Case Report

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Puzzle of the Fifth Ventricle: Left Ventricular Dissection Post Myocardial Infarction Resulting into Formation of a Neo-cavity: A Case Report and Literature Review

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Abstract

Left ventricular dissection is an exceedingly rare and atypical mechanical complication of myocardial infarction with high mortality. It is considered a sub-acute phase of cardiac rupture, which has a subtle presentation and often remains undiagnosed. Presented here is a case report of a 54-year-old female who developed features of acute heart failure 1 month following an anteroseptal wall myocardial infarction and was later diagnosed to have left ventricular dissection on echocardiogram. This case highlights few facts on ventricular dissection, which has been sparsely reported or explained in literature.

Keywords: Cardiac rupture, echocardiogram, myocardial infarction, ventricular dissection

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Introduction

Left ventricular dissection is a mechanical complication following myocardial infarction, explained by the dissection of spiral myocardial fibers. The literature describes ventricular dissection as an acute or chronic mechanical complication following myocardial infarction.[1] However, there are only a few reported cases worldwide where acute myocardial infarction is complicated by a rarely encountered left ventricular dissection. Moreover, due to its subtle presentation, the diagnosis is often missed.

Case report

A 54-year-old ex-smoker female presented to the emergency department with a chief complaint of chest discomfort associated with shortness of breath and generalized body swelling. There was no history of fever. She was diagnosed to have recent anteroseptal wall myocardial infarction with apical aneurysm and heart failure with reduced ejection fraction (LVEF: 30%) about a month back. She was on guideline-directed standard medical therapy for myocardial infarction with heart failure with reduced ejection fraction.

On examination, she was in cardiogenic shock. Hence, she was immediately resuscitated with inotropic support (dobutamine). The laboratory profiles were grossly normal except for a slightly raised International Normalized Ratio (INR) of 1.6. An Electrocardiogram (ECG) depicted Q waves in V1-V3, poor R wave progression, and persistence of ST elevation in precordial leads (Figure 1). Echocardiogram revealed an intramural area of significant echo-lucent region (neo-cavity) involving mid and apical anteroseptal segment within the myocardium mimicking appearance of an extra ventricle. However, there was no color flow within the neo-cavity. Moreover, there was dyskinetic movement of anteroseptal wall

with borderline dilated LV and grossly reduced left ventricular ejection fraction (Figure 2, Figure 3, Figure 4). Relying on ECG & echocardiogram, a working diagnosis of left ventricular dissection was made. Anti-platelet therapy was stopped from guideline directed medical therapy while supplemental oxygen and inotropic support were continued. Gradually, her hemodynamics stabilized over the next few days. Dobutamine was tapered and discontinued on day 3. A cardiac MRI was planned; however, due to financial constraints in our resource-limited setting, it could not be performed. She was continued with oxygen therapy, guideline directed medical therapy and other supportive treatments when, on day 10, her family decided to care for her at home before successful weaning from supplemental oxygen could be achieved despite proper counseling regarding diagnosis, treatment, and prognosis.

Discussion

This case highlighted the need to consider ventricular dissection as one of the differential diagnoses for complications following myocardial infarction. Though rare in occurrence, it is a known grave complication. As patient presented at emergency department in cardiogenic shock, our focus was stabilization of the patient and thus we failed to acquire the standard echocardiographic views. Moreover, because of various social and financial constraints in resource limited setting like ours, we have to discharge the patient on family request and thus we could not exactly know the outcome of the patient. Despite these challenges, with this case we were at least able to highlight few informative facts on ventricular dissection.

Left ventricular dissection is a commonly mentioned mechanical complication of myocardial infarction; however, its clinical encounter is rare. Ventricular dissection is described as the splitting or cracking of the myocardium of the ventricles, creating a fracture in the ventricle. The outer wall of the ventricle consists of pericardium, epicardium, and some portions of myocardium, whereas the inner wall consists of the remaining myocardium and endocardium resulting in formation of new cavity in between these layers.[2]

Left ventricular dissection in the setting of myocardial infarction can be portrayed in three different ways: a) sequel of myocardial infarction with the remodeling process, b) reperfusion injury following resuscitation, and c) as a no-reflow phenomenon following revascularization.3 The myocardium of the left ventricle after myocardial infarction, loses its integrity thus providing poor support to adjacent tissues, which is further aggravated by the abrupt rise of intracavitary pressure following increased perfusion to coronary capillaries as a part of compensatory/reperfusion phenomenon, and finally contributes to initiating the rupture of small vessels resulting in intramyocardial hematoma formation. This is the initial step in the pathological process. With the nidus of hematoma, a series of events occur inside the infarcted myocardium. Subsequently, this hematoma can have different fates. It may expand gradually to form a new cavity, expand rapidly, rupture into adjacent structures, expand and communicate to adjacent cavities, or get partially/completely reabsorbed to remain silent, depending on the spectrum of ischemic injury, local as well as systemic hemodynamic response to the applied revascularization.[2-7]

