ECRONICON OPEN ACCESS

EC CARDIOLOGY
Case Report

Left Anterior Descending Coronary Artery Thrombus in Young Patient with Systemic Lupus Erythematosus (SLE) Causing Acute Myocardial Infarction

Hanan B Al Backr*

Associate Professor, Department of Cardiac Sciences, King Fahad Cardiac Center, College of Medicine, King Saud University, Saudi Arabia

***Corresponding Author:** Hanan B Al Backr, Associate Professor, Department of Cardiac Sciences, King Fahad Cardiac Center, College of Medicine, King Saud University, Saudi Arabia.

Received: July 27, 2021; **Published:** August 16, 2021

Abstract

A 39 years old gentleman smoker with l history of SLE and DVT presented with (NSTEMI) electrocardiogram was normal with elevated troponin I. Laboratory data revealed elevated cholesterol, positive ANA. Cardiac catheterization revealed (LAD) thrombus. His LAD thrombus was treated with aspiration thrombectomy and given triple antithrombotic therapy. APL antibodies negative.

Keywords: *Systemic Lupus Erythematosus; Left Anterior Descending Coronary Artery; Thrombus*

Introduction

Ischemic heart disease is a common complication of systemic lupus erythematosus (SLE) and may be observed in up to 16% of SLE patients [1]. Most coronary events in adults are related to atherosclerosis; however, approximately 20% of coronary heart disease in young adults is related to non-atherosclerotic factors such as coronary abnormalities, connective tissue disorders, and autoimmune diseases [2]. Autoimmune diseases, such as systemic lupus erythematosus (SLE), are associated with thrombosis and atherosclerosis. Myocardial infarction is observed in patients with SLE in all age groups; it appears as the first presentation of SLE disease or during the course of the disease [3,4]. The most common underlying etiologies of acute myocardial infarction (AMI) in SLE patients include coronary atherosclerosis, thrombosis, arteritis and myocarditis. We report a case of AMI in SLE patient due to left anterior descending artery thrombosis treated with aspiration thrombectomy and triple antithrombotic therapy.

Case Presentation

This is 39-year-old young man who is known to have SLE diagnosed based on arthralgia, malar rash and positive serology. He is on Cellcept, hydroxychloroquine, prednisolone. Had history of Lupus nephritis based on biopsy. He gave History of hypertension on valsartan 160 mg. He had history of left lower limb DVT, was on anticoagulation for 3 months, currently off anticoagulation. He had history of depression/insomnia on olanzapine and escitalopram.

His SLE is controlled on medications, no joints pain or stiffness, no ulcers or skin changes, presented with 1 day history of chest pain, left sided radiating to left shoulder and neck, sudden onset and exertional, no relation to food or position, lasted for about 2 hours, improved after receiving aspirin, not associated with diaphoresis, palpitations or syncope, no SOB, orthopnea or PND. He is a chronic

smoker, no family history of premature CAD or sudden cardiac death, no alcohol or illicit drug abuse, no fever, productive cough or contact with sick patients. No recent upper respiratory tract infection, no prolonged travel, lower limb swelling or leg pain. On examination, Temperature: 36.7°C (Oral), Respiratory rate: 20/min, Blood Pressure: 119/67, HR = 85 beats/min, regular and equal pulses bilaterally, pO_2: 99% on room air. His weight is 90 kg, jugular venous pulse (JVP) normal, no murmurs or added sounds, no lower limb edema nor tenderness, Chest examination normal breath sounds. ECG showed Normal sinus rhythm with no ischemic changes (Figure 1).

