" Lupus Cystitis Leading to Post-renal AKI and Renal Vein Thrombosis: A Case of Multi-organ SLE"

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Background

- **Lupus cystitis** is a rare manifestation of systemic lupus erythematosus (SLE) characterized by urinary bladder inflammation. Although uncommon, severe bladder dysfunction can lead to obstructive uropathy.
- This condition may, in extremely rare cases, cause compression of the renal veins, leading to venous stasis and subsequent renal vein thrombosis (RVT).

Objective

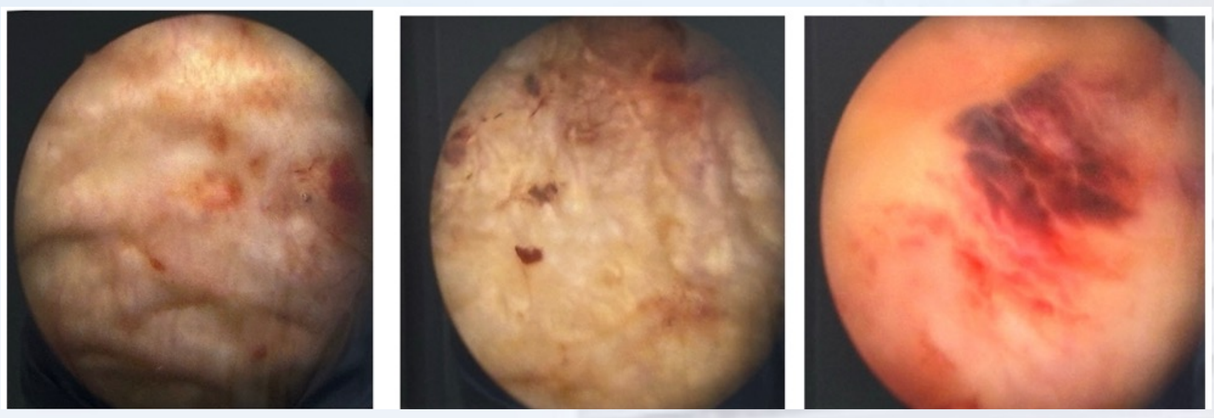
- This report presents a case of post-renal AKI in a patient with active SLE, characterized by lupus cystitis with obstructive uropathy and concurrent bilateral renal vein thrombosis.

Case Report

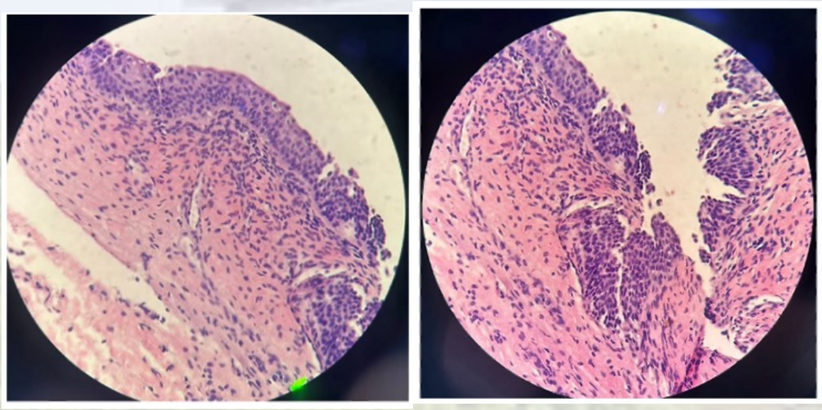
- A 28-year-old Thai woman with a one-year history of polyarthritis presented with recent gastrointestinal (GI) symptoms and seizure. Computed tomography (CT) revealed GI vasculitis, and neuropsychiatric SLE was suspected. Laboratory findings were also consistent with autoimmune hemolytic anemia. She was diagnosed with active SLE and started on intravenous(IV) dexamethasone 10 mg/day.
- On hospital day 13, she developed severe colicky suprapubic pain lasting approximately one hour, exacerbated by urination. Physical examination revealed low-grade fever with marked suprapubic tenderness, without guarding or costovertebral angle tenderness. Laboratory tests showed AKI (serum creatinine increased from 0.96 to 1.3 mg/dL).
- CT imaging demonstrated diffuse bladder wall thickening, bilateral hydroureteronephrosis, and newly developed thrombosis of both renal veins and the inferior vena cava (IVC). The provisional diagnosis was lupus cystitis, which led to post-renal AKI and subsequent thrombosis from bilateral renal vein compression.

