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CASE REPORT

The Rare Occurrence of Tissue PLA2R-Positive Membranous Nephropathy in a Patient With Sjögren's Syndrome and Renal Tubular Acidosis

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Abstract

Aims/Background Membranous nephropathy (MN) is categorized into primary and secondary forms based on its etiology, with autoimmune diseases significantly contributing to the secondary form. The phospholipase A2 receptor (PLA2R) antibody has been widely used as a specific marker for primary MN. However, explaining PLA2R positivity in MN patients who also exhibit typical features of Sjögren's syndrome (SS) is challenging.

Case Presentation A 51-year-old male patient presented with acute systemic edema, hypertension, macroalbuminuria, and hypoproteinemia, confirming a diagnosis of nephrotic syndrome. Concurrently, SS, characterized by dry mouth and dry eye symptoms, positive specific antibodies, and positive labial gland biopsy results, was also confirmed. Renal pathology indicated MN (stage I–II) with moderate secondary chronic renal tubulointerstitial lesions. The cluster of differentiation 3 (CD3)+ and CD20+ immunostaining of renal tissue was consistent with renal interstitial damage associated with SS, and the clinical manifestations aligned with the typical presentation of renal tubular acidosis (RTA). PLA2R was negative in the serum but positive in the renal tissue.

Conclusion SS is one of the autoimmune diseases associated with secondary membranous nephropathy, which has a low clinical incidence. In this particular case, the diagnosis of SS was confirmed, and the histopathological examination revealed typical tubulointerstitial lesions clinically accompanied by RTA. However, the specific antibody PLA2R, which is typically associated with primary MN, was positive in renal tissue. Additionally, IgG immunodeposition was not observed. Further investigation is warranted to ascertain whether the MN is secondary to SS.

Key words: renal tubular acidosis; membranous nephropathy; phospholipase A2 receptor; Sjögren's syndrome; case report

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Introduction

Primary Sjögren's syndrome (pSS) is a systemic autoimmune disease characterized by lymphocytic infiltration of exocrine glands (Patel and Shahane, 2014), resulting in multisystem involvement. Among renal manifestations, tubulointerstitial nephritis (TIN) is the most common, accounting for 85% of SS cases with kidney lesions, while glomerulonephritis (GN), another primary lesion, arises from

secondary immune complex-mediated mechanisms (Evans et al, 2015; Ren et al, 2008). Membranous nephropathy (MN) is the predominant type of GN in pSS, accounting for 36% and 50% of renal lesions in recent studies of pSS renal biopsy patients (Luo et al, 2019; Pan et al, 2020; Yang et al, 2018). Furthermore, renal tubular acidosis (RTA) is a specific complication of pSS, primarily affecting the renal tubules and typically manifesting as metabolic acidosis, urolithiasis, bone disease, hypokalemia, and, in severe cases, respiratory failure and cardiac dysfunction related to myasthenia gravis (Ohtani et al, 1999).

MN is a leading cause of nephrotic syndrome in adults, characterized by the accumulation of sub-epithelial immune complexes, predominantly Immunoglobulin G (IgG), leading to the thickening of the glomerular basement membrane (Ruggenenti and Remuzzi, 2019). Approximately 20–25% of MN cases are secondary, arising from various etiologies, such as malignant tumors, autoimmune diseases, infections, and exposure to certain drugs or toxins (Keri et al, 2019). SS is one of the rare causes of secondary MN. The phospholipase A2 receptor (PLA2R) serves as a target antigen in about 70% of patients with primary MN (Beck et al, 2009). However, Chen et al (2021) reported that serum PLA2R was positive in 8 cases out of 13 patients with MN and SS. Here, we report a case of membranous nephropathy associated with SS, characterized by PLA2R-positive renal tissue, tubulointerstitial injury, and RTA.

Case Report

Patient History

This case report is compiled in accordance with the CARE consensus guidelines to ensure high-quality reporting. The completed CARE checklist is available for reference as **Supplementary Material**. A 51-year-old male patient was admitted to Jiaxing Hospital of Traditional Chinese Medicine with "bilateral lower limb edema persisting for over 2 months". The edema developed gradually without any identifiable cause. One month before hospital admission, he experienced progressive lower limb edema, swollen eyes, blurred vision, dizziness, and headache. He sought medical attention at a local eye hospital, where his blood pressure was recorded at 190/120 mmHg, with urine protein at 3+, serum albumin level at 19 g/L, and serum creatinine at 105.9 μmol/L. He was diagnosed with "nephrotic syndrome with suspected chronic renal failure", and he was prescribed oral antihypertensive therapy. However, despite clinical intervention, his edema gradually worsened. Furthermore, his medical history showed "hypertension" diagnosed one year before, with a maximum recorded blood pressure of 190/120 mmHg. Upon admission, clinical examination revealed severe edema in both lower limbs and a blood pressure of 144/104 mmHg.

