Massive inferior wall aneurysm presenting with ventricular tachycardia and refractory cardiomyopathy requiring multiple interventions - a case report

MASSIVE INFERIOR WALL ANEURYSM WITH VT

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Abstract

BACKGROUND
Inferior wall left ventricular aneurysms are rare, they develop after transmural myocardial infarctions and may be associated with poorer prognosis. We present a unique case of a large aneurysm of the inferior wall complicated by ventricular tachycardia and requiring surgical resection and mitral valve replacement.

CASE SUMMARY
A 59-year-old male was admitted for ventricular tachycardia one month after he had a delayed presentation for an inferior ST-segment elevation myocardial infarction (STEMI) and was discovered to have a large true inferior wall aneurysm on echocardiography and confirmed on coronary CT angiography. Due to the sustained ventricular tachycardia, concern for aneurysm expansion, and persistent heart failure symptoms, the patient was referred for surgical resection of the aneurysm with patch repair, mitral valve replacement, and AICD insertion with significant improvement in functional and clinical status.

CONCLUSION
Inferior wall aneurysms are rare and require close monitoring to identify electrical or contractile sequelae to ameliorate life-threatening complications.

Key Words: Inferior wall aneurysm; true aneurysm; ventricular tachycardia; electrophysiology; structural interventional cardiology; case report

Anuforo A, Charlamb J, Draytsel D, Charlamb M. Massive inferior wall aneurysm presenting with ventricular tachycardia and refractory cardiomyopathy requiring multiple interventions - a case report. World J Cardiol 2024; 0(0): 0000-0000

URL: https://www.wjgnet.com/1949-8462/full/v0/i0/0000.htm
DOI: https://dx.doi.org/10.4330/wjc.v0.i0.0000
Core Tip: This case report is intended to assist clinicians anticipate and recognize complications arising from true inferior wall aneurysms in a bid to expedite timely pharmacologic and surgical interventions. It will also help outline the role of multidisciplinary care in managing inferior wall aneurysm complications to improve quality of care and help provide guidance in utilizing different imaging modalities to evaluate ventricular aneurysms and help guide therapy.

INTRODUCTION
The prevalence of myocardial infarction in American adults aged 20 and older is 3.1 percent.¹ That equates to about 1.5 million patients in the United States and the overall incidence of left ventricular aneurysms is 30-35 percent of acute transmural myocardial infarctions.² The clinical implications of these aneurysms are profound, manifesting as wall motion abnormalities, reinfarction, ventricular tachyarrhythmias, and an increased risk of sudden cardiac death.³

Ventricular aneurysms have two major risk factors. The first is total occlusion of the left anterior descending artery and the second is failure to obtain patency of the infarcted artery. This can lead to a true ventricular aneurysm or a ventricular pseudoaneurysm. A true aneurysm is a full-thickness outpouching of the ventricular wall. While a pseudoaneurysm is a ventricular wall rupture that remains contained within the pericardium.² Differentiating between a true and pseudo-aneurysm can be difficult but is vital as pseudo-aneurysms have a propensity to rupture leading to cardiac tamponade, shock, and death. True aneurysms generally do not carry these same risk factors.⁴ Nonetheless, they can lead to mural thrombus, arrhythmia, and heart failure.⁵ This report details the case of a patient with a true aneurysm found after sustained ventricular tachycardia (VT) post-cardiac catheterization and placement of a drug-eluting stent. The patient subsequently had surgical resection of the aneurysm,
mitral valve replacement, and intracardiac cryoablation below the mitral valve to prevent future VT episodes.

**CASE PRESENTATION**

*Chief complaints*

We present a 59-year-old male with cardiovascular comorbidities. He presented to our emergency department (ED) with new onset palpitations that had lasted about 90 minutes, wide complex tachycardia tracing on his smartwatch with a heart rate of around 160/min, and concerns for ventricular tachycardia (VT).

*History of present illness*

Before presentation, the patient had no fever, nausea, vomiting, chest pain, syncope, or dyspnea. His clinical presentation was most consistent with scar-related ventricular tachycardia arising from the aneurysm wall. Potential differentials include supraventricular tachycardia with aberrancy, pre-excited supraventricular tachycardia, and antidromic atrioventricular reciprocating tachycardia.

*History of past illness*

One month earlier the patient had a delayed presentation for an acute inferior wall ST segment elevation myocardial infarction (STEMI) (Figure 2) with admission high sensitivity cardiac Troponin (hs-cTn) elevated at 988 ng/L, peaked at 1,225 ng/L and trended down to 954 ng/L. He underwent coronary angiography that revealed an occluded right coronary artery RCA and normal flow through the proximal, mid, and distal LAD and LCx. He then received percutaneous coronary intervention (PCI) with the placement of a drug-eluting stent (DES) to the occluded RCA and this hospital course was complicated by pericarditis managed with Aspirin and colchicine.

