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Case Report

An unforeseen complication: left ventricular thrombus formation in a patient with membranous nephropathy

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ABSTRACT

Membranous nephropathy (MN) is an autoimmune glomerulopathy and one of the leading causes of adult nephrotic syndrome (NS). It is characterized by subepithelial immune complex deposition, complement activation, and podocyte injury, leading to proteinuria, hypoalbuminemia, hyperlipidaemia, and systemic oedema. A major complication of NS is a hypercoagulable state due to urinary loss of anticoagulants, hepatic overproduction of clotting factors, endothelial dysfunction, and platelet hyperactivity. Thromboembolic events are well-documented in MN, with venous thromboses predominating, whereas arterial thrombosis such as left ventricular thrombus (LVT) remains a rare but clinically significant event. We report the case of a 57-year-old male with type 2 diabetes mellitus who presented with generalized anasarca, dyspnoea on exertion, productive cough, weakness, and cutaneous lesions. Laboratory evaluation revealed nephrotic-range proteinuria (urine protein-creatinine ratio 19.78), hypoalbuminemia (2.0 g/dL), elevated serum creatinine (2.0-2.4 mg/dL). Renal biopsy demonstrated Stage II membranous nephropathy with characteristic basement membrane thickening and IgG-dominant immune deposits. Cardiac evaluation revealed a left ventricular apical clot with mildly reduced ejection fraction (45-50%), in the absence of prior cardiac disease history. Importantly, physicians promptly initiated a target-specific treatment approach addressing the glomerular pathology with immunosuppressive therapy, mitigating hypercoagulability with anticoagulation, and relieving fluid overload with diuretics. This individualized, timely strategy directly targeted the dual pathology of MN and its thrombotic complication, preventing further morbidity and demonstrating the value of precision-based decision-making. This case highlights LVT as a rare but severe thromboembolic complication of MN-induced nephrotic syndrome, even in the absence of pre-existing cardiac disease. Early recognition of hypercoagulability, prompt anticoagulation, and multidisciplinary management are crucial to prevent life-threatening embolic events in such patients.

Keywords: Membranous nephropathy, Nephrotic syndrome, Hypercoagulability, Left ventricular clot, Targeted therapy, Case report

INTRODUCTION

Membranous nephropathy (MN) is a primary glomerular disease commonly causing adult nephrotic syndrome, about 80% of MN cases don't have clear secondary cause, called idiopathic membranous nephropathy (IMN) or primary membranous nephropathy (PMN), and about 20% of MN cases are secondary to autoimmune diseases, infections, malignant tumours, medicine use, and heavy-metal toxicity, called secondary membranous nephropathy(SMN), It presents as diffuse subepithelial immune complex deposition on the glomerular basement membrane (GBM) which leads to GBM thickening, the alteration of podocyte structure, causing the appearance of substantial proteinuria.[1,2]

Immunofluorescence demonstrates diffuse granular deposits of IgG and C3, and electron microscopy typically reveals electron-dense subepithelial deposits. In 70% of cases of IMN, autoantibodies against the M-type phospholipase A2 receptor circulate and bind to a conformational epitope present in the PLA2R on human podocytes, producing characteristic in situ deposits. Three to 10% of IMN patients alternatively have autoantibodies to thrombospondin type-1 domain containing 7A (THSD7A). Both antigens co-localize within glomerular subepithelial deposits with IgG4 (PLA2R). Circulating deposits and glomerular deposits of these autoantibodies have correlated with the likelihood of a spontaneous remission, severity of IMN, and the response to therapy. Eighty percent of patients with MGN present with nephrotic syndrome and nonselective proteinuria. Microscopic haematuria is seen but less commonly than in IgA nephropathy or FSGS.

