The spectrum of myocardial stress: Concurrent spontaneous coronary artery dissection and stress-induced cardiomyopathy in a patient presenting with acute ST-segment elevation myocardial infarction.

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Abstract

A 72-year-old lady without any conventional cardiovascular risk factors presented to the emergency room with severe anginal chest pain. ECG showed lateral wall ST-elevation and serial serum troponins were elevated. Emergent cardiac catheterization showed spontaneous coronary artery dissection involving the first diagonal artery with angiographically normal other epicardial coronary arteries. Left ventriculogram and echocardiogram showed a moderately reduced left ventricular systolic function with akinetic mid to distal myocardial segments and normal basal contraction suggestive of stress-induced cardiomyopathy. Spontaneous coronary artery dissection presenting with ST-elevation myocardial infarction and stress-induced cardiomyopathy is very rare.

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Abstract

A 72-year-old lady without any conventional cardiovascular risk factors presented to the emergency room with severe anginal chest pain. ECG showed lateral wall ST-elevation and serial serum troponins were elevated. Emergent cardiac catheterization showed spontaneous coronary artery dissection involving the first diagonal artery with angiographically normal other epicardial coronary arteries. Left ventriculogram and echocardiogram showed a moderately reduced left ventricular systolic function with akinetic mid to distal myocardial segments and normal basal contraction suggestive of stress-induced cardiomyopathy. Spontaneous coronary artery dissection presenting with ST-elevation myocardial infarction and stress-induced cardiomyopathy is very rare.

Keywords: Spontaneous coronary artery dissection, stress-induced cardiomyopathy, ST-elevation myocardial infarction

Introduction:

Spontaneous coronary artery dissection (SCAD) and stress-induced cardiomyopathy (SC) are different clinicopathologic entities that frequently present with the acute coronary syndrome (ACS) without underlying atherosclerotic coronary artery disease. SCAD and SC commonly present with ACS in post-menopausal women and are believed to be due to stress-induced neurohormonal surge, exposure to female sex hormones; and catecholaminergic toxicity leading to coronary microvascular dysfunction and apical stunning respectively.[1,2] SCAD is the most common cause of ACS in young women without any conventional cardiovascular risk factors, including those who are pregnant, parturient, and on oral contraceptives.[3] It is a nontraumatic tear in the epicardial coronary artery and the spectrum ranges from the intimal rupture to expanding intramural hematoma obstructing the distal flow leading to ischemia. The etiology is multifactorial and is hypothesized to be associated with exposure to female sex hormones, environmental and emotional stressors, and underlying fibromuscular dysplasia.[4] SC is usually preceded by physical or emotional stress. It causes a catecholaminergic excess state, leading to hypokinesis of the cardiac apex and hypercontractility of the base, causing classical apical ballooning. It occurs predominantly in post-menopausal women soon after exposure to emotional and physical stressors and presents with typical chest pain, ST-segment changes, and troponin elevation. It is the diagnosis of exclusion and epicardial coronary artery disease needs to be ruled out by invasive coronary angiogram. It is generally reversible but sometimes systolic dysfunction may persist, leading to chronic non-ischemic cardiomyopathy. Stress causes a surge of adrenaline (Epi) from the adrenal medulla and nor-adrenaline (NE) from cardiac nerve endings after excitation of the medullary autonomic system. Circulating adrenal Epi exerts stronger hormonal effects on the cardiac tissue than NE.[5] The basal myocardium is rich in sympathetic nerve terminals and noradrenaline whereas the apex is rich in β_1-receptors.[6,7] High NE thus results in vigorous basal contraction. However, excess Epi in the apex due to low clearance due to sparse neural uptake leads to excitotoxicity of the apical adrenoreceptors and intracellular Ca $^{2+}$overload.[2,8] This is compounded by an increased afterload state created by increased Nadr surge to the peripheral arteries leading to apical myocardial stunning which presents as classical "ballooning" of the cardiac apex. [5]

Although both SCAD and SC are common in post-menopausal women and are believed to result from stress-induced neurohormonal surge, they are rarely present concomitantly. We report a rare case of a post-menopausal lady who presented with ST-segment elevation myocardial infarction and was found to have SCAD of a non-culprit vessel and typical echocardiographic features of SC.

Case report:

A 72-year-old lady with a past medical history of recurrent stage IIA invasive ductal carcinoma of the breast, grade III, estrogen receptor-positive status post bilateral mastectomy, radiation therapy, chemotherapy, and adjuvant endocrine therapy with Anastrozole which was completed a month prior to presentation and on

remission presented with anginal chest pain. The patient was hemodynamically stable, and the cardiovascular system examination was unremarkable. ECG showed ST segment elevation in leads I and aVL, figure 1, and serial high-sensitivity troponins were elevated to 910 ng/L, 2000 ng/L, and 1400 ng/L. She was treated with Aspirin 324mg and Clopidogrel 600 mg and was emergently taken to the cardiac catheterization lab. Coronary angiogram showed type 3 spiral dissection in the first diagonal artery and normal other major epicardial coronary arteries (Video a, Video b, Video c, Video d, Video e, and Video f). The left ventriculogram showed an ejection fraction of 35%, apical akinesis, and basal hyperkinesis suggestive of stress-induced cardiomyopathy (Video g). Transthoracic echocardiography showed a moderately reduced ejection fraction of 35% with mid to distal anterior, lateral, inferior, inferoseptal, inferolateral, and anteroseptal segments with preserved basal contraction suggestive of stress-induced cardiomyopathy, Video h and Video i. Her baseline echocardiography was normal. The wall motion abnormalities along with a moderate reduction in left ventricular systolic function were typical for stress-induced cardiomyopathy and not explained by SCAD of the first diagonal artery (D1). This established the fact the initiating event was SCAD of D1 which presented with acute lateral wall STEMI and stress-induced cardiomyopathy. The patient was medically managed with dual Antiplatelets, Metoprolol, Atorvastatin, and Valsartan and discharged home in stable condition.

