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Renal Vein Thrombosis as an Initial Presentation for Systemic Lupus Erythematosus in a 32-Year-Old Sudanese Male: A Case Report in Palestine

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Abstract

Systemic lupus erythematosus (SLE) is a complex autoimmune disease known for its diverse clinical presentations, and one severe complication is lupus nephritis (LN), which significantly contributes to morbidity and mortality. While LN often presents within the first 5 years of SLE diagnosis, renal vein thrombosis (RVT) is a rare vascular complication with a high risk of mortality and morbidity. This case report discusses the rare occurrence of RVT as the initial presentation of SLE in a 32-year-old Sudanese male patient, currently working in Palestine, presenting with flank pain, hematuria, fever, and lower limb edema. The case details the patient's symptoms, examination findings, and extensive laboratory and imaging workup leading to the diagnosis. This report highlights the rare association between RVT and SLE, emphasizing the importance of maintaining a high index of suspicion for SLE in patients with multisystem involvement, especially in males, where the diagnosis may be overlooked due to its lower prevalence. Early recognition can improve patient outcomes and reduce the risk of complications. Further research is needed to better understand the connection between RVT and SLE and to develop more effective treatment strategies.

Keywords

SLE, male, RVT

Introduction

Renal vein thrombosis (RVT) is an exceedingly rare complication of systemic lupus erythematosus (SLE), occurring in only 2% of cases.[1] This report presents a unique case of RVT as the initial manifestation of SLE in a male patient, highlighting the diverse and unpredictable ways this autoimmune disease can present.

Systemic lupus erythematosus is an autoimmune disease affecting multiple systems.[2] Its prevalence is notably higher in females, approximately 10 times more than in males.[3] The disease manifests in various phenotypes, exhibiting diverse clinical presentations ranging from mild mucocutaneous manifestations to severe involvement of multiple organs which can be difficult to identify.[4] Therefore, SLE can manifest with severe symptoms like lupus nephritis (LN), a significant cause of morbidity and mortality.[5]

Notably, renal manifestations can be the initial presentation of the disease,[2,6] often appearing within the first year of diagnosis or typically within the initial 5 years.[7] This is particularly concerning for certain ethnicities with a higher prevalence of SLE, such as African American, Asian, Arab, Hispanic, and Mestizo, where up to 50% of patients develop nephritis.[7]

Lupus nephritis can manifest as nephritic and/or nephrotic syndrome, presenting with symptoms like edema, proteinuria, hematuria, impaired renal function, abnormal lipid profile, hypertension, and thrombosis. Patients with SLE are at high risk of thrombosis, with prevalence exceeding 10% and rising to over 50% in high-risk cases.[8] Thrombosis is a major cause of mortality in SLE, accounting for 26.5% of deaths.[9]

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Contributing factors include antiphospholipid antibodies (APLAs), inflammation, genetic mutations, and LN.[10] Renal vein thrombosis occurs in 7% of nephrotic syndrome cases, often triggered by a hypercoagulable state.[11]

Lupus-associated nephrotic syndrome (stage V) accounts for 20% of LN cases,[12] potentially causing a severe hypercoagulable state leading to thrombosis.[11] Renal vein thrombosis is an infrequent occurrence in SLE, constituting only 2% of renovascular complications associated with LN.[1]

This case report aims to contribute to the understanding of RVT as an initial manifestation of SLE in males and discuss the potential mechanisms involved.

Case Presentation

A 32-year-old male with no significant medical history presented with a gradual onset of constant, bilateral stabbing flank pain for 3 weeks, partially relieved by paracetamol. The pain was not associated with food, medication, nausea, vomiting, or urinary symptoms, except for frothy urine. This was associated with a documented fever reaching 38.8 °C, and was treated for a suspected urinary tract infection with cefuroxime, but without improvement. He later experienced worsening bilateral pleuritic chest pain radiating to the scapulae, along with shortness of breath.

Subsequently, the patient complained of bilateral lower limb swelling extending to the thigh, in addition to right upper arm swelling, which led him to seek medical advice from our hospital. The patient had not been taking any medications, and there was no documented history of deep vein thrombosis (DVT) or systemic disease.

