

A 6-Year-Old With Becker Muscular Dystrophy and Catatonia

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Catatonia is often underrecognized and underdiagnosed, particularly in young children. Catatonia occurs secondary to a medical and/or psychiatric etiology. The diagnosis can be supported by validated rating scales, including the Pediatric Catatonia Rating Scale and/or response to a dose of a benzodiazepine, often lorazepam. We present the case of a 6-year-old boy with acute behavioral changes in speech, affect, awareness, and new odd behaviors, admitted to a quaternary care pediatric hospital for further assessment and work-up of symptoms consistent with catatonia. Laboratory testing was notable for previously undiagnosed Becker muscular dystrophy (BMD), although otherwise unremarkable. He received many medication treatments for catatonia, including escalating doses of lorazepam and augmentation with memantine, zolpidem, quetiapine, and ultimately clozapine, without adequate symptom improvement and with side effects including significant weight gain. Neither behavioral interventions nor empirical treatment of presumed seronegative autoimmune encephalitis with intravenous immunoglobulin and methylprednisolone conferred benefit. He ultimately received electroconvulsive therapy, with significant and relatively rapid improvement occurring over 2 weeks after a 4-month hospitalization. This case highlights the importance of the identification of catatonia to guide evidence-based treatment and the value of timely access to electroconvulsive therapy for pediatric patients. While the etiology of his catatonia remains undetermined, his diagnosis of BMD could play a role and further supports the importance of a comprehensive medical work-up.

INTRODUCTION

Catatonia is a neuropsychiatric disorder characterized by abnormalities in movement, affect, and behavior that is associated with significant morbidity and mortality.¹ Catatonia occurs in the context of psychiatric and/or medical diagnoses.² Presentations vary widely³ and have been further characterized as: 1) hypokinetic/stuporous catatonia, the most common presentation consisting of symptoms such as mutism, immobility, and staring; 2) hyperkinetic/excited catatonia, accounting for approximately 20% of catatonia presentations and consisting of symptoms such as agitation and impulsivity; and 3) malignant catatonia, accounting for approximately 5% of presentations and marked by autonomic instability and hyperpyrexia.⁴ Pediatric catatonia incidence appears to be rising,^{5,6} likely in part due to increased catatonia identification and recognition of medical etiologies, which account for approximately 20% of pediatric catatonia cases. First-line treatment of catatonia consists of benzodiazepines, most commonly lorazepam, with electroconvulsive therapy (ECT) for severe or refractory cases.^{7,8}

abstract

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Dr Lichtor conceptualized and designed the case study, drafted the initial manuscript, and critically reviewed and revised the manuscript. Dr Robinson was involved in the figure creation, drafting the initial manuscript. Drs Pumphrey and Luercio were involved in conceptualization, drafting the initial manuscript, and critically reviewing and revising the manuscript. Drs Okopie and Landi were involved in drafting and critically reviewing the manuscript. Drs Gorman and Siegel critically reviewed and revised the manuscript. Dr Samsel was involved in the conceptualization and design of the case study, and critically reviewing and revising the manuscript. All authors approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

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Medication Doses and Catatonia Rating Scale Scores