Literature has many names for ventricular dissection, viz, dissecting hematoma, intramural hematoma, and intramyocardial dissecting hematoma.[2-7] Lunseth was the pioneer to describe this pathophysiological phenomenon in his necropsy series in 1956, where he observed that the hematoma, rather than infiltrating and destroying the adjacent connective tissue, dissects and separates the myocardial fibers.8 The hematoma behaves like a chisel, mechanically splitting helical fibers of the infarcted myocardium, forming a neo-cavity. This temporal progression of hematoma leads to a varied spectrum of clinical entities, ranging from ventricular dissection, communicating pseudoaneurysm formation, and complete cardiac rupture.[2,3]

Vargas-Barŕon et al. in their case series discovered the incidence of ventricular dissection to be 21% of total cases of cardiac rupture, whereas cardiac rupture occurred in 3.5% of myocardial infarction cases.[2,3] Hence, in such scenario of limited available epidemiological evidence, a deductively calculation claims the incidence of ventricular dissection to be 0.7-0.8%. However, because of rarity of ventricular dissection, its actual incidence is not yet known. The paucity of data and literature on ventricular dissection is the reason for explaining it in case reports and case series. To date, about 50 cases of ventricular dissection have been reported in the literature, including this case.[9]

Patient presents with varied symptoms and signs, including chest pain, shortness of breath, palpitation, syncope, arrhythmias, and blocks, features of heart failure, or cardiogenic shock.[7,9] Persistence of ST elevation beyond 72 hours of myocardial infarction should produce high suspicion for ventricular dissection.[2] Echocardiography remains the investigation of choice for diagnosis and subsequent follow-up and management, as it discloses various spectrums of ventricular dissection. ie, extension, reabsorption, and communication with other ventricles. Echocardiographic diagnosis of ventricular dissection is made with the presence of at least three of the following signs: (1) the formation of one or more neo cavitations within the tissue with an echo-lucent center, (2) a thinned and mobile endomyocardial border surrounding the cavitary defect, (3) ventricular myocardium identified in the regions outside of the cystic areas, (4) changes in the echogenicity of the neo-cavitation suggesting blood content, (5) partial or complete absorption of the cystic structure, (6) continuity between the dissecting hematoma and one of the ventricular cavities, (7) communication between the two ventricular chambers through the myocardial dissection, and (8) Doppler recording of flow within the dissected myocardium.[2,3,10] In the present day, with the advent of medical sciences and technology, cardiac Magnetic Resonance Imaging (MRI) remains the gold standard for diagnosis. Steady state-free precision MRI protocol using T1 & T2-weighted images with fluid and fat flair defines pathology, anatomical abnormality, its extension, and differentiation from other differential diagnoses.[11,12] The differential diagnoses of ventricular dissection are pseudoaneurysm, takotsubo cardiomyopathy, and LV cavity thrombus.[2]

A meta-analysis by Leitman et al. found that 80% ventricular dissection was due to myocardial infarction. Other causes included cardiac surgery and intervention, trauma, takotsubo cardiomyopathy, and a few rarer causes, including echinococcus.[9,13,14] The mean time of diagnosis of ventricular dissection was 9 days, and in-hospital mortality was 23%. The predictors of mortality with ventricular dissection were ejection fraction <35%, age> 60 years, myocardial infarction, and late diagnosis (after 24 hours of symptoms).[9] Ventricular dissection was considered equivalent to subacute myocardial rupture.[9,15]

The rarity of diagnosis has compelled us to follow an individualized approach for management of ventricular dissection. As a rule of thumb, conservative management for clinically stable patients

carrying the theme of non-maleficence and surgical correction for high-risk individuals remains the treatment strategy.[16-18] Various surgical approaches, including patch repair, necrotomy, including cardiac transplantation, have been highlighted in the literature.[17,18] Mortality after dissection is high, ranging from 47% to 78% depending on the part of the LV wall involved.[2] Prognostically, apical dissection has benign courses and can be managed conservatively; however, septal and or extensive dissection has a poor prognosis and should be considered in the line of surgical management. Spontaneous resolution occurs in some cases over 6-12 months. A multi-disciplinary team approach is the key for management.[1]

Conclusion

Left ventricular dissection is a rare presentation of a common clinical entity. Gaining knowledge about the natural history of ventricular dissection and its pathophysiology lays the foundation for recognizing this uncommon diagnosis promptly, especially when encountering atypical presentations of common diseases. Moreover, the insights gained from this report contribute to the growing body of knowledge regarding Left ventricular dissection. Further case reports/series are needed to explore the optimal management and long-term prognosis for ventricular dissection.

Declaration of patient consent

Written informed consent was obtained from the patient for publication of clinical details and images.

