International Journal of Science and Research (IJSR)
ISSN: 2319-7064
Impact Factor 2024: 7.101

Adult-Onset Nephrotic Syndrome with Membranous Nephropathy Complicated by Right Renal Vein Thrombosis and Acute Pyelonephritis: A Case Report

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Abstract: ***Nephrotic syndrome in adults presents significant diagnostic and therapeutic challenges, particularly in the presence of thrombotic and infectious complications. We report a 31-year-old male with biopsy-proven membranous nephropathy who developed right renal vein thrombosis and acute pyelonephritis. CT imaging revealed thrombosis extending into the inferior vena cava along with features of pyelonephritis. The patient improved with anticoagulation, intravenous antibiotics, and supportive therapy. This case underscores the importance of early imaging and coordinated multidisciplinary management in complicated nephrotic syndrome.***

Keywords: Membranous nephropathy, Nephrotic syndrome, Renal vein thrombosis, Acute pyelonephritis, Direct oral anticoagulants (DOACs)

1. Introduction

Membranous nephropathy (MN) is one of the leading causes of nephrotic syndrome in adults and is classically characterized by subepithelial immune complex deposition along with thickening of the glomerular basement membrane (1, 2). The disease may present with edema, heavy proteinuria, and variable degrees of renal dysfunction. In addition to these renal manifestations, patients with nephrotic syndrome are at substantially increased risk of thromboembolic complications due to alterations in the coagulation–fibrinolytic balance, including urinary losses of natural anticoagulants, increased hepatic synthesis of clotting factors, enhanced platelet aggregation, and haemoconcentration (3).

Renal vein thrombosis (RVT) is a serious yet often underrecognized complication of nephrotic syndrome. Its presentation may range from incidental detection to acute flank pain, haematuria, or worsening renal function. Imaging modalities such as Doppler ultrasonography and contrast-enhanced CT are essential for diagnosis (4). Furthermore, simultaneous occurrence of infection—especially **acute pyelonephritis**—can complicate clinical assessment. Infection-driven systemic inflammation may worsen hypercoagulability and influence the choice and timing of anticoagulation therapy (5).

In this case, we describe a young adult male with biopsy-proven membranous nephropathy who developed both renal vein thrombosis and acute pyelonephritis—two important but diagnostically challenging complications. The case highlights the importance of early recognition, appropriate imaging, and coordinated management.

2. Case Presentation

A 31-year-old male presented with a six-day history of persistent, dull left flank discomfort that gradually progressed in severity. During the same period, he developed swelling of his left lower limb that worsened over a few days, accompanied by intermittent low-grade fever and generalized fatigue. He denied urinary symptoms, recent travel, trauma, or previous renal disease. On examination, he was afebrile with stable vital signs and had mild pallor without icterus. Cardiovascular and respiratory examinations were unremarkable, while abdominal examination revealed localized tenderness over the left flank without guarding or rigidity. The left lower limb showed mild, non-pitting edema without calf tenderness or erythema. Laboratory investigations demonstrated nephrotic-range proteinuria (3+), serum albumin of 2.9 g/dL, elevated CRP of 64.8 mg/L, serum cholesterol of 266 mg/dL, and normal renal function, with sterile blood and urine cultures—findings consistent with nephrotic syndrome with systemic inflammation. Ultrasonography revealed an enlarged, mildly hyperechoic right kidney, prompting further evaluation with contrast-enhanced CT, which showed features of acute right-sided pyelonephritis, including a striated nephrogram and perinephric fat stranding, along with a partially occlusive right renal vein thrombosis extending into the inferior vena cava; no pulmonary embolism or lower limb deep venous thrombosis was detected. Renal biopsy performed to investigate the nephrotic-range proteinuria revealed thickened glomerular basement membranes on light microscopy and granular IgG and C3 deposits on immunofluorescence, confirming membranous nephropathy (1,2). The patient received **IV Ceftriaxone 1 g once daily** for acute pyelonephritis and **Enoxaparin 1 mg/kg subcutaneously every 12 hours** for renal vein thrombosis. Anticoagulation was later transitioned to **Apixaban 10 mg twice daily for 7 days**, then **5 mg twice daily**. Edema was

managed with **Furosemide 40 mg once daily**, along with **sodium restriction** and **moderate fluid control**. Serial Doppler studies and laboratory monitoring assessed thrombus stability, renal function, electrolyte status, and therapeutic response throughout treatment. He showed steady clinical improvement, with reduction in flank pain and inflammatory markers, and was discharged with outpatient nephrology follow-up and a plan to begin immunosuppressive therapy after resolution of the infection.

