

Cardiomyopathy Following Chloralose Intoxication: A Case Report

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Abstract

Alpha-chloralose is a poisonous compound used as a rodenticide. Self-poisoning with alpha-chloralose is a rather common reason for referral to the ICU in Tunisia compared to Europe.

We present an unusual case of chloralose rodenticide voluntary intoxication in a young woman with life-threatening neurological, hemodynamic and respiratory distress requiring the use of norepinephrine and ventilatory support. ECG abnormalities, elevated cardiac troponin level, severe global hypokinesia with a collapsed left ventricular ejection fraction (LVEF) revealed by trans-thoracic echocardiography justified the utilization of high dose of dobutamine in combination with norepinephrine and anti-ischemic therapy. An improvement in cardiac function followed and both of neurological and respiratory patient's status well evolved.

Myocardial damage during chloralose intoxication can be explained by the context of stress, severe hypoxia and tachycardia, but toxic cardiomyopathy remains to be evoked.

Keywords: *Chloralose; Intoxication; Hemodynamic Distress; Global Hypokinesia; Cardiomyopathy*

Introduction

Alpha-chloralose is a poisonous compound formed by a combination of chloral and glucose. It has originally been used in Europe since the eighties as a human and veterinary hypnotic agent [1]. It is currently no longer used in medical practice. Nevertheless, it is still utilized as a rodenticide. Self-poisoning with alpha-chloralose is a rather common reason for referral to the ICU in Tunisia compared to Europe. The incidence of chloralose poisoning is superior to 3 cases per 100,000 inhabitants in Tunisia since 1997 versus 63 to 108 cases, per year, of chloralose intoxication, in France, in overall from 1999 to 2012 [2,3].

There is no specific antidote. The final outcome is good on average with a mortality of 0.4% in Tunisia [4].

Neurological signs are at the forefront involving jerks and coma. Cardiorespiratory features are not specific [3]. In fact, cardiac toxicity is not well known. There are only few reports of acute heart failure related to chloralose poisoning.

Aim of the Study

The aim of this paper is to present an unusual case of chloralose rodenticide voluntary intoxication in a young woman complicated with cardiomyopathy and acute heart failure.

Case Report

A 34 year-old woman with no medical history ingested two raticide bags, i.e. a toxic dose of 4.2g of chloralose, in a suicide attempt subsequently to a conflict with her husband. Admitted to the Emergency Department after an hour, physical examination initially revealed a comatose patient with myoclonic jerks, mottled skin of the whole body, cyanosis, hypersialorrhea and bradypnea. The blood pressure was of 70/50 mm of Hg, her heart rate was at 130 flapping/min. Norepinephrine was used up to a dose of 2 mg per hour to reach a mean arterial pressure of 65 mm Hg. Ventilatory support was required. The patient had a gastric lavage and a single dose of activated charcoal.

In the ICU, the electrocardiogram performed to the patient showed sinus tachycardia at 150 bpm, poor R wave progression and ST-segment depression in inferior leads (DIII and aVF) and from V3 to V5 (Figure 1). The chest X-Ray was in favor of pulmonary edema (Figure 2). Myocardial necrosis markers were positive with a cardiac high-sensitivity troponin I peak of 9911 ng/L (Normal range is inferior to 4

ng/L). Trans-thoracic echocardiography revealed severe global hypokinesia with a depressed left ventricular ejection fraction (LVEF) of 16% (Figure 3) and elevated LV filling pressures justifying the utilization of high dose of dobutamine in combination with norepinephrine and anti-ischemic therapy which consists in heparin, clopidogrel, aspirin, statin.