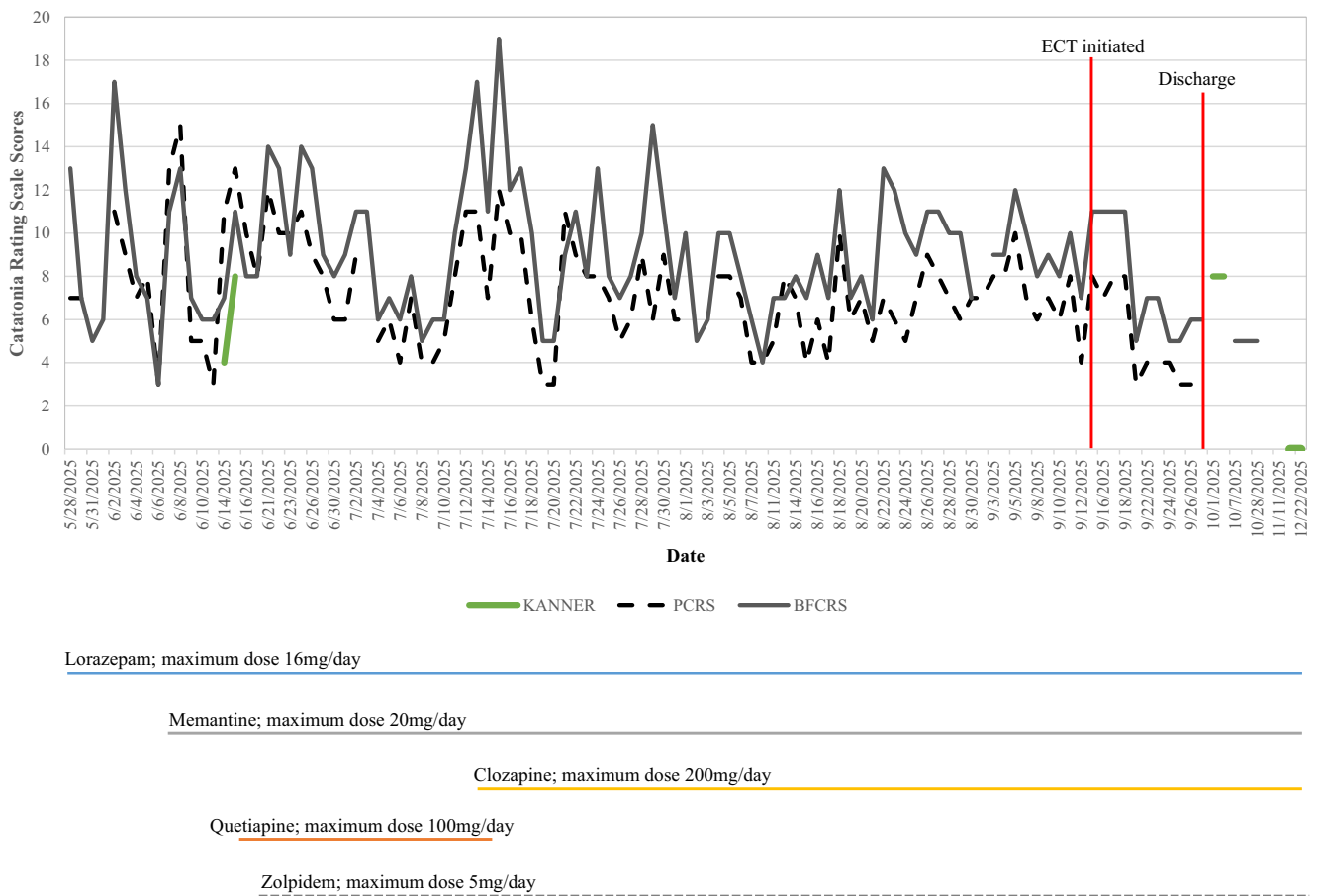


FIGURE 1.

Catatonia treatment and associated rating scale scores. Length and location of lines for each medication treatment across the timeframe of hospitalization. There was no clinically significant response to initiation or dose escalation of lorazepam, memantine, zolpidem, or quetiapine. Catatonia rating scales declined when clozapine reached maximum dose and after ECT initiation.

Abbreviations: BFCRS, Bush Francis Catatonia Rating Scale; ECT, Electroconvulsive Therapy; KANNER, Katatonia Autism Neuropsychiatric and Neuromovement Examination Rating; PCRS, Pediatric Catatonia Rating Scale.

We present the case of a 6-year-old admitted for medical work-up for acute behavioral change, ultimately diagnosed with catatonia. A previously unidentified in-frame mutation within the dystrophin gene consistent with Becker muscular dystrophy (BMD) was identified via rapid genome sequencing. He received multiple catatonia treatments over 4 months without adequate improvement (Figure 1). Ultimately, symptoms resolved in a matter of weeks after initiation of ECT. His case highlights the importance of a high index of suspicion for catatonia in children with acute behavioral change or regression, as well as the need for access to evidence-based definitive treatment, including ECT, even for younger children.

CASE

This patient is a 6-year-old boy with attention deficit/hyperactivity disorder (ADHD), combined type, who

presented to a quaternary pediatric hospital with changes in motor, affect, and behavior domains occurring over days. Symptoms included repetitive movements such as tapping his head or repeatedly reaching out as if to grab something, not responding to questions posed, not eating or drinking, and having episodes of urinary incontinence.

