Membranous Glomerulonephropathy Associated with Psoriasis vulgaris

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Dear Sir,

Many pathological conditions are linked to membranous glomerulonephropathy (MGN). Torres and Donadio [1] described dermatological diseases such as bullous pemphigoid and dermatitis herpetiformis associated with MGN, but there are few detailed descriptions. We have reported on a rare case of MGN associated with psoriasis vulgaris (PV). To our knowledge, this is the second case of MGN in a patient with PV [2]. This case might throw light on the relation between MGN and PV.

A 50-year-old man was admitted to a nearby hospital because of systemic edema and itching on his back in April 1988. Physical examination on admission revealed no obvious eruption on his back. Laboratory data were as follows: proteinuria 2 g/day and serum total protein (TP) 5.6 g/dl. A diagnosis of MGN was made by renal biopsy and he was treated with diuretics and antiplatelet drugs as an outpatient. Six months later, he developed an eruption on his back with itching as well as persistent edema and received an initial daily dose of 20 mg of prednisolone (PSL) with gradual tapering because of a good effect on skin lesions. Ten months later, however, in October 1990 when 7.5 mg of PSL were given daily, the nephrotic syndrome (NS) worsened with resulting deterioration of the renal function from 0.91 mg/dl in serum creatinine levels to 1.73 mg/dl, accompanied by the development of red colored papulae with marked scale which had spread over his whole skin. The diagnosis of PV was made by a dermatologist at the other hospital. He was transferred to our hospital for the treatment of PV (fig. 1) and NS on March 22, 1991. Laboratory data on admission were as follows: proteinuria 4 g/day, TP 4.9 g/dl, albumin 2.5 g/dl, creatinine 2.18 mg/dl, total cholesterol 415 mg/dl, IgG 990 mg/dl, IgA 239 mg/dl and IgM 153 mg/dl. The rheumatoid factor, antinuclear antibody and anti-DNA antibody were all negative. No hypocomplementemia was observed. Circulating immune complexes were not measured.

Renal biopsy showed thickening of glomerular capillary walls. An immunofluorescence study showed deposits of IgG, IgA and C3 along the glomerular capillary walls (fig. 2). Electron microscopy showed stage III which is characterized by numerous in-tramembranous or subepithelial deposits encircled by newly formed glomerular basement membrane-like materials (fig. 3).

A diagnosis of MGN associated with PV was made. A 4-week course of oral PSL (60 mg/day) with subsequent cyclophosphamide administration was started and urinary protein excretion gradually decreased to 2-3 g/day with an increase in serum albumin levels to 3.2 g/dl and skin lesions completely resolved. Mild skin lesions recurred when PSL was decreased to 10 mg/day. About 3 years later, in February 1994 when he received 12.5 mg/day of PSL, mild skin lesions were observed and proteinuria was 2-3 g/day, TP 5.3 g/dl, serum albumin 3.1 g/dl and serum creatinine 3.25 mg/dl.

Fig. 1. Skin lesions on admission.

An immune mechanism is considered to be involved in the pathogenesis of both PV [3] and MGN [4]. Immunological examinations revealed high serum IgA and IgG [5] and defective suppressor T cell function [6]

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Fig. 2. Granular deposits of IgG along the glomerular capillary walls. Fig. 3. Numerous intramembranous or subepithelial deposits encircled by newly formed glomerular basement membrane-like materials are seen. × 5,000. in PV. The association of PV with autoimmune diseases such as systemic lupus erythematosus (SLE) was reported [7]. Since secondary causes of MGN include autoimmune diseases such as SLE and rheumatoid arthritis, the same immune mechanism may be involved in the pathogenetic association of PV with MGN. In our case, NS with a resulting deterioration of renal function appears to have run parallel to the development of PV and oral administration of PSL improved the skin lesions, followed by a decrease in proteinuria and attenuation of renal deterioration. These results suggested the possible involvement of the same immune abnormality in the pathogenesis of these two diseases.

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