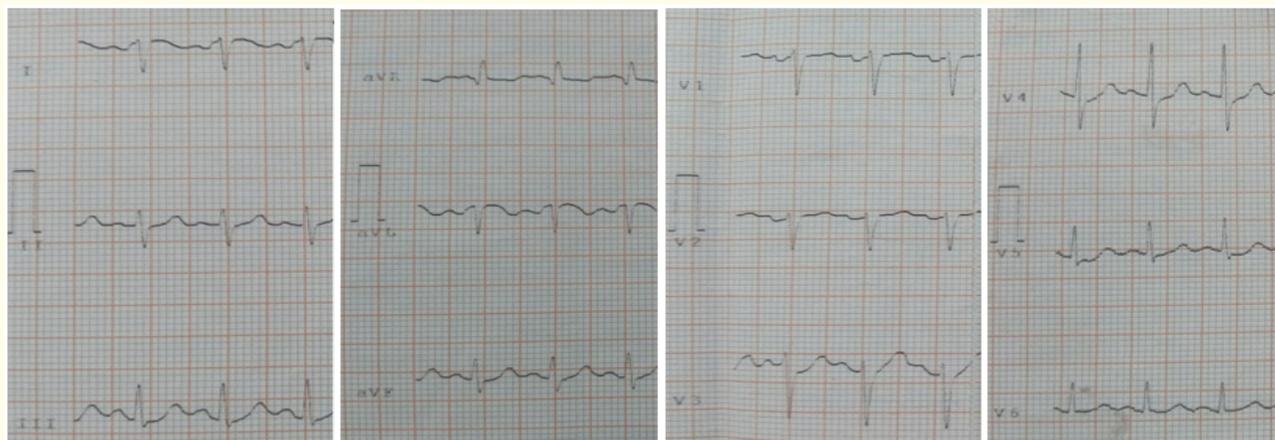


Figure 1: Initial ECG.



Figure 2: Chest X-Ray showing pulmonary edema.



Figure 3: LFEF of 16,9%, Simpson method.

An improvement in cardiac function followed with a normal controlled ECG and an increase in LVEF controlled at 40% and then at 50% (Figure 4) and norepinephrine weaning on day 4. Both of neurological and respiratory patient’s status well evolved with extubation on day 5 with dobutamine kept. Withdrawn from vasoactive drugs was on day 8 and referral to a specialized psychiatry department was on day 10.

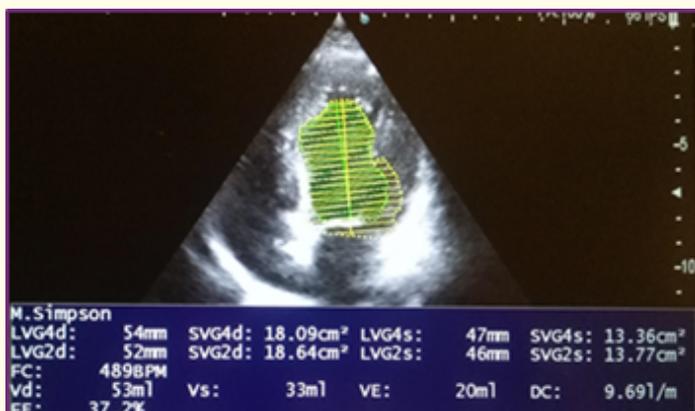


Figure 4: LVEF of 37,2%, Simpson method.

Discussion

Chloralose has two paradoxical properties: a sedative and a motor hyperexcitability effect. Coma and jerks are almost constant. Respiratory signs are not specific and bronchial hypersecretion is the main symptom [4].

Generally, hemodynamic tolerance is very good during chloralose intoxication [4]. Except that toxic myocardial damage can be observed, yet literature data studies are to this day still scarce [5]. Indeed, apart from the sedative but also synaptic neuroexcitatory effect, cardiac dysfunction has been reported [6]. Plural mechanisms are incriminated, including direct cardiac toxicity and transient negative inotropic effect [7] Nevertheless, stress cardiomyopathy precipitated by intense sympathetic pathways stimulation can cause coronary or microcirculatory spasm and therefore should be considered.

Myocardial damage during chloralose intoxication can be explained by the context of stress, severe hypoxia and tachycardia, but toxic cardiomyopathy remains to be considered.

The management of chloralose poisoning is symptomatic and its aims to maintain the vital functions integrity. A single dose of activated charcoal is usually recommended within 2 hours after the ingestion, after upper airway protection if required [4]. The treatment of the cardiogenic shock is based on using inotropic agent like dobutamine to increase cardiac output and thereby increase mean arterial blood pressure and maintain perfusion to vital organs and tissues [8].

Final outcome is generally favorable. Prognosis is related to the delay of management.

Conclusion

Acute heart failure related to chloralose poisoning must be evoked. Cardiomyopathy is reversible when management is early.

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