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WHEN LUPUS STRIKES THE VESSELS: CONCURRENT CEREBRAL AND PULMONARY VASCULITIS AS THE INITIAL MANIFESTATION OF SYSTEMIC LUPUS ERYTHEMATOSUS

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ABSTRACT

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease with protean clinical manifestations. Vasculitis represents a severe and potentially life-threatening complication, especially when involving critical organs such as the central nervous system and lungs. We report a rare case of a 41-year-old male who presented with acute neuropsychiatric manifestations, mucocutaneous bleeding, tremulousness, and pulmonary involvement, later diagnosed as SLE-associated small-vessel vasculitis with secondary antiphospholipid syndrome (APS).The patient presented with bleeding gums, altered behavior, tremors, unsteady gait, and new-onset seizures. Examination revealed oral ulcers, erythematous papules, cognitive impairment, and generalized weakness. MRI brain demonstrated bilateral multiple small infarcts suggestive of vasculitis, while CT chest showed centrilobular nodules with ground-glass opacities. Autoimmune work-up revealed ANA positivity (3+) and elevated anti-β2 glycoprotein I IgG, confirming SLE with APS. The patient showed dramatic clinical and radiological improvement following immunosuppression with corticosteroids and anticoagulation.This case underscores the importance of considering autoimmune vasculitis in patients with multisystem involvement and highlights the aggressive nature of SLE-related vasculitis in males.

KEYWORDS: Systemic lupus erythematosus • *Vasculitis* • *Neuropsychiatric lupus* • *Antiphospholipid syndrome* • *CNS infarcts* • *Pulmonary vasculitis.*

BACKGROUND

Systemic lupus erythematosus (SLE) is a chronic multisystem autoimmune disease characterized by loss of immunological tolerance to nuclear antigens, leading to immune-complex formation, complement activation, and inflammatory tissue injury, resulting in protean clinical manifestations due to widespread vascular and organ involvement.[1,4] The global prevalence of SLE ranges from 20 to 150 per 100,000 population, with marked geographic and ethnic variation, and although the disease predominantly affects women of reproductive age with a female-to-male ratio of approximately 9:1, male patients tend to present with more severe disease, higher activity indices, and increased frequency of major organ involvement, including vasculitis, neuropsychiatric manifestations, and antiphospholipid syndrome.[1,4]

Vasculitis is a recognized manifestation of active SLE and occurs in approximately 10–20% of patients, most commonly affecting small cutaneous vessels, while visceral vasculitis involving vital organs such as the central nervous system and lungs is rare, occurring in less than 5% of cases, but is associated with significant morbidity and mortality.[1,4] The pathogenesis of SLE-associated vasculitis is predominantly immune-complex mediated, resulting in endothelial injury, fibrinoid necrosis, luminal narrowing, and a prothrombotic milieu, leading to tissue ischemia, hemorrhage, or infarction, particularly during periods of high disease activity.[1,4] Neuropsychiatric involvement in SLE encompasses a wide spectrum of manifestations, and central nervous system disease may result from immune-mediated vasculitis, antiphospholipid antibody-related thrombosis,

or direct neuronal injury mediated by inflammatory cytokines and autoantibodies; clinical presentations include cognitive dysfunction, seizures, acute confusional states, psychosis, and ischemic stroke, with MRI typically demonstrating multifocal cortical or subcortical infarcts suggestive of small-vessel involvement.[2,4] Pulmonary involvement is common in SLE; however, pulmonary vasculitis is uncommon and potentially life-threatening, arising from immune-mediated injury to the pulmonary microvasculature and manifesting radiologically as ground-glass opacities, centrilobular nodules, or diffuse infiltrates that often mimic infection or pulmonary edema, leading to diagnostic delay.[3,4] The coexistence of antiphospholipid antibodies, particularly anti-β2 glycoprotein I IgG, further amplifies vascular injury by promoting endothelial activation, platelet aggregation, and thrombosis, thereby increasing the risk of both cerebrovascular and pulmonary vascular events.[1,4] The simultaneous involvement of the central nervous system and pulmonary vasculature therefore represents a rare but severe disease phenotype in SLE, posing significant diagnostic challenges due to overlap with infectious and thrombotic disorders, and underscores the importance of early recognition and prompt immunosuppressive therapy to prevent irreversible organ damage and improve clinical outcomes.

