

## A Rare Case of Anterior Wall Myocardial Infarction Presenting as Acute Limb Ischemia

Paul N, Vasudev R\*, Shah P, Rampal U, Patel H, Virk H, Bikkina M and Shamoof F

St Joseph's University Medical Center, Paterson, NJ, USA

### Abstract

Left ventricular thrombus (LVT) formation after an acute myocardial infarction (MI) is dependent on age, sex, co morbid conditions of the patient as well as the characteristics of the MI like the type of infarction, area of endocardium involved, duration of MI, inflammatory markers and tissue acoustic properties of the thrombus itself. LVT carries a great risk of embolism, which also varies as per the thrombus location and mobility. It is rare to have arterial occlusion by emboli from LVT after MI. We present a forty two years old female with acute limb ischemia as a result of arterial emboli originating from LVT after one week of acute MI. Percutaneous intervention was done for occluded coronary vessels as well as thrombolysis of the arterial clot, was performed. In this case two interventions were successfully done alongside. Timely intervention and initiation of anticoagulation is the most valuable therapy in such cases.

**Keywords:** Left ventricular thrombus; Acute myocardial infarction; Acute limb ischemia; Left ventricular aneurysm

### Introduction

Thromboembolism resulting from acute myocardial infarction is secondary to the friability of LVT. Early medical therapy for MI can prevent clot formation, however if formed, early diagnosis can reduce the chances of embolism. Several cases of strokes have been reported, however there are only a few cases of limb ischemia post myocardial infarction seen. With the advent of anticoagulation and novel techniques of imaging of left heart chamber, the incidence of thromboembolism is decreasing. The thrombus is situated within the apex of left ventricle, especially when there is a large infarction or aneurysm of the left ventricle. As the chamber deforms into an aneurysm, there is collection of fibrous tissue, which makes the blood coming into its contact to clot formation.

### Case Description

42 years old female with past medical history of hypertension and Type II diabetes mellitus, non-compliant to medication, presented to the emergency department with complaint of numbness and pain in left leg for three days. There was history of having experienced intermittent severe sub sternal chest pain for one week. On examination, left lower extremity had diminished peripheral pulses and was cold. Electrocardiogram showed ST elevations in leads V1 to V4 and hence, a STEMI code was called and patient was rushed to catheterization laboratory. There was total mid left anterior descending artery (LAD) occlusion. Two drug eluting stents were placed, one in the mid LAD and one in the mid to distal left anterior descending. Transthoracic echocardiogram showed thrombus in the left ventricle. Angiogram of the lower extremities was taken which showed acute occlusion of anterior tibial artery, posterior tibial artery and profunda femoris artery on the left side. Vascular surgeon was consulted, while patient was still in the Catheterization lab. Catheter assisted thrombolysis (EKOS) was performed. The EKOS device was set up and patient had localized thrombolysis using TPA infusion. After the procedure, patient was kept under monitoring in cardiac care unit. Peripheral pulses returned and patient's symptoms had resolved. Patient was discharged after 4 days, on dual antiplatelet therapy and counseled for with close follow up.

### Discussion

Incidence of left ventricle thrombus as a result of myocardial infarction has decreased significantly in the current era of anticoagulation. There is a strong temporal relation between LVT formation and occurrence of MI. On the basis on clinical evidence, it is noted that LVT forms within 5 days of acute MI or 12.0 days after MI [1]. Patients receiving timely therapy [hospitalization and treatment within 12 hours of acute chest pain], have a lower incidence of LVT [15.4%] compared to

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#### \*Correspondence:

Rahul Vasudev, St Joseph's University Medical Center, Paterson, NJ, USA.

E-mail: [drrahulvasudev@gmail.com](mailto:drrahulvasudev@gmail.com)

Received Date: 12 Feb 2018

Accepted Date: 13 Mar 2018

Published Date: 16 Mar 2018

**Citation:** Paul N, Vasudev R, Shah P, Rampal U, Patel H, Virk H, et al. A Rare Case of Anterior Wall Myocardial Infarction Presenting as Acute Limb Ischemia. *J Clin Case Rep.* 2018; 1(1): 1004.

ISSN 2643-8194

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those diagnosed after 12 hours [63.6%] [2].

Thrombus formation is more likely in a STEMI [43.1%], as compared to NSTEMI [15%] [3]. LVT formation is highly dependent on clinical presentation and epidemiological risk factors. Interestingly, family history of myocardial infarction is believed to be a predictor of LVT. Presence of Anterior infarction, left ventricular dilatation, dyskinesia of LV, aneurysm of LV and a reduced systolic left ventricular function (ejection fraction < 40%) are known to promote LVT [4]. There was anterior wall infarction, which is highly prone to give rise to left ventricle thrombus, especially in this case where there was possible stasis for about seven days, prior to diagnosis.

Abnormal flow pattern [LV Vortex depth and relative strength] are strongly associated with LVT formation [5-7]. Virchow's triad of blood stasis, endothelial injury and hypercoagulability, is the pathophysiology of intracavitary thrombus. Areas of infarction have sluggish blood flow as compared to normally perfused areas with well contracting muscles. Therefore, patients at highest risk are those with large anterior ST elevation myocardial infarction with anteroapical aneurysm formation. Dyskinesia or akinesia in the LV aneurysm as well as contact of blood with fibrous tissue in the aneurysm rather than normal endocardium is also thought to trigger clot formation.

In the setting of risk factors, thrombus formation is associated with presence of anticardiolipin antibodies (ACA). Complexes are formed between autoantibody and beta-2 glycoprotein 1 on activated platelets at sites of endothelial injury, such binding promotes further platelet activation and augments thrombosis [8]. C-reactive protein (CRP), fibronectin, mean Platelet Volume [MPV], neutrophil lymphocyte ratios and Factor V Leiden, are the biochemical markers directly related to LVT [9-12].

LVT can shower emboli and may present as acute limb pain, abdominal pain, flank pain or stroke [arterial ischemia, splenic and renal infarction] [13-19].

Thirteen fold higher risk of embolism with LVT. Majority of emboli are cerebrovascular accidents. Predictors of embolisation vary from patient characteristics (age, gender and onset of symptoms) and thrombus variables (thrombus movement or mobility, shape, heterogeneity, layering, presence within an aneurysm and pendulousness). Thrombus is more prevalent in age more than 50 years [20-22]. Patients with LVT formed within 48 hours of acute MI, have higher in-hospital mortality (42.5% versus 13%) as compared to those who develop thrombus later [23]. Diagnosis of LVT can be done by transthoracic echocardiogram, abnormal flow pattern by Doppler echocardiography, Power Doppler-derived speckle tracking image of intraventricular flow, myocardial performance index or cardiac magnetic resonance [24-29]. There is evidence suggesting the role of prophylactic anticoagulation or thrombolysis for LVT in high risk patients with acute MI. Also there is less risk of thromboembolism [30-34].

## Conclusion

LVT formation after acute MI is not uncommon. However embolic shower from LVT resulting in arterial occlusion is rare. It is critically important to prevent its formation, diagnose as early as possible and early intervention to prevent embolization. Our case describes a female hypertensive and diabetic patient who was diagnosed with LVT after seeking medical attention for lower extremity pain. The cause of LVT was acute anterior wall MI, onset of which was one week

from the day of presentation. Early hospitalization and intervention could have prevented embolization in this case.

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