Serum Laboratory Examination

The serum laboratory results obtained upon admission revealed the 24-hour urinary protein was 11,618 mg. Blood biochemistry showed the following: Total protein: 37 g/L, Albumin: 16.2 g/L, Serum total cholesterol: 6.37 mmol/L, Triglyc-

erides: 1.98 mmol/L, Low-density lipoprotein cholesterol (LDL-C): 4.38 mmol/L, Serum Creatinine: 118 μmol/L, Serum Potassium: 2.75 mmol/L, and Serum Chloride: 113.5 mmol/L. Considering the presence of severe proteinuria, hypoproteinemia, and hyperlipidemia, a diagnosis of nephrotic syndrome was established. Furthermore, blood gas analysis revealed the following: pH: 7.34, Standard residual base: –4.0, Actual base excess: –3.8, Standard bicarbonate: 20 mmol/L, Actual bicarbonate: 20.8 mmol/L, Potassium: 2.57 mmol/L, and Chloride: 109 mmol/L. Moreover, urine routine analysis showed Urine protein: 4+, pH: 7.0, and Erythrocytes: 24.71/HP.

Immunological and Hematological Analysis

Autoantibody profiling revealed: Anti-nuclear antibody (ANA): 1:320, Anti-Sjögren's-syndrome-related antigen A (anti-SSA): Positive, Anti-RO-52 antibody: Positive, Anti-Sjögren's-syndrome-related antigen B (anti-SSB): Positive, Anti-smith antibody (anti-SM): Negative, and Anti-double-stranded DNA (anti-dsDNA): Negative. Furthermore, immunoglobulins and complement levels were as follows: Immunoglobulin G (IgG): 8.75 g/L, Immunoglobulin A (IgA): 2.19 g/L, Immunoglobulin M (IgM): 1.91 g/L, Complement C3: 0.74 g/L, and Complement C4: 0.22 g/L.

The increased anti-nuclear antibody (ANA) titer and positive SSA, SSB, and Ro-52 antibodies, were suggestive of Sjögren's syndrome, warranting further clinical investigation. Furthermore, hematological indicators were as follows: Leukocyte count: 2.99×10^9/L, Red blood cell count: 3.45×10^{12}/L, and Hemoglobin: 107.0 g/L. Inflammatory markers revealed Rheumatoid factor (RF): 28.6 IU/mL and Erythrocyte sedimentation rate (ESR): 44 mm/h, and other key findings included a negative serum PLA2R.

Lip Gland Biopsy

The patient also complained about ocular and oral dryness, warranting further assessment. Immunofluorescence analysis for ANA was positive, with positive SS-A (anti-SSA), SS-B (anti-SSB), and RO-52 antibodies. A salivary gland biopsy revealed chronic sialadenitis with peri-acinar lymphocytic infiltration, yielding a focus score greater than 1. This score indicates the presence of at least 50 mononuclear inflammatory cells per 4 mm^2 of glandular section (Fig. 1). Based on the 2016 American College of Rheumatology/European League Against Rheumatism (ACR/EULAR) Classification Criteria for Sjögren's Syndrome, the patient meets the histological diagnostic criteria for SS, as confirmed by an experienced pathologist (Franceschini et al, 2017).

Renal Biopsy: Histopathological Examination

Light microscopy showed glomerular sclerosis in one out of twelve glomeruli. The remaining glomeruli exhibited diffuse thickening of the basement membrane with spike and bubbling formations and mild mesangial cell proliferation. The renal interstitium demonstrated multifocal fibrosis affecting more than 25% of the area, accompanied by moderate inflammatory infiltration, primarily including lymphocytes, plasma cells, and monocytes (Fig. 2A).

