Acute myocarditis complicating severe chloralose intoxication: A case report

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Abstract

Background: 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.

Case report: We report here a case of chloralose rodenticide voluntary intoxication complicated by myocarditis and an acute heart failure five hours after admission. Echocardiography showed diffuse hypokinesia and decreased left ventricular ejection fraction. The evolution was favorable within five days.

Why should an emergency physician be aware of this? Cardiac toxicity following acute chloralose poisoning remains a life-threatening condition. This cardiomyopathy is usually reversible in a few days when management is early.

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

INTRODUCTION

Alpha chloralose is a rodenticide commonly used in pesticide phytosanitary preparations in the fight against rodents. The toxic or lethal threshold of chloralose in humans is difficult to state due to variable subject sensitivity. The rate of chloralose poisoning is estimated to be 1000 hospitalizations yearly in Tunisia [1]. The toxidrome consists namely of neurological signs. Cardiac toxicity is a life-threatening presentation and is rare. The mechanisms of this cardiac toxicity are not yet well established. We report a case of acute myocarditis secondary to chloralose intoxication.

CASE PRESENTATION

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).

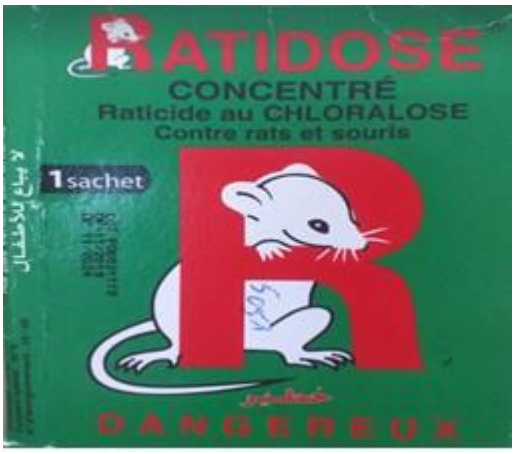

Figure 1: Ingested chloralose

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 had no abnormalities.

Two hours after admission, both neurological and respiratory states were deteriorating, and we performed 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).

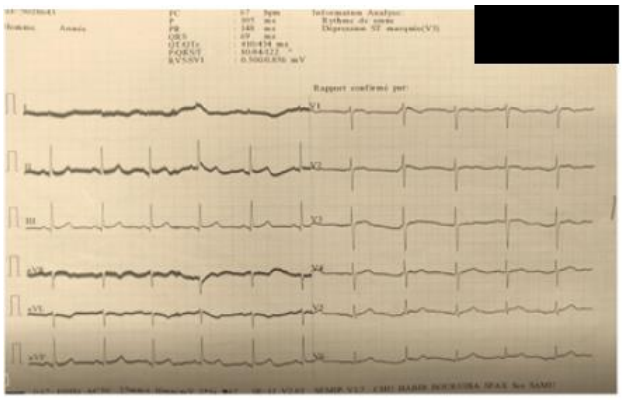

Figure 2: The control ECG showing an ST-segment depression in V2-V4 leads

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 has progressively improved. Extubation had been successful five days after admission. The cardiac troponin level declined to 0.023, and the control echocardiography on day 6 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.

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	139	138	136-145
Potassium, mmol/L	3.5-5.1	3.7	4.3	3.5-5.1
Chloride, mmol/L	98-107	100	105	98-107
PH	7.38-7.42	7.28	7.38	7.38-7.42
PaCO2, mmHg	38-42	36	36	38-42
PaO2, mmHg	≥80	87	82	≥80
bicarbonates level, mmol/l	22-26	16	23.6	22-26
Base excess		-8.6	0.3	
Creatinine, µmom/l	62-106	24	22	62-106
Alanine transaminase, IU/L	10-41	32	37.7	10-41
Aspartate transaminase, IU/L	10-37	34	36.8	10-37
Lactate, mmol/L	0.50-2.20	0.68	0.79	0.50-2.20
Lipase, U/L	13-60	247	49	13-60

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 be 1000 hospitalizations yearly in Tunisia [1].

