Pyrethroid insecticide-induced takotsubo syndrome

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Introduction

Pyrethroid insecticides are a synthetic formulation of naturally occurring pyrethrins, which are extracted from the flowers of chrysanthemum cinerariaefolium. Pyrethroids make up the majority of common insecticides used against household pests such as mosquitoes, house flies, bed bugs, and cockroaches (1).

Takotsubo syndrome is described as a reversible left ventricular (LV) variable wall motion abnormality that occurs in the absence of coronary artery stenosis or acute plaque rupture on angiography (2). Emotional trauma and some medical conditions, such as acute respiratory failure, pancreatitis, cholecystitis, pneumothorax, thyrotoxicosis, pregnancy, carbon-monoxide poisoning, exposure to sympathomimetic drugs, stroke, and intracerebral hemorrhage, have been implicated as triggers of takotsubo syndrome (2). Postmenopausal women are commonly affected by this syndrome.

In this article, we report a case of a 96-year-old woman who developed takotsubo syndrome after an accidental exposure to a large amount of pyrethroid insecticide (deltamethrin) via inhalation in a closed space.

Case Report

A 96-year-old woman was admitted to the emergency department with complaints of substernal chest pain and dyspnea. The pain started few minutes after intense exposure to the pyrethroid (deltamethrin) when spraying the insecticide in a closed space. Shortness of breath began shortly after chest pain. Her past medical history indicated only hypertension, for which she was taking a combination of candesartan and hydrochlorothiazide. Examination revealed a tachypneic, orthopneic elderly woman. She had a respiratory rate of 30 breaths per minute, peripheral oxygen saturation of 86% on room air, a regular pulse rate of 100 beats per minute, and a blood pressure of 160/100 mm Hg. Laboratory workup revealed a hemoglobin level of 13 g/dL, a hematocrit level of 40%, white blood cell count of 9.2×10^9/L, platelet count of 310×10^9/L, creatinine level of 1.2 mg/dL, and C-reactive protein level of 5.9 mg/L (normal range, 0–8 mg/L). An initial cardiac panel demonstrated a mildly elevated troponin T level at 19.2 pg/mL (normal range, 0–14 pg/mL) and a normal creatine kinase myocardial band (CK-MB) mass level of 1.3 ng/mL (normal range, 0–4.88 ng/mL), However, the troponin T and CK-MB mass levels peaked to 791.7 pg/mL and 19.6 ng/mL, respectively. Electrocardiogram (ECG) showed sinus rhythm with a ventricular rate of 98 beats per minute and anterior ST segment elevation myocardial infarction appearance (Fig. 1a). Transthoracic echocardiogram showed akinesis of the apical, midventricular segments, and ballooning of the apical segment with reduced ejection fraction of 35% (Fig. 2a, 2b; Video 1). A diagnosis of ST segment elevation myocardial infarction was made and the patient underwent a coronary angiography for primary percutaneous coronary intervention. Oxygen, nitroglycerine, acetylsalicylic acid, ticagrelor, and furosemide therapy were administered to the patient in the emergency department. Her coronary angiogram revealed no epicardial coronary artery stenosis or evidence of plaque rupture (Fig. 3a–3c; Videos 2–4). Because of the absence of stenosis on coronary angiography, the presence of ST elevation in leads V1–V6, DI, and augmented vector left (aVL) on electrocardiography, and ballooning of the apical segment on echocardiography, the patient was diagnosed with takotsubo syndrome and treated conservatively. She was discharged home in stable condition on Day 6 after hospital admission with acetylsalicylic acid, metoprolol, candesartan plus hydrochlorothiazide, and furosemide treatment. One week after discharge, the patient returned to our outpatient clinic for follow up. An echocardiogram showed significant improvement of the ejection fraction to 65% and a disappearance of the apical bal-

