Journal of Cardiovascular Diseases & Diagnosis

Rana et al., J Cardiovasc Dis Diagn 2016, 4:5
DOI: 10.4172/2329-9517.1000256

Case Report Open access

A Rare Case of Pseudoaneurysm of the Aortic Root Compressing the Left Anterior Descending Artery and Causing Myocardial Infarction: A Case Report and Review

Chirag Rana[1*], Kunal Patel[2], Hiten Patel[2], Fayez Shamoon[2] and Mahesh Bikkina[2]

[1]*Department of Internal Medicine, St. Joseph's Regional Medical Center, New York Medical College, Paterson, USA*

[2]*Department of Cardiology, St. Joseph's Regional Medical Center, New York Medical College, Paterson, USA*

[*]**Corresponding author:** Chirag Rana, Department of Internal Medicine, St. Joseph's Regional Medical Center, New York Medical College, Paterson, NJ 07503, USA, Tel: 973-807-8945; Fax: 973-754-3376; E-mail: chirana87@gmail.com

Received date: Aug 09, 2016; **Accepted date:** Aug 24, 2016; **Published date:** Aug 30, 2016

Abstract

Aortic pseudoaneurysm is extremely rare and a catastrophic sequelae of aortic surgery and blunt chest trauma involving the aorta. They can be asymptomatic or present with non-specific cardiac symptoms. In some instances, they present with life threatening complications such as rupture or even sudden death. Symptoms are usually due to the anatomical location, size and pressure on the neighboring anatomical structures. Here we report on a 37-year-old male with history of multiple cardiac surgeries including aortic root and aortic valve surgeries who developed a pseudoaneurysm two weeks after an aortic root patching. The pseudoaneurysm was found to be externally compressing the left anterior descending artery, leading to a myocardial infarction.

Keywords: Pseudoaneurysm; Aortic root; Ascending aorta; Left anterior descending

Introduction

Aortic pseudoaneurysm is an outpouching of the aorta caused by a defect in the tunica intima and media. Since they are bound by only the tunica adventitia, risk of rupture is very high. It is different from a true aneurysm which is an outpouching involving all three layers of the vessel wall (intima, media and adventitia). It is extremely rare and catastrophic sequelae of cardiac surgery, trauma, infection or genetic diseases. The incidence is thought to be about 1% following aortic surgery and about 3-4% following blunt chest trauma involving the aorta [1].

It is often difficult to differentiate pseudoaneurym from other etiologies that cause chest pain based on symptoms alone. Most common symptoms include dyspnea and chest pain; therefore it closely mimics acute coronary syndrome, aortic dissection and pulmonary embolism [1]. About 20% of cases are asymptomatic and are discovered incidentally [1]. Some cases present with rupture leading to serious outcome including death. Ascending aortic pseudoaneurysm, in particular, is associated with high morbidity and mortality rate [2]. Here we present a very rare case of pseudoaneurysm of the aortic root compressing the left anterior descending (LAD) artery and causing a myocardial infarction.

Case Report

A 37-year-old Hispanic male with a significant medical history of multiple cardiac morbidities including aortic and mitral valve disease due to a childhood history of rheumatic fever affecting the mitral valve, atrial fibrillation, atrial flutter, endocarditis, first degree heart block, hyperlipidemia, and hypertension presented to emergency department (ED) with generalized weakness and a near-syncopal episode. He reports intermittent left sided chest pain at rest that kept him up at night and prompted him to present to the ED. He denied palpitation, shortness of breath, nausea, vomiting, fever, and diaphoresis. He was recently admitted for methicillin-sensitive *Staphylococcus aureus* (MSSA) prosthetic valve endocarditis and had undergone a third re-operation for aortic and mitral valve replacement with aortic root/cusp abscess patching. The repair of the aortic root abscess was performed in close proximity to the left main coronary artery. Patient was discharged home two weeks ago with oxacillin and rifampin. He was compliant with the antibiotics and was doing well at home until this new presentation.

