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Case Report

Lupus podocytopathy presenting as lupus cerebritis: a rare diagnostic challenge in a young female

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ABSTRACT

Lupus cerebritis is a serious complication usually seen in patients with lupus nephritis. However, lupus podocytopathy presenting as lupus cerebritis is extremely rare and, to the authors' knowledge, has not been reported. We present the case of a 19-year-old female with no known comorbidities who presented with seizures, headache, and altered sensorium. Cerebrospinal fluid analysis showed lymphocytic pleocytosis, and brain imaging revealed diffuse encephalitis. She was initially treated as viral encephalitis, but subsequent serology confirmed systemic lupus erythematosus with high-titer ANA and positive nucleosome and histone antibodies. The diagnosis of lupus cerebritis was made, and she improved with high-dose corticosteroids and immunosuppressive therapy. Within days, she developed nephrotic-range proteinuria and hypoalbuminemia (1.8 g/dl). Kidney biopsy demonstrated diffuse podocyte foot process effacement without immune complex deposition, consistent with lupus podocytopathy. She was treated with corticosteroids, hydroxychloroquine, and mycophenolate mofetil, resulting in remission. This case highlights that lupus podocytopathy can rarely present with neurological symptoms, and timely diagnosis and treatment can lead to excellent outcomes.

Keywords: Lupus podocytopathy, Lupus cerebritis, Nephrotic syndrome, Systemic lupus erythematosus, Kidney biopsy

INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease affecting multiple organ systems, with renal involvement being a common and serious manifestation. Approximately 50–60% of patients with SLE develop renal disease, and lupus nephritis remains a major cause of morbidity and mortality.[1] Classical lupus nephritis is characterized by immune complex-mediated injury with granular deposits on immunofluorescence. In contrast, lupus podocytopathy is a rare subset of lupus nephritis, accounting for about 1% of cases, and is defined by minimal change disease (MCD) or focal segmental glomerulosclerosis (FSGS)-like histology with diffuse podocyte foot process effacement and no significant immune complex deposition.[2,3] It typically presents with abrupt-onset nephrotic syndrome and tends to respond well to corticosteroids, often with a more favorable prognosis than classical immune complex-mediated lupus nephritis.[4]

SLE can also affect the central nervous system (CNS), producing a spectrum of neuropsychiatric manifestations. When encephalopathy and seizures predominate, the term "lupus cerebritis" is often applied.[5] This condition may present at any stage of the disease, including as the initial symptom, and may mimic infectious, metabolic, or vascular causes.[6] The concurrent presentation of lupus cerebritis and lupus podocytopathy is exceedingly rare, with very few reported cases. This report describes a young female who initially presented with neurological features consistent with lupus cerebritis and was later

found to have biopsy-confirmed lupus podocytopathy. This case underscores the importance of maintaining a broad differential diagnosis and performing renal biopsy in SLE patients with atypical renal presentations.

CASE REPORT

A 19-year-old female presented to the emergency department in January 2023 with acute-onset headache, generalized tonic-clonic seizures, blurred vision, and altered sensorium. She had a two-year history of intermittent low-grade fever, arthralgia involving small joints, malar rash, generalized fatigue, and intermittent pedal edema, none of which had been previously investigated.

On arrival, she was disoriented, drowsy, and responsive only to painful stimuli. Her pulse was 120/min, blood pressure 160/90 mmHg, and temperature 38.3°C. Physical examination revealed pallor and bilateral pedal edema. Neurological examination showed decreased power (3/5) in the right lower limb with hyporeflexia and no meningeal signs.

Laboratory investigations were summarized in Table 1.

Table 1: Laboratory investigations.

Parameter	Value	Reference range
Hemoglobin (g/dl)	9.2	12–16
Total leukocyte count (per mm^3)	6,800	4,000–11,000
Platelet count (per mm^3)	2.1×10^5	1.5-4.0×10^5
ESR (mm/hour)	60	<20
Serum creatinine (mg/dl)	0.8	0.6–1.2
Serum urea (mg/dl)	22	10–40
Serum albumin (g/dl)	1.8	3.5–5.0
24-hour urine protein (g/dl)	4.2	<0.15

Cerebrospinal fluid analysis (Table 2) showed lymphocytic pleocytosis.

Autoimmune workup (Table 3) revealed high-titer ANA, positive nucleosome and histone antibodies, and low complement levels. Nerve conduction studies suggested mononeuritis multiplex.

Contrast-enhanced CT brain showed a hypodense lesion in the right capsuloganglionic region. MRI brain with MR angiography demonstrated diffuse cerebral edema and patchy hyperintensities in the parietal and occipital lobes bilaterally, consistent with lupus cerebritis. She was treated with intravenous methylprednisolone (1 g/day for five days), followed by tapering oral prednisolone, resulting in marked neurological improvement.

Despite neurological recovery, nephrotic-range proteinuria and pedal edema persisted. On day 10 of hospitalization, a kidney biopsy was performed. Light microscopy (Figure 1) showed preserved glomerular architecture without segmental sclerosis or crescents. Immunofluorescence was negative for immune complex deposits. Electron microscopy revealed widespread podocyte foot process effacement without electron-dense deposits, consistent with lupus podocytopathy.

Table 2: Cerebrospinal fluid analysis.

