

Case Report

Gabapentin Abuse and Overdose: A Case Report

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Abstract

Gabapentin is a commonly prescribed medication for neuropathic pain, seizure disorder, and anxiety. It is deemed a safer alternative to benzodiazepines, with lower abuse potential, but it has been misused. There is growing concern about gabapentin's abuse potential and withdrawal. A 20 year old man presented to the psychiatry inpatient unit after being medically cleared from the emergency room after ingesting a large amount of gabapentin and oxazepam in order to become intoxicated. He was monitored closely for signs of withdrawal, and was encouraged to take acetylcysteine solution, at the recommendation of poison control. Patient was subsequently discharged in stable condition after two days of inpatient treatment. He returned several hours later, having again ingested a large amount of gabapentin, but was discharged from the emergency room.

INTRODUCTION

The abuse potential of drugs that stimulate dopamine release has driven the search for drugs that influence alternate neurotransmitters, and the glutamergic pathway is a potential target for the development of new therapeutic practices [1]. Gabapentin is one such drug that modulates GABA and glutamate levels; its clinical effectiveness has been reported across a wide variety of disorders ranging from neuropathic pain and epilepsy to psychiatric disorders. While abuse potential for gabapentin is less than that for dopamine agonists, relatively large amounts of the drug can induce the "high" that abusers seek. Little research and attention has been generated with regards to gabapentin abuse potential. Also, the role of acetylcysteine in treating gabapentin overdose has received virtually no attention, and yet, this is the recommended treatment by poison control. It is for these reasons that we present a case of gabapentin abuse and overdose.

CASE PRESENTATION

Mr G is a 20 year old man with history of polysubstance use disorder and bipolar 2 disorder who presented to the emergency department after taking eight 600mg pills of gabapentin as well six to seven 15mg pills of oxazepam. Additionally, he was smoking marijuana and drinking alcohol at the time of the overdose. He was initially unresponsive when found by EMS, but was quickly awoken with smelling salts. In the ED, the patient appeared intoxicated, with slurred speech, and was verbally abusive to staff. AST and ALT were 66 and 96 respectively. Urine

drug screen was positive for cannabinoids, and his gabapentin level was 6.2. He was given one dose of acetylcysteine solution at 140mg/kg, and transferred to psychiatry. Psychiatry team was told to continue the acetylcysteine at 70 mg/kg every four hours for a total of 17 doses. This was at the recommendation of poison control. Patient refused all treatment with acetylcysteine solution after he was given the initial dose. The patient denied suicidality and reported that he takes gabapentin for anxiety, but periodically takes large doses in order to "get high". He described the high as similar to the one he gets when smoking marijuana. Patient also takes bupropion 300mg for depressed mood and divalproex 500mg twice per day for mood stability. During his admission, patient became more cooperative with staff, attended group meetings and expressed interest to be discharged. On day two of admission, his liver enzymes dropped to 51(AST) and 85(ALT) and his gabapentin level was <1.5. He was discharged, and instructed to stop taking gabapentin, and to follow up with outpatient psychiatry within one week.

Several hours after discharge, he was brought back via EMS to the emergency department after falling and having a "seizure" witnessed by his girlfriend. Patient again admitted to taking multiple gabapentin pills "to get a buzz". Patient was discharged from the emergency department after a negative workup.

DISCUSSION

While the mechanism by which Gabapentin (GBP) exerts its therapeutic effect is not fully understood, it is has been proposed that the drug acts on Gamma-Aminobutyric Acid

(GABA) and glutamate levels in the brain. Goldlust [2] found that gabapentin modulates a number of enzymes involved in the synthesis and breakdown of both GABA and glutamate, and in summary, weakly inhibited GABA aminotransferase (GABA-T), decreasing conversion of GABA to Glutamate; it increased glutamate dehydrogenase (GDH), increasing glutamate breakdown, and competitively inhibited branched chain amino acid transferase (BCAA-T), which decreases conversion of BCAAs to glutamate. With our patient we were prompted by poison control to administer acetylcysteine solution. N-acetylcysteine (NAC), an amino acid derivative of cysteine, is available as a health supplement in health food stores and pharmacies and has been used as a hepatoprotective agent in the treatment of acute acetaminophen overdoses. Moran et al. [3] proposed that activation of cysteine/glutamate exchange through NAC administration restores extracellular glutamate levels and reduces drug-seeking behavior. Similar mechanisms were proposed by Grant, et al. [4] for the treatment of trichotillomania, which is believed to be associated with decreased extracellular glutamate concentrations in the nucleus accumbens. In the context of gabapentin overdose, it is not entirely clear what the net deviation in glutamate and GABA levels may be in the brain, however, it appears that NAC administration may exert a therapeutic effect by increasing concentration of extracellular glutamate. Another reason to give NAC in cases of overdose is to cover for potential acetaminophen overdose, especially in cases where suicidality is a concern.

A case report published in 1997 reports a 42 year old woman who was dependent on crack cocaine, and when she would go through withdrawal, she used her husband's gabapentin. She reported that the gabapentin relaxed her and helped her diminish cocaine cravings [5]. Foltinet al. [6] reported similar outcomes, that gabapentin reduced cocaine cravings. Gabapentin diversion was also reported in Florida correctional facilities, with some inmates admitting to snorting gabapentin in order to get "high" [7]. In several case reports, the dangers of gabapentin abuse are apparent and potential for severe and potentially deadly withdrawal is clear. Pittenger and Desan [8] reported two cases of gabapentin withdrawal, leading to delirium tremens, which only subsided fully after gabapentin was resumed. Another case reported the development of withdrawal symptoms common to

those seen in ethanol withdrawal in a 53 year old woman 2 days after gabapentin discontinuation. Withdrawal symptoms did not respond to lorazepam, and improved only after gabapentin was resumed [9].

Although the abuse of gabapentin remains limited, it can be potentially dangerous, especially due to the similar withdrawal pattern as that seen with alcohol or benzodiazepines. Similarly, patients withdrawing from this medication require careful monitoring for the development of delirium tremens and seizures. At this time, there is a need for further investigation into the epidemiology of gabapentin abuse, the potential for dependency in known drug addicts, as well as a discussion about the need for more stringent prescription monitoring for this common and increasingly-prescribed neuromodulator.

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