Catatonia: Clinical Aspects and Neurobiological Correlates

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Catatonia is a neuropsychiatric syndrome that can occur due to medical or psychiatric disorder. This review synthesizes over 20 years of original research and comprehensive review articles with attention to the most recent findings. Though catatonia is common and highly treatable, there have been few research studies investigating the syndrome. Pooled case reports suggest that catatonia due to an underlying general medical condition and catatonia due to a psychiatric illness can be treated similarly and that the catatonic symptoms and the underlying illness must be addressed in both types. Benzodiazepines and ECT continue to be mainstays of treatment. Evidence is mounting for the use of NMDA antagonists in catatonia refractory to lorazepam.

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Catatonia is a neuropsychiatric syndrome most commonly characterized by mutism, stupor, refusal to eat or drink, posturing, and excitement or hypokinesis. Though catatonia had been associated with schizophrenia throughout the 20th century, it is most often caused by affective disorders and medical or neurologic illness. Catatonic patients are often treated on hospital medical floors by internists, medical specialists, and consultation-liaison psychiatrists. There have been very few randomized, controlled clinical trials to investigate the nature of this illness and its treatments; most of the evidence reviewed in this article is descriptive data from almost 100 case reports published over the past 20 years. Despite the difficulty in conducting larger clinical trials, very successful treatments for catatonia have been discovered. Benzodiazepines and ECT have been the mainstay of treatment for years. As the neurobiologic understanding of catatonia has deepened, a number of new treatments have come to light, including *N*-methyl-D-aspartic acid (NMDA) antagonists, the judicious use of atypical antipsychotics, and repetitive transcranial magnetic stimulation (rTMS).

Epidemiology

The prevalence of catatonia is unknown. Catatonia is likely underdiagnosed by psychiatrists and other physicians.[1] It is common among psychiatric inpatients

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(7%–31% in six prevalence studies conducted after 1976 [total N=1,081]), and it occurs most often in patients with mood disorders (28%–31% of catatonic patients had mixed mania or mania in three studies conducted since 1977 [total N=280]).[2] Only 10%–15% of catatonic patients have an underlying diagnosis of schizophrenia as measured in five studies dating from 1932 to 1986.[2]

Catatonia caused by a medical condition is believed to be common. There are no known prevalence studies regarding the frequency of catatonia on inpatient medical units. On psychiatric units, the frequency of catatonia due to a general medical condition was measured at 20%–25%, based on three prospective prevalence studies (pooled N=65) conducted after 1985.[3–6] Case reports have been published describing a wide array of medical conditions, ranging from multiple sclerosis to uremia and metabolic ketoacidosis.

Clinical Presentation and Characterization

Catatonia includes behavioral, motor, cognitive, affective, and sometimes autonomic disturbances. To organize and quantify these symptoms for research and diagnostic purposes, a number of rating scales have been developed. The Bush-Francis Catatonia Rating Scale (BFCRS)[4] is the most widely used in research studies and case reports. Signs included on this scale—excitement, immobility/stupor, mutism, staring, posturing/catalepsy, grimacing, stereotypy, mannerisms, verbigeration, rigidity, negativism, waxy flexibility, echolalia, echopraxia, and withdrawal—should raise a high suspicion for the presence of catatonia. Impulsivity, automatic obedience, *mitgehen*, *gegenhalten,* ambitendency, perseveration, combativeness, and autonomic abnormalities are signs which can be also found in catatonia and are included in the scale. Other scales, including the Bochum-German rating scale, the University of Frankfort scale, the Stony Brook scale, and the Catatonia Rating Scale (CRS), have been published and report good inter-rater reliability within each instrument and across instruments.[7,8] In 1994 the DSM-IV included catatonia due to a general medical condition and catatonia as a specifier in mood disorders; these criteria are also often used in research studies and clinical case reports.[9]

No difference in the expression of catatonic symptoms in patients with underlying psychiatric or medical causes has been reported; the same rating scales have been used for clinical and research purposes in both populations.

