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Cocaine Induced Postictal Psychosis: a Rare Cause of Antipsychotic Resistant Psychosis

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Abstract

Cocaine induced postictal psychosis is a rare and underreported phenomenon. When patients ingest large amounts of cocaine, patients can experience seizures. Despite returning to baseline after several days to weeks, patients can suddenly decompensate, exhibiting what is called postictal psychosis despite being abstinent from the substance. This condition can lead to chronic psychosis if not properly treated. In this case, a male in his twenties, presented with 1 month of auditory hallucinations, visual hallucinations, and paranoid delusions. After his last cocaine use 1 month ago, the patient developed seizure-like activity and shortly after returned to baseline. Approximately 1 week later, the patient started to experience psychosis. Due to persistent symptoms, the patient was admitted and started on Risperidone 2mg twice a day but did not respond. Clonazepam 1mg twice a day was added, which helped patient to return to baseline. Cocaine induced postictal psychosis is underreported, and it can be difficult to recognize. Similarly, there are few guidelines as to how to treat this condition. Clinicians need to rapidly address if they suspect that the patient is experiencing possible postictal psychosis given that this can lead to chronic psychosis.

Background

Seizures are defined as alterations of normal neurologic function, primarily caused by excessive hypersynchronous discharge from neurons in the brain and can be caused by drugs, such as cocaine.[1,2] Cocaine is a stimulant derived from coca leaves in South America and can cause psychosis, lasting hours to a few days.[3] Cocaine can induce seizures, however, the majority of cases are managed without hospital admissions.[2] There are a handful of case reports, most notably involving patients ingesting massive cocaine amounts; another way to induce seizures is through "kindling" where there is a repetitive administration of subconvulsive doses of cocaine leading to status epilepticus.[2]

Once a seizure subsides, patients may experience symptoms such as confusion, drowsiness, and headaches, which can last between 5 to 30 minutes. This is also known as postictal state. After being in a postictal state, patients return to baseline.[3] In postictal psychosis, despite several days to weeks after returning to this baseline, patients can suddenly decompensate, or get worse. This includes exhibiting psychotic symptoms including thought disorders, auditory and visual hallucinations, and paranoid delusions, which can last between 15 hours to less than 2 months.[4] Annual incidence of postictal psychosis is estimated at 6.4%,[5] and has been associated with epilepsy. Overall, postictal psychosis is not benign and can lead to chronic psychosis.[5] This highlights the need to diagnose and rapidly treat the patient if the clinician suspects that the patient is experiencing postictal psychosis.

A case of a man with a history of cocaine-induced seizure and psychosis, who presented with 1 month of psychosis after experiencing an episode of cocaine-induced seizure is presented.

Case Report

A male in his late twenties with a significant history of cocaine use (documented in the charts and from self-reports from patient) and depression presented to the Queens Medical Center-Punchbowl with auditory and visual hallucinations lasting for 1 month. The patient was sober for 7 months but relapsed 3 months prior to admission, using cocaine intermittently. His family members noted that he was having auditory hallucinations, which lasted 1 to 2 days after each cocaine use. One month prior to admission, the patient was living with his family. The patient's sister found him unconscious, "stuck" on the stair case, and unable to move. The patient's body was stiff with his shoulders shrugging up and down, which lasted for a few minutes. Shortly after, the patient went back to his baseline. Five years prior, the patient had a similar episode after using cocaine and there were concerns that he had a seizure. In the emergency department, however, the brain computerized tomography (CT) scan and electroencephalogram (EEG) were normal. On exam, the patient was noted to have blood in his mouth, but no urinary incontinence or tongue lacerations were seen on exam. After this emergency department visit, family did not seek additional medical care.

Several days after the momentary paralysis activity on the staircase, the family noted the patient started to exhibit psychotic symptoms, including auditory hallucinations, visual hallucinations, and paranoid delusions, which did not resolve. Due to persistent psychotic symptoms, the outpatient physician urged the patient to be admitted to the emergency department for further workup where most notably the patient's lab work, including urine drug screen, was negative. The patient was admitted to the inpatient psychiatric unit and was prescribed risperidone 2mg twice a day for his psychosis. However, the patient had a minimal response to risperidone. His family members, and later the patient, confirmed that his last cocaine use was about 1 month prior to admission, around the same time as when he experienced momentary paralysis in the stairwell. Given

Table 1. Logsdail and Toone's Criteria for Postictal Psychosis[7]

1-Episode of psychosis developed within one week after a seizure or a cluster of seizures.
2-Psychosis lasting at least 15 hours and less than 2 months.
3-Mental state characterized by delirium or delusions (paranoid, non-paranoid, delusional, misidentifications) or hallucinations (auditory, visual, somatosensory, olfactory) in clear consciousness.
4-No evidence of:
(a)Previous history of treatment with antipsychotic medication or evidence of psychosis within the past three months. (b)Antiepileptic drug toxicity. (c)EEG evidence of non-convulsive status epilepticus.

that there were concerns for postictal psychosis, clonazepam 1mg twice a day was also added to the regimen on the 3rd day of hospitalization, which helped the patient return to his baseline by the 4th hospital day. On the 5th hospital day, an EEG was completed given that the patient was no longer agitated, and the EEG results were normal. At the time of discharge, the patient denied any auditory and visual hallucinations.