Discussion:

SCAD and stress-induced cardiomyopathy are the different manifestations of cardiac neurohormonal stress. The first stress-induced cardiomyopathy was first described by Iga et al[9] as reversible left ventricular dysfunction associated with pheochromocytoma. Sato et al[10] first described it as tako-tsubo-like left ventricular dysfunction in 1990. The incidence is rising, and patients present with typical chest pain, classical apical hypokinesis, and basal hyperkinesis in echocardiography with elevated cardiac enzymes mimicking acute coronary syndrome and have normal coronary angiography. These patients are treated with beta-blockers, renin-angiotensin-aldosterone system (RAAS) inhibitors with improvement in left ventricular ejection fraction. First described by Pretty in 1931,[11] SCAD has been found to be increasingly associated with myocardial infarction (MI) in premenopausal and postmenopausal women without any conventional cardiovascular risk factors; 90 % of SCAD patients are women who present with ACS in their 5^{th} and 6^{th} decades of life.[12] SCAD accounts for 35% of MIs in women <50 years and is the most common cause of MI in pregnancy. Studies suggest SCAD accounts for less than 1% of all MI overall and is very uncommon in males.[1,13] There are three types of SCAD, type 1: multiple radiolucent lumens or contrast staining of the vessel wall, type 2: diffuse stenosis with a sudden change in vessel caliber, and type 3: focal or tubular stenosis.[14] SCAD has a predilection for the distal vessel and in terms of coronary distribution, the left anterior descending artery is most affected, and its diagonal and septal branches are involved in 45%-61% of cases, followed by the circumflex artery and its branches in about 30% and the right coronary artery and its branches in abound 15% .[15] SCAD is initially diagnosed by invasive coronary angiography. Intracoronary imaging modalities like intravascular ultrasound (IVUS) and optical coherence tomography (OCT) have better spatial resolution and can be performed if a coronary angiogram is inconclusive.[14] Conservative management is recommended as studies show spontaneous angiographic healing in 90% of patients within a month.[16] Percutaneous coronary intervention (PCI) can lead to an increased risk of complications. The coronary guidewire may enter the false lumen and balloon angioplasty and stent placement may lead to the propagation of dissection or stent malapposition.[17] PCI is only indicated for patients with ongoing ischemia or hemodynamic instability. Coronary artery bypass grafting is performed if PCI fails, or the patient has proximal or left main dissection, or refractory ischemia despite conservative management.[16] These patients should be hospitalized for 3-5 days as chances of early clinical deterioration and new recurrent dissection are high.[18] Treatment with antiplatelets is controversial and dual antiplatelets are used for 12 months only after PCI as per the guidelines. Beta-blockers are proven to reduce recurrence.[19] Mortality is low at 1-2% and the incidence of recurrent SCAD and MI is about 18%.[19]The patients frequently present to the clinic after discharge with chest pain, due to sequelae of dissection or underlying psychologic stress, depression, and anxiety from SCAD, hence should be closely followed up.

Conclusion

Stress-induced cardiomyopathy and SCAD are different pathophysiologic manifestations of the same etiologic process and may present concurrently. They masquerade as acute myocardial infarction due to atherosclerotic coronary artery disease in their clinical presentation and differentiation is important as treatment, outcome, and prognosis are different. Detailed evaluation for SCAD in these patients is important during a coronary angiogram; IVUS or OCT should be used additionally if the angiogram is inconclusive as SCAD tends to affect the branch vessels and distal small vessels which may be undetected with conventional angiogram only.

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Figure 1: ECG showing ST-elevation in leads I and aVL with reciprocal ST-depression in leads III and aVF

Video a: Coronary angiogram LAO cranial view after left coronary artery intubation showing type 3 dissection involving proximal first diagonal artery (D1) and normal circumflex (Circ) and left anterior descending coronary artery (LAD)

Video b: Coronary angiogram RAO cranial view after left coronary artery intubation showing type 3 dissection involving D1 and normal LAD

Video c: Coronary angiogram LAO caudal view after left coronary artery intubation showing type 3 dissection involving proximal first diagonal artery and normal circumflex and LAD

Video d: Coronary angiogram RAO caudal view after left coronary artery intubation showing type 3 dissection involving the first diagonal artery and normal circumflex and LAD

Video e: Coronary angiogram LAO cranial view after right coronary artery intubation showing a normal proximal and distal right coronary artery (pRCA and dRCA), right posterior descending artery (RPDA) and right posterolateral (RPL) branches

Video f: Coronary angiogram RAO cranial view after right coronary artery intubation showing a normal proximal, mid and right coronary artery and branches

Video g: Left ventriculogram in RAO cranial projection showing anteroapical (A) akinesis with hypercontractile base (B)

Video h: Transthoracic echocardiography, apical two chamber view showing akinetic mid to distal inferior and anterior walls

Video i: Transthoracic echocardiography, apical four chamber view showing LV ejection fraction of about 35% with akinetic mid to apical inferoseptal, septal and lateral segments with preserved basal contractility

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