A Case of Minimal Change Disease Treated Successfully with Mycophenolate Mofetil in a Patient with Systemic Lupus Erythematosus

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The World Health Organization classifies lupus nephritis as class I to V or VI. However, a few cases of minimal change glomerulopathy have been reported in association with systemic lupus erythematosus (SLE). Mycophenolate mofetil has been shown to be effective for treatment of minimal change disease and lupus nephritis. A 24-year-old woman diagnosed with SLE five years prior to presentation complained of a mild generalized edema. The urinalysis showed microscopic hematuria and proteinuria. The assessed amount of total proteinuria was 1,618 mg/24 hours. A renal biopsy demonstrated diffuse fusion of the foot processes of podocytes on electron microscopy. Mycophenolate mofetil was started in addition to the maintenance medications of prednisolone 10 mg/day and hydroxychloroquine 400 mg/day. After six months of treatment, the microscopic hematuria and proteinuria resolved, and the total urine protein decreased to 100 mg/24 hours.

Keywords: Nephrosis, lipoid; Lupus erythematosus, systemic; Mycophenolate mofetil

INTRODUCTION

Mycophenolate mofetil (MMF) has been reported to be effective in the treatment of minimal change disease and lupus nephritis. However, there is no consensus on its use for minimal change disease especially in systemic lupus erythematosus (SLE).

CASE REPORT

A 24-year-old woman diagnosed with SLE five years previously and maintained on prednisolone 5 mg/day and hydroxychloroquine 400 mg/day complained of mild generalized edema. At the time of the diagnosis of SLE, the patient was referred to the emergency room for cough, sputum production and bruising. Severe thrombocytopenia was associated with oral ulcerations, arthritis, high titer speckled fluorescent antinuclear antibody test (FANA), ds-DNA Ab, anti-Smith Ab, positive Venereal Disease Research Laboratory (VDRL) and normal bone marrow cellularity and maturity. These abnormalities normalized after treatment with intravenous immunoglobulin (IVIG) 400 mg/kg for five days, prednisolone 60 mg/day and hydroxychloroquine 400 mg/day.

In order to evaluate the newly developed generalized edema, the patient had a 24 hours urine test for total protein and a duplex scan for renal vascular patency and blood flow. The white blood cell (WBC) count was 4,800/
μL, Hgb 13 g/dL, platelet 279,000/μL, aspartate aminotransferase/alanine aminotransferase (AST/ALT) 44/15 U/L, and blood urea nitrogen/creatinine (BUN/Cr) 9.8/0.6 mg/dL on blood testing. The urinalysis demonstrated 2 (++) proteinuria on albustick, and the total urine protein was 1,618 mg/24 hr. The duplex scan showed patent renal vasculature and normal flow.

To evaluate the glomerular proteinuria in this patient with SLE a renal biopsy was performed. On light microscopy, no abnormalities were found; immunofluorescent staining showed normal findings (Fig. 1). However, electron microscopy revealed diffuse fusion and microvilli formation of the foot processes, no electron dense material in the mesangium, electrolucent and absorptive features of epimembranous deposits and tubulo-reticular bodies in the endothelial cell cytoplasm (Fig. 2). These findings were compatible with a podocytopathy. With no other possible causes for the glomerular podocytopathy the patient was diagnosed as minimal change disease associated with active SLE presenting with glomerular proteinuria and generalized edema.

To control minimal change disease, high-dose glucocorticoid therapy remains the mainstay. But because the patient had experienced glucocorticoid-related adverse effects such as severe edema, weight gain, gastro-intestinal

Figure 1. Kidney biopsy. No specific pathology was found on light microscopy (Periodic acid-Schiff stain, ×200).

Figure 2. Kidney biopsy. The electron microscopy showed diffuse fusion of the foot processes, microvilli formation of foot processes, no electron dense material in the mesangium, electrolucent and absorptive features of epimembranous deposits and tubulo-reticular bodies in the endothelial cell cytoplasm (Uranyl acetate and lead citrate double stain; A: × 2,000; B, C: × 5,000).