In the ED, patient denied any recurrence of chest pain and was not in any obvious distress. On physical examination, he was found to be tachycardic (pulse of 105 beats per minute); otherwise, the rest of the exam was unremarkable. An electrocardiogram (ECG) was performed which did not reveal any ST-T-wave changes indicating acute myocardial infarction; however, it did show sinus tachycardia with first degree A-V block, presence of premature atrial complexes, and non-specific T-wave abnormalities, which was worse in the inferior leads. His cardiac enzymes were elevated, with a troponin of 9.94 ng/mL (normal<0.045), suggesting non-ST elevation myocardial infarction. Given the location of his previous aortic root abscess, clinical presentation and elevated troponin, there was concern for left main coronary artery compromise. Therefore, an emergent cardiac catheterization was performed. The cardiac catheterization showed a normal left main coronary artery but severe stenosis (95%, long, smooth tubular occlusion) of the LAD, involving both the ostium and the proximal LAD (Figure 1). There was also a 60% smooth tubular occlusion in the mid LAD. The mid LAD lesion was suspected to be due to mild atherosclerotic disease and thus the subsequent inflammation likely caused coronary vasospasm. These occlusions were suspected to be due to coronary spasm. Multiple doses of intracoronary nitroglycerine and nicardipine were given without any response, raising suspicion for possible extrinsic compression. Patient

started experiencing severe chest pain despite all these medications and his systolic blood pressure dropped to 70s; therefore, a dose of norepinephrine was given. A drug eluting stent was then placed in the osteal and proximal LAD. An intra-aortic balloon pump was also placed for his hemodynamic instability. He tolerated these procedures without any complication and was transferred to the cardiac care unit (CCU) on intravenous heparin and low dose norepinephrine.

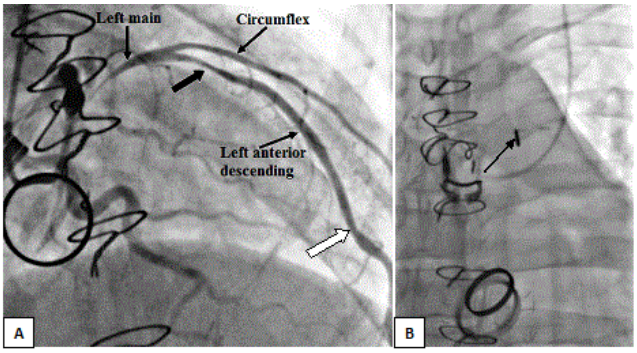

Figure 1: Coronary angiogram showing severe stenosis in LAD. **(A):** Coronary angiography showing a severe stenosis (95% long, smooth tubular occlusion) of the left anterior descending (LAD) artery involving both the ostium and proximal LAD (thick black arrow). Another stenosis (60% smooth tubular occlusion) can be noted in the mid LAD (thick white arrow). **(B):** Post-stenting angiography showing the restoration of blood flow in the LAD. The drug eluting stent can be noted in the osteal and proximal LAD (arrow).

While in the CCU, a transesophageal echocardiogram showed that the previously patched aortic root had increased in size from 3 cm (two weeks ago) to about 5 cm and there was now a new evidence of communication/flow from aorta at the aortic root level into this collection, suggesting a development of a pseudoaneurysm (Figure 2). This collection appeared to be externally compressing the LAD region. Previous records do not reveal a cardiac catheterization prior to the multiple cardiac surgeries.

A decision was made for possible bridging versus reoperation and follow up at another facility was advised. Further follow-up revealed that the patient received cardiac surgery which entailed aortic and mitral valve replacement with aortic root graft and reconstruction. Patient recovered well and has not had any further complications or complaints.