Upon arrival, the patient's blood pressure was 160/102 mmHg, heart rate at 96 bpm, temperature at 37.2 °C, and oxygen saturation at 97% in room air. Percussion of both lung bases showed dullness and reduced air entry. In addition, there was diffuse lymphadenopathy. No organomegaly or other palpable masses were detected. There was no tenderness in the costovertebral angle on both sides. Bilateral +2 pitting edema was observed in the lower limbs and tender erythematous cord-like swelling in the lateral aspect of the right arm. Examinations of all other systems were within normal limits.

In his laboratory results, the Complete Blood Count (CBC) showed an Hb 11.4, Mean Coruscular Volume (MCV) 87, and Red Cell Distribution Wedth (RDW) 11.4. Iron studies showed Fe 19.5, ferritin 701.9, and Total Iron Binding Capacity (TIBC) 56. In addition, the C-Reactive Protein (CRP) was elevated at 123.8, and the Erythrocyte Sedimentation Rate (ESR) was 114. The urinalysis showed +4 proteins, +2 blood, and an Red Blood Cell (RBC) count from 18 to 20, accompanied by dysmorphic cells and granular casts. Concurrently, the serum albumin level was low at 1.8. The lipid profile showed elevated levels of cholesterol 271.6, triglycerides 145.4, and low-density lipoprotein 180. Adding to the elevated D-dimer level to 12.88 and fibrinogen level to 787, nephrotic syndrome was suspected due to the elevated protein-to-creatinine ratio, along with a Blood Urea Nitogen (BUN) of 24.4 and a creatinine level of 1, confirming the diagnosis.

The combination of flank pain, hematuria, proteinuria, and elevated D-dimer and fibrinogen levels raises the suspicion of RVT. An abdominal and pelvic computed tomography (CT) with intravenous (IV) contrast was done, which revealed a filling defect in the left main renal vein and the upper half of its branch which confirmed the RVT (Figure 1).

Because of suspecting hypercoagulable status, bilateral lower limbs venous duplex ultrasound and spiral chest CT were performed, ruling out venous thromboembolism (VTE). Chest X-ray revealed a bilateral pleural effusion (Figure 2), and pleural analysis showed exudative effusion (Table 1). Also, abdominal Doppler ultrasound indicated normal kidneys. Upper limb Doppler ultrasounds showed a thrombosed superficial vein in the right arm with no DVT along with bilateral enlarged lymph nodes. A left axillary lymph node excisional biopsy revealed a negative result for malignancy, ruling out lymphoma. Echocardiography showed normal results, ruling out infective endocarditis as a potential cause for glomerulonephritis.

Furthermore, due to the presence of a hypercoagulable state, pleurisy, elevated ESR, and nephrotic syndrome in this young male, SLE was suspected, and further investigations were done to confirm this diagnosis. He had 5 of 11 American Rheumatism Association criteria for SLE, so he was diagnosed as SLE. Also, according to the American College of Rheumatology (ACR)/European League Against Rheumatism (EULAR) classification, the patient demonstrated a positive antinuclear antibody and scored 15 points on the scale (Table 2).

The patient's in-hospital treatment included ongoing enoxaparin. His discharge medication regimen addressed both his SLE and his elevated risk of thrombosis. Hence, he was prescribed a combination of hydroxychloroquine and prednisone for long-term SLE management, alongside a 3-month course of apixaban or until his serum albumin level exceeded 2.5.

Discussion

Systemic lupus erythematosus is a chronic, multisystemic autoimmune disease distinguished by an unknown cause, where a multifactorial etiology, marked by a genetic, hormonal, and environmental predisposition appears to contribute to a breakdown of immunological tolerance.[2] One of the striking features of SLE is its overwhelming preference for females. Women are diagnosed with SLE at a rate of 10 to 1 compared with men,[3] suggesting a strong link to female sex hormones. Researchers hypothesize that heightened autoimmune reactivity and the immunological disturbances that drive SLE may be influenced by estrogen and other female hormones.[13]

The diagnostic challenge in SLE arises from its broad spectrum of symptoms, ranging from mild joint and skin manifestations to severe complications affecting vital organs such as the kidneys, hematologic system, or central nervous system. Nevertheless, the primary initial manifestations typically encompass arthritis, rash, and pyrexia.[9]