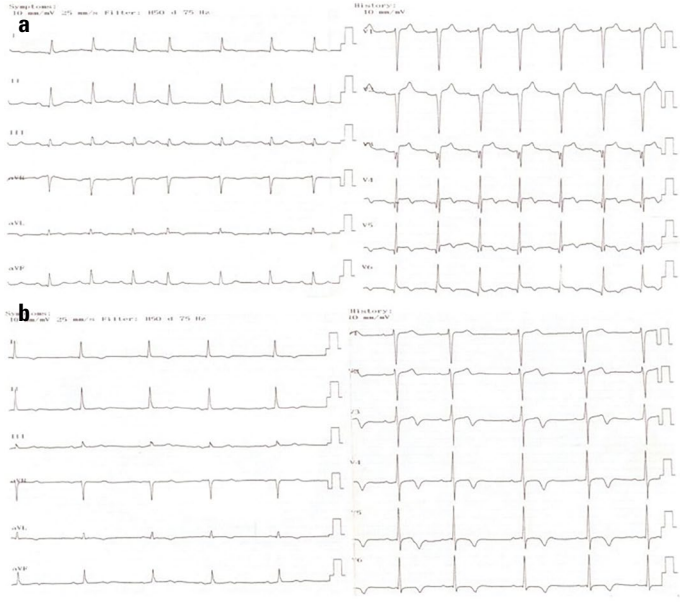

Figure 1. Twelve-lead electrocardiogram on admission showing ST-segment elevation in leads V1–V6, DI, and aVL with no reciprocal ST-segment depression (a). One week after discharge, biphasic T in V3–V4 and T negativity in V5–V6, DI, and aVL leads were present (b)

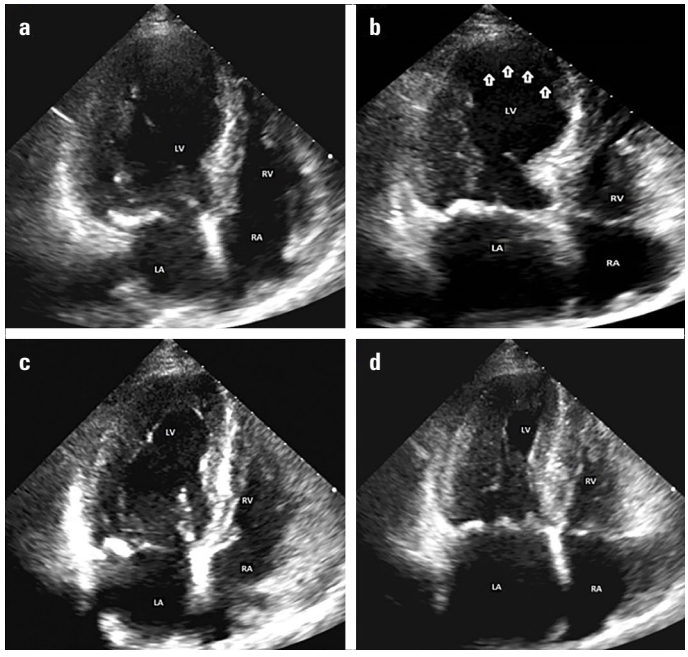

Figure 2. Transthoracic echocardiogram (apical 4 chamber view) on admission showing apical ballooning of the left ventricle in diastole (a) and systole (b). One week after discharge, a repeated echocardiogram showed disappearance of the apical ballooning in diastole (c) and systole (d). LV, left ventricle; RV, right ventricle; LA, left atrium; RA, right atrium

looning in accordance with the course of takotsubo syndrome (Fig. 2c, 2d; Video 5). A repeated electrocardiogram also showed biphasic T in leads V3–V4 and T negativity in the V5–V6, DI, and aVL leads (Fig. 1b). Furosemide treatment was discontinued.

Discussion

Takotsubo syndrome is defined by chest pain, elevated cardiac enzymes, and ST elevations resembling acute coronary syndrome with no culprit coronary artery stenosis (2). Although takotsubo syndrome is a reversible condition, one-fifth of takotsubo syndrome patients exhibit hemodynamic and electrical instability during the acute phase (3).

Diagnostic workup of takotsubo syndrome includes an ECG, cardiac biomarkers, echocardiography, and coronary angiography (4). The initial ECG abnormalities typically include ST segment elevation, T-wave inversion, or both. Resolution of initial ST segment elevation is followed by progressive T-wave inversion and QT interval prolongation over several days, as in our case. Peak values of troponin and CK-MB are substantially lower compared with classical acute coronary syndrome. Our patient also had mildly elevated cardiac troponin and CK-MB peak levels consistent with takotsubo syndrome. Typical findings on transthoracic echocardiography include a large area of regional wall motion akinesia of the LV extending beyond the distribution of a single coronary artery territory. Apical (typical), midventricular, basal, and focal (atypical) forms of takotsubo syndrome have been described according to the segments that are involved. In our case, apical ballooning of the LV with normal basal contractility was observed on admission. Regional ventricular wall motion abnormality has been proposed to be transient as a diagnostic confirmation criteria for takotsubo syndrome. Our patient had resolution of cardiac function with full recovery by 1 week after discharge. Final differential diagnosis to distinguish takotsubo syndrome from acute coronary syndrome requires a coronary angiogram demonstrating perfusion-contraction mismatch. In our case, there was an absence of obstructive coronary disease and no angiographic evidence of acute plaque rupture. Lastly, myocarditis must be excluded (4). Our patient demonstrated no signs and symptoms of viral infection, elevated C-reactive protein levels, or pericardial effusion, confirming the diagnosis of takotsubo syndrome.

