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Chronic Kidney Disease Secondary to Sjögren's Syndrome Associated Membranoproliferative Glomerulonephritis in A Male Patient: A Case Report

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Abstract

Sjögren's syndrome (SS) is a chronic autoimmune disease that occurs primarily in women, with a female-to-male ratio of 9:1. We report a case of chronic kidney disease (CKD) associated with SS in a male patient, diagnosed according to the 2016 ACR/EULAR criteria, with positive SSA/RO antibodies and a Schirmer test result of < 5 mm/5 min. Renal biopsy revealed 18 glomeruli. Pathological findings included focal and segmental endocapillary proliferation (involving 3 glomeruli), a nonspecific lymphocytic tubular infiltrate (sometimes nodular) and fibroproliferative endarteritis. Immunofluorescence revealed deposits of IgA, IgG, C3, kappa, and lambda chains. These features were consistent with membranoproliferative glomerulonephritis (MPGN). A treatment regimen consisting of prednisone and mycophenolate mofetil (MMF) led to an improvement in glomerular filtration rate (GFR). This case highlights the possibility of severe glomerular involvement such as MPGN, in the course of primary SS, even in men.

Keywords

Sjögren's Syndrome, Chronic Kidney Disease, Membranoproliferative Glomerulonephritis, Tengandogo University Hospital, Burkina Faso

1. Introduction

Sjögren's syndrome (SS) is an autoimmune exocrine disorder characterized by dry mouth and eyes, resulting from a chronic and progressive loss of secretory function in the salivary and lacrimal glands. This pathology affects nine women for every man, and its prevalence was estimated between 2 and 4 million people in the United States in 2004 [1]. In addition to glandular manifestations, SS can affect other organs, particularly the kidneys. Renal involvement in primary SS is predominantly tubular, while glomerular involvement is rarer. In primary SS, the types of glomerulonephritis encountered are predominantly membranoproliferative glomerulonephritis, membranous nephropathy, and IgA nephropathy [2] [3]. We report a case of membranoproliferative glomerulonephritis associated with primary SS that occurred in a male patient.

2. Case Presentation

A 33-year-old man presented to the nephrology clinic at Tengandogo University Hospital in January 2025 for evaluation of worsening renal failure (Serum creatinine of 193 µmol/L, eGFR using CKD-EPI formula = 40 ml/min/1.73m^2) and proteinuria. Symptoms began eight months prior with dry mouth, photophobia, and inflammatory polyarthralgia. His past medical history was notable for hemoglobin AC (sickle cell trait) and hypertension diagnosed three months ago which has been treated with losartan (50 mg/day). Notably, his renal function had declined since September 2024, when his serum creatinine was 161 µmol/L, corresponding to an estimated GFR of 54.88 ml/min/1.73 m^2.

Physical examination showed conjunctival pallor. There was no peripheral edema and no joint deformities. Blood pressure was 132/75 mmHg, with a pulse rate of 63 beats per minute. The patient's weight was 60 kg and his height was 1.75 m, resulting in a body mass index (BMI) of 19.59 kg/m^2.

Laboratory tests confirmed renal insufficiency with a serum creatinine of 272.5 µmol/L (eGFR using the CKD-EPI formula: 29.1 mL/min). Serum calcium and phosphorus levels were normal, at 2.25 mmol/L and 1.07 mmol/L, respectively. C-reactive protein and fasting venous blood glucose were normal, at 1.99 mg/L and 5.1 mmol/L, respectively. The complete blood count revealed microcytic anemia at hemoglobin (9.5 g/dL), with a mean corpuscular volume (MCV) of 77.5 fl, and a mean corpuscular hemoglobin (MCH) of 26.5 pg. White blood cell and platelet counts were normal, at 4,750 cells/mm^3 and 218,000 cells/mm^3, respectively. Urinalysis revealed nephrotic range proteinuria at 3.2 g/24 hours. Urinary sediment examination showed significant dysmorphic hematuria accompanied by red blood cell and granular casts. Serum protein electrophoresis showed normal total protein (70.2 g/L), hypoalbuminemia (35.5 g/L), and polyclonal hypergammaglobulinemia (26.57 g/L). Immunological workup was strongly positive for antinuclear antibodies (ANA > 55 IU/mL), anti-SSA/Ro (>241 IU/mL), and anti-SSB/La (>18 IU/mL). Native anti-DNA antibodies (<10 IU/mL), viral serologies (for human immunodeficiency virus (HIV), hepatitis B and C viruses), and syphilis were