Discussion

The most common predisposing factors for developmental of a thoracic aortic pseudoaneurysm are blunt chest trauma involving the aorta and aortic surgery. In particular, surgeries for repair of coarctation, aneurysm, dissection, or aortic cannulation during cardiopulmonary bypass and trauma such as deceleration injury or penetrating injuries are the more common causes of pseudoaneurysm of thoracic aorta [1]. Pseudoaneurysm related to surgery usually develops at the following sites: aortotomy or anastomotic suture lines, aortic and cardioplegia canulation sites, cross-clamp sites, and needle puncture holes [3,4]. Those related to deceleration injury usually develops at aortic isthmus [1,5]. Other known etiologies include infective endocarditis, vasculitis (Behcet disease), Marfan syndrome and Kommerell diverticulum [1]. Rarely, it can develop from non-cardiovascular thoracic surgery. In the case presented above, patient's pseudoaneurysm was most likely related to his previous cardiac surgeries including the recent patching of aortic root abscess.

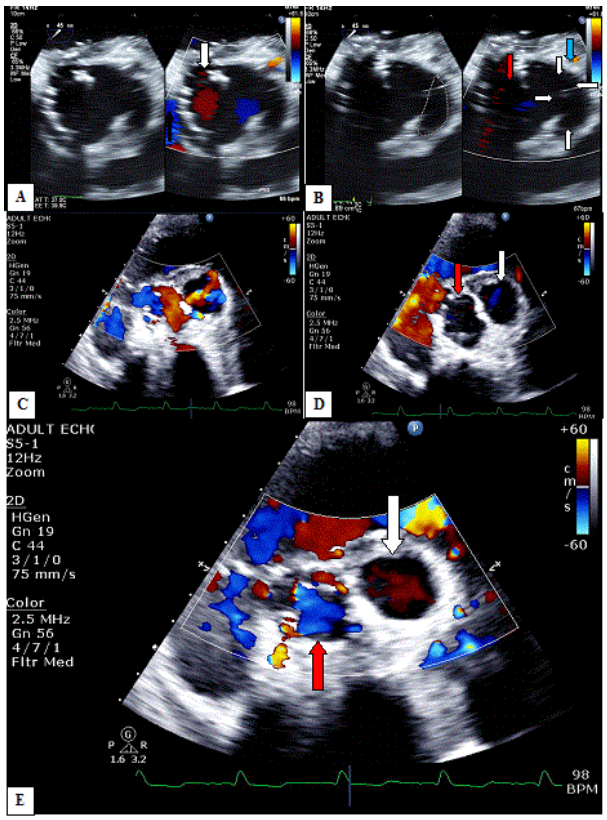

Figure 2: Visualization of pseudoaneurysm by transesophageal echocardiography (TEE). **(A):** TEE showing the aorta (black arrow) in systole and on the right side to it, the pseudoaneurysm with multicolor flow (thick white arrow) can be noted. (B)**:** In this image, the aorta (red arrow) is shown during diastole and the borders of pseudoaneurysm are denoted by white arrows. The left main coronary artery filling during diastole may be visualized by the blue arrow. Note the proximity of the pseudoaneurysm to the coronary artery. The pseudoaneurysm was identified to be externally compressing the left anterior descending artery. **(C):** The psuedoaneurysm can be seen on the right side of the aorta denoted by the dotted circle. The size of the pseudoaneurysm was approximated to be about 5 cm by 3.5 cm. **(D) and (E):** The pseudoaneurysm (white arrow) can be seen on the right side of the aorta (red arrow) during diastole and systole, respectively.

Clinical presentation of pseudoaneurysm is highly variable and therefore a high index of suspicion is necessary in order to investigate and diagnose it. Some patients are asymptomatic and are detected incidentally while others present with life-threatening complications such as rupture or even sudden death. The risk of rupture for those resulting from trauma, Marfan's, and infection is higher than those that result from surgery or vasculitis [1]. In general, symptoms are largely due to anatomical location, size, and pressure on the neighboring anatomical structures. Some of the reported complications include

SVC obstruction, aorto-pulmonary fistula, RV inflow obstruction, fistula into RA, RV and LV and angina [4].

