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Case Report

Takotsubo cardiomyopathy: a hidden enemy of the hypovolemic patient?

Abstract

Takotsubo cardiomyopathy (TCM) is an underrecognized transient left ventricular dysfunction that mimics clinically an acute coronary syndrome. It has been linked to emotional stress and several clinical entities that provoke a catecholamine surge in the blood stream. We investigated the case of a young female patient who was admitted to the intensive care unit after a significant blood loss due to miscarriage. The patient was fully monitored and was treated for the hypovolemia. A dramatic aggravation of her clinical status was directly linked to the appearance of TCM 1 hour after her admission. This study sought to assess the appearance of TCM in a hypovolemic patient. We found no reports in the literature linking these 2 clinical entities. The blood loss and the pathophysiology of hypovolemia, especially through the excess of catecholamines that are released in the blood stream, seem to provoke the appearance of a subclinical form of TCM. As a result, TCM worsened further the general condition of the patient. There is a high possibility that TCM and hypovolemia can be closely related, and therefore, the treatment of hypovolemia can be adjusted to new standards. Because it has already been proved that TCM can be induced by emotional stress and various pathological entities, further investigations are necessary.

We present the case of a 36-year-old woman who was admitted to the intensive care unit with stage 3 hypovolemia (blood pressure, 90/50 mm Hg, 150 beats per minute; inspiratory rate, 35 to 40 per minute) after significant blood loss due to miscarriage. She was 12 weeks pregnant, with an unremarkable medical history.

The hemogram confirmed the hypovolemic status (hematocrit, 21.7%; hemoglobin, 7.4 g/dL; PLT, 100 000 k/mL), and blood gas results showed a metabolic acidosis (pH 7.30; PO_2, 59 mm Hg; PCO_2, 19 mm Hg) and poor tissue oxygenation (Lac, 5.3 mmol/L). The electrocardiogram (ECG) revealed sinus tachycardia, and a performed transthoracic echocardiogram (TTE) was normal. The patient was administered with IV fluids to restore the volume depletion, esmolol (100 μg/kg per minute) for the sinus tachycardia, fresh-frozen plasma, and packed red blood cells.

One hour after admission, the patient presented dyspnea while her vital signs and blood gas results worsened significantly (pH 7.05; PO_2, 77 mm Hg; PCO_2, 43 mm Hg), leading to the patient's intubation. A new TTE revealed an ejection fraction of 35% and severe hypokinesis of the apex of the left ventricle (Fig. 1), whereas a chest computed tomography showed pulmonary edema (Fig. 2). Coronary angiography and ventriculography were negative for obstructive coronary disease. The diagnosis of takotsubo cardiomyopathy (TCM) was established.

The patient was supplementary administered with IV furosemide; inotropic agents were not used because her condition improved within hours. The patient was weaned from mechanical ventilation the next day when respiratory and circulatory functions were adequately restored. The laboratory results indicated mild renal charge (blood urea nitrogen, 80 mg/dL; creatinine, 2.2 mg/dL) and confirmed the myocardial dysfunction (creatine kinase, 1470 IU/mL; TnT, 0.15 ng/mL). Continuous heart monitoring did not reveal ECG changes, and an echocardiogram revealed restoration of the contractility of the left ventricle and of the ejection fraction (60%) at the fifth day. The patient was discharged the seventh day and has not had from any kind of relapse 11 months after this incident.

Takotsubo cardiomyopathy is an acquired transient dysfunction of the apical or midlevel segments of the left ventricle [1]. It is today categorized as an acquired cardiomyopathy, and the diagnosis is established reductio ad absurdum [2]. Most of the researches indicate that the myocardial stunning is a result of a catecholamine surge in the blood stream after emotional or physical stress due to enhanced sympathoneural activity [3]. Hypercatecholaminaimia possibly affects the myocardium causing direct toxicity (potential source of oxygen-derived free radicals), coronary vasoconstriction (indicated by the various ECG changes), and microvascular spasm (sympathetically mediated dysfunction).