One-third of patients continue to have relapsing nephrotic syndrome but maintain normal renal function and approximately another third of patients develop renal failure or die from the complications of nephrotic syndrome. Male gender, older age, hypertension, and the persistence of nephrotic-range proteinuria are associated with worse prognosis. MGN has the highest reported incidences of renal vein thrombosis, pulmonary embolism, and deep vein thrombosis. Prophylactic anticoagulation is controversial but has been recommended for patients with hypoalbuminemia.[3]

Thromboembolism is a well-recognized and serious complication of NS with the highest reported rates in patients with PMN.[1] Venous thromboembolism (VTE) typically manifests as deep vein thrombosis (DVT), pulmonary embolism, or renal vein thrombosis with incidence rates ranging between 2% and 37% in studies.[4] Arterial thromboembolism manifests as cardiovascular and cerebrovascular events and is relatively less frequent; however, its risk is eight times higher in patients with NS compared with age-matched and sex-matched controls.[4]The pathogenesis of this acquired hyper coagulopathy in PMN is not well elucidated and is proposed to be multifactorial. Hypoalbuminemia stimulating hepatic synthesis of prothrombotic factors (fibrinogen, factors 5, 8, 10), urinary loss of antithrombotic proteins (protein c and s, antithrombin 3), and elevated platelet count/altered platelet function have been discussed as possible mechanisms; however, evidence is weak.[5] Serum albumin of <2.5 g/dl and severity of proteinuria indicated by an elevated proteinuria/serum albumin ratio have been described as primary risk factors for development of thromboembolic complications.[6] More recently, antiphospholipase A2 receptor (anti-PLA2R) antibody has been implicated as an independent risk factor for VTE in PMN with incidence of VTE and D-dimer levels strongly correlating with higher levels of anti-PLA2R.[7,8]

CASE REPORT

A 57-year-old male presented to the Department of General Medicine with complaints of gradually progressive generalized anasarca for the past 6 months, dyspnoea on exertion and dry cough for 1 year (MMRC grade 2-3), and productive cough with expectoration for the past 2 days. He also reported generalized weakness and body ache for 15-20 days, abdominal pain, and pruritic skin lesions involving the face, neck, buttocks, and groin for 6 months. The patient has a history of type 2 diabetes mellitus and secondary haemoptysis associated with vigorous coughing. He had a history of bidi smoking for 6-7 years, which he discontinued 1 year ago.

Table 1: Investigation chart: clinical presentation (on admission).

Test name	Day 1	Biological reference
Haemoglobin	7.8	13 -17 g/dl
Red blood cell count	2.72	4.5 - 5.5 million/ul
Hematocrit	24.6	40 - 54 %
RDW CV	14.4	11.5 - 14 %
Neutrophils	78.5	50 - 62 %
Lymphocytes	11.6	20 - 40 %
Absolute lymphocyte count	854	1000-3000//µl
Serum creatinine	2.4	0.66 - 1.25 mg/dl
EGFR	28.0	CKD stage 3 (30 - 59 ml/min/1.73m^2) CKD stage 4 (15 - 29 ml/min/1.73m^2)
TSH	6.749	0.400 - 4.049
Serum total protein	4.40	6.3 - 8.2 g/dl
Serum albumin	2.0	3.5 - 5.0 g/dl
Protein urine	1250	0 -20 mg/dl
C- reactive protein (CRP) test	-	0-6 mg/l
Protein urine	1250	0 - 20 mg/dl
Creatinine urine	63.2	-
Protein creatinine ratio urine	19.78	0.1-1.0

Continued.

Test name	Day 1	Biological reference
Activated partial thromboplastin time - APTT	37.8	22.7 - 35.0 seconds
Prothrombin time patient value	9.9	9.39- 12.91 seconds
International normalized ratio (INR)	0.83	0.8 - 1.1

Table 2: Urine analysis.

Test name	Day 1	Biological reference
Appearance	Hazy	-
Protein	Present (+++)	Absent/ present
Blood	Present (+)	Absent
Nitrite	Absent	-
Pus cells	Occasional	0 - 10 /HPF
RBCS	6 - 8	0 - 10 cells/HPF
Epithelial cells	Occasional	0 - 2

On the day of admission, the patient's vital signs were within normal limits.

Anti-Nuclear Antibody (ANA) was negative.