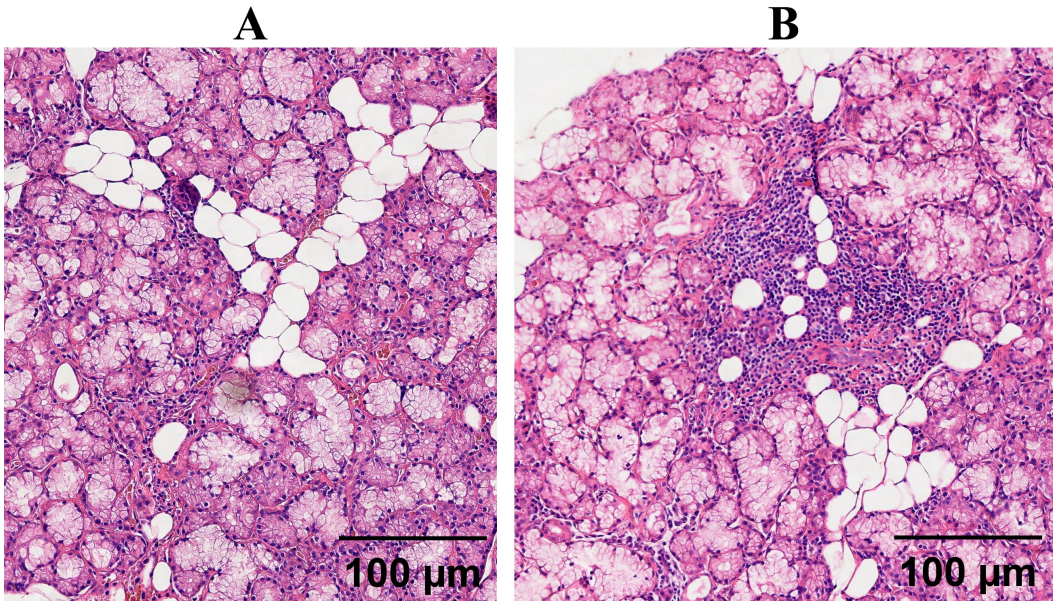

Fig. 1. Salivary gland lesions in the patient. (A) Peri-acinar lymphocytic infiltration was observed in hematoxylin-eosin staining (HE, 200×). (B) Another area of lymphocytic infiltration around a dilated acinus (HE, 200×).

The immunohistochemistry showed that both cluster of differentiation 3 (CD3) and CD20 were multifocally positive. The number of CD20-positive B cells exceeded the CD3-postive T cells in some areas, indicating a higher presence of B cells than T cells (Fig. 2C,D). The immunofluorescence (IF) staining revealed that IgA, IgG, and IgM were positive along the glomerular basement membrane (GBM) and the mesangial area. IgG subclass analysis showed positivity for both IgG1 and IgG4 (Fig. 2B). Furthermore, C3 was positive, while complement component 1q (C1q) was lacking. Similarly, PLA2R staining demonstrated granular positivity along the GBM with a 2+ intensity.

Electron microscopy showed GBM thickening with diffuse subepithelial granular electron-dense deposits. Segmental mesangial hyperplasia and granular deposits were observed. Moreover, diffuse effacement of foot processes was also found (Fig. 2E).

Renal tissue mass spectrometry identified PLA2R as a predominant antigen, with high spectral counts (67), confirming PLA2R-associated MN.

Final pathological diagnoses were consistent with PLA2R-associated MN and chronic interstitial nephritis, likely secondary to SS.

Treatment and Clinical Follow-Up Outcomes

A middle-aged male presented with acute-onset systemic edema, hypertension, massive proteinuria, and hypoproteinemia, confirming a diagnosis of nephrotic syndrome. Furthermore, Sjögren's syndrome was diagnosed based on dry mouth and dry eye symptoms, seropositivity for specific autoantibodies, and a positive lip gland biopsy. Renal biopsy findings were consistent with MN (stage I–II) and moderate chronic renal tubulointerstitial lesions. CD3+ and CD20+ immunostaining in renal tissue suggested secondary lesions associated with SS-related tubulointersti-

