Case Report

Tuberculosis as a Microbiologically Proven Etiology of Membranous Nephropathy and Interstitial Nephritis

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ABSTRACT. Secondary causes of membranous glomerulonephritis (GN) include systemic lupus erythematosus, other autoimmune diseases, neoplasms, and infections such as hepatitis B and C viruses, syphilis, and parasites. The association of tuberculosis (TB) with membranous GN is rare. We report the first case of microbiologically proven tubercular interstitial nephritis and membranous nephropathy (MN) occurring concurrently in the same patient. The patient improved with the use of antitubercular therapy alone. TB should be recognized as a potentially treatable infectious cause of secondary MN.

Introduction

Glomerulonephritic lesions seldom have known primary factors. Membranous nephropathy (MN) is uncommonly secondary to known infectious, autoimmune or neoplastic lesions. No cases of microbiologically proven tubercular etiology are, however, on record. We describe a case of MN and interstitial nephritis concomitant with the presence of tubercle bacilli in renal parenchyma and its subsequent resolution following antitubercular therapy.

Case Report

A 32-year old Indian male presented with generalized edema for six months. There was no history of fever or cough. No history of diabetes or hypertension was present. His serum creatinine was 0.80 g/dL, and serum albumin was 3.04 g/dL. Urine examination revealed proteinuria of 9 g/day and 5–8 red blood cells per high-power field. He was non-reactive for human immunodeficiency virus and hepatitis B and C viruses. A renal biopsy revealed 10 glomeruli with diffusely thickened capillary walls. The interstitium was infiltrated by collections of epithelioid histiocytes, chronic inflammatory cells, and foci of necrosis. Immunofluorescence showed coarse granular deposits of immunoglobulin (Ig) IgG (+) and C3 (+) along the glomerular basement membrane. In view of the necrotizing interstitial
nephritis, a Ziehl–Neelson staining was done which revealed numerous acid-fast tubercle bacilli.

In view of tuberculosis (TB) detected incidentally on renal biopsy, a workup for systemic TB was done. Chest X-ray did not reveal any lesions suggestive of active pulmonary TB. Sputum culture was negative for tubercle bacilli. Computed tomogram of the abdomen showed a hypodense mass lesion in the middle third of the left kidney (Figure 1). A diagnosis of MN and interstitial nephritis with renal TB was made. The patient was treated with antitubercular therapy as per standard WHO regimen for extrapulmonary TB. He was also treated with angiotensin-converting enzyme inhibitor (ramipril). Steroids or cytotoxic agents were not added. On a follow-up visit, four months after biopsy and starting antitubercular therapy, the patient showed positive response to treatment, and his proteinuria settled to 0.15 g in 24 h along with resolution of hematuria.

Informed consent was obtained from the patient before presenting the report.

Discussion

We report the first case in the English literature of microbiologically proven renal TB and MN occurring concurrently in the same patient. TB can affect the kidney as a manifestation of disseminated miliary TB or as localized urinary tract infection as was true in the present case.\(^1\) Patients with localized renal TB usually have cavitory lesions in unilateral kidneys. Such lesions are clinically silent, with preservation of renal function. Localized renal lesions are believed to be a sequel of remote pulmonary TB in immunocompetent patients.\(^2\) Such lesions show the presence of well-formed epithelioid cell granulomas on histology, similar to the ones seen in the present case.

The occurrence of glomerulonephritis (GN) in association with TB is extremely rare. Solak et al in a review of the English literature identified 15 cases of GN associated with TB. Of these, IgA nephropathy was noted in six; crescentic GN in four; mesangioproliferative
in two; and collapsing, mesangiocapillary, and membranous GN (MGN) in one case each. The case of MGN in this review occurred in a patient with lumbar TB, but tubercle bacilli could not be identified in the renal biopsy or urine culture. In a case reported from India, a 23-year-old female presented with MN and granulomatous interstitial nephritis. Although no acid-fast bacilli could be detected in her renal biopsy, she showed an ulcerated tuberculin test and responded to the use of antitubercular therapy. A case of renal TB with MN and nephrotic syndrome (NS) has also been reported in the Japanese literature. In the present case, the identification of acid-fast bacilli in the biopsy lend credence to the tubercular etiology of the membranous lesions. The etiologic link is further strengthened by the amelioration of symptoms and proteinuria with treatment by antitubercular drugs alone. Recognized secondary causes of MGN include systemic lupus erythematosus, other autoimmune diseases, neoplasms, and infections such as hepatitis B and C viruses, syphilis, and malaria. TB should also find place in this list as a potentially treatable cause of secondary MGN. It can be surmised in the current case that a silent renal TB became manifest by the onset of MGN and resultant NS.

**Conclusion**

TB is a potentially treatable infectious cause of secondary MN. Antitubercular therapy alone aided in improving the outcome in the current case. Immunosuppression without the use of antitubercular therapy would be detrimental in such cases. Appreciation of tubercular etiology and institution of antitubercular therapy will greatly influence the prognosis of such cases. A misdiagnosis of such a case as idiopathic MGN can worsen the prognosis if immunosuppression alone is used without antitubercular therapy.

**Conflict of interest:** None declared.

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