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CASE REPORT

PERIPHERAL ARTERIAL THROMBOSIS AS AN ATYPICAL INITIAL PRESENTATION OF SYSTEMIC LUPUS ERYTHEMATOSUS IN A YOUNG MALE: A CASE REPORT

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Abstract:

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease with heterogeneous clinical manifestations and a predilection for affecting women of childbearing age. Thrombotic events, particularly venous thrombosis, are well-documented complications of SLE, often associated with antiphospholipid syndrome (APS). However, arterial thrombosis, especially involving large peripheral vessels, is a relatively rare initial manifestation, and even more unusual in young males without traditional cardiovascular risk factors.We report the case of an 18-year-old previously healthy male who presented with acute peripheral arterial thrombosis of the superficial femoral artery. Comprehensive laboratory evaluation showed positive antinuclear antibody (ANA) and anti-double stranded DNA (anti-dsDNA) antibodies, with negative antiphospholipid antibodies and a normal thrombophilia panel. CT angiogram confirmed a long-segment thrombotic occlusion of the right superficial femoral artery. The diagnosis of SLE with vascular involvement was established in the absence of APS. The patient was successfully managed with immunosuppressive therapy and antiplatelet agents.This case highlights the diagnostic complexity of SLE in atypical presentations and emphasizes the importance of considering SLE in young patients with thrombotic events, even in the absence of classic risk factors.

Keywords: *Systemic Lupus Erythematosus, Young Male, Arterial Thrombosis, Rare Case Report*

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Introduction:

Systemic Lupus Erythematosus (SLE) is a multisystem autoimmune disorder characterized by the production of autoantibodies and immune complex deposition, leading to inflammation and tissue damage[1]. The disease predominantly affects women, with a female-to-male ratio of approximately 9:1, and commonly presents between the ages of 15 and 45 years[2]. Clinical manifestations vary widely, ranging from mild cutaneous lesions to severe organ involvement including lupus nephritis, central nervous system disease, and hematological abnormalities[3].

Vascular complications are a significant cause of morbidity and mortality in SLE patients. These include accelerated atherosclerosis, vasculitis, and thrombotic events. Venous thrombosis, especially deep vein thrombosis and pulmonary embolism, are well documented and are often associated with the presence of antiphospholipid antibodies, collectively known as antiphospholipid syndrome (APS)[4,5]. Arterial

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thrombosis, particularly involving large peripheral vessels, is much less common and typically occurs in the setting of APS or traditional cardiovascular risk factors such as hypertension, diabetes, or smoking[6].

Arterial thrombosis as an initial presentation of SLE, especially in young males without conventional risk factors or APS, remains exceedingly rare and poses a diagnostic challenge. Here, we report such a case with detailed clinical, laboratory, and imaging findings, emphasizing the importance of considering SLE in atypical vascular presentations.

Case report:

An 18-year-old male, with no significant past medical or family history, presented to our clinic with a 2-month history of pain, numbness, and progressive weakness in his right lower limb. He described the limb as cold and noted difficulty in walking. There was no history of trauma, fever, rash, joint pain, oral ulcers, photosensitivity, or weight loss. He denied smoking, alcohol consumption, or use of illicit substances. There was no history of chest pain or palpitation.

Vital signs were within normal limits. Examination of the right lower limb revealed absent dorsalis pedis, anterior tibial, and popliteal artery pulsations. The limb was cooler compared to the left side with mild muscle wasting noted in the calf region. Sensory and motor examination revealed decreased power (4/5) in the ankle dorsiflexors and plantarflexors. The left lower limb exhibited normal peripheral pulses and no wasting. Initial laboratory investigations were largely unremarkable (Table 1).The complete blood count was within normal limits. Renal function tests, liver function tests, and electrolyte levels were all normal. Urinalysis revealed no proteinuria or hematuria.The coagulation profile, including prothrombin time (PT), activated partial thromboplastin time (aPTT), and international normalized ratio (INR), was within normal range.

Immunological testing showed a positive antinuclear antibody (ANA). Anti-double stranded DNA (anti-dsDNA) antibodies were also positive, with levels exceeding 50 IU/mL. Antiphospholipid antibodies were negative, including lupus anticoagulant, anticardiolipin IgG and IgM, and anti-â2 glycoprotein I antibodies.