On day 4 of admission, laboratory evaluation. A significantly elevated erythrocyte sedimentation rate (ESR) of 120 mm/hr (reference: 3-12 mm/hr) indicated a high inflammatory state. Notably, D-dimer levels were grossly elevated (>99,990 ng/mL; reference: 0-500 ng/mL), pointing towards a hypercoagulable state.

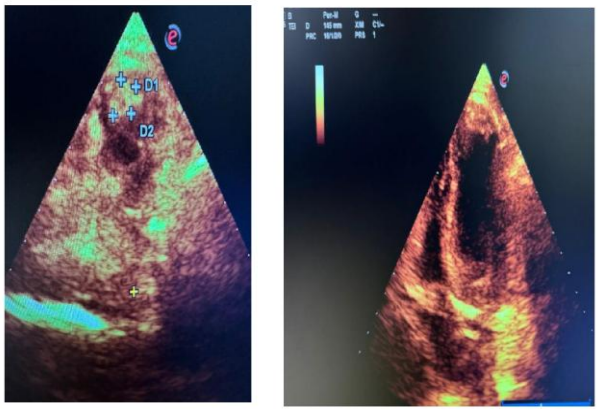

Figure 1: 2D echocardiography and colour doppler.

Regional wall motion abnormality (RWMA) noted. Left ventricular ejection fraction (LVEF): 45-50%.

Normal left ventricular (LV) size with mild LV systolic dysfunction. Presence of left ventricular apical clot. Grade I LV diastolic dysfunction observed.

Biopsy assessment / histopathology assessment

Silver methenamine stain

This section shows up to 9 glomeruli with thickened glomerular basement membranes with characteristic "spikes and holes" appearance, consistent with stage II membranous nephropathy as described in the microscopy report. The glomeruli have open capillary loops, normal mesangial cellularity, and no evidence of crescents, necrosis, or mesangial nodules. These findings align with the light microscopy description in the renal biopsy.

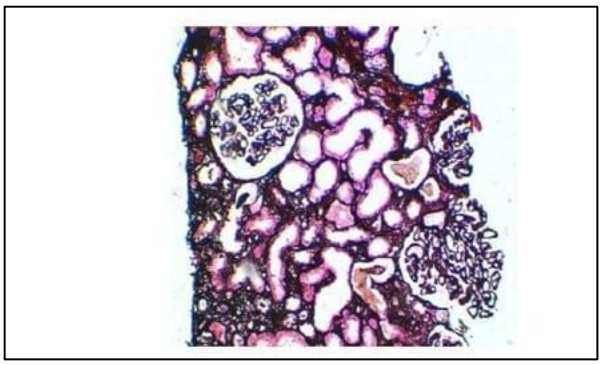

Figure 2: Silver methenamine - stained section.

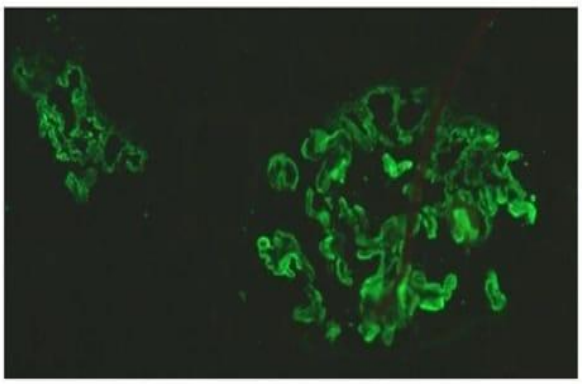

Figure 3: Immunofluorescence microscopy.

Immunofluorescence – igG FITC

Granular peripheral capillary wall positivity for IgG (+3) is demonstrated, confirming immune complex deposition

along the capillary loops. The granular capillary fluorescence pattern strongly supports the diagnosis of membranous nephropathy and corresponds to the immunofluorescence findings detailed in the biopsy report (IgG +3, with additional weaker staining for IgA, C3, Kappa, and Lambda). while IgM and C1q are negative. These findings confirm the presence of immune complex–mediated injury restricted to the glomerular capillary wall without significant vascular or tubular involvement.