negative. Complement levels (C3: 0.91 g/L and C4: 0.24 g/L) were within normal limits. Serum cryoglobulins were not measured due to local technical constraints. Renal ultrasound showed normal-size kidneys (110 mm) with well-defined borders and normal echogenicity.

An ophthalmological consultation revealed dry eye syndrome with a Schirmer test result of < 5 mm/5 minutes.

Based on the 2016 ACR/EULAR classification criteria, the patient scored a total of 4 points (3 points for positive SSA/RO antibodies and 1 point for the Schirmer's test result of < 5 mm/5 minutes).

A renal biopsy was performed, yielding 18 glomeruli. Light microscopy revealed highly proliferative and chronic glomerular changes. A segmentally and focally distributed endocapillary proliferation was observed. Chronic architectural changes were prominent, featuring flocculo-capsular synechiae and the formation of pseudotubules. Notably, there was evidence of an old fibrinoid necrosis of the glomerular basement membrane. The tubulointerstitial compartment showed severe tubular atrophy. The tubules contained hyaline casts and Tamm-Horsfall proteins, which were associated with intracytoplasmic hyaline droplets. The interstitium had been infiltrated by a non-specific, occasionally nodular lymphocytic infiltrate. The intrarenal vasculature exhibited severe sclerotic and inflammatory changes, characterized by fibro-proliferative endarteritis and arteriolosclerosis, alongside morphological signs of old thrombotic microangiopathy (TMA).

Immunofluorescence microscopy demonstrated significant immune complex-mediated disease. There was a predominance of immunoglobulin A (IgA), immunoglobulin G (IgG), and complement C3 deposits trapped within the glomerular structures.

Electron microscopy was unavailable at the institution due to resource limitations. These findings were highly suggestive of immune-complex mediated membranoproliferative glomerulonephritis (MPGN) secondary to primary SS.

The patient was treated with induction therapy consisting of three pulses of intravenous methylprednisolone (500 mg daily for 3 days), followed by oral maintenance with prednisone (60 mg/day for twelve weeks then gradually tapered over a 6-month period). Mycophenolate mofetil (MMF) was initiated at a dose of 2 g daily in 2 divided doses for 3 months. Adjuvant therapy included calcium and vitamin D supplementation, omeprazole, continued losartan, oral Iron and Erythropoietin. Following the initiation of immunosuppressive therapy, the patient showed significant clinical and biological improvement. Serum creatinine decreased to 167 μmol/L (eGFR: 53.6 mL/min/1.73 m^2) and proteinuria decreased to 1.23 g/24 hours. Therefore, anemia persisted. **Figure 1** shows the trends in serum creatinine levels and estimated glomerular filtration rate (eGFR). The patient remains clinically stable at 12 months follow-up. The current treatment includes: Calcium + Vitamin D 500 mg/250 IU (1 tablet daily), Losartan 50 mg (1 tablet daily), Polymaltose iron hydroxide complex 100 mg (1 tablet daily), and Epoetin alfa (4000 IU twice weekly) by subcutaneous injection. Written informed consent was ob-

tained from the patient for the publication of this case report.