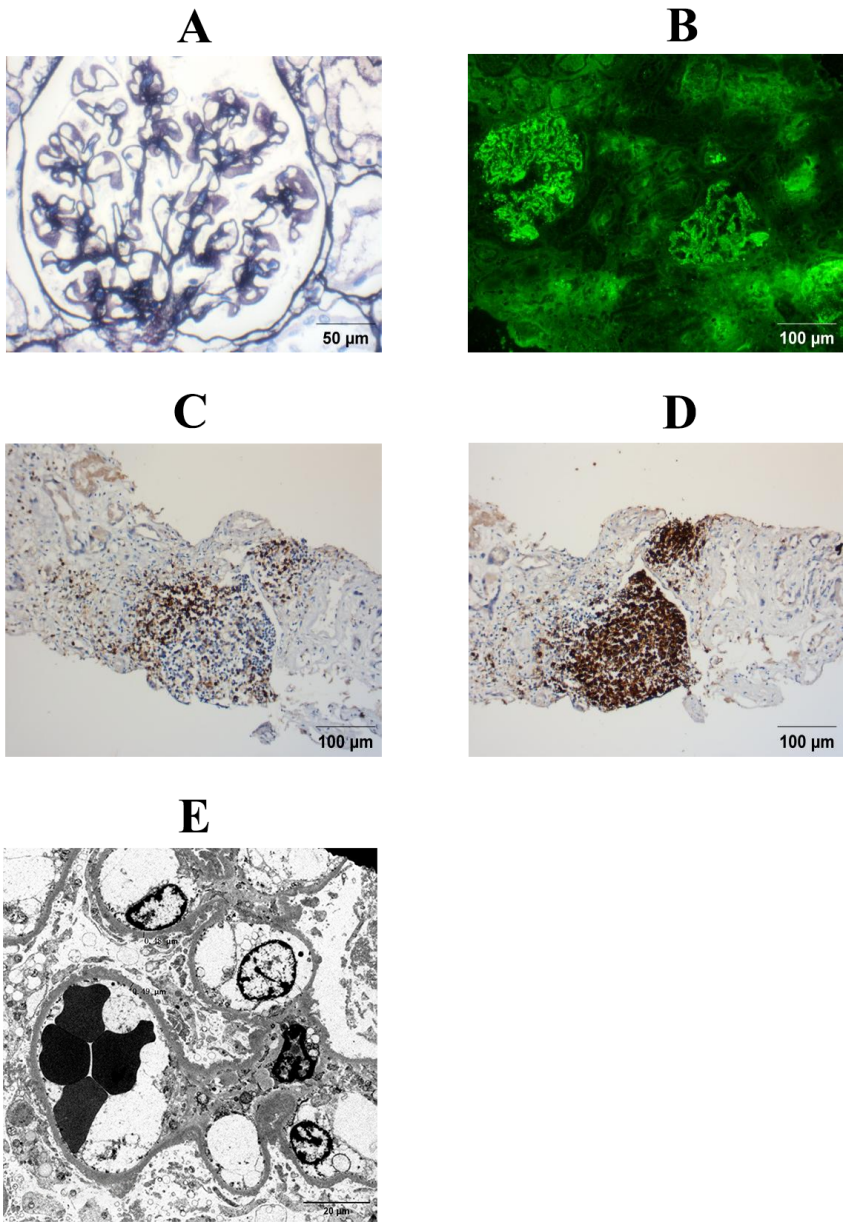

Fig. 2. Renal Pathology of the Patient. (A) Diffuse thickening of the basement membrane with spike and bubbling formations and mild mesangial cell proliferation (Periodic acid-methenamine silver (PAM) staining, 400×). (B) Immunoglobulin G (IgG) was granularly positive along the glomerular basement membrane (GBM) (Immunofluorescence (IF): staining, 200×). (C) Cluster of differentiation 3 (CD3) showed focal positive (Immunohistochemistry (IHC): 200×). (D) CD20 showed more positive cells than CD3 (IHC, 200×). (E) Diffuse thickening of GBM with subepithelial deposits and diffuse foot process effacement (Electron microscope (EM): 2000×).

tial nephritis. The patient started on methylprednisolone at a dose of 40 mg/day (body weight 76 kg), with gradual reduction based on renal function. The dose was reduced 8 weeks after initiation, with 10% reductions from the initial dose until it was discontinued. Furthermore, the patient received intravenous cyclophosphamide at a dose of 0.8 g/month as an immunosuppressive therapy, with a cumulative dose of 6.4 g. Additionally, the patient received supportive treatment, such as calcium supplementation, gastric protection, and blood pressure control. The

overall treatment regimen aimed to manage renal disease progression and systemic complications associated with SS and MN.

After 6 months of follow-up, the patient showed substantial clinical improvement. Laboratory examination revealed that plasma albumin increased to 34 g/L, blood creatinine decreased to 98 μmol/L, and the 24-hour urinary protein excretion reduced to 640 mg. Clinically, the patient experienced partial remission, and renal tubular acidosis was successfully resolved.

Clinical Considerations

The possibility of MN secondary to SS could not be excluded based on clinical results. The negative serum PLA2R result was consistent with the typical characteristics of secondary MN. Immunofluorescence analysis of the patient's renal biopsy revealed IgG1 and IgG4 expression, but without a predominant expression of IgG1, a pattern usually noticed in secondary MN. However, positive PLA2R in the renal tissue conflicted with secondary MN diagnosis. Given the conflicting clinical and pathological findings, renal tissue mass spectrometry was performed to identify potential antigens associated with MN. The results of this analysis are presented in Table 1.