*Personal and family history*
He had a history of essential hypertension, dyslipidemia, and type 2 diabetes mellitus and was on Amlodipine, Metoprolol, Atorvastatin, Metformin, and dual antiplatelet with Aspirin and Ticagrelor. Past surgical history was significant for left total shoulder and total knee replacements. The family history was significant for diabetes mellitus and heart disease in his father.

**Physical examination**

Vitals were most significant for tachycardia with a heart rate of 160/min, BP of 122/98 mmHg, respiratory rate of 16/min, and SPO2 of 97% on ambient air. He was alert, oriented, and in no painful distress. He was well hydrated, with no neck vein distension. Chest auscultation was clear bilaterally and cardiac auscultation revealed an apical pansystolic murmur without rubs or gallops. Abdominal exam was within normal limits and he was neurologically intact with warm and well-perfused extremities, with no lower extremity edema.

**Laboratory examinations**

An electrocardiogram (EKG) in the ED confirmed monomorphic VT (Figure 1). Lab testing was most significant for WBC of 13.6 x 10³/L and elevated hs-cTn at 117 ng/L, which increased to 140 ng/L. Other lab values were within normal limits.

**Imaging examinations**

The echocardiogram showed hypokinetic and dyskinetic left ventricular walls, a large basal and mid-inferior-inferoseptal aneurysm with a wide neck, reduced systolic function, and an LVEF 44% as well as moderate mitral insufficiency from a restricted posterior mitral valve leaflet and mild aortic insufficiency (AI) with a dilated ascending aorta of 4.4cm (Figure 3). Prior echocardiogram during the STEMI had shown a dyskinetic segment involving the inferoseptal, basal, and mid-inferior wall which subsequently became aneurysmal. To further evaluate the aneurysmal segment better, a cardiac gated computed tomography (CT) angiography was performed which showed a
large true aneurysm arising from the inferobasal part of the left ventricle with a large neck (3 x 4 cm), dome (6.8 cm diameter) and rightward displacement of the basal septum, with no evidence of a pseudoaneurysm and without any significant stagnation of blood within the aneurysmal cavity (Figure 4 and 5). Cardiac catheterization showed patent RCA stent and 75% stenosed mid-LAD lesion. Intravascular ultrasound (IVUS) demonstrated a mid-LAD fibrofatty plaque with an area of plaque rupture. The lesion was dilated and 3.5 x 33 mm DES was placed, with final 0% stenosis and TIMI 3 flow.

MULTIDISCIPLINARY EXPERT CONSULTATION
Interventional Cardiology
General Cardiology
Critical care Cardiology
Electrophysiology
Cardiac Surgery

FINAL DIAGNOSIS
Sustained scar-related ventricular tachycardia secondary to inferior wall Left ventricular aneurysm post inferior STEMI

TREATMENT
He received a 150mg Amiodarone bolus followed by an infusion at 1mg/min and Esmolol at 50mcg/kg/min in the ED. The esmolol dose was doubled and he received another 150mg Amiodarone bolus for persistent VT and converted spontaneously to sinus rhythm just before anticipated electrical cardioversion. Repeat EKG showed loss of R wave progression in precordial leads and q waves with slight ST elevation in leads V3-V6. After the LAD stent placement, he was continued on dual antiplatelet therapy (DAPT), high-intensity statin, GDMT with Enalapril, Furosemide, Metoprolol succinate, Amiodarone, Dapagliflozin and a wearable cardioverter defibrillator (LifeVest).
Two months after the VT episode, due to concern for the aneurysm expansion, he was referred for LV aneurysm patch repair with polytetrafluoroethylene (PTFE) graft and bioprosthetic mitral valve replacement. He also received a surgical intracardiac cryoablation of the tissue between the mitral annulus and the scar of the inferior MI to prevent future recurrence of mitral annular VT. Surgery was complicated by prolonged mechanical ventilation and pneumosepsis. Due to the history of VT and persistently low pre-op LVEF of around 35%, the patient also had a dual-chamber automated implantable cardioverter defibrillator (AICD) placed and Amiodarone was discontinued due to bradycardia and prolonged QTc interval of 598ms.

OUTCOME AND FOLLOW-UP
Three months postoperatively, he developed recurrent left-sided pleural effusions requiring two sessions of thoracentesis with drainage of 1.5L and 0.7L respectively. Due to postoperative persistent symptomatic LV dysfunction, GDMT was progressively optimized to include Entresto (replacing Enalapril), and Eplerenone in addition to previous medications with improvement in functional status and LVEF up to 50%.

DISCUSSION
This case outlines the complicated clinical course of a patient who developed a true inferior wall aneurysm complicated by sustained ventricular tachycardia one month after presenting with an inferior wall STEMI despite reperfusion. It presents a unique intersection of ischemic complications combining ventricular arrhythmias post-STEMI with structural pathologies requiring combined AICD insertion and mitral valve replacement. In this case, due to unknown magnetic resonance imaging (MRI) compatibility of the patient's orthopedic implants, it was agreed to obtain a cardiac gated coronary CT angiography (CCTA) scan as opposed to the gold standard for LV aneurysm diagnosis, which is cardiovascular MRI.

