Membranous Nephropathy in a Treatment Naïve Patient of Rheumatoid Arthritis

Dear Editor,

A 45-year-old woman presented with a one-month history of generalized body swelling Over the preceding month, she had gained 8 kg in weight, experienced reduced urine output, and reported easy fatigability. The patient also disclosed a two-year history of inflammatory polyarthritis affecting small joints in her hands and wrists, with no prior treatment. Clinically, she was afebrile, with normal vitals and her weight was noted to be 92 kg.

Evaluation revealed normocytic normochromic anemia (hemoglobin 9.3 g/dL), low serum albumin (1.2 g/dL), elevated serum cholesterol (333 mg/dL), hypertriglyceridemia (311 mg/dL), serum creatinine within normal limits (1.0 mg/dL), and an estimated glomerular filtration rate (eGFR) of 71 ml/min/1.73 m^2. Urine analysis showed proteinuria (3+ on dipstick, 8 g/day on the 24-hour collection). Chest X-ray displayed blunting of bilateral costophrenic angles. While an ultrasound of the kidneys indicated normal size, her renal biopsy confirmed membranous nephropathy (MN).

Notably, the patient's anti-cyclic citrullinated peptide antibody (Anti-CCP Ab) was highly positive (>500 U/ml), leading to a definite diagnosis of rheumatoid arthritis (RA) based on the 2010 ACR/EULAR criteria. Management involved diuretics, parenteral albumin, ACE inhibitors, steroids, anticoagulants, and septran prophylaxis. A percutaneous renal biopsy [Figure 1] confirmed MN, anti-PLA2R antibody was negative, and subsequent treatment with the anti-CD20 monoclonal antibody Rituximab was initiated. Conventional disease-modifying antirheumatic drugs (DMARDs) such as Methotrexate and Hydroxychloroquine were prescribed for RA.

The patient responded well to the treatment (Rituximab and Methotrexate, Hydroxychloroquine), experiencing resolution of anasarca and albuminuria. Ongoing follow-up revealed sustained remission for both MN and RA.

MN is a significant cause of nephrotic syndrome in adults, comprising about 30% of cases, with 80% being primary (pMN) and 20% secondary (sMN).[1] While the patient's history and workup excluded causes of pMN, sMN, the positive response to RA treatment indicated a rare case of MN secondary to RA.

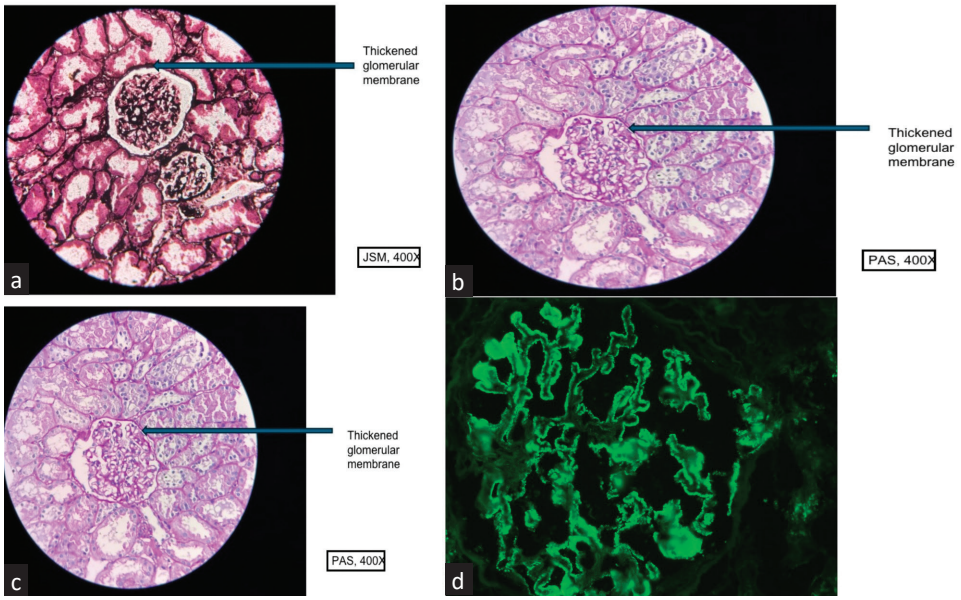

Figure 1: (a and b) show thickening and stiffening of the glomerular capillary loop (400x, PAS and 400x JSM). (c) shows in addition to the thickened loops, there were pockets of lymphomononuclear inflammatory cell infiltrate in the cortical parenchyma and chronic mild inflammation (400x, PAS). (d) Direct immunofluorescence showed coarse granular deposits for anti sera specific against IgG (3+), Kappa (3+), Lambda (3+), C3 (1+) in the sub epithelial aspect of glomerular capillary loop. Rest of the panel IgA, IgM, C1q was negative, PAS: periodic acid schiff.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

Conflicts of interest

There are no conflicts of interest.