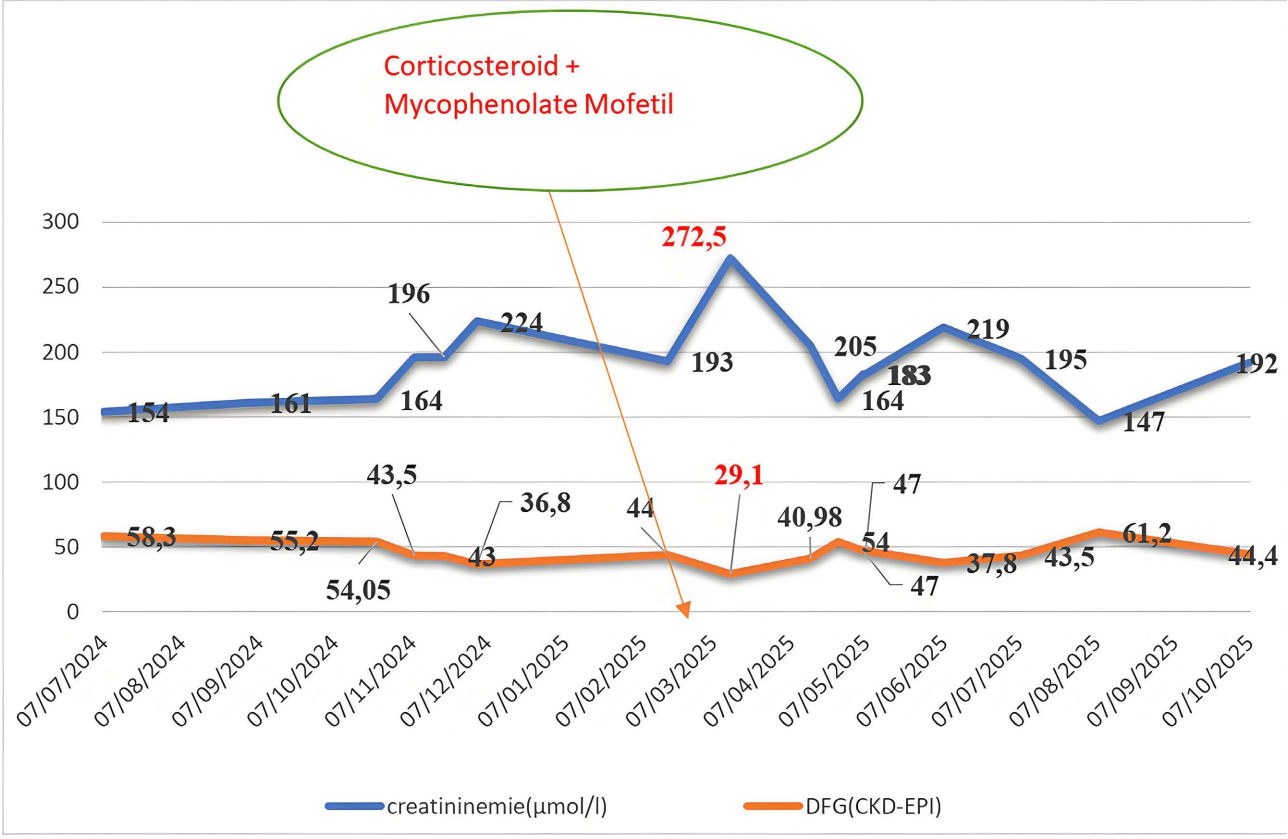

Figure 1. Changes in serum creatinine levels and eGFR before and after treatment.

3. Discussion

Our case presents a male patient with primary SS according to the 2016 ACR/EULAR criteria [4] complicated by concomitant MPGN. While SS is significantly more common in women, several studies have shown that in men, the disease is often diagnosed earlier and is associated with significantly higher positivity for ANA, SSB/La, and SSA/Ro antibodies. According to Sepúlveda JIR *et al.*, these sex-based differences may be explained by the X chromosome and sex hormones [5]. Furthermore, in 2020, Park Y *et al* noted that SS in men carries a higher risk of pulmonary involvement and lymphoma [6]. In addition to glandular involvement, other organs such as the kidneys may also be affected by this condition.

