Volume 1 Issue 3
www.escientificlibrary.com

ISSN: 2768-0533

ES Journal of Cardiology

Delayed Rupture of Left Ventricular Aneurysm with Resultant Coexisting Pseudoaneurysm

Case Report

Adel Ekladious

Department of General Medicine, Bunbury Regional Hospital (South West Health Campus), Australia

Received: May 28, 2020; **Accepted:** June 15, 2020; **Published:** June 18, 2020

***Corresponding author:** Dr Adel Ekladious, Department of General Medicine, Bunbury Regional Hospital (South West Health Campus), Bunbury, Western Australia, Australia

Abstract

A 60 year old gentleman was transferred to a regional hospital following an admission for non-ST elevation myocardial infarction (NSTEMI) complicated by true left ventricular apical aneurysm and pyopericardium. During admission he had delayed rupture of the aneurysm leading to the development of a concurrent pseudoaneurysm – a rare event. The onset of pseudoaneurysm was difficult to determine as the patient was asymptomatic, and limited access to cardiac CT (computerised tomography) and cardiac MRI (magnetic resonance imaging) made diagnosis difficult.

Background

Left ventricular aneurysm (LVA) is an uncommon complication of transmural myocardial infarction (MI). There are two types of left ventricular aneurysm, true aneurysm and false aneurysm (pseudoaneurysm), which differ in pathophysiology and risk of rupture. True aneurysm is a late stage complication of MI caused by cardiac remodelling and characterised by well-delineated thinning of the ventricular wall that lacks muscle and is either akinetic or dyskinetic during systole.(1) Due to the development of fibrotic scar tissue after two to three weeks, the wall of a true aneurysm is able to withstand left ventricular pressure, and rupture of an established true LVA is very rare.(1, 2) In contrast, pseudoaneurysm tends to occur early after MI when rupture of left ventricular free wall endocardium and myocardium is contained only by pericardial adhesions [1,2]. Even after fibrosis occurs, left ventricle pseudoaneurysm is high risk for fatal rupture, and for this reason pseudoaneurysm requires urgent surgical repair. This is in contrast to true aneurysm, which can often be managed medically.

The incidence of LVA has declined over the past decades due to major improvements in the management of patients with acute myocardial infarction, in particular the use of thrombolytic agents and/or percutaneous coronary intervention, and administration of afterload-reducing agents [3]. LVA is associated with a number of complications including ventricular arrhythmias, thromboembolism due to mural thrombus, congestive heart failure, and very rarely rupture of the ventricular wall. Small to moderately-sized true ventricular aneurysms can be treated with medications to reduce left ventricular afterload and prevent ventricular remodelling; however large true ventricular aneurysms (and those that do not respond to medical management) can be treated with elective surgery which aims to reverse ventricular remodelling and resolve heart failure [4].

Case Presentation

A 60-year-old male publican with a history of alcohol excess was transferred to a regional hospital for ongoing

care after a prolonged complex admission to the Coronary Care Unit at a tertiary service for management of non-ST elevation myocardial infarction and pyopericardium with methicillin-sensitive staphylococcus aureus requiring urgent pericardiocentesis and treatment with intravenous flucloxacillin. His coronary angiogram demonstrated chronic total occlusion of the left anterior descending coronary artery (LAD) with retrograde collateral circulation from the right coronary artery (RCA) and left circumflex artery (LCx); and he had severe ostial OM1 disease which was stented. Left ventriculography showed severe segmental systolic dysfunction with mid-distal antero-apical, distal infero-apical hypokinesia, and moderate LV apical aneurysm, which was confirmed on transthoracic echocardiogram.

One month into his admission at our hospital, the patient developed increasing abdominal pain and abdominal distention. He remained haemodynamically stable and a febrile, but abdominal examination revealed ascites without evidence of peritonism. The patient underwent CT Abdomen which noted bilateral small pleural effusions, ascites and liver cirrhosis with portal hypertension, as well as an incidental finding of interval development of large aneurysm/pseudoaneurysm arising from the aneurysmal left ventricular wall measuring approximately 10 cm by 5 cm.

