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From right to left heart failure: An unexpected transition

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ABSTRACT

Right and left heart failure are very common clinical syndromes with close correlation. Right-sided or right ventricular heart failure usually occurs as a result of left-sided failure. We report a very rare case of transition from right heart failure due to pulmonary embolism, followed by its resolution, to left heart failure due to Tako-tsubo syndrome within 48 h of hospitalization.

Introduction

Right and left heart failure are highly prevalent clinical syndromes with close correlation but different etiologies. The 2 main causes of right ventricle (RV) failure are: chronic pulmonary arterial hypertension and pulmonary embolism (PE). Acute massive PE can lead to RV dilation and failure, primarily due to acute increase in afterload. This RV dysfunction is a well known predictor of morbidity, mortality and recurrence of PE.[1] Aggressive intervention with thrombolytic therapy, vasoactive agents, or mechanical embolectomy may improve right ventricular function and clinical outcomes.[2] On the other hand, Tako-tsubo cardiomyopathy, a transient and reversible left ventricular apical ballooning, is typically precipitated by an emotional stress or acute medical illness[3]; it mimics acute coronary syndrome in the absence of significant angiographically coronary artery stenosis.

Presentation of case

Ms. EG is a 58 year old woman, who presented to our emergency department (ED) for shortness of breath and palpitations of few hours duration. History goes back to one day prior to presentation when the patient went to her gastroenterologist complaining of chronic constipation. She was tachycardic with irregular heart rate at the office. Thus, she was sent to the ED for further evaluation. On arrival, the patient was complaining of shortness of breath and palpitations. The onset of symptoms was associated with generalized weakness, lightheadedness and right calf pain of few days duration. Review of systems revealed 20 lbs weight loss over the past two months.

Her past medical history is significant for asthma, ethanol abuse, chronic constipation, chronic low back pain, early menopause, and laxative abuse. She is allergic to penicillin.

On physical examination, her blood pressure was 152/87 mm Hg, respiratory rate 22 breaths per minute, irregular pulse of 140 beats per minute and no fever ($T = 95.9$ F). The patient appeared in mild respiratory distress. There was mild distention in the neck veins. Examination of the heart showed irregular S1 and S2 with no murmurs. Wheezes were heard throughout in both lungs. Her abdomen was soft with mild diffuse tenderness on deep palpation but no rebound tenderness. Her peripheral pulses were palpable. Right calf tenderness was noticed on palpation and on dorsal extension of the right foot. No lower extremities edema was noted.

The results of other laboratory tests were within normal limits except for normocytic anemia (hemoglobin of 10.9 g/dl). The brain natriuretic peptide (BNP) was 1056 pg/ml with two sets of negative cardiac enzymes.

Her electrocardiogram (ECG) showed atrial fibrillation with rapid ventricular response of 140 beats per minutes with no ischemic ST-T

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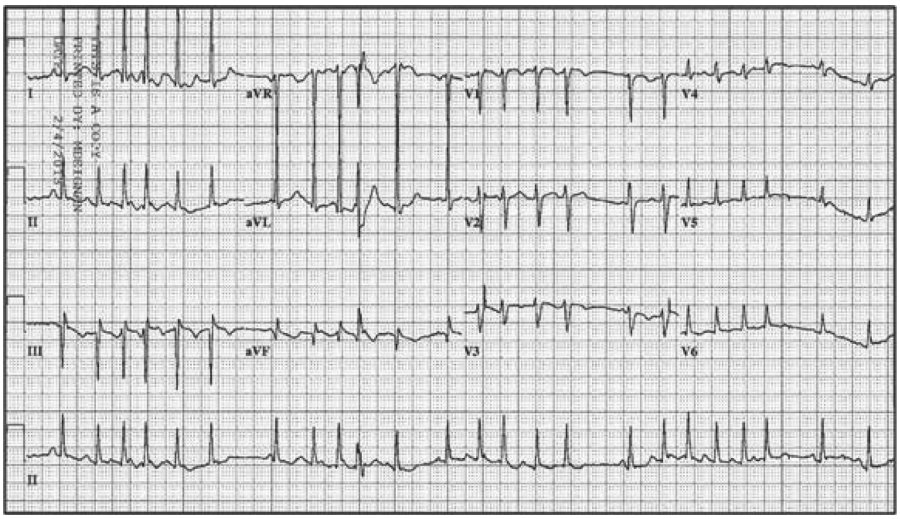

