CASE REPORT

A Left Ventricular Pseudoaneurysm Caused by a Traumatic Fall

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A 51-year-old man with chest pain, increasing dyspnea, and a cardiac murmur was thought to initially have a myocardial infarct-related ventricular septal defect (VSD) and was referred for consideration of percutaneous closure. However, imaging studies revealed an intact ventricular septum and a large left-to-right shunt through an inferior wall pseudoaneurysm (LVPA) that had burrowed into the right ventricle. Although the initial incorrect diagnosis delayed appropriate treatment, the defect was successfully repaired.

Introduction

Left ventricular pseudoaneurysms (also called false aneurysms) can result from myocardial infarction, prior cardiac surgery, endocarditis, and chest wall trauma.[1] Free wall rupture is contained by adherent pericardium that over time expands by incessant systolic inflations. Unlike true aneurysms, the wall is devoid of myocardium and the communication with the LV cavity is narrower than the aneurysm diameter.

The presenting symptoms are usually those of congestive heart failure or angina. Sudden cardiac death was the presenting manifestation in about 3%, while 12% of patients were asymptomatic.[2-4]

Untreated pseudoaneurysms have a 30% to 45% risk of rupture and, with medical therapy alone, a mortality of almost 50 percent.[2,5] In contrast to patients with true ventricular aneurysms, those with false aneurysms most commonly die of hemorrhage.[6] Thus, surgery is the preferred therapeutic option. With current techniques, perioperative mortality is less than 10%. The risk is greater among patients with severe mitral regurgitation requiring concomitant mitral valve replacement.[7, 8]

Case Report

A 51-year-old Hispanic laborer was admitted to a local hospital with increasing difficulty breathing progressing over 2 weeks and new onset chest pressure. His electrocardiogram (ECG) demonstrated QS complexes in lead III and QR in aVF with concordant T waves, left ventricular hypertrophy by voltage, and flattened T waves. Maximum serum Troponin level was 2.8 ng/ml. Left heart catheterization with coronary and left ventricular angiography revealed non-critical coronary artery disease in the right coronary artery and left anterior descending branch of the left coronary with calcified plaques. Cardiac output determined by thermodilution was 6.5 L/min (Cardiac Index 3.6 L/min/M^2), and the left ventricular diastolic pressure was 20 mmHg. Left ventriculography in the left anterior oblique projection demonstrated uniform wall motion with a transient blush of contrast on the right ventricular side of the interventricular septum. An infarct-related VSD was suspected. The patient remained in the hospital for intensive management of his heart failure for 12 additional days but failed to improve, prompting his referral to Harbor-UCLA for closure of the defect.

Examination on admission was notable for jugular venous elevation of 14 cm with dominant a-waves, and a systolic thrill over the left mid precordium. A grade 4/6 harsh, low-pitched holosystolic murmur was loudest over the mid precordium and easily heard over the entire precordium where it was higher in pitch. The second heart sound was persistently split with a loud pulmonic closure sound. No gallops were detected.

Transthoracic echocardiography was initially interpreted to show a “large muscular VSD measuring 3.9 cm” and repeat right and left heart catheterization confirmed a large left-to-right shunt (pulmonary blood flow 11 L/min, systemic blood flow 3.5 L/min, (qP/qS 3.14/1). Left ventriculography depicted a vigorously contracting ventricle with contrast medium entering a thin-walled oval chamber on the diaphragmatic surface of the left ventricle followed by opacification of the right ventricle (Figure 1). The magnitude of left-to-right opacification was significantly greater than on the study performed 2 weeks prior, while the systemic cardiac output had declined by 46%.

CT imaging with contrast confirmed the thin-walled, inferior wall pseudoaneurysm with a narrow neck leading to a fistula entering the inferior wall of the right ventricle adjacent to the septum (Figures 2, 3).

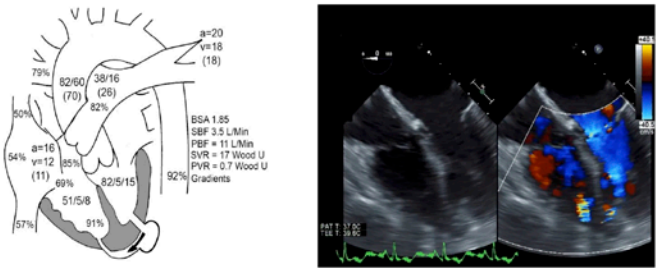

Figure 1: Figure (left) demonstrating schematic of inferior wall pseudoaneurysm with a narrow neck communicating with the right ventricle adjacent. Saturations and hemodynamics are also shown. Transesophageal echocardiography (right) showing four-chamber view with color Doppler

