Case report

Spontaneous coronary dissection revealed by ventricular tachycardia in a female patient: A case report and review of literature

Abstract:

Spontaneous coronary artery dissection is an unusual cause of acute coronary syndrome or sudden death, described in patients with few atherosclerotic risk factors.

A 53-years-old female patient presented to the emergency department with palpitations and spontaneous severe retrosternal chest pain followed by lipothymia. The admission EKG showed a ventricular tachycardia spontaneously resolutive [11]. A transthoracic echocardiogram demonstrated dilated ventricles with severe regional wall motion abnormality and impaired left ventricular ejectional fraction. Cardiac catheterization showed an intimal dissection involving the mid segment of the left anterior descending artery. The patient had a conservative treatment with a stable angiographic control 1 month later.

Key words: Artery, Coronary, Dissection, Coronary Angiography

Introduction

Spontaneous coronary artery dissection (SCAD) is a nonatherosclerotic etiology of acute coronary syndrome, including sudden cardiac death, which frequently affects younger women. SCAD is usually diagnosed on invasive coronary angiography; intracoronary imaging can be used for

confirmation in indeterminate cases, often by visualizing an intramural hematoma. In some, intravascular imaging demonstrates the presence of hematoma without a discrete tear in the coronary wall, suggesting that SCAD may also arise from spontaneous bleeding within the vasa vasorum [12]. The present case study discuss about spontaneous coronary dissection revealed by ventricular tachycardia in a female patient.

Case presentation:

A 53-years-old female patient presented to the emergency department complaining of spontaneous severe retrosternal chest pain occurring 6 hours previously followed by lipothymia. Those symptoms appeared while the patient was climbing stairs. There was no history of hypertension, diabetes mellitus, dyslipidemia or smoking. However, the patient was menopaused a year ago, and obese with a body mass index of 35. She had no significant family history of premature coronary artery disease. The patient had an orthostatic hypotension for twenty years with no actual treatment and a podagra under allopurinol. The review of symptoms was otherwise negative.

On admission, the patient was in no acute distress. Vital signs showed a regular spontaneous pulse at 75 and a blood pressure of 117/67. Carotid impulses were normal and the jugular venous pressure was not raised. Cardiac examination revealed normal heart sounds and there were no appreciable cardiac murmurs, rubs, or gallops. Lungs were clear.

Differential diagnosis:

1. Acute coronary syndrom
2. MINOCA
3. Coronary dissection

Investigations:

The ECG (figure 1) performed right while the episode of palpitations showed a regular wide QRS tachycardia with a TV score of 6 suggesting a ventricular tachycardia. This arrythmia had spontaneously resolved, and the next ECG (figure2) showed a complete left bundle branch with a left axis and a premature ventricular complex.

Serial troponin level drawn on admission were 617 times the normal amount.

A transthoracic echocardiogram (Figure 3a and b) demonstrated dilated ventricles with severe regional wall motion abnormality over the apex and adjacent walls, anteroseptal and anterior walls with a severe left ventricle dysfunction (left ventricle ejectional fraction of 25%). A moderate mitral regurgitation was noted.

A cardiac MRI (Figure 5) was performed 7 days later to explore further the left ventricular dysfunction and showed an ischemic cardiomyopathy with dilated left ventricle with severe left ventricular dysfunction. A non-viable necrosis focus in the left anterior descending artery's territory was noted.

Medical management and interventions:

The patient was given 300mg of clopidogrel and 320 of aspirin orally. Low molecular weight heparin at a curative dose and the patient was taken to the cath lab.

Cardiac catheterization with coronary angiography was performed using a right radial artery approach. Coronary angiography (figure 4) showed a radiolucent linear defect suggestive of an intimal dissection involving the mid segment of the left anterior descending artery with Thrombolysis in Myocardial Infarction (TIMI) grade 2 flow.