Radiological assessment

Day 1 CT KUB plain: Small left kidney with bilateral significant perinephric fat stranding and fascial thickening (Left > Right). Moderate to gross ascites, bilateral gross hydrocele, significant subcutaneous oedema with minimal intramuscular oedema and fat stranding involving the abdominal wall and upper thighs. No renal calculi detected.

Day 1 USG whole abdomen + pelvis: Mildly altered liver parenchymal echotexture (correlation with LFT advised). Gross ascites with internal echoes, reactive cholecystitis, mild subcutaneous oedema of abdominal wall, and mild right- and minimal left-sided pleural effusion with lung collapse and consolidation.

Chest X-ray day 1: Homogeneous opacity involving the right mid and lower lung zones, silhouetting the right dome of the hemidiaphragm and right cardiac border, obscuring the right costophrenic angle, and extending along the left lateral chest wall – suggestive of right-sided pleural effusion.

Treatment given

The patient was initiated on supportive therapy including IV ceftriaxone, IV pantoprazole, electrolyte supplementation, and diuretics (IV furosemide later augmented with oral metolazone) for edema control. Symptomatic agents—codeine–triprolidine, antihistamines, and topical fusidic acid, clotrimazole–beclomethasone, and clobetasol—were used for cough and dermatological lesions.

Following the diagnosis of membranous nephropathy, the patient received IV pulse methylprednisolone (500 mg daily for 3 days), after which he was transitioned to oral prednisolone (20-50 mg/day). Metabolic issues were managed with insulin, rosuvastatin, and thyroxine.

On identification of a left ventricular apical thrombus, IV unfractionated heparin was started and later switched to oral apixaban (2.5 mg twice daily) along with low-dose aspirin. Respiratory support included nebulized Levo salbutamol–ipratropium and oral etofylline–theophylline.

At discharge, the patient was maintained on apixaban, a prednisolone taper, torsemide with metolazone, rosuvastatin, thyronorm, pantoprazole, cefixime–clavulanate, inhaled budesonide–formoterol, and supportive skin and cough medications.

DISCUSSION

MN is one of the most frequent causes of nephrotic syndrome in adults and is increasingly recognized as an autoimmune disease driven by circulating antibodies directed against podocyte surface antigens, most commonly the phospholipase A2 receptor (PLA2R).[9] The resulting subepithelial immune complex deposition and complement activation led to podocyte injury and heavy proteinuria, generating the classical nephrotic state characterized by hypoalbuminemia, edema, and dyslipidaemia. A less widely appreciated but clinically important feature of nephrotic syndrome is the profound hypercoagulability that arises through loss of endogenous anticoagulants—such as antithrombin III, protein C, and protein S—combined with increased hepatic synthesis of coagulation factors, platelet hyperreactivity, and endothelial dysfunction.[10] While venous thrombosis is far more common, arterial thrombosis and intracardiac thrombus formation remain rare but potentially catastrophic complications.

The present case illustrates an uncommon manifestation of MN: the development of a left ventricular thrombus (LVT). Intracardiac thrombosis in nephrotic syndrome typically occurs in the presence of severe left ventricular dysfunction or acute myocardial infarction; however, emerging clinical observations suggest that nephrotic-syndrome–related hypercoagulability alone may be sufficient to trigger intracardiac clot formation even in the absence of significant cardiac pathology.[11] This case supports that possibility, as the patient developed an apical LVT despite having only mildly reduced ejection fraction and no prior ischemic heart disease. Recognition of such presentations is essential because LVT carries a high risk of systemic embolization, including stroke, peripheral arterial occlusion, and visceral infarction.

The management of LVT in the setting of MN requires a dual therapeutic strategy: rapid stabilization of the thrombus and control of the underlying glomerular disease. Anticoagulation remains the mainstay of treatment for LVT. Historically, vitamin K antagonists have been used; however, recent reports support the growing use of direct oral anticoagulants (DOACs) as safe and effective alternatives with easier monitoring requirements.[12] In this case, initiation of intravenous heparin followed by transition to apixaban was in line with current practice trends and resulted in clinical stability without embolic complications.