CASE PRESENTATION

A 41-year-old male daily-wage worker presented to the emergency department with complaints of bleeding gums for one day, altered behavior for one week, and generalized tremulousness for four days associated with unsteady gait. During evaluation, he developed new-onset generalized tonic–clonic seizures. There was no history of fever, headache, vomiting, neck stiffness, trauma, loss of consciousness prior to seizures, substance abuse, recent vaccination, or prior neurological illness. He reported progressive loss of weight and appetite over the preceding few months, with no past history of hypertension, diabetes mellitus, tuberculosis, chronic kidney disease, thyroid disorder, or prior hospitalizations. On examination, the patient was conscious and cooperative but inattentive, poorly built and poorly nourished, with stable vital signs (blood pressure 110/80 mmHg, pulse 88/min, respiratory rate 18/min, SpO_2 96% on room air). Higher mental function assessment revealed impaired attention and calculation with a Mini–Mental State Examination score of 23/30. Oral ulcers with scabbing over the lips and multiple erythematous papules over the face and upper trunk (Figure 1) were noted, along with generalized tremulousness. Neurological examination showed no cranial nerve deficits; motor examination revealed generalized weakness with power graded 4/5 in all four limbs, normal tone, and preserved deep tendon reflexes, with no sensory, cerebellar, extrapyramidal, or meningeal signs. Cardiovascular, respiratory, and abdominal examinations were unremarkable. On the third day of hospitalization, the patient developed generalized tonic–clonic seizures followed by involuntary micturition and was treated with antiepileptic drugs and catheterization, after which no further seizures occurred. Hematological investigations revealed anemia and thrombocytopenia, with hemoglobin 9.8 g/dL (normal 13–17 g/dL), total leukocyte count 14,200 cells/mm³ (normal 4,000–11,000 cells/mm³) with neutrophilic predominance and left shift, and platelet count 22,500 cells/mm³ (normal 150,000–450,000 cells/mm³); peripheral smear showed dimorphic anemia with a leucoerythroblastic picture and thrombocytopenia without schistocytes. Coagulation profile was normal (PT 14 s, aPTT 28.3 s, INR 1.12), and liver and renal function tests were within normal limits. Serological tests for HBsAg, anti-HCV, and HIV were negative. Cerebrospinal fluid analysis revealed elevated protein of 81 mg/dL (normal 15–45 mg/dL), glucose 117 mg/dL (normal 45–80 mg/dL), positive globulin, nil cells, and acellular cytology. MRI brain demonstrated bilateral multiple small cortical and subcortical infarcts suggestive of small-vessel cerebral vasculitis (Figure 3), while CT thorax revealed centrilobular nodules and ground-glass opacities in bilateral upper lobes consistent with pulmonary microvascular involvement (Figure 4); chest radiograph and CT brain were unremarkable. Infectious etiologies including tuberculosis were excluded with negative sputum AFB smear, CBNAAT, blood cultures, and viral serology. Autoimmune evaluation revealed strong antinuclear antibody positivity (3+) and elevated anti-β2 glycoprotein I IgG (normal < 20 GPL units), confirming systemic lupus erythematosus with secondary antiphospholipid syndrome. The patient was initiated on high-dose systemic corticosteroids and anticoagulation therapy, following which he showed rapid clinical improvement with resolution of tremulousness and mucocutaneous lesions, cognitive recovery with MMSE improving to 30/30, radiological regression of pulmonary lesions, and no further seizure episodes; he was discharged in stable condition with appropriate immunosuppressive and anticoagulant therapy and advised close follow-up.

