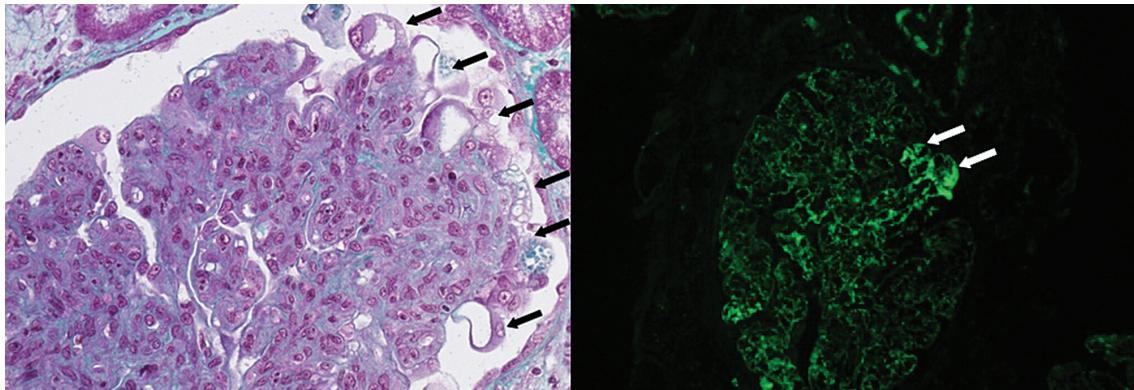


Podocyte Injury in Segmental Garland-pattern Poststreptococcal Glomerulonephritis

IJKD 2013;7:356
www.ijkd.org



A 13-year-old boy presented with anasarca and hypertension after pharyngitis. Laboratory tests showed serum creatinine, 1.6 mg/dL; albumin, 2.9 g/dL; low C3; elevated antistreptolysin O; urine protein 7.25 g/24 h. A kidney biopsy revealed proliferative glomerulonephritis with humps. A segmental podocytes cap with marked hypertrophy was also identified in some glomeruli (Left). By immunofluorescence, immunoglobulin G and C3 were diffusely scattered in a granular pattern, predominantly along glomerular basement membrane, but sometimes confluent subepithelial deposits showed intense peripheral staining in glomerular segments that resembles a garland (Right). A diagnosis of garland-pattern acute poststreptococcal glomerulonephritis was made. Six months after treatment, serum creatinine level was 0.8 mg/dL and proteinuria was 2.4 g/24 h.

Acute poststreptococcal glomerulonephritis appears to be induced by specific nephritogenic strains of group A beta-hemolytic streptococcus. Subepithelial immune complex deposition causes local inflammation and proteinuria by podocyte disruption and may induce podocyte proliferation¹. Acute poststreptococcal glomerulonephritis is manifested by a discrete, more densely packed and confluent heavy disposition of immunoglobulin G and complement C3, corresponding to numerous humps noted on the subepithelial side of the capillary walls.² Patients with garland-pattern more frequently develop nephrotic syndrome compared to those with the other types. There were also cases with a complete disappearance of proteinuria, especially in younger patients, but other patients still had a distinct proteinuria after months to years indicating a protracted or chronic course.³

ACKNOWLEDGMENTS

We are very grateful to Osmar M Silva and Edienny V Lobato.

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