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Concomitant Left Ventricular Aneurysm and Ventricular Septal Defect Following Acute Inferior Myocardial Infarction from In-Stent Thrombosis in a Post CABG Patient

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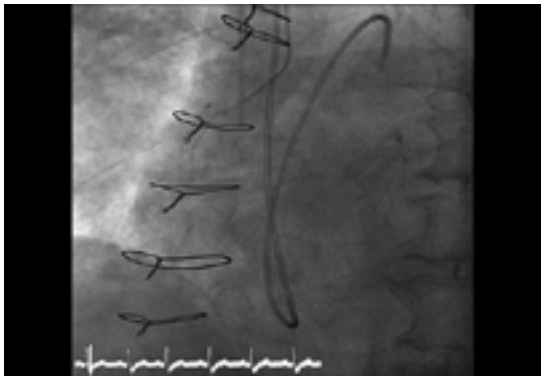
Abstract

Mechanical complications of acute myocardial infarction are rare. Additionally, concomitant complications in the same patient are even even less common, particularly in the revascularized patient. Here, we present a case of concomitant left ventricular inferior/inferoseptal aneurysm and ventricular septal defect as parallel complications of an acute inferior ST elevation myocardial infarction from in-stent thrombosis of a drug-eluting stent to the SVG-RCA graft in a patient after coronary artery bypass graft surgery. Our case is also unique in that we employed multiple cardiac imaging modalities, particularly cardiac magnetic resonance imaging, to delineate the anatomic complexity of the two concomitant complications, which was essential in defining the appropriate course of surgical management.

Keywords: Myocardial Infarction; Ventricular Septal Rupture; Left Ventricular Aneurysm; Echocardiography; Cardiac Magnetic Resonance Imaging

Abbreviations: CAD: Coronary Artery Disease; PCI: Percutaneous Coronary Intervention; DES: Drug-Eluting Stent; PAP: Pulmonary Artery Pressure; 2D TTE: Two-dimensional Transthoracic Echocardiogram, CABG: coronary artery bypass graft, STEMI: ST segment elevation myocardial infarction

Case Report



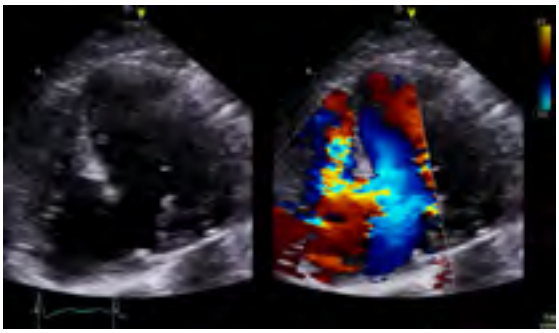
A 73 year-old male with a history of hypertension, coronary artery disease (CAD), triple vessel CABG (LIMA-LAD, SVG-ramus, SVG-RCA in 2008) (Video Clip 1), and an unrepaired abdominal aortic aneurysm was transferred for management of acute decompensated heart failure. He had multiple admissions to the outside hospital. His first hospitalization was August 2015. At that time, he presented with chest pain and was found to have an acute inferior STEMI for which he underwent percutaneous coronary intervention (PCI) with placement of a single drug-eluting stent (DES) to his SVG-RCA graft. He was discharged on aspirin and clopidogrel. However, he returned 6 days later with

recurrent chest pain. He was found to have a new inferior STEMI. Coronary angiogram revealed complete occlusion of the recently placed DES due to in-stent thrombosis. He underwent aspiration thrombectomy and PCI with placement of 3 DESs in the SVG-RCA graft.

Case Presentation

It was believed our patient failed on clopidogrel. Consequently, he was discharged on ticagrelor only to return 12 days after his initial hospitalization. He reported chest pain and shortness of breath. His ECG revealed fragmented QRS complexes in leads III and aVF consistent with his prior inferior myocardial infarction. He was diagnosed with acute decompensated heart failure and placed on dobutamine and furosemide drips. Despite diuresis, his symptoms persisted. The echocardiogram revealed LVEF 60-65%, hypokinesis of the septal and inferior walls, a possible VSD, RV systolic dysfunction, and severe pulmonary hypertension. Given these findings and his persistent dyspnea, he was transferred to our coronary care unit for further evaluation and management.