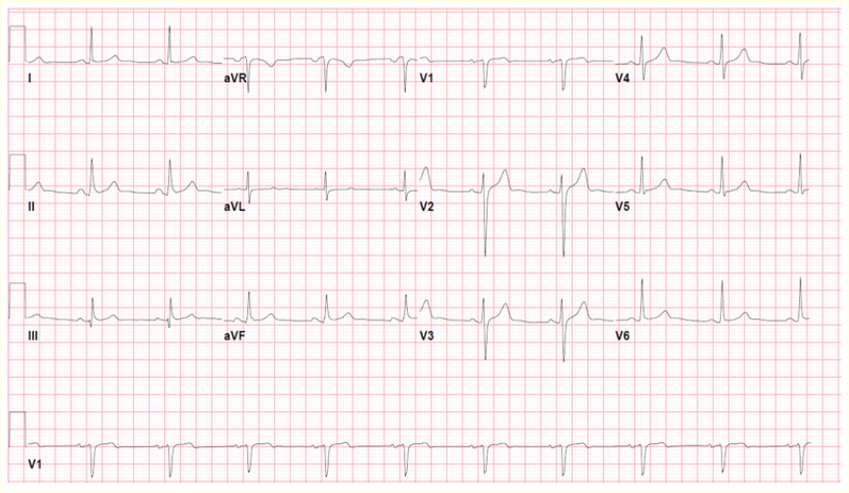
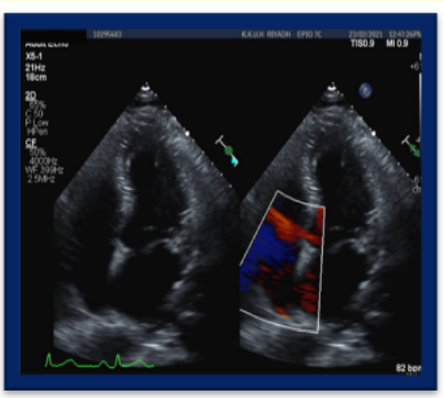


Figure 1: *12 lead ECG.*

Laboratory data revealed the following, no leukocytosis, Hemoglobin is normal, normal platelet no, renal panel and electrolytes with in normal, NT pro BNP is 41 ng/L, and troponin I is 8168 ng/L (normal 00.0 - 19.00) which went down by half to 4000 ng/l within nine hours of admission and declined over his hospital course. ANA 1:80 (positive), C3, C4 were normal both, normal CRP and ESR and total cholesterol high 6 mmol/L (normal 3.2 - 5).

Hospital course and management: The patient admitted to the hospital as case of known SLE with positive cardiac markers, the working diagnosis was non-ST elevation myocardial infarction (NSTEMI) and treated as such with aspirin, Plavix, heparin, beta blockers and high dose statins. Other possible causes include pulmonary embolism and myocarditis was also entertained as differential diagnoses of his chest pain.

Echocardiogram (Figure 2), showed left ventricle is normal in size, left ventricular systolic function normal, the left ventricular wall motion normal. The right ventricle is normal in size and function. No pericardial effusion. No valve pathology.



a

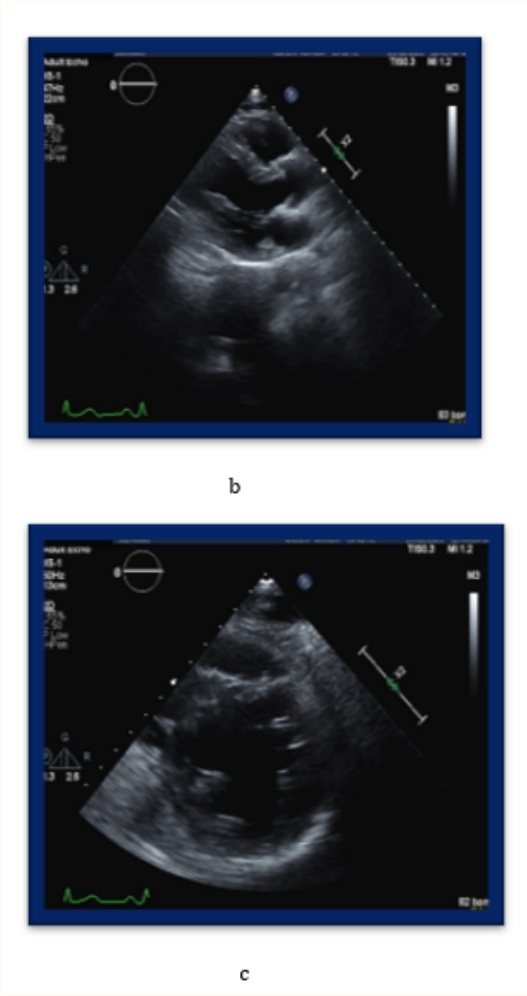


Figure 2a-2c: *Shows adult transthoracic echocardiogram of the patient. a. Four chamber view of the heart with color doppler, b. Parasternal long axis window of left ventricle and c. Short axis window of left ventricle.*

