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Pathology Page

Acute post-streptococcal glomerulonephritis

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A 16-year-old girl complained of rhinorrhea, oliguria, body weight gain, and generalized edema for 1 week.

Urinalysis showed occult blood (4+) with numerous dysmorphic red blood cells and protein (3+). Her blood urea nitrogen was 77 mg/dL and creatinine was 2.6 mg/dL. Serology showed that antistreptolysin O (ASLO) was 1:1280. She received a renal biopsy and histopathology showed hypercellular proliferation with infiltration of numerous neutrophils in all glomeruli (Fig. 1). An immunofluorescence stain showed immunoglobulin (Ig)A, IgG, and C3 granular deposition in the capillary walls and mesangial area (Fig. 1, inset). Post-streptococcus glomerulonephritis (PSGN) was diagnosed. After supportive treatment, she was discharged with regular follow-up at our hospital.

Acute PSGN is caused by glomerular deposition of immune complexes, resulting in proliferation of endothelial and mesangial cells and infiltration of neutrophils. The classic case of PSGN develops in a child 1–4 weeks after recovery from a group A streptococcal infection. The initial infection is localized to the pharynx or skin.

PSGN is an immune complex disease, in which tissue injury is primarily caused by complement activation by the classic pathway. Histopathology shows diffuse proliferation of endothelial and mesangial cells and infiltration of neutrophils and monocytes. Immunofluorescence studies reveal scattered granular deposits of IgG and complement within the capillary walls and some mesangial areas, corresponding to the deposits visualized on electron microscopy.

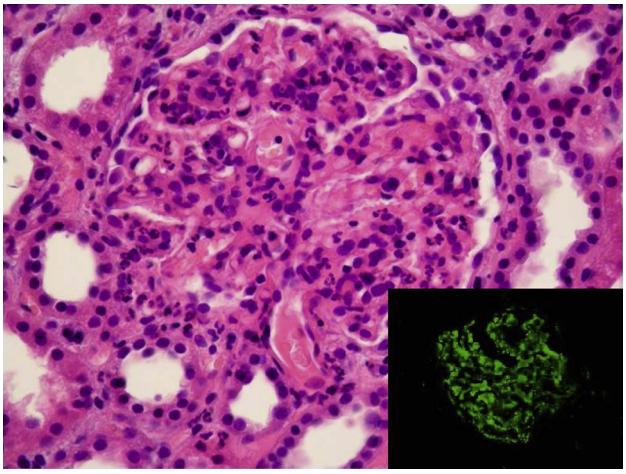

Fig. 1. Histopathology shows diffuse endothelial and mesangial cell proliferation and infiltration of numerous neutrophils (hematoxylin and eosin stain, 400×). Inset: immunofluorescence stain shows immunoglobulin G deposition in the capillary wall and mesangial area (fluorescein isothiocyanate, 400×).

The classic clinical presentation of PSGN is gross hematuria, with the urine appearing smoky brown, rather than bright red. Serum complement levels are low during the active phase of the disease, and serum ASLO antibody titers are elevated in PSGN. Recovery occurs in most children in epidemic cases. Some children develop rapidly progressive glomerulonephritis, owing to severe injury, with a formation of crescents.

Further reading

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Conflicts of interest: none.

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