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Acute Inferior Wall ST-Elevation Myocardial Infarction in a Young Woman with Systemic Lupus Erythematosus and Lupus Nephritis: A Case Report

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Abstract: ***Background: Systemic lupus erythematosus (SLE) is associated with a significantly increased risk of premature cardiovascular disease due to chronic inflammation, immune-mediated vascular injury, and thrombosis. Myocardial infarction (MI) in young patients with SLE is uncommon and frequently occurs in the absence of traditional cardiovascular risk factors.[1-3] Case Presentation: We report the case of a 22-year-old woman, a known case of SLE with lupus nephritis diagnosed at 14 years of age, who presented with acute onset chest pain associated with nausea, vomiting, and giddiness. Electrocardiography revealed ST-segment elevation in the inferior leads (II, III, and aVF). Coronary angiography demonstrated total occlusion of the distal right coronary artery. Transthoracic echocardiography showed a left ventricular ejection fraction of 45% with regional wall motion abnormalities involving the inferior, infero-septal, and infero-lateral walls. Conclusion: This case highlights the occurrence of acute myocardial infarction in a young woman with SLE and lupus nephritis, emphasizing the need for high clinical suspicion of coronary events in SLE patients presenting with chest pain, even in the absence of conventional risk factors.[1,4]***

Keywords: Systemic lupus erythematosus, Lupus nephritis, Myocardial infarction, Young female, Coronary thrombosis

1. Introduction

Systemic lupus erythematosus is a chronic autoimmune disease characterized by multisystem involvement and immune-mediated inflammation. Cardiovascular disease has emerged as a leading cause of morbidity and mortality in patients with SLE.[1] The risk of myocardial infarction is markedly increased in young women with SLE compared with the general population.[2,3]

The mechanisms responsible for premature coronary artery disease in SLE include accelerated atherosclerosis, endothelial dysfunction, immune complex–mediated vascular injury, steroid-related metabolic abnormalities, and a prothrombotic state.[4] We report a case of acute inferior wall ST-elevation myocardial infarction in a young woman with longstanding SLE and lupus nephritis.

2. Case Presentation

A 22-year-old female presented with sudden onset severe retrosternal chest pain associated with nausea, vomiting, and giddiness of 2 hours duration. She had no history of hypertension, diabetes mellitus, smoking, or dyslipidemia.

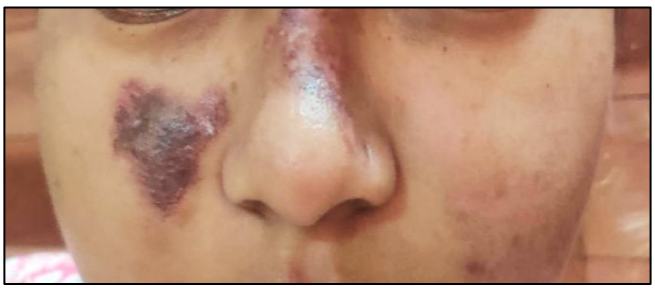


Figure 1: Clinical photograph showing malar rash involving the nasal bridge and malar areas, characteristic of cutaneous involvement in systemic lupus erythematosus.

She was a known case of systemic lupus erythematosus with lupus nephritis, diagnosed at 14 years of age when she initially presented with malar rash (Figure 1). At diagnosis, immunological evaluation revealed antinuclear antibody positivity with a speckled pattern (1:100), anti–double stranded DNA antibody positivity, and markedly elevated erythrocyte sedimentation rate (104 mm/hr). Renal involvement was documented with a 24-hour urinary protein excretion of 532 mg/day. She had been receiving oral prednisolone (5 mg/day), mycophenolate mofetil (360 mg/day), and hydroxychloroquine regularly.

Electrocardiography showed ST-segment elevation in leads II, III, and aVF, consistent with acute inferior wall ST-elevation myocardial infarction (Figure 2). Transthoracic echocardiography demonstrated a left ventricular ejection

fraction of 45% with hypokinesia of the inferior, infero-septal, and infero-lateral walls.

