Case Report

Accidently Discovered Big Basal Left Ventricular Pseudoaneurysm: Case Report and Review of Literature

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Abstract

The left ventricular pseudoaneurysm (LVPA) is rare but potentially fatal complication post myocardial infarction (MI). The surgical experience is limited and its surgical treatment remains still a challenge with an elevated mortality. We present our experience with acquired post MI huge LVPA that was accidently discovered in a 39 years old man with a history of more than one year posterior MI and 1ry percutaneous coronary intervention/obtuse marginal coronary artery (PCI/OM).

Keywords: Left ventricular pseudoaneurysm; Myocardial infarction and Rupture LV wall

Abbreviations: Myocardial infarction: MI; Left ventricular pseudoaneurysm: LVPA; Percutaneous coronary intervention: PCI; Transesophageal echocardiography: TEE; Ejection fraction: EF; computerized tomography: CT; Magnetic resonance imaging: MRI

Introduction and Background

Rupture of the free wall of the left ventricle (LV) is a catastrophic mechanical complication occurring in 23% of those who die of MI. About 0.2% of infarcted patients were complicated with LV pseudoaneurysm, but yet in general the precise incidence is unknown, but it is even less common than cardiac rupture after MI, which has an incidence of 2-4% [1,2]. Rupture of the free wall is four to five times more common than septal rupture and is usually immediately fatal except when it converts to LVPA [3, 4]. The LVPA results when there is a small rupture occurs in the myocardium where the overlying pericardium is sufficiently adherent to the epicardium which leads to a localized hemopericardium. Persistent communication of the hemopericardium with the LV cavity results in gradual expansion of the hemopericardium into a false aneurysm [4, 5].

Pseudoaneurysms are often asymptomatic and incidentally discovered on imaging tests [5]. Even if symptoms and signs are present, there are neither sensitive nor specific for aneurysm or pseudoaneurysm and high index of suspicion is needed to avoid missing the diagnosis [5,6]. One should be vigilant to look out for mechanical complications in patients presenting with delayed MI especially when there is a sudden change in the hemodynamic status [6].

Given the propensity for pseudoaneurysms to rupture leading to cardiac tamponade, shock, and death, compared with a more benign natural history for true aneurysms, accurate diagnosis of these conditions is important.

True aneurysm, usually, calls for an elective surgery while a pseudoaneurysm is a calling for urgent surgical repair [7]. Echocardiography is the modality most studied with respect to distinguishing between ventricular aneurysms and pseudoaneurysms [7,8]. Transesophageal echocardiography (TEE) has a diagnostic accuracy of more than 75% compared to angiography, but data about its use are limited [5,7]. Echocardiography can usually distinguish a pseudoaneurysm from a true aneurysm by the appearance of the connection between the aneurysm and ventricular cavity. Only small ruptures of the ventricular wall are compatible with survival. As a result, pseudoaneurysms have a narrow neck, typically less than 40 percent of the maximal aneurysm diameter, that causes an abrupt interruption in the ventricular wall contour. In contrast, true aneurysms are nearly as wide at the neck as they are at the apex [6,8]. The nature of flow within a pseudoaneurysm has been used to distinguish it from true aneurysm based on results with echocardiographic Doppler techniques. The presence of turbulent flow by pulsed Doppler at the neck of a cavity or within the cavity itself suggests a pseudoaneurysm [9,10]. Other useful

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Sub Date: October 26th 2017, Acc Date: November 20th 2017, Pub Date: November 20th 2017


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findings could be obtained by echocardiography include; evaluation of the valvular status, estimation of the ejection fraction (EF), assessment of the continuity of the myocardium. Intraoperative TEE can be very useful for assessing the location, size and number of communications of aneurysm/pseudoaneurysm, the presence of mural thrombus, involvement of mitral apparatus, inter-papillary distance, global and regional function of ventricles, valvular regurgitation, left atrial size and diastolic dysfunction, etc [8-10].

Unfortunately, when clots are present, it is difficult to determine by echocardiography whether they are inside or outside the myocardium. Even though, presence of clots lead to underestimation of the orifice-cavity diameter which leads to incorrect diagnosis [1,10].