Table 1. Laboratory data on admission (Cont.)

Serology	
ANA	Homogenous pattern 1 : 160
Anti-dsDNA	< 1:10
Lupus anticoagulant	Negative APTT 27.2 (25.7-33.3) dRVTT 48 (33.2-48.2)
Beta-2 -glycoprotein1 (IgG IgM IgA)	Negative
Anti-cardiolipid (IgG IgM IgA)	Negative
Protein S activity	57 % (59-118%)
Protein C level	161 (70-140)



Cystoscope : Trabeculation at posterior and lateral wall of bladder, hemorrhagic cystitis, submucosal edema



Histopathological : Lymphocytic infiltration in lamina propria

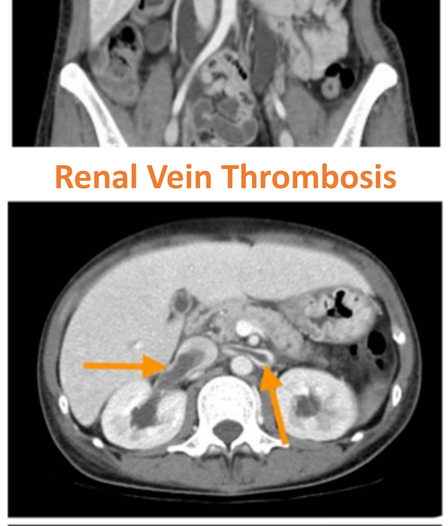
Case Report (Cont.)

Table 1. Laboratory data on admission

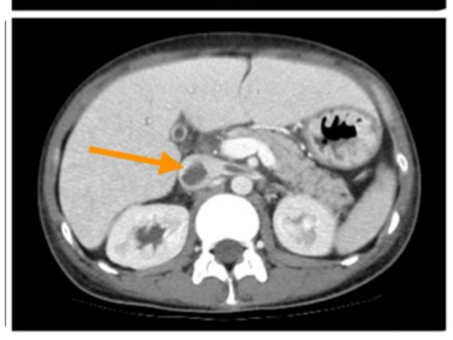
Parameters	Results	Reference Range
Hb	8.7	12.0-14.9 g/L
Hct	25.8	37.0-45.7 %
MCV	80.6	83-97 fl
WBC	11340	4400-10300/ul
Platelet	93 x10^3	179-435 x10^{3}/ul
INR	1.11	0.91 - 1.08 sec
Creatinine	0.96	0.51-0.95 mg/dL
Sodium	136	135 - 145 mmol/L
Potassium	2.8	3.5 - 5.1 mmol/L
Chloride	107	98 - 107 mmol/L
Bicarbonate	17	22 - 29 mmol/L
Albumin	2.4	3.5-5.5 g/dL
C3	38.4 (L)	90-180 mg/dL
C4	3.0 (L)	10-40 mg/dL

Urine	Results	Reference Range
Sp.gr.	1.014	1.003-1.030
Protein	2+	Negative
Blood	3+	Negative
WBC	5-10	0-5/HPF
RBC	50-100	0-2/HPF
24 hr urine protein	1824	mg/24hours
24 hr urine creatinine	638	mg/24hours

Obstructive uropathy



Renal Vein Thrombosis



- Cystoscopy confirmed diffuse bladder inflammation with a trabeculated, hyperemic mucosa, Hunner's ulcers, and petechial hemorrhages.
- A random bladder tissue biopsy showed minimal lymphocytic infiltration in the lamina propria, with no evidence of vasculitis, granulomas, dysplasia, malignancy, or microorganisms.
- She was treated with high-dose IV methylprednisolone for 3 days, followed by IV cyclophosphamide (800 mg), hydroxychloroquine, and low molecular weight heparin. Although her condition initially improved, she developed a spontaneous bladder rupture two days after receiving cyclophosphamide, which was managed conservatively.
- By hospital day 40, she was discharged in stable condition with complete resolution of AKI (serum creatinine 0.7 mg/dL). Mycophenolate mofetil was initiated as maintenance therapy in the outpatient setting.

Conclusion

- Although uncommon, lupus cystitis can lead to post-renal AKI and renal vein thrombosis.
- Early recognition and prompt immunosuppressive therapy are key to favorable outcomes.

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