Case Report

A woman with Typical Chest Pain and Broken Heart

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Abstract

Stress-induced cardiomyopathy is an increasingly recognized syndrome characterized by transient apical or midventricular dysfunction that mimics myocardial infarction in the absence of significant coronary artery disease (CAD), but with essential difference in their management; thus our report present one of these cases.
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Introduction

Transient left ventricular apical ballooning syndrome also called (Takotsubo cardiomyopathy, Stress-induced cardiomyopathy (SICM), Broken heart syndrome, Ampulla cardiomyopathy) was initially described in Japanese literature in 1990 and has since been diagnosed by transient LV apical hypokinesia in the absence of significant angiographic coronary artery stenosis or cardiomyopathy.[1] he affected heart, when viewed by echocardiography or catheterization, mid-ventricle and apex has a rounded bottom and narrow neck in systole which resembles the traditional Japanese octopus trap called "Tako-tsubo".[2] SICM is an emerging and under-diagnosed disorder mimicking ST elevation infarction, and high clinical suspicion is required for diagnosis. We present here a patient with this clinical disorder resembling an acute coronary syndrome.

Case report

A 40-year-old afghan woman presented to urology department with urinary incontinency. She was diagnosed with vesicovaginal fistula and was scheduled for elective surgery. She underwent transabdominal vesicovaginal repair and reimplantation of bilateral distal ureters because of left distal ureter obstruction. After her surgery, a cardiology consultation was requested due to chest pain, hypotension and electrocardiogram (ECG) changes. Her chest pain was began after half hour of recovery, located at the mid sternal area, and radiated to her neck and inferior jaw associated with cold diaphoresis, nausea and orthopnea. She had no coronary risk factors and her family history of CAD was negative. She was transferred to coronary care unit (CCU) for further evaluation. Vital signs at the time of CCU admission included: Blood pressures=90/55 mmHg, respiratory rate=24/min, pulse rate=110/min, body temperature=37˚C. Jugular venous pressure was elevated. Cardiac examination revealed normal heart sounds without murmurs. Bilateral basal fine crackles were heard. Her ECG showed extensive anteroseptal and inferior ST-segment elevation (Figure 1) and her serum troponin level was elevated to10 ng/ml (Normal up to 8 ng/ml). Coronary angiography was done which revealed no obstructive coronary lesions (Figure 2A and 2B). The left ventriculogram demonstrated apical ballooning with an appearance of Tako-Tsubo cardiomyopathy and an ejection fraction of 20% (Figure 2C). Conservative medical treatment recommended with inotropic agents, angiotancin converting enzyme inhibitor, ASA and anticoagulation. Yet the patient developed shock and remained unstable on high doses of epinephrine, vasopressin, dobutamine, dopamine, norepinephrine. Unfortunately she died three days after admission due to sepsis.