He was born full term and adopted shortly after birth. There were no known complications in the pregnancy or delivery. He met his developmental milestones, although he received occupational therapy and counseling in the school setting due to the impact of his ADHD, combined type, diagnosed via neuropsychological testing that he underwent related to noted dysregulation and impulsivity. He did not meet the criteria for autism spectrum disorder. He had a first-degree biological relative with bipolar disorder and ADHD, although the rest of his family's psychiatric history, including catatonia, is unknown. He was prescribed

immediate-release guanfacine 3 mg at bedtime and methylphenidate 3 mg in the morning, 1 mg in the afternoon by his outpatient psychiatrist for ADHD without a recent dose change. Approximately 2 weeks before presenting to the emergency department, he was seen by his psychiatrist because of concerns for anxiety, for which fluoxetine 8 mg liquid formulation daily was initiated. This was discontinued 3 days later after he started to exhibit changes in behavior. There were no other identified precipitants (ie, trauma or stressors or toxic ingestion) to the behavioral change.

He was brought to the emergency department approximately 1 week after he started to exhibit abnormal behaviors, including odd repetitive movements, not interacting or responding appropriately, and was not able to engage in school, prompting his pediatrician to refer him for further assessment and expedited work-up. On evaluation, the psychiatry consultation service reviewed his history and the videos provided by his parents. His symptoms of excitement, negativism, stereotypies, staring, grimacing, and mutism were consistent with catatonia. He received intravenous (IV) lorazepam 0.5 mg with symptom improvement and Pediatric Catatonia Rating Scale⁹ scores decreasing from 6 to 3, supporting a diagnosis of catatonia.

He underwent extensive testing, including investigation for potential infectious, autoimmune, endocrine, oncologic, metabolic, genetic, and hematologic contributors to his presentation (Table 1). Notable abnormalities included serum autoimmune encephalitis panel with anti-GAD-65 antibodies 0.11 nmol/L (normal ≤ 0.02) with negative cerebral spinal fluid (CSF) anti-GAD-65 antibodies and autoimmune encephalitis panel including anti-NMDA receptor antibodies, and elevated creatine kinase (initially 1483, reaching maximum 14 632 U/L). Notably, rapid genome sequencing identified a pathogenic hemizygous in-frame deletion of exons 48–51 in the Duchenne muscular dystrophy (DMD) gene, resulting in a diagnosis of X-linked DMD-related dystrophinopathy, consistent with BMD.

Given the severe symptoms, lack of definitive alternative explanation, limited improvement with lorazepam, and after extensive multidisciplinary discussions with the parents, he received empirical courses of methylprednisolone 30 mg/kg/d for 5 days and IV immunoglobulin 2 g/kg twice for possible seronegative autoimmune encephalitis,¹⁰ without improvement. Treatment of catatonia featured IV then oral lorazepam, increased to a maximum of 16mg/d (approximately 0.6 mg/kg/d). Given inadequate catatonia improvement, treatment was augmented with memantine up to 20 mg/d with improvement in emotional lability but no significant improvement in catatonia. Zolpidem 5 mg at bedtime was added as a dual insomnia and catatonia treatment augmenting agent. Guanfacine was optimized to a maximum daily dosing of 4.5 mg to attempt to address impulsivity and emotional dysregulation that could

have been attributed to ADHD rather than catatonia. Board Certified Behavior Analyst and Speech Language Pathologist were consulted, although therapeutic interventions did not significantly impact his behavior. Quetiapine was added and titrated to 100 mg/d without improvement or sedation (see Figure 1 for medication timeline). Lorazepam was transitioned to clonazepam because of the potential that the neuroplasticity of a developing brain could impact acclimation to a shorter-acting benzodiazepine.^{11,12} This led to some improvements in motor signs of catatonia, although he became more emotionally labile, less engaged, and stopped swallowing pills, and his parents preferred resumption of lorazepam. Clozapine was initiated and increased to a maximum of 200 mg/d, reaching a level of 290 ng/mL, while tapering quetiapine, with mild improvement in interactivity and speech. He experienced common but significant clozapine side effects of weight gain (weight increased from 76.8 to 97.5th percentile over the course of hospitalization) and tachycardia.

After 4 months of ongoing severe symptoms despite intensive assessment and treatment, including maximal pharmacotherapy, he was referred for ECT. Per Massachusetts regulations, ECT for patients under age 16 requires the approval of the Department of Mental Health. Given the patient's young age, they required in-person assessment by their psychiatrists and multiple discussions with the treatment team and parents, with regulatory approval taking approximately 1 month to obtain.

