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Poststreptococcal glomerulonephritis (PSGN) is an immunological-mediated glomerular lesion caused by prior infection, with specific nephritogenic stains of group A β-hemolytic streptococcus. Histologically, it is characterized by exudative glomerular injury, dominant C3 staining, and subepithelial humps. The clinical picture varies from asymptomatic to rapidly progressive glomerulonephritis. Although it is considered a self-limited condition, especially in children, it may occasionally follow a rather persistent course, leading to chronic kidney insufficiency. Patients with pre-existing pathology, where PSGN is superimposed, have been reported to have a poor outcome. Specifically, patients with comorbidities, such as diabetes mellitus, hypertension, smoking, and alcoholism, are more likely to end up on chronic dialysis.

For patients with PSGN who require or are progressing toward dialysis treatment, a kidney biopsy is mandatory in the acute phase to confirm the diagnosis and exclude other conditions that may present with acute glomerulonephritis and hypocomplementemia, such as C3 glomerulopathy, cryoglobulinemic glomerulonephritis, lupus nephritis, membranoproliferative glomerulonephritis, and IgA-dominant, staphylococcus-associated glomerulonephritis. The pattern of hypocomplementemia and its duration (the disease time-course), i.e., acute onset versus persistent-recurrent active glomerulonephritis over a prolonged period, in correlation with histopathology, depict the diagnosis. Clinically, a critical distinction pertains to the time of infection occurrence; prior to PSGN or coincidentally with the IgA-dominant, staphylococcus-associated glomerulonephritis.

Left image: A large glomerulus from the first biopsy with a significant number of inflammatory cells, mostly neutrophils, into distended capillary lumena. Thick basement membranes, diffuse mesangial matrix increase, and a well-formed mesangial nodule at the bottom right can be appreciated (H&E ×400).

Left center image: A large glomerulus from the second biopsy with extensive neutrophil infiltration in association with distension of glomerular lumena, superimposed on mesangial expansion caused by diabetes (H&E ×400).

Right center image: Diffuse, coarsely granular deposits along the glomerular capillary walls and focally in the mesangial regions, in a starry sky appearance (C3 ×400).

Right image: Electron microscopy revealed numerous and large subepithelial, electron-dense deposits (humps) situated on moderately thickened basement membranes. Plentiful neutrophils were seen in the glomerular lumena (uranyl acetate and lead citrate ×4400).

Images provided by Sophia Lionaki,* MD, Maria Pappa, MD, and John N Boletis, MD, Nephrology Department & Transplantation Unit, Laiko Hospital, National and Kapodistrian University of Athens, Faculty of Medicine, Greece, and George Liapis,* MD, Pathology Department, Laiko Hospital, National and Kapodistrian University of Athens, Faculty of Medicine, Greece, with acknowledgment to Sophia Haraki, PhD, Department of Histology and Embryology, National and Kapodistrian University of Athens, Faculty of Medicine, Athens, Greece, for technical support in electron microscopy.

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