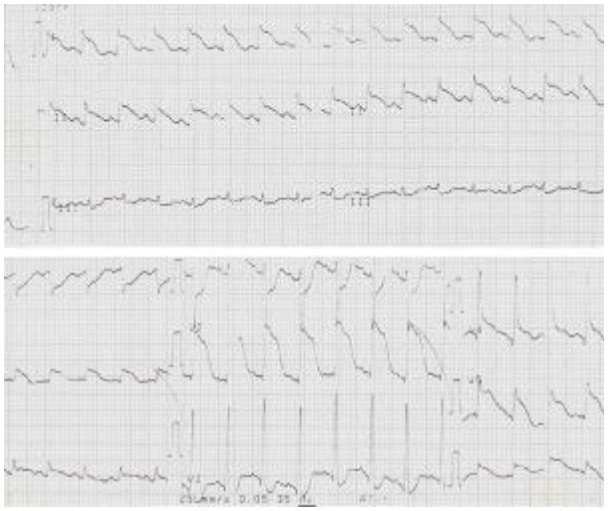

Fig 1- Electrocardiogram of the patient

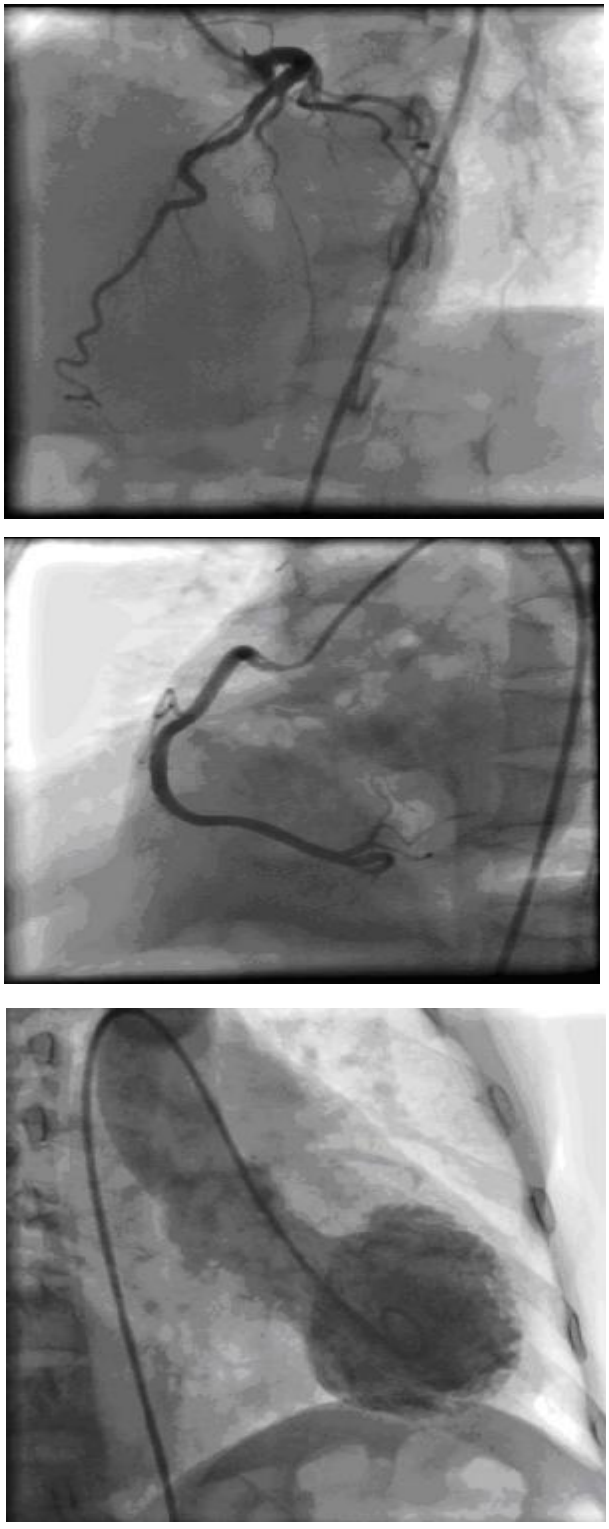

Fig 2C- Left Ventriculogram of the Patient

SICM is diagnosed in 1–2% of patients presenting with symptoms suggestive of acute coronary syndrome.[3] Most patients with SICM are postmenopausal women. A meta-analysis of 14 studies by Gianni et al.[4] and Prasad et al.[1] found 89% and 90% female predominance with median age being 58–77 and 58-75 years old respectively.[1] The etiology of the SICM has not been clearly established but catecholamine-mediated neurogenic myocardial stunning provoked by stressors is confirmed by elevated plasma catecholamine levels during the acute phase in more than 70% of the patients.[3] Strong evidences support the hypothesis that sympathetic hyperactivity can lead to SICM. Myocardial scintillography with 123I-metaiodobenzylguanidine (MIBG) in these patients cleared a reduced uptake of radiotracer in several segments of the heart, confirming an intense discharge of adrenalin induced by stress.[5] Studies showed that the density of beta-adrenergic receptors is at the apex of the heart, so the circulating catecholamine influences this segment excessively which result in apical negative cardiac myocyte inotropy.[6] Typically, the precipitating event is severe emotional or physiological stress. Emotional stressors were important precipitating events for SICM in case series report. As a result, the name "Broken heart syndrome" was used in some reports.[4] Alternatively, physiological stressors can trigger an episode of apical ballooning syndrome, such as an acute medical illness (ex. pancreatitis), exacerbation of a chronic disease (ex. congestive heart failure), or noncardiac surgical procedure, such as our patient.[3] The most common symptoms are chest pain (two thirds of the patients) and dyspnea similarly to those with acute myocardial infarction.[4] Cardiogenic shock may present in patients with severe left ventricular dysfunction,[7] Our patient presented with pulmonary edema. In ECG, ST-segment elevation is absent in two thirds of patients with SICM,[8] however our patient had ST- segment elevation on her ECG. The ECG changes at presentation time do not correlate with the severity of the ventricular dysfunction or prognosis.[8] Cardiac biomarkers are typically moderately elevated in SICM.[3] The principal criteria of SICM are: (1) acute emotional/physical stress before the onset of chest pain; (2) ischemic

abnormalities on the ECG; (3) normal epicardial coronary arteries on angiography; (4) apical ballooning with basal hyperkinesis on the left ventriculogram or echocardiogram; (5) disproportionately low release of cardiac biomarkers with respect to severity of left ventricle dysfunction and (6) rapid resolution of LV dysfunction.[9] Management of patients with SICM is overall supportive and conservative. The administration of thrombolytic agents should be avoided.[3] Left ventricular dysfunction is treated with beta blockers, angiotensin converting enzyme inhibitors, and diuretics. Additionally, beta blockers may block catecholamine excess which is the potential mechanism of SICM. Also, beta blockers decrease left ventricular outflow tract obstruction by reducing basal segment hyperkinesis.[3] The overall prognosis for SICM is good without treatment and most patients recover normal cardiac function in less than 1 month;[10] however, mortality rates range from 0% to 8%.[11] The most frequent complication is left-sided heart failure with and without pulmonary edema. Other complications are cardiogenic shock, ventricular arrhythmias, left ventricular mural thrombus, mitral valve regurgitation, pulmonary thromboembolism, and left ventricular wall rupture.[3] Physicians should assess left ventricular function recovery during outpatient follow-up and emphasize on possible complications of the disease and explain the possible causes to the patients and their families.

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