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Monoamine Oxidase Deficiency Symptom Management with Memantine: A Case Report

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Abstract

A patient with developmental delays, agitation, disordered sleep, cataplexy and anxiety was recently diagnosed with monoamine oxidase deficiency using whole exome sequencing. The overactive monoamine system explained the symptom complex and suggested a pharmacological approach to reduce glutamate activity while avoiding monoamine stimulation. Memantine has helped this young man better benefit from active treatment in a community setting. Further exploration of the genetics and management of monoamine oxidase abnormalities is encouraged.

Keywords

Monoamine Oxidase, Brunner's Syndrome, Memantine, Glutamate, Whole Exome Sequencing

1. Introduction

Rare genetic disorders present challenges for the patient, their family, and their caregivers. These disorders also offer opportunities to better understand mechanisms and treatment for severe emotional distress. We present a patient with Monoamine Oxidase (MAO) deficiencies and associated severe agitation whose daily life improved with memantine.

Genetic variations in MAO isoenzymes play a role in a wide range of neuropsychiatric disorders. Brunner's syndrome, also known as Monoamine Oxidase A (MAOA) deficiency, is a rare X-linked recessive developmental disorder characterized by cognitive and behavioral disabilities. This condition is caused by mutations in the MAOA gene, located on chromosome Xp11, which encodes the enzyme that is responsible for metabolizing neurotransmitters such as serotonin and norepinephrine. MAOB is fundamental to dopamine metabolism [1]-[3].

Individuals with genetic or acquired MAO deficiencies typically exhibit impul-

sive agitation, mild intellectual disabilities, insomnia, affective lability, emotional rigidity, lack of exploratory behavior, episodes of skin flushing, sweating, headaches, gastrointestinal problems, and stereotypies. There are often many symptoms of Autism Spectrum Disorder (ASD). MAOA deficiency is usually identified in childhood when the symptoms come to professional attention and genetic testing is completed [4].

2. Case Report

2.1. Patient's History

M is a 23-year-old male with MAOA deficiency. At the time of the initial history and examination, he lived in a community-based intermediate care facility for patients with developmental disabilities. The pregnancy was described as "difficult". Developmental delays were identified by 4 months. Occupational and physical therapies began at 15 months. He has had sensory processing problems, hypersensitivity to noise, anxiety, cataplexy, and severe insomnia most of his life. Central sleep apnea was diagnosed at 3 years of age but later resolved. He is diagnosed with a rapid-eye-movement sleep disorder. Evaluation for narcolepsy was negative, but cataplexy has been challenging. Heat intolerance and excessive sweating became more apparent as he got older. Agitation and its management became more difficult in the teenage years. Strengths include his ability to enjoy sports, music, reading, puzzles, and time with others. He has never engaged in imaginative play.

Early genetic testing at ages 4 and 10 was unremarkable but testing as a 20-year-old, using whole exome sequencing (Next-Generation sequencing (XomeDxPlus), described an individual with a pathogenic copy number variant within cytogenetic band Xp11.3. The patient's mother tested negative for the abnormality, suggesting the variant arose de novo in this patient [5]-[7].

The patient was admitted to the group home in June 2023. Behavioral support was developed and implemented along with a schedule of regular activities. He was on a diet commonly used for patients on MAO inhibitors, avoiding tyramine-containing foods. Caution was used to avoid medications like dextromethorphan, which might interact with MOA inhibitors. Unfortunately, there were many explosive tantrums with aggression, self-injury and "meltdowns" in the group home and on home visits. Small disappointments were associated with severe agitation. He refused van transportation and was unable to go into buildings like the day program or restaurants. Food preferences were very limited.

2.2. Medication Trial

Preadmission trials of FDA-approved medications for ASD agitation, risperidone and aripiprazole, did not help. Medications at the time of this initial evaluation were escitalopram oxalate 5 mg/5 ml, 1 ml qhs, Colace 100 mg per day, and vitamin D3 1000 units per day. M's behavior early in the group home stay was marked by severe agitation and cataplexy. He was on a diet commonly used for patients on MOA

inhibitors, avoiding tyramine-containing foods.

The treatment team, including his mother, discussed several medication ideas such as memantine and donepezil. Orexin/hypocretin antagonists seemed unwise in the context of cataplexy. Crisis medications or pre-treatment for medical/dental visits with hydroxyzine were also considered. With guardian permission, memantine was begun at 2.5 mg per day and raised to 5 mg per day two weeks later. Soon after the medication started, caregivers reported, "he is a lot calmer". Activities of daily life showed "a huge improvement" according to his direct care staff. He was much more tolerant of basic hygiene requests and began showering. He began trying different foods, traveled by van, attended the day program and went to restaurants. His mother noted no "meltdowns" from initiation of memantine to one episode in May 2025. No formal data was collected on cataplexy, but the global impression was fewer events. All relationships improved, with better connections to peers and staff plus more successful home visits. This improvement has continued for over 20 months. Clonidine 0.05 mg qhs was added on January 24 with mild improvement in sleep. Vital signs, done weekly, were normal. Admission height was 66 inches, and weight was 177 lbs. with BMI of 28.7 (pre-obesity). October 23 BMI 24, and January 24, weight was 149 lbs. with BMI of 24 (normal). Laboratory work on admission, however, June 24 blood glucose was 103 but the HbA1c was 4.7. Jan 2025 identified low blood glucose (62) and HbA1c (4.7).