In addition to behavioral disturbances, catatonia is also characterized by affective and cognitive changes. Patients with catatonia, though they often appear to have a flat affect, describe experiencing very intense emotional states. In an ongoing study (with an unreported design) Rosebush and Mazurek[10] found that the majority of patients felt very anxious and 15% thought they were either already dead or going to die. Northoff et al.[11] studied the subjective experience of 24 patients 3 weeks after their catatonia had resolved by using a self-questionnaire. He found that despite being aware of their very anxious emotional state, most patients were not aware of motor disturbances that had occurred during their previous episode of catatonia. This phenomenon has been called "motor anosognosia" or "postural anosognosia."

Catatonia Subtypes and Differential Diagnosis

The expression of the catatonic syndrome has most often been divided into excited and withdrawn types, though patients often exhibit signs of both. The underlying cause does not appear to predict which type is expressed. Patients with classic withdrawn-type catatonia appear awake and watchful, but with minimal spontaneous speech and movement. Stupor, mutism, negativism, and posturing are common signs. Excited-type catatonia is characterized by excessive purposeless motor activity associated with disorganized speech, disorientation, aggression, and violence.[7]

Catatonia with escalating fever and autonomic instability is known as "lethal" or "malignant" catatonia. Before 1960, the death rate was found to be 75%–100%, but since 1986 the death rate has dropped to 9% for reported cases, which has led to proposals to change the name from "lethal catatonia" to "malignant catatonia."[12,13]

Malignant catatonia resembles neuroleptic malignant syndrome (NMS) in many ways, but was described long before the introduction of neuroleptics.[14] Neuroleptic malignant syndrome is an idiosyncratic response to dopamine receptor antagonist medications. The incidence of NMS is estimated at 0.01%–0.02% of patients treated with neuroleptic medications.[15] Due to a number of case reports and a case-controlled study, catatonia, in particular catatonic excitement, has been thought to be a risk factor for NMS.[16,17]

Some authors also consider toxic serotonin syndrome as a subtype of malignant catatonia. It has similar characteristics and course, but is precipitated by serotonergic medications and typically has gastrointestinal

symptoms, hyperreflexia or clonus.[18] Unlike NMS or malignant catatonia, it is a toxic response that occurs in a dose-related fashion[19] whereas NMS is an idiosyncratic reaction which is not dose related.[20]

Periodic catatonia is most often described as a rare heritable subtype of catatonic schizophrenia with a chronic degenerating course.[21–23] It has been characterized by specific genetic mutations and autosomal dominant inheritance.[24,25] Gjessing described the disorder and systematically studied metabolic disturbances in these patients. He suggested that the behavioral fluctuations of periodic catatonia were related to a cyclic nitrogen imbalance and could be treated with thyroid hormone extract, which seemed to control the symptoms but not cure the disorder.[26] Periodic catatonia has yet to reach nosological definition in the DSM-IV.[27]

Neurobiology of Catatonic Symptoms

Lesions in many different brain regions (including the frontal lobes, basal ganglia, cerebellum-pons, parietal lobe, and corpus callosum[28]) as well as frontal lobe degeneration and ruptured anterior artery aneurysms[7] have been reported to cause catatonic symptoms in case reports, but it is very rare for one focal lesion to do so. Reports of medical catatonias caused by a diffuse CNS etiology are far more common than those reporting catatonia due to a single CNS lesion, supporting the hypothesis that catatonia is caused by pathway dysregulation and not as a result of a focal insult.[29] No consistent postmortem changes have been found in patients with medical catatonia.

In recent years, some authors have attempted to create a coherent neurobiological explanation for the catatonic syndrome. Northoff[11] has been the most active researcher, conducting imaging, neuropsychological, and neurochemical testing. However, clinical experience has provided the basis for most hypotheses regarding catatonia and its neurology. Because current pharmacologic interventions modify γ-aminobutyric acid (GABA)-A, glutamate, and dopamine systems, it is thought that dysregulation of each of these neurotransmitter systems may be involved in catatonia.