Coronary angiogram (Figure 3) is done through the right radial artery approach showed, dominant right system, left ventricle end diastolic pressure high 17 mmHg, left main coronary artery normal. Left anterior descending artery (LAD) showed Minimal atherosclerotic disease with a thrombus occluding more than 95% of LAD after first Diagonal artery (D1). Left circumflex artery Normal, right coronary artery showed mild 30% lesion at mid segment. So, Impression was, this is non-obstructive coronary artery disease (CAD) with LAD thrombus after D1. After first left heart catheterization (Cath) the plan was to give him Aggrastat infusion (GP2b/3a inhibitor) for 24 hours with low dose of Heparin 500 IU per hour, Full dose Heparin after stopping Aggarastat for 48 hours, continue Plavix and Aspirin, considering repeat angiogram after few days. The patient was doing well, his chest pain improved and he did not have any bleeding complications form the antiplatelet and the anticoagulants. Two days later sent for the second coronary angiogram (Figure 4) and he underwent aspiration thrombectomy of LAD thrombus as a therapeutic intervention with good results. Seen by Rheumatologist to assess his SLE status and antiphospholipid antibodies was negative. In the second left heart cathetrization, Residual mid LAD thrombus

was found despite pharmacological treatment with GP2b/3a inhibitor and anticoagulation for 48 hrs so treated currently with balloon angioplasty and aspiration thrombectomy. Post cath recommendations to Start Warfarin for 3 months with INR between 2 - 3, Continue aspirin and clopidogrel indefinite, follow up with cardiology clinic in one month to repeat coronary angiogram if still persistent he will go for intracoronary imaging and possible percutaneous coronary stent intervention (PCI). The patient sent home in a very stable condition.

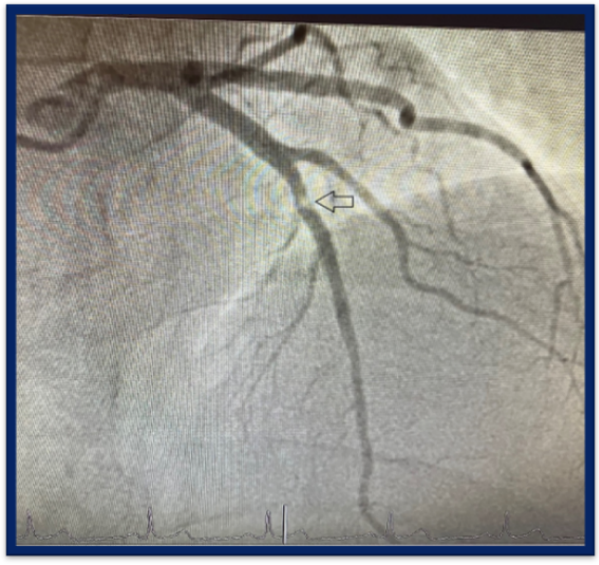
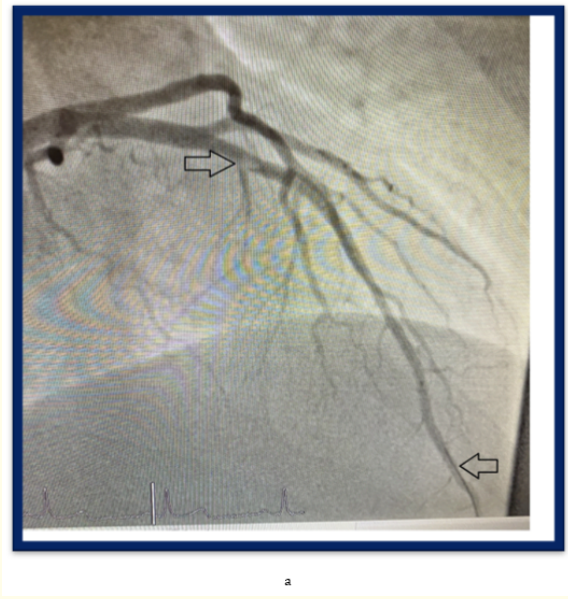
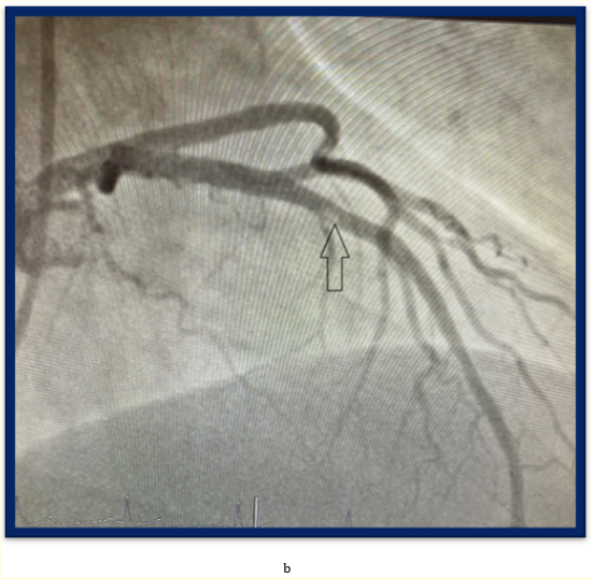