3. Discussion

Caring for patients with developmental disabilities requires a team approach with structure, social support, skill building, and often medication management. Often, several factors need to change for a patient to feel better.

The monoamine system is present in bacteria, yeasts, fungi, parasites, invertebrates and vertebrates. Given oxidative stress produced by these enzymes, this system must have strong adaptive value [8]-[11].

Monoamine activity and metabolism are important processes in neurology and psychopharmacology. Iproniazid, first used as an anti-tubercular drug and one of the earliest antidepressants, is a monoamine oxidase inhibitor. The treatment of Parkinson's disease includes the use of selective MAOB inhibitors such as selegiline, rasagiline and safinamide [12]-[16].

This system is underactive in Brunner's syndrome and other polymorphisms of the Monoamine A genes. Underactivity of the MAOA system may also be acquired by prescription of monoamine oxidase inhibitors and other environmental events [17] [18].

Neurotransmitter-based diagnosis uses physical and emotional symptoms to guide interventions. There is not yet a direct way to measure brain neurotransmitter activity. In this case, the genetic abnormality is known, and central monoamine elevations are predicted. MAOA Deficiency causes elevated levels of serotonin, norepinephrine, and dopamine in the brain, increased urinary levels of metanephrine and normetanephrine [19]. It would be nice to have urinary metabolites pre- and

post-medication trial, but these tests were not done.

There is a small literature on the use of SSRIs in patients with MAOA deficiency. Observations from animal models and an earlier case report led to cautious recommendation of treatment with an SSRI. It is puzzling that a reuptake inhibitor may down-regulate serotonin effects. There is some evidence that inhibition of platelet serotonin transporters may lower blood serotonin levels in individuals treated with SSRIs. There may be different neurotransmitter effects at varying doses of SSRIs [20]-[25].

Emotional stability depends on a functional window of neurotransmitter activity working in balance with other neurochemicals within healthy anatomical structures. A psychopharmacologist may adjust activators or inhibitors of a certain process to get healthier emotional responses. In this case, extreme anxiety, explosive outburst, cataplexy, and insomnia caused by excess monoamines may have been settled by a reduction in glutamate activity. Genetics may become a guide to personalized psychopharmacology [26]-[28].

Memantine helps slow the progression of Alzheimer's dementia. It is also prescribed, with less rigorous research, in severe vascular dementia, PTSD, GAD, pathological gambling, OCD, treatment-resistant schizophrenia, ADHD, adjunctive treatment of borderline personality disorder, skin picking-excoriation behavior, traumatic brain injury, and other neuropsychiatric challenges. Memantine is believed to interact with a variety of ligand-gated ion channels to block current flow through channels of N-methyl-d-aspartate (NMDA) receptors. It is one of the few psychotropic drugs with anti-glutamate activity. Glutamate excitotoxicity appears fundamental to neuroprogression, impaired neural energy supply, and kindling [29]-[44].

One challenge in theoretical pharmacology is understanding neurotransmitter interactions in health and disease states. There is likely feedback, feedforward, and other interactions as brain systems adapt to changes. Also, memantine not only mitigates glutamate activity, but also has effects on other neuro-messengers.

In this case, the addition of memantine seemed safer than raising the SSRI. The relatively better side-effect profile suggests memantine might be used in younger children with Brunner's syndrome and certain cases of Norrie Disease, when deletions extend to include the MAOA and MAOB genes. There is some hope that calming during the early developmental stages would permit activation of prosocial behaviors and subsequent improved relationships. Memantine is generally well-tolerated in children and may have potential benefits for other pediatric neurodevelopmental disorders, particularly those with MAOA gene polymorphisms [45] [46].

Patients with cataplexy experience muscle weakness or atonia with intense emotions. Cataplexy is linked to narcolepsy and abnormalities of the hypocretin/orexin system of the hypothalamus. Noradrenergic, cholinergic, and GABAergic processes are involved in REM sleep. Reducing emotional reactivity likely explains reduced cataplexy. However, there may be a direct effect of glutamate reduction on central

atonia [47]-[54].

4. Conclusion

In this case report, a patient with Monoamine Oxidase deficiency and severe agitation had a rapid and sustained improvement when treated with memantine. Causal attribution in psychiatry is complex and often uncertain. Confounding factors include addition of low-dose clonidine, normalization of body mass index, and the development of mild hypoglycemia. The improvement noted here may also be related to growing comfort in a supportive environment, placebo response, caregiver-response bias, and other factors. A single case does not determine safety or efficacy. Objective measurements of brain function related to psychiatric change have not been developed, so the glutamate mediation hypothesis remains speculative. While stopping memantine might be scientifically interesting, this step is not in the patient's best interest right now. A series of case reports and then controlled trials are needed to gain scientific confidence about this treatment. Medication intervention for younger children with MAOA and B polymorphisms is worth exploring. The genetics of mitochondrial processes and monoamine metabolism is an area of active study. Whole exome testing is better than chromosomal microarray for children with complex developmental disabilities. The newest testing was important for this patient and his family. The research and clinical care of patients with genetic disorders offer unique opportunities to learn neurochemical and psychopharmacological processes.

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Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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