Table 1. Renal tissue mass spectrometry (dominant expression of specific antibodies associated with membranous nephropathy).

Item	Description	PSMs	MSRRB17511	Protein FDR confidence
1	Secretory phospholipase A2 receptor (PLA2R)	67	782.598301	High
2	Early endosome antigen 1 (GEN1)	23	83.57356927	High
3	Serine protease HTRA1	3	11.06758406	High
4	Low-density lipoprotein receptor-related protein 2 (LRP2)	9	7.103855536	High
5	Protein NDNF	2	6.872050461	Low
6	EGF-containing fibulin-like extracellular matrix protein 2 (EFEMP2)	2	6.767388713	High
7	Thrombospondin type-1 domain-containing protein 7A (THSD7A)	1	6.376155582	Medium
8	Macrophage mannose receptor 1 (TSP-1)	1	6.196741203	Low
9	Insulin-degrading enzyme (IDE)	5	4.519606512	High
10	Reversion-inducing cysteine-rich protein with Kazal motifs (RECK)	1	2.872293394	Medium
11	Extracellular sulfatase Sulf-1	3	1.687265733	Medium

Supplementary Diagnostic Approach: A comprehensive mass spectrometry analysis of renal tissue was performed to detect MN-related antibodies, including Exostosin 1, Exostosin 2, TGFBR3, NCAM1, and others. Mass Spectrometry Findings: The proteomic expression of these antigens was weak, and the findings were not robust enough to be included in the data table. NDNF, neuron derived neurotrophic factor; HTRA1, high-temperature requirement A serine peptidase 1; PSMs, Physical Self-Maintenance Scale; FDR, false discovery rate; TGFBR3, Transforming Growth Factor Beta Receptor 3; NCAM1, Neural Cell Adhesion Molecule 1.

Discussion

MN is traditionally divided into two main categories: primary MN (~70%) and secondary MN (~30%). The primary MN occurs without any identifiable systemic disease, while the secondary MN type is associated with underlying systemic diseases (Couser, 2017; Glassock, 1991; Glassock, 1992; Ronco et al, 2021). These conditions include, but are not limited to, autoimmune diseases, infections, neoplasia, hematopoietic stem cell transplantation, sarcoidosis, and exposure to drugs or toxins. Secondary MN can occur as a result of well-defined autoimmune diseases, such as systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), urticarial vasculitis, sarcoidosis, SS, systemic sclerosis or ankylosing spondylitis (Jara et al, 2009; Makino et al, 2002; Mok, 2009; Rosato et al, 2018; Shanmugam and Steen, 2012; Strobel and Fritschka, 1998; Tonsawan et al, 2019; Vinicki et al, 2015). Among these diseases, SS is a well-recognized but relatively rare cause of secondary MN. Despite its low prevalence, it should be considered in the differential diagnosis of MN, mainly when clinical features of SS are observed.

PLA2R was first identified in 2009 and has since become the most characteristic target antigen in MN (Beck et al, 2009). Using Western blot immunoassay, a 185 kDa protein band was identified in human glomerular extract, which was found to be expressed in 70% of patients with primary MN but not in those with secondary MN. Mass spectrometry analysis confirmed this protein M-type PLA2R (Beck et al, 2009).

Anti-PLA2R reactivity is primarily carried by IgG4, although other IgG subclasses may also be involved. In some cases, PLA2R positivity in kidney tissue by negativity in serum may be due to the "sinking effect", a phenomenon observed in 15% of primary MN cases (Couser, 2017). A study focusing on MN secondary to SS reported PLA2R positivity in only 1 out of 6 patients (Larsen et al, 2013). PLA2R-positive MN associated with SS has been reported in only a few cases (Hari Krishna Reddy et al, 2018), making it more challenging to determine whether a patient has primary MN with SS or secondary MN caused by SS.

Tubulointerstitial nephritis (TIN) and renal tubular acidosis (RTA) are common manifestations of pSS-associated MN (Bossini et al, 2001; Maripuri et al, 2009). Research indicates that approximately 75% of pSS patients undergoing renal biopsy demonstrate TIN (Maripuri et al, 2009; Ren et al, 2008). In this case report, the patient exhibited moderate chronic tubulointerstitial disease, consistent with findings of pSS-associated renal involvement. Glomerular diseases associated with pSS is relatively rare. When glomerular involvement occurs, it frequently manifests as membranoproliferative glomerulonephritis (MPGN). Other glomerular diseases associated with pSS include minimal change disease, IgA nephropathy, membranous nephropathy, fibrillary glomerulonephritis (GN), and vasculitis (Bossini et al, 2001; Goules et al, 2019; Kidder et al, 2015). While these glomerular diseases may be part of the pSS disease spectrum, the possibility of coexisting glomerular lesions unrelated to pSS should be considered.