Tests for rheumatoid factor (RF), perinuclear ANCA (p-ANCA), and cytoplasmic ANCA (c-ANCA) were negative.The complement levels showed a normal C4 level of 0.12 g/L (normal 0.10-0.40 g/L) but a low C3 level of 0.68 g/L (normal 0.90-1.80 g/L). D-dimer was elevated 1.98 µg/mL (normal <0.50 µg/mL)

A thrombophilia screen, including Protein C, Protein S, Antithrombin III, homocysteine levels, and testing for the Factor V Leiden mutation, showed all parameters within normal limits.

Table I

Laboratory results

Investigation	Result	Normal Range
WBC count	7.46 × 10^9/L	4-10 x 10^9/L
Platelet	210 K/µL	150-410 K/µL
Haemoglobin	13.50 g/dL	13-17 g/dL
ESR	32 mm in 1st hour	0-10 mm in 1st hour
CRP	21 mg/L	<5.00 mg/L
Procalcitonin	0.02 ng/mL	<0.05 ng/mL
ALT	31 U/L	Upto 41 U/L (Male)Upto 33 U/L (Female)
S. Creatinine	1.08 mg/dL	0.70-1.20 mg/dL
Urea	32.90 mg/dL	16.60-48.50 mg/dL
Serum Albumin	4.11 g/dL	3.50-5.20 g/dL
Calcium	8.78 mg/dL	8.80-10.60 mg/dL
p-ANCA	<2.50 AU/mL	Nonreactive: <16AU/mlReactive: >24 AU/ml
c-ANCA	<2.00 AU/mL	Nonreactive: <16AU/mlReactive: >24 AU/ml
Anti-phospholipid IgG	2.35 U/mL	<10: Negative>10: Positive
Anti-phospholipid IgM	2.88 U/mL	<10: Negative>10: Positive
Anti Cardiolipin IgG	2.42 GPLU/mL	Nonreactive: <8GPLU/mLReactive: >12 GPLU/mL
Anti Cardiolipin IgM	2.73 GPLU/mL	Nonreactive: <8 GPLU/mLReactive: >12 GPLU/mL

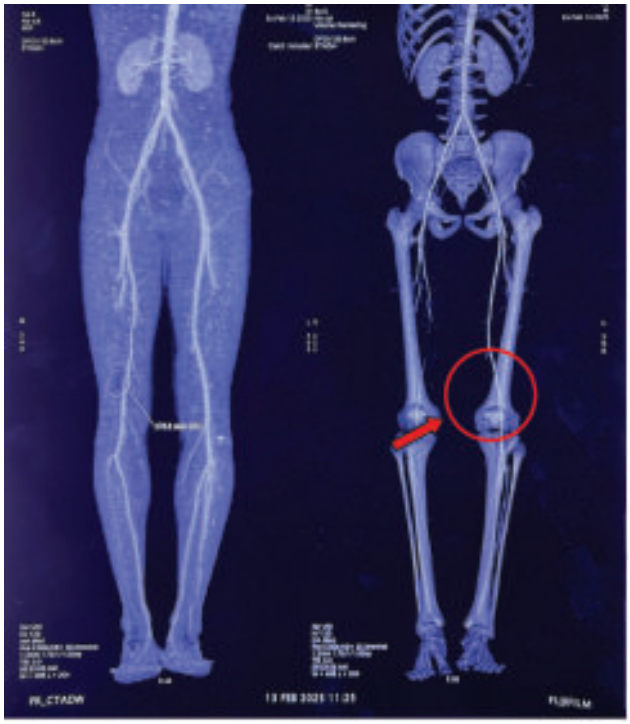

Figure 1: *CT angiogram of abdominal aorta and both lower limbs revealed thrombotic occlusion of the right superficial femoral artery (Indicated by circle & arrow)*

CT angiography of the abdominal aorta and lower limbs was done (Figure 1). It showed a thrombotic occlusion of the right superficial femoral artery extending for approximately 17.6 cm. There was reconstitution of moderate blood flow distally in the lower superficial femoral, popliteal, and posterior tibial arteries. Narrowing with thin flow was noted in the right anterior tibial and peroneal arteries. Echocardiogram and electrocardiogram were normal, ruling out cardiac embolic sources.