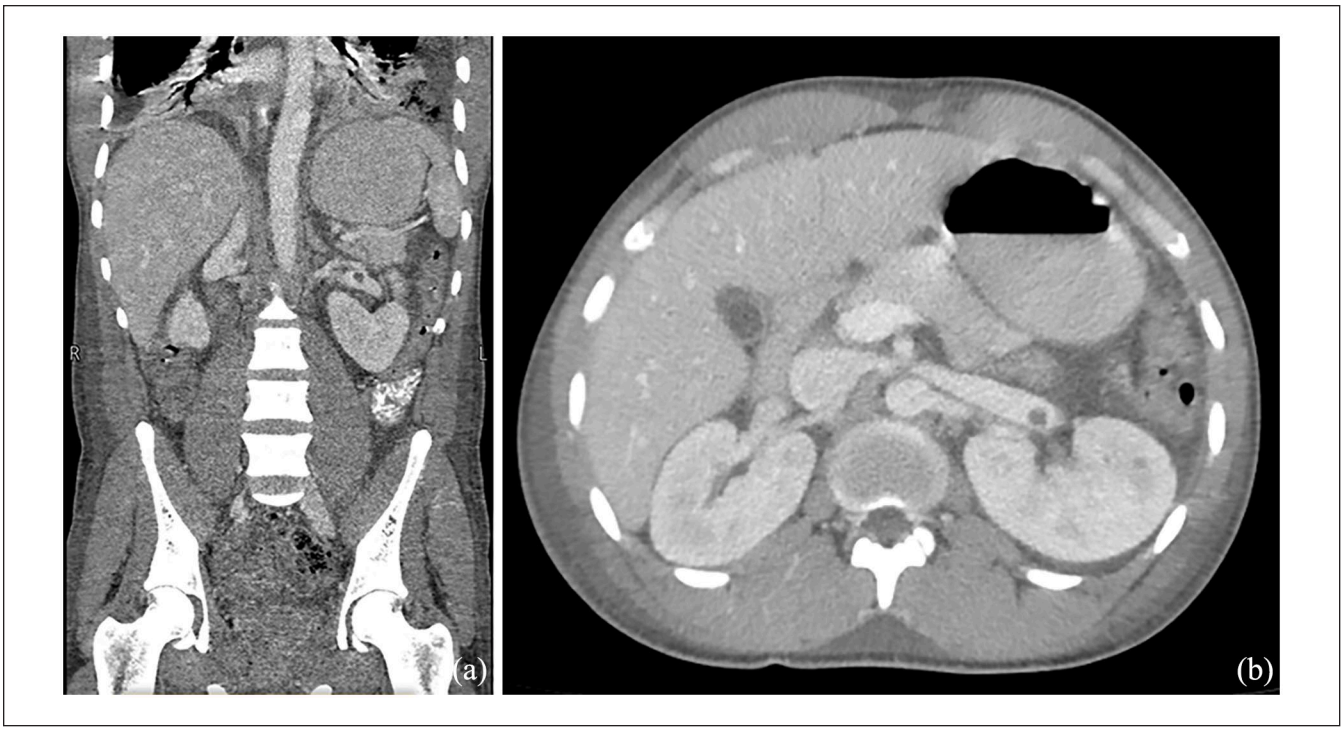

Figure 1. (a) Coronal view: An abdominal and pelvic computed tomography with intravenous contrast reveals a filling defect in the left main renal vein and the upper half of its branch. (b) Axial view: An abdominal and pelvic computed tomography with intravenous contrast reveals a filling defect in the left main renal vein and the upper half of its branch.

