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Have you Considered Catatonia? Cases of Catatonia in Individuals with Autism Spectrum Disorder

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Introduction

Catatonia is a neuropsychiatric syndrome that affects movement, affect, behaviors, and at times, the autonomic nervous system [1]. Catatonia is most often associated with psychiatric illnesses such as affective or psychotic disorders, but can also occur in neurodevelopmental disorders, various medical conditions such as infections, metabolic disturbances, autoimmune encephalitis, focal neurological lesions, neurodegenerative disorders, and endocrine disorders, and has several other reported etiologies such as administration or withdrawal of certain medications [1,2]. This case report highlights the challenges of diagnosing catatonia in pediatric patients with autism spectrum disorder (ASD) and briefly describes the impacts of delayed treatment on prognosis. In 2013, catatonia was added as a specifier for ASD. However, many experts suspect that catatonia is still underdiagnosed in patients with ASD, especially in those with a co-occurring intellectual disability or language impairment [2,3]. For patients with ASD who are diagnosed with catatonia, the time of diagnosis from symptom onset is often much longer than for neurotypical patients [3]. There are a few possible explanations for these discrepancies. First, many of the core features of ASD overlap with the core features of catatonia, such as stereotypies, mannerisms, mutism, poor eye contact, echolalia, verbigeration, perseveration, negativism, and other motor abnormalities [2-6]. Additionally, many patients with ASD who develop catatonia present with unique symptoms that are not listed in current diagnostic guidelines or validated catatonia rating scales [3-8,10]. Examples of such symptoms include late developmental regression and severe or treatment-refractory self-injurious behaviors, both of which have been suggested as signs of catatonia in patients with ASD [3-8,10]. Furthermore, there are no validated catatonia rating scales for patients with ASD that account for all these unique symptoms. The Kanner Scale is a validated catatonia rating scale for patients with neurodevelopmental disorders, but it does not include regression or self-injurious behaviors [11]. The most commonly used validated catatonia rating scales, the Bush-Francis Catatonia Rating Scale and the Pediatric Catatonia Rating Scale, are based on neurotypical patients and often misrepresent catatonia in ASD. The diagnostic guidelines for catatonia in the DSM-5-TR are also based on neurotypical patients and are not specific to pediatric catatonia. All of these factors likely contribute to the under recognition of catatonia in patients with ASD, which frequently delays diagnosis and treatment [3]. Here, we discuss a unique case of an 11-year-old male with level two ASD, mild intellectual disability, epilepsy, and cerebral palsy who was admitted to our neuropsychiatric specialty care partial hospitalization program due to worsening seizures and a significant functional decline over six months.

Case 1

Mr. E is an 11-year-old male born prematurely with past medical history of neonatal intraventricular hemorrhage, cerebral palsy, congenital diplegia, epilepsy, and obstructive sleep apnea, and past psychiatric history of autism spectrum disorder (ASD). He presented to Neuropsychiatric Special Care for significant regression across multiple functional domains – speech, comprehension, ability to meaningfully interact with his environment, and self-care over the course of 6 months. Additionally, he had worsening of his seizures, which were previously well-controlled for seven years. Medical work-up included CSF studies, which demonstrated elevated GAD65 antibody at 0.04 nmol/L (ref: <0.02 nmol/L). Brain MRI at age 3 showed remote periventricular leukomalacia with corresponding mild ex vacuo enlargement of the lateral ventricles, left more than right, and small amount of hemosiderin staining in the lateral ventricles from prior intraventricular hemorrhage noted after birth. Hippocampi were asymmetric, with the left smaller than the right. Repeat MRI at age 11 was unchanged. Additional work-

up including brain imaging, EEGs, CMP, CMC, and thyroid panel, were unremarkable and did not show an organic cause of his regression. The patient exhibited subtle signs of catatonia, such as stereotypy, verbigeration, and hypoactivity, though a diagnosis of catatonia was clinically indeterminant.