Based on the clinical presentation of acute limb ischemia, serological positivity for ANA and anti-dsDNA, negative antiphospholipid antibody panel, and exclusion of inherited thrombophilia, a diagnosis of SLE with large vessel arterial thrombosis was established.Since this case receives two (2) points for fever, three (3) points for low C3, and six (6) points for a positive anti-dsDNA antibody, it satisfies the 2019 EULAR/ACR criteria for diagnosing SLE.So, we have classified this case as SLE with a score of 11 and it also fulfills the entry criterion which is positive ANA.

The patient was initiated on high-dose corticosteroids (oral prednisone 1 mg/kg/day) alongside hydroxychloroquine (400 mg daily) to control underlying autoimmune activity. Given the absence of antiphospholipid antibodies and stable distal limb perfusion, antiplatelet therapy (aspirin 75 mg daily) was started without full anticoagulation. The vascular surgery team advised conservative management as the limb was viable and surgical thrombectomy carried high risks.

The patient was monitored closely for symptom progression and potential development of further SLE manifestations.

At 3 months, the patient reported improvement in pain and functional use of the affected limb. On physical examination, peripheral pulses remained diminished but there was no progression of ischemia or tissue loss. Repeat imaging demonstrated partial recanalization of the occluded segment.

At 6 months, the patient remained clinically stable without new SLE symptoms or thrombotic events. The pain subsided and functional capacity is improved. On physical examination, popliteal pulsation was found to be sluggish. There was no progression of ischemia or tissue loss and corticosteroids were gradually tapered. He continues hydroxychloroquine therapy and is under regular rheumatology follow-up.

Discussion:

Vascular complications in SLE are predominantly due to accelerated atherosclerosis and antiphospholipid syndrome (APS)-related thrombosis[7]. However, the occurrence of large-vessel arterial thrombosis in the absence of APS remains uncommon and poorly understood. The pathogenesis likely involves multiple mechanisms such as immune complex–mediated endothelial injury, complement activation, inflammatory cytokine release, and vasculitis, which together promote a prothrombotic state[8,11].

This case is unique given the patient's young age, male gender, and lack of traditional cardiovascular risk factors or APS antibodies, highlighting the heterogeneous and sometimes atypical presentations of SLE. Arterial thrombosis without APS in SLE patients is often under-recognized and can delay diagnosis and treatment[9]. Recent studies have emphasized the role of neutrophil extracellular traps (NETs) in SLE-associated vascular damage, further supporting immune-mediated endothelial dysfunction as a key mechanism for thrombosis[12].

Clinicians should maintain a high index of suspicion for autoimmune etiologies in young patients presenting with unexplained arterial occlusion, particularly when conventional risk factors are absent. A comprehensive autoimmune panel, including ANA and anti-dsDNA, is crucial for early diagnosis and appropriate management.

Therapeutic strategies focus on immunosuppression to control lupus activity and prevent further vascular injury. Hydroxychloroquine has been shown to have antithrombotic properties and reduce SLE flare rates, while corticosteroids help control acute inflammation. Antithrombotic therapy should be individualized; anticoagulation is recommended in patients with APS, but in APS-negative patients, antiplatelet therapy may suffice[10]. Surgical or endovascular interventions are reserved for critical ischemia or limb-threatening cases.

Early recognition and prompt immunosuppressive treatment are essential to minimize ischemic complications and improve long-term outcomes. Our case underscores the importance of considering SLE as a differential diagnosis for arterial thrombosis even in atypical demographics and presentations.

Conclusion:

This case exemplifies an atypical presentation of SLE in a young male patient manifesting as peripheral arterial thrombosis without antiphospholipid syndrome. Awareness of such rare presentations is critical for timely diagnosis and management.

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Consent for publication:

Informed written consent was taken from the parents of the patient to publish details relevant to the disease and management.

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Conflict of interests: None

Authors' contributions:

All authors were involved in the management of the patient and all authors contributed to the conception, writing, and editing of the case report.

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