized by abnormalities of movement and behavior. Most commonly, physicians associate catatonia with extreme withdrawal and vegetative symptoms, but agitated and hyperkinetic states also exist.[1,2] Catatonia is most often associated with psychiatric conditions, with a prevalence of 10%-25% in acute psychiatric patients.[1-4] However, neurological and medical etiologies also cause catatonia; 3% of acute neurology patients and 4% of patients in intensive care units have catatonia.[2,3] Still, this condition is underdiagnosed, by both psychiatrists and internists.[3] A recent study within a general medical hospital found that 59% of patients who retrospectively met criteria for catatonia were not diagnosed, underscoring the need for greater recognition of this condition.[3]

Catatonia can result in several medical complications including malnutrition, stasis ulcers, contractures, rhabdomyolysis, and venous thromboembolisms.[2,4,5] Additionally, catatonia can progress to a lethal variant called malignant catatonia (MC), with a mortality rate of up to 20%.[6,7] Unfortunately, delays in diagnosis and treatment are common, with a 2010 study reporting an average delay in treatment of 15 days.[8] Early diagnosis and treatment are of critical importance because delays have been shown to worsen outcomes.

Internists, especially hospitalists, are likely to encounter patients with catatonia. Therefore, it is valuable for the internist to be familiar with this syndrome so that they can recognize concerning signs, consider psychiatric consultation, and begin treatment. Psychiatric consultation can be helpful and can play a crucial role in detection of this syndrome.[3] However, psychiatric services are not universally available, so it can be helpful for the hospitalist to be comfortable understanding the differential diagnosis, workup, and treatment of catatonia. Additionally, some aspects of the management of catatonia are paradoxical, so even if psychiatry is providing recommendations, it can be helpful for internists to understand the standard treatments. Finally, the internist has an important role in monitoring for the development of the adverse sequelae associated with catatonia and preventing further morbidity. Here we present a narrative review of catatonia, with practical guidance targeted for the internist.

Catatonia Subtypes

Catatonia, for much of history, has been separated into excited/hyperkinetic or withdrawn/hypokinetic subtypes[2,6]; however, patients may experience symptoms of both hyperkinetic and hypokinetic states, and switching from one subtype to another is possible within the course of the syndrome.[7,9] Examples of hyperkinetic symptoms include excitement and combativeness; examples of hypokinetic symptoms include mutism, immobility, staring, negativism, and withdrawal; and examples of parakinetic (abnormal) symptoms include posturing, automatic obedience, waxy flexibility, and grasp reflex.[2,6,7,10] These symptoms are summarized and defined in Table 1. Generalized analgesia and bowel and bladder incontinence and/or retention may also

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Table 1
Catatonic Symptoms on the Bush Francis Catatonia Rating Scale.

Movement category	Symptom	Definition
Hypokinetic	Immobility/stupor	Decreased activity, minimal response to stimuli
	Mutism	Decreased verbal output, ranging from only a few words to total silence
	Staring	Minimal gaze movement; decreased blinking
	Posturing/catalepsy	Maintenance of a posture for a prolonged period
	Rigidity (Gegenhalten)	Maintenance of a rigid position, or resistance or stiffness with passive range of motion
	Withdrawal	Decreased oral intake; decreased eye contact
	Muscle resistance	Resistance to passive movement of limb to repositioning, with increased resistance to increased pressure
	Ambitendency	Appearance of being stuck in motor movements
	Negativism	Refusal to follow instructions or other oppositional behavior
Hyperkinetic	Excitement	Excessive motor activity
	Impulsivity	Sudden inappropriate behavior (ie, suddenly running down hallway, taking off clothing)
	Combativeness	Sudden aggression or agitation without explanation or provocation
Parakinetic	Grimacing	Contracting the face into odd facial expressions; can be episodic or maintained
	Echopraxia	Mimicking examiner's movement
	Echolalia	Mimicking examiner's speech
	Stereotypy	Repetitive non-goal-directed motor activity (ie, fiddling thumbs, patting leg)
	Mannerisms	Repetitive purposeful motor activity (ie, waving hand, combing hair); the act is unusual either in its context or its persistence
	Verbigeration	Repetition of words or phrases, "like a broken record"
	Waxy flexibility	Initial resistance to repositioning, then allowing repositioning (like a wax candle bending)
	Automatic obedience	Exaggerated cooperation with examiner (ie, continues shaking hand when told to stop; sticks tongue out if told, "stick your tongue out for me, I'm going to stick a pin in it")
	Passive obedience (Mitgehen)	Raises arm in response to light pressure of finger, even if told not to raise arm
	Grasp reflex	Grasps the examiner's fingers when the palm is rubbed
	Perseveration	Repetition of the same topic or motor movement

