

Acute poststreptococcal glomerulonephritis with acute interstitial nephritis related to streptococcal pyrogenic exotoxin B

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A 20-year-old man presented with symptoms of edema. Three weeks prior to admission, he experienced an upper respiratory tract infection. On admission, blood pressure was 177/118 mmHg. Initial laboratory investigations revealed serum creatinine, 170 $\mu\text{mol/L}$; complement C3, 130 mg/L and antistreptolysin O titer, 538 IU/mL (reference, <239 IU/mL). Urinalysis showed nephrotic range proteinuria and hematuria.

A percutaneous kidney biopsy was performed. Light microscopy contained 20 glomeruli, 4 of which were globally sclerotic and 1 of which showed cellular crescent formation. Diffuse endocapillary proliferative glomerulonephritis was identified (Figure 1A). Forty percent of the interstitium was infiltrated by inflammatory cells (Figure 1B). Immunofluorescent staining revealed complement C3, mainly on capillary walls.

Acute poststreptococcal glomerulonephritis (APSGN) with acute interstitial nephritis (AIN) was diagnosed. We administered methylprednisolone pulse therapy (1 g/day) for 3 days, followed by prednisolone at 80 mg/day. The serum creatinine level declined to 74 $\mu\text{mol/L}$ and proteinuria decreased to <1 g/day. Transient type 4 renal tubular acidosis was observed during steroid treatment.

AIN is found in the infectious diseases of children, particularly in streptococcal infection [1]. We investigated the

localization of two streptococcal antigens, streptococcal pyrogenic exotoxin B (SPEB) and nephritis-associated plasmin receptor (NAPlr), using an anti-SPEB antibody (Abcam, Cambridge, MA) and a fluorescein isothiocyanate-conjugated anti-NAPlr antibody [1F10] (Abcam). Although NAPlr was not detected, SPEB was positive in the interstitium as well as glomeruli (Figure 1C–F). Chang *et al.* [2] reported a case of streptococcal infection-related AIN, in which SPEB was found in both tubular epithelial cells and interstitium. In the present case, negative results were obtained for SPEB in tubular epithelial cells. Similarly, SPEB and its precursors have been detected only in glomeruli and interstitium in the rat kidney [3].

SPEB has been reported in association with the pathogenesis of APSGN. The mechanisms of leukocyte infiltration could be mediated by chemotactic effects and the migration inhibitor factor-like activities of SPEB [3]. SPEB also induces proliferation of human mononuclear leukocytes [4]. SPEB may be one of the causes of streptococcal infection-related AIN.

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Conflict of interest statement. None declared.

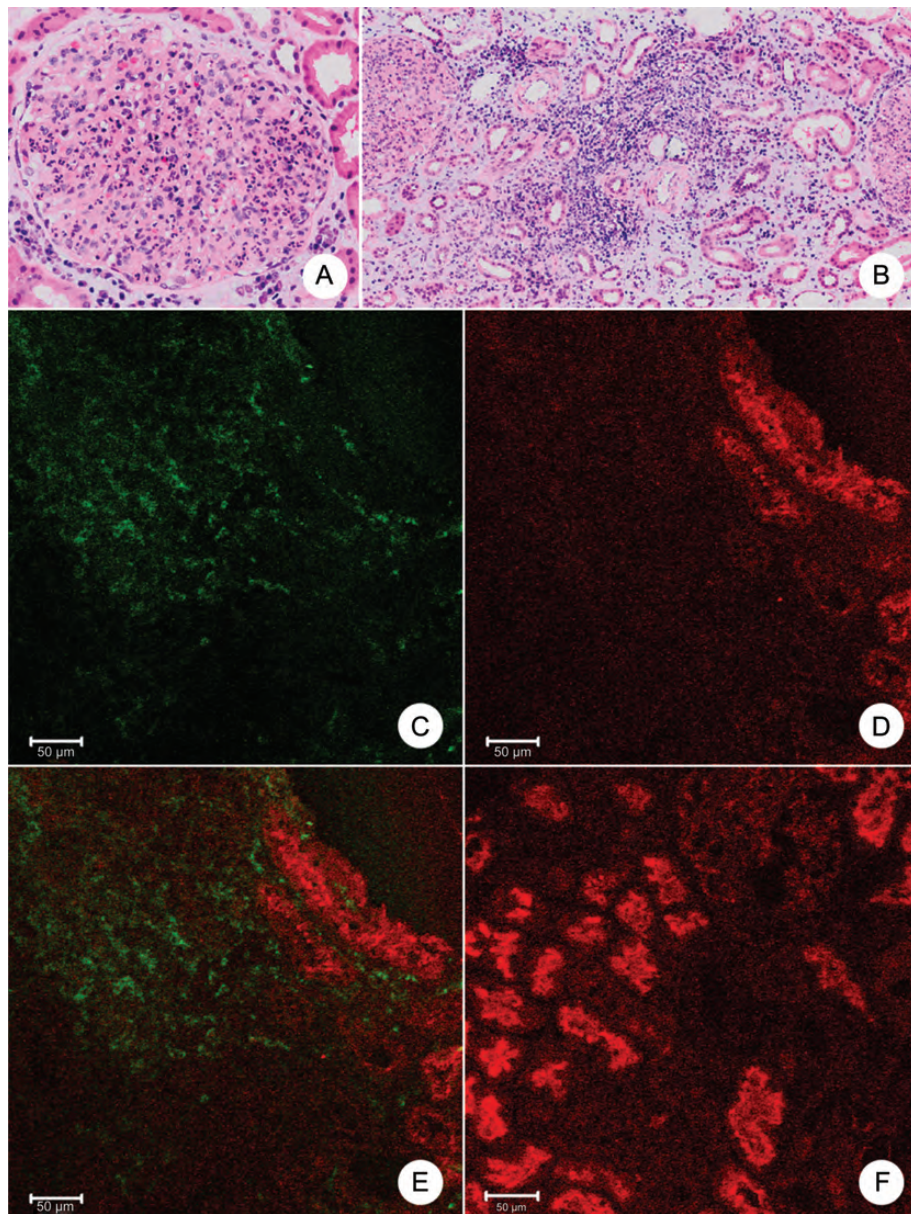


Fig. 1. (A and B) Findings of the kidney biopsy. Hematoxylin and eosin-stained section showing diffuse endocapillary proliferation with massive infiltration of neutrophils (A) and inflammatory cells infiltration in the cortical interstitium (B). (C–F) SPEB localization relative to the tubulointerstitium. Confocal microscopy images of double immunofluorescence staining for SPEB (C, FITC) and CD10 (D, Alexa Fluor 594) with a merged image (E). IgA nephritis case as a negative control (F).

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