Case Report

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Interventional Cardiology Journal
ISSN 2471-8157

2021
Vol.7 No.5:129

DOI: 10.36648/2471-8157.7.5.129

Ominous triad of LV thrombus, acute limb ischemia and triple vessel SCAD

Vivek Singh Guleria*, G Keshavamurthy

Department of Cardiology, Army Hospital Research & Referral, New Delhi, India

*Corresponding author:
Vivek Singh Guleria

viveksguleria@gmail.com

Department of Cardiology, Army Hospital Research & Referral, New Delhi, India

Citation: Guleria VS, Keshavamurthy G (2021) Ominous triad of LV thrombus, acute limb ischemia and triple vessel SCAD. Interv Cardiol J Vol.7 No.5:129

Abstract

Triple vessel spontaneous coronary artery dissection (SCAD) has rarely been reported in males. We herein document a case of young male who presented in ED with acute limb ischemia. Evaluation revealed inferior wall myocardial infarction, left ventricular thrombus and SCAD of all three coronary vessels.

Received: April 22, 2021; Accepted: May 14, 2021; Published: May 20, 2021

Introduction

Spontaneous coronary artery dissection (SCAD) was first described in 1931 by [1]. It is one of the rare and fatal causes of acute coronary syndrome (ACS) in young females. With advancement of intracoronary imaging techniques, it is increasingly being reported. SCAD is important cause of coronary events in otherwise healthy young individuals without traditional risk factors. Risk factors for SCAD comprise pregnancy, Ehlers-Danlos disease, Marfan's Syndrome, intensive exercise, or cocaine abuse. SCAD in males is rare and triple vessel SCAD is seldom seen. Only 7 cases of triple vessel SCAD have been reported in literature [2].

Left ventricular thrombus (LVT) is common complication after ST elevated anterior wall myocardial infarction and is rarely seen in inferior wall infarction [3]. We present a case of triple vessel SCAD, complicated by acute limb ischemia because of thromboembolism of left ventricular thrombus in a 37-year-old man with inferior wall myocardial infarction.

Case Report

37 years old male with BMI of 31 kg/m2 presented with sudden onset pain right leg in emergency department. He also gave history of mild chest discomfort 5 days back which resolved off its own. He had tachycardia with heart rate of 100/min, and blood pressure 170/100 mmHg. Local examination revealed absent right popliteal, anterior, and posterior tibial arteries with capillary filling time of >3 seconds. In view of acute limb ischemia colour doppler was done which showed no flow beyond superficial femoral artery (SFA) and a large thrombus in SFA. CT angiography showing left ventricular clot (Figure 1a) and thrombus in right common femoral artery, external iliac artery, profundal femoris and SFA (Figure 1b).

Electrocardiogram of the patient had "q" waves in inferior leads. His cardiac biomarkers were raised but other biochemical and haematological profile was normal. Transthoracic echocardiography was done immediately which showed left ventricular hypertrophy, akinetic basal and mid inferior wall with ejection fraction of 40%. There was a large 51x23 mm oscillating thrombus in left ventricle (Figure 2).

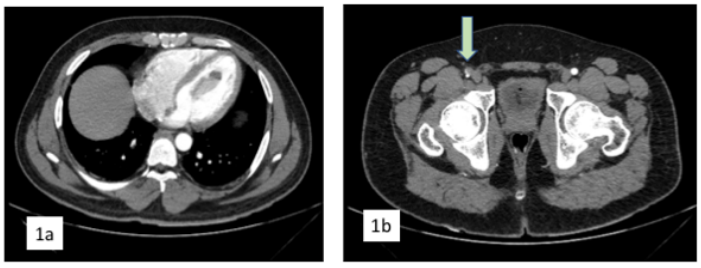

Figure 1 a) CT angiography showing left ventricular clot. b) Thrombus in right superficial femoral artery.

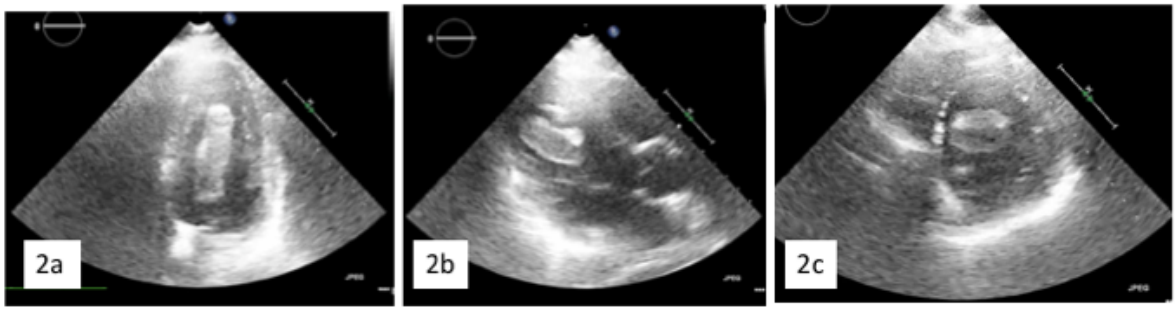

Figure 2 Transthoracic echocardiography showing a large organized left ventricular clot in 4 chamber view (2a), in parasternal view (2b) and short axis view(2c).