Figure 1: Oral ulcers with scabbing over lips and Erythematous papular rash over face and upper trunk.

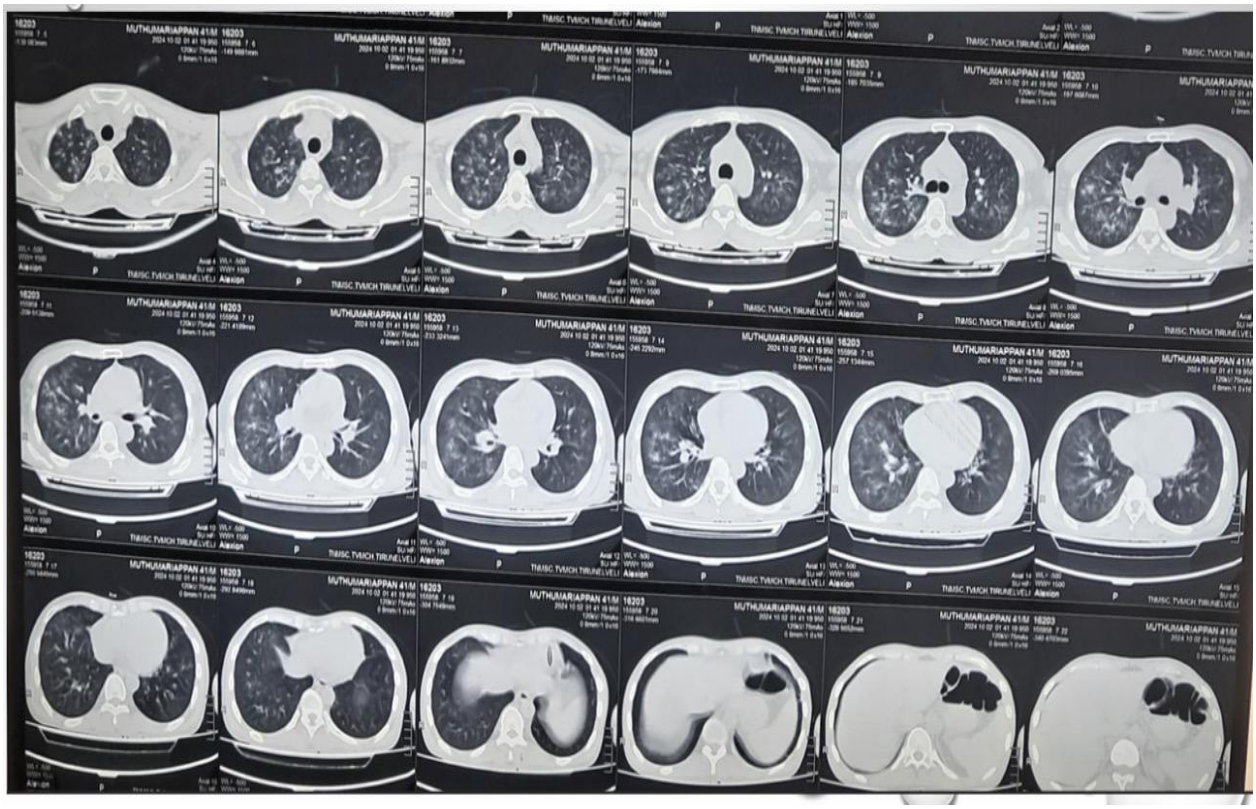

Figure 2: CT chest showing centrilobular nodules and ground-glass opacities in bilateral upper lobes suggestive of pulmonary vasculitis.