Mortality rates are estimated to be 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 [3,4]. The precocity of clinical signs seems to be proportional to the dose. The severity of the clinical manifestations depends on the ingested dose and the product concentration in chloralose[3,5].

The toxidrome consists namely of neurological signs. In this type of intoxication, hemodynamic tolerance is usually good. A collapsed circulatory system with low central venous pressure and shock has been described as 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 electrolyte disturbances, and acid-base balance troubles due to cellular anoxia, as well [6, 8].

Several mechanisms explaining the direct cardiac dysfunction in chloralose poisoning are worth to be mentioned. First, a transient negative inotropic effect was described in 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 evoked 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 improved 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 case [3, 12]. The gastric decontamination has to be early achieved. Gastric lavage is still performed in some countries [13]. 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, 14].

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.

REFERENCES

1. *Fekih Hassen M., Dalla Ayed S., Ben Sik Ali H., Gharbi R., Elatrous S. Acute heart failure following severe chloralose poisoning: A case report. Egyptian Journal of Anaesthesia. 2013 ; 29 ; 87–88. Doi : http://dx.doi.org/10.1016/j.egja.2012.05.003*
2. *Kouraichi N, Brahmi N, Bouzidi H, et al. Intoxication aiguë par le chloralose. Étude de la corrélation entre la dose ingérée et le tableau neurologique. Reanimation 2006;15(Suppl. 1):S128 [abstract].*
3. *Kouraichi N., Brahmi N., Elghord H., Béji O., Thabetb H., Amamou M. Chloralose poisoning: Prognostic factors and management. Réanim. 2010: 19; 581-6. DOI: https://doi.org/10.1016/j.reaurg.2010.05.014*
4. *Richet C. L'anesthésie générale par le chloralose. CM Acad Sci. 1918; 166: 1026-33. [cross reference]*
5. *Shimokawa A, Kunitake T, Takasaki M, Kannan H. Differential effects of anesthetics on sympathetic nerve activity and arterial baroreceptor reflex in chronically instrumented rats. J Auton Nerv Syst. 1998 ;72:46-54. doi: 10.1016/s0165-1838(98)00084-8.*
6. *Hamouda C, Amamou M, Jedidi S, Hedhili A, Ben Salah N, Thabet H. Graded classification of acute chloralose poisoning. 509 cases. Press Med 2001; 30: 1055-8.*
7. *Zhioua M, Kouraichi N, Makhloufi M, Benzarti A, Riahi F, Hamdoun M. Acute chloralose poisoning: 752 observations. Journal de Médecine Légale Droit Médical 51:343-356.*
8. *ederici S, Claudet I, Laporte-Turpin E, Marcoux M.O, Cheuret E, Maréchal K. Severe chloralose intoxication in a toddler. Arch Pediatr. 2006; 13: 364-6. doi: 10.1016/j.arcped.2006.01.014*
9. *Dardaine V, Legras A, Lanotte R, Brasset N, Furet Y. Unrecognised chloralose poisoning. Intensive Care Med. 1992; 18:497. Doi: doi: 10.1007/BF01708591.*
10. *acy 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. Doi: 10.1016/s0002-9149(99)80590-6.*
11. *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; 143:448–55. Doi: 10.1067/mhj.2002.120403.

12. Landier C, Kaskes S. État de choc lors d'une intoxication par le chloralose. Rev SAMU 1995;2:54—5.

13. Touil Yosr., Abidi A., Daly F., Amri S., Ben Amor G., Trifi A., et al. Cardiomyopathy Following Chloralose Intoxication: A Case Report. EC Emergency Medicine and Critical Care. 2019: 101-104. Available on: https://www.ecronicon.com/ecec/pdf/ECEC-03-00041.pdf.

14. Harry P. Pratique de l'administration du charbon active en toxicologie aiguë. Reanim Urgence 1993;2:210—4.