The prevalence of renal involvement in primary SS varies widely (1% to 50%) due to the variability of diagnostic criteria, and geographic differences, with higher rates reported in Asian studies compared to European cohorts [7] [8]. While tubulo-interstitial lesions account for approximately 70% of cases, glomerular lesions including membranoproliferative glomerulonephritis, cryoglobulinemic vasculitis, membranous nephropathy and segmental and focal hyalinosis occur in about 30%. Thus, primary SS is considered the leading cause of non-hepatitis C related cryoglobulinemia [9]. In 2019, Goules *et al.* reported three cases of

MPGN among five patients with SS associated glomerulonephritis. All three cases presented with cryoglobulinemia and C4 complement consumption [10]. This report highlights an atypical renal presentation of pSS characterized by a MPGN histological pattern but completely normal serum C3 and C4 levels. Classically pSS-associated MPGN is driven by mixed cryoglobulinemia, which aggressively consumes complement via the classical pathway, leaving a footprint of profound hypocomplementemia (especially near-zero C4). The normal complement levels observed here represent a significant clinical variance. This profile can be explained pathophysiologically by dynamic complement fluctuations during periods of systemic quiescence, or by an upregulated hepatic production that fully compensates for localized, low grade renal consumption. Alternatively, the structural "tram-track" glomerular basement membrane remodeling may stem from non-cryoprecipitable immune complexes driven by chronic polyclonal B-cell activation, or a chronic endothelial injury pattern from localized microvascular vasculitis mimicking an MPGN phenotype. A major diagnostic challenge and study limitation was the inability to screen the serum cryoglobulins due to institutional resource constraints. Other classic causes of immune-complex-mediated GN have been ruled out using a targeted diagnostic panel. SLE was excluded by negative anti-dsDNA and anti-Sm antibodies, alongside the absence of extra-renal lupus criteria. Infection-related-glomerulonephritis and chronic infections were ruled out through negative serology for Hepatitis B (Ag HBS), hepatitis C (Ac HCV), and HIV, combined with no signs of bacterial endocarditis. Absence of serum and urinary monoclonal protein spike ruled out monoclonal gammopathy of renal significance. By eliminating these pathways, the glomerular lesion could be confidently attributed to intense polyclonal B-cell activation and autoimmune framework of the patient's established pSS. Additionally, we found no data in the literature linking Sickle cell trait (hemoglobin AC) to this specific glomerular pattern.

The pathophysiology of renal involvement in SS mirrors that of glandular involvement. Indeed, environmental factors and genetic susceptibility stimulate the production of interferons and other cytokines, which contribute to polyclonal activation of B lymphocytes. This results in the production of gamma globulins and autoantibodies that bind to antigens on epithelial cells (glandular and others), causing tissue damage and clinical manifestations. Tubulointerstitial involvement is thought to result from the infiltration of T and B lymphocytes, plasma cells, and more rarely, autoantibodies. While the mechanism of glomerulonephritis, is poorly understood, the deposition of immune complexes on the glomerular membrane appears to be the most plausible explanation [8].

The management of SS -associated glomerulonephritis depends on the histological findings. Commonly used agents include corticosteroids combined with immunosuppressants such as cyclophosphamide, mycophenolate mofetil (MMF), rituximab, azathioprine, and even plasma exchange. A treatment regimen consisting of prednisone and MMF, with or without rituximab, would be a reasonable therapeutic option for cases of glomerulonephritis in SS, as it offers a benefit in terms of renal function [10]. Indeed, Kidder *et al.* in 2015 demonstrated that treat-

ment with prednisolone and MMF, with or without rituximab, in patients with SS associated MPGN was beneficial, resulting in either an improvement or stabilization of their glomerular filtration rate (GFR) [11]. In our case, due to limited access to rituximab, the combination of steroids and MMF was utilized, resulting in a favorable renal outcome.

4. Conclusion

This case highlights the possibility of severe glomerular involvement, especially MPGN, in primary SS, even in a male patient. A renal biopsy is essential for definitive diagnosis and therapeutic guidance. Early initiation of immunosuppressive therapy combining corticosteroids and MMF can significantly improve the renal prognosis.

Author Contributions

Conception and study design: Kissou P. François, Manuscript drafting: Kissou P. François, Lengani YH. Aida and Sawadogo Amidou. Manuscript revision: Kissou P. François, Lengani YH. Aida, Sawadogo Amidou and Tchoupé D Marius. All the authors have read and agreed to the manuscript.

Conflicts of Interest

There is no conflict interest in this study.

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