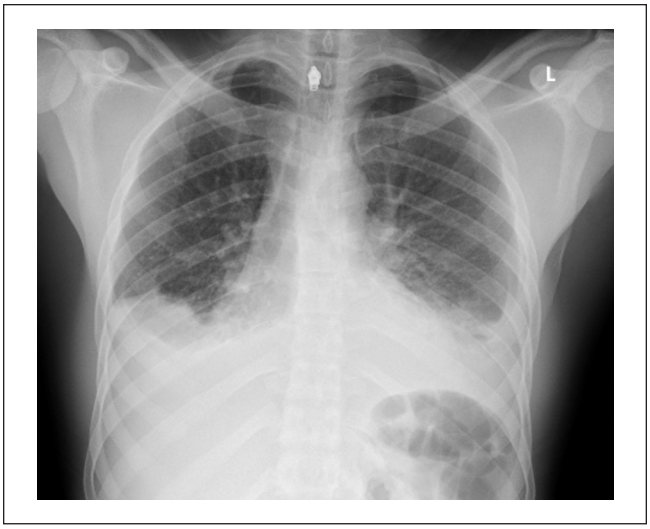

Figure 2. PA view of chest X-ray revealing a bilateral pleural effusion.

Systemic lupus erythematosus presents with a wide range of manifestations, with certain aspects being more prevalent than others. Constitutional symptoms, the most common of which include weight loss, fatigue, and low-grade fever, often occur with arthralgias or arthritis, though joint deformities are less common.[14] Cutaneous features affect 75% to 80% of patients, ranging from rash and photosensitivity to ulcerations.[15] Renal involvement, a serious feature, affects up to 50% of patients,[16] while respiratory manifestations, especially pleuritis, are present in 30% to 50% of cases.[17] Hematologic manifestations, including cytopenia, are common, with lymphopenia indicating high disease activity.[18] Neuropsychiatric symptoms, ocular manifestations, gastrointestinal issues, and immunocompromised risks are also noted.[2,19]

Findings from different studies suggested that in men, disease onset typically occurs before age 50, marked by acute inflammation. In contrast, women often experience manifestations during their reproductive years, with milder symptoms, followed by remission until chronic complications arise later in life.[20] Moreover, studies suggest that SLE in men follows a more complex clinical course, with a higher occurrence of renal impairment, central nervous system involvement, and vascular diseases compared with women. In addition, there is a 1-year higher mortality due to these complications. Men are more prone to immune-mediated anemia and elevated APLA levels, which may lead to thrombogenesis. Serositis is a cardinal symptom of SLE and occurs more frequently in male patients.[21-24] Our patient complained of both pleuritis and thrombophlebitis as early manifestations of this disease.

The risk of VTE in inflammatory rheumatic diseases is notably elevated, as evidenced by a cumulative incidence of 7.29% in SLE patients. Existing studies on SLE predominantly

Table 1. Pleural Tap Analysis.

Proteins	2.3
Lactate Dehydrogenase (LDH)	219
Glucose	123.5

Table 2. Rheumatologic Workup.

ANA	Positive
Anti-dsDNA	25.8
Direct Coombs	Positive
Indirect Coombs	Positive
RF	Negative
SS-A	>100
SS-B	<3
ANCA-PR3	<3
ANCA-MPO	<3
C3 (70-176)	135
C4 (20-50)	30
Cardiolipin IgG (<12)	3.9
Cardiolipin IgM (<12)	7.7

Abbreviation: ANA, antinuclear antibody.

emphasize heightened risks linked to positive antiphospholipid status, rather than delving into the inherent hypercoagulable nature intrinsic to this inflammatory condition.[25,26] Approximately, 50% of SLE patients exhibit the presence of APLA, as documented in the literature.[27,28] Yet our patient lacks a history of arteriovenous thrombosis and tests negative for anticardiolipin antibodies (ACLs), as additional tests are unavailable in our facility.

Significantly, these antibodies have been consistently established as a substantial and independent risk factor for thrombotic events.[20] Limited investigations have explored the prevalence of RVT in SLE.[1,29-31] Further investigations have been conducted to identify the risk factors influencing the development of RVT in SLE. These inquiries have specifically examined associations with conditions such as nephrotic syndrome, membranous glomerulonephritis, hypercoagulable states, the presence of ACL, and occurrences of thrombophlebitis, as influencing factors and integral components of the pathogenesis.[32-38] Due to the presence of nephrotic-range proteinuria, over 90% dysmorphism in RBCs, low albumin levels, ESR, positive anti–double-stranded DNA, and hyperlipidemia, we attribute our patient's hypercoagulability status to LN.