Fig. 1. Electrocardiogram showing atrial fibrillation with rapid ventricular rate at 140 beats per minute.

changes (Fig. 1). A portable chest x-ray showed small right pleural effusion and elevated left hemidiaphragm. Right deep femoral vein, right calf vein and left common femoral vein thrombosis were detected on a venous Doppler of the lower extremities. Therefore, the patient was admitted to the telemetry unit. She was started on intravenous (IV) diltiazem drip, to control her heart rate, and subcutaneous enoxaparin 1 mg/kg every 12 h. Given the high clinical suspicion of pulmonary embolism, a ventilation perfusion scan of the lung was performed and showed multiple segmental perfusion defects.

Moderate dilation and systolic dysfunction of the (RV), large right atrial thrombus extending into the RV and normal left ventricle ejection fraction (LVEF) were detected on the transthoracic echocardiography (TTE) (Fig. 2). The decision was to transfer the patient to the ICU for thrombolysis with alteplase. Enoxaparin was switched later to intravenous unfractionated heparin and the patient was started on warfarin. A repeat TTE on the next day showed significant resolution of the atrial thrombus.

The patient was then transferred to a regular floor. Given her clinical presentation, the presumed diagnosis was a hypercoagulable state related most probably to a colon cancer. A CT scan of the abdomen and pelvis showed large bowel obstruction with enhancing soft tissue mass lesion in the descending colon with hypodense lesions within the liver reflecting metastatic disease.

A surgical consult was obtained for diverting colostomy and inferior vena cava filter (IVC) placement. All these findings and the plan were discussed with the patient. Few hours later, she started to complain of shortness of breath. ECG showed normal sinus rhythm, frequent premature atrial beats with anterolateral ST-T changes (Fig. 3). Serial cardiac enzymes were negative and CXR showed no changes.

A repeat TTE showed acute dilation of the mid and apical segments of the LV with an EF of 15–20%. The RV was within normal in size and function. There was no visualization of any thrombus in the right atrium (Fig. 4). Since the patient had no history of coronary artery disease, these findings were consistent with an acute stress-induced dilated cardiomyopathy or Tako-tsubo syndrome.

Diltiazem was stopped and the patient was started on low dose carvedilol and enalapril. Few days later, and after being assessed by the cardiologist, the patient underwent an exploratory laparatomy, Hartman's procedure and insertion of an IVC filter. A repeated TTE showed improved left ventricular systolic function with normal RV function.

Discussion

Acute pulmonary embolism (PE) is a common and often fatal disease. It can be classified as massive or submassive. The diagnosis of massive PE should be considered whenever there is hypotension accompanied by an elevated central venous pressure (or neck vein distension) not otherwise explained by acute myocardial infarction,

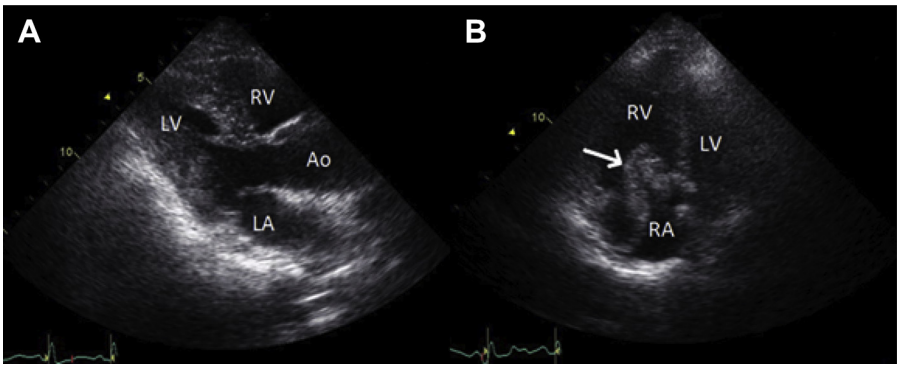

Fig. 2. Transesophageal echocardiogram showing A) Normal left ventricle size and function; B) Dilated right ventricle with large right atrial thrombus extending into the right ventricle (white arrow).