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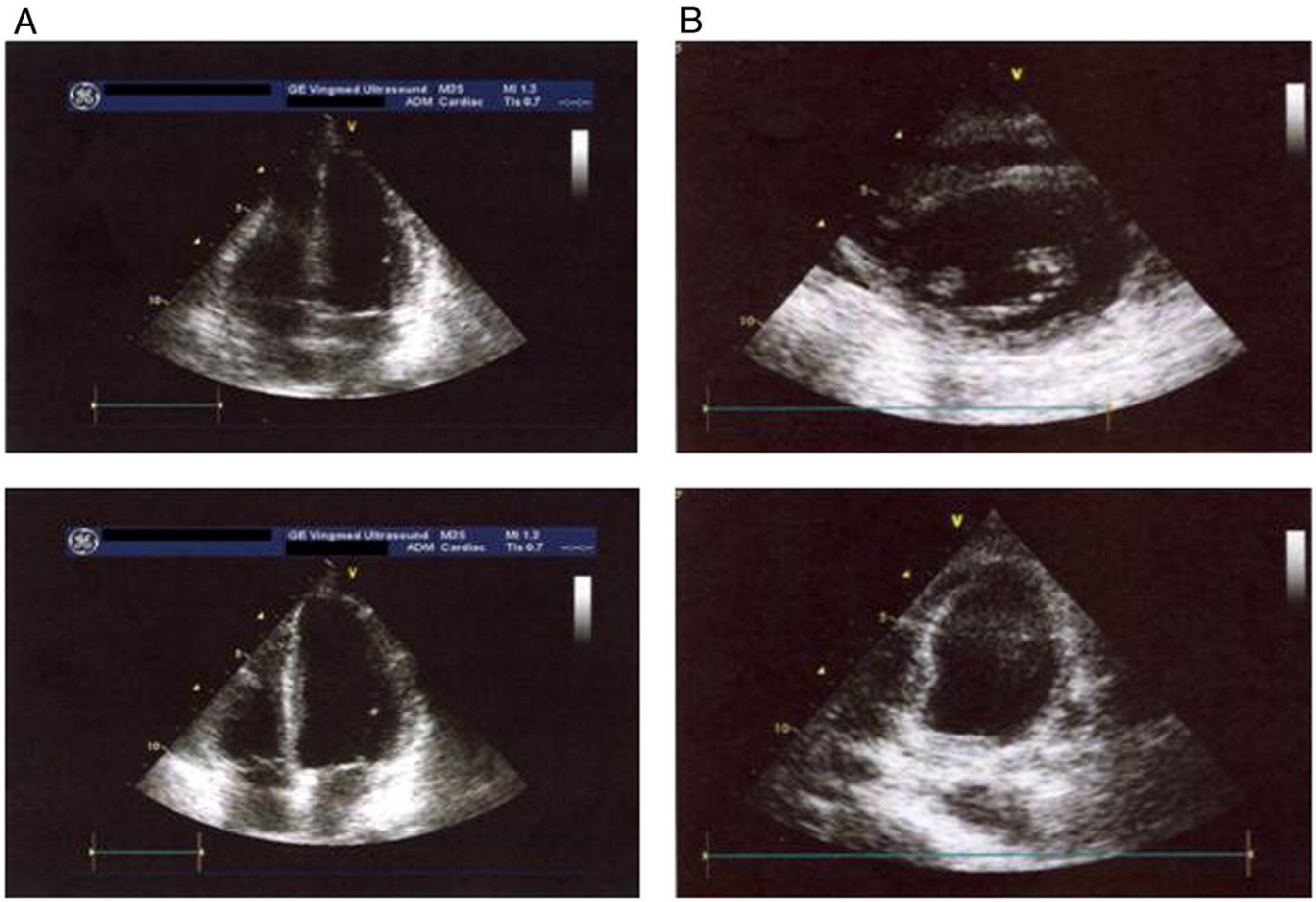

Q5 **Fig. 1** A, Normal TTE at the time of admission. B, Image of TCM in the TTE 1 hour after the patient's admission.

Hypovolemia is a clinical state that causes secretion of catecholamines in the blood stream. Reduced volume leads to sympathetic activation and catecholamine release to balance the reduced blood irrigation of vital organs.

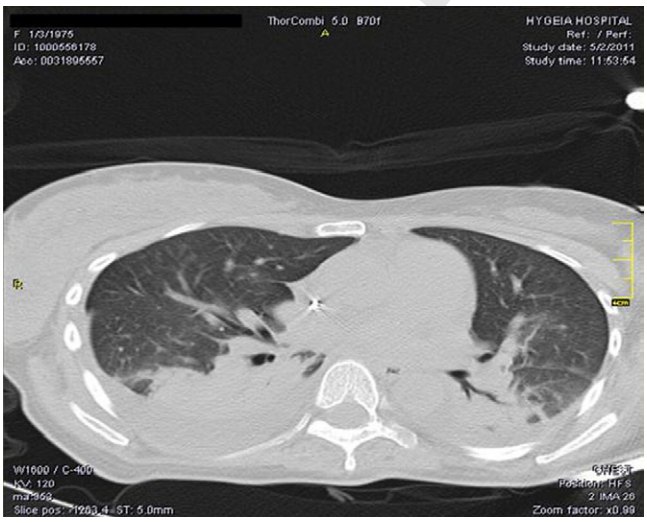

Fig. 2 Chest computed tomography confirming the pulmonary edema.

Our patient had lost almost 30% to 40% of her blood volume at the time of admission, and the effects of blood loss were visible until the intravascular volume was restored. A normal ECG on admission was critical because it excluded the possibility of the cardiomyopathy being present. The sudden onset of dyspnea and aggravation of hypotension and tachypnea were the result of TCM establishment. The most probable cause was hypercatecholaminaimia due to hypovolemic shock. Most importantly, the patient's condition worsened dramatically because of pulmonary edema and cardiogenic shock. It is highly likely that there is a bidirectional relationship between hypovolemia and TCM, and our team was able to observe it in a controlled environment. Because the patient did not present ECG changes, it would be quite possible that such a condition be missed, for example, in the emergency department.

Our hypothesis was that there are strong indications that catecholamines' secretion in a hypovolemic patient can lead to TCM, and we strongly believe that this fact needs to be subjected to further studies. The importance of a possible subclinical form of TCM in the hypovolemic

patient stands in the treatment of hypovolemia and how it is affected.

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