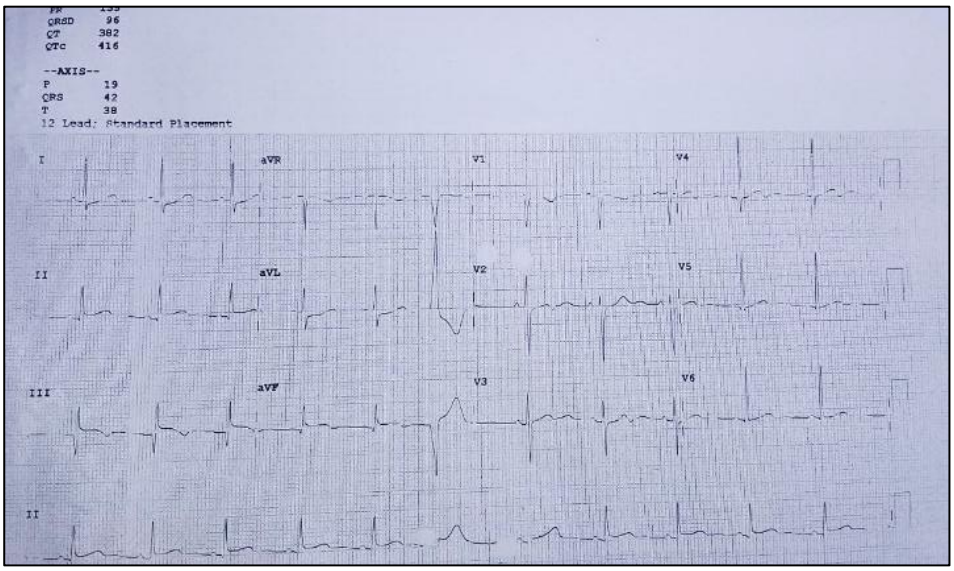


Figure 2: Twelve-lead electrocardiogram showing ST-segment elevation in leads II, III, and aVF, consistent with acute inferior wall ST-elevation myocardial infarction.

Table 1: Summary of Clinical Findings

Parameter	Finding
Age / Sex	22 years / Female
SLE duration	8 years
Initial SLE presentation	Malar rash
Immunology	ANA speckled (1:100), anti-dsDNA positive
Renal involvement	Lupus nephritis, proteinuria 532 mg/24 h
Medications	Prednisolone, MMF, Hydroxychloroquine
Presenting symptoms	Chest pain, nausea, vomiting, giddiness
ECG	ST elevation in II, III, aVF
Coronary angiography	Distal RCA total occlusion
Echocardiography	EF 45%, inferior wall hypokinesia

Management and Outcome

The patient was managed according to standard ST-elevation myocardial infarction protocol, including antithrombotic therapy and revascularization strategy as appropriate.

1) The Left Main Coronary Artery (LMCA) was cannulated with a JR 3.5 6F guiding catheter and diagnostic shoots taken in various views. Coronary angiography revealed total occlusion of the distal right coronary artery (RCA) (Figure 3), while the left coronary arteries were angiographically normal (Figure 4).



Figure 3: Left coronary angiography showing normal left main, left anterior descending, and left circumflex arteries without significant obstructive disease