be present; while not part of the diagnostic criteria, these features may raise concern for catatonia.[10-13]

MC is any form of catatonia with vital sign instability.[6,10,14] Prior to the development of treatment of MC with benzodiazepines and electroconvulsive therapy (ECT), the mortality rate was 75%-100%; now, mortality has dropped to 9%-20%.[6,7]

Pathophysiology

While the pathophysiology of catatonia is not fully understood, abnormal motor pathways and alterations in neurotransmitter systems have been implicated. These alterations include reduction in GABA activity, increased glutamate activity (particularly at the NMDA receptor), and decreased dopamine activity.[2,4,15,16] Pharmacologic treatment aims to restore balance to these neurotransmitter systems.

Diagnosis

There are 2 generally accepted criteria for diagnosing catatonia. The first is the criteria set in the Diagnostic and Statistical Manual, Fifth Edition Text Revision (DSM-5 TR), with diagnosis of catatonia made in the presence of any 3 of the following 12 features: stupor, catalepsy, waxy flexibility, mutism, negativism, posturing, mannerisms, stereotypy, agitation, grimacing, echolalia (verbal repetition), and echopraxia.[17]

The second is the Bush Francis Catatonia Rating Scale (BFCRS) (see supplemental), which consists of 23 items; a diagnosis of catatonia is made with the presence of 2 or more positives on the first 14 items.[16,18] The BFCRS can also be helpful when used regularly to assess response of symptoms to treatment.

Though internists would not be expected to administer the BFCRS, they may encounter the scale in consultation with psychiatry or neurology. Therefore, it is helpful for the internist to have a general understanding of BFCRS terms and scoring. Similarly, while the internist would not be expected to memorize these terms, it is useful to have a general understanding and to consider psychiatric consultation for further examination and diagnostic clarity when these findings are present (Table 1).

The most common symptoms of the BFCRS in hospitalized patients are staring, immobility/stupor, mutism (decreased verbal output), and withdrawal.[19] Underdiagnosis has been reported to be more common if agitation, echolalia, and/or grimacing are present.[3] When these symptoms are present, catatonia should be considered, especially if the patient's status is a departure from baseline. Collateral information from family, friends, caregivers, or prior medical records will be critical when the patient's baseline is unknown.

The most common misidentified items on the BFCRS are immobility/stupor, posturing/catalepsy, negativism, withdrawal, and poor eye contact.[20] As such, these terms warrant increased attention and discussion. Immobility/stupor may present as decreased physical activity or decreased responsiveness to environmental stimuli. Posturing/catalepsy may present as holding a physical position (such as a raised arm or leg) for an extended period of time and can be assessed by moving the patient's limb; if posturing is present, patients may not retract the limb to a resting position after examination. Negativism may present as oppositional behavior; as only answering "no" to yes/no questions; or as persistent refusal to follow instructions, without clear reason or explanation. Withdrawal may present as decreased oral intake, or as decreased eye contact with interviewers. Poor eye contact, which is considered under both "staring" and "withdrawal," may also include decreased vi-

Table 2
Differential Diagnosis of Catatonia.

