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Case Report

Organophosphate poisoning presenting as out-of-hospital cardiac arrest: A clinical challenge

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ABSTRACT

Out-of-hospital cardiac arrest (OHCA) remains a challenge for physicians since effective management and definitive salvage depend upon correct determination of the etiology and the extent of injury. Definitive diagnosis of organophosphate poisoning (OP) requires physicians' clinical awareness of a typical toxidrome, that is, characteristic signs and symptoms of poisoning, and laboratory confirmation. Here we report a case of an OHCA patient with OP, which was initially misdiagnosed as an acute ST segment elevation myocardial infarction based on the patient's medical history and clinical manifestations.

<**Learning objective:** Organophospate poisoning is associated with an increasing mortality with widely used pesticides in the developing world. Differential diagnosis of out-of-hospital cardiac arrest should include such etiology that can be reversed by early intervention.>

Introduction

Studies show that the incidence and survival rate of out-of-hospital cardiac arrest (OHCA) vary both regionally and globally [1]. Effective OHCA management with appropriate intervention depends upon determining the etiology and extent of injury. Among patients with OHCA, cardiovascular events account for the majority of deaths, while coronary artery disease (CAD) is often present even in the absence of an acute ST segment elevation myocardial infarction (STEMI). The presence of ventricular fibrillation or pulseless ventricular tachycardia is also highly associated with CAD. Several studies have also investigated the prevalence of non-cardiac etiology of OHCA and reported trends that have occurred in various populations [2]. Park et al.[3] described the epidemiologic features and outcomes found in the Korean emergent medical system and reported that poison-induced OHCA was responsible for 4.4% of non-cardiac etiology OHCAs. However, heterogeneity of poison-induced OHCA makes it especially difficult to differentiate the underlying mechanisms responsible for each type of poisonous substance. Here we report a 60-year-old OHCA male patient with a history of undergoing coronary intervention for triple-vessel disease, whose cardiac status on admission gave the initial impression of new-onset STEMI. However, an emergency coronary angiogram showed negative results and laboratory examination indicated organophosphate poisoning (OP). We suggest, therefore, that poisoning, especially OP, should be considered in the differential diagnosis of OHCA.

Case report

A 60-year-old man with a history of triple coronary disease and stent implantation was found unconscious and unresponsive in the park for an unknown amount of time and was brought to our hospital at midnight by emergency medical technicians. There were no witnesses to his collapse and no suspicious bottles,

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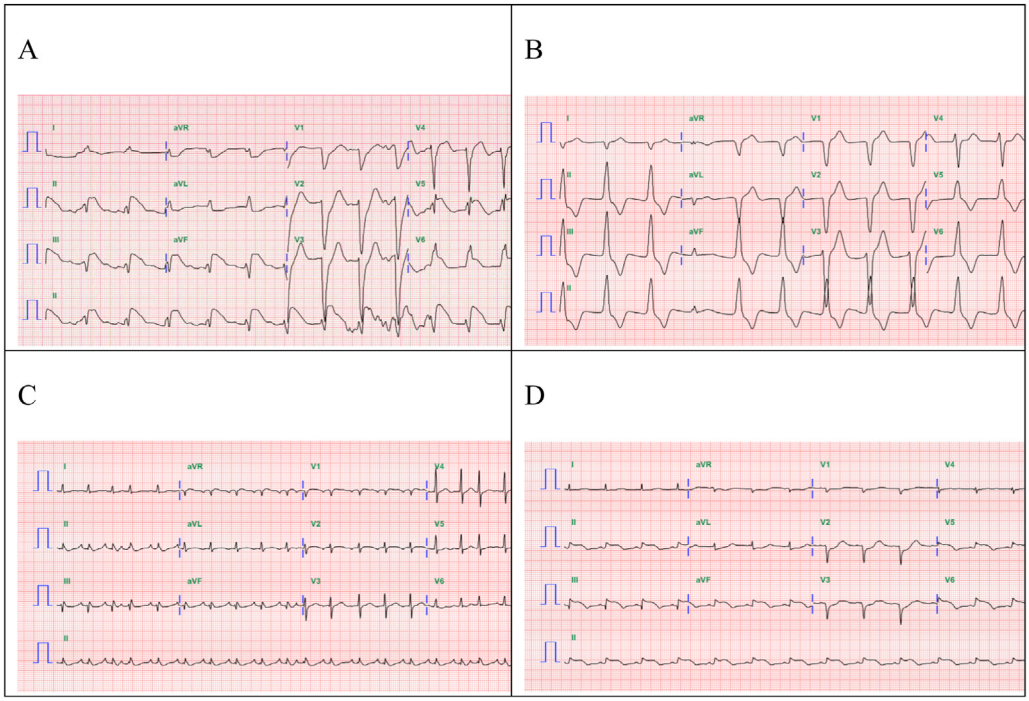