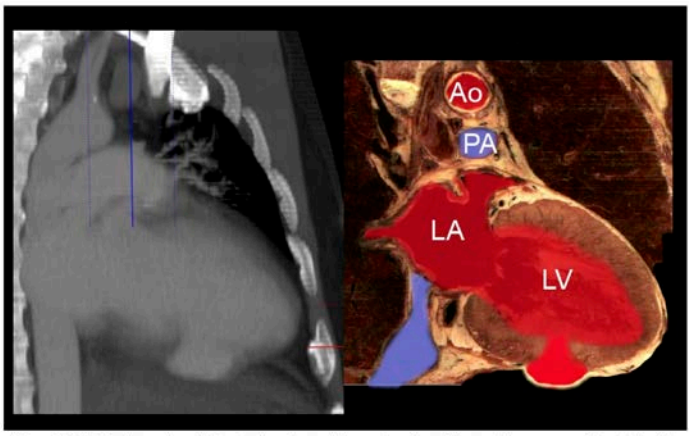

Figure 2: CT Angiography right anterior view oblique showing LV pseudoaneurysm. Created with images provided by the Visible Human Project® Gallery

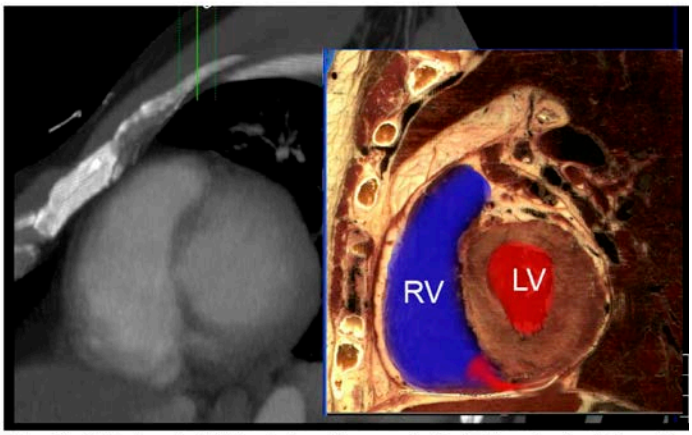

Figure 3: CT Angiography left lateral view with communicating tubular extension between the left ventricle and right ventricle. Created with images provided by the Visible Human Project® Gallery

Given findings of pseudoaneurysm without significant coronary artery disease, the patient was questioned further and revealed that he had fallen off a 14-foot ladder seven years prior, landing flat on his back and was admitted to a hospital where he underwent back surgery with implantation of pedicle screws at L4, L5, and S1 for a presumed spinal fusion necessitated by the trauma. We concluded that the fall had also resulted in an inferior wall rupture that in turn led to a pseudoaneurysm that was quiescent for 7 years but had recently burrowed into the adjacent right ventricle, causing an enlarging shunt that greatly increased the work of the left ventricle.

Surgical repair was initially delayed for concern that the shunt was caused by a recent myocardial infarct-related VSD. Early repair of infarct-related VSDs are often complicated by failure of sutures to hold in the friable tissue surrounding the defect. The uniformly vigorous left ventricular wall motion required to eject over 11 L/min, 24 hours a day, belied the presence of ischemic impairment or evidence of extensive infarction.

When it was agreed that the shunt was not caused by a septal defect, open heart surgery with cardiopulmonary bypass was undertaken via median sternotomy approach. However, when the pericardium was incised, the right ventricle rapidly expanded and the arterial pressure dropped precipitously, necessitating urgent cannulation for initiation of cardiopulmonary bypass. The relief of “girdle constraint” by opening the pericardium massively increased the magnitude of the shunt, but rapid cannulation and institution of cardiopulmonary bypass decompressed the heart, restored systemic arterial pressure, and allowed the surgeon to explore the arrested heart.

A 3-cm thin-walled sac on the diaphragmatic surface of the left ventricle communicated with the left ventricular cavity through a 2.5 cm defect. The false aneurysm’s bulbous body and narrow tubular extension entering the right ventricle resembled a “syringe bulb” which entered the floor of the right ventricle via a 1-cm ostium adjacent to the interventricular septum. Both orifices were buttressed by strong fibrous tissue supporting the sutured Dacron® patches. Additionally, the left anterior descending artery was bypassed. Once stable, the patient was discharged with cardiology follow up.

Discussion

Left ventricular aneurysms (LVAs) and pseudoaneurysms are two complications of myocardial infarction that can lead to death or serious morbidity. The latter forms when cardiac rupture is contained by adherent pericardium or scar tissue. This differs from left ventricular true aneurysms, which contain endocardium or myocardium, and seldom rupture.[2]

Transthoracic echocardiography, CT or MRI imaging are all appropriate diagnostic modalities in patients suspected of having an LVA or pseudoaneurysm. Pseudoaneurysms are typically located in a more posterior or inferior position and

may be observed to expand during ventricular contraction, exhibiting dyskinesis.[9] Untreated, left ventricular pseudoaneurysms have an increased risk of rupture and death by cardiac tamponade. Surgical intervention remains the definitive treatment modality.[10]

This case demonstrates that repeating a comprehensive past history interrogation after employment and interpretation of pertinent imaging modalities resulted in a crucial difference in outcome. The absence of significant coronary artery obstruction and the presence of uniformly hyperdynamic left ventricular wall motion prompted consideration of an alternative diagnosis. Such steps were instrumental in appropriately managing this patient's trauma-related pseudoaneurysm.

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