Investigations if relevant

Repeat transthoracic echocardiogram was performed to further assess the pseudoaneurysm, and demonstrated large haemopericardium communicating with the left ventricle across a perforated apex, with severely impaired left ventricular function. Further targeted imaging with CT Chest confirmed interval rupture of broad moderate-sized aneurysm of the mid and distal LV into the pericardial or epicardial space – this was not distinguishable on CT imaging (Figure 1). The defect was 30 mm in diameter, located inferior to the apex wrapping around the lateral wall of the LV and was concealed (that is, a pseudoaneurysm). The pseudoaneurysm was 60 mm thick at the apex and 40 mm overlying the LV. There was no chamber compression, specifically, no RV compromise.

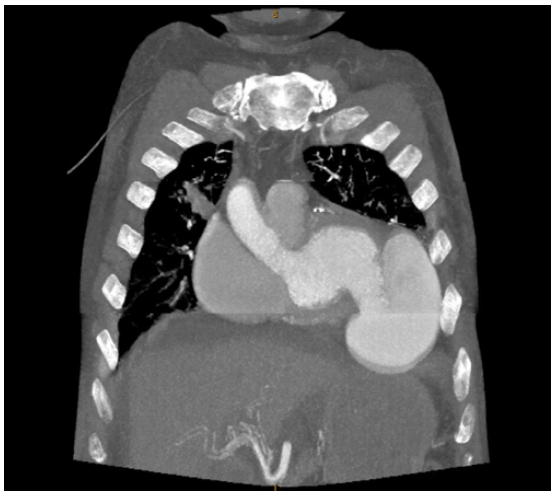

Figure 1: CT imaging of ruptured LV aneurysm

Differential diagnosis if relevant

Treatment if relevant

Outcome and follow-up

Following diagnosis of an asymptomatic ruptured true LV apical aneurysm resulting in coexisting true LV aneurysm and pseudoaneurysm, he was urgently transferred to a tertiary hospital for cardiothoracic opinion. He was ultimately deemed not suitable for surgical intervention given his high surgical risk, compounded by existing medical comorbidities and medical frailty. He was subsequently transferred to a peripheral hospital for palliation and died two weeks later.

Discussion

Left ventricular aneurysms and pseudoaneurysms are two potential complications of myocardial infarction that can lead to death. True aneurysm usually results from gradual cardiac remodelling following myocardial infarction, and more frequently involves the anterior wall of the left ventricle [1,4]. The scar tissue of the aneurysm wall gradually fibroses and is therefore very unlikely to rupture. Other causes of true aneurysm include hypertrophic cardiomyopathy and Chagas disease, both of which can lead to apical aneurysm. A pseudoaneurysm, or false aneurysm due to myocardial infarction occurs when there is acute intrapericardial rupture of the ventricular free wall, contained by adhesions of the overlying pericardium to the epicardium. Without myocardial involvement, pseudoaneurysm walls are weak, and are associated with a 30 to 45% risk of rupture, and mortality of almost 50% with non-surgical therapy. In contrast to true aneurysms, pseudoaneurysms occur more frequently in the posterior basal segment of the left ventricle [1,4]5

This case is significant for a number of reasons; the first is the atypical nature of presentation. While free

left ventricular wall rupture (usually resulting in death) can occur acutely following myocardial infarction, the occurrence of an established true left ventricular aneurysm resulting in delayed rupture is extremely uncommon [6]. In this case the exact timeframe between diagnosis of left ventricular aneurysm and rupture is unclear, but serial echocardiogram monitoring demonstrated a period of 2 months where the left ventricular aneurysm remained largely unchanged. Differentiation between true ventricular aneurysm and pseudoaneurysm can be very difficult. True ventricular aneurysm does not have specific physical signs, but patients with pseudoaneurysm may have pericardial friction rub, new machinery murmur, reduced heart sounds, sinus bradycardia, or junctional rhythm (our patient did not have any of these).