Because of the patient's BMD, ECT was performed using medications that do not provoke malignant hyperthermia, as patients with BMD are prone to anesthesia-induced rhabdomyolysis. This included methohexital induction and nondepolarizing neuromuscular blockade with rocuronium, guided by quantitative neuromuscular monitoring and followed by full reversal with sugammadex. ECT was administered with bitemporal electrode placement due to the severity of his condition and long history of bitemporal ECT for catatonia, on a twice-weekly schedule.

He had rapid and profound improvement in his symptoms with ECT and was able to be discharged from the inpatient hospital following 4 treatments over the course of 2 weeks. He received a total of 10 acute course treatments on a twice-weekly schedule, 8 weekly continuation treatments, and one final treatment 2 weeks later for a total of 15 weeks of ECT, while tapering lorazepam and clozapine. He had no significant medical or psychiatric side effects from treatment. After 15 weeks, he was judged to have returned to baseline functioning by his parents and school staff, scoring 0 on catatonia rating scales.

DISCUSSION

This case highlights the importance of identifying catatonia and the role of ECT when pediatric catatonia is not responsive to benzodiazepines, even in very young children.⁸

TABLE 1. Labwork and Imaging Completed During Hospitalization		
System	Laboratory Testing	Imaging/Procedure
Cardiology	Troponin T Gen5 (25 ng/L reference range: ≤ 14), CK-MB, B-Type Natriuretic Peptide, troponin I, calcium (min 7.2 mg/dL reference range: 8–10.5)	Cardiac echocardiogram
Endocrinology	Free T ₄ , antithyroid antibody, T ₃ , inhibin B, hemoglobin A1c, TSH (max 5.99 mcunit/mL, reference range 0.7–5.7), glucose	
Gastroenterology	GGT (min 4 U/L, reference range: 12–55), ammonia, copper, ceruloplasmin, vitamin A , vitamin B1, vitamin B12 (1112 pg/mL reference range: 211–911), folate (29.46 ng/mL (reference range: 4.2–20 ng/mL), vitamin E, 25-hydroxyvitamin D (19.9 ng/mL, reference range: 30–80), zinc (45 mcg/dL, reference range: 60–120), total bilirubin, direct bilirubin, alkaline phosphatase, total protein (min 4.5 g/dL reference range: 5.5–8.2), AST (max 316 U/L reference range: 2–40 U/L), ALT (max 117 U/L reference range: 3–30), albumin, retinol binding protein	Abdominal US
Genetics/metabolism	Cholesterol, triglycerides, HDL cholesterol, LDL cholesterol, carnitine free and total, amino acids x 2 (tyrosine initially high then normal on repeat), aminolevulinic acid, homovanillic acid, vanillylmandelic acid urine: porphyrins, lactic acid, 3-hydroxypropionic acid, 3-hydroxybutyric acid, methylmalonic acid, 4-hydroxybutyric acid, ethylmalonic acid, succinic acid, fumaric acid, propionylglycine, isobutyrylglycine, glutaric acid, 2-methylbutyrylglycine, isovalerylglutamine, adipic acid, 3-methylcrotonylglycine, tiglylglycine, 2-hydroxyglutaric acid, 2-ketoglutaric acid, hexanoylglycine, suberic acid, orotic acid, citric acid, sebamic acid, 4-hydroxyphenyllactic acid, 3-phenylpropionylglycine, suberylglycine, 3-hydroxyisovaleric acid, 2-methyl-3-hydroxybutyric acid, 3-methylglutaconic acid, succinylacetone, 3-hydroxy-3-methylglutaric acid, 2-methylcitric acid, 4-hydroxyphenylpyruvic acid, in-frame DMD deletion of exons 48–51-hemizygous	