Initially, we proposed a monistic explanation, attributing the MN and tubulointerstitial lesions to SS. However, several findings challenged this hypothesis:

• High PLA2R expression in renal tissue is typically associated with primary MN.

• Negative serum immune-related antibodies are often observed in secondary MN.

• Immunofluorescence indicates balanced IgG1 and IgG4 expression, which is not typical for primary MN.

These findings suggest a more complex pathophysiological scenario where tubulointerstitial lesions could be secondary to SS, while MN might be a distinct primary pathology.

With the identification of new target antigens, our understanding of MN continues to advance. In 2023, the Mayo Clinic Consensus introduced a revised classification system for MN (Sethi et al, 2023), impacting the diagnostic approach to the disease. Based on this updated classification, mass spectrometry was utilized to analyze related antibodies for further characterization in this case. Given that PLA2R is positive in this case, the distinction between primary MN and secondary MN associated with SS remains uncertain. To address this uncertainty, a comprehensive examination of all autoimmune markers outlined in the Mayo Clinic's diagnostic criteria was conducted.

In the evaluation of this case, several antigens commonly associated with autoimmune diseases were not detected:

• Exostosin 1/Exostosin 2: These antigens are the most commonly detected proteins linked to autoimmune diseases (Sethi, 2021). They were not found in the present case.

• Transforming Growth Factor Beta Receptor 3 (TGFBR3): Reported in nearly 6% of patients with membranous lupus nephritis (Caza et al, 2021a), this antigen was not identified in the current case.

• Neural Cell Adhesion Molecule 1 (NCAM1): Typically found in a subset of MN patients with membranous lupus nephritis and other autoimmune-related diseases (Caza et al, 2021b), NCAM1 expression was not found in this case.

The absence of these antigens in the renal tissue analysis does not support a diagnosis of MN associated with the autoimmune diseases typically linked to these markers. This finding is essential for differential diagnosis and may guide further investigation into the etiology of the patient's MN. NCAM1 is detected in 1–2% of MN cases and approximately 6% of membranous lupus nephritis cases. However, no NCAM1 expression was observed in current cases.

Conclusion

When MN coexist with primary SS, establishing a correlation between these two conditions is essential. The presence of PLA2R in serum and kidney tissue serves as an effective diagnostic tool. However, a comprehensive assessment should include the detection of other specific antigens.

While it is more likely that PLA2R-positive MN coexisting with an autoimmune disease represents a coincidental occurrence of both conditions, this possibility does not eliminate the need for a thorough investigation. The possibility of

PLA2R-negative secondary MN remains crucial and warrants further exploration by the global medical community. Understanding this distinction is essential for fully characterizing MN and its associations with autoimmune diseases.

Key Points

- The patient has Sjögren's syndrome, and serum PLA2R antibodies are negative, yet renal-tissue PLA2R staining is positive, raising the key question of whether the membranous nephropathy is primary or secondary.
- When the positive expression of PLA2R in renal tissue conflicts with the diagnosis of secondary MN, and the immunofluorescence of renal tissue shows a balanced expression of IgG1 and IgG4, making it unclear whether MN is primary or secondary, renal tissue mass spectrometry for the detection of related antigens can be an option.
- In some cases, PLA2R positivity in renal tissues that are negative in serum may be due to the "sinking effect", a phenomenon observed in some cases of primary MN.
- This case offers the possibility of PLA2R-negative secondary MN, which is worthy of further exploration.

Availability of Data and Materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Author Contributions

CLL designed the research study and wrote the first draft. XLT, YJZ and XQX performed the research. KH, XPF and PXH analyzed the data. XJW and BF collected the data of this work. XJW and BF made significant guidance and supervision. All authors contributed to important editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

This study complies with the relevant principles and regulations of the Declaration of Helsinki, and the study was reviewed and approved by Jiaxing Hospital of Traditional Chinese Medicine (Ethics number: JXTCM-IRB-2025-007). The patient signed the informed consent.

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Conflict of Interest

The authors declare no conflict of interest.

Supplementary Material

Supplementary material associated with this article can be found, in the online version, at https://www.magonlinelibrary.com/doi/suppl/10.12968/hmed.2025.0132.