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Conflict of interest

None.

Puzzle of the fifth cavity: A case of left ventricular dissection following acute myocardial infarction

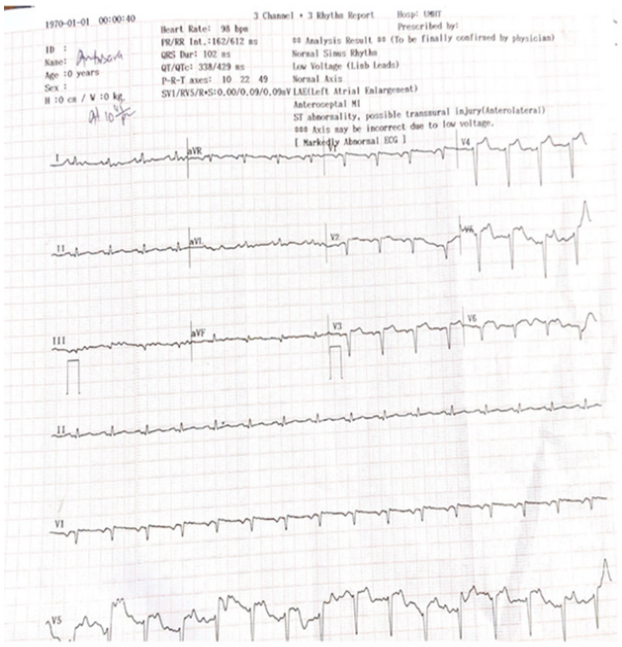

Figure 1: *ECG showing Q wave in V1-V3 and persistent ST elevation in all precordial leads (V2-V6) with poor R wave progression*

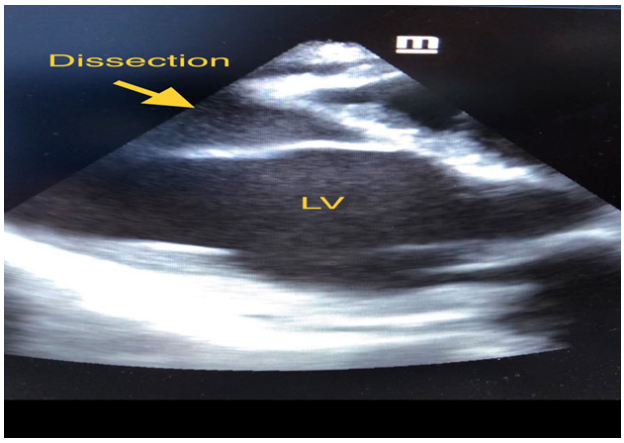

Figure 2: *Echocardiography imaging showing left ventricular dissection involving mid and apical anteroseptal segment*

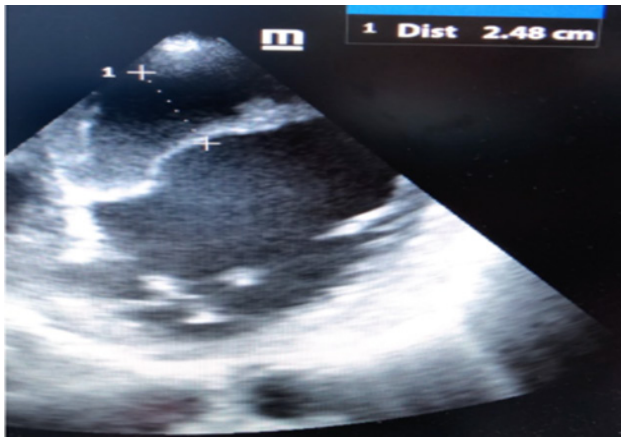

Figure 3: *Echocardiography image showing the size of the dissected cavity between the outer layer (myocardium and pericardium) & inner layer (thin myocardium and endocardium)*

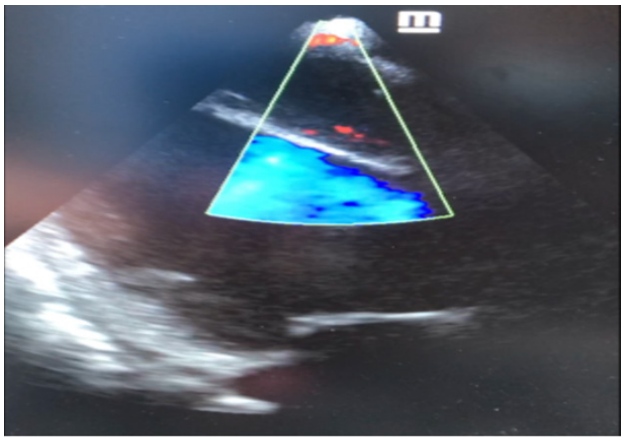

Figure 4: *Echocardiography image showing absent color flow doppler effect inside neo-cavity*

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