Hematology	White blood cell count (max 14.78 K cells/uL, reference range: 4.63–10.37), red blood cell count (min 3.71 M cells/uL, reference range: 4.18–5.02), hemoglobin 10.9 g/dL (reference range 11.3–14.6), hematocrit (min 30.2%, reference range: 34.4–42.9), MCV, MCH (max 30.3 pg, reference range 25.5–29.5), MCHC (36.1 g/dL, reference range 32.2–34.8), RDW (max 14.8%, reference range 11.9–13.7), platelets, MPV (min 7.7 fL, reference range: 9.2–11.3), Von Willebrand ag, iron, ferritin, transferrin, total iron binding capacity	
Infectious disease	CSF Gram stain and cultures, CSF meningitis/encephalitis panel (includes bacterial and viral PCR), B. burgdorferi ab; ESR, CRP, HIV 1 and 2 PCR, Streptolysin O ab, hepatitis B sAg, hepatitis B, sAb, HSV 1 and 2 PCR, COVID, urinalysis	
Immunology	CSF glucose (min 53 mg/dL, reference range: 60–80 mg/dL), CSF protein (min 12.5 mg/dL, reference range 15–45), CSF WBC, CSF RBC, CSF and serum oligoclonal bands, neopterin, beta-2-macroglobulin, serum and CSF autoimmune encephalitis panel, Total IgG, IgG CSF, IgM, IgG, IgA, serum GAD65 (titer 0.11 nmol/L, reference range: ≤ 0.02 nmol/L)	Lumbar puncture
Musculoskeletal	CK (max 11 234 U/L, reference range: 4–175), aldolase (max 89.7 U/L, reference range: 2.7–8.8 U/L), lactate dehydrogenase (max 612 U/L, reference range: 165–430 U/L)	
Neurology		Brain MRI x 2, EEG
Oncology	hCG, AFP	Scrotal US, chest radiograph: mild peribronchial cuffing
Rheumatology	ANA, RF, SCL-70, SSA-52 and 60 IgG, Smith/RNP Ab, IgG, Jo-1 Ab, IgG, PL-12 ab, PL-7 ab, EJ ab, OJ ab, SRP ab, Ku ab, PM/ScI 1-ab, fibrillarin Ab, Mi-2 ab, P155/140 ab, TIF-1 gamma ab, SAE1 ab, MDA5 ab, NXP2 ab, Ha ab, Ks ab, Zo ab, HMGR ab screen	
General	Sodium, potassium (max 5.73 mmol/L reference range: 3.2–4.5), chloride (max 115 mmol/L reference range: 99–111), CO₂ (min 16 mmol/L reference range: 22–30), BUN (min 2 mg/dL reference range: 5–18), creatinine, phosphorus, magnesium (max 2.4 reference range: 1.5–2.2 mg/dL), lactate, cystatin C, lead	
Abbreviations: AFP, alpha-fetoprotein; ALT, alanine aminotransferase; ANA, antinuclear antibody; AST, aspartate aminotransferase; BUN, serum urea nitrogen; CK, creatine kinase; CK-MB, creatine kinase muscle and brain hybrid; CO ₂ , carbon dioxide; CRP, C-reactive protein; CSF, cerebral spinal fluid; DMD, Duchenne muscular dystrophy; EEG, electroencephalogram; ESR, erythrocyte sedimentation rate; EJ ab, anti-glycyl-tRNA synthetase; GAD65, glutamic acid decarboxylase 65-kDa isoform; GGT, gamma-glutamyltransferase; hCG, human chorionic gonadotropin; HDL, high-density lipoprotein; HMGR, HMG-CoA reductase; HSV, herpes simplex virus; IgA, immunoglobulin A; IgG, immunoglobulin G; IgM, immunoglobulin M; Jo-1 ab, anti-histidyl-tRNA synthetase; Ks, asparaginyl-tRNA synthetase; LDL, low-density lipoprotein; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; MPV, mean platelet volume; MRI, magnetic resonance imaging; NXP2, nuclear matrix protein 2; OJ ab, anti-isoleucyl-tRNA synthetase; PCR, polymerase chain reaction; PL-12 ab, anti-alanyl-tRNA synthetase; PL-7 ab, anti-threonyl-tRNA synthetase; RBC, red blood cell; RDW, red cell distribution width; RF, rheumatoid factor; RNP Ab, ribonucleoprotein antibody; SAE1, small ubiquitin-like modifier activating enzyme 1; SCL-70 ab, anti-topoisomerase I antibody; sAb, surface antibody; sAg, surface antigen; SRP, anti-signal recognition particle; SSA, anti-Ro; SSB, anti-La; T ₃ , triiodothyronine; T ₄ , thyroxine; Troponin T Gen5, 5th generation high sensitivity troponin T; TSH, thyrotropin; US, ultrasonography; WBC, white blood cell; Zo, anti-synthetase. Abnormal values highlighted in bold.		