Pyrethroid insecticides do little harm to humans because they are rapidly metabolized into nontoxic metabolites (1). Despite their low toxicity to humans, however, they can lead to toxicity in humans in rare cases. Pyrethroid insecticide toxicity can affect the gastrointestinal system, central nervous system, cardiopulmonary system, and renal system (5). Poisoning due to pyrethroid insec-

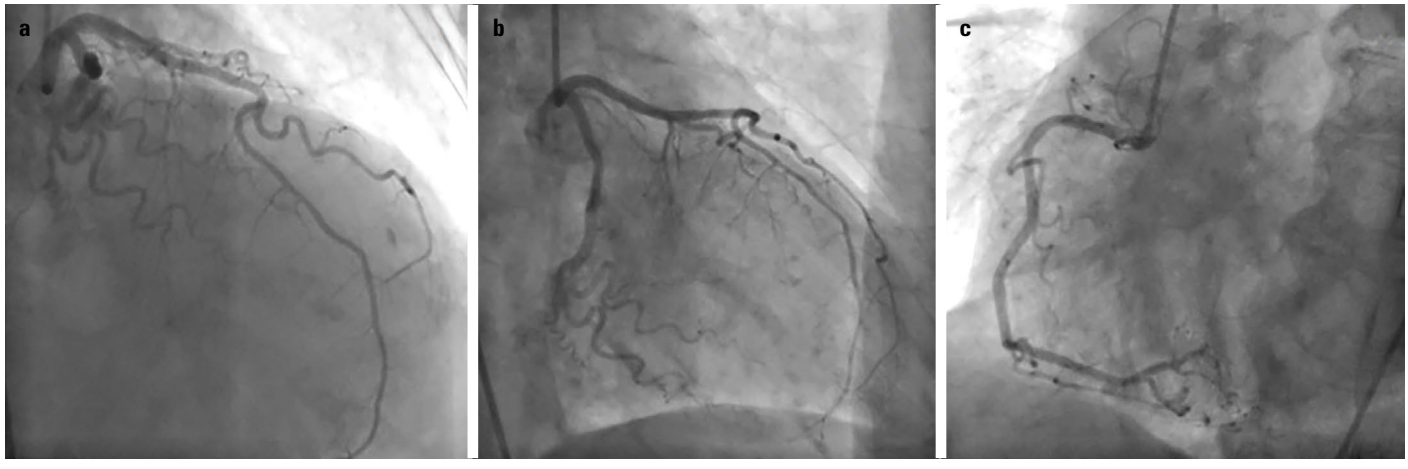

Figure 3. Coronary angiogram showing normal left anterior descending (a), circumflex (b), and right coronary arteries (c)

ticides can occur as a result of dermal exposure, inhalation, or ingestion (1). Inhalational poisoning can occur when pyrethroid insecticides are sprayed in closed spaces, as with our case.

Although the exact pathophysiological mechanism of takotsubo syndrome is not known, there is evidence that takotsubo syndrome is caused by a catecholamine increase and enhanced sympathetic stimulation (2). Catecholamine increase has also been thought to be the mechanism of pyrethroid insecticide toxicity (6). It has been shown that pyrethroid insecticide (deltamethrin) increases catecholamine release (6-8). To date, only 2 cases of pyrethroid insecticide (cypermethrin)-induced takotsubo syndrome have been reported in the literature (9, 10). Together with our case, these cases raise the possibility that takotsubo syndrome after pyrethroid insecticide exposure may occur because of its effect in increasing catecholamine secretion.

Conclusion

Takotsubo syndrome should be considered after acute coronary syndrome is excluded in elderly patients presenting with acute chest pain after exposure to a high amount of insecticide via inhalation, especially in a closed spaces. In conclusion, our case demonstrates that takotsubo syndrome may occur with pyrethroid insecticide poisoning.

Informed consent: Written informed consent was obtained from the patient.

Video 1. Transthoracic echocardiogram (apical 4 chamber view) on admission showing apical ballooning of the left ventricle in diastole (A) and systole.

Video 2. Coronary angiogram showing normal left anterior descending and circumflex coronary arteries.

Video 3. Coronary angiogram showing normal left anterior descending coronary arteries.

Video 4. Coronary angiogram showing normal right coronary arteries.

Video 5. Transthoracic echocardiogram showing significant improvement of the ejection fraction to 65% and disappearance of the apical ballooning

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