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Case Report

Acute Pulmonary Edema following Panic Attack in a Patient without Evidence of Heart Disease

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ABSTRACT

A 35-year-old woman was admitted because of organophosphate pesticide self-poisoning. At the time of admission in the emergency department of clinical toxicology, she was agitated. Evaluation of vital signs revealed a pulse rate of 148/min, systolic/diastolic blood pressure of 109/88 mm Hg, and respiratory rate of 20/min. She was afebrile and had plenty of oral secretions. Her pupils were mydriatic and reactive to light. Examination of the chest showed bilateral rales. Other organs revealed no pathologic sign or symptoms on physical examination. Computed tomography scan of the brain was normal. Serum cholinesterase level was 5%, and red-cell acetylcholinesterase activity was 0.3. She had no premorbid illness. After the injection of 4 mg of atropine, all muscarinic signs disappeared. This was followed by the infusion of atropine at a rate of 0.5 mg/h; the dose was titrated as per her clinical response and signs of atropinization. Over the next 2 days, she did not need further atropine. On day 4 after admission (i.e., after she had not need any atropine infusion or other treatments for organophosphate poisoning for 2 days), she suddenly developed hypertension crisis with systolic/diastolic blood pressure of 230/150 mmHg, cold sweating, tachycardia, and tachypnea. Chest examination revealed basilar wet rales. Chest X-ray presented diffuse bilateral alveolar infiltration. ECG was normal. Considering the clinical diagnosis of acute cardiogenic pulmonary edema, we started an intravenous infusion of nitroglycerin and furosemide and an intravenous injection of morphine. Twelve hours later, blood pressure was controlled and the rales disappeared. Bedside echocardiography showed normal left ventricular systolic and diastolic functions and normal right ventricular size and function. There was no significant valvular heart disease. Psychiatric consultation confirmed anxiety disorder and panic attack. Treatment with fluoxetine and clonazepam was commenced. During the course of her hospital stay and after her hospital discharge, outpatient follow-up showed no hypertension crisis. We conclude that panic attack and its hypertensive crisis may be severe enough to develop pulmonary edema even in young healthy adults with no comorbidity and with structurally normal heart. ***(Iranian Heart Journal 2016; 17(1): 71-73)***

Keywords: ■Acute pulmonary edema ■Panic attack ■Hypertension

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Cardiovascular events during a panic episode may be triggered by increased sympathetic outflow.[1] During a panic attack, sympathetic nerve firing increases, as does the adrenomedullary secretion of epinephrine. It has been suggested that some patients may experience angina-like attacks secondary to coronary artery spasm. Anxiety or panic disorder is one of the major causes of paroxysmal hypertension.[2,3]

Emotional stress and panic attack can also precipitate severe, reversible left ventricular (LV) dysfunction (Takotsubo's syndrome), which is accompanied by chest pain, pulmonary edema, and cardiogenic shock in patients without coronary disease.[4,5]

In this article, we present a case of pulmonary edema and hypertension crisis due to panic attack.

CASE PRESENTATION

A young woman, aged 35 years, was admitted to the emergency department of clinical toxicology with organophosphate pesticide self-poisoning. She had consumed undetermined amounts of an organophosphate pesticide 4 hours before hospitalization following an emotional stress. At the time of admission to the emergency department of clinical toxicology (EDCT), she was restless and agitated. Evaluation of vital signs revealed a pulse rate of 148/min, systolic/diastolic blood pressure of 109/88 mm Hg, and respiratory rate of 20/ min. She was afebrile and had a brief increase in salivation. Arterial O2 saturation was 88% based on pulse oximetry. Her pupils were mydriatic and reactive to light. In respiratory examination, dispersed rhonchi and bilateral rales were heard. Other organs revealed no pathologic sign or symptoms on physical examination. Laboratory investigations at admission showed normal renal function tests, liver enzymes, and normal serum levels of sodium, potassium, calcium, blood sugar, and magnesium. Computed tomography (CT) scan showed a normal picture of brain structure. Serum cholinesterase level was 5%, and red-cell acetylcholinesterase activity was 0.3. She had no history of physical illness or substance abuse. During the transfer to the hospital, 2 mg of atropine was injected because of increased salivation by emergency medical services. Thereafter, another 2 mg of atropine was given in the EDCT until the patient's mouth mucus was completely dried and auscultation of her lungs was clear without any crackles or rhonchi. This was followed by the infusion of atropine at a rate of 0.5 mg/h; the dose was titrated as per her clinical response and signs of atropinization in the clinical toxicology ward. Response to atropine treatment was desirable; and over the next 2 days, she did not need further atropine. She was awake, although she had no verbal communication at this time. Neurological examination was completely normal. She had only mild tachycardia (96/min), and her other vital signs were normal. On day 4 after admission (i.e., when she had required no atropine infusion or other treatments for organophosphate poisoning for 2 days), she suddenly developed hypertension crisis with systolic/diastolic blood pressure of 230/150 mm Hg, cold sweating, pink frothy sputum, tachycardia, and tachypnea. Chest examination revealed widespread rales wet rales. Chest X-ray illustrated diffuse bilateral alveolar infiltration (Fig. 1). ECG revealed no ST-T changes or other abnormalities (Fig. 2). Considering a clinical diagnosis of acute cardiogenic pulmonary edema, we started an intravenous infusion of nitroglycerin and furosemide and injection of 3 mg of ampule morphine every 4 hours after a bolus of 2 times 40 mg of furosemide and also 2 times 3 mg of morphine during the first 30 minutes. Twelve hours later, the patient's blood pressure was controlled and her rales disappeared. Bedside echocardiography showed normal left ventricular systolic and diastolic functions and normal right

ventricular size and function. There was no significant valvular heart disease. Psychiatric consultation confirmed anxiety disorder and panic attack. Treatment with fluoxetine and clonazepam was started. During the patient's hospital stay and after hospital discharge, outpatient follow-up showed no hypertension crisis.

DICUSSION

There are a small number of case reports that complement our observation and suggest that pulmonary edema may be precipitated in some individuals without evidence of heart disease if there is an appropriate trigger such as idiosyncratic emotional stress. There is a report of hemoptysis and pulmonary edema during sexual intercourse in a patient with mild mitral regurgitation. Pulmonary edema was reported in 2 athletes during a 90-km race.

These observations suggest that some individuals who appear to have normal hearts may develop pulmonary edema during emotional stress, extreme exertion, or sexual intercourse. These individuals appear to be susceptible to pulmonary edema when swimming and appear predisposed to hypertension.[2] The cause of the episodes of pulmonary edema is uncertain, but left ventricular diastolic dysfunction may be a contributor.[6] Coronary artery spasm may be involved in the etiology of the patients who experience chest pain; however, it seems most probable that in these patients, the episodes of acute left ventricular failure were precipitated by acute hypertension as a result of neurohormonal stimulation. Patients with panic disorder exhibit acute episodes that evoke sympathetic neuronal and adrenomedullary activation, with precipitation of hypertension crisis.

CONCLUSIONS

We conclude that panic attack and its hypertensive crisis may be severe enough to develop pulmonary edema even in young healthy adults with no comorbidity and with structurally normal heart.

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