

# A sound sleep

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Abstract

Benzodiazepine is a commonly encountered agent of poisoning, which is readily diagnosed by a pertinent history of drug ingestion and the clinical scenario. In the absence of a proper drug history, the diagnosis becomes challenging. Proper clinical assessment, urine assays and imaging play a very important role in reaching the diagnosis. We present a case of acute benzodiazepine poisoning without a history of drug intake. The key pointers toward diagnosis were an unarousable state with obstructive apnea. Magnetic resonance imaging (MRI) of the brain revealed peculiar symmetrical isolated globus pallidus T2 hyperintensity. We believe this to be the first report of isolated bilateral basal ganglia T2-weighted hyperintensity in MRI in the setting of acute benzodiazepine poisoning from India.

**Keywords:** Benzodiazepine, bilateral basal ganglia hyperintensity, India, MRI, poisoning

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## Introduction

Benzodiazepines are among the most frequently prescribed drugs worldwide. The ease of availability makes them one of the most commonly abused drugs. Although common as an agent for suicidal or homicidal poisoning, these drugs are remarkably safe. Deep coma is a rarity for benzodiazepine poisoning, with severity of central nervous system (CNS) depression depending on the dose, the age of the patient and his or her clinical status. Treatment of a certain case of acute benzodiazepine poisoning is with supportive measures and the administration of flumazenil.

If a proper drug history is available, the diagnosis can be made with confidence. On the other hand, in the absence of a proper history, diagnosis depends on high index of a clinical suspicion and reliance of laboratory parameters, which are most commonly nonchalant.

We present a case of acute benzodiazepine poisoning without any guiding history of drug intake, which was

successfully diagnosed and treated. The key pointers toward diagnosis were an unarousable state with obstructive apnea. Magnetic resonance imaging (MRI) of the brain revealed peculiar symmetrical isolated bilateral globus pallidus T2 hyperintensity (BGPTH) without any other focal lesion or altered signal anywhere else. This report emphasizes the importance of high index of clinical suspicion for proper diagnosis. We believe this to be the first report of such a peculiar MRI finding in the setting of acute benzodiazepine poisoning from India.

## Case Report

In July 2010, a 52-year-old male was admitted in an unarousable state. His relatives found him in this condition the morning before, when he was apparently sleeping past his normal waking time. There was no history of any drug intake in the recent or distant past. On immediate evaluation, his vitals were: blood pressure – 130/78 mmHg, pulse rate – 108/min, respiratory rate – 16/min and no evidence of cyanosis. Capillary blood glucose was 108 mg/dL. A quick neurological examination revealed an unconscious state with absent vestibule-ocular reflexes, unresponsive plantar reflexes with generalised hypotonia and depressed deep tendon reflexes. Pupillary reflexes were normal. Systemic examination was entirely nonchalant except for the fact that the patient appeared to snore prominently. An upper airway assessment did not reveal any obstruction.

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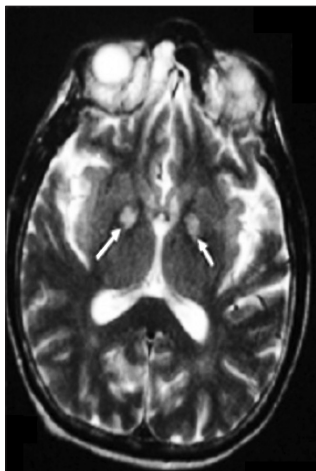
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Pulse oximetry showed SpO<sub>2</sub> of 96%. The ECG was normal. Arterial blood gas showed a normal pH (7.38) with, PaO<sub>2</sub> of 96 mmHg, PaCO<sub>2</sub> of 41 mmHg with normal bicarbonate (23 mmol/L) and anion gap (12 mEq/L). While collecting the gastric lavage samples for toxicological purposes, a plain computed tomography (CT) scan of the brain was done, considering the patient was in neurological shock following a cerebrovascular accident, which came to be absolutely normal.

The complete hemogram, liver and renal function tests and electrolytes were normal (Hb – 11.6 gm/dL, total leucocyte count – 7600/μL [neutrophil 73%], creatinine – 0.9 mg/dL).

Awaiting the reports of the gastric lavage samples and without any improvement (or deterioration of the patient), an MRI brain with contrast was done, which revealed BGPTH [Figure 1]. Among the common causes of BGPTH (discussed below), carbon monoxide poisoning and the encephalopathies were excluded by clinical setting and cyanide and H<sub>2</sub>S poisonings were excluded by the absence of lactic acidosis (blood lactate – 0.6 mmol/L).

Therefore, we were facing an unconscious patient who was snoring with borderline carbon dioxide retention and had a BGPTH inexplicable by common causes. Keeping in mind that benzodiazepines sometimes cause coma, snoring and obstructive apnea,<sup>[1]</sup> we screened the patient's urine sample. Benzodiazepine was strongly positive (1380 ng/mL) by fluorescent polarization immunoassay. The patient or his relatives could not give any reference regarding the source of benzodiazepine – he was not used to taking benzodiazepine as sedative or hypnotic. Presence of benzodiazepine in urine



**Figure 1:** Magnetic resonance imaging of the brain of the patient showing bilateral T2-weighted globus pallidus hyperintensities (indicated by arrows)

and absence of similar past history ruled against “endozepine-4 stupor syndrome”.<sup>[2]</sup> The patient was given a trial dose of flumazenil (500 μg IV), to which he dramatically responded with almost full recovery of consciousness level. This was followed by a therapeutic dosage of flumazenil (500 μg IV up to 2 mg). The patient recovered uneventfully by the second day of therapy and fifth day of admission, and was discharged the next day.

The patient had been on regular follow-up for the past 1 year and did not show any neurological symptoms.

## Discussion

Benzodiazepine poisonings cause up to 30% emergency admissions. Most of these cases come with a positive drug history.<sup>[3]</sup> Deep coma requiring assisted ventilation is rare. The severity of the CNS depression is influenced by the dose, the age of the patient and his or her clinical status prior to the ingestion.<sup>[4]</sup> A large retrospective study including 702 benzodiazepine poisoning cases found that 56% had severe CNS depression, with 47% requiring orotracheal intubation and 18% requiring artificial ventilation. Only five cases were fatal.<sup>[3]</sup>

Among the disorders that may cause bilateral GPTH are carbon monoxide,<sup>[5]</sup> cyanide, hydrogen sulfide, methanol, disulfiram, osmotic myelinolysis, uremic or hepatic encephalopathies, radiation therapy or cerebrovascular accidents.<sup>[6]</sup> There is only a single report of a 62-year-old patient with suicidal olanzapine poisoning with choreoathetosis and MRI of brain showing bilateral small hyperintense foci in globus pallidi.<sup>[7]</sup> In this regard, it is important to remember that BGPTH may occur as part of the normal ageing process, especially after the age of 60 years, and it is rare in younger people.<sup>[8]</sup>

In the absence of a definite history of drug intake, an unconscious elderly man becomes a diagnostic nightmare. Three clinical pointers helped us crack our dilemma – snoring, carbon dioxide retention and BGPTH. These led us to consider poisoning as the etiology. We would like to emphasize that poisoning is an important differential in similar clinical scenarios, even without any definite drug history. A high index of clinical suspicion and urine screening tests become of paramount importance for diagnosis.

This report emphasizes the importance of a high index of clinical suspicion for proper diagnosis. We believe this to be the first report of such a peculiar MRI finding in the setting of acute benzodiazepine poisoning from India.

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