Diagnosis	Similarities to catatonia	How it differs from catatonia
Neuroleptic malignant syndrome	Vital sign instability Altered mental status Low serum iron Elevated CPK	Requires exposure to newly started dopaminergic agent, or recent change in dose
Serotonin syndrome	Altered mental status Rigidity Vital sign instability	GI distress Hyperreflexia Clonus
Delirium	Hypoactive and hyperactive types Decreased attention Withdrawal from environment	Worsened by benzodiazepines Other psychomotor abnormalities (echolalia, echopraxia, rigidity, etc.) not present
Coma	Hypoactivity Mutism Incontinence	Reflexes lost in coma Parakinetic symptoms not present No response to benzodiazepine challenge
Extrapyramidal side-effects (EPS)	Akathisia Dystonia (may look like rigidity or posturing)	No altered mental status No other motor symptoms present; no impaired volition No negativism Hypoactivity not present
Parkinsonism	Rigidity Motor freezing Staring	No other motor symptoms present Tremor present in Parkinsonism Patients generally interactive
Status epilepticus	Immobility Withdrawal	Specific EEG findings

sual scanning of the environment, or difficulty in switching attention between different examiners.

Differential Diagnosis

Differential diagnoses to consider along with catatonia are broad and include neuroleptic malignant syndrome (NMS), serotonin syndrome (SS), delirium, coma, extrapyramidal side effects from medications, Parkinsonism, and status epilepticus. These diagnoses may be challenging to differentiate from catatonia and will be discussed further and summarized in Table 2.

NMS is particularly challenging to distinguish from MC. Some authors consider NMS, MC, and SS to be on a spectrum of illnesses, and some consider SS and NMS to be drug-induced forms of MC.[6,10,14,16] When viewed as separate entities, NMS is more likely than MC to have lead-pipe rigidity (although it can occur in both), and NMS requires recent exposure to a change in dopaminergic agent (eg, newly started or a change in dose).[14] However, both conditions may have altered mental status (AMS), vital sign instability, low serum iron, and elevated creatine phosphokinase (CPK).[14]

Like NMS and MC, SS commonly presents with elevated temperature, rigidity, and AMS.[21] SS requires recent exposure to a serotonergic agent; however, serotonergic agents are common and include antidepressants, antiemetic agents, first- and second-generation antipsychotics, antiepileptics, St. John's wort, antimicrobials (particularly linezolid), and illicit substances (amphetamines, lysergic acid diethylamide, ecstasy, etc.).[22,23] SS is less likely to present with elevated CPK, LFT alterations, or low serum iron and is more likely to present with gastrointestinal distress, hyperreflexia, and clonus.[22,23]

Delirium is defined by a fluctuating sense of orientation and attention and, like catatonia, has both hypoactive and hyperactive subtypes.[6] Both syndromes may present with withdrawal and decreased attention. The other psychomotor abnormalities seen in catatonia, such as waxy flexibility, rigidity, echolalia, and echopraxia, are not present in delirium alone.[24] The distinction between the two is important, as benzodiazepine therapy is likely to worsen delirium but improve catatonia, and antipsychotic management of agitation in delirium may worsen catatonia.[6] Complicating matters, delirium and catatonia may coexist in some patients[24,25]; a recent paper found that catatonia is present in 12%-30% of patients with delirium.[25]

Patients with catatonia who primarily exhibit hypokinetic symptoms, such as withdrawal, mutism, and stupor, may be misdiagnosed with coma, especially those who also have incontinence and generalized analgesia.[12] Reflexes may be used to distinguish between these; in catatonia, reflexes are preserved, whereas they may be lost in coma.[12] Additionally, parakinetic symptoms such as waxy flexibility, posturing, and automatic obedience would not be expected in comatose patients. A benzodiazepine challenge, discussed in detail under treatment, would be expected to differentiate between these 2 states, as one would expect a positive response in catatonia but not coma.