Left ventricular Angiography (LVA) has been historically recommended as the imaging modality of choice and still considered the most reliable method for diagnosis of a pseudoaneurysm [5,11]. Angiographic findings that help distinguish false aneurysms include a narrow orifice leading into a saccular aneurysm and lack of surrounding coronary arteries. In addition, coronary angiography shows the need for concomitant bypass grafting and may also prevent inappropriate surgical ligation of the left circumflex artery [11]. In the literature review, LVA resulted in a definitive diagnosis in over 85 percent of patients, with only 2 percent having a normal examination [11,12]. Currently, there are no specific guidelines on the performance of LVA at the time of coronary angiography and the decision is left to the opinion of the operator. Critics of LVA often argue that it adds to the cost, risk, higher volume of contrast and radiation exposure and it may predispose to embolisation if LV clots are there and it may be replaced by noninvasive imaging [11,13].

Cardiovascular computerized tomography (CT) and magnetic resonance imaging (MRI) are an alternative to angiography or echocardiography that may be useful in order to distinguish a pseudoaneurysm from a true aneurysm [5,10,11]. The high spatial resolution and tissue characterization of cardiac MRI make it ideal for evaluation of pseudoaneurysm of the ventricles and for distinguishing pseudoaneurysm from true aneurysms. In such cases, the use of late gadolinium enhancement to identify the location and transmural extent of prior infarcts is particularly valuable [6,13].

Nevertheless, not all patients can undergo cardiac MRI because of either patient contraindication (e.g., implanted cardiac devices, claustrophobia) or lack of availability. Cardiac CT offers high spatial resolution and provides an excellent visualization of the LV myocardium, coronary arteries, and bypass grafts. Although cardiac CT is more widely available than cardiac MRI, it involves radiation and the use of intravenous contrast exposure [12,13].

Approximately 45% of false aneurysms due to coronary artery disease will rupture. Even small undiagnosed aneurysms can rupture and cause sudden death. Since few patients referred to surgery for aneurysm repair more than 12 months after their initiating event, we recommend that once a diagnosis of a false aneurysm is made, operative management is mandatory unless serious circumstances exist [11,14].

The aim of surgical therapy in the case of pseudo-aneurysms is to repair the ventricular defect before its lethal rupture and to relief from ischemia, by performing coronary artery bypass grafting (CABG), diminishing ventricular volume and restoring it to its normal geometry and, when appropriate, bringing further decrease in volume overload by performing mitral valve repair if needed. Ventricular volume reduction diminishes wall stress, and thus reduces myocardial oxygen consumption. By excluding the mass of abnormal myocardium, it improves wall compliance, reduces filling pressure, and enhances coronary blood flow. Thus the systolic shortening of fibers is increased in extent and velocity bringing about enhanced contractile performance [1,15].

The results of operative repair of false ventricular aneurysms have improved greatly. Even considering the very early cases to undergo surgery in the mid1950’s, the overall operative mortality for patients in the literature review was only 14% [11,16,17].

We present here our experience in a case of repair of LVPA which was accidentally discovered in a young patient one year post myocardial infarction.

**Case Presentation**

The study protocol was approved by the Local Ethical and Research Committee of our institute and fully informed written patient’s consent was obtained and available.

Our case is 39 years old gentleman, works as a soldier; he is 170cm height, 69kg weight and body mass index (BMI) = 23, will known to have diabetes mellitus (DM II), Hypertension, and heavy smoker for the last 25 years, with a history of inferolateral MI one year ago when 1ry PCI to OM1 was done at that time. After that his follow up period was unremarkable and he went back to his normal work and usual life.

After one year he presented to the outpatient clinic (OPD) with shortness of breath (SOB), New York heart association classification (NYHA) class II-III and other vital signs were normal. Routine investigations were done including laboratory work up which was unremarkable except small troponin I and CkMB enzyme leak. Echocardiography surprisingly showed a huge mass at the lateral-posterior basal area of the heart about

Figure 1 (a, b, c): Spiral cardiac CT with 3-D reconstruction showing big LVPA with its communicating neck to the LV cavity very close to AV groove and base of the heart.

Figure 2 (a, b): Cardiac CT showing the LVPA communicating LV cavity with two orifices (blue arrow).

8x3 cm with huge organized blood clot inside it and communicating orifice to the LV cavity with blood flow to and fro inside it. Multiple regional wall motion abnormalities were seen, severe hypokinesia to akinesia of the posterolateral LV wall with an inferolateral and posterior area of thinned out myocardium. EF was 35-40%, with grade II-III diastolic dysfunction and mild mitral regurgitation, minimal pericardial effusion with massive fibrinous adhesions between the heart and the pericardium. Cardiac spiral CT was done and it confirmed the echo findings in addition to discovering of two communicating holes between the LVPA and LV cavity (figure 1, 2).