GABA-A agonists have been found to be very effective in quickly alleviating catatonic symptoms in most patients. Northoff et al.[30] evaluated GABA-A binding in 10 akinetic catatonic patients in a case-controlled SPECT study. He found that the GABA-A radioligand binding was significantly lower in the right lateral orbitofrontal cortex of catatonia patients compared with both psychiatric patients and healthy comparison subjects and that cerebral perfusion was significantly decreased in the right parietal cortex of catatonia patients.[1,30] Lesions in the lateral orbitofrontal cortex have previously been found to cause imitation behaviors[31] which could be likened to echophenomena. The orbitofrontal cortex is also reciprocally connected with the amygdala which could account for the state of extreme fearfulness which catatonia patients often report.[13] Administration of a GABA-agonist has been postulated to correct dysregulation of GABA-ergic tone in the orbitofrontal cortex.

There is growing clinical evidence that NMDA-antagonists can treat catatonia, suggesting that glutamate hyperactivity might be related to catatonic symptoms. It has been postulated that NMDA hyperactivity causes dysregulation of GABA-A function and that NMDA antagonists can indirectly restore GABA-A function in the frontal lobes, though more slowly than GABA-A agonists.[32] Northoff[11] hypothesizes that either excess glutamate or hyperactivity of glutamate receptors causes dysfunction of the posterior parietal lobe. This could lead to symptoms such as posturing and impairment in spatial positioning that occur in patients with lesions of parietal cortex and in patients with catatonia. Carroll et al.[33] reported a case in which a patient with vascular dementia with no perfusion to the left posterior parietal area (as measured by SPECT) and catatonia refractory to multiple treatments finally responded to memantine, an NMDA antagonist. The authors proposed that memantine may have reduced excess glutamate at the NMDA receptor within a circuit that connects the parietal lobe, the subcortex and the frontal cortex, thus reregulating neurotransmitter function in these areas.[33]

The role of dopamine in catatonia is very complex. Dopamine pathways play a central role in catatonia, malignant catatonia, and neuroleptic malignant syndrome in conjunction with other neurotransmitter systems; however, the exact role of dopamine has yet to be clarified. Atypical antipsychotic medications which block D_2 receptors (among other receptor actions) have been found to cause and treat catatonia as well as cause progression to neuroleptic malignant syndrome. The lateral and medial orbitofrontal cortical areas are each connected to the subcortex through a "thalamocortical loop" which connects the cortical area to the striatum, pallidum/substantia nigra, the thalamus and then back to the cortex.[34] These loops are heavily modulated by

dopamine.[29] The dopaminergic circuit is in turn modulated by GABA and by serotonergic projections from the dorsal raphae.[7]

Northoff[11] proposes that the initial neurochemical dysregulation in catatonia occurs in the cortex, with involvement of the GABA-A system and subsequent dopamine dysregulation in the thalamocortical loops and subcortex. He also postulates that the "top-down" dysfunction of dopamine could lead to catatonia and malignant catatonia, whereas NMS may originate as a dopamine dysregulation in the subcortex and subsequently cause catatonic symptoms (in some cases) via "bottom-up" dysregulation of the GABA-A function in the orbitofrontal cortex.[11] Mann[34] proposes that the medial orbitofrontal cortex thalamocortical loop to the lateral hypothalamus causes the autonomic symptoms of malignant catatonia/NMS.

