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CASE REPORT

An unusual cause of focal segmental glomerulosclerosis: psoriasis vulgaris

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ABSTRACT

Psoriasis, being limited to the skin, is generally a chronic inflammatory disorder. Several glomerular diseases have been distinguished due to renal histological findings of psoriatic patients to date. The underlying pathogenetic mechanisms of these associations remain unclear because of the limited number of cases. We report a second case of focal segmental glomerulosclerosis in a psoriatic patient.

Keywords: focal segmental glomerulosclerosis; psoriasis vulgaris; glomerulonephritis; proteinuria; nephrotic syndrome

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INTRODUCTION

Psoriasis is a chronic skin disorder that is occasionally associated with arthritis and inflammatory bowel disease.[1] Generally, the psoriatic process is limited to the skin; however, internal organs such as the kidneys may be involved in the course. It may be rarely complicated by the occurrence of nephrotic syndrome and hematuria secondary to immunoglobulin A (IgA) nephropathy, focal segmental glomerulosclerosis (FSGS), membranous glomerulonephritis (MGN), membranoproliferative glomerulonephritis (MPGN), and amyloidosis.[2–6] But the underlying pathogenetic mechanism of these associations remains unclear because of the limited number of cases. We described a case of FSGS in a psoriatic patient.

CASE REPORT

A 43-year-old female was referred to nephrology department for persistent proteinuria. She has been followed with a diagnosis of plaque psoriasis for 18 years. Her symptoms were under control with topical steroid preparations. She had no significant family history such as chronic diseases, hypertension, diabetes, and heart and liver diseases. Except for bilateral pitting pedal edema, physical findings of patient were normal. There were no erythrocyte, leukocyte, and cast on urine analysis. The laboratory examinations revealed the presence of proteinuria (1962 mg/day), normal renal function (creatinine 49.5 μmol/L, BUN 4.64 mmol/L), total serum protein 70 g/L, and serum albumin 37 g/L. Total cholesterol level was elevated at 6.86 mmol/L and triglyceride levels at 2.94 mmol/L. Liver function tests were normal. The anti-neutrophilic cytoplasmic antibody (ANCA), anti-nuclear antibody (ANA), anti-dsDNA, the serum complement, and the IgG, IgM, IgA values were within normal limits. Viral serologies for HIV and hepatitis B and C were negative. Sonogram of the kidney was normal.

Percutaneous kidney biopsy was performed due to persistent proteinuria. On light microscopy, focal and segmental glomerulosclerosis was described with adhesion bowman capsule in the affected segments, periglomerular fibrosis, and interstitial inflammatory infiltrates. Immunofluorescent microscopy only demonstrated positive fibrinogen in the segmental lesions but was negative for C3, C4, C1q, and immunoglobulin deposits. FSGS was diagnosed.

Kidney biopsy findings were compatible with FSGS due to dietary modifications; thereafter we started on immunosuppressive treatment with methylprednisolone (1 mg/kg/day), ramipril (2.5 mg/day), and atorvastatin (20 mg/day). Three months after the initiation of this treatment, she presented with proteinuria (1643 mg/day) and serum albumin (36 g/L), but no altered renal function. In view of late steroid resistance, she

took therapy with cyclosporine A (CsA) (2 mg/kg/day) in addition to methylprednisolone. While she was taking CsA therapy, she did not tolerate because of adverse gastrointestinal effects. She was switched to azathioprine therapy (2 mg/kg/day) after 2 months of initiation. The dosage of methylprednisolone was tapered to 4 mg/day over the next 4 months. At follow-up, while having no improvement about proteinuria, kidney function was normal at the end of the first year of treatment.

DISCUSSION

Psoriasis is a common inflammatory and proliferative disorder of the skin, characterized by sharply, demarcated dull-red scaly plaques.[3] Although the etiology of psoriasis is not certain, evidences suggest a primary T-lymphocyte-based immunopathogenesis. In this condition, up-regulation of proinflammatory cytokines and reduced levels of the anti-inflammatory cytokine interleukin-10 (IL-10) and IL-4 occur; also dominance of T-helper 1 cytokines, such as interferon-gamma and IL-2, increases. In addition, there is a possible linkage of the psoriasis-susceptibility gene *PSORS2* with a gene involved in the regulation of IL-2.[1] Similarly, there is considerable evidence that cell-mediated immunity plays an important role in the pathogenesis of minimal change disease and FSGS. Therefore, the resolution of psoriatic skin lesions and remission of proteinuria following CsA therapy supports the hypothesis of defective cell-mediated immunity in both disorders. As is well known, the mechanism of action of CsA involves blocking translocation of the cytosolic component of nuclear factor of activated T cells (NFAT) and inhibiting transcription of multiple cytokines which are the products of cell-mediated immunity.[7] We used CsA for treatment but she did not tolerate the therapy because of adverse gastrointestinal effects.

Patients with psoriasis might show that it has varying degrees of glomerular involvement, presenting with microalbuminuria, hematuria, and nephrotic syndrome and deteriorating renal functions.[8] The occurrence of patients with glomerular diseases affected by psoriasis has been rare despite the fact that the number of documents has been increasing over recent years. IgA nephropathy, usually presenting with sub-nephrotic proteinuria, is the most frequent glomerulonephritis reported in association with psoriasis.[3,9] The pathogenesis of psoriasis may be raleted to disregulation of the IgA system like development of IgA nephropathy. This disregulation may be lead to elevation of serum IgA levels or IgA containing immune complexes, or an intirinsic defect in the structure of IgA due to genetically predisposed, leading to IgA deposition in the mesangium, may be present in psoriasis.[3]

MGN, which is another form of recorded glomerulonephritis association with psoriasis, is considered to be a complication of drugs (NSAIDs, gold salts).[4] Moreover, psoriasis has been rarely documented between the association of mesangiocapillary glomerulonephritis, MPGN, amyloidosis, and minimal change disease.[5,6,10,11]

According to our on-line research on PubMed, we found only one case which was reported by Sirolli et al. Our patient is a second case of FSGS with psoriasis who has an ongoing sub-nephrotic proteinuria despite immunosuppressive therapy. As like the other, she has no obvious predisposing cause for FSGS such as a history of pyelonephritis, vesicoureteral reflux, HIV, hepatitis B, or obesity and excluded a secondary FSGS. Although an immune mechanism is probable, the potential pathogenic significance of rare association between psoriasis and FSGS remains unknown. A circulating "permeability factor," possibly a lymphokine or cytokine, or abnormalities of soluble immune mediators have been suggested as potential etiologic factors in primary FSGS.[3]

In conclusion, the clinical appearance of glomerular involvement may be seen more commonly in psoriasis, but there is little data which were confirmed by renal biopsies in literature. Renal biopsy should be performed when the glomerular involvement is considered in early course of follow-up with abnormal urinalysis. Because of this, annually urinalysis should be done for early diagnosis of glomerular lesions in psoriasis.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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