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Echocardiography Findings in an Interesting Presentation of Stress-Induced Cardiomyopathy

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Abstract: *We report a case of a middle-aged man who presented in ventricular tachycardia/ventricular fibrillation cardiac arrest and subsequently developed stress-induced cardiomyopathy (SIC). What makes this case intriguing is when the patient first presented to the emergency department, the bedside echocardiogram showed a normal ejection fraction within hours of the initial event. The next day, a repeat echocardiogram revealed stress cardiomyopathy, which has now resolved. The event included two defibrillator shocks in the field and one after cardiac catheterization. This presentation was similar to previously reported cases of SIC, but we could find no other reports of similar cases where the patient had a normal initial echocardiogram and then developed SIC post-VT/VF arrest.*

Keywords: *Takotsubo, Echocardiography, Cardiomyopathy, Ampulla, Arrhythmia.*

CASE: A 50-year-old male with no significant past medical history was admitted to the ICU after being found in the field status post cardiac arrest. The patient was found to be in ventricular fibrillation and achieved return of spontaneous circulation after receiving two shocks via defibrillator. He was intubated in the field. On arrival to the emergency department, the patient's initial vital signs were temp 98.4F, heart rate 82, respiratory rate 18, blood pressure, 127/81, and oxygen saturation 100% on the ventilator. He was given a bolus of amiodarone in the emergency department and started on amiodarone drip. Physical examination showed a well-developed middle-aged man. His neck was supple with no jugular venous distension, cardiovascular examination revealed normal S1, S2, with a regular rate and rhythm. There were no murmurs, rubs, or gallops, and his lungs were clear to auscultation bilaterally. His Glasgow Coma Scale was 15.

The initial electrocardiogram on arrival showed normal sinus rhythm, 62 beats per minute, with ST segment depressions in the lateral leads, troponin was mildly elevated at 0.117ng/mL. The initial echocardiogram showed a normal ejection fraction with no obvious wall motion abnormalities. He was then transferred to the ICU on ventilatory support and continued on amiodarone drip. The patient was placed on cardiac monitoring for any arrhythmia activity. Overnight, telemetry showed normal sinus rhythm. The next day, repeat echocardiogram showed the left ventricle was normal in size and wall thickness. However, the left ventricular systolic function was severely decreased to an ejection fraction of 20-25% with basal segments contracting, and apical ballooning and hypokinesis of all other segments, thus strongly suggesting signs of takotsubo cardiomyopathy (Figure 1). These new findings, coupled with his presentation of cardiac arrest due to ventricular fibrillation, warranted a diagnostic cardiac catheterization to rule out ischemic causes. The left main coronary artery was normal caliber and left anterior descending was normal in caliber type III vessel with 10% luminal irregularities. The left circumflex also had normal caliber with 10% luminal irregularities and 10% luminal irregularities as well in the right coronary artery.

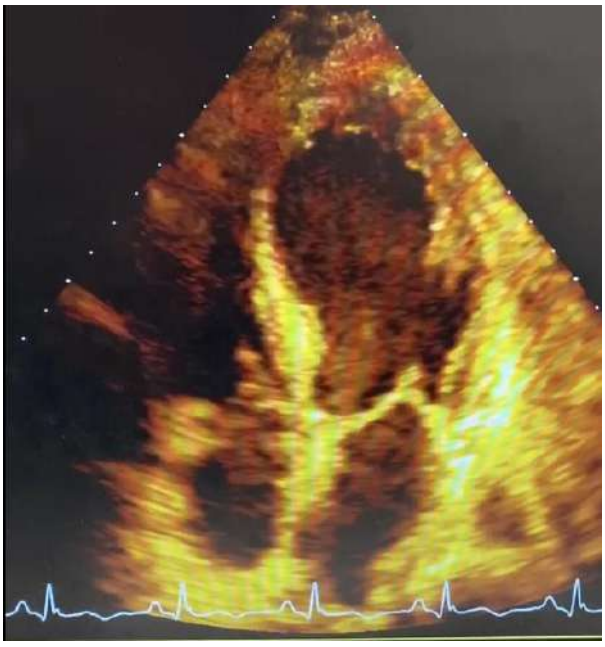

Figure 1

Following catheterization, the patient was then treated for takotsubo cardiomyopathy. He was started on carvedilol 3.125mg, lisinopril 5mg, and aspirin 81mg. He was also started on apixiban 5mg twice a day due to high risk of a thromboembolic event occurring. The final echocardiogram repeated 12 days later showed the left ventricle to be normal in size and wall thickness. The patient had a return to a normal left ventricular segmental wall motion, with LVEF of 55-60% (Figure 2).

