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Case Report Open Access

Biventricular Stress-induced Cardiomyopathy and Cardiogenic Shock Secondary to Rheumatoid Arthritis Flare

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Abstract

Stress-induced cardiomyopathy has a wide variety of clinical presentations ranging from angina symptoms to cardiogenic shock, as well as an array of echocardiographic findings and etiologies. We present a unique case of severe biventricular dilation with cardiogenic shock but rapid recovery within 96 hrs. Severe biventricular stress-induced cardiomyopathy secondary to acute rheumatoid arthritis (RA) flare was diagnosed and the patient later regained full recovery after circulatory support and RA treatment.

Keywords: Biventricular stress-induced cardiomyopathy; Cardiogenic shock; Rheumatoid arthritis

Case Presentation

Biventricular stress-induced cardiomyopathy is rare and often associated with severe hemodynamic compromise and fatal outcomes. Here we describe the first reported biventricular stress-induced cardiomyopathy with cardiogenic shock caused by a rheumatoid arthritis flare that was successfully treated with immunosuppressant in addition to mechanical and pharmacological circulatory support.

A 59-year-old African American male with history of hypertension and seronegative rheumatoid arthritis (RA) was evaluated for chest pain, hypotension and ST-segment elevations on electrocardiogram (ECG). The patient had been on weekly methotrexate and daily prednisone for long standing RA, but these were stopped 5 weeks prior to his presentation in preparation for left knee surgery due to worsening erosive arthritis. He began experiencing arthritic pain, fatigue and chest pressure shortly after discontinuation of these medications. On the day of presentation, however, he developed acutely worsening chest pain and shortness of breath. Due to refractory symptoms, the patient then presented to his primary care physician's office. There, he was found to be hypotensive with a systolic blood pressure of 70 mmHg. An ECG revealed atrial fibrillation with anterolateral ST-segment elevations (Figure 1). He was sent to the emergency department where he was given 325 mg of aspirin and started on a dopamine infusion for ongoing hypotension. Emergent coronary angiography showed no evidence of coronary artery disease or vasospasm (Figure 2). A left ventriculogram revealed severely impaired left ventricular contractile function in a global pattern and a right heart catheterization demonstrated profoundly decreased cardiac output. An echocardiography confirmed severe biventricular dilation (Figure 3) and dysfunction with an estimated left ventricular ejection fraction (EF) of 30%. Cardiogenic shock secondary to acute biventricular failure was diagnosed. A dobutamine infusion was initiated and an intra-aortic balloon pump (IABP) was inserted for additional hemodynamic support.

Initial cardiac troponin levels were normal, and remained so serially over the next 24 hours. A comprehensive urine toxicology screen showed no evidence of recent substance abuse. An extensive infectious evaluation was unrevealing. Due to significant RV dysfunction, a CT angiogram of the chest was performed and no pulmonary emboli were detected. Although a cardiac magnetic resonance imaging study could not be performed due to a metallic eye implant, a whole body PET/CT scan revealed no apparent malignancy or abnormal myocardial uptake suggestive of either myocarditis or myocardial scar.

C-reactive protein and erythrocyte sedimentation rate levels were significantly elevated at 39.3 mg/dL and 94 mm/hr, respectively. The patient had active knee and hand joint synovitis consistent with a RA flare and prednisone was started at 20 mg daily on hospital day #3 by rheumatology. The patient's condition steadily improved with supportive measures. He was weaned off of all intravenous vasoactive medications and the IABP was successfully removed. Electrocardiographic ST-segment elevations persisted for 3 days, but completely normalized on day #4 (Figure 4). A repeat echocardiogram on day #4 demonstrated near-complete resolution of the patient's biventricular dysfunction, with a reported LVEF 50% and normal right ventricular (RV) function.