Parameter	Value	Reference range
Appearance	Clear	Clear
Opening pressure (mm H_2O)	170	100–200
Cells (cells/mm^3)	60 (90% lymphocytes)	0–5
Protein (mg/dl)	85	15–45
Glucose (mg/dl)	58	40–70
Gram stain	No organisms seen	Negative
Culture	Sterile	Sterile

Table 3: Autoimmune workup.

Parameter	Result	Reference range
ANA	Positive	Negative
Anti-nucleosome antibody	Positive	Negative
Anti-histone antibody	Positive	Negative
Anti-dsDNA	Negative	Negative
C3 complement (mg/dl)	40	90-180
C4 complement (mg/dl)	6	10-40

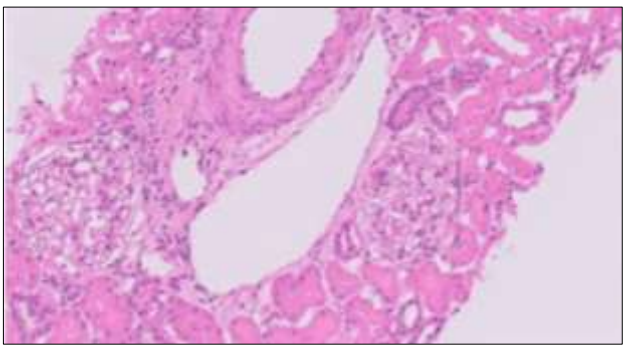

Figure 1: Light microscopy of renal biopsy showing preserved glomerular architecture without segmental sclerosis or crescents; electron microscopy revealed diffuse podocyte foot process effacement without electron-dense deposits, consistent with lupus podocytopathy.

She was treated with oral prednisolone, hydroxychloroquine 200 mg twice daily, and mycophenolate mofetil 500 mg twice daily, escalated to 1

g twice daily. Over two months, her proteinuria declined and serum albumin normalized. Corticosteroids were tapered off, and mycophenolate reduced to 360 mg twice daily. Follow-up MRI brain in June 2025 showed complete resolution of lesions. She remains in remission under regular nephrology and neurology follow-up.

DISCUSSION

Lupus podocytopathy is a rare histological variant of lupus nephritis, characterized by diffuse podocyte foot process effacement without immune complex deposition, resembling MCD or FSGS.[2,3] Its pathogenesis may involve podocyte injury mediated by cytokines or circulating autoantibodies rather than immune complex deposition. Patients typically respond well to corticosteroids, with an overall favorable renal prognosis compared to proliferative lupus nephritis.[4]

Neuropsychiatric lupus is a recognized manifestation of SLE and may occur at disease onset or during flares. Lupus cerebritis, presenting with encephalopathy and seizures, is one of the more severe forms, requiring prompt immunosuppressive therapy to prevent long-term sequelae.[5,6]

The simultaneous presentation of lupus cerebritis and lupus podocytopathy is rare, with only a few isolated cases reported.[7,8] In our patient, renal involvement was not initially suspected due to the dominant CNS features. The diagnosis of lupus podocytopathy was established only after renal biopsy, emphasizing its importance in atypical cases. Early and targeted immunosuppression led to rapid improvement in both neurological and renal manifestations, consistent with prior reports.[4,7]

This case highlights the need for heightened clinical suspicion for dual organ involvement in SLE, especially in young women presenting with new-onset seizures and nephrotic syndrome. Early biopsy and appropriate therapy can significantly alter the disease course.

CONCLUSION

This case illustrates an unusual and diagnostically challenging dual presentation of SLE as lupus cerebritis and lupus podocytopathy. It emphasizes the importance of considering renal biopsy in SLE patients with proteinuria even when other organ manifestations dominate. Timely diagnosis and appropriate immunosuppressive therapy can lead to excellent outcomes in such rare overlapping presentations.

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REFERENCES

1. Almaani S, Meara A, Rovin BH. Update on lupus nephritis. Clin J Am Soc Nephrol. 2017;12:825-35.
2. Kraft SW, Schwartz MM, Korbet SM, Lewis EJ. Glomerular podocytopathy in patients with systemic lupus erythematosus. J Am Soc Nephrol. 2005;16:175-9.
3. Dube GK, Markowitz GS, Radhakrishnan J, D'Agati VD. Minimal change disease in systemic lupus erythematosus. Clin Nephrol. 2002;58:120-6.
4. Chen D, Hu W. Lupus podocytopathy: a distinct entity of lupus nephritis. J Nephrol. 2018;31:629-34.
5. Unterman A, Nolte JE, Boaz M, Abady M, Shoenfeld Y, Zandman-Goddard G. Neuropsychiatric syndromes in systemic lupus erythematosus: a meta-analysis. Semin Arthritis Rheum. 2011;41:1-11.
6. Oliva-Damaso N, Payan C, Oliva-Damaso E, Pereda T, Bomback AS. Lupus podocytopathy: an overview. Adv Chronic Kidney Dis. 2019;26:369-75.
7. Hu WX, Chen Y, Wang S, Chen H, Liu Z, Zeng C, et al. Lupus podocytopathy: a retrospective analysis of 50 cases from a single center. Clin J Am Soc Nephrol. 2016;11:2003-10
8. Weening JJ, D'Agati VD, Schwartz MM, Surya VS, Charles E Alpers, Gerald B Appel, et al. The classification of glomerulonephritis in systemic lupus erythematosus revisited. Kidney Int. 2004;65:521-30.

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