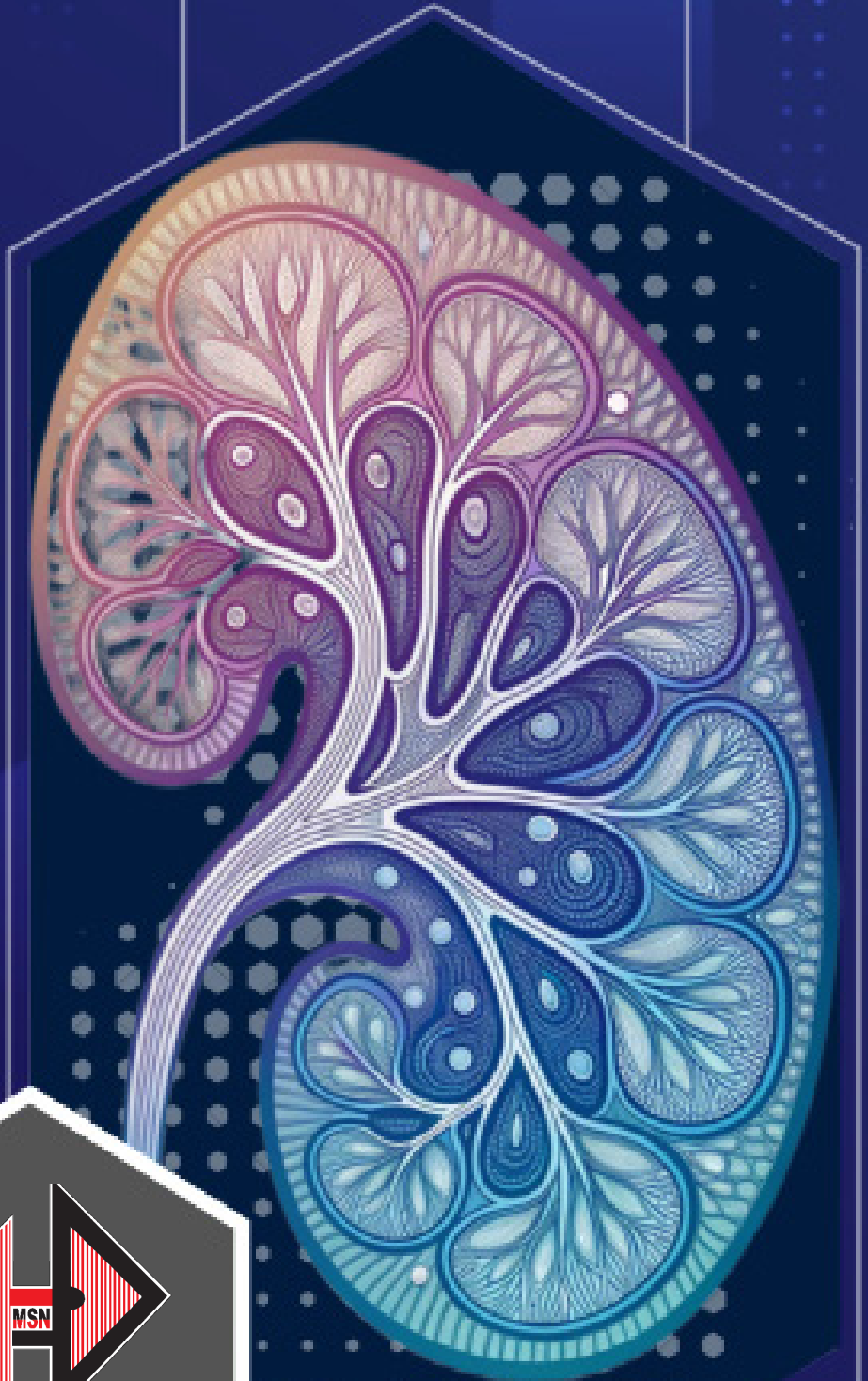


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**JCTN RENAL CPC  
HIGHLIGHTS**

**A Headache Beyond the Brain: Cerebral Venous Sinus Thrombosis Revealing IgA Nephropathy**

*Wong Jze Wei, Christopher Thiam Seong Lim*

**Necrotising Lymphadenitis with Bicytopenia as Prelude to Systemic Lupus Erythematosus**

*Nor Izzati Mohd Zuki, Christopher Thiam Seong Lim, Fauzah Abdul Ghani*



# A HEADACHE BEYOND THE BRAIN: CEREBRAL VENOUS SINUS THROMBOSIS REVEALING IgA NEPHROPATHY

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## CASE PRESENTATION

A 49-year-old gentleman, ex-smoker with no known medical illness, presented with 2-week history of thunderclap headache (sudden onset, occipital and right-sided, associated with nausea and photophobia), frothy urine, and pedal oedema. On examination, he was alert and hemodynamically stable with unremarkable systemic findings.