Given the patient's severity of regression without organic cause, a lorazepam challenge (0.1 mg/kg/dose) to rule out catatonia was performed. His initial Bush Francis Catatonia Rating Scale before the lorazepam trial between three raters was 9, 10, and 12, though he scored positive in areas that overlap with ASD symptoms. The patient had minimal response to the initial lorazepam trial, though showed subtle improvement in motor domains without evidence of sedation. He was continued on Lorazepam 2 mg three times daily. His BFCRS four days after initiating treatment continued to average between 9-12, despite anecdotal improvement in interactiveness and regression per staff and caregiver reports. As such, the BFCRS was not a particularly helpful measure for objective data to monitor behavioral changes. Given some subjective improvement without evidence of sedation, his dose was increased to 3 mg lorazepam three times daily and 100 mg amantadine twice daily. The patient was discharged on this medication regimen. After 17 days of treatment on this course, the patient demonstrated significant improvement, nearing his baseline functioning prior to the regression. He was sitting independently, vocalizing full, purposeful sentences such as "I want chocolate cake for my birthday" per parental report. A 4 month follow up indicated that Mr. E remained at baseline on Lorazepam 4 mg TID, Amantadine 100 mg BID and Depakote 125 mg BID. He was able to re-

enter school and is described as having an improved quality of life with the return of speech and an ability to perform his activities of daily living as he once was.

Case 2

Mr. R is a 17-year-old male born full-term with past medical history of dental caries and past psychiatric history of possible autism spectrum disorder (ASD), moderate intellectual disability, attention-deficit/hyperactivity disorder with predominantly hyperactive-impulsive presentation, mixed receptive-expressive language disorder, and semantic-pragmatic disorder. At age eight, he was evaluated by Developmental Pediatrics and determined not to have ASD, and then at age 17, was diagnosed with ASD and moderate intellectual disability through neuropsychological testing in the community. Mr. R had been experiencing hallucinations and delusions for one year before he presented to the hospital for workup of six months of cognitive decline, staring, maintaining odd postures, and escalating aggression. His medical workup included a brain MRI, lumbar puncture, autoimmune encephalitis panel, lactate, ammonia, B12, ferritin, thiamine, copper, TSH, genome analysis, C3, C4, and ANA panel. C4 was low and ANA was positive with borderline elevated titers and negative for specific antibodies, requiring no further rheumatologic testing in the absence of signs of systemic inflammation.

The remainder of his workup was also reassuring against organic causes for his cognitive and behavioral changes. Prior to the initial psychiatric consultation, the patient received olanzapine 5mg for agitation. On initial evaluation, he was found to be catatonic with an initial Bush-Francis Catatonia Rating Scale (BFCRS) score of 19 for stupor/immobility, staring, mutism, catalepsy, mitgehen, ambitendancy, echopraxia, and echolalia; of note, patient's medical record indicated a history of echolalia as a young child. After a one-day trial of lorazepam 1mg three times daily, the patient responded with temporary resolution of catalepsy, mitgehen, and echolalia, increased engagement and talkativeness, and a BFCRS score of 8. Periods of aggression during subsequent evaluations prevented full use of the BFCRS as a measure of his symptoms, though based on concern for ongoing catatonia symptoms including catalepsy and agitation, lorazepam was scheduled and titrated to a maximum of 3mg four times daily. Of note, multiple psychiatric providers who assessed Mr. R during his ten-day medical hospitalization felt that he likely met criteria for ASD. On his last day of medical hospitalization, his BFCRS score was 9 with intermittent excitement, verbigeration, impulsivity, perseveration, and combativeness; he also endorsed auditory hallucinations and exhibited a disorganized thought process concerning for an underlying psychotic disorder. He was transitioned to Neuropsychiatric Special Care for further stabilization. On his initial exam, he demonstrated significant thought blocking, thought disorganization, and speech latency, and while he did not allow for administration of BFCRS, he was not posturing or rigid. The patient's catatonia was felt to be improved enough to start risperidone to address his psychosis.

Risperidone was titrated up to 2mg two times daily before he received two loading doses of Invega sustenna long-acting injectable. During this time, the patient did not demonstrate signs of worsening catatonia or development of neuroleptic malignant syndrome. After 16 days on NSC, one provider assessed the patient to have a BFCRS score of 9 based on immobility/stupor, staring, echopraxia/echolalia, rigidity, gegenhalten, and autonomic abnormality. Despite this, family members and behavioral health staff reported clinical improvement with increased communication and engagement, clearer speech, and organized, goal-directed thought process; occasional psychomotor slowing and difficulty understanding and expressing himself were felt to be consistent with his baseline per parent report. He was discharged on a 2-week bridge of oral risperidone 2 mg twice daily and lorazepam 4 mg three times daily, with a plan to receive maintenance doses of Invega sustenna and taper lorazepam outpatient.

