IgA Nephropathy Complicated by Ulcerative Colitis

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Dear Sir,

IgA nephropathy [1] has been found to coexist with extrarenal diseases in an undetermined number of patients. The association of IgA nephropathy with ankylosing spondylitis, dermatitis herpetiformis, scleritis and gastrointestinal disease has been reported. Ulcerative colitis is an inflammatory bowel disease known to have many systemic complications occurring in the skin, joints, eyes and vascular system. Recently, the coexistence of glomerulonephritis with ulcerative colitis has been reported in several cases [2, 3]. We present here a case report of ulcerative colitis which developed in a patient with IgA nephropathy.

A 21-year-old man was referred to our hospital because of persistent microscopic hematuria and proteinuria in October 1985. He had an episode of sore throat, fever and gross hematuria in May 1985. His past history was noncontributory. Upon admission his temperature was 36.7 °C, pulse rate 84 beats/min and blood pressure 120/86 mm Hg. His throat was intact. The abdomen was flat and not tender, and his liver, spleen and kidneys were not palpable. His skin and joints were normal. There was no peripheral edema. Urinalysis showed a 1 + test results for protein amounting to 0.2–0.3 g over a 24-hour period. The sediment contained 15–20 red blood cells, a few red blood cell casts, granular casts and hyaline casts per high-power field. Blood urea nitrogen was 10 mg/100 ml, serum creatinine 1.0 mg/100 ml and creatinine clearance 95 ml/min. Serum IgA was 169 mg/100 ml, C3 68 mg/100 ml and C4 22.6 mg/100 ml. A percutaneous renal biopsy was performed on the 5th day of hospitalization. Light-microscopic examination of glomeruli showed mild mesangial proliferation with slight hypercellularity. Immunofluorescent microscopy of glomeruli revealed granular deposits of IgA and C3 predominantly in the mesangium and IgG, IgM and C1q were negative. Electron microscopy revealed large and small electron-dense deposits in the mesangium and paramesangium. Capillary loops were patent with a normal basement membrane. He was diagnosed as having IgA nephropathy and clinical follow-up was performed without medication at our clinic. In June 1987, the patient began to experience lower abdominal pain, diarrhea and bloody stool. Upon examination, the abdomen was soft and not distended, and tenderness was elicited in the lower part of the abdomen. He did not have joint pain, purpura or edema of the extremities. His urine was yellow and tested a 2 + for protein,

and the sediment contained 30–40 red blood cells and a few hyaline cast per high-power field. Blood urea nitrogen was 14 mg/100 ml and serum creatinine 1.1 mg/100 ml. Serum IgA was 202 mg/100 ml, CH₅₀ 35 U/ml and circulating immune complexes measured by Clq solid-phase assay were negative. There was no apparent change in urinalysis, renal function or immunological examination at the onset of gastrointestinal symptoms. A barium enema and endoscopic examination of the colon and rectum were performed. Multiple erosions, which were exudate and consisted of mucus, blood and pus were observed in the mucosa of the sigmoid colon and rectum. A biopsy of the colonic and rectal mucosa showed inflammatory changes and ulcerations consistent with ulcerative colitis. He was treated with salazosul-fapyridine and showed clinical improvement of ulcerative colitis.

The present case was found to have ulcerative colitis 2 years after the onset of IgA nephropathy. The systemic nature of IgA nephropathy has recently been emphasized, and it is considered that this disease should be regarded as a syndrome [4]. Similar glomerular changes are shown in Henoch-Schönlein purpura [5], lupus nephritis and hepatic cirrhosis [6]. In this patient, Henoch-

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Schönlein purpura was unlikely because of the lack of purpura, and lupus nephritis and hepatic cirrhosis were excluded based on clinical and laboratory findings. Ulcerative colitis is a chronic inflammatory and ulcerative disease of the colon and rectum clinically characterized by abdominal pain, diarrhea, bloody stool and weight loss. It is well known that ulcerative colitis can have many extraintestinal manifestations involving the skin, joints, eyes and vascular system. The association of glomerular diseases with ulcerative colitis is also reported. The study of pathologic conditions associated with IgA nephropathy by Makdassy et al. [2] demonstrated 1 case of ulcerative colitis out of 322 patients with IgA nephropathy. Although a clinical link between IgA nephropathy and ulcerative colitis was not apparent in the present case, it is speculated that IgA nephropathy could be pathogenetically related to ulcerative colitis via IgA class immune complexes originating in the diseased mucosa. The association between IgA nephropathy and ulcerative colitis may be supported by their common complications such as ankylosing spondylitis and scleritis known common to both diseases. Further studies are required to explore the association between IgA nephropathy and inflammatory bowel disease, and their pathogenetic mechanisms.

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