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Dekkers, Bart G J; Eck, Ruben J; Ter Maaten, Jan C; Touw, Daniël J

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Case Report

An acute oral intoxication with haloperidol decanoate

Bart G.J. Dekkers, PharmD, PhD a,⁎, Ruben J. Eck, MD b, Jan C. ter Maaten, MD, PhD c, Daniël J. Touw, PharmD, PhD a

a Department of Clinical Pharmacy and Pharmacology, University Medical Center Groningen, University of Groningen, Groningen, The Netherlands
b Department of Critical Care, University Medical Center Groningen, University of Groningen, Groningen, The Netherlands
c Department of Internal Medicine, Emergency Department, University Medical Center Groningen, University of Groningen, Groningen, The Netherlands

Abstract

Haloperidol decanoate is a typical antipsychotic drug used as maintenance therapy for schizophrenia and mood disorders formulated as an ester for intramuscular injection. Cases of oral haloperidol decanoate intoxications have not been described in literature. In this report, we present for the first time a case of an oral ingestion of haloperidol decanoate of a young woman who presented to the emergency department following an intentional oral ingestion of 1 ampoule of haloperidol decanoate 100 mg. At presentation, she had a bilateral rest tremor of both hands and mild hypothermia. No other obvious signs of an intoxication were observed. She was treated with a single dose of activated charcoal and laxative and was admitted to the intensive care for rhythm monitoring and observation. During the night the QTc interval increased to 453 ms, but stayed within the normal range. Haloperidol plasma levels increased as well, but also stayed within therapeutic ranges. These findings indicate that treatment with oral activated charcoal was sufficient to prevent any serious events.

1. Introduction

Haloperidol is an effective typical antipsychotic drug, primarily used for the treatment of schizophrenia, mood disorders and delirium [1]. In therapeutic doses (up to 20 mg daily), its antipsychotic actions are thought to be mainly mediated by inhibiting dopaminergic D2 receptors. In addition, haloperidol interacts with muscarinic, histamine and adrenergic receptors and the delayed rectifier potassium channel. Adverse effects, including extrapyramidal effects, QTc prolongation and torsade de points relate to its pharmacological actions [1]. Haloperidol decanoate, formulated as an ester for intramuscular injection, is administered in 2- to 4-weeks intervals as maintenance therapy. The effects of oral intake of haloperidol decanoate are unknown. This case report describes an acute ingestion of 100 mg haloperidol decanoate.

2. Case report

A young woman with a psychiatric background presented to the emergency department following an intentional oral ingestion of 1 full ampoule of haloperidol decanoate 100 mg during a panic attack. She had a history of suicide attempts and had been treated with haloperidol decanoate intramuscularly every 4 weeks for 3 months. The last regular dose was 4 weeks earlier. At presentation, approximately 1.5 h after ingestion, she had a respiratory rate of 12–14 breaths/min with an oxygen saturation of 99% on room air. Her blood pressure was 110/70 mm Hg and heart rate 75 beats/min. The Glasgow coma score was 15 without clear signs of an intoxication, except for a bilateral rest tremor of her hands that had been present for over a week, but had increased after intake of the haloperidol decanoate. Her body temperature was reduced to 35.3 °C. Initial laboratory testing revealed normal serum electrolytes, liver and renal function tests. The haloperidol plasma concentration was 4 μg/l (therapeutic 1–15 μg/l), which was similar to levels two weeks earlier (5 μg/l). Paracetamol was undetectable. Electrocardiography showed a QTc interval of 412 ms, which was also comparable to earlier measurements. At the emergency department activated charcoal (50 g) in combination with sodium sulphate 15% 200 ml was administered to eliminate non-absorbed drug. The patient was admitted to the intensive care overnight for rhythm monitoring and continued observation. During the night the QTc interval increased steadily to a maximum of 453 ms and stabilized at the early morning. The haloperidol plasma concentration was 8 μg/l. Her tremor remained stable and no other symptoms developed. The next day, she was transferred to an outpatient clinic for psychiatric follow-up.

3. Discussion

Currently, the clinical course of an intoxication with oral haloperidol decanoate has not been reported. In the current case the effects were relatively mild, probably due to the early presentation and treatment.
Symptoms of an intoxication with the active drug include adverse reactions, such as extrapyramidal, anticholinergic and cardiac effects, including QTc prolongation [1-3]. Although haloperidol intoxications are seldom life threatening, fatalities after high dosages (≥100 mg) have been described [4,5]. Moreover, higher doses of haloperidol increase the risk of developing neuroleptic malignant syndrome [6]. The QTc interval increased during the night, but stayed within the reference range (<460 ms). In line with previous non-fatal haloperidol intoxications [1-3], an increase in extrapyramidal adverse effects and a reduction in body temperature were observed. After overnight observation, the patient was released with some minor tremor, which had already been present before.

After intramuscular injection, haloperidol decanoate is gradually released to the circulation. After release, the ester is quickly hydrolysed by endogenous esterases to haloperidol [7]. The biopharmaceutical effects of oral intake are unknown, however, the ester is likely to be hydrolysed to haloperidol by the acidic environment in the stomach or by digestive enzymes in the small intestine. As the complete dose of haloperidol could be released, the intoxication was judged as potentially life threatening. Oral activated charcoal and laxative were administered to capture and eliminate remaining haloperidol and haloperidol decanoate, because of the recent intake and the potential delay in release from the oil and cleavage of the prodrug. To establish a reference haloperidol level, an early blood sample was drawn, which was within the therapeutic range [8]. The patient was admitted to the intensive care for continued monitoring and observation. The next morning, a doubling of the haloperidol concentration was found, which was well within the therapeutic range and below toxic levels [8]. No further haloperidol levels were determined due to the mild course of the intoxication and resolution of the initial symptoms. Based on this, we would suggest to treat patients with an acute oral haloperidol decanoate intoxication with oral activated charcoal and to observe the patients in the emergency department or intensive care unit for at least 8 h until symptoms have resolved.

4. Conclusion

After oral ingestion of haloperidol decanoate adverse effects, associated with the mechanism of action of haloperidol, may occur. In this single case, without other co-ingestants or comorbidities, treatment with a single dose of oral activated charcoal and laxative was sufficient to prevent any serious events.

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