Symptoms of catatonia are frequently underrecognized in younger children, often because clinicians might not be familiar with the condition or consider it on the differential.¹³ Particularly in younger children, and given the overlap of symptoms with other conditions such as delirium, it requires a high index of suspicion.¹⁴ Awareness of the variety of potential presentations, including hyperkinetic/excited catatonia, and Diagnostic and Statistical Manual criteria¹⁵ increases the likelihood that the diagnosis can be made. Diagnosis is facilitated by evidence-based rating scales, particularly the Pediatric Catatonia Rating Scale,⁹ validated for pediatric patients, although the Bush Francis Catatonia Rating Scale¹⁶ is widely used for individuals of all ages. The KANNER scale,¹⁷ more specific for individuals with neurodevelopmental disorders, was also used by clinicians to monitor symptom improvement. Catatonia rating scales help clinicians monitor response to treatment, as noted in Figure 1, and are a mainstay of a psychiatrist's daily assessment.

Catatonia may be caused by a variety of conditions. While catatonia is often associated with psychiatric diagnoses, including schizophrenia or bipolar disorder, such disorders are exceedingly rare in young children,^{18,19} and a broad search including contributing medical etiologies is essential.^{20,21} This includes a variety of genetic conditions.^{22,23} This patient's work-up was notable for the identification of BMD, a condition that carries increased risk of neuropsychiatric conditions such as ADHD and autism.^{24–27} While the pathophysiology of catatonia is not entirely understood, an imbalance of excitatory and inhibitory gamma-aminobutyric acid (GABA)ergic neurons is thought to play a role.²⁸ As dystrophin isomers have a role in clustering GABA receptors in the prefrontal cortex, there could be a link between dystrophin dysfunction that occurs in BMD/DMD and catatonia.²⁹ His diagnosis of BMD likely would not have been made at his age while asymptomatic if not for the laboratory and genetic testing sought to further understand the potential etiology of catatonia. It is not clear if this diagnosis impacted the development of catatonia in his case. Per the extant literature, individuals identified with BMD and catatonia typically were older than our patient and also had an associated psychotic disorder.²⁶

Treatment of catatonia often involves high-dose lorazepam. In this case, there was not an adequate response to escalating lorazepam doses, thus other agents were added as augmentation that have evidence for treatment of catatonia, including: zolpidem,³⁰ memantine,^{19,31,32} and quetiapine^{7,33} without adequate benefit. Generally, antipsychotic medications are avoided in catatonia because of the risk of neuroleptic malignant syndrome and thus must be used with caution. Clozapine has demonstrated efficacy for the treatment of catatonia in case series,³⁴ although it carries the possibility of significant side effects, including weight gain and tachycardia, as experienced by our patient and

others, including myocarditis and agranulocytosis, and thus requires monitoring.³⁵ Clozapine has a unique binding profile, including affinity for serotonergic, α -adrenergic, cholinergic, and histaminergic receptors, although it also serves as a modulator at the GABA_B receptor, which might account for its effectiveness in treating catatonia. However, while attempts at medication optimization occurred over several months, improvement with ECT was noted within 2 weeks. In consideration of the risks of delay in definitive treatment to this child and family while he remained significantly debilitated and catatonic, risks of medication side effects, and the benefit of ECT, we posit that ECT should be considered earlier in seemingly refractory catatonia. Furthermore, improved access is necessary for individuals to benefit from this treatment.

ECT has demonstrated safety and efficacy for catatonia in pediatric patients, including young children and children who are medically complex.^{13,36–38} However, there are significant barriers for youth to receive this potentially lifesaving treatment in many states because of ongoing stigma surrounding this treatment.^{39–41} This led to our attempt at optimizing medication management despite the potential for side effects and an extended multimonth hospital stay. Ultimately, the addition of ECT led to catatonia remission, and lorazepam and clozapine tapering. The importance of timely access to ECT deserves particular emphasis. Access for this patient was made possible by the recent integration of ECT within our hospital system. This case highlights the need for streamlined procedures that ensure safety and appropriate referral for ECT, and also lower barriers to access to this vital treatment nationwide.

Written permission was obtained from the patient's guardians to publish this case.

ABBREVIATIONS

ADHD: attention deficit/hyperactivity disorder
BMD: Becker muscular dystrophy
DMD: Duchenne muscular dystrophy
ECT: electroconvulsive therapy
IV: intravenous
KANNER: Katatonia Autism Neuropsychiatric and Neuromovement Examination Rating

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