Physicians should consider pseudoanaeurysm in patients with unexplained chest symptoms, especially if the patient has risk factors such as chest trauma involving aorta or prior aortic surgeries. CT and MRA have a high sensitivity and specificity for diagnosing it [1]. Trans-esophageal echocardiography and chest x-ray are helpful in making the diagnosis. Once a pseudoanaurysm is identified, surgical repair is recommended even if patient is asymptomatic because of the risk for rupture. Pseudoaneurysm formation is one of the leading causes of late reoperation after surgical repair of acute type A aortic dissection [6]. Open surgical repair or endovascular repair are used for the correction of pseudoaneurysm. Compared to open surgical repair, endovascular repair is associated with decreased morbidity (paraplegia, stroke, renal failure) and mortality [1,7]. Endovascular techniques include using stent grafts, coils for the closure of pseudoaneurysm, thrombin injections, septal occlude devices and vascular plugs [8].

In summary, pseudoaneurym is a very rare yet fatal complication of previous aortic surgeries or aortic trauma. Although various symptoms have been reported, acute coronary syndrome from an ascending pseudoaneurysm is rare. There have been rare cases in which patients developed MI due to compression of RCA caused by the pseudoaneurysm. Kar et al. presented two cases of right ventricular dysfunction, one presenting as volume overload (pseudoaneurysm-RA fistula) and another presenting as pressure overload with intermittent ischemia caused by RCA compression by pseudoaneurysm [4]. To our knowledge, there are even fewer cases of LAD compression by pseudoaneurysm in the literature [9]. Therefore, we believe that the case we presented is an extremely rare finding and could have led to catastrophic events if the diagnosis and treatment were delayed.

Conflict of Interest

Authors declare that there is no conflict of interest regarding the publication of this manuscript.

References

1. Alla VM, Suryanarayana PG, Thambidorai SK (2010) Thoracic aortic pseudoaneurysm following noncardiovascular surgery: a rare complication that can mimic common chest emergencies. South Med J 103: 1186-1188.
2. Garisto JD, Medina A, Williams DB, Carrillo RG (2010) Surgical Management of a Giant Ascending Aortic Pseudoaneurysm. Tex Heart Inst J 37: 710-713.
3. Sullivan KL, Steiner RM, Smullens SN, Griska L, Meister SG (1988) Pseudoaneurysm of the ascending aorta following cardiac surgery. Chest 93: 138-143.
4. Kar P, Gopinath R, Padmaja D, Kumar RV (2015) Complex pseudoaneurysm of ascending aorta: Unusual cause of right heart dysfunction-implications to the anesthesiologist. J Anaesthesiol Clin Pharmacol 31: 246-249.
5. Kim S, Park JS, Yoo SM, Kim KH, Yang WI, et al. (2014) Traumatic aortic regurgitation combined with descending aortic pseudoaneurysm secondary to blunt chest trauma. Cardiovasc J Afr 25: e5-8.
6. Klvacek A, Konecny J, Santavy P, Lonsky V, Simek M, et al. (2015) An unusual symptoms caused by huge pseudoaneurysm formation of ascending aorta. J Cardiothorac Surg 10: A337.
7. Kotelis D, Bischoff MS, Rengier F, Ruhparwar A, Gorenflo M, et al. (2016) Endovascular repair of pseudoaneurysms after open surgery for aortic coarctation. Interact Cardiovasc Thorac Surg 22: 26-31.
8. Quevedo HC, Santiago-Trinidad R, Castellanos J, Atianzar K, Anwar A, et al. (2014) Systematic review of interventions to repair ascending aortic pseudoaneurysms. Ochsner J 14: 576-585.
9. Bouabdallaoui N, Achouh P, Lacaze-Gadonneix J, Ennezat PV (2014) Unusual cause of acute coronary syndrome: dynamic coronary compression by an aortic pseudoaneurysm. Eur Heart J Cardiovasc Imaging 15: 468-469.