## INVESTIGATIONS

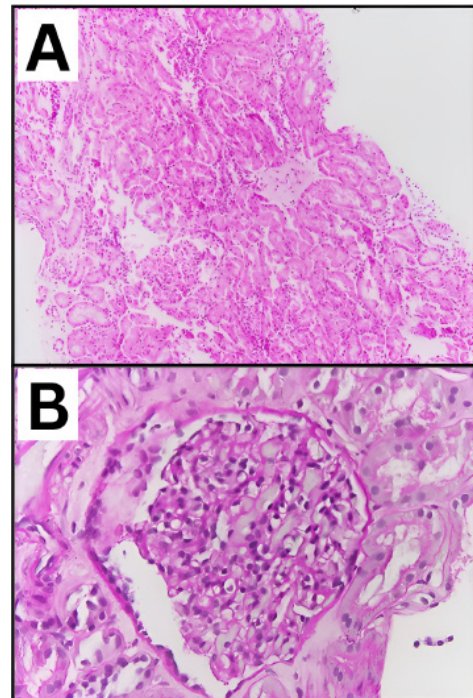
Blood tests revealed severe hypoalbuminemia (13 g/L) with nephrotic-range proteinuria (8.73 g/day) and microscopic haematuria (RBC 2+), alongside hypercholesterolemia (12.8 mmol/L). Renal function was preserved (creatinine 85  $\mu\text{mol/L}$ , eGFR  $>90$  mL/min/1.73 m<sup>2</sup>). Immunology showed: ANA and ANCA negative, normal C3 and C4, with weakly positive lupus anticoagulant and negative anti-PLA<sub>2</sub>R. Brain MRI revealed cerebral venous sinus thrombosis. Renal ultrasound showed normal-sized kidneys.

## DIFFERENTIAL DIAGNOSIS

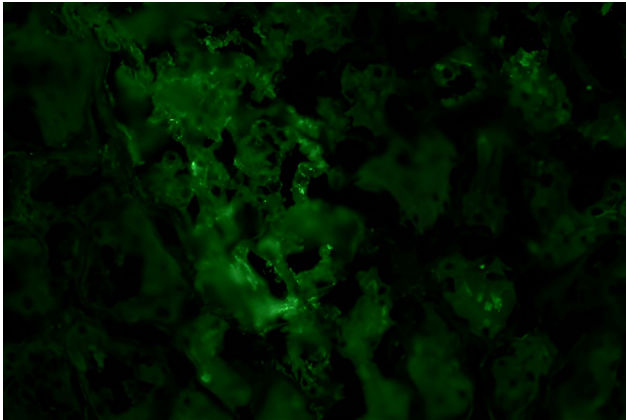
1. Membranous nephropathy (primary or secondary) with hypercoagulable state
2. IgA nephropathy with thrombotic complications
3. Lupus nephritis with antiphospholipid syndrome

## RENAL BIOPSY FINDINGS

A renal biopsy was performed on 29th September 2025.



**Figure 1:** A) One (1) globally sclerosed glomerulus seen, in the background of back-to-back tubules (H&E, original magnification x100). B) There is patchy and mild mesangial hypercellularity seen. Otherwise the capillary loops are delicate with no active proliferative lesion (PAS, original magnification x400).



**Figure 1:** Immunofluorescence studies show IgA small foci of mesangial granular positivity (2+) (original magnification x400)

### FINAL DIAGNOSIS

IgA Nephropathy (Oxford Classification M0 E0 S1 T0 C0) complicated by Cerebral Venous Sinus Thrombosis

### LEARNING POINTS

1. Although IgA nephropathy typically presents with synpharyngitic haematuria or mild urinary abnormalities, the development of nephrotic-range proteinuria suggests more aggressive pathology and a poorer prognosis. In less common situations, as seen in this case, it may even be associated with severe extra-renal complications such as cerebral venous sinus thrombosis.
2. Patients with nephrotic syndrome are at increased risk of thrombotic events due to urinary loss of anticoagulant proteins (antithrombin III, protein C and S). The weakly positive lupus anticoagulant in this case is likely a transient finding related to the hypercoagulable state of nephrotic syndrome, rather than true antiphospholipid syndrome.
3. Negative serology should not deter renal biopsy when clinical presentation suggests glomerulonephritis. Histology remains the diagnostic gold standard
4. Thunderclap headache in the setting of nephrotic syndrome should raise suspicion for cerebral venous sinus thrombosis. Prompt neuroimaging with MRI and magnetic resonance venography is crucial for diagnosis and initiation of anticoagulation therapy.