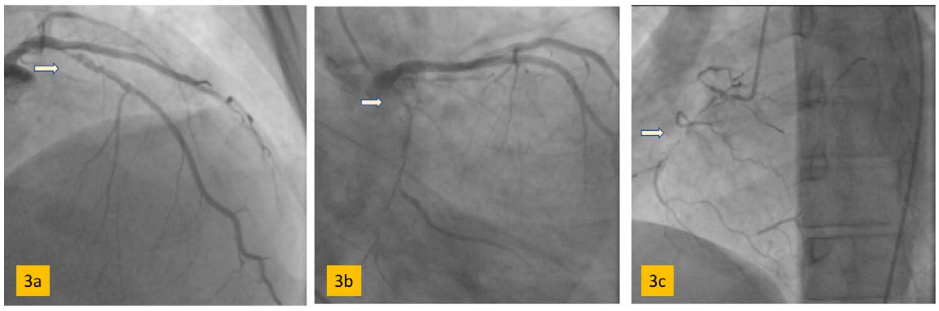

Figure 3 Coronary angiography of the patient. RAO cranial view showing SCAD of left anterior descending (LAD) artery extending from proximal to middle part (3a). RAO caudal view showing SCAD in left circumflex artery extending from ostial to distal part(3b). LAO cranial view SCAD OF right coronary artery (RCA) extending from ostium to distal part; LAD being refilled retrogradely(3c).

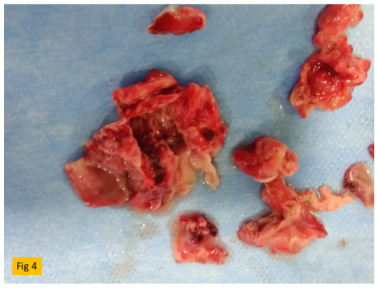

Figure 4 Large well-organized clot removed from left ventricle.

Patient was taken up coronary angiography which revealed spontaneous coronary dissection of right coronary artery (RCA) (Figure 3a). Mid Left anterior descending artery (LAD) and proximal to distal left circumflex (LCx) also showed SCAD (Figure 3b,3c).

Patient was immediately taken up in hybrid operation theatre for coronary artery bypass grafting, LV clot removal and thromboembolectomy. Per op findings revealed large, organized LV clot adherent to inferior wall and septum which was removed (Figure 4) followed by right thromboembolectomy and CABG. Post op period was uneventful.

Discussion

SCAD occurs in those patients who lack traditional cardiovascular risk factors. Of all ACS cases SCAD is the etiological factor in 1-4% [4]. However, it is responsible for about 35% of ACS cases in women <50 years of age [5] [6] have reported that male SCAD patients present at a slightly younger age than women. LAD is the most affected artery (32-46%) followed by LCx (15-45%). RCA is involved in 10-39% of patients [7]. Regarding triggering factors for SCAD, two theories have been proposed. According to the first theory there is an intimal tear which allows seepage of blood from true lumen to enter the intimal tear and create a false lumen. In the second theory the inciting event is proposed to be a spontaneous haemorrhage arising from vasa vasorum.

Presenting symptoms in SCAD are like atherosclerotic ACS. 3-11% SCAD patients can present as ventricular arrhythmias or sudden

cardiac death.7 Intravascular ultrasonography (IVUS) and optic coherence tomography (OCT) are imaging modalities of choice for diagnosing SCAD as they provide detailed visualization of vessel wall. OCT because of its better resolution has become the gold standard for diagnosing SCAD. In our case even though CAG had revealed SCAD with high diagnostic accuracy we tried to utilize intracoronary imaging, but coronary wire could not be negotiated. Our patient had LVT that had started embolizing post inferior wall infarction.

The incidence of LVT in patients with acute anterior MIs in the prethrombotic era is ranged from 20% to 40% and with a non-anterior acute myocardial infarction (AMI) the risk of LVT was <5%. In present era it is estimated to be around 5%.3

Traditionally, the causes of LVT formation after acute ST-segment elevation MI include segmental dysfunction of the infracted myocardium resulting in the stasis of blood, endocardial tissue inflammation that provides a thrombogenic surface and a hypercoagulable state [8]. In our patient LVT cannot be attributable to inferior wall infarction alone. Since he is on oral anticoagulants, he will be worked up for prothrombotic state eventually.

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