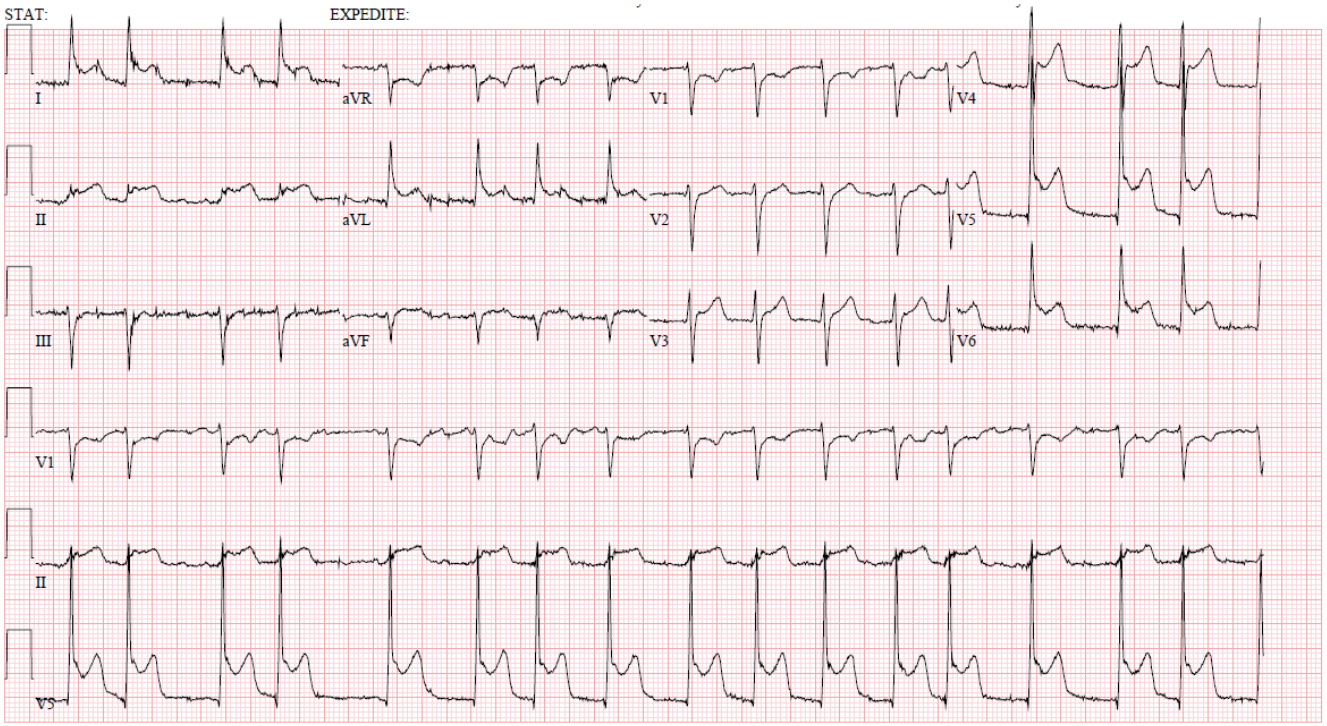

Figure 1: An ECG revealed atrial fibrillation with anterolateral ST-segment elevations.