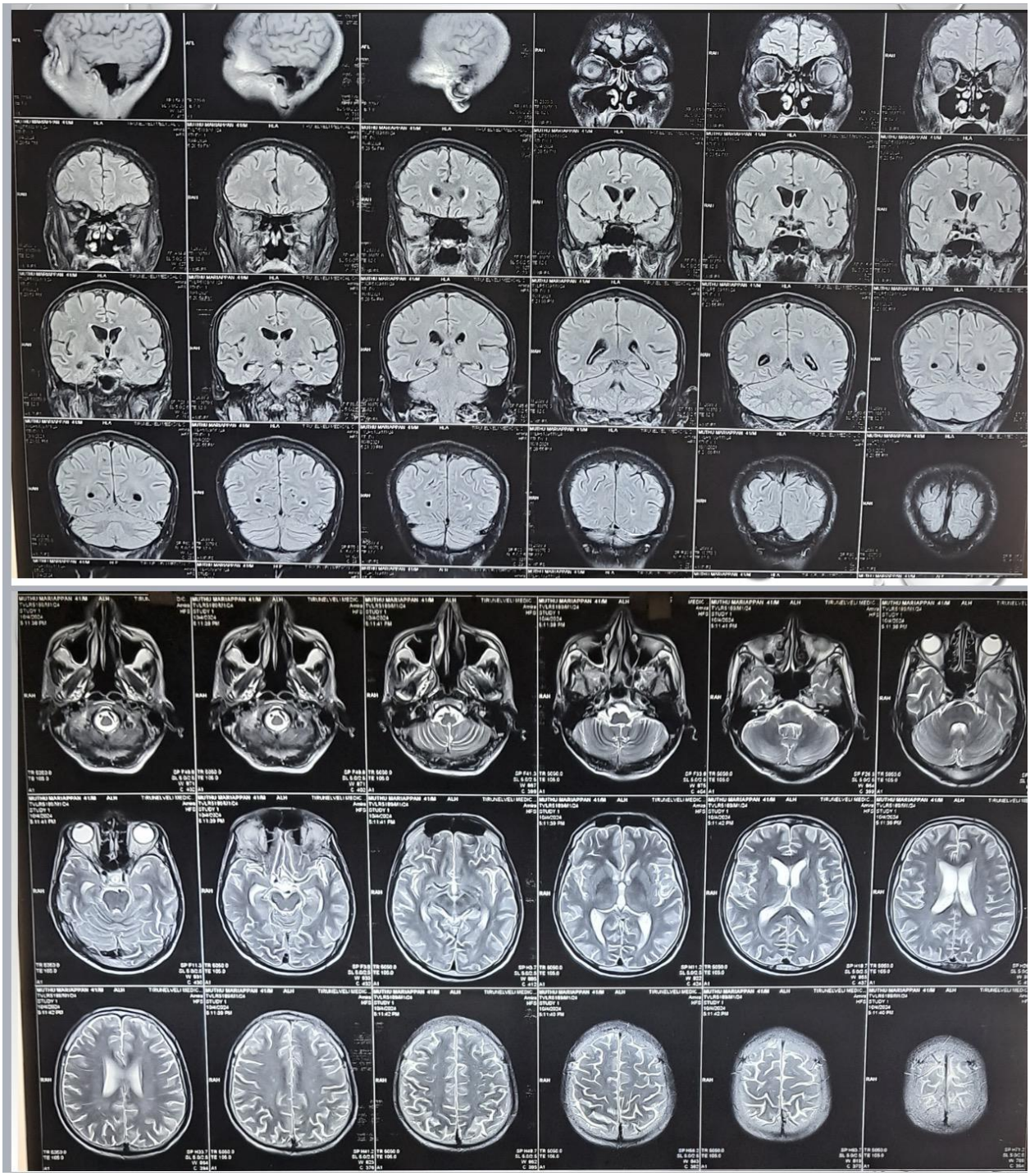

Figure 3: MRI brain showing bilateral multiple small cortical and subcortical infarcts suggestive of cerebral vasculitis.