Extrapyramidal symptoms (EPS) are motor side effects, such as akathisia, dystonia, or tardive dyskinesia, associated with the use of first- and second-generation antipsychotics.[26] These movement side effects may appear similar to the movement abnormalities seen in catatonia; however, patients with EPS alone are unlikely to present with other signs and symptoms of catatonia including negativism, impaired volition, AMS, withdrawal, echolalia, posturing, or vital sign abnormalities.[26]

Parkinsonism—which includes forms of Parkinson's disease, as well as EPS—can include rigidity, motor freezing, and staring, sharing overlap with catatonia. Patients with Parkinsonism are generally interactive and are unlikely to present with other signs of catatonia. In addition, patients with Parkinsonism usually have a characteristic tremor, further distinguishing this diagnosis from catatonia.[27]

Status epilepticus can present with immobility and withdrawal like hypokinetic catatonia; an EEG can differentiate the two.[15,26]

Etiologies of Catatonia

Medical Etiologies of Catatonia

When catatonia is due to an underlying medical cause, it is less likely to respond to standard catatonia treatments; therefore, it is crucial that medical causes are identified and treated.[28,29] Catatonia due to a psychiatric illness should be considered a diagnosis of exclusion.[16] The mnemonic MINDSET (Miscellany, Inflammation of the CNS, Neural Injury, Developmental disorder, Structural CNS pathology, Epilepsy, Toxin or medication) may be of benefit to help clinicians recall the most common etiologies.[30] Additional medical etiologies of catatonia are summa-

Table 3
Summary of More Common Medical Etiologies of Catatonia.

Neurological	Poisoning
Encephalitis	Carbon monoxide
Seizures	Ethylene
Structural abnormalities	Organophosphates
Vascular insults	**Endocrine**
TBI	Hypothyroidism
Hydrocephalus	Hyperthyroidism
Narcolepsy	Hyperparathyroidism
Hypoxia	DKA
PRES	Hypoglycemia
Substance Use	Wilson's disease
Benzodiazepine intoxication or withdrawal	Cushing's disease
Alcohol intoxication or withdrawal	Adrenal abnormalities
Stimulant intoxication	**Infectious**
Hallucinogen intoxication	UTI
Inhalant intoxication	Respiratory infections (esp. COVID)
Medications	Cellulitis
Disulfiram	Systemic infections
Aspirin	HIV/AIDS
Baclofen	**Autoimmune**
Bupropion	SLE
Discontinuation of clozapine	Demyelinating conditions
Steroids	Crohn's disease
Anticonvulsants	Paraneoplastic syndrome
Antibiotics	PANDAS
Antiretrovirals	**Metabolic**
Chemotherapy	Hyponatremia
Developmental	Hypercalcemia
Autism spectrum disorder	Hyperammonemia
Down's syndrome	Porphyria
Mitochondrial abnormalities	Renal failure
22q11.2 deletion syndrome	Hepatic failure
Tay-Sachs' disease	**Nutritional Deficiencies**
Tuberous sclerosis	Vitamin B1
	Vitamin B3
	Vitamin B12
	Niacin

rized in Table 3, although this table is also not all-inclusive, given the breadth of conditions that may cause catatonia.

Symptoms that favor a primary diagnosis of medical catatonia rather than psychiatric catatonia include presence of delirium, autonomic abnormalities, excitement, presence of the grasp reflex, and a history of a neurological condition including seizures.[30] As such, when these are present, suspicion of an underlying medical cause should be especially high.