A conservative treatment was opted and the patient was maintained on medical management, including clopidogrel, aspirin, betablocker, amiodarone, spironolactone and sacubitril/valsartan association with close outpatient follow-up. Advice on lifestyle modifications were provided to the patient, physical exercise and body weight loss.

Follow-up coronary angiography at 1 month demonstrated a stable appearance of the coronary angiography.

Discussion:

The first case of SCAD was described in 1931 in the autopsy of a 42-year-old woman. (1) The true prevalence of SCAD remains uncertain. SCAD can affect both sexes across the life span. However, approximately 90% of patients are woman who present between 47 and 53 years of age (2) which is consistent with our case.

Vessel wall weakness is an important substrate for development of spontaneous coronary artery dissection. It may also be associated with autoimmune and collagen vascular diseases, or intense physical exercise (3).

In fact, further biological tests were carried out in our case to look for a systemic disease; all of them were normal.

In more than 90% of patients who survive to initial evaluation, SCAD manifests as myocardial infarction. Approximately 20 to 50% of patients present with STEMI (4–6) (7,8), 3 to 5% present with ventricular arrythmias (9,10) and 2% present in cardiogenic shock.

The suspicion for SCAD is typically instigated by clinical presenting features such as patient demographics, especially young age, female sex, and few or no conventional cardiovascular risk factors. Conventional coronary angiography remains the first-line diagnostic imaging method because it is available and recommended for early invasive management of ACS. The left anterior descending artery is the most commonly affected.

Dedicated intracoronary imaging methods, including intravascular ultrasonography IVUS and optical coherence tomography OCT, provide detailed visualization of the arterial wall that aids the diagnosis of SCAD. Neither IVUS nor OCT were provided in our case and the diagnosis was based only on the coronary angiography aspect.

There are no guidelines regarding optimal treatment of this condition. Treatment is guided by the clinical symptoms, extent and location of the dissection, and the hemodynamic status of the patient. Percutaneous coronary strategies are reasonable in acute cases with proximal dissection and arterial occlusion in order to restore coronary perfusion and hemodynamic

stability. However, PCI in a coronary dissection presents a unique challenge as it may prove difficult to identify the true arterial lumen and secure proper guidewire placement.(1)

Conservative medical therapy is reasonable in cases of distal dissection with preserved coronary flow. Reduction of vessel wall sheer stress with the use of beta-blockade is a reasonable therapeutic choice. Use of heparin, thrombolysis, and glycoprotein IIb–IIIa inhibitors during the acute presentation is controversial. A conservative medical therapy was chosen for our case.

Conclusion:

Accurate diagnosis of SCAD in the early stages of ACS presentation is important because management and investigation are different from those for atherosclerotic forms of coronary artery disease.

An understanding of the inherent difficulties and potential complications of manipulating dissecting coronary arteries should help guide the risk/benefit assessment of the therapeutic decision-making when confronted with this unusual angiographic finding.

Learning points:

- The prevalence of SCAD can be underestimated because patients may receive alternative diagnoses
- Accurate diagnosis of SCAD in the early stages of ACS presentation is important because management and investigation are different
- There are no guidelines regarding optimal treatment of this condition