Discussion

Cocaine is a psychostimulant, which can cause psychosis independent of seizures. In the state of Hawai'i, stimulants including methamphetamine and cocaine have contributed to significant hospitalizations with methamphetamine-related hospitalizations having risen from 532 hospitalized patients a year in 2009 to more than 2100 hospitalized patients a year in 2019.[6] Although cocaine use is less common than methamphetamine use in Hawai'i, the rates of cocaine hospitalizations have also risen by two-fold in the last decade.[6] In 2009, there were 167 patients hospitalized due to cocaine. By 2019, the number had risen to about 312 patients. Therefore, cocaine and its effects should continue to be clinically monitored by medical providers in Hawai'i.

Providers in this case were concerned that the patient had been unresponsive to the risperidone which commonly works well to manage stimulant-induced psychosis. Therefore, providers evaluated whether other diagnoses and therefore treatments were better suited for the patient. When evaluating postictal psychosis diagnosis for this patient, providers looked to the Logsdail and Toone's criteria for postictal psychosis[7] (see **Table 1**). Given that cocaine is a short acting psychostimulant, intoxication as well as withdrawal were unlikely to be the primary causes of patient's underlying psychosis, lasting for 1 month. The patient was started on risperidone due to the treatment recommendations based on stimulant-induced psychosis as well as possible postictal psychosis, as antipsychotics are a common treatment for substance-induced psychosis. Both first-generation antipsychotics and second-generation antipsychotics are equally efficacious. The team chose to first prescribe the patient risperidone as there was less likely to be drug-drug interactions and there was a lower seizure risk associated with this antipsychotic.[7] However, the patient was minimally responsive to treatment.

The recommended treatment for postictal psychosis consists of both an antipsychotic and a benzodiazepine.[4] Given the limited literature available, it is unclear how this combination works to treat postictal psychosis, however, in animal models both antipsychotic and benzodiazepines are shown to have additive effects; benzodiazepines are antagonists to the central nervous system (CNS), while the antipsychotics can mediate the neurotransmitters, dopamine and serotonin.[8] The theory is that cocaine over activates the CNS sympathetic system through dopamine, muscarinic, and sigma receptors and these therapies work to reverse these effects.[8] It is therefore important for providers to be cognizant of cocaine postictal psychosis as the treatment differs from substance induced psychosis. Not only is an antipsychotic required, but benzodiazepine may also be warranted. While the downstream effects specific to postictal cocaine psychosis are unknown, there is an elevated risk of the postictal psychosis progressing to chronic psychosis when it is not treated.[9] This highlights the need to rapidly address this condition if the clinician suspects that the patient is experiencing postictal psychosis.

Limitations

There were several limitations to this case report. Although the patient denied ever using methamphetamine, the authors cannot rule out methamphetamine or other substance induced psychosis as cocaine may contain contaminants. Although his urine drug screen was negative, it is important to remember that urine drug screens are screeners and cannot definitively say whether patient has used a substance. The result may have been a false negative and therefore would have required a confirmatory test to be completed. However, the patient improved during hospitalization prior to the confirmatory tests being completed. Patients can exhibit methamphetamine induced psychosis due to seizures however this is less common. Due to its longer half-life, mmethamphetamine more commonly can cause psychosis that has a longer duration compared to cocaine. Current national drug trends show that cocaine can be contaminated with fentanyl. Another concern was that although the urine drug screen was negative and the patient stated his last drug use was the prior month, interim drug use within the month cannot be definitively ruled out. It also must be pointed out that while the patient was started on an antipsychotic and did not respond to treatment for sev-

eral days, it is unclear whether the patient would have responded had the team tried another antipsychotic or waited for a longer period. It is unclear how long it would take an antipsychotic to fully reverse substance induced psychosis given that it is heavily dependent on the amount ingested as well as the extent of chronic drug use. While a primary psychiatric disorder was initially considered, ultimately it was less likely as the patient had no family history of schizophrenia and denied any prodromal symptoms including cognitive decline and changes in mood and behavior. Still, given that the treatment team evaluated the patient for a short duration in the setting of an acute hospitalization, a primary psychotic disorder cannot be definitively ruled out.

Conclusion

Health care providers should consider cocaine induced postictal psychosis when a patient presents with a recent history of cocaine induced seizures and now exhibits psychotic symptoms. Given such a clinical diagnosis, providers should consider treating the symptoms with both an antipsychotic and a benzodiazepine. Without treatment, patients risk experiencing chronic psychosis, which may result in self-harm or harm to others.

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Conflicts of interest

None

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