Parameter	Result	Reference Range	Interpretation
Hemoglobin	11 g/dL	13–17 g/dL	Mild anemia
Serum Albumin	2.9 g/dL	3.5–5.0 g/dL	Hypoalbuminemia (nephrotic syndrome)
Urine Protein (Dipstick)	3+ (Nephrotic-range)	Negative / <150 mg/day	Heavy proteinuria
Serum Cholesterol	266 mg/dL	<200 mg/dL	Hyperlipidemia
C-Reactive Protein (CRP)	64.8 mg/L	<6 mg/L	Markedly elevated; inflammation/infection
Serum Creatinine	Normal	0.7–1.2 mg/dL	Preserved renal function
Blood Culture	Sterile	—	No bacteremia
Urine Culture	Sterile	—	Possible culture-negative pyelonephritis
Ultrasound Abdomen	Bulky right kidney	—	Suggestive of acute pyelonephritis
Contrast CT Abdomen (CECT)	Acute right pyelonephritis; right renal vein thrombosis extending into IVC	—	Confirms infection + thrombosis
Renal Biopsy	Thickened GBM with granular IgG & C3 deposits	—	Membranous nephropathy
Thrombosis Assessment	RVT confirmed; partial IVC extension	—	Supports need for anticoagulation

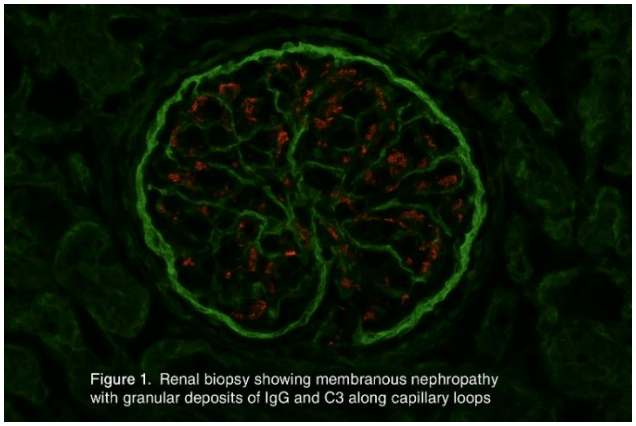

Figure 1: Granular IgG/C3 subepithelial deposits along glomerular capillary loops on immunofluorescence, characteristic of membranous nephropathy

3. Discussion

Membranous nephropathy is a prototypical cause of nephrotic syndrome in adults and carries a high risk of venous thromboembolism. The pathogenesis involves urinary loss of key anticoagulant proteins (e.g., antithrombin III), increased levels of fibrinogen, and reduced fibrinolytic activity, all contributing to a hypercoagulable state (3). RVT is among the most serious complications and may present acutely or remain clinically silent until discovered on imaging (4).

In this patient, RVT was symptomatic, presenting with flank pain—a classic but nonspecific feature. The extension of the thrombus into the IVC is clinically significant due to the risk of propagation and pulmonary embolism. CT imaging remains the gold standard for diagnosis and was crucial in this case (6,7).

Concurrently, the patient showed features of acute pyelonephritis, demonstrated radiologically despite sterile cultures. Culture-negative pyelonephritis is frequently observed in partially treated infections or early presentations (5). Infection also amplifies systemic inflammatory markers and may exacerbate the hypercoagulable state, potentially accelerating thrombosis.

The management of RVT has evolved significantly. While warfarin was historically the mainstay, direct oral anticoagulants such as **apixaban** have shown increasing safety and efficacy in renal vein thrombosis—including in nephrotic syndrome and COVID-associated thrombosis—making them an attractive alternative (8,9).

This case underscores several important clinical lessons:

Persistent flank pain in nephrotic syndrome should prompt evaluation for RVT. Early imaging facilitates timely diagnosis and treatment. Coexisting infections should be actively sought and treated. Anticoagulation strategies should consider both thrombotic risk and infection status. Multidisciplinary care (nephrology, radiology, infectious diseases) significantly improves outcomes. Overall, early diagnosis and coordinated management were key in achieving a favorable recovery in this patient.

4. Conclusion

Membranous nephropathy complicated by renal vein thrombosis and pyelonephritis represents a serious but treatable clinical scenario. Early recognition and coordinated management are essential for improved outcomes.

Consent for participation
"Not applicable"

Consent for publication
"Not applicable"

Availability of data and materials
"Not applicable"

Competing interests
"The authors declare that they have no competing interests"

Funding/support
"This study received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors."

Conflicts of interest
"All authors have disclosed no conflicts of interest.

Ethical approval and Consent to participate
"Not Applicable"

Acknowledgements
"Not Applicable"

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