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A Case of Takotsubo-Like Cardiomyopathy Induced By Methomyl (Organophosphate) Poisoning

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Abstract: An 82-year-old man intentionally ingested methomyl after arguing with his son. He had a history of alcoholism. Upon arrival, his vital signs were as follows: Glasgow Coma Scale, E1V1M4; blood pressure, 214/94 mmHg; pulse rate, and 116 beats per minute. The physiological findings included fasciculation of the eyelids and moist rales at the right lung. Electrocardiography (ECG) showed sinus tachycardia. He was admitted to the intensive care unit with a diagnosis of organophosphate poisoning and aspiration pneumonia. ECG on the 2nd hospital day showed negative T-waves at the precordial and limb leads with prolongation of the QT correction interval. Echocardiography revealed akinesis of the apical portion of the left ventricle with compensatory hyperkinesis of the basal walls, suggesting Takotsubo-like cardiomyopathy. His cardiac motion and abnormal electrocardiography findings finally improved. Although the pathogenesis of Takotsubo-like cardiomyopathy is not clear at present, the cardiotoxicity induced by methomyl of itself and/or catecholamine-induced microvascular spasms may result in the occurrence of Takotsubo-like cardiomyopathy.

Keywords: Takotsubo cardiomyopathy; organophosphate poisoning; cardiotoxicity; catecholamine-induced microvascular spasms.

INTRODUCTION

Methomyl is an organophosphate insecticide. Methomyl is placed in EPA Toxicity Category I (the highest toxicity category out of four) based on acute toxicity testing in studies that have investigated exposure via the oral route and eye irritation [1]. In Japan, there were 533 fatal cases of methomyl poisoning between 1997 and 2003[2]. Takotsubo cardiomyopathy is a disorder characterized by a transient dysfunction of the apical portion of the left ventricle with compensatory hyperkinesis of the basal walls that produces ballooning of the apex with systole in the absence of coronary artery disease [3]. The most remarkable clinical characteristics of Takotsubo cardiomyopathy are that the disease is much more common in females than in males and that it is more common in females over 60 years of age than in females younger than 60 years of age [3]. In addition, the onset of Takotsubo cardiomyopathy is associated with preceding acute medical illness and emotional or physical stress [3]. No cases of organophosphate poisoning-induced Takotsubo cardiomyopathy have been reported. We herein report the case of a patient with methomyl poisoning complicating Takotsubo-like cardiomyopathy.

CASE REPORT

An 82-year-old man intentionally ingested methomyl after arguing with his son. When the doctor helicopter staff members checked him, he was comatose and respiratory secretion was observed; he was therefore intubated at the scene and was transported to our department by ambulance. Traces of vomit were also observed in the patient's home. He had a history of alcoholism. Upon arrival, his vital signs were as follows: Glasgow Coma Scale, E1V1M4; blood pressure, 214/94 mmHg; pulse rate, 116 beats per minute; and peripheral oxygen saturation on 10 liters of oxygen per minute with a reservoir mask, 90%. His pupils were 2 mm in size with a prompt light reflex. The physiological findings included fasciculation of the eyelids and moist rales at the right lung. Chest roentgenography showed a cloudy right lung field. Electrocardiography (ECG) showed sinus tachycardia with monofocal premature ventricular contraction (Figure 1). A complete blood count revealed the following: white blood cells, 12,800/mm^3; hemoglobin, 13.3 g/dl; and platelets, 25.0 x10^4/mm^3. Serum biochemistry revealed decreased levels of cholinesterase (41 [normal range; 180-450] IU/L). The patient was treated with cathartic and activated charcoal after gastric lavage. He was admitted to the intensive care unit with a diagnosis of organophosphate poisoning

and aspiration pneumonia requiring Sulbactam/Ampicillin. ECG on the 2nd hospital day showed negative T-waves at the precordial and limb leads with prolongation of the QT correction interval (536 ms) (Figure 2). Echocardiography revealed akinesis of the apical portion of the left ventricle with compensatory hyperkinesis of the basal walls, suggesting Takotsubo-like cardiomyopathy (Figure 3). The ejection fraction (EF) was 48%. His circulation was stable. His respiratory function improved and extubation was performed on the 4th day. However his condition was complicated with chemical pneumonia and his respiratory function deteriorated to the point that he required temporary nasal high flow oxygen therapy. His cardiac motion and abnormal electrocardiography findings improved. He discharged on foot on 26th hospital day, after the improvement of cardiorespiratory function.

The electrocardiogram shows sinus tachycardia with monofocal premature ventricular contraction.

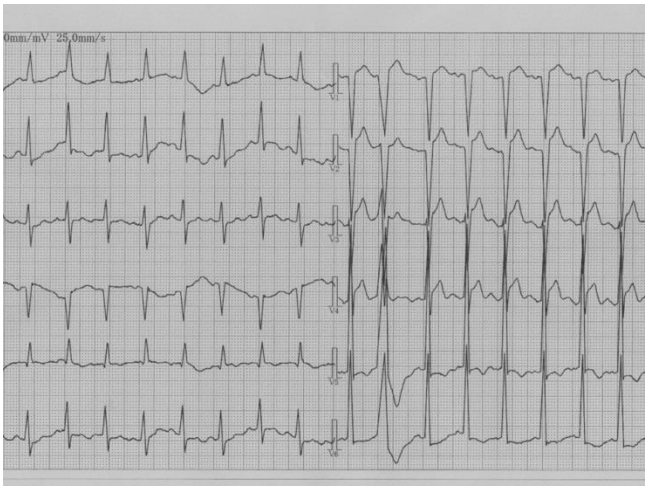

Fig-1: Electrocardiography on arrival

The electrocardiogram shows negative T-waves at the precordial and limb leads with prolongation of the QT correction interval (536 ms) (Fig-1).