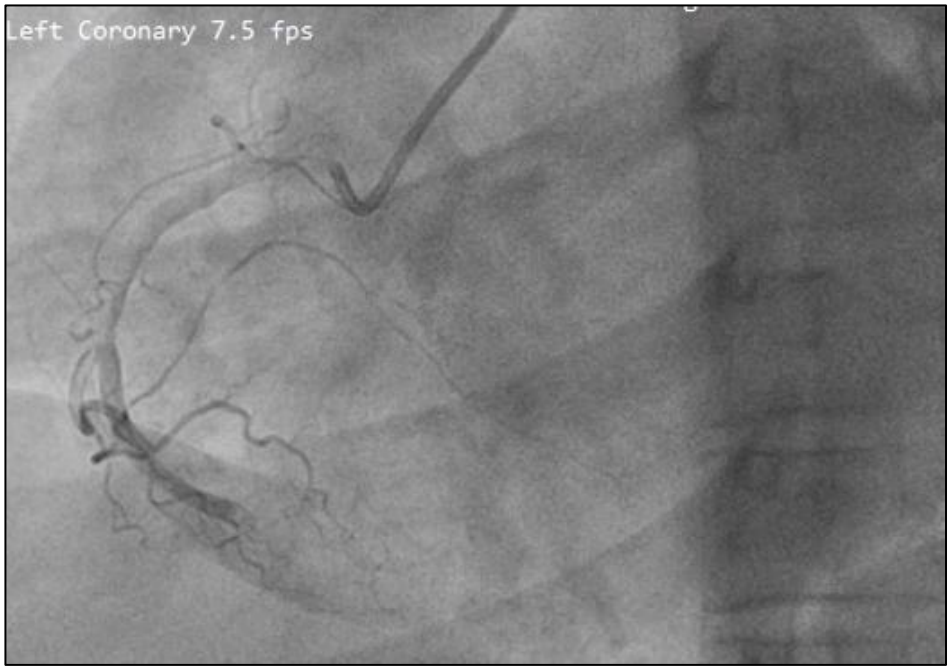


Figure 4: Right coronary angiography demonstrating total occlusion of the distal right coronary artery

2) The RCA lesion was crossed with Sion Blue wire. (Figure 5)



Figure 5: Primary Angioplasty in Myocardial Infarction to right coronary artery showing crossing of wire

3) Pre-dilatation was done with 2.0 x 15 mm Vector NC balloon at 6 atmosphere for 10 seconds each. (Figure 6)

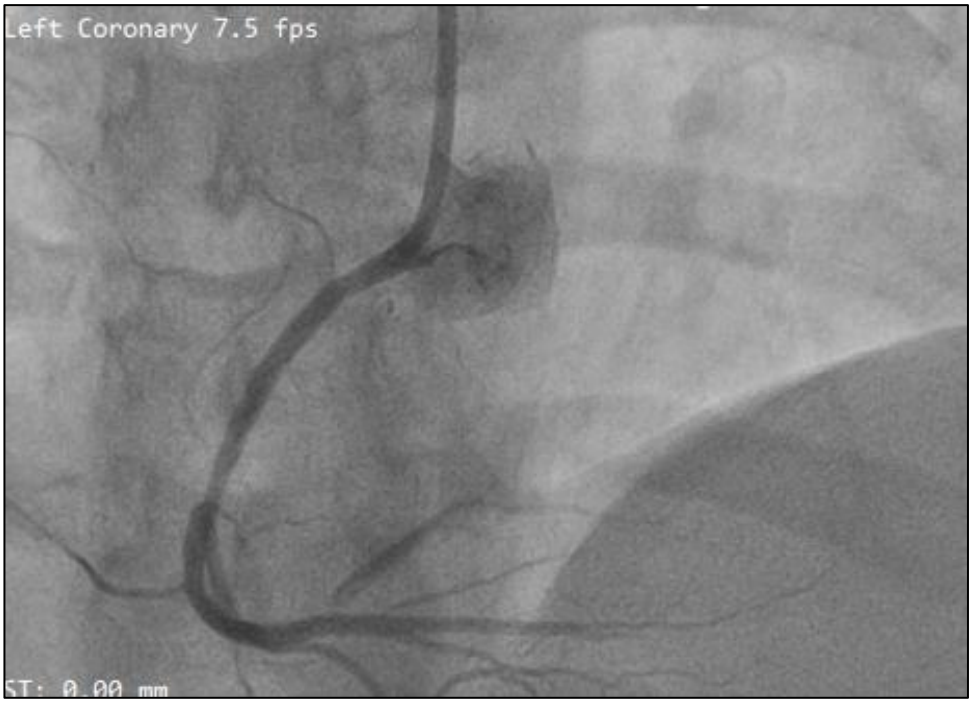


Figure 6: Coronary angioplasty view showing pre-dilatation of lesion

4) 2.5 x 23 mm Xience Xpereience Prime DES was deployed at 10 atm in for 10 seconds and check shoots repeated.