# NECROTISING LYMPHADENITIS WITH BICYTOPENIA AS PRELUDE TO SYSTEMIC LUPUS ERYTHEMATOSUS

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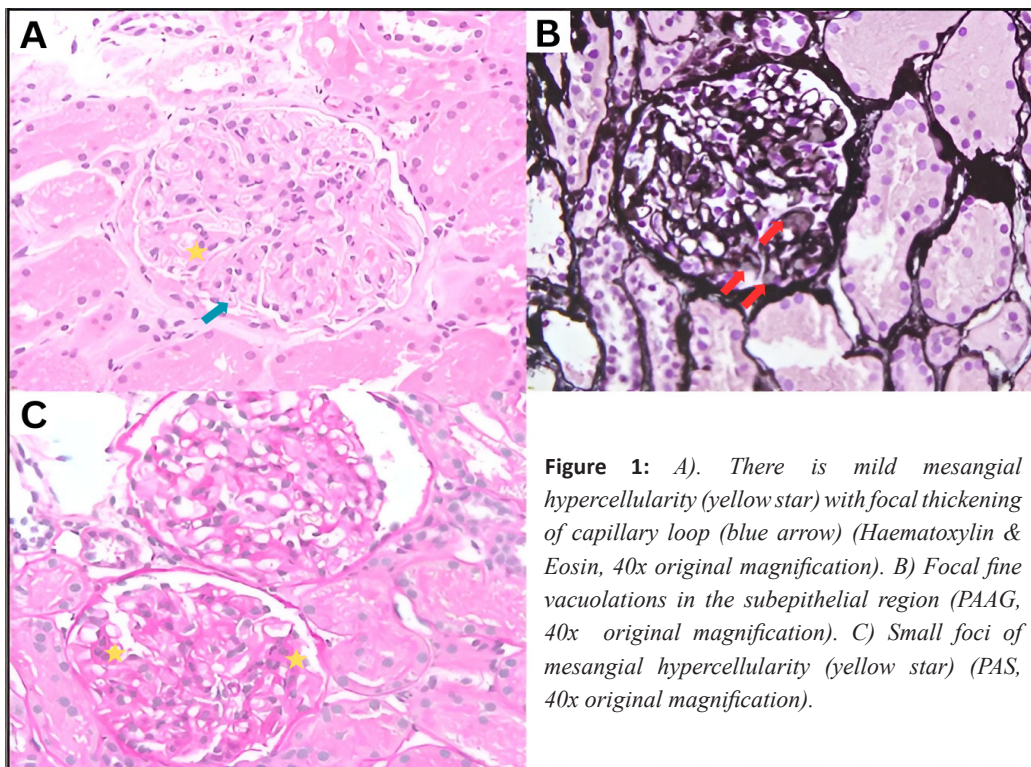
## CASE PRESENTATION

A 32-year-old divorced, nulliparous laboratory assistant presented with a three-month history of intermittent fever, significant weight loss (40 → 34 kg), loss of appetite, and polyarthralgia with early morning stiffness. She also developed painful left cervical swelling three days prior to seeking treatment. There was no history of cough, tuberculosis contact, or travel. She had a prior hospital admission in 2024 for thrombocytopenia, where a bone marrow biopsy showed normal cellularity with increased megakaryopoiesis, suggestive of peripheral platelet destruction.