Satish Kumar[1], Santosh Kumar Singh[2], Rahil Arora[2], Ahmed Waheed Kashif[3]

[1]Department of Internal Medicine, Military Hospital, Yol Cantt, Yol, Departments of [2]Internal Medicine and [3]Pathology, Armed Forces Medical College, Wanowrie, Pune, India.

Corresponding author:
Rahil Arora, Department of Internal Medicine, Armed Forces Medical College, Wanowrie, Pune, India. E-mail: rahilarora1992@gmail.com

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Collagenofibrotic Glomerulopathy—An Unexpected Finding in a Young Female

Dear Editor,

Collagenofibrotic glomerulopathy (CG) is a rare kidney disease, seldom suspected clinically. A 31-year-old lady was referred for the evaluation of hypertension and proteinuria, which she had developed in the third trimester of her second pregnancy and persisted for eight months postdelivery. Her first pregnancy was uneventful. Appropriate patient consent was taken.

On evaluation, she had proteinuria without haematuria or pyuria. The urine protein creatinine ratio was 2.52, serum creatinine was 0.57 mg/dl, and her kidneys looked normal on ultrasound examination. Serological examination for HIV, HBV, and HCV were negative. Evaluation for connective tissue disorders was not contributory. Hence, a kidney biopsy was performed with a spring-loaded gun under real-time ultrasound guidance for a definitive diagnosis.

On light microscopy, all the sampled glomeruli were viable, enlarged with diffuse mesangial widening which stained weakly positive by PAS, pale blue on Masson stain, and was negative with Jones silver methenamine stain. No immune deposits on immunofluorescence. The first impression was that of non-light chain amyloid, yet Congo red was negative [Figure 1]. Electron microscopy revealed haphazardly arranged curvilinear frayed bundles of banded collagen showing distinct periodicity and a diameter of 29.15 nm present in the subendothelial region and the mesangium, suggesting collagen fibrils and Type III collagen was confirmed

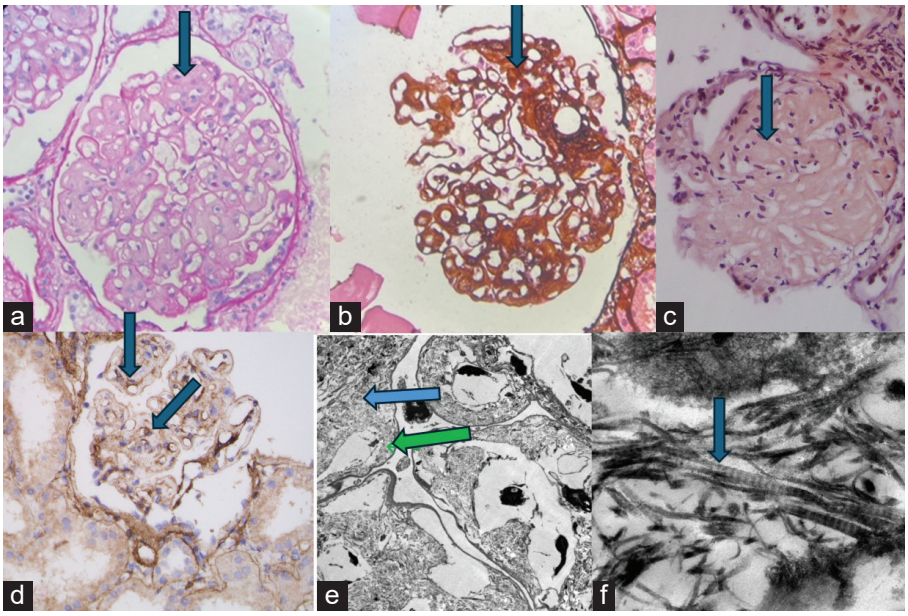

Figure 1: (a) PAS negative widened mesangium (400x, blue arrow), (b) Silver negative widened mesangium, (400x, blue arrow). (c) Congo red negative widened mesangium (400x, blue arrow), (d) Type III collagen IHC, blue arrows marking peripheral and mesangial location of IHC stain. (e) Electronmicroscopy, blue arrow pointing mesangial location, green arrow pointing subendothelial location of fibrils. Original magnification 600x. (f) Electronmicroscopy, Curvilinear banded fibrils, 29.15 nm, original magnification 10000x, blue arrow.