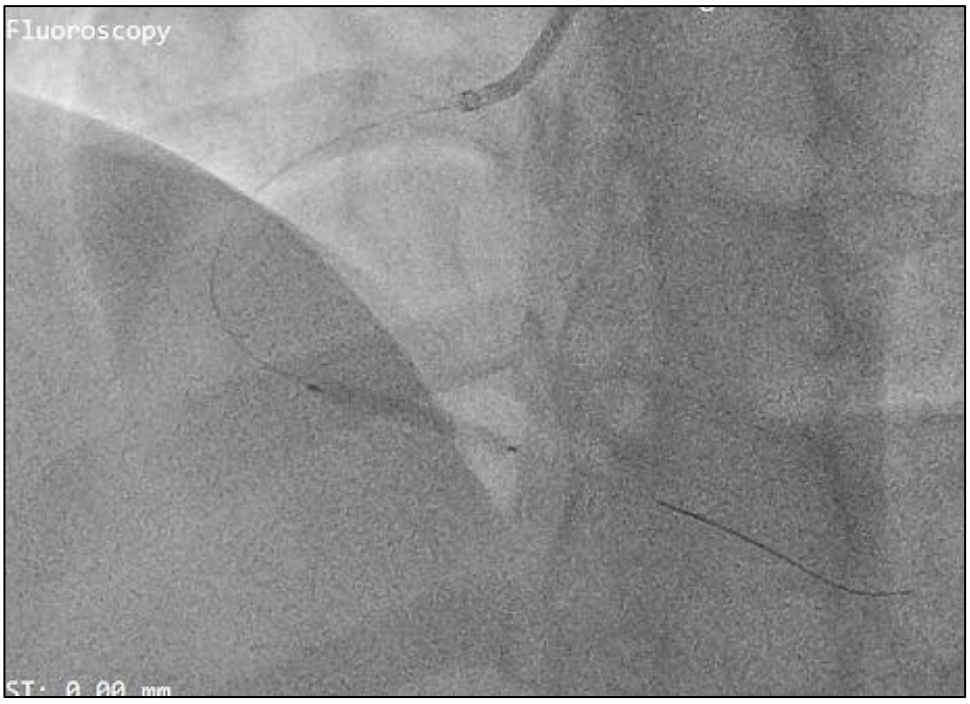


Figure 7: Coronary angioplasty view showing stent deployment

5) Post dilatation was done with 2.75 x 13 mm Raiden NC balloon at 12 atm for 10 seconds.
6) There was no dissection. An adequate deployed stent with distal TIMI III flow was seen.

Her immunosuppressive therapy was continued, and she showed clinical improvement with an uneventful hospital course. She was discharged with advice for close cardiology and rheumatology follow-up.

3. Discussion

Cardiovascular disease represents the predominant cause of late mortality in patients with systemic lupus erythematosus (SLE), with acute myocardial infarction (AMI) occurring substantially earlier than in the general population. Population-based and registry-derived studies have consistently demonstrated a two- to three-fold higher risk of myocardial infarction among individuals with SLE compared with age- and sex-matched controls, an excess risk that persists even after adjustment for conventional cardiovascular

risk factors.[12] This underscores the contribution of disease-specific mechanisms beyond traditional atherosclerotic pathways. The present case exemplifies this phenomenon, with a 22-year-old woman developing an acute inferior wall ST-elevation myocardial infarction eight years after the diagnosis of SLE and lupus nephritis.

Premature and Severe Coronary Disease in SLE

Evidence from nationwide registries and matched cohort studies suggests that myocardial infarction in SLE is most frequently related to **accelerated obstructive coronary artery disease**, rather than myocardial infarction with non-obstructive coronary arteries (MINOCA).[1] In a large Swedish cohort, patients with SLE demonstrated a significantly higher incidence of myocardial infarction with angiographically obstructive coronary lesions, supporting the concept that premature atherosclerosis is the dominant pathological substrate in this population.[1] This observation is concordant with our patient's coronary angiographic finding of total occlusion of the distal right coronary artery, rather than normal or minimally diseased epicardial vessels.