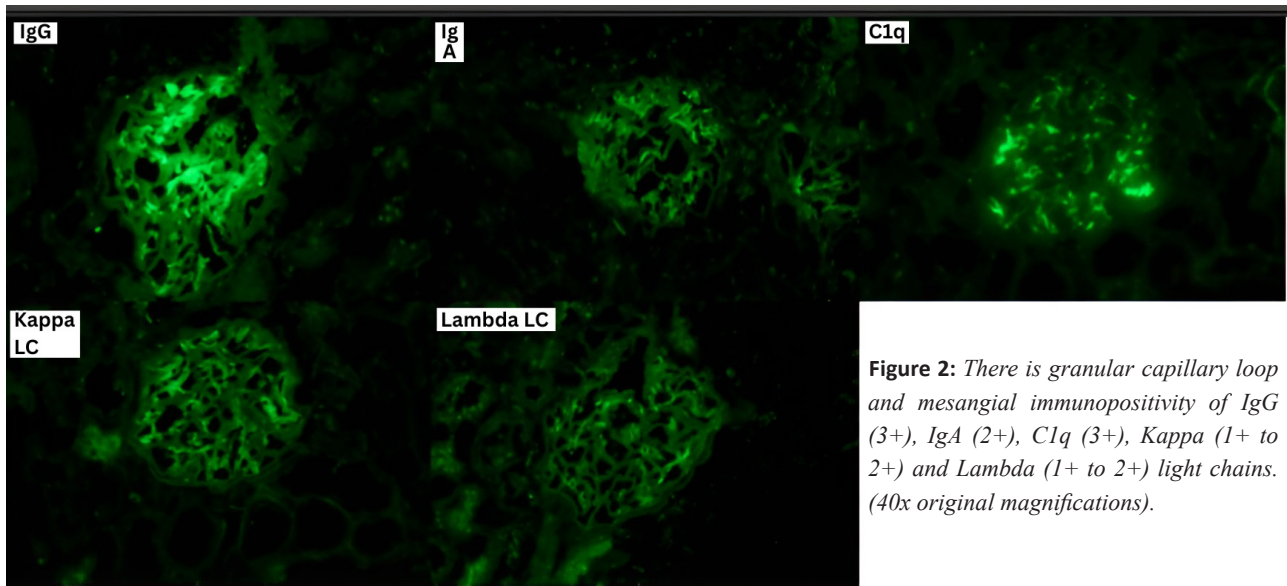
On examination, she was pale, tachycardic (114 bpm), and normotensive (117/69 mmHg), with malar rash and tender left cervical lymphadenopathy. Laboratory investigations revealed normocytic anaemia (Hb 8.5 g/dL), leukopenia, ESR 84 mm/hr, CRP 37.6 mg/L, and normal renal function (creatinine 40 µmol/L). Urine protein was 1+ with a protein-creatinine index of 0.55 g/day. Liver enzymes showed raised AST (79 U/L) with low albumin (26 g/L). Immunological studies revealed high-titre ANA (≥1:1280, speckled), negative anti-dsDNA, but strongly positive anti-Sm, U1RNP/Sm, and ribosomal P-protein. Complement C3 was low (40 mg/dL), C4 normal, and Coombs' test positive. Ferritin was markedly elevated (4953 µg/L). Fine-needle aspiration cytology of the cervical node demonstrated necrotising lymphadenitis without malignant cells. Renal biopsy confirmed ISN/RPS Class II lupus nephritis with membranous components.

## DIFFERENTIAL DIAGNOSIS

Systemic lupus erythematosus (SLE), tuberculous lymphadenitis, and haematological malignancy.



**Figure 1:** A). There is mild mesangial hypercellularity (yellow star) with focal thickening of capillary loop (blue arrow) (Haematoxylin & Eosin, 40x original magnification). B) Focal fine vacuolations in the subepithelial region (PAAG, 40x original magnification). C) Small foci of mesangial hypercellularity (yellow star) (PAS, 40x original magnification).



**Figure 2:** *There is granular capillary loop and mesangial immunopositivity of IgG (3+), IgA (2+), C1q (3+), Kappa (1+ to 2+) and Lambda (1+ to 2+) light chains. (40x original magnifications).*

## LEARNING POINTS

1. Early recognition of systemic inflammatory features such as fever, weight loss, rash, and polyarthritis in a young woman is crucial for early diagnosis of SLE and prevention of organ damage.
2. Autoimmune cytopenias, including thrombocytopenia and Coombs-positive anaemia, may precede the full clinical spectrum of lupus and serve as early diagnostic clues.
3. Serological specificity: Negative anti-dsDNA but positive anti-Sm and anti-U1RNP antibodies support SLE diagnosis, as anti-Sm remains highly specific even in the absence of dsDNA positivity.
4. Lupus lymphadenitis (necrotising lymphadenitis without malignancy) can mimic tuberculous lymphadenitis, especially in endemic regions, underscoring the importance of histopathological confirmation before initiating anti-TB therapy.

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