Another remarkable component of the case is the resultant coexisting left ventricular true aneurysm and pseudoaneurysm following rupture. There are few cases of coexisting true aneurysm and pseudoaneurysm described in the literature, and like this case, most false aneurysms arose from the anterior and antero-apical regions. Usually rupture of aneurysm or pseudoaneurysm leads to haemopericardium which results in cardiac tamponade and sudden death [7], but this was not the case with our patient. We suggest that the patient did not die suddenly because the site of aneurysmal rupture (postero-lateral and inferior to the apex) resulted in containment by the pericardium and inflammatory adhesions, thereby preventing cardiac tamponade.

Diagnosing coexistence of left ventricular aneurysm and pseudoaneurysm in the same patient is technically difficult, often requiring cardiac CT and cardiac magnetic resonance imaging (MRI). An added challenge in our case was the difficulty of differentiating between true aneurysm and pseudoaneurysm in the setting a regional hospital where advanced imaging modalities such as cardiac CT and cardiac MRI were not available. Particularly in a case of asymptomatic rupture, history and examination (including ECG) are often non-specific, and definitive diagnosis by transthoracic echocardiogram is made in only 26% of patients [8]. The most reliable method for diagnosing pseudoaneurysm is angiography, with typical findings of a narrow orifice leading to a saccular aneurysm on left venticulography [9]. Recent literature suggests cardiac MRI is the most ideal non-invasive imaging modality for diagnosis of pseudoaneurysm due to the high spatial resolution and tissue characterisation, as well as the use of late gadolinium enhancement in assessing the extent of prior transmural infarcts. If patients are unable to undergo cardiac MRI, cardiac CT also offers excellent visualisation of the myocardium and coronary arteries. At the regional hospital where the patient was admitted, there were no facilities for cardiac MRI and the CT scanner was unable to be programmed for cardiac CT, so pseudoaneurysm was diagnosed using a combination of transthoracic echocardiogram and non-cardiac protocol contrast CT.

Learning points/take home messages 3-5 bullet points

- Left ventricular aneurysm is a rare occurrence after myocardial infarction
- While true LV aneurysm doesn't often rupture, it is still possible
- Coexisting LV true aneurysm and pseudoaneurysm is a very rare event
- Coronary angiography is gold standard for diagnosis of true vs pseudoaneurysm, with cardiac MRI used preferentially for non-invasive imaging; but it can still be diagnosed on CT chest

References

1. Marzlin KM. Ventricular Aneurysm: Diagnosis and Treatment. AACN Adv Crit Care. 2017;28(4):391-4.
2. Das AK, Wilson GM, Furnary AP. Coincidence of true and false left ventricular aneurysm. Ann Thorac Surg. 1997;64(3):831-4.
3. Tikiz H, Balbay Y, Atak R, Terzi T, Genc Y, Kutuk E. The effect of thrombolytic therapy on left ventricular aneurysm formation in acute myocardial infarction: relationship to successful reperfusion and vessel patency. Clin Cardiol. 2001;24(10):656-62.
4. Zoffoli G, Mangino D, Venturini A, Terrini A, Asta A, Zanchettin C, et al. Diagnosing left ventricular aneurysm from pseudo-aneurysm: a case report and a review in literature. J Cardiothorac Surg. 2009;4:11.
5. Sousa P, Santos W, Cordeiro P, Pereira S, Ferrinha R, Brandao V, et al. Pseudoaneurysm inside of a true aneurysm. J Cardiothorac Surg. 2013;8:97.
6. Chao HH, Lu MJ, Hung CR. Spontaneous rupture of left ventricular true aneurysm. Ann Thorac Surg. 2005;80(3):1106-8.
7. Frances C, Romero A, Grady D. Left ventricular pseudoaneurysm. J Am Coll Cardiol. 1998;32(3):557-61.
8. Kupari M, Verkkala K, Maamies T, Hartel G. Value of combined cross sectional and Doppler echocardiography in the detection of left ventricular pseudoaneurysm after mitral valve replacement. Br Heart J. 1987;58(1):52-6.
9. Alapati L, Chitwood WR, Cahill J, Mehra S, Movahed A. Left ventricular pseudoaneurysm: A case report and review of the literature. World J Clin Cases. 2014;2(4):90-3.