Methamphetamine-Induced Catatonia

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Catatonia is a complex syndrome with affective, behavioral, attentional, motor, and vocal abnormalities. It can accompany various psychiatric disorders and occur in association with medical illness and pharmacologic agents. It may also be precipitated by various substances of abuse, including cannabis, synthetic cannabis, phencyclidine, methoxetamine, and stimulants (cocaine, MDMA [3,4-Methylenedioxymethamphetamine], methylphenidate, and synthetic cathinones).[1] Methamphetamines are a newer substance of abuse in recent years and have become increasingly prevalent in drug markets. In the United States, 1.4 million individuals reported abuse within the past year, with 667,000 reporting current use in 2016.[2] Although often compared to cocaine, methamphetamines can be even more addictive and potent due to dopamine release from synaptic vesicles, in addition to blocking dopamine reuptake. Furthermore, 50% of cocaine may be removed within an hour, whereas it may take 12 hours for such clearance with methamphetamines, thus leading to a prolonged duration of action.[3] There are currently no reports of methamphetamine-induced catatonia. Here, we present the case of a young man who developed an episode of acute catatonia due to methamphetamine abuse.

Case Report

Mr A was a 21-year-old man with no prior psychiatric or medical history who was admitted to the hospital due to being found by emergency services in a "frozen" state. He was unable to verbally share any significant history. His mother provided collateral information, revealing that the patient had been abusing methamphetamines recently. On examination, he was found to be disheveled and with a flat affect. He exhibited prominent immobility, mutism, abnormal staring, upper extremity paratonia, and withdrawal. His Bush-Francis Catatonia Rating Scale (BFCRS)[4] score was 24. His urine drug screen (via straight catheter) was positive for amphetamines. His laboratory results were otherwise unremarkable for any infectious, metabolic, or hematologic derangements. Computed tomography and magnetic resonance imaging of his brain revealed no abnormalities. A bedside provocative electroencephalogram revealed only generalized, symmetric background slowing (mainly theta frequency). A lorazepam challenge of 2 mg was administered intravenously, with less withdrawal and staring and increased rates of response to questions observed within 60 minutes. Lorazepam intravenous 2 mg every 8 hours was then scheduled. The following day, he was found to still exhibit significant hypoactive features, though with moderate improvement. His BFCRS score was 18. Thus, the frequency of his lorazepam administration was increased to every 6 hours. On the third day, he was alert and fully oriented, with mainly residual features of occasional staring, posturing, and grimacing. His BFCRS score was 5. He shared that he had been injecting and smoking methamphetamines regularly over the past month. He was unable to quantify the amount that he injected just prior to admission; however, he recalled that it was more than usual. Lorazepam was gradually tapered over the next 2 days, and he was subsequently discharged from the hospital.

Discussion

The pathophysiology of catatonia remains complex and not fully understood. Dopamine and γ-aminobutyric acid (GABA) depletion and dysfunction are 2 potential neurotransmitter derangements observed.[5] With methamphetamines, the acute intoxication and withdrawal phases may lead to rapid shifts in dopamine regulation in particular. This may account for the mostly hypoactive features observed in our case, especially given their direct/indirect modulation of the pre-supplementary and supplementary motor areas, as aberrant hyperactivity in these regions has been well described by neuroimaging studies.[6] Despite the adequate response to benzodiazepines in our case, the use of dopamine agonists should likely be considered in cases involving substances (eg, methamphetamines) that are known to cause such severe dysregulation of dopamine in particular.[7]

In summary, this case highlights the importance of screening for methamphetamines appropriately as potential etiologies for catatonia. They remain a popular drug of abuse in the United States especially. Further study and reports of their link to catatonia should be investigated given their potent and severe capability of precipitating this syndrome, even with an acute increase in the setting of chronic abuse.

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