Acute myocarditis complicating severe chloralose intoxication: A case report

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Abstract

Chloralose self- poisoning is frequent in Tunisia. Neurological signs are the most common. Cardiac toxicity is potentially serious and rarely reported. Its mechanism is not well known.
We report here a case of chloralose rodenticide voluntary intoxication complicated by myocarditis and an acute heart failure fife hours after admission. Echocardiography showed diffuse hypokinesia and decreased left ventricular ejection fraction. The evolution was favorable within five days.

Keywords: Intoxication; Chloralose; Acute Heart Failure; Myocarditis, Shock.

CASE REPORT

A 26-year-old woman, with no medical history, was admitted to the emergency department for a suicidal attempt. She had ingested one hour before hospitalization one sachet (4 grams) of chloralose rodenticide (4g) (Figure1).

On admission, her Glasgow coma scale score was 13/15. She had myoclonic jerks and pinpoint pupils. The respiratory rate was at 20 breaths per minute with pulse oximetry at 98 % in ambient air. There were no hemodynamic disorders (arterial blood pressure at 130/70 mm Hg and heart rate at 80 b/min). Within the first hour of admission, we performed an electrocardiogram and blood tests performed. They all were with no abnormalities.

Two hours after admission, both neurological and respiratory states were deteriorating, and we performed an urgent invasive mechanical ventilation.

After intubation, we indicated an abundant gastric lavage (18 liters) with the administration of activated charcoal (50g).

Three hours later, the patient developed bradycardia at 50 b/min and shock (blood pressure:70/55 mmHg). The control ECG showed an ST-segment depression in V2-V4 leads (Figure 2). Control blood test analyses have shown heart injury with lactic acidosis (Table 1). Chest X-ray bilateral pulmonary edema. Transthoracic echography showed acute heart failure with a left ventricular ejection fraction (LVFE) at 32% and diffuse hypokinesia.

A fluid replacement was administered (40ml/kg). Regarding persisting low blood pressure, with ECG and echography findings, we also decided to begin continuous intravenous perfusion of adrenaline (2.5mg/hour). The patient's respiratory and hemodynamic status have progressively improved.

Table1. Results of the biological tests

	Reference Range	On admission	At h-6 of hospitalization	At h-12 of hospitalization
Sodium, mmol/L	136-145	134	140	141
Potassium, mmol/L	3.5-5.1	3.3	3.7	4
Chloride, mmol/L	98-107	99	109	109
PH	**7.38-7.42**	**7.43**	**7.21**	**7.21**
PaCO2, mmHg	38-42	34	43	59
PaO2, mmHg	**≥80**	**82**	**181**	**86**
bicarbonates level, mmol/l	22-26	22.6	17.2	23.6
Base excess		-1.2	-10.3	-5
PaO2/FiO2		**390**	**362**	**172**
Creatinine, µmom/l	62-106	57	48	57
CPK, U/l	**26-192**	**65**	**234**	**2180**
Troponines, ng/ml	**<0.014**	**0.01**	**0.126**	**0.145**
Pro BNP, pg/mL	**<400**			**8240**
Lactate, mmol/L	**0.50-2.20**	**1.2**		**2.6**
Cholinesterase activity, U/l	**5320-12920**	**7308**		

Extubation had been successful five days after admission. The cardiac troponin level declined to 0.023, and the control echocardiography at day-6 day showed an improvement of the LVFE to 65%. She was discharged home without sequels.

DISCUSSION

Alpha chloralose is a rodenticide formed by condensing chloral with a pentose or hexose sugar. Formerly used medicinally for its sedative and hypnotic properties, chloralose is commonly used in pesticide phytosanitary preparations in the fight against rodents. The rate of chloralose poisoning is estimated to 1000 hospitalizations yearly in Tunisia (1).

Mortality rates are estimated to 0.4% (2, 3). The toxic or lethal threshold of chloralose in humans is difficult to state due to variable subject sensitivity. Richet has set the minimum active dose in adults at 0.004 g/kg, the toxic dose at 1 g, and the lethal dose at 0.1 g/kg, with large individual variations (4). The precocity of clinical signs seems to be inversely proportional to the dose. The severity of the clinical manifestations depends on the ingested dose and the product concentration in chloralose (5).

The toxidrome consists namely of neurological signs. In this type of intoxication, hemodynamic tolerance is usually good. Collapse circulatory system with low central venous pressure and shock have been described in massive intoxication (6, 7). Cardiac toxicity is a life-threatening presentation and is rare. The mechanisms of this cardiac toxicity are not yet well established. Moderate sinus tachycardia was consistently found. Arrhythmias have also been reported. These ECG abnormalities have often been explained by electrolytes' disturbances, acid-base balance due to cellular anoxia, as well (6, 8).

Several mechanisms explaining the direct cardiac dysfunction in chloralose poisoning worth to be mentioned. First, a transient negative inotropic effect was described the first few minutes after intoxication. This effect is often masked by early increased heart flow and rate, secondary to stimulating endogen catecholamines. This adaptation mechanism is transient (3, 9). Second,

chloralose has direct toxicity on the cardiomyocytes, which is usually reversible within a few days (1). Third, sudden emotional stress, also named Takotsubo cardiomyopathy, induces myocardial stunning (10, 11). That induces cardiac ischemia, via three pathways: 1) epicardial coronary arterial spasm; 2) alteration of the coronary flow (via microvascular spasm, or sympathetic mediated microcirculation dysfunction); 3) and direct myocyte injury (1). In our case, the echocardiography findings were evoking a cardiac dysfunction due to intoxication.

The prognosis depends on the early management after ingestion; including gastric decontamination, and symptomatic procedures (3). Intubation and mechanical ventilation are required in case of neurologic and/or respiratory distress. The restlessness, myoclonus, and seizures are usually improving by benzodiazepines. Fluid replacement is commonly sufficient to restore blood pressure. The rare cases of sustaining cardiovascular collapse are successfully managed with vasoactive agents, as in our example (3, 12). The gastric decontamination has to be early achieved. Gastric lavage is still performed in some countries (13, 14). A single dose (50 g) administration of activated charcoal is nowadays more recommended and safer, within two hours of ingestion and in the absence of contraindications (3, 15).

CONCLUSION

Cardiac toxicity following acute chloralose poisoning remains a life-threatening condition. Its mechanism is still poorly understood. Direct cardiac toxicity has to be evoked in case of electrocardiogram abnormality, hemodynamic disorders, troponin elevation, or chest x-ray abnormality. Echography confirms the diagnosis. This cardiomyopathy is usually reversible in a few days when management is early.

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