Notably, myocardial infarction in SLE occurs at a substantially younger age—often one to two decades earlier-than in individuals without autoimmune disease. Coronary angiographic studies have shown that the extent and severity of coronary artery disease in SLE patients presenting with AMI may resemble that seen in non-SLE patients nearly 20 years older.[2] This striking age disparity highlights the aggressive nature of vascular disease in SLE and is clearly illustrated by the occurrence of significant epicardial coronary occlusion in early adulthood in the present case.

Role of Chronic Inflammation and Immune Dysregulation

The mechanisms underlying coronary artery disease in SLE are complex and multifactorial. Persistent immune activation, circulating immune complex deposition, and chronic systemic inflammation contribute to endothelial dysfunction and promote early plaque formation. Dysregulation of both innate and adaptive immune responses enhances oxidative stress, modifies lipoprotein composition, and facilitates inflammatory cell infiltration within the arterial wall, resulting in plaques that are particularly unstable and prone to rupture.[2]

Several immune-mediated processes have been implicated in plaque destabilization and thrombosis in SLE, including enhanced formation of neutrophil extracellular traps, activation of plasmacytoid dendritic cells, sustained interferon-α signalling, and impaired regulatory T-cell function.[2] These abnormalities create a vascular environment that is simultaneously pro-atherogenic and pro-thrombotic, predisposing even young patients to acute coronary events. Importantly, this risk may be present despite the absence of traditional cardiovascular risk factors, emphasizing the central role of ongoing inflammatory activity in driving clinically significant coronary artery disease in SLE.

Impact of Lupus Nephritis and Disease Duration

Renal involvement is a well-recognized amplifier of cardiovascular risk in SLE. Lupus nephritis contributes to endothelial dysfunction, systemic inflammation, dyslipidemia, and the development of hypertension, all of which accelerate atherosclerotic processes. Large observational cohorts have consistently reported higher rates of cardiovascular events among SLE patients with renal disease compared with those without nephritis.[2]

Disease duration further modulates cardiovascular risk. The cumulative inflammatory burden over time, combined with prolonged exposure to corticosteroids and other immunosuppressive therapies, contributes to progressive vascular injury. In the present patient, an eight-year duration of SLE with lupus nephritis likely played a significant role in the early development of obstructive coronary artery disease, even though she was receiving relatively low-dose maintenance corticosteroid therapy at presentation.

Outcomes and Prognosis of AMI in SLE

Recent nationwide inpatient analyses have shown that patients with SLE hospitalized for acute myocardial infarction experience higher in-hospital mortality, longer lengths of stay, and increased healthcare resource utilization compared with non-SLE patients.[3] Identified predictors of adverse outcomes include female sex, advanced age, prolonged hospitalization, and greater comorbidity burden. These findings highlight the intrinsic vulnerability of SLE patients during acute coronary events.

Encouragingly, contemporary data suggest that when treated with guideline-directed medical therapy and appropriate revascularization strategies, outcomes following percutaneous coronary intervention in patients with SLE are comparable to those observed in non-SLE populations.[4] This supports the principle that invasive management should not be deferred solely on the basis of underlying autoimmune disease and reinforces the importance of early diagnosis and aggressive treatment of acute coronary syndromes in this population.

4. Clinical Implications

This case underscores several important clinical considerations. Acute coronary syndrome should be promptly suspected in young patients with SLE presenting with chest pain, irrespective of age or the presence of traditional cardiovascular risk factors. Coronary angiography remains crucial for defining the underlying mechanism of myocardial infarction and guiding definitive management. Long-term cardiovascular risk reduction in SLE requires not only modification of traditional risk factors but also sustained control of disease activity and minimization of cumulative corticosteroid exposure.

Hydroxychloroquine remains the only immunomodulatory agent with consistent evidence of cardiovascular benefit in SLE, exerting anti-thrombotic, lipid-modifying, and anti-inflammatory effects.[2] Close multidisciplinary collaboration between cardiology and rheumatology is therefore essential to optimize both short- and long-term outcomes in patients with SLE who develop coronary artery disease.

5. Conclusion

Acute myocardial infarction can occur in young patients with systemic lupus erythematosus and lupus nephritis, even in the absence of traditional cardiovascular risk factors. This case highlights the importance of early recognition, prompt coronary angiography, and integrated management strategies to improve cardiovascular outcomes in patients with SLE.

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