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Stimulant-Induced Chorea

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Introduction

Adderall, a Dextroamphetamine/Levoamphetamine salt, a synthetic compound similar to methamphetamines, is a widely used stimulant type medication commonly used to treat Attention Deficit Hyperactivity Disorder and Attention Deficit Disorder in children, adolescents, and adults. It works through a mechanism of increasing the release of both norepinephrine and dopamine in the central nervous system, thereby enhancing cognitive effects of increased wakefulness, improved cognitions of focus and concentration, and decreasing hyperactivity, impulsivity and inattentiveness. But at higher doses or through methods of abuse Adderall can cause signs of impaired cognitive function, signs of psychosis, and produce movement disorders, such as chorea.

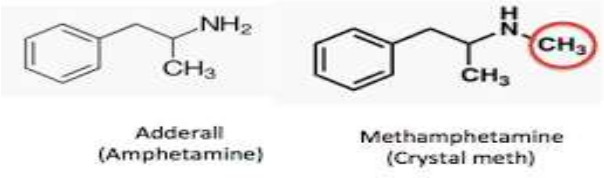

Figure 1: Chemical Structures of Amphetamine and Methamphetamine

Chorea is characterized by abnormal involuntary movements that can consist of short, irregular, asymmetrical, sudden, unexpected, non-stereotypy movements.

Figure 2: Image of Chorea

Chorea can have various causes including:

- Hereditary diseases (Huntington's, Wilsons, Lesch Nyhan, Friedreich's Ataxia),
- Post-infectious syndromes (Sydenham's chorea),
- Medical (thyrotoxicosis, HIV, cerebrovascular),
- Immune-mediated (SLE, Celiac),
- Endocrine and metabolic (pregnancy, polycythemia vera),
- Drug-induced (levodopa, neuroleptics, anti-convulsants, oral contraceptive pills),
- Misuse of psychostimulants (cocaine, methamphetamine, amphetamine)

Case Report

A 39 year old female with a past psychiatric history of mood disorder, anxiety, and attention deficit disorder was initially admitted to the hospital ICU unresponsive status post-cardiac arrest of unknown origin. While in the ICU and intubated, the patient had an additional episode of ventricular tachycardia and subsequent cardiac arrest, necessitating defibrillation and shock.

Days after admission:

- Day 1: Cardiology consultation for evaluation of causation of cardiac arrest. Likely etiology peripartum cardiomyopathy. The patient, G15P10, recently delivered her 10th child less than 1 month prior to admission.
- Day 3: Neurology consultation for concerns of unresponsiveness. Patient starting to regain consciousness, still intubated, but requiring chemical and physical restraints due to agitation. Assessment of encephalopathy and recommended MRI brain.
- Day 5: Psychiatry consultation for assessment and management of mood disorder. On MSE, patient presented pleasant, cooperative, no use of restraints noted, and did not show any signs of mood alteration, depression, or anxiety. Most prominent feature on MSE was cognitive impairment with difficulties in attention, concentration, registration, and short-term memory. Cause of cognitive impairment speculated as related to anoxia from recent cardia arrest. History of "street drug" use elicited by family; patient denied.
- Day 9: Psychiatry re-consultation for behavior of severe agitation, aggression towards hospital staff, requiring the use of physical restraints after being transferred from the ICU to the medical floor. On MSE, patient was AOx1, very disorganized, hyperverbal, loud, agitated, and displaying prominent choreoathetotic movements of : involuntary jerking surrounding the face, shoulders, and pelvis. Patient necessitated assistance when walking due to the movements and instability. Patient's home medications of Celexa 20 mg daily, Adderall 30 mg twice daily, and Ativan 1 mg daily. Patient denied any history of drug abuse, but did admit to possibly taking more than prescribed after recent birth of her 10th child (22 days prior).

Labs

CMP: AST/ALT (47/17), Alk Phos (148)
CBC: WBC (21.9), H/H (10.5/33.7), Platelets (454), Neutrophils (19.27)
TSH (1.59), Troponin (0.02), Lactate (6.6), Procalcitonin (1.36), UA (-)
UDS: (+) amphetamine, (-) benzodiazepine, (-) EtOH
MAPS: Adderall script filled 4 days prior to hospital admission

Imaging

CT Head: (-)
EKG: sinus rhythm with occasional PVCs and LAD
ECHO: EF 35%, global hypokinesis with LV dilation
24 hour EEG: moderate diffuse encephalopathy, (-) epileptiform activity

Discussion

It is well known that at high doses or through forms of misuse and abuse that stimulants can cause movement disorders, such as chorea. But there is additional research supporting evidence that at lower doses or commonly prescribed doses over an extended long period of time that stimulants can cause changes in cell processes and surface structure in the brain, most notably in the nigrostriatal pathway of the dorsal striatum and substantia nigra, leading to a possible dose-dependent relationship to some of the adverse effects seen with stimulants as is such a possibility in this case.

Upon regaining consciousness, this patient demonstrated movements of: involuntary, abrupt, random, repetitive, non-stereotypied, jerking, which are consistent with choreathetosis. After discussion with the patient and obtaining collateral information with family members and the Michigan MAPS system, it was concluded that the patient's movements were due a combination of prolonged Adderall use through prescription and the recent self-increase in the amount of Adderall. Patient believed the increase would help with fatigue and low energy after the birth of her 10th child. This unprecedented increase of the stimulant lead to the development of acute chorea, which resolved within days after no stimulant use.

Treatment: In this case, treatment included stopping the suspected offending agent, which was Adderall. Providing supportive care as needed, including benzodiazepines for agitation. We recommended avoiding the use of antipsychotic medications altogether due to the potential cardiac complications as the patient was status-post 2 cardiac arrests and diagnosed with a peripartum cardiomyopathy.

Conclusion

This case illustrates the importance of considering prolonged prescribed stimulant use in the context of misuse as a development of chorea.

References

1. Asser A and Taba P (2015). Psychostimulants and movement disorders. *Frontiers in Neurology.* 6(75): 1-12.
2. Diek A, Saunders-Pullman R, and San Luciano M (2012). Substances of abuse and movement disorders: complex interactions and comorbidities. *Current Drug Abuse Reviews.* 5(3): 243-253.
3. Enevoldson TP (2004). Recreational drugs and their neurological consequences. *Journal of Neurology, Neurosurgery, and Psychiatry.* 75(Suppl III): iii9-iii15.
4. Walker BH. (2010). Approach to the patient with chorea. *Movement Disorder Society.*
5. Bhidayasiri R and Truong DD. 2004. Chorea and related disorders. *BMJ Postgraduate Medical Journal.* 80(947)
6. Berman SM, et. al. 2009. Potential Adverse Effects of Amphetamine Treatment on Brain and Behavior: A Review. *Molecular Psychiatry.* 14(2): 123-142.
7. Moszczynska A and Callan SP. 2017. Molecular, Behavioral, and Physiological Consequences of Methamphetamine Neurotoxicity: Implications for Treatment. *The Journal of Pharmacology and Experimental Therapeutics.* 362(3): 474-488.