The final diagnosis was big post MI LV pseudoaneurysm. Consensus was done between the cardiologists, radiologists and cardiac surgeons and the decision was urgent surgical repair. The patient admitted to cardiac surgery department and after a high risk consent was taken, he was taken to the operating theater (OR).

In the OR, intraoperative TEE was done and it confirmed the diagnosis of big LVPA and its difficult location at the base of the heart and very close to the atrioventricular (AV) groove and to the mitral valve apparatus.

The approach to cardiopulmonary bypass was through right femoral groin (femro-femoral) canulation due to massive adhesions in the pericardium and to avoid possible rupture of the big friable LVPA during manipulation after chest opening and to decrease the chance of clots embolisation.

After going on cardiopulmonary bypass and administration of cold blood interrupted cardioplegia, the very thin transparent cystic wall adhering to the pericardial cavity was dissected carefully and opened and the huge blood clots were carefully removed and two holes communicating to LV cavity were seen (one big and one small) then the LV wall was cleaned and the mitral apparatus including lateral papillary muscle and its chordate tendiae could be seen clearly from the defect (figure 3, 4, 5). The wall of the cystic cavity did not show any myocardium. The two defects (the neck of the LVPA) was closed with one haemashield synthetic patch with interrupted 3/0 Prolen sutures with enforcement with pledged Teflon (figure 6). Then we applied a tissue glue over the patch and the LVPA false wall was closed over it for more haemostasis and support.

Figure 3: The very thin transparent LVPA wall with massive Hemorrhagic fibrinous pericardial adhesions.

Figure 4: Video-assisted endoscopic camera through LVPA cavity we can see both orifices (blue arrow) and visualize the chordate tendinae and part of the papillary muscle.
Figure 5: Huge blood clot taken out from the LVPA cavity.

Figure 6: Synthetic patch closure of the communicating neck between LVPA and LV cavity.

Discussion

The earliest report introduced in the literature about LVPA was in 1797 by Corvisart [1,18,19]. Potaine in 1892 first one reported a traumatic false aneurysm. Since then there have been additional reports largely describing the pathological and clinical features [10,11,18]. A literature review of 253 patients with LVPA in whom the cause was reported, most of the cases are related to acute MI (particularly of the inferior wall, which was twice as common as anterior infarction) and cardiac surgery (particularly mitral valve replacement and previous aneurysmectomy) [4,19,20]. In another literature review of 290 patients, the median patient age was found 60 years, and more than two thirds of patients were men and three quarters were white [14,20,21].

Unlike a true ventricular aneurysm, the LVPA is contained by the overlying adherent pericardium or scar tissue with no endocardium or myocardium, while the true LV aneurysm is an area of thinned myocardium that is dyskinetic and involves the full thickness of the wall [3,4,22]. The LV true aneurysm can be safely treated medically with an anticipated five-year survival of up to 90%, while LV pseudoaneurysm are prone to rupture (estimated risk 30%-45%) thus, a surgical approach is often undertaken with an operative mortality 23 % compared to 48 % with conservative management [7,8,23]. There were only four patients in the literature who had surgical treatment of post myocardial infarction false aneurysm more than a year after infarction, and a single patient was operated on after 48 months. This suggests that few patients survive for prolonged periods [9,24]. It is believed that the location is one of the most easily documented potential features proposed for distinguishing true aneurysms from pseudoaneurysms [10,25]. This finding is consistent with the location of pseudoaneurysms on the posterior, lateral, apical or inferior surface of the LV. Unlike LVPA, only 4% of LV true aneurysms are located at the posterolateral or diaphragmatic surface. The reason for the detection of fewer anterior LVPA may be due to the fact that an anterior LV wall rupture terminates in rapid hemopericardium, tamponade, shock, and death; whereas, posteriorly it tends to get contained by the pericardium since the patients are usually recumbent and blood tends to pool in the dependent posterior pericardium forming a pseudoaneurysm rather than cardiac tamponade [3,25]. Extensive posterior infarction usually involves papillary muscle which usually results in severe mitral regurgitation and death, and thus these patients never go on to develop aneurysms. Another explanation suggested for predominance of anterior wall true aneurysm is that true aneurysm in the posterior wall is difficult to detect by usual imaging methods, resulting in a discovery bias in favor of anteroapical aneurysms [10,24,25].