At the time of transfer he was on dobutamine 3mcg/kg/min. His vitals were stable. Physical exam was notable for bibasilar crackles, jugular venous distention, harsh V/VI holosystolic murmur at the LLSB with widespread radiation, a palpable thrill, and 2+ lower extremity edema to the knees bilaterally. Labs were notable for brain natriuretic peptide of 903 pg/ml and troponin I levels of 0.11 ng/ml, 0.06 ng/ml, and 0.05 ng/ml. His ECG showed normal sinus rhythm and fragmented QRS complexes in III, aVF. Chest X-ray displayed bilateral and diffuse opacities consistent with pulmonary edema. Right heart catheterization data was as follows: right atrial pressure 8 mmHg, right ventricular pressure 67/3 mmHg, pulmonary artery pressure (PAP) 62/21 mmHg, mean PAP 38mmHg, and pulmonary capillary wedge pressure 21mmHg (with V waves to 40mmHg). A shunt study revealed a 21% step up in going from the RA to the RV suggestive of a VSD with left to right shunting. The Qp: Qs was 2.2. Graft angiography illustrated patent LIMA-LAD and SVG-Ramus grafts. The SVG-RCA graft was visualized with numerous stents and displayed poor overall flow (TIMI 2 flow); yet there was no evidence of complete obstruction (Video Clip 1).



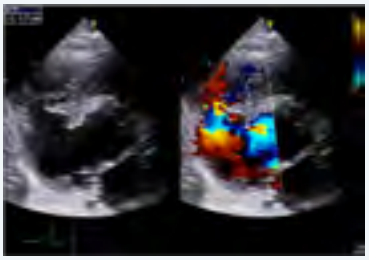


Figure 1A: Two-dimensional echocardiogram revealing concomitant LV aneurysm and VSD (measuring 1.7cm) with color flow illustrating left to right shunt flow across the VSD.

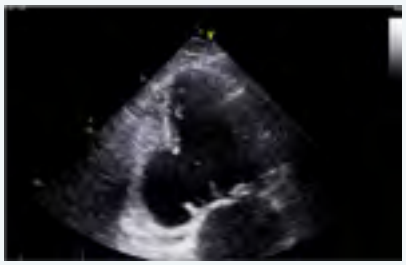


Figure 1B: Two dimensional echocardiogram apical 2-chamber views revealing large inferior LV aneurysm.

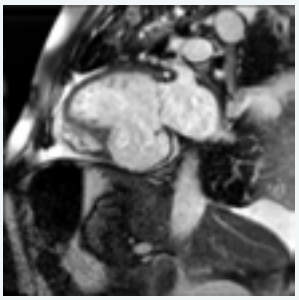


Figure 2A: Cardiac MRI two chamber still cine revealing large inferior basal to mid left ventricular aneurysm.

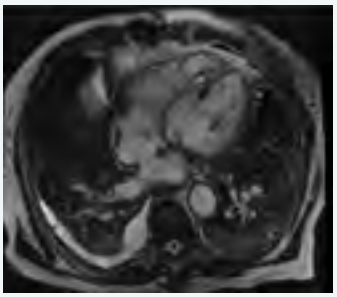


Figure 2B: Cardiac MRI four chamber still cine revealing large basal to mid inferoseptal aneurysm.

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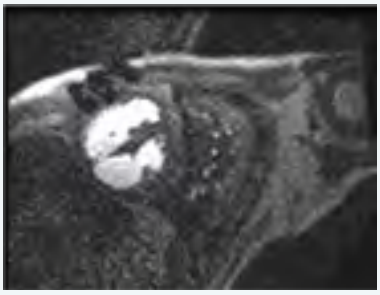


Figure 2C: Cardiac MRI still first pass phase contrast revealing inferior/inferoseptal LV aneurysm and concomitant VSD within LV aneurysm.

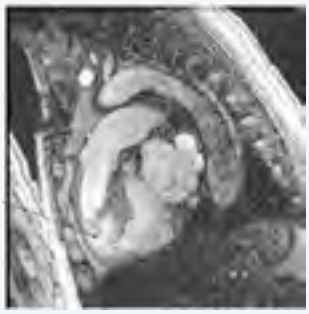
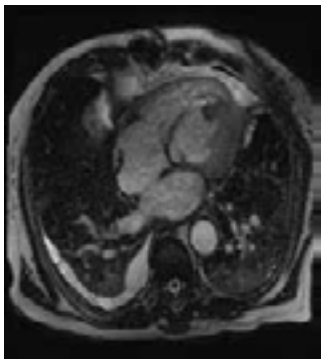
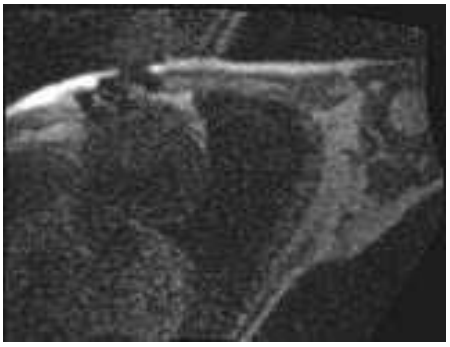
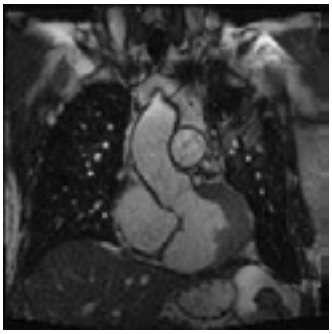