Figure 3: *Left coronary angiogram of the patient showing, left anterior descending artery (LAD) showed minimal disease with a thrombus after first diagonal artery (arrow).*



a



b

Figure 4: *Left heart Cath for therapeutic aspiration thrombectomy, a. Showing the thrombus and the wire (arrwos) in LAD, b. Showing the final result with significant decrease in the thrombus burden in LAD (arrow) after aspiration.*

Before discharge, he was counselled about importance of smoking cessation,also adviced for strict control of his cardiac risk factors hyperlipidemia and hypertension, he was also counsleled extensively about warfarin use, including dietary and medical caution, the importance of compliance and frequent monitoring. His discharge medications was as follows, aspirin 81 mg, atorvastatin 80 mg, bisoprolol 2.5 mg, Clopidogrel 75 mg for one month, warfarin 5 mg for three months, CellCept Escitalopram, hydroxychloroquine, olanzapine, pantoprazole 40 mg, prednisone and Valsartan 160 mg. He was seen in the cardiology clinic after on month of discharge remained stable, aspirin was stopped after one month, continued on palvix and warfarin.

Discussion

39-year-old man with documented SLE experienced an acute myocardial infarction due to thrombus in mid-portion of the left anterior descending coronary artery, he was initially treated with aggressive antiplatelet therapy including GP2b/3a inhibitors and heparin followed by aspiration thrombectomy and aggressive triple antithrombotic therapy. His symptoms improved, howevere he had multiple cardiac risk factor that he was councelled carefully about their control and to be compliant with his anticoagualtion therapy. He was adviced to follow up with rhumatologist to rule out antiphospholipid syndrome (APL). Our patient SLE status was not active according to his symptoms and his lab data, however anticoagulant therapy and antiinflammatory medication may be necessary to prevent myocardial infarction in patients with SLE, even if there is no immunological evidence of active SLE [5]. The pathophysiology of lupus-mediated coronary events seems to be different according to multiple case report of acute MI in lups patients [5,6]. Patients with SLE are predisposed to acute thrombosis from clotting dysfunction rather than age-related atherosclerosis. Furthermore systemic lupus erythematosus is a chronic inflammatory disorder that predisposes to acute coronary thrombosis which could be treated with medical threrapy alone [6]. Cardiac complications of lupus affect most parts of the heart, these include pericarditis, myocarditis, endocarditis and coronary artery disease.Acute myocardial infarction (AMI) is relatively rare in systemic lupus erythematosus (SLE), although other cardiac complications, such as pericarditis myocarditis and mitral valve thickeining occur more frequently in this disease [7]. Many case reports in SLE patients with AMI or CAD, looked at the role of APLS and found that due to the proinflammatory and prothrombotic nature of APS, the

presence of APL is a predictor of coronary artery disease in SLE patients [8]. Additionally, APS can precipitate a thrombosis, resulting in myocardial infarction in the absence of coronary artery disease [9]. Reynaud Q and his collegues reported that a thrombotic event in SLE patients is mostly related to antiphospholipid syndrome (APS) [10]. APS can manifest as venous or arterial thrombosis with abnormally elevated levels of serologic markers anticardiolipin antibody (aCL) and/or lupus anticoagulant (LA). Moreover LA and aCL were significantly associated with an increased risk of thrombosis, especially arterial, in patients without SLE. Our patient did not have lupus anticoagulant or other serologic markers.There are no definitive guidelines regarding anticoagulation therapy in an SLE patient with thrombosis. In patients with APS and a thrombotic event, anticoagulation therapy is recommended [11]. Our case have other cardiac risk facors including hypertensoin, hyperlipidemia and somking. A review of literature about CAD in SLE patients done from 1980 to 2000, suggests that SLE is a significant risk factor for coronary atherosclerosis independent of the classic risk factors of hypertension, tobacco use, and hyperlipidemia [12]. The etiology of the accelerated atherosclerosis in SLE is not known but has been linked to inflammation and endothelial dysfunction. Most of the case reports similar to our case share the fact that Acute myocardial infarction (AMI) resulting from CAD has various pathophysiology depending on the clinical scenario [13]. Because the pathophysiology of coronary involvement in SLE is multifactorial and there are no guidelines to tell us how to manage those patients, the diagnosis and appropriate therapy may be complex. In our patient, only 1 month of anticoagulation was recommended for him then dual antiplatelet therapy for 1 year was palnaned. When the cause of coronary artery occlusion is a thrombus, aspiration may be helpful as a primary therapy. In many cases similar to ours, predilatation, thrombus aspiration, and stent implantation are routinely performed. Although stent implantation was not performed in this case, follow-up angiography was planned to be done in one to three months. This case suggests that AMI in this patient was due to thrombosis, and evaluation of a lesion after thrombus aspiration can avoid unnecessary procedures, especially stent implantation.