The cardiopulmonary bypass and aortic cross clamp time were 93 minutes and 64 minutes respectively. Post bypass intraoperative TEE showed good LV repair with no residual communicating defects were seen, mild mitral regurgitation and same regional wall motion abnormalities and same LV function.

The patient tolerated the procedure well and shifted to the intensive care unit (ICU) in a stable condition. The post operative course was unremarkable and discharged home at 7th post-operative day. Post operative follow up was done at 1 week, one month, 3 months then 6 month and one year respectively. The serial echocardiography was acceptable and EF improved and reached 50% at one year and the NYHA class improved to class I-II.
In a literature review of 290 patients with pseudoaneurysms, the most reported symptoms or conditions were congestive heart failure (36%), chest pain (30%), and dyspnea (25%) [14,26]. In contrast to LV true aneurysm, angina is the most frequent symptom in most series of operated patient with LV aneurysm. Given that three-vessel coronary artery (CAD) is present in 60% or more of these patients, the frequency of angina is not surprising [16,17]. Many patients have an audible nonspecific systolic murmur. McNiel, et al. reported a patient with a false left ventricular aneurysm and a murmur which mimicked mitral regurgitation. Cone and Hawley described a to-and-fro murmur at the apex which was similar to a murmur noted by Scanlon, et al. A murmur of this type may be an aid to early diagnosis [11,24]. The electrocardiogram is of little help in diagnosing false ventricular aneurysm, a persistent ST segment elevation may suggest a ventricular aneurysm, but it is present in only approximately one-half of the patients [11,23]. The plain-film finding of a paracardiac mass in a diaphragmatic or posterolateral location in conjunction with a history of previous myocardial infarction is highly suggestive of pseudoaneurysm and is sufficient indication for angiographic studies [17,22]. Thrombus is frequently found in the false aneurysm, and remains nonadherent and less well organized than in true aneurysm because of the avascular wall of false aneurysm. The true incidence of embolism is unknown. However, it may be less than with true aneurysm since the aperture communicating with the left ventricle is usually smaller (typically less than 40% of maximal aneurysm diameter) [10,11,24].

Historically, the operative approach to false aneurysm of the left ventricle is very interesting. Courageous surgeons first attempted repair before cardiopulmonary bypass was available. Through a purse string suture in the false aneurysm, the communicating aperture was closed blindly by sutures. Currently, standard cardiopulmonary bypass technique is utilized. As with true aneurysms, it is important to avoid manipulation of the pseudoaneurysm until ventricular ejection ceases because thrombus may be dislodged. Total excision of the aneurysmal sac is not necessary since the crucial step is closure of the communicating aperture [2,3,11,25].

Regardless of treatment, patients with LV pseudoaneurysms had a high mortality rate, especially those who did not undergo surgery [19]. However, the prognosis for patients with pseudoaneurysms, including the risk of rupture or other complications, such as congestive heart failure, embolic events, or arrhythmias, is not known [10,23,25]. In sharp contrast to true aneurysm, false aneurysm may rupture after the fibrous stage has been reached even when the aneurysm is small [11,19,26]. Patients with false aneurysms of the left ventricle do well after surgical repair, except when concomitant mitral valve replacement is necessary because it is proved that combination of mitral valve replacement and LV aneurysm surgery increase mortality [16,27].

We think that our case is unique because this the 1st reported case of LVPA in “Saudi Arabia and Gulf countries” and accidentally discovered after more than 1 year post MI and PCI/OM in a relatively young and active man (soldier). Also its location very close to the basal Atrio-Ventricular groove and mitral apparatus plus massive pericardial adhesions and decision of femor-femoral bypass represents a surgical challenge in the OR.

**Conclusion**

We conclude that Cardiac pseudoaneurysms are rare but clinically significant potentially lethal lesions. Although often challenging to diagnose, advances in noninvasive imaging improve the ability to distinguish LV true aneurysm from pseudoaneurysm specially echocardiography, cardiac CT and MRI. LVPA requires urgent surgical intereference to avoid its devastating complications including heart failure symptoms, systemic embolisation, mitral regurgitation and high possibility of fatal rupture and tamponade.

**References**