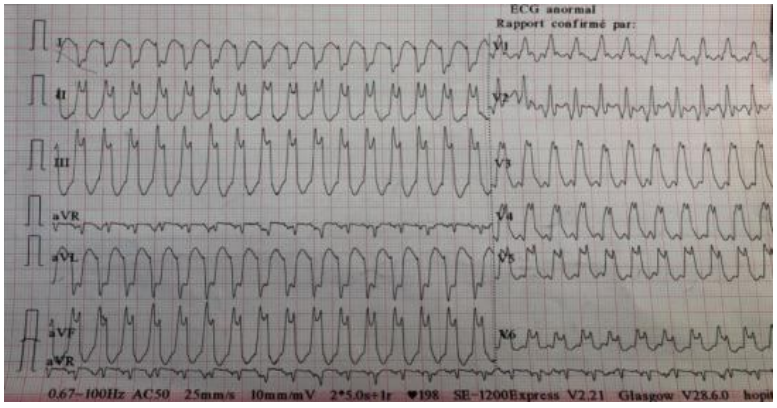

Figure 1:EKG showing a regular wide QRS tachycardia

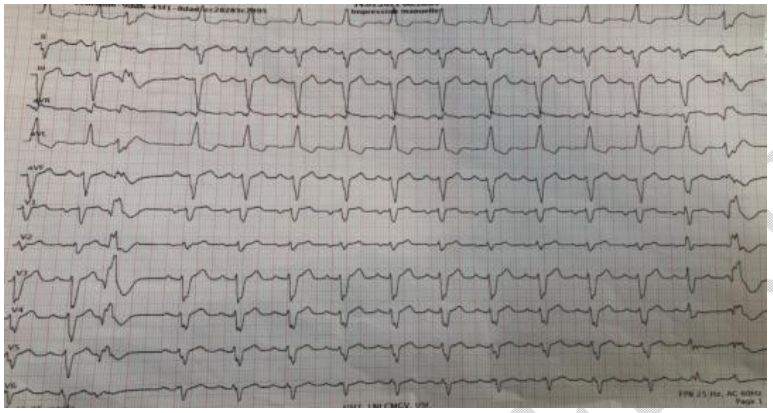

Figure 2: EKG showing a complete left bundle branch

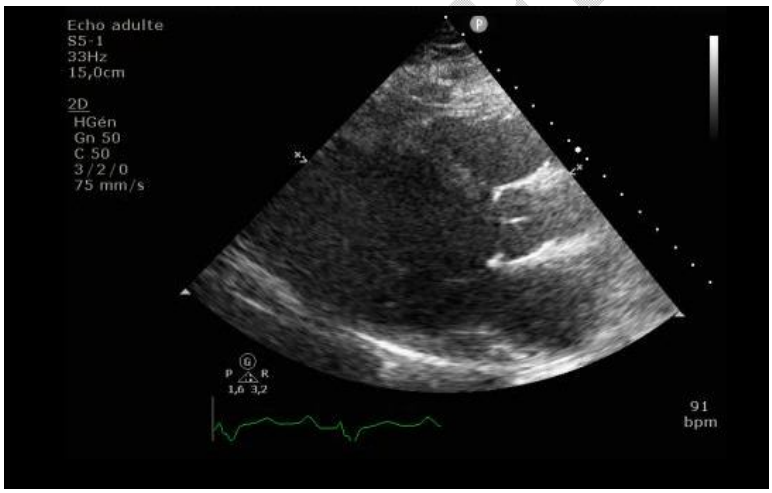

Figure 3a: Parasternal long axis showing dilated left ventricle

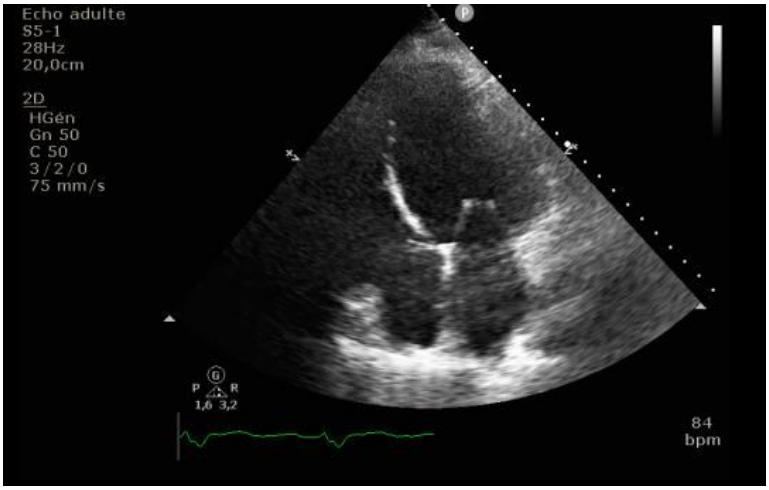

Figure 3b: Apical-4-chamber view showing globular shape of left ventricle