Around 70% of cases of medical catatonia are due to a neurological etiology, most commonly encephalitis, particularly NMDA receptor encephalitis.[4,6,30,31] Other neurological causes include seizures, structural abnormalities or space-occupying lesions, vascular insults (such as strokes, hematomas, venous thrombosis, and aneurysms), traumatic brain injury, hydrocephalus, narcolepsy, hypoxia, and posterior reversible encephalopathy syndrome.[4,6,15,30-33]

Dementia, including Alzheimer's disease, dementia with Lewy bodies, frontotemporal dementia, Creutzfeldt-Jakob's disease, and dementia due to medical conditions such as AIDS, may also present with catatonia.[15,34,35] However, patients with catatonia may also be mistakenly diagnosed with dementia instead of catatonia; it is important to obtain a careful history including collateral, perform longitudinal assessments, and assess for response to benzodiazepine therapy to distinguish between these diagnoses.[34,35]

Intoxication or withdrawal from various substances of abuse may cause catatonia. Examples include alcohol or benzodiazepine intoxication or withdrawal, and intoxication with stimulants, opioids, hallucinogens (including LSD, PCP, MDMA, and cannabis), and inhalants.[4,6,15,30,33,36] Benzodiazepine withdrawal precipitating catatonia typically occurs when the benzodiazepine use is chronic and may occur when a benzodiazepine is tapered too quickly or inadvertently held on admission. Fortunately, this type of catatonia is highly responsive to reinitiation of home benzodiazepine regimen.[36]

Many other medications have also been implicated in causing catatonia, including disulfiram, aspirin, lithium, baclofen, steroids, anticonvulsants, antibiotics, antiretrovirals, bupropion, chemotherapy agents (particularly tacrolimus), and the discontinuation of clozapine.[4,6,15,32,33,36] Poisonings, including carbon monoxide, ethylene, and organophosphates, may also cause catatonia.[33] It is crucially important to obtain a detailed history and home medication list and obtain appropriate lab work when catatonia is diagnosed.

Endocrine etiologies of catatonia include both hypo- and hyperthyroidism, hyperparathyroidism, diabetic ketoacidosis, hypoglycemia, Wilson's disease, Cushing's disease, and adrenal abnormalities.[4,6,15,29,30,32,33]

Infections of various etiologies may cause catatonia, including those of the urinary tract, respiratory system, cellulitis, and systemic infections.[6,15,30,32] Examples of systemic infectious that may lead to catatonia include HIV, syphilis, CMV, and EBV.[32] Recently, COVID-19 has led to catatonia in a portion of affected patients.[37]

Autoimmune conditions to consider include systemic lupus (with or without cerebral involvement), demyelinating conditions, Crohn's disease, paraneoplastic syndromes, and Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal infections (PANDAS).[6,15,30,32,33]

Metabolic abnormalities to consider include hyponatremia, hypernatremia, hypercalcemia, hyperammonemia, porphyria, renal failure, and hepatic failure.[4,30,33] Interestingly, recent transplantation, particularly of the liver, has also been implicated as the precipitating factor in some cases.[33] Nutritional deficiencies to consider include vitamin B1, vitamin B3, vitamin B12, and niacin deficiencies.[15,32,33]

Developmental causes of catatonia include autism spectrum disorder (ASD), Down's syndrome, mitochondrial abnormalities, 22q11.2 deletion syndrome, Tay-Sachs' disease, tuberous sclerosis, and Prader-Willi's syndrome.[16,30,32,33,38]

Psychiatric Etiologies of Catatonia

A prior history of psychiatric illness makes the diagnosis of primary psychiatric catatonia, rather than medical catatonia, more likely; however, a prior history of psychiatric illness does not exclude an underlying medical cause.[30] Careful history, exam, and appropriate workup remain important in these patients.