References

Beck LH, Jr, Bonegio RGB, Lambeau G, Beck DM, Powell DW, Cummins TD, et al. M-type phospholipase A2 receptor as target antigen in idiopathic membranous nephropathy. The New England Journal of Medicine. 2009; 361: 11–21. https://doi.org/10.1056/NEJMoa0810457

Bossini N, Savoldi S, Franceschini F, Mombelloni S, Baronio M, Cavazzana I, et al. Clinical and morphological features of kidney involvement in primary Sjögren's syndrome. Nephrology, Dialysis, Transplantation. 2001; 16: 2328–2336. https://doi.org/10.1093/ndt/16.12.2328

Caza TN, Hassen SI, Kenan DJ, Storey A, Arthur JM, Herzog C, et al. Transforming Growth Factor Beta Receptor 3 (TGFBR3)-Associated Membranous Nephropathy. Kidney360. 2021a; 2: 1275–1286. https://doi.org/10.34067/KID.0001492021

Caza TN, Hassen SI, Kuperman M, Sharma SG, Dvanajscak Z, Arthur J, et al. Neural cell adhesion molecule 1 is a novel autoantigen in membranous lupus nephritis. Kidney International. 2021b; 100: 171–181. https://doi.org/10.1016/j.kint.2020.09.016

Chen R, Wang J, Xie Q, Xue J, Hao C. Sjögren's syndrome complicated with membranous nephropathy, a cause or coincidence? International Journal of Rheumatic Diseases. 2021; 24: 1086–1094. https://doi.org/10.1111/1756-185X.14168

Couser WG. Primary Membranous Nephropathy. Clinical Journal of the American Society of Nephrology. 2017; 12: 983–997. https://doi.org/10.2215/CJN.11761116

Evans R, Zdebik A, Ciurtin C, Walsh SB. Renal involvement in primary Sjögren's syndrome. Rheumatology. 2015; 54: 1541–1548. https://doi.org/10.1093/rheumatology/kev223

Franceschini F, Cavazzana I, Andreoli L, Tincani A. The 2016 classification criteria for primary Sjogren's syndrome: what's new? BMC Medicine. 2017; 15: 69. https://doi.org/10.1186/s12916-017-0837-1

Glassock RJ. Secondary membranous glomerulonephritis. Nephrology, Dialysis, Transplantation. 1992; 7: 64–71.

Glassock RJ. The therapy of idiopathic membranous glomerulonephritis. Seminars in Nephrology. 1991; 11: 138–147.

Goules A, Geetha D, Arend LJ, Baer AN. Renal involvement in primary Sjögren's syndrome: natural history and treatment outcome. Clinical and Experimental Rheumatology. 2019; 37: 123–132.

Hari Krishna Reddy M, Kumar AC, Sangeetha B, Ram R, Siva Kumar V. Anti-PLA2R antibody in a secondary cause of membranous nephropathy. Nephrology. 2018; 23: 94–95. https://doi.org/10.1111/nep.12997

Jara LJ, Navarro C, Medina G, Vera-Lastra O, Saavedra MA. Hypocomplementemic urticarial vasculitis syndrome. Current Rheumatology Reports. 2009; 11: 410–415. https://doi.org/10.1007/s11926-009-0060-y

Keri KC, Blumenthal S, Kulkarni V, Beck L, Chongkrairatanakul T. Primary membranous nephropathy: comprehensive review and historical perspective. Postgraduate Medical Journal. 2019; 95: 23–31. https://doi.org/10.1136/postgradmedj-2018-135729

Kidder D, Rutherford E, Kipgen D, Fleming S, Geddes C, Stewart GA. Kidney biopsy findings in primary Sjögren syndrome. Nephrology, Dialysis, Transplantation. 2015; 30: 1363–1369. https://doi.org/10.1093/ndt/gfv042

Larsen CP, Messias NC, Silva FG, Messias E, Walker PD. Determination of primary versus secondary membranous glomerulopathy utilizing phospholipase A2 receptor staining in renal biopsies. Modern Pathology. 2013; 26: 709–715. https://doi.org/10.1038/modpathol.2012.207

Luo J, Xu S, Lv Y, Huang X, Zhang H, Zhu X, et al. Clinical features and potential relevant factors of renal involvement in primary Sjögren's syndrome. International Journal of Rheumatic Diseases. 2019; 22: 182–190. https://doi.org/10.1111/1756-185X.13429