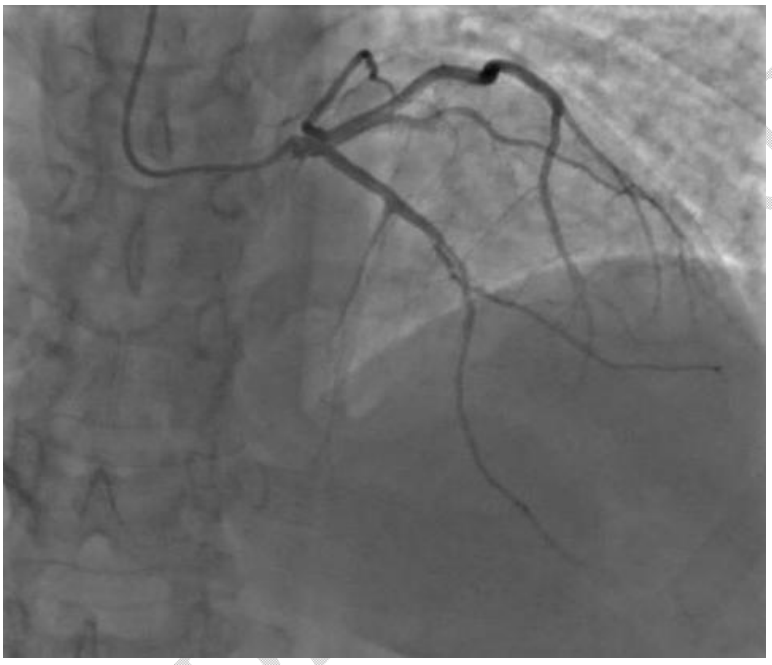

Figure 4: Coronary angiography demonstrating an intimal dissection involving the mid segment of the left anterior descending artery

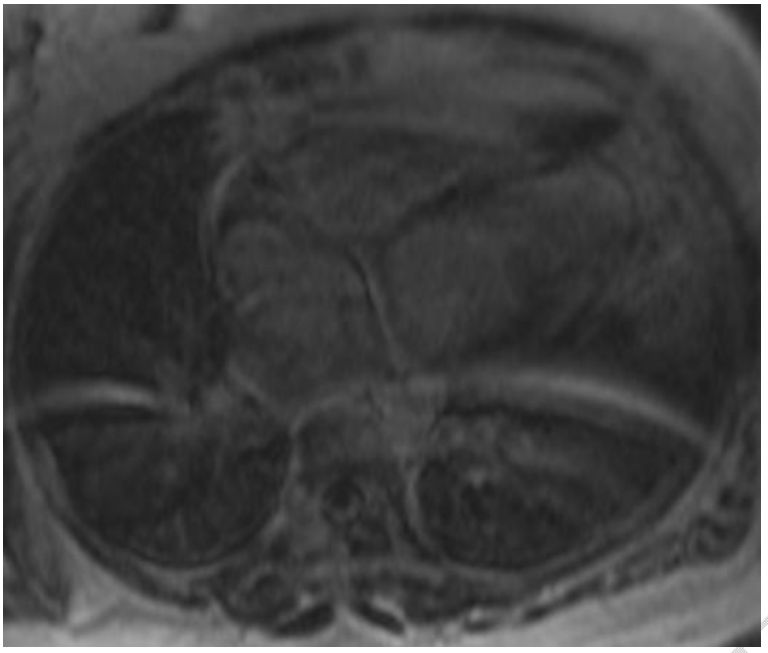

Figure 5: Cardiac MRI showing a non-viable necrosis focus in the left anterior descending artery's territory

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