Echocardiography revealed akinesis of the apical portion of the left ventricle with compensatory hyperkinesis of the basal walls, suggesting Takotsubo-like cardiomyopathy (Fig-2).

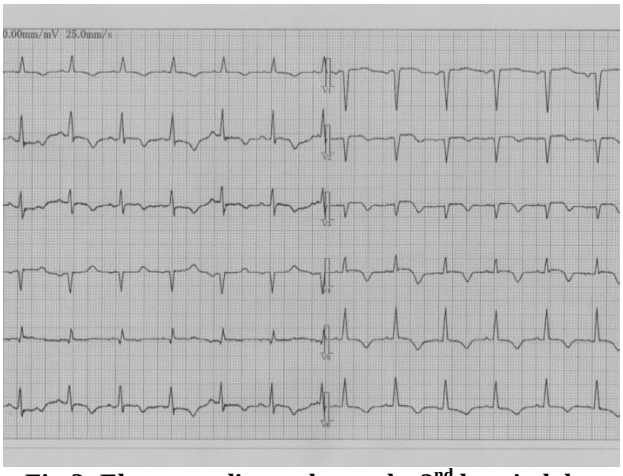

Fig-2: Electrocardiography on the 2nd hospital day

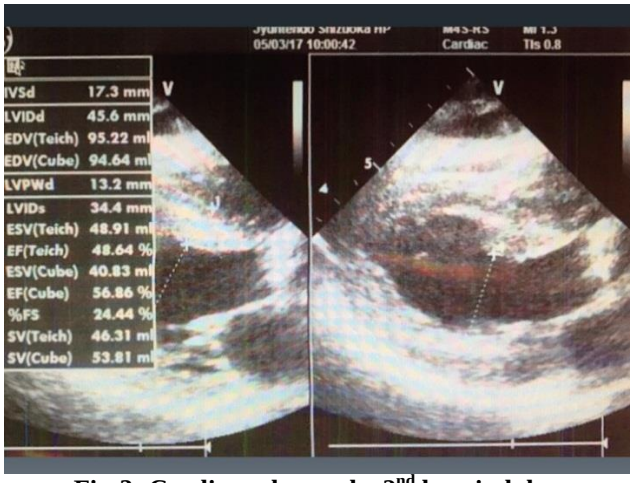

Fig-3: Cardiac echo on the 2nd hospital day

DISCUSSION

This is the first case of organophosphate poisoning with Takotsubo-like cardiomyopathy. This combination has not been previously reported. We diagnosed the patient with Takotsubo-like cardiomyopathy based on the transient abnormalities in cardiac wall motion, elevated cardiac enzyme levels and ECG findings that were suggestive of Takotsubo-like cardiomyopathy without significant coronary disease.

Lee *et al*. reported on the cardiotoxicity of methomyl [4]. They reviewed 14 consecutive cases of methomyl poisoning. An ECG analysis revealed ST depression and T-wave inversion in five patients (35.7%) and one patient (7.1%), respectively. A cardiac biochemical marker analysis revealed initial TnI was elevation in 11 patients (78.6%). Cardiac echo was performed in 9 of the 11 TnI-positive patients. Among them, three patients (33.3%) showed a reduced ejection fraction (EF), and regional wall motion abnormalities were noted in two patients. However, Takotsubo-like movement was not described. One patient expired due to pneumonia, and one patient was transferred in a moribund state. They followed up 12 patients who survived to discharge for 6-44 months. One patient (8.3%) died during the follow-up period, and 11 survived without any further complications. They concluded that methomyl exposure could cause direct myocardial injury and reversible cardiac dysfunction. Lee *et al*. reported the cardiotoxicity of methomyl in comparison to patients poisoned with other Class I organophosphates [5]. In the methomyl group, 7 of 17 patients suffered cardiogenic cardiac arrest, three died from multiple organ dysfunction syndromes after resuscitation from cardiac arrest. In the Class I organophosphate group, 4 of 42 patients died from pneumonia and complicating acute respiratory distress syndrome. Accordingly, the cardiotoxicity of methomyl may cause temporal Takotsubo-like cardiomyopathy.

In organophosphate poisoning, the activation of the vagus nerve system (muscarine reaction), which is characterized by symptoms such as visual disturbance, bradycardia, hypotension, wheezing due to bronchoconstriction, increased bronchial secretion, increased salivation, lacrimation, sweating, peristalsis, diarrhea and urination, is one of main problems. The nicotine reaction, which has effects such as fasciculation followed by muscle paralysis, is also important in organophosphate poisoning. In addition, the nicotine reaction causes the activation of the sympathetic nervous system. Previous reports revealed that the temporal over activation of the sympathetic nervous system was induced by organophosphate poisoning, both experimentally and clinically [6-8]. The present case showed hypertension and tachycardia, which suggested the activation of the sympathetic nervous system. Accordingly, the patient's Takotsubo-like cardiomyopathy might have been induced by the over activation of the sympathetic nervous system by organophosphate poisoning.

CONCLUSION

We reported the first case of Takotsubo-like cardiomyopathy induced by methomyl (organophosphate) poisoning. Although the pathogenesis of Takotsubo-like cardiomyopathy is not clear at present, the cardiotoxicity induced by methomyl of itself and/or catecholamine-induced microvascular spasms may result in the occurrence of Takotsubo-like cardiomyopathy.

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