Historically, catatonia was regarded as a subtype of schizophrenia, and this has resulted in a misconception that persists today.[4,7,15] The most common causes of psychiatric catatonia are the affective (mood) disorders, predominately bipolar disorder and major depressive disorder.[4,7,9,10]

Workup

Initial workup should include a complete blood count, comprehensive metabolic panel, ammonia, TSH with reflex T3 and T4, urine toxicology, iron, creatine phosphokinase, and vitamin B12 and folate levels.[7,9,10,16,32] Brain imaging, ideally MRI, and an EEG should be considered in most cases.[16,32] Additional workup to consider based on history and presentation may include HIV, RPR, COVID-19, ANA, CRP, serum copper, and ceruloplasmin, as well as a lumbar puncture with serologies for encephalitis.[16,32,33,37] A detailed history of recent exposures, including medications, should be obtained.[7]

Management

Treatment

It is worth reiterating that if the underlying cause of catatonia is medical, patients are less likely to respond to treatments described be-

low.[28,29] As such, underlying causes should be promptly identified and treated appropriately.

The first-line treatment for catatonia is benzodiazepines, most commonly lorazepam. An initial treatment is referred to as a "benzodiazepine challenge," a positive response to which is considered diagnostic of catatonia, although a negative response does not exclude the diagnosis.[39] A positive response is denoted by rapid improvement in catatonic symptoms, typically measured by comparing a BFCRS both pre- and post–benzodiazepine administration. The response to the challenge in hypokinetic patients is somewhat paradoxical, as post-lorazepam, these patients tend to become more interactive and may even convert to a hyperkinetic state. It is crucial to explain to nursing the counterintuitive response to benzodiazepines in these patients, as without education, they may hold the medication due to concerns for oversedation, which can worsen the catatonia and prolong the course. The lorazepam should be held for respiratory depression, but it should not be held when the patient is sleeping.

A lorazepam challenge is administered as 0.5-2 mg for a first dose, with lower initial dosages used for medically frail patients. Most commonly, lorazepam is administered IV, as this formulation achieves the most effective response; however, PO and IM can also be given. A lower dose can be repeated in 10 minutes if the initial results are equivocal. A positive response will typically be observed quickly, within minutes and up to 30 minutes after administration. Subsequent treatment and dosing are adjusted based on response, with a goal to improve symptoms but avoid oversedation; doses usually vary from 2 to 24 mg/day, scheduled every 6-8 hours.[6,15,16,26,40] Typically, the benzodiazepine will be titrated to achieve full resolution of catatonia symptoms before converting from IV to oral, and then tapered slowly, over weeks to months if necessary. A recent review[40] found that response rates to benzodiazepines are in the range of 65%-100%, typically seen within a few days of treatment initiation.[7,39]

In cases not responsive to benzodiazepine treatment, or if a benzodiazepine is contraindicated (such as with respiratory depression or co-occurring delirium), the NMDA receptor antagonists amantadine and memantine may be trialed.[26] Amantadine can be started at 100 mg daily and increased by 100 mg every few days up to a total daily dose of 400 mg daily. Amantadine carries the risk of precipitating psychosis, and it can lower the seizure threshold. Memantine, as a more selective NMDA antagonist, has less dopamine activity and therefore is not thought to carry the risk of psychosis.[41] Memantine is usually started at 5 mg twice a day and titrated up to 10 mg twice a day after a few days.

Antipsychotic therapy in catatonia is a matter of debate, as both first- and second-generation antipsychotics have the potential to worsen catatonia or even cause progression to MC or NMS.[40] However, when catatonia is due to underlying psychosis, initiation of an antipsychotic may be beneficial.[27] Similarly, discontinuing an antipsychotic too rapidly in these cases may cause clinical worsening. As such, careful consideration of these nuances is needed, and if psychiatry is not already involved, a consultation should be considered. In general, a second-generation antipsychotic with lower D2 affinity, such as olanzapine or quetiapine, is typically preferred over higher potency agents in the setting of catatonia, and if catatonia is not thought to be due to underlying psychosis, antipsychotics should generally be avoided.[27,40,42]

For life-threatening catatonia, such as when vital sign abnormalities (ie, MC) are present or nutritional status is gravely compromised, ECT is first-line.[2,6] ECT may also be considered in pharmacologic-resistant catatonia.[2,6] ECT is usually started at 3 times a week; scheduling may be adjusted subsequently based on response. Response rates are up to 80%, even in treatment-resistant cases.[4,6,7]