Catatonia and Seizure Activity

Because of the effectiveness of benzodiazepines and ECT in catatonia, two treatments that usually raise seizure threshold, it has been proposed that catatonia may be caused by underlying seizure activity.[35] Though seizures are one of the most commonly reported causes of medical catatonia, there is no evidence that seizure activity underlies every case of catatonia. EEG changes have been found in catatonic schizophrenia patients, but it is not clear whether these changes were state indicators or trait indicators.[36] No study has ever consistently found seizure activity by EEG in catatonia, but subcortical seizures cannot be eliminated as a cause.[10]

Making a Diagnosis and Finding an Underlying Cause

Catatonia is always caused by an underlying medical, neurological, or psychiatric illness. Identification of the symptoms of catatonia must be made while at the same time determining its cause.

Syndromes and symptoms to be differentiated from catatonia include coma,[37] akinetic mutism, abulia, hypoactive delirium, and locked-in syndrome.[38] An accurate diagnosis can change the treatment and prognosis dramatically. Alisky[39] reports the case of a 90-year-old man who was found unresponsive and was thought to be in a coma. Though the patient did not respond to any stimuli, he was found to be purposefully squeezing his eyes closed; he was diagnosed with catatonia after "waking up" with a dose of lorazepam.

To thoroughly identify and monitor signs and symptoms of catatonia, it can be helpful to use a standardized examination and rating scale for catatonia. History from family and friends will be particularly important as catatonic patients are often mute.

The broad array of medical causes that can underlie catatonia demands an extensive workup including a thorough history, physical and neurologic exam. Reports of severe psychiatric symptoms suggest a catatonic state of severe intensity. A drop in weight may suggest poor PO intake. Malingering may need to be investigated. HIV risk factors should be assessed. A very thorough medication and substance abuse history can provide valuable information. Suggested laboratory tests include CBC, BUN, and creatinine, serum glucose level, thyroid function tests, RPR, B12, folate, liver function tests, creatinine phosphokinase, therapeutic medication levels, HIV/AIDS test, serum iron, urine drug screen, urinalysis, EEG, brain MRI, lumbar puncture (if signs of encephalitis or meningitis). A workup for systemic lupus erythematosis may also be indicated. Finally, a "test dose" of lorazepam as described below may also help to make the diagnosis of catatonia.

Medical and toxic causes of catatonia must always be considered, even if a psychiatric cause is suspected, because there are often multiple causes of catatonia.[40] Over one hundred medical conditions have been reported to cause catatonia; most commonly these are processes that cause diffuse cerebral dysfunction such as encephalitis, seizures, metabolic disturbances, phencyclidine exposure, systemic lupus erythythematosus, corticosteroids, and disulfiram toxicity.[27,41] Focal disturbances such as CNS structural damage, infections, and focal seizures can also cause catatonia.

Immediately dangerous etiologies such as NMS, epilepsy,[33] encephalitis, mass lesion or cerebrovascular infarction[42] need to be ruled out quickly. Most authors recommend an EEG and brain imaging be performed in all patients with catatonic symptoms.[43] Seizures should be suspected especially if episodic bradycardia is present.[44]

Many nonpsychiatric medications can cause neuropsychiatric symptoms and potentially lead to catatonia. In one case report, sibutramine was postulated to cause psychosis and subsequent catatonia via dopamine uptake inhibition and increased dopaminergic tone. Clarithromycin, azithromycin and amoxicillin have all been reported to cause psychotic symptoms at times leading to catatonic symptoms. Minocycline has been described as improving catatonic symptoms

in two patients with schizophrenia, possibly via NMDA antagonism.[45]