DISCUSSION

Systemic lupus erythematosus (SLE) is a prototypical immune-complex–mediated autoimmune disorder with heterogeneous clinical manifestations arising from multisystem involvement, and vasculitis represents a severe expression of active disease resulting from immune-complex deposition within vessel walls, complement activation, and subsequent endothelial injury leading to fibrinoid necrosis and luminal compromise.[1,4] While cutaneous small-vessel vasculitis is relatively common, clinically significant visceral vasculitis involving critical organs such as the central nervous system and lungs is rare and associated with substantial morbidity and mortality.[1,4] In the present case, central nervous system involvement manifested as seizures, altered behavior, cognitive dysfunction, and multifocal cerebral infarctions, a constellation suggestive of small-vessel cerebral vasculitis; such manifestations may result from immune-mediated vascular inflammation, antiphospholipid antibody–related thrombosis, and cytokine-mediated neuronal injury.[2,4] The absence of infectious or metabolic etiologies,

combined with cerebrospinal fluid findings of elevated protein without pleocytosis and characteristic MRI features, supports an inflammatory vasculitic process rather than infectious encephalitis or primary thromboembolic disease. Although pulmonary involvement in SLE is common, pulmonary vasculitis and microvascular disease are uncommon and frequently underrecognized, with immune-mediated injury to the pulmonary microcirculation resulting in capillaritis and microthrombosis, producing radiological findings such as centrilobular nodules and ground-glass opacities that often mimic infection, as observed in this patient.[3,4] The coexistence of secondary antiphospholipid syndrome, evidenced by elevated anti-β2 glycoprotein I IgG antibodies, likely amplified vascular injury by promoting endothelial activation, platelet aggregation, and thrombosis, thereby synergistically contributing to both multifocal cerebral infarcts and pulmonary microvascular lesions.[1,4] An additional distinguishing feature of this case is the occurrence of this aggressive disease phenotype in a male patient, as male sex has been associated with higher disease activity, increased frequency of vasculitis, neuropsychiatric involvement, hematological abnormalities, and poorer outcomes in SLE.[1,4] The rapid clinical and radiological improvement following initiation of high-dose systemic corticosteroids and anticoagulation further supports an immune-mediated inflammatory and thrombotic pathogenesis and underscores the importance of early recognition and prompt targeted therapy. This case is unique due to its rare initial presentation with simultaneous central nervous system and pulmonary vasculitis, coexistence of secondary antiphospholipid syndrome, and aggressive disease course in a male patient, highlighting the need for heightened clinical suspicion when evaluating patients with unexplained multisystem ischemic and inflammatory manifestations.

CONCLUSION

This case highlights a rare and severe initial presentation of systemic lupus erythematosus manifesting as simultaneous central nervous system and pulmonary vasculitis, complicated by secondary antiphospholipid syndrome, in a male patient. Such a constellation of findings is distinctly uncommon, as SLE typically presents with mucocutaneous, musculoskeletal, or renal involvement, and clinically overt multisystem vasculitis at disease onset is infrequently encountered.[1,4] The occurrence of seizures, cognitive impairment, and multifocal cerebral infarctions alongside pulmonary microvascular involvement underscores the aggressive inflammatory and thrombotic potential of active SLE, particularly when compounded by antiphospholipid antibodies.[2,4] This case is clinically important as it illustrates the diagnostic challenges posed by atypical and overlapping neurological and pulmonary manifestations, which can mimic infectious, metabolic, or primary thromboembolic disorders and lead to delays in appropriate therapy. The dramatic and sustained clinical as well as radiological improvement following timely initiation of high-dose immunosuppressive therapy and anticoagulation emphasizes that early recognition and aggressive targeted treatment can be organ-saving and potentially life-saving.[1,4] Additionally, the presentation in a male patient is noteworthy, as male sex has been associated with higher disease severity, increased vasculitic involvement, and poorer outcomes, necessitating heightened clinical vigilance.[1,4] This case therefore adds to the limited existing literature on multisystem vasculitis as an initial manifestation of SLE and reinforces the need for clinicians to maintain a high index of suspicion for autoimmune vasculitis in patients presenting with unexplained multisystem neurological and pulmonary involvement, even in the absence of classical features of SLE.

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