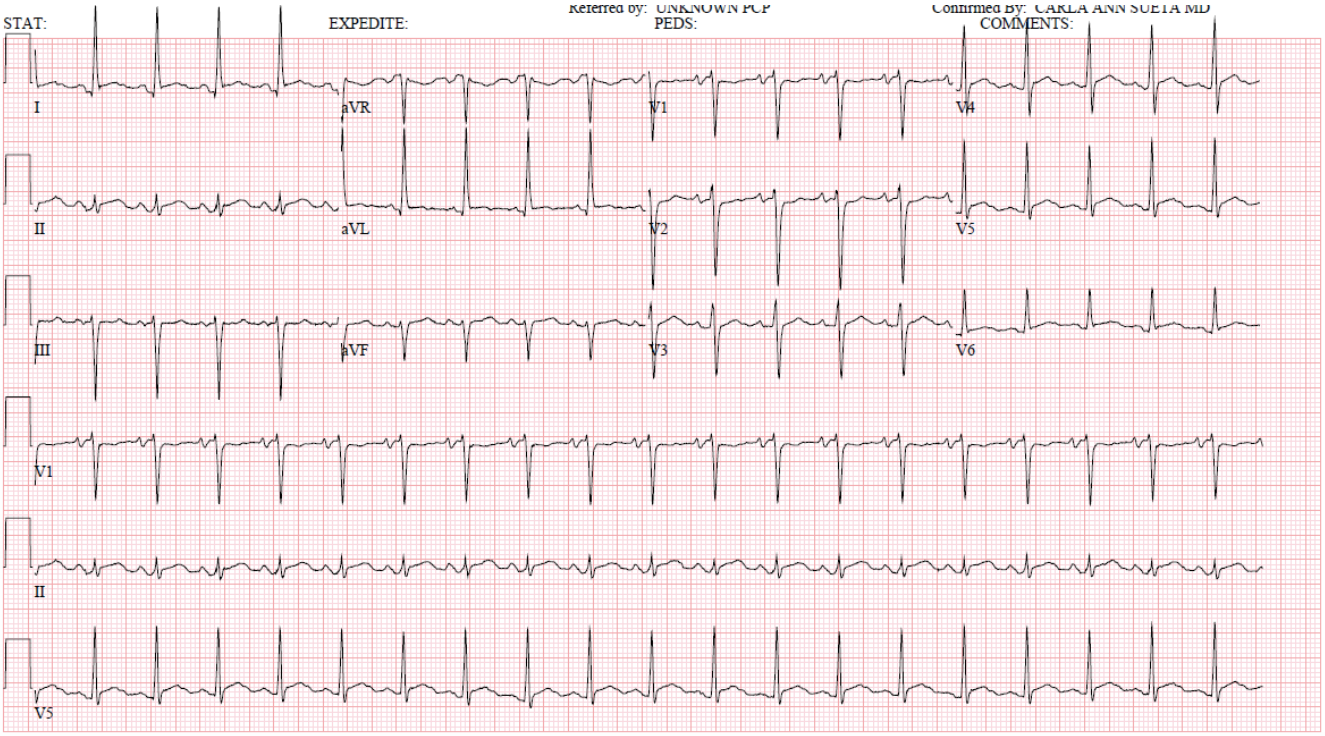

Figure 2: Electrocardiographic ST-segment elevations persisted for 3 days, but completely normalized on day #4.

Given the patient's transient global biventricular dysfunction and electrocardiographic changes in the absence of coronary abnormality, myocarditis, or pheochromocytoma, a diagnosis of biventricular stress-induced cardiomyopathy (SICM) was made [1]. The presumptive etiology of this patient's SICM was his acute RA flare. The patient was continued on prednisone, initiated on oral, evidence-based chronic heart failure pharmacotherapies (ACE inhibitor and beta blocker), and discharged to home on hospital day #12. At 6-week follow-up, he was without any cardiac symptoms and his functional status had completely returned to baseline. An echocardiogram then showed normal left ventricular (EF >55%) and normal RV size and function (Figure 4). At 3- months follow up, the patient had no recurrent heart failure symptoms or clinical events.

Discussion

We report a unique case of severe biventricular SICM and cardiogenic shock precipitated by a RA flare. SICM was first described in 1990 and given the moniker Takotsubo Cardiomyopathy to illustrate the characteristic apical ballooning wall motion abnormality that was commonly seen. However, since its original description, there have been many anatomical variations identified, including mid-ventricular, basal, focal and global hypokinesis (as seen in this case) [2]. SICM is typically thought of as a disease occurring predominately in postmenopausal women from emotional stress resulting in catecholamine surge. More recent analysis of the International Takotsubo Registry instead found that physical triggers are more common than emotional ones, and that 28.5% of cases have no known trigger at all [3]. In the current case, there was profound biventricular failure, which necessitated mechanical and pharmacological circulatory support. RV involvement is often associated with worse outcomes and hemodynamic compromise [4], as seen with this patient.

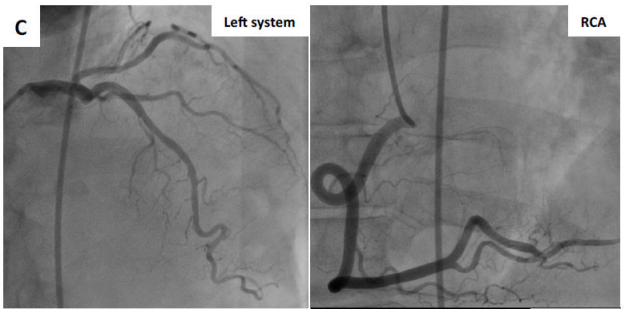

Figure 3: Emergent coronary angiography showed no evidence of coronary artery disease or vasospasm.

Autoimmune disorders have been associated with SICM in rare occasions [5]. Here we describe the first reported case caused by a RA flare. The pathophysiology of SICM in the setting of systemic inflammation is unclear, but is thought to be due to the release of tumor necrosis factor and activation of the complement cascade – all culminating in the development of cardio-suppression. In this patient, we hypothesize that the discontinuation of prednisone and methotrexate precipitated an exacerbation of the patient's RA and led to subsequent systemic inflammation. This ultimately resulted in biventricular SICM and cardiogenic shock.

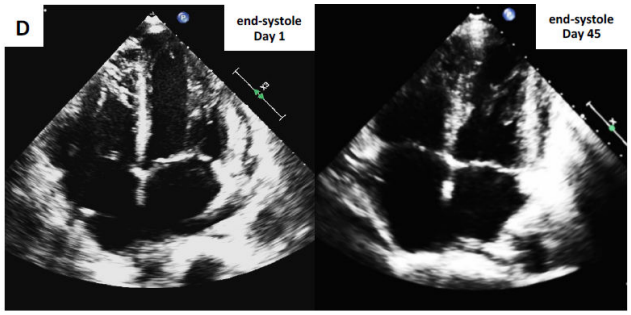

Figure 4: An echocardiogram then showed normal left ventricular (EF >55%) and normal RV size and function.

Disclosures

The authors have no conflicts of interest to disclose.

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