For MN itself, treatment selection depends on risk stratification based on proteinuria magnitude, renal function, and serologic activity. Rituximab has become a preferred first-line therapy in many centres due to evidence supporting its favourable safety profile and durable remission rates compared with calcineurin inhibitors or

cyclophosphamide-steroid regimens.[13] In the present case, pulse steroid therapy followed by oral corticosteroids was initiated, resulting in partial clinical improvement.

This case highlights the necessity for clinicians to maintain high suspicion for thrombotic complications in patients with MN, especially those presenting with severe hypoalbuminemia. Routine screening for hypercoagulability, early imaging when symptoms suggest cardiac involvement, and prompt anticoagulation can prevent fatal outcomes.

CONCLUSION

LVT is a rare but serious complication of membranous nephropathy, arising from the pronounced hypercoagulable state inherent to nephrotic syndrome. This case reinforces the need for early recognition, aggressive anticoagulation, and targeted immunosuppression. Greater awareness of such atypical presentations can improve patient outcomes and guide clinicians in managing both renal and cardiovascular risks associated with MN.

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REFERENCES

1. Couser WG. Primary Membranous Nephropathy. Clin J Am Soc Nephrol. 2017;12(6):983-97.
2. Ronco P, Beck L, Debiec H, Fervenza FC, Hou FF, Jha V, et al. Membranous nephropathy. Nat Rev Dis Primers. 2021;7(1):69.
3. Jameson JL, Fauci AS, Kasper DL, Hauser SL, Longo DL, Loscalzo J. Harrison's Principles of Internal Medicine. McGraw Hill. 2022.
4. Parker K, Ragy O, Hamilton P, Thachil J, Kanigicherla D. Thromboembolism in nephrotic syndrome: controversies and uncertainties. Res Pract Thromb Haemost. 2023;7(6):102162.
5. Greenberg KI, Choi MJ. Understanding hypercoagulability with nephrotic syndrome: how the plot thickens. Clin J Am Soc Nephrol. 2023;18(2):149-51.
6. Radhakrishnan J. Venous thromboembolism and membranous nephropathy: so what's new?. Clin J Am Soc Nephrol. 2012;7(1):3-4.
7. Zhu H, Xu L, Liu X, Liu B, Zhai C, Wang R, et al. Anti- PLA2R antibody measured by ELISA predicts the risk of vein thrombosis in patients with primary membranous nephropathy. Ren Fail. 2022;44(1):594-600.
8. Li J, Wang X, Jiang S, Li W. Serum PLA2R antibody as a predictive biomarker for venous thromboembolism risk in primary membranous nephropathy. Med Clin (Barc). 2023;161(10):417-21.
9. Ronco P, Beck L, Debiec H, Fervenza FC, Hou FF, Jha V, et al. Membranous nephropathy. Nat Rev Dis Primers. 2021;7(1):69.
10. Kerlin BA, Ayoob R, Smoyer WE. Epidemiology and pathophysiology of nephrotic syndrome–associated thromboembolic disease. Clin J Am Soc Nephrol. 2012;7(3):513-20.
11. Mak SK, Wong PN, Lee KF, Fung LH, Wong AK. Intracardiac thrombus in an adult patient with nephrotic syndrome. Nephrol Dial Transplant. 1996;11(8):1627-30.
12. Cubilier E, Chergui Y, Garrouste C, Ramos I, Philipponnet C, Atenza A, et al. Nephrotic syndrome thromboprophylaxis with direct oral anticoagulants or vitamin K antagonists. 2025;10(5):1468-75.
13. Fervenza FC, Appel GB, Barbour SJ, Rovin BH, Lafayette RA, Aslam N, et al. Rituximab or cyclosporine for membranous nephropathy. N Engl J Med. 2019;381(1):36-46.

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