Medical problems that cause diffuse neuropsychiatric symptoms, such as systemic lupus erythematosus and HIV/AIDS, have been reported to cause catatonia in a number of case reports. Systemic lupus erythematosus causes neuropsychiatric disturbances in 50–70% of patients, even in cases without cerebritis. Ahuja et al.[46] support treating catatonia due to systemic lupus erythematosus with corticosteroids, immunosuppressive agents and plasma exchange, in addition to treating the catatonia with benzodiazepines and ECT. Neuropsychiatric disturbances due to the direct effect of HIV/AIDS, as well as CNS opportunistic infections, have also been reported to cause catatonia. Ferrando and Nims reported a case of HIV-associated mania that progressed to catatonia in a man with no focal neurologic lesions and a CD4 lymphocyte count of 33 cells/mm^3. The mania and catatonia were effectively treated with benzodiazepines, ECT and highly active antiretroviral therapy.[47] Cancer can cause both focal CNS masses and leptomeningeal disease, as well as paraneoplastic encephalitis, all of which can lead to catatonia. Treatments can be aimed at eliminating focal lesions with radiation and chemotherapy, or treatment of the encephalitis while treating catatonic symptoms.[48] One case report describes vitamin B_{12} deficiency as a possible cause of mental status changes and catatonia; treatment with vitamin B_{12} supplementation alone was an effective treatment.[49]

Interestingly, a number of case reports describe catatonia following liver transplantation. Liver transplant recipients have the most neuropsychiatric sequelae among transplant patients; the relationship to alcoholism, hepatitis C, and immunosuppressant medications remains unclear. Three case reports describe patients who received liver transplants (diagnosed with cirrhosis related to hepatitis C, alcohol abuse, and one for which the cause was not reported). All patients developed catatonic signs on postoperative day two or three and were successfully treated with benzodiazepines alone. There are no case reports describing catatonia in other types of organ transplantation.

Patients with NMS or malignant catatonia can have signs of catatonia as well as dysarthria, diaphoresis, sialorrhea, incontinence, myoclonus, tremors or tachypnea[50] along with autonomic instability, muscle rigidity, delirium, and leukocytosis. Laboratory abnormalities include a very high creatine phosphokinase, leukocytosis, thrombocytosis, and elevated LFTs. Low serum iron has been found in catatonia and NMS; one study found that NMS was more likely to occur in catatonic patients with low serum iron, but this has not been replicated.[51–53] It has been postulated that up to 10% of patients with catatonia treated with neuroleptics will develop NMS.[54]

Treatment

While identification and treatment of underlying etiology are sought, treatment of catatonia begins with supportive care to reduce the risk of morbidity and mortality caused by immobility and poor nutrition. A number of case reports have described the risks of catatonic immobility: bed sores, flexion contractures, rhabdomyolysis, deep venous thrombi, and pulmonary emboli, urinary retention, infection, aspiration pneumonia, and death.[55–58] Treatment with subcutaneous heparin, urinary catheterization and a high level of nursing care can prevent these complications. Catatonic patients who refuse to eat or drink can become dehydrated and malnourished and require IV fluids. Nasogastric tube feeds and PEG tube placement may be required. Severe deconditioning along with malnutrition may require extensive physical rehabilitation.

Pharmacologic treatments and ECT are the primary treatment interventions for catatonia. Among these treatments, there has only been one prospective randomized placebo-controlled trial that was conducted with lorazepam in chronic catatonia patients with schizophrenia. In case reports, catatonic symptoms due to both psychiatric and medical causes seem to respond similarly to the standard treatments for catatonia; in both cases the underlying illness must also be addressed.[6]

Benzodiazepines and ECT have been the first-line treatments for almost all types of catatonia. In 1995, Hawkins et al.,[59] in a retrospective literature review including 178 psychiatric inpatients with catatonia, found that lorazepam was the most commonly used treatment, resolving symptoms in 70% of reported cases. ECT alone resulted in resolution of symptoms in 85%, whereas antipsychotics alone were effective in only 7.5% of cases. In malignant catatonia, the response to ECT was 89%, response to benzodiazepines was 40% and response to antipsychotics was 0% (0 of 2).[59]

Benzodiazepines

Benzodiazepines are all GABA-A agonists. It has been hypothesized that GABA-A agonists correct deficient

GABA-ergic function in the orbitofrontal cortex.[10] Lorazepam is the most commonly used benzodiazepine in the treatment of catatonia, but other benzodiazepines such as diazepam,[60] oxazepam,[61] and clonazepam[62–64] have also been reported to treat catatonia. An open prospective study found that of 18 psychiatric inpatients with catatonic signs, two patients showed an immediate response to intravenous lorazepam and 16 patients overall showed significant clinical improvement; nine patients of the responding patients later required ECT for complete remission.[65] Numerous authors have found that giving lorazepam 1–2 mg intravenously to a catatonic patient can dramatically reduce catatonic signs and symptoms within minutes; this can be helpful but not definitive in making a diagnosis of catatonia.

