CASE REPORT

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Patient with diffuse mesangial and endocapillary proliferative glomerulonephritis with hypocomplementemia and elevated anti-streptolysin O treated with prednisolone, angiotensin-converting enzyme inhibitor, and angiotensin II receptor antagonist

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Abstract A 24-year-old woman was admitted to Toyosaka Hospital with proteinuria, hematuria, lymphopenia, hypocomplementemia, positive anti-nuclear antibody (ANA), and elevation of anti-streptolysin O (ASO). Renal biopsy specimen revealed diffuse mesangial and endocapillary glomerulonephritis with crescent formation and duplication of the capillary loop on light microscopic examination. Mild to moderate proliferation of mesangial matrix and cells were observed. On immunofluorescence (IF) examination, deposition of IgG, IgA, IgM, C1q, C3, and C4 to the mesangium and capillary wall were observed. By electron microscopy (EM), mesangial, subendothelial, and subepithelial deposits were recognized. However, microtubular structure in glomerular endothelial cells, fingerprint structures, and circumferential mesangial interposition were not observed by EM. The patient was referred to our hospital, but there was no change in her proteinuria 3 weeks after admission. The elevation of ASO, hypocomplementemia, and endocapillary proliferation suggested acute glomerulonephritis, while lymphocytopenia, positive ANA, the persistent hypocomplementemia, and various deposits detected by IF and EM suggested lupus nephritis; however, she did not fulfill the classification criteria of systemic lupus erythematosus. We started prednisolone (40 mg/day) with the diagnosis of chronic glomerulonephritis revealing diffuse mesangial and endocapillary proliferative glomerulonephritis, but it was not effective for the proteinuria. Quinapril (10 mg/day) and losartan (25 to 50 mg/day) were administered and the proteinuria decreased. It is possible that this use of an angiotensin converting-enzyme inhibitor and an angiotensin II receptor antagonist was effective in reducing the proteinuria in this patient.

Key words Mesangial and endocapillary proliferative glomerulonephritis · Systemic lupus erythematosus · Anti-streptolysin O · Hypocomplementemia · Angiotensin-converting enzyme inhibitor · Angiotensin II receptor antagonist

Introduction

The prognosis of acute poststreptococcal glomerulonephritis (PSGN) is favorable, and steroid and cytotoxic agents have no confirmed role in the treatment of the condition and may be rather harmful.1 However, steroid therapy is necessary for the treatment of lupus nephritis.2 We experienced a patient showing endocapillary proliferative glomerulonephritis in whom it was difficult to differentiate between PSGN and lupus nephritis. We observed her without any treatment for 2 months, but there was no decrease in hematuria or proteinuria. Steroid therapy, followed by an angiotensin-converting enzyme inhibitor (ACEI) and an angiotensin II receptor antagonist (AT1a) was started. The renal pathology and the clinical course appeared to be unique.

Case report

In October 1999, a 24-year-old woman showed proteinuria when she underwent a physical check-up. She had a history
of tonsillectomy performed when she was 10 years old. She was referred to Toyosaka Hospital on November 10. Proteinuria (1.0 to 2.8 g/day), hematuria (urinary red blood cells [U-RBC], 5–10/high-power field [HPF]), lymphopenia (1338/mm³), hypocomplementemia (C3, 10 mg/dl; C4, 9 mg/dl; CH50, <12.0 U/ml), positive anti-nuclear antibody (ANA; measured by an immunofluorescence method [FANA], × 640), elevated anti-streptolysin O (ASO; 185 IU/ml; normal, under 165 IU/ml) were recorded.

She was admitted to Toyosaka Hospital on December 2. She denied a preceding history of sore throat or skin rash. She had not experienced dry eyes or dry mouth. The creatinine clearance rate (Ccr) was 83.5 ml per min. A renal biopsy was performed on December 7. On light microscopy (LM) examination, diffuse mesangial and endocapillary proliferation with focal fibrocellular or fibrous crescent formation and diffuse segmental or global duplication of the capillary loops was observed. Glomerular endocapillary proliferation consisted of an infiltration of polymorphonuclear cells and mononuclear cells (Fig. 1). On immunofluorescence (IF) microscopic examination, diffuse coarse granular deposition of immunoglobulin G (IgG; Fig. 2a), IgA, IgM, C1q (Fig. 2b), C3 (Fig. 2c), and C4 to the mesangium and capillary wall were noted. Mesangial, subendothelial, and subepithelial deposits, as well as deposits in the glomerular basement membrane (GBM) were seen by electron microscopy (EM; Fig. 3). The mesangial and paramesangial deposits were dominant. Circumferential mesangial interposition was not observed. Microtubular structure (MTS) in the glomerular endothelial cells, fingerprint structures, and dense deposits were not observed by EM. Subendothelial deposits did not include organized tubular structures or virus-like particles. The elevated ASO, hypocomplementemia, and various deposits detected by IF and EM suggested lupus nephritis.

She was referred to Niigata University Hospital on December 17 for the differential diagnosis of AGN and lupus nephritis. On admission, her height was 159 cm, body weight was 50 kg, body temperature was 37°C, and blood pressure was 124/74 mmHg. She did not have oral ulcers, hair loss, photosensitivity, joint pain, joint swelling, or rash. There was no pedal edema.

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Fig. 1. Light microscopic observation shows diffuse mesangial and endocapillary proliferative glomerulonephritis with focal segmental duplication of the capillary loop. Endocapillary proliferation reveals the infiltration of polymorphonuclear cells and mononuclear cells.

Fig. 2. a Strong diffuse coarse granular deposition of immunoglobulin G is observed by immunofluorescence microscopy (IF). b C1q deposition is weak. c C3 deposition is very strong.