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Case report

Acute heart failure following severe chloralose poisoning: A case report

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Abstract Self-poisoning with alpha chloralose is frequent in Tunisia. Neurological signs are prominent involving myoclonic jerks and coma. Cardiac toxicity is not well known.

We report the case of a 19 year-old woman who ingested a chloralose rodenticide and was admitted to our intensive care unit. Four hours after admission she developed acute heart failure with pulmonary edema and elevated troponin. Echocardiography showed diffuse hypokinesia. Patient's cardiac and respiratory status improved progressively and echocardiography performed 5 days after was normal.

Cardiac toxicity following acute chloralose poisoning is uncommon and potentially serious. Its mechanism is poorly understood.

1. Introduction

Alpha-chloralose (AC) is a rodenticide, formed by condensing a chloral with a pentose or a hexose sugar. Self poisoning with alpha-chloralose in humans was seldom described in the literature according to its low frequency in European country [1–3]. In Tunisia the rate of chloralose poisoning was estimated to 1000 hospitalizations yearly. We report here a case of chloralose rodenticide voluntary intoxication in a teenager complicated by an acute heart failure.

2. Clinical record

A 19 year-old woman was admitted to the emergency department three hours following a suicide attempt with a chloralose rodenticide (8 g). Its clinical features included coma (GCS: 7) with myoclonic jerks. The initial chest radiograph was normal. She was mechanically ventilated and did not had gastric lavage. Afterward she was admitted to our intensive care unit. Four hours later, she developed severe hypoxemia (PaO_2/FiO_2: 150 mmHg) and the hemodynamic status remained stable. A second chest radiograph showed bilateral pulmonary edema (Fig. 1). The electrocardiogram showed an ST-segment depression in V3-V4 leads. The troponin level was elevated (13.36 ng/ml). Echocardiography showed diffuse hypokinesia

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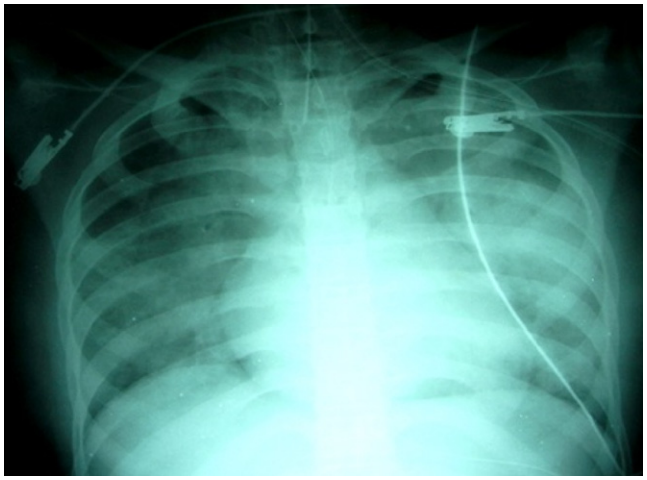

Figure 1 Chest radiography showing an acute pulmonary edema.

with a left ventricular ejection fraction (LVFE) of 30%. The patient's respiratory status was improved progressively and was extubated two days later. The cardiac troponin level declined to 1.06 ng/ml and the echocardiography performed 5 days after showed a LVFE of 65%. She was discharged home without sequelae.

3. Discussion

This report shows that acute heart failure with pulmonary edema might be a complication of chloralose poisoning; therefore, clinicians should look for signs of heart failure consistently.

Acute intoxication with chloralose is generally well supported hemodynamically. In central nervous system, chloralose was sedative and causes hyperexcitability. The coma is almost constant. Respiratory signs are not specific and bronchial hypersecretion is the main symptom.

Shock was reported in the mass poisoning [3]. However, in our knowledge no cases of acute pulmonary edema have been reported in the literature. The absence of inhalation, the echocardiography results and elevated troponin I points to left ventricular dysfunction as the cause of pulmonary edema.

Several mechanisms may be involved in the pathogenesis of cardiac dysfunction following chloralose poisoning:

- Direct toxicity of chloralose on the heart and this toxic cardiomyopathy was reversible within few days.
- Myocardial stunning due to sudden emotional stress or Takotsubo Cardiomyopathy [4,5]. Three mechanisms might explain the association between sympathetic stimulation and myocardial stunning: ischemia resulting from epicardial coronary arterial spasm, microvascular spasm secondary to an abnormality in coronary flow or the presence of sympathetically mediated microcirculation dysfunction and direct myocyte injury.

The management of chloralose poisoning is symptomatic and its aims to maintain the vital functions integrity. Activated charcoal (50 g) is recommended during the two hours following the intoxication. The treatment of acute pulmonary edema without shock is based on nitrates and diuretics. In the absence of complications, the evolution was favorable. The mortality is around 0.4%.

Acute chloralose poisoning is mainly complicated by neurological signs. The cardiac toxicity remains scarce and potentially serious. Its mechanism is poorly understood.

References

[1] Richelme C, Duval G, Gérard J, Corbin JC, Chuet C. Voluntary chloralose poisoning. Cah Anesthesiol 1985;33:589–92.
[2] Dardaine V, Legras A, Lanotte R, Brasset N, Furet Y. Unrecognised chloralose poisoning. Intensive Care Med 1992;18:497.
[3] Clement S, Savy FP, Dumont D, Lachatre G, Froge E, Piva C. Le chloralose. Persistance des intoxications volontaires en milieu rural. Á propos de douze observations. J Med Leg Droit Med 1992;35:105–9.
[4] Lacy CR, Contrada RJ, Robbins ML, Tannenbaum AK, Moreyra AE, Chelton S, et al. Coronary vasoconstriction induced by mental stress (simulated public speaking). Am J Cardiol 1995;75:503–5.
[5] Kurisu S, Sato H, Kawagoe T, Ishihara M, Shimatani Y, Nishioka K, et al. Tako-tsubo-like left ventricular dysfunction with ST-segment elevation: a novel cardiac syndrome mimicking acute myocardial infarction. Am Heart J 2002;143(3):448–55.