Pathophysiology	Atherosclerosis	Acute thrombosis	Coronary vasculitis	Coronary spasm
Presentation	CAD risk factors diffuse disease on angiography, characteristic IVUS findings	APS history of acute thrombosis, characteristic IVUS findings	Presence of aneurysm	Response to intracoronary nitrates
Treatment	Angioplasty and stenting	Aggressive antiplatelet therapy	Systemic steroids 8 angioplasty and stenting	Calcium channel blockade, long-acting nitrates

Table 1: *Pathophysiology of AMI in a patient with lupus [6].*

Conclusion

Patients with SLE are predisposed to acute thrombosis from clotting dysfunction rather than age-related atherosclerosis. There is little evidence based decisions the acute management of AMI in patients with SLE. We presented our management for this case of AMI in patients with SLE. There are case reports of AMI treated differently, and successfully, depending on the clinical situation. Our case makes an argument for the management of acute coronary thrombosis in SLE patient with aggressive anticoagulation and antiplatelet therapy plus thrombus aspiration without stent implantation. There is a need for more evidence-based studies to better understand the natural disease course in this condition and to asses what is the best management strategy for those patients.

Conflict of Interest

The authors has no conflicts of interest to declare.

IRB Approval

This case report was approved by institutional review board of King saud universiy, Riyadh, Research Project No. E-21-5815.

Acknowledgement

This project is supported by college of medicine research center, deanship of scientific research, King Saud University, King Saud university medical city.

Funding Support

None.

Bibliography

1. Badui E., *et al*. "Cardiovascular manifestations in systemic lupus erythematosus. Prospective study of 100 patients". *Angiology* 36 (1985): 431-441.
2. Jessica B Rubin and William B Borden. "Coronary Heart Disease in Young Adults". *Current Atherosclerosis Reports* 14 (2012): 140-149.
3. H González-Pacheco., *et al*. "Acute left main coronary artery thrombosis as the first manifestation of systemic lupus erythematosus and catastrophic antiphospholipid syndrome". *American Journal of Emergency Medicine* 32 (2014): 197.e3-197.e5.
4. Kota Murai., *et al*. "Acute myocardial infarction in a patient positive for lupus anticoagulant: a case report". *BMC Cardiovascular Disorders* 19 (2019): 167.
5. H Kurokawa., *et al*. "Myocardial infarction due to thrombi in coronary aneurysms in a young woman with systemic lupus erythematosus". *Journal Cardiograpy* 16.1 (1986): 249-258.
6. Medical Therapy., *et al*. "Effective Management of Acute Coronary Thrombosis in a Young Woman with Lupus Using Aggressive Cardiology 118 (2011): 45-49.
7. D Jain and M K Halushka. "Cardiac pathology of systemic lupus erythematosus". *Journal of Clinical Pathology* 62 (2009): 584-592.
8. Petri M. "Detection of coronary artery disease and the role of traditional risk factors in the Hopkins Lupus Cohort". *Lupus* 9 (2000): 170-175.
9. Davies JO and Hunt BJ. "Myocardial infarction in young patients without coronary atherosclerosis: assume primary antiphospholipid syndrome until proved otherwise". *International Journal of Clinical Practice* 61 (2007): 379-384.
10. Reynaud Q., *et al*. "Risk of venous and arterial thrombosis according to type of antiphospholipid antibodies in adults without systemic lupus erythematosus: a systematic review and meta-analysis". *Autoimmunity Reviews* 13 (2014): 595-608.
11. Koniari I., *et al*. "Antiphospholipid syndrome; its implication in cardiovascular diseases: a review". *Journal of Cardiothoracic Surgery* 5 (2010): 101.
12. A Karrar., *et al*. "Coronary artery disease in systemic lupus erythematosus: A review of the literature". *Seminars in Arthritis and Rheumatism* 30.6 (2001): 436-443.
13. C Korkmaz., *et al*. "Myocardial infarction in young patients (≤35 years of age) with systemic lupus erythematosus: a case report and clinical analysis of the literature". *Lupus* 16 (2007): 289-297.

Volume 12 Issue 9 September 2021