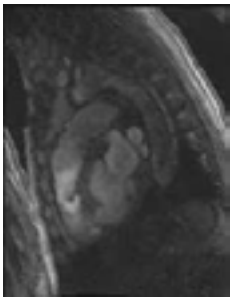
Figure 2D: Cardiac MRI RV outflow tract still phase contrast illustrating LV aneurysm and contained VSD with shunt flow across the VSD (arrow).

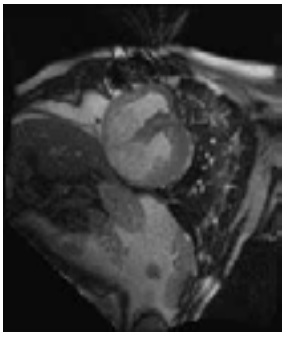
Two-dimensional transthoracic echocardiogram (2D TTE) revealed mild LV dilation, an LVEF of 64%, inferior wall akinesis, and a basal to mid to mid inferior and inferoseptal aneurysm with bidirectional shunt flow (Figure 1) (Video Clip 2). To better characterize the lesion, cardiac MRI was performed. Cardiac MR revealed a large basal to mid inferoseptal and inferior LV wall aneurysm measuring 6.0 x 4.5 cm with associated dyskinesis of this region (Figure 2). Within the LVA, there was also a VSD measuring 0.9cm; the shunt fraction was 17% and Qp: Qs was 1.2 (Figure 2). The VSD extended from the mid to apical aspect of the LV aneurysm (Figure 2, Video Clips 3-8).











At surgery, there was a true aneurysm of the inferior LV; this defect was large and extended from the base to the apex measuring approximately 4.5cm in depth and 1.7cm in width at the neck. The aneurysm extended into the RV toward the apex and measured approximately 2.5cm. Both defects were patch repaired using a bovine pericardial patch completely excluding the LV aneurysm and closing the VSD. The post repair TEE revealed complete exclusion of the LV aneurysm from the LV

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cavity without evidence of flow into the aneurysm. Likewise, the VSD was closed and there was no evidence of left to right shunt with Qp: Qs of 1.0. Post-operatively, the patient did fairly well and was ultimately discharged to home 2 weeks after arriving at our institution.

In the era of coronary revascularization, the prevalence and incidence of post MI complications is low [1,2]. Additionally, the co-existence of multiple complications is even less common, particularly in the re-vascularized patient [3,4]. There have been prior reports of concomitant LVA and VSD. For example, Heath and colleagues described a case of concomitant inferior LV aneurysm and apical VSD discerned at surgery in a patient following an inferior ST elevation MI [5]. Rogers et al. [6] identified concomitant posterior VSD with a discrete aneurysm of the posterior interventricular septum in 6 consecutive patients with acute inferior myocardial infarctions. Also, numerous studies have described anterior, anteroapical, or apical aneurysms and associated VSDs identified by 2D TTE in patients suffering an acute left anterior descending ST elevation MI [7-9].

Conclusion

There are several novel aspects of our case. First, nearly all of the case reports in the literature detail anterior, anteroapical, or apical LVAs while we describe a case of an inferior/inferoseptal LVA. Second, we describe concomitant LVA and VSD in a revascularized patient, which has not been illustrated previously. Third, the mechanism of post MI complications in our case is unique in that in-stent thrombosis of the DES to his SVG-RCA graft proved to be the ultimate cause of the concomitant LVA and VSD. Fourth, in terms of imaging the complications, 2D TTE by and large was the imaging modality of choice. However in our case 2D TTE, while it did reveal the inferior/inferoseptal LVA, did not illustrate the complex interplay between the LVA and VSD. Cardiac MRI was essential in order to detail the complex anatomy of the LVA and associated VSD and consequently aid in surgical decision making. In the era of coronary revascularization, we believe our case is the first of its kind.

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