Renal vein thrombosis by itself represents a rare clinical occurrence, manifesting either acutely or insidiously and potentially culminating in acute kidney injury or chronic kidney disease. This condition is frequently linked with nephrotic syndrome, primary hypercoagulability disorders, malignant renal tumors, extrinsic compression, infections, trauma, or a complication following renal transplant. The consequential severe passive congestion induces renal swelling and engorgement, resulting in nephron degeneration and the onset of symptoms such as flank pain, hematuria, and diminished urine output,[3,5] as observed in our patient experiencing these manifestations.

Despite the infrequent occurrence of RVT, its rarity in SLE makes its precise incidence challenging to determine. A particular study reported an approximate RVT incidence of nearly 2% within the kidneys of a cohort of 100 patients diagnosed with SLE.[1] Interestingly, the research findings imply that RVT tends to manifest more commonly during the therapeutic course of SLE patients[30,37] rather than at the initial presentation as observed in our unique case.

Gilsanz et al conducted angiography on 20 SLE patients to investigate RVT.[39] Renal vein thrombosis was identified in 2 out of 6 patients with nephrotic syndrome but only in 1 out of 14 without it. Nephrotic syndrome is a distinct and prevalent risk factor for RVT in SLE due to its role in inducing a hypercoagulable state.[30] Another study found that patients with peripheral thrombophlebitis had a higher RVT risk (61.5%) compared with those with nephrotic syndrome (27%).[40] Our patient exhibited both peripheral thrombophlebitis in his right hand and nephrotic syndrome, possibly leading to RVT.

The foundation of diagnosis relies heavily on imaging. In early acute RVT, about 90% of patients show radiological signs, with the affected kidney appearing enlarged and hyperechogenic.[41] Ultrasound of our patient revealed normal-sized kidneys with mild bilateral increased echogenicity. Computed tomography angiography is a non-invasive, highly accurate method for visualizing renal veins. The administration of contrast enhances renal vein visualization, with nearly 100% sensitivity and specificity. Our patient's CT scan with IV contrast (venous phase) showed a filling defect in the left main renal vein and the upper half of its branch, suggesting thrombosis.

The management of SLE with RVT mirrors that of RVT in other conditions. Initial treatment involves heparin, transitioning to warfarin for long-term anticoagulation with a target International Normalized Ratio (INR) of 2 or 3. While direct oral anticoagulants are promising, they lack sufficient data for nephrotic patients, so they aren't recommended. Suprarenal Inferior Vena Cava (IVC) filters can be lifesaving for those with RVT and pulmonary Embolism (PE) who can't tolerate anticoagulation. Anticoagulation typically lasts 6 to 12 months, but it may be extended with persistent nephrotic syndrome. Thrombolysis, due to high bleeding risk, and local thrombolytic therapy, due to renal failure risk, are generally avoided. Surgical thrombectomy is a last resort.[18,42]

Conclusion

In conclusion, this case emphasizes the importance of recognizing atypical SLE presentations, particularly in assessing renal complications like RVT. Renal vein thrombosis as an initial manifestation of SLE is rare, highlighting the need for thorough diagnostics. Early identification is key, stressing interdisciplinary collaboration among rheumatologists, nephrologists, and imaging specialists. Further research is needed to understand the link between SLE and RVT for

enhanced diagnostic strategies. Systemic patients should always be screened for SLE, preventing delays in management, especially in males prone to atypical manifestations. This underscores the pivotal role of early diagnosis in improving prognosis for cases with complications.

Authors' Contributions

A.A., A.I., and L.A. contributed to writing the first draft.
A. A., A.H., and A.D. contributed in data collection and writing the final manuscript.
A. S. supervised the project.
All authors contributed to the article and approved the submitted version.

Declaration of Conflicting Interests

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Ethics Approval

The study is exempt from ethical approval in our institution in Al-Makassed Hospital.

Informed Consent

Written informed consent was obtained from the patient for the publication of this case report and accompanying images. The patient is a legally competent person to do so in accordance with the applicable law. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Grantor

Aya Abu Lehia is the guarantor of this case report, taking full responsibility for the integrity of the manuscript and ensuring that all questions related to its accuracy or completeness are addressed.

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