It has been noted that patients with catatonia due to a medical or affective illness may respond better to lorazepam than do patients with schizophrenia with catatonic features. A 12-week-long randomized double-blind, placebo-controlled cross-over study conducted in 18 patients diagnosed with schizophrenia and exhibiting chronic catatonic symptoms as measured by the Bush-Francis Catatonia Rating Scale found no statistical difference between groups treated with 6 mg/day of lorazepam and those not treated with lorazepam.[66]

No consensus on the dosing schedule of lorazepam for catatonia has been established. Most authors suggest starting at 1–2 mg intravenous lorazepam every 4–12 hours and increasing this over the course of days with the goal of relieving catatonic signs and symptoms but not sedating the patient, which carries the risk of aspiration. The duration of treatment with benzodiazepines has not been firmly established, but a number of authors report that it is necessary to continue the benzodiazepines until the causative illness has been fully treated[67,68]; otherwise the patient will be at high risk for relapse. One case report describes a patient with idiopathic recurrent catatonia who required chronic lorazepam treatment.[69]

Zolpidem

Zolpidem, like the benzodiazepines, is a GABA-A agonist and has been reported in one case series to be effective in the treatment of catatonia.[70] Zolpidem has a quick onset (15 to 30 minutes) which makes it a useful diagnostic tool, but it lasts only 3–4 hours, requiring frequent administration. It was reported to be effective in one case where lorazepam failed.[71]

Electroconvulsive Therapy

Available studies and case reports suggest a high success rate in treating all types of catatonia with ECT.[72] It has been found to be effective in catatonia refractory or partially refractory to lorazepam.[69] There are few studies regarding electrode placement, assessment of seizure threshold, and frequency of trials—these have yet to be standardized. Most reports of successful treatment of catatonia use bitemporal ECT, though there is one case report using biparietal ECT, which was used to augment stimulus intensity.[73] There has been a general consensus among specialists that temporal or frontal placement is the most effective. One case report described the rapid discontinuation of benzodiazepines in anticipation of ECT treatment which caused an exacerbation of catatonia.[74] Some physicians describe discontinuing benzodiazepine treatment just prior to ECT, whereas others recommend continuing benzodiazepines during and beyond the ECT treatments.[45] A synergistic effect between benzodiazepines and ECT has also been postulated.[75]

ECT poses a risk in hypokinetic patients; ECT normally causes a transient rise in serum potassium levels, but when administered to a patient after a prolonged immobilization, significant hyperkalemia leading to life-threatening arrhythmias may occur.[76] In addition, there is a recommended waiting time of 3 months after a myocardial infarction[77] and concern for increased intracranial pressure with ECT in patients with CNS space-occupying masses. In these cases, a risk-benefit ratio needs to be assessed.

Repetitive Transcranial Magnetic Stimulation (rTMS)

A number of case reports have found that rTMS can be used successfully to treat catatonia,[78,79] and one showed success in a case in which lorazepam had failed.[80]

NMDA Antagonists

N-methyl-D-aspartic acid antagonists may provide clinicians with another option when benzodiazepines and ECT fail or are not an option. Case reports have been published describing the success of NMDA antagonists amantadine and memantine in treating catatonia.[32,81,82] Amantadine can have anticholinergic side effects and can also increase dopaminergic tone.[83] One case report described successful use of amantadine 100 mg daily in a 38-year-old man with catatonia related to end stage renal failure and major depression, after ECT failed.[84] More recently, a review of glutamate antagonist ther-

apy in the treatment of catatonia was published by Carroll et al.[85] They found 25 reported cases of catatonic patients who had received either amantadine (200–500 mg PO or IV) or memantine (5–20 mg oral) in addition to their original regimen for catatonia. All showed a strong positive response, with initial response time ranging from 1 to 7 days. The effect of NMDA antagonists is slower than that of benzodiazepines—effects can be seen within 24 hours with complete recovery occurring within 3 weeks.[86]

