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Membranous Glomerulonephritis Associated With Ulcerative Colitis

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Glomerulonephritis is reported as a rare extraintestinal manifestation of inflammatory bowel disease. We report a case of a 69-year-old woman who suffered from membranous glomerulonephritis, 3 years after diagnosis of nephrotic syndrome. She was admitted because of acute kidney failure, bloody diarrhea, and gastrointestinal symptoms. Further evaluation confirmed the diagnosis of ulcerative colitis.

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Case Report

INTRODUCTION

Membranous glomerulonephritis (MGN), one of the most common types of idiopathic nephrotic syndrome in adults, is usually of unknown etiology. It may be associated with infections, neoplasm, rheumatoid disorders, and drugs consumption. In the medical literature, glomerulonephritis is reported as a rare extraintestinal manifestation of inflammatory bowel disease (IBD).[1-5] Herein, we report a unique case of accumulating IBD during MGN progression which was led to acute kidney failure.

CASE REPORT

A 69-year-old woman presented with bloody diarrhea, mucoid defecation, and confusion. She had been diagnosed with MGN 7 years earlier when referred with generalized edema, hypertension, and nephrotic-range proteinuria. During the therapeutic period, serum creatinine had increased to 3 mg/dL.

At presentation, she reported symptoms of bloody diarrhea 7 to 8 times per day, mucoid defecation, and a history of 3-month rectorrhagia. Hypovolemia was remarkable on physical examination and serum creatinine was elevated up to 6 mg/dL. Hypokalemia was also noted on laboratory studies. Therefore, urgent hemodialysis was done for the patient. Meanwhile, the patient underwent colonoscopy due to intestinal manifestations. Severe disseminated ulcerative proctitis was revealed in the rectal area on total colonoscopy. As a result, the patient received mesalamine, oral sulfasalazine, 3 g/d, and folic acid, 5 mg/d, for gastrointestinal disturbances. During the treatment of ulcerative colitis, acute kidney failure was controlled, the kidney function was alleviated, and proteinuria decreased. The results of laboratory tests were as follows: white blood cell count, 7 $\times 10^9$/L (neutrophils, 70% and lymphocytes, 30%); estimated sedimentation rate, 74 mm/h; blood hemoglobin, 8 g/dL; hematocit, 24%; platelet count, 300 $\times 10^9$/L; stool exam: many white blood cells, many red blood cells, and negative for parasites; urinalysis: 2 to 3 red blood cells per high-power field, 5 to 6 white blood cells per high-power field, and 4+ proteinuria; and negative urine culture for microorganisms.

On laboratory studies, serum levels of complement, antinuclear antibody, and anti-dsDNA were normal. Serum protein electrophoresis and virus serology did not show abnormal results.

Meanwhile, the small intestine transit was normal. Proteinurea was 4200 mg per 24 hours at first, which decreased to 979 mg after the treatment period. Serum creatinine level was 3 mg/dL simultaneous to diagnosis of MGN that increased to 6 mg/dL when ulcerative colitis presented. After control of ulcerative colitis, serum level of creatinine decreased to 3.4 mg/dL and became stable without hemodialysis.

In the specimen from kidney biopsy, increased disseminated thickness in the glomerular capillaries was observed with subepithelial deposits associated with spike presentation (Figure 1). There was no Goblet cell in the glandular epithelium taken from the rectum. Exocytose neutrophil and cryptus abscess was present. In addition, acute and chronic inflammatory cell infiltrations were seen in the lamina propria (Figure 2).

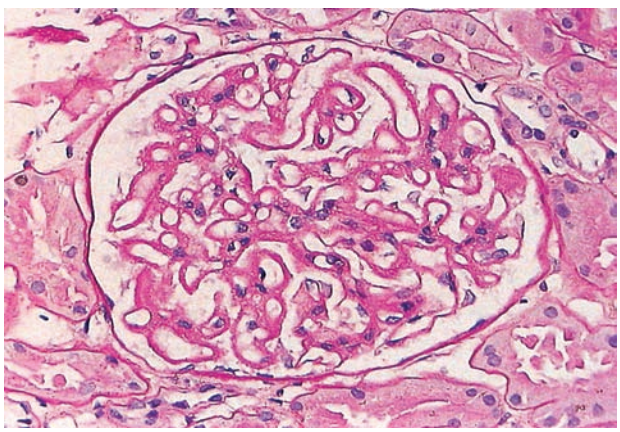

Figure 1. Pathologic examination of a kidney specimen showed disseminated thickness in the glomerular capillaries and subepithelial deposits associated with spike presentation.

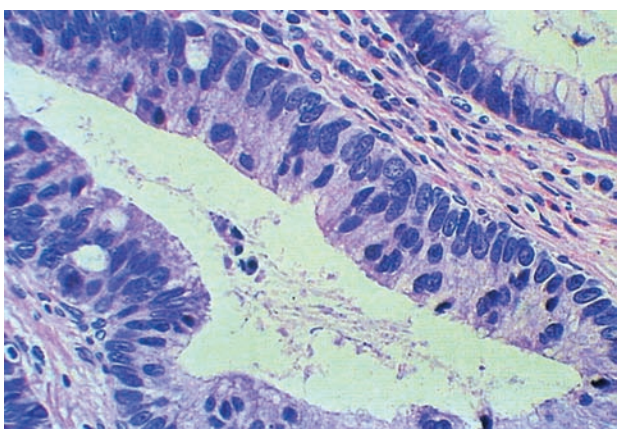

Figure 2. Pathologic examination of a specimen from the rectal mucosa was indicative of acute and chronic inflammatory cell infiltrations in the lamina propria.

During a 4-year follow-up period, 2 recurrent ulcerative colitis episodes were seen because of discontinuing of conservative treatment by the patient that was controlled by medical therapy.

DISCUSSION

While most of extraintestinal manifestations of IBD are present parallel to the activity of IBD, primary sclerosing cholangitis and ankylosing spondylitis may run a clinical course independent of the bowel disease.[6] Therefore, the possibility of presenting MGN, 3 years before ulcerative colitis as an extraintestinal manifestation of IBD is not considerable. Membranous glomerulonephritis is caused by immune complex deposit in the subepithelial area of the glomerular capillaries. It seems that ulcerative colitis has common etiologic factors with other immunity-based diseases such as MGN. Undoubtedly, diarrhea and rectorrhagia caused kidney dysfunction since during the therapeutic period, fluid therapy and blood transfusion were effective. Acute kidney failure due to acute tubular necrosis directed to kidney dysfunction, which was controlled by hemodialysis. Kidney function stability might be related to the control of ulcerative colitis, because after initiating the treatment of ulcerative colitis, kidney failure was controlled without the need for hemodialysis.

We recommend that in patient presenting MGN associated with intestinal manifestations, association of IBD with glomerulonephritis should be considered.

CONFLICT OF INTEREST

None declared.

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