Most LV aneurysms (75-80%) arise from the apical or anterior wall and are often
associated with established risk factors like absence of collateralization in the setting of left anterior descending (LAD) coronary artery total occlusion, and incomplete or delayed reperfusion. True inferior aneurysms are rare but have similar risk factors with the culprit vessel often being the right coronary artery (RCA) and persistent inferior ST elevation on EKG is usually consistent with the development of an aneurysm. Aneurysm development arises from infarct expansion, which occurs in about 35-45% of anterior MI and less frequently at other locations.

Following the advent of thrombolysis and percutaneous coronary intervention (PCI), large ventricular aneurysms have become uncommon complications of myocardial infarctions. A true aneurysm occurring a month after a STEMI raises questions about the underlying substrate and the potential role of delayed myocardial healing and remodeling. While pseudoaneurysms are at a higher risk of rupture, true aneurysms balloon out in systole causing a loss of kinetic energy required to maintain cardiac output, thus carrying a higher risk of heart failure, the stasis favors thrombus formation, and the scar tissue forms an arrhythmogenic substrate for ventricular arrhythmias. Complications of aneurysm formation are responsible for a six-fold increase in the mortality of acute coronary syndrome, death is usually from sudden cardiac death.

This case highlights a greater need for arrhythmia risk stratification in post-infarction patients with regional myocardial dysfunction as well as the need for further exploration of potential underlying arrhythmogenic substrates, the possible role of scar-related reentry mechanisms and the impact of worsening myocardial dysfunction. Furthermore, this patient had a dual-chamber AICD placed for secondary prevention of recurrent ventricular tachycardia managed with Amiodarone, and worsening LVEF of 35% after a trial of a LifeVest while on guideline-directed medical therapy (GDMT). Due to the patient’s elevated risk of sudden cardiac death on account of depressed LVEF, history or recurrent ventricular tachycardia, and STEMI within the past 40 days, the patient had
been placed on a LifeVest. More specific indications would include LVEF ≤35% within 90 days of coronary artery bypass graft (CABG), newly diagnosed but potentially reversible nonischemic cardiomyopathy, or severe heart failure awaiting transplantation. However, some months after VT ablation and after a device check revealed no recurrent episodes of VT, it was decided to discontinue Amiodarone due to persistent bradycardia, markedly prolonged QTc interval of 598ms, and the increased risk of Torsades.

Inferior aneurysms could be associated with mitral regurgitation (MR) from disruption of papillary muscle anatomy due to scar formation and subsequent mitral leaflet tethering. This patient had moderate MR from dyskinetic walls and a restricted posterior mitral valve leaflet further complicating his heart failure symptoms, hence mitral valve replacement was required. The coexistence of mitral valve disease emphasizes the complexity of managing concurrent structural heart disease and arrhythmias.

Uncomplicated ventricular aneurysms are usually managed pharmacologically with GDMT and anticoagulation. However, in the setting of persistent ventricular arrhythmia or refractory heart failure unresponsive to GDMT (in this case), surgical intervention is strongly indicated. Surgical repair significantly reduces heart failure symptoms and VT episodes. Of note, the perioperative risk of mortality for aneurysmal repair is high particularly when additional coronary artery bypass or valvular surgery is required especially when the LVEF is 35% or less. This patient had an inferoseptal/inferobasal aneurysmectomy with patch repair, concomitant bioprosthetic mitral valve replacement, and AICD implantation with significant improvement at one-year follow-up. VT Cryoablation was guided by anatomical landmarks and performed from the scar area to the posterior mitral annulus (P2/P3 area) both from the LV endocardium side and epicardium side during the MV replacement procedure. This emphasizes the challenges of managing post-infarction patients, including the potential
impact of ventricular mechanics and arrhythmia substrate.

Also noteworthy was the fact that the decompensated heart failure state postoperatively was complicated by symptomatic pleural effusions requiring thoracentesis procedures with drainage of 2.2L. In this patient, further postoperative optimization of GDMT was associated with improvements in LVEF, pleural effusion, and functional status. This case underscores the evolving landscape of collaborative care involving interventional cardiology, cardiac surgery, and electrophysiology in addressing multifaceted cardiac pathologies.

CONCLUSION
This case provides valuable insights into the management of high-risk patients with complex post-infarction complications and underscores the importance of vigilant monitoring and risk assessment in the post-STEMI period in patients with inferior wall involvement. Additionally, it reveals the significant complications, perioperative challenges, and guideline-directed interventions for a true inferior wall left ventricular aneurysm and aims to contribute to the enhancement of treatment protocols within the realm of cardiology.
## ORIGINALITY REPORT

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**SIMILARITY INDEX**

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