Fig 1. (A) The initial 12-lead electrocardiogram (ECG) at emergency room arrival disclosed wide QRS rhythm and infero-lateral wall ST elevation. (B) After admission to cardiac care unit, accelerated idioventricular rhythm was noticed after administering atropine and pralidoxime (Day 1). (C) After receiving 3 ampules of atropine infusion, the 12-lead ECG then showed sinus tachycardia (Day 2). (D) On the second night of hospitalization, the 12-lead ECG showed infero-lateral wall ST segment elevation.

containers, knives, or liquid spills were found near him. Initial arterial blood gas studies revealed severe acidemia, metabolic and respiratory acidosis (pH 6.9, $PaCO_2$ 103.5 mmHg, HCO_3• 19.9 and base excess −15.5), and severe hypoxemia (PaO_2 28.8 mmHg). Cardiopulmonary resuscitation and cardiac defibrillation were implemented and the patient regained pulses after 13 min of these attempts to resuscitate. The initial 12-lead electrocardiogram (ECG) showed wide QRS rhythm and infero-lateral ST segment elevation (Fig. 1A), raising high suspicion of OHCA due to STEMI. However, emergent coronary angiogram showed no obvious stenotic artery or thrombosis (Fig. 2). Clinical laboratory tests on the patient's blood sample drawn when he arrived at the hospital revealed no significantly elevated cardiac enzymes [hs-TnT: 0.015 ng/ml (<0.1)] suggestive of myocardial infarct. Routine urinalysis was performed as well as urine screening for illicit drugs, and all results were negative.

The patient was admitted to the cardiac care unit with vital signs as follows: body temperature 31.2 °C, pulse 58/min, respiratory rate 16/min under intubation and mechanical ventilation, and blood pressure 86/43 mmHg. The fluid challenge technique was employed, and he was given dopamine infusion (13.6 mcg/kg/min) and warm coverings but shock status and hypothermia were still evident. We noticed that he had a large amount of sweat that drenched his whole body. He also had massive tears, saliva, and naso-gastric tube aspiration with the smell of organic-solvent. Pin-point pupils (bilateral 1 mm) with poor light reflexes were also found. Because of these abnormal physical findings as well as high anion gap metabolic acidosis, we strongly suspected cholinergic toxidrome, which we considered to possibly be associated with organophosphates or carbamyl agent intoxication. Additional blood tests revealed low cholinesterase activity, which corresponded clinically with organophosphate and carbamate intoxication (Table 1). We then administered continuous infusion of atropine 1 mg/h and pralidoxime 500 mg/h. The subsequent 12-lead ECG showed accelerated idioventricular rhythm (Fig. 1B). After receiving 3 ampules of atropine infusion, the 12-lead ECG then showed sinus tachycardia (Fig. 1C), and the patient's pupil size returned to 3 mm bilaterally with positive light reflexes. His overall physical status

Table 1 Detailed laboratory data during hospitalization. Reference range of CHE: >5000 U, AchE activity >20 μmol/s/L. CHE: cholinesterase; AchE: acetylcholinesterase; CK: creatine kinase; QTc: corrected QT interval.

		Day 1 09:00	Day 1 21:00	Day 2 09:00	Day 2 21:00	Day 3 09:00	Day 3 21:00	Day 4 09:00	Day 4 21:00
CK (U/L)		1571	1493	1189	793	1123	1016		
CK-MB (ng/ml)		237	268	177	47.6	120	104		
RBC AchE activity (μmol/s/L)		2.1	10.8	17.5	15.1	12.1	14.1	13.8	19.3
Pseudo CHE activity (μmol/s/L)		0.02	0.3	0.02	0.01	1.8	0.6	0.1	2.0
CHE (U/L)		<200.0							
Infusion rate	Atropine (mg/h)	1		0.5		1		1	
	Pralidoxime (mg/h)	500		500		500		500	
QTc		587 (D1-11:55)	539 (D1-23:08)	478 (D2-09:45)					