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Discussion

Catatonia is a serious neuropsychiatric condition that is complicated to diagnose and treat. This challenge is especially amplified in special populations, like those with autism spectrum disorder [ASD]. The case of Mr. E illustrates these diagnostic hurdles and highlights the urgent need for tools to assess catatonia in this population and serves as a model for effective evaluation and treatment. Catatonia is associated with significant morbidity and mortality [12]. Associated medical complications include malnutrition, stasis ulcers, rhabdomyolysis and venous thromboembolisms. Additionally, catatonia can also progress to malignant catatonia, where mortality rates range from 9-20% [13]. Diagnosis of catatonia in the general population has remained a challenge, with an average delay in treatment of 15 days [14]. Early and aggressive intervention is associated with improved outcomes, making prompt diagnosis and treatment essential [15].

Increased rates of catatonia have been seen in those with autism spectrum disorder and other developmental disorders [12]. Recent studies have focused on identifying the incidence of catatonia, specifically in the pediatric population [16]. However, due to inconsistent reports, the exact prevalence in those with ASD remains unclear [16]. The overlapping clinical features that exist in both catatonia and ASD pose a challenge for diagnosis and treatment. These overlapping symptoms include agitation, echolalia, echopraxia, grimacing, stereotypy, posturing, mutism, mannerisms, and negativism [7,8]. Furthermore, the higher rates of medical comorbidity in individuals with ASD add an additional layer of complexity, contributing to a wide variability in observed symptoms.

Currently, the primary method for diagnosing catatonia in this population includes monitoring changes from baseline and focusing on clinical features that are unique to catatonia [17]. Instruments such as the Bush Francis Catatonia Rating Scale (BFCRS), the Pediatric Catatonia Rating Scale (PCRS), the Kanner Scale, and DSM-V-TR criteria are utilized, yet none have been specifically validated for individuals with ASD [4,17]. Of note, only the Pediatric Catatonia Rating Scale is validated for the monitoring of catatonia in pediatric populations [11]. The BFCRS failed to adequately capture the symptomatic improvements achieved through Mr. E's treatment. Our team relied on subjective reports of Mr. E's changes in behavior that coincided with medication changes and the data collected by our Applied Behavior Analysis (ABA) team. While no significant change on the BFCRS was appreciated, our other measures demonstrated vast improvements in the patient's symptoms with a return to his mental status baseline. This underscores the need for better diagnostic and monitoring tools tailored for the ASD and IDD populations.

From a pharmacological standpoint, we treated Mr. E's catatonic symptoms with lorazepam and amantadine, common interventions for managing catatonia. What sets this case apart are the psychological interventions employed by our behavioral team, which focused on activities of daily living such as brushing teeth and combing hair. While these interventions did not address the underlying pathology, they effectively targeted deficits resulting from catatonia, ultimately enhancing the patient's functioning and quality of life. This case illustrates how psychotherapeutic techniques that encourage active engagement and continued demands can be a vital component of a positive clinical course. Delays in accessing optimal treatment for catatonia are common, particularly when electroconvulsive therapy is warranted. However, initiating psychotherapeutic interventions early in the clinical course could significantly improve prognosis, even in the face of treatment delays. Based on the graph, after assessing Mr. E's baseline of these daily living activities using a task analysis for brushing hair and brushing teeth, treatment began with least-to-most (LTM) prompting provided by staff. For each step of brushing his teeth, if Mr. E did not complete a step independently, after a wait time a LTM prompting hierarchy was used, a vocal prompt by staff, gestural model, partial physical, and then full physical as needed. The next phase of treatment incorporated visual supports concurrent with LTM prompting. Following data analysis, based on patient need, most-to-least prompting was incorporated (immediate use of physical prompting, then fading to gestural prompts, then vocal prompts). Finally, treatment concluded using LTM prompting with visual supports. As these behavioral treatments were implemented, medication changes occurred as well.

Overall, catatonia is a complex neuropsychiatric condition that can be associated with significant morbidity and mortality. Understanding this diagnosis in individuals with intellectual and developmental disabilities remains challenging, but is nonetheless crucial. Our case highlights the necessity for specialized diagnostic tools and emphasizes the value of a psychotherapeutic approach in the treatment of catatonia.

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