Antiepileptics

Topiramate is a novel antiepileptic agent that is thought to increase GABA-ergic tone and augment GABA activity as well as indirectly reduce the activation of AMPA (non-NMDA glutamate receptor).[82] One case series found that topiramate was effective in four cases of catatonia refractory to benzodiazepines and divalproex, though two patients relapsed while still taking the medication.[87]

One case report found valproate to be effective in 2 siblings with catatonic schizophrenia who do not respond to benzodiazepines, though their therapeutic effects took longer.[88] Its efficacy was postulated to be related to its GABA-ergic properties.

Atypical Antipsychotics

The role of atypical antipsychotics in the treatment of catatonia remains unclear; it is possible that atypicals directly treat catatonia. In addition to causing a D_2 receptor blockade, atypicals have weak GABA-agonism and $5HT_2$ antagonism that could stimulate dopamine release in the prefrontal cortex and help alleviate catatonic symptoms.[89] Though many case reports describe the successful use of antipsychotics in catatonia (in patients with schizophrenia and psychotic symptoms in particular), a few case reports suggest that they can cause catatonia.[90] Moreover, all atypical antipsychotics have been found to cause NMS. Given that catatonia patients are believed to be at high risk for NMS,[91] some authors recommend against their use entirely.[92] In the literature, multiple case reports and retrospective studies[93] indicating the successful treatment of catatonia with atypical antipsychotics (olanzapine, risperidone, ziprasidone, aripiprizole, and clozapine)[89,94–96] have far outnumbered reports of atypical antipsychotics causing catatonia, though these studies were largely in patients with schizophrenia and only one focused on a patient with a medical illness.[97,98] In a recent review of the use of atypicals in the treatment of catatonia that included 10 case reports of successful treatment of catatonia with atypical antipsychotics, Van Den Eede et al.[99] advised using atypical antipsychotics in catatonic patients with caution, given the risk of NMS. They postulated that treatment with an antipsychotic might target the psychiatric illness that is driving the catatonia, rather than treating the catatonia itself. This would imply that using atypical antipsychotics in patients with catatonia due to a general medical condition would not be appropriate. It remains unclear as to whether atypical antipsychotics should be used in cases of catatonia due to a general medical condition.

CONCLUSION

Psychiatrists are in a position to diagnose and treat catatonia on both medical and psychiatric units, given the combination of medical and psychiatric problems that catatonic patients often present. Though catatonia is highly treatable and more common than thought, there have been very few research studies investigating the prevalence, pathophysiology, and treatment of the syndrome. Much more research is required to understand its biological underpinnings and the most effective treatments. Pooled case reports suggest that catatonic symptoms due to medical conditions can be treated similarly to those due to psychiatric conditions and that the catatonic symptoms and the underlying illness must be addressed in both types. Benzodiazepines and ECT continue to be mainstays of treatment, and evidence is mounting for the use of NMDA antagonists in catatonia refractory to lorazepam. The safety and usefulness of atypical antipsychotic medications in catatonia due to a general medical condition remain unclear, though there appears to be some evidence for their effectiveness in patients with catatonic schizophrenia.

An increased awareness of the diagnosis and treatment of catatonia among clinicians has the potential to significantly improve morbidity and mortality of catatonic patients. In addition, catatonia provides us with a unique window into how the mind works. Further research into its pathophysiology and clinical treatment will offer us an excellent chance at expanding our understanding of psychiatric illness and its causes.

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