Makino H, Yoshinaga Y, Yamasaki Y, Morita Y, Hashimoto H, Yamamura M. Renal involvement in rheumatoid arthritis: analysis of renal biopsy specimens from 100 patients. Modern Rheumatology. 2002; 12: 148–154. https://doi.org/10.3109/s101650200025

Maripuri S, Grande JP, Osborn TG, Fervenza FC, Matteson EL, Donadio JV, et al. Renal involvement in primary Sjögren's syndrome: a clinicopathologic study. Clinical Journal of the American Society of Nephrology. 2009; 4: 1423–1431. https://doi.org/10.2215/CJN.00980209

Mok CC. Membranous nephropathy in systemic lupus erythematosus: a therapeutic enigma. Nature Reviews. Nephrology. 2009; 5: 212–220. https://doi.org/10.1038/nrneph.2009.14

Ohtani H, Imai H, Kodama T, Hamai K, Komatsuda A, Wakui H, et al. Severe hypokalaemia and respiratory arrest due to renal tubular acidosis in a patient with Sjögren syndrome. Nephrology, Dialysis, Transplantation. 1999; 14: 2201–2203. https://doi.org/10.1093/ndt/14.9.2201

Pan Y, Cui Z, Wang S, Zheng D, Deng Z, Tian X, et al. Idiopathic multicentric Castleman disease with Sjögren's syndrome and secondary membranous nephropathy: a case report and review of the literature. BMC Nephrology. 2020; 21: 528. https://doi.org/10.1186/s12882-020-02191-z

Patel R, Shahane A. The epidemiology of Sjögren's syndrome. Clinical Epidemiology. 2014; 6: 247–255. https://doi.org/10.2147/CLEP.S47399

Ren H, Wang WM, Chen XN, Zhang W, Pan XX, Wang XL, et al. Renal involvement and followup of 130 patients with primary Sjögren's syndrome. The Journal of Rheumatology. 2008; 35: 278–284.

Ronco P, Beck L, Debiec H, Fervenza FC, Hou FF, Jha V, et al. Membranous nephropathy. Nature Reviews. Disease Primers. 2021; 7: 69. https://doi.org/10.1038/s41572-021-00303-z

Rosato E, Gigante A, Barbano B, Gasperini ML, Cianci R, Muscaritoli M. Prognostic Factors of Renal Involvement in Systemic Sclerosis. Kidney & Blood Pressure Research. 2018; 43: 682–689. https://doi.org/10.1159/000489740

Ruggenenti P, Remuzzi G. A First Step toward a New Approach to Treating Membranous Nephropathy. The New England Journal of Medicine. 2019; 381: 86–88. https://doi.org/10.1056/NEJMe1906666

Sethi S, Beck LH, Jr, Glassock RJ, Haas M, De Vriese AS, Caza TN, et al. Mayo Clinic consensus report on membranous nephropathy: proposal for a novel classification. Kidney International. 2023; 104: 1092–1102. https://doi.org/10.1016/j.kint.2023.06.032

Sethi S. New 'Antigens' in Membranous Nephropathy. Journal of the American Society of Nephrology. 2021; 32: 268–278. https://doi.org/10.1681/ASN.2020071082

Shanmugam VK, Steen VD. Renal disease in scleroderma: an update on evaluation, risk stratification, pathogenesis and management. Current Opinion in Rheumatology. 2012; 24: 669–676. https://doi.org/10.1097/BOR.0b013e3283588dcf

Strobel ES, Fritschka E. Renal diseases in ankylosing spondylitis: review of the literature illustrated by case reports. Clinical Rheumatology. 1998; 17: 524–530. https://doi.org/10.1007/BF01451293

Tonsawan P, Talabthong K, Puapairoj A, Foocharoen C. Renal pathology and clinical associations in systemic sclerosis: a historical cohort study. International Journal of General Medicine. 2019; 12: 323–331. https://doi.org/10.2147/IJGM.S221471

Vinicki JP, Pellet SC, De Rosa G, Dubinsky D, Laborde HA, Marini A, et al. Analysis of 65 Renal Biopsies From Patients With Rheumatoid Arthritis (1976–2015): Change in Treatment Strategies Decreased Frequency and Modified Histopathological Findings. Journal of Clinical Rheumatology. 2015; 21: 335–340. https://doi.org/10.1097/RHU.0000000000000302

Yang HX, Wang J, Wen YB, Fei YY, Jiang MD, Zhou MY, et al. Renal involvement in primary Sjögren's syndrome: A retrospective study of 103 biopsy-proven cases from a single center in China. International Journal of Rheumatic Diseases. 2018; 21: 223–229. https://doi.org/10.1111/1756-185X.13182