Given that NMDA receptor encephalitis is among the most common causes of medical catatonia—and that catatonia presents with high frequency in NMDA receptor encephalitis—increased awareness of this condition and its treatment is warranted.[30,43,44] Catatonia due to NMDA receptor encephalitis can be treated with benzodiazepines, ECT, and NMDA receptor antagonists; NMDA receptor antagonists may be particularly useful for catatonia in these cases, given the pathophysiology.[43,44] NMDA receptor encephalitis is particularly sensitive to antipsychotics; when these agents are administered, they may cause progression to MC and/or NMS. Antipsychotics must therefore be used with caution and discontinued if signs of MC or NMS become present.[43,44] Treatment of the underlying encephalitis may include steroids, immunotherapy (intravenous immunoglobin, or IVIG), or plasma exchange.[43]

Complications

Medical complications have been reported in up to 25% of patients with catatonia.[45] Possible complications include DVTs/PEs, nutritional deficiencies, rhabdomyolysis, pressure ulcers, deconditioning, and contractures.[5,6,33,46]

Patients with catatonia are at increased risk of developing DVTs and subsequent PEs, which may be fatal; the incidence of DVT has been reported as up to 35% in hypokinetic catatonia.[46] DVT prophylaxis should be considered in any patient hospitalized with catatonia.[5,46]

Dermatologic and neuromuscular complications, such as contractures, deconditioning, and skin breakdown, are complications of catatonic immobility.[5,6] Early engagement in physical therapy and occupational therapy, as well as mobility protocols, should be utilized as preventative measures.[5,6] Fall risk should be assessed, and appropriate precautions placed.[33] Repositioning and skin assessments should be performed regularly.[5,6]

Nutritional status, including electrolytes and hydration, in patients with catatonia should be assessed regularly, and when possible, dietician support should be sought.[5,6] Artificial nutrition may need to be considered if spontaneous oral intake is insufficient for around 5 days. Care must be taken to avoid refeeding syndrome.[5] Due to the risk of aspiration pneumonia, there is a low threshold to engage in speech therapy.[5,6]

Low serum iron in patients with catatonia has been correlated with reduced response to benzodiazepines; as such, iron should be repleted if low.[6]

Conclusion

Catatonia is a complex psychomotor syndrome that is underdiagnosed in hospital settings. Due to the significant morbidity, mortality, and medical complications, early recognition of catatonia is crucial. An appropriate workup, identification of underlying medical comorbidities, and treatment of these comorbidities is necessary.

Catatonia is less likely to respond to catatonia therapy alone if the underlying cause is medical in nature; prognosis is improved by treating the underlying medical cause. As the most common medical causes of catatonia are neurologic, emphasized by the MINDSET mnemonic, workup should generally include consideration of neurological causes. Treatment of catatonia most commonly consists of benzodiazepines, ECT, NMDA receptor antagonists, or a combination of these.

Medical complications due to catatonia can include DVTs/PEs, malnutrition, aspiration, and dermatologic and neuromuscular damage. As these complications worsen morbidity and mortality, appropriate prevention and treatment of these conditions when they occur is of utmost importance.

Psychiatric consultation can assist with diagnostic clarity and treatment recommendations and should be considered when catatonia is on the differential and psychiatric services are available. However, even when psychiatric services are available, the internist has an invaluable role in medical workup, medical management of underlying comorbidities, and prevention and treatment of complications.

Disclosure

The authors do not have any financial relationships or conflicts to disclose. Both authors were involved in the conceptualization, research, and writing of this manuscript.

Declaration of Competing Interest

The authors do not have any financial relationships or other conflicts to disclose.

CRediT authorship contribution statement

Alyssa C. Smith: Conceptualization, Methodology, Writing – original draft. **Emily G. Holmes:** Conceptualization, Methodology, Writing – review & editing, Supervision.

Supplementary materials

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