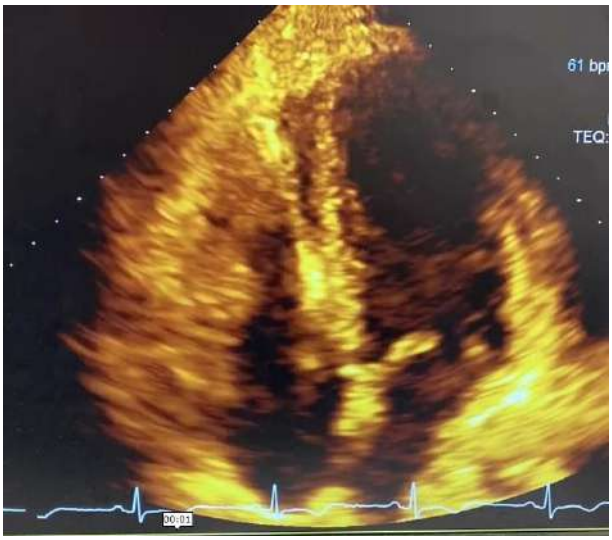

Figure 2

3. DISCUSSION

SIC is also known as takotsubosyndrome, broken heart syndrome, and ampulla cardiomyopathy[1]. SIC presents similarly to an acute myocardial infarction, however it has been attributed to severe emotional or physical stressors with an absence of obstructive cardiomyopathy, acute plaque rupture, and angiographic obstructive coronary artery disease[2].

SIC has reversible and transient systolic dysfunction of the left ventricle[3]. Although the workup usually mimics acute coronary syndrome, coronary angiography does not show adequate blockage [34]. SIC usually presents with left ventricular apical akinesia, systolic ballooning effect with preserved and or hyperdynamic basal function[5]. SIC is correlated with electrocardiogram findings indicating ischemia, with a rise in troponins. However, no studies were able to distinguish SIC from acute coronary syndrome based on noninvasive studies[6]. The majority of patients with SIC present with minimal elevations in cardiac enzymes on the time of admission.[4] Research has shown these slight increases in cardiac enzymes have dropped rapidly, without having significant prognostic value[4]. It is imperative coronary angiography is completed to definitively rule out acute coronary syndrome[4]. The imaging modality most frequently used to assess left ventricular changes is the echocardiography[7]. Important features seen on echocardiogram are left ventricle apical ballooning, or dyskinesia of mid-apical myocardial segments[7]. There may also be anterior or entire interventricular septum, midventricular anterolateral wall involvement, or inferior wall involvement seen on echocardiogram[7]. The pathogenesis of SIC is currently unknown[4]. At this time catecholamine-induced cardiotoxicity, along with microvasculature dysfunction are the most supported theories[4]. Studies have shown serum catecholamine concentrations were on average three times higher than patients with myocardial infarctions[8]. The microvasculature dysfunction seen in patients with SIC include abnormalities in endothelium-dependent vasodialation, impaired myocardial perfusion, and excessive vasoconstriction[9]. When observing patients who underwent myocardial biopsies there were areas of contraction band necrosis, localized fibrosis, and infiltration of inflammatory cells[10]. Treatment of SIC during the acute phase is primarily symptomatic[4]. If the patient is hemodynamically unstable intra-aortic balloon pumps can be required for cardiopulmonary circulatory support, and continuous veno-venous hemofiltration[111213]. Although controversially used cardiac stimulants are used in around 20%-40% of patients treated for SIC[142]. If the patient has severe left ventricular outflow obstruction with hemodynamic compromise, it is recommended to use beta-blockers or alfa-adrenocepter agonist therapy[4]. Calcium channel blockers are commonly used to decrease left ventricular outflow tract gradient[4]. When patients are hemodynamically stable, diuretics, angiotensin-converting enzyme inhibitors, and beta-blockers are recommended[4]. When treating SIC you must also take into account the risk of thromboembolism[4]. In patients with loss of motion of the left ventricular apex, anticoagulation is recommended until the apex contractility has improved[4]. As of now there is not overall agreement regarding long-term management of SIC[4]. It is seen as reasonable to treat patients with beta-blockers, and angiotensin-converting enzyme inhibitors during the ventricular recovery period[4].There is no data that supports continuous use of these medications for the prevention of SIC recurrence, or increasing survival rates[4]. In regards to prognosis, patients with SIC have been observed to regain full recovery in around 96% of cases[15]. Studies have shown hospital mortality rates ranging from 1%-2%[1617].

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