

the fact it is most commonly carried out in the home, the ready availability of the materials used, and widespread perceptions about the method. Feasible strategies for the prevention of suicide by hanging must involve greater vigilance in institutional settings and adequate management of patients with previous attempts and patients with mental disorders. The scarcity of effective preventive measures to reduce escalation of this form of suicide demands a comprehensive discussion among professionals, families, and health managers who are at the frontline of care of suicidal people. Future research should focus on the details of deeply rooted perceptions about the meaning of death by hanging in different cultures and the origins of this knowledge as used by suicidal individuals, in order to reduce their willingness to plan suicide by this method.

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Treating nightmares in PTSD with doxazosin: a report of three cases

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This paper reports three cases of patients successfully treated with doxazosin, as an alternative to prazosin, for nightmares associated with posttraumatic stress disorder (PTSD). All patients were male, middle-aged, severely ill,

and had comorbid major depressive disorder (MDD). They developed PTSD after the Kiss nightclub fire in 2013, and were receiving care at a specialized outpatient clinic at the University Hospital of Santa Maria, Rio Grande do Sul, Brazil. A thorough clinical evaluation was conducted, including of specific sleep aspects (latency, length, frequency of nightmares, etc.)

The first patient was a relative of a victim and a survivor. He was taking venlafaxine 225 mg, quetiapine 125 mg, and chlorpromazine 200 mg, and reported nightmares at least three times a week. Doxazosin 1 mg at night was prescribed. After 2 weeks, he reported no more nightmares, but insomnia persisted as at presentation. Doxazosin was progressively increased to 4 mg in an attempt to improve insomnia, and chlorpromazine was reduced to 100 mg to avoid side effects. After 4 months of follow-up, he remained free of nightmares, but insomnia continued.

The second patient had helped rescue the victims. He also had type 2 diabetes mellitus and hypercholesterolemia. He was taking sertraline 200 mg, chlorpromazine 300 mg, and clonazepam 2 mg. He reported nightmares twice a week and consequent intermediate insomnia. He was very reluctant to reduce chlorpromazine or clonazepam doses, fearing that his symptoms would worsen. Doxazosin 2 mg at night was prescribed. He returned 7 weeks later without nightmares or insomnia.

The third patient was a survivor of the fire. He was taking sertraline 200 mg, lithium carbonate 1,500 mg, and clonazepam 1 mg. He was depressed and reported nightmares and insomnia. Doxazosin was started at a dose of 1 mg; after 2 weeks, nightmares had decreased. After 45 days, doxazosin was increased to 2 mg. At follow-up, he reported nightmares less than once a week.

Nightmares are core symptoms of PTSD, and even contribute to increasing rates of suicide among these patients.¹ Although antidepressants are considered the first-line treatment for PTSD, no evidence has found that these drugs are superior to placebo in treating PTSD-related insomnia and nightmares.²

Doxazosin, an alpha1-adrenergic antagonist prescribed to treat benign prostatic hyperplasia, is widely available for users of the Brazilian public health system. Furthermore, it has a long half-life (16 to 30 hours), allowing not only easy dosage, but maintenance of effect throughout the night. Finally, its good absorption profile reduces the risk of hypotension. All of these characteristics positively differentiate doxazosin from its analogue, prazosin, which has been described as an efficacious adjuvant treatment for PTSD-associated nightmares.³

To the best of our knowledge, this is the first case series of Brazilian patients with PTSD successfully treated with adjuvant doxazosin for associated nightmares. After initiation of adjunctive doxazosin, two patients were free from nightmares and one experienced improvement for up to 7 weeks. Our findings are in accordance with those of a review of medical records⁴ and a pilot clinical trial,⁵ but placebo-controlled studies are needed to confirm it.

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Life-threatening hypoglycemia attributable to haloperidol in a patient with schizophrenia

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Haloperidol is an antipsychotic drug widely used in emergency departments, general medicine wards, and psychiatry units. Long-term therapy is used in psychotic disorders, including schizophrenia and dementia, and bipolar disorders¹; for short-term use, it is frequently the drug of choice in acutely confused states. We present a case of a patient with an established diagnosis of

schizophrenia who developed prolonged, life-threatening hypoglycemia under haloperidol therapy.

A 67-year-old woman was admitted to our emergency department due to altered level of consciousness. She had been diagnosed with schizophrenia in 1980 and been under psychiatric care ever since. She had a previous history of epilepsy, endometrial adenocarcinoma (treated with hysterectomy), and dyslipidemia. Two weeks prior to admission, her haloperidol dose had been increased from 5 mg oral bid to 5 mg tid and 100 mg intramuscular (haloperidol decanoate) every 4 weeks to 100 mg every 2 weeks. She was also on simvastatin (20 mg oral qd), valproic acid (200 mg oral bid), and mirtazapine (15 mg oral qd).

On admission, the patient had sinus bradycardia (heart rate 38 bpm) and was comatose with a Glasgow Coma Score of 3 (O1V1M1), profoundly hypoglycemic (capillary blood glucose 24 mg/dL), and hypothermic (axillary temperature 30.7 °C). Passive rewarming measures and 40 mL of 30% glucose were administered intravenously with a positive response; the patient regained consciousness and became alert without any neurologic deficit. Continuous intravenous infusion of glucose plus 30% glucose boluses and multiple feedings were administered, but during the following 36 hours, recurrent episodes of hypoglycemia developed, as shown in Figure 1. Extensive diagnostic studies were performed to determine the cause of hypoglycemia; infection, organ failure, thyroid abnormalities, suspicion of insulinoma, and adrenal insufficiency were ruled out. A prolonged fasting period or accidental administration of hypoglycemic agents were also excluded. In line with previous cases of hypoglycemia due to haloperidol, insulin levels were elevated, although within normal limits, and C-peptide levels were normal (these tests were added to the first blood sample collected in the emergency department). After the exclusion of other causes of hypoglycemia and due to high suspicion, haloperidol was discontinued after 12 hours in the emergency department. At 36 hours after discontinuation of haloperidol, hypoglycemia had resolved.

All of the patient's medications had been in long-standing use, only in haloperidol has hypoglycemia been described as an adverse effect, and only the dose of haloperidol had been increased recently before the development of hypoglycemia. According to the Naranjo algorithm, this case presents a score of 9, indicating a definite adverse drug reaction.

Although the presence of high or relatively high levels² of insulin during haloperidol-induced hypoglycemia in the cases previously described in the literature suggest that glucose sensitization is implicated, the precise mechanism is still unclear.^{3,4} Some studies conducted in rats revealed that haloperidol inhibits glucose-stimulated insulin release² through the inhibition of ATP-sensitive potassium channels in the pancreatic beta cells.⁵ In our patient, the hypoglycemic episode was prolonged and refractory to intravenous glucose (in 48 hours, 554 g were administered, plus oral feedings). We present this case to raise awareness in the medical community to