

## Acute Glomerulonephritis

Acute glomerulonephritis (AGN) may be a primary event or a manifestation of a systemic disorder that can range from minimal to severe. Common features include oliguria, edema, hypertension and circulatory congestion, hematuria, and proteinuria. Most cases are postinfectious and have been associated with pneumococcal, streptococcal, and viral infections. **Acute poststreptococcal glomerulonephritis (APSGN)** is the most common of the postinfectious renal diseases in childhood and the one for which a cause can be established in the majority of cases. APSGN can occur at any age but affects primarily early school-age children, with a peak age of onset of 6 to 7 years old. It is uncommon in children younger than 2 years old, and boys outnumber girls two to one.

### Etiology

APSGN is an immune-complex disease that occurs after an antecedent streptococcal infection with certain strains of the group A beta-hemolytic streptococci (GABHS). Most streptococcal infections *do not* cause APSGN. A latent period of 10 to 21 days occurs between the streptococcal infection and the onset of clinical manifestations. Disease secondary to streptococcal pharyngitis is more common in the winter or spring, but when APSGN is associated with pyoderma (principally **impetigo**), it may be more prevalent in late summer or early fall, especially in warmer climates. Second episodes of APSGN are rare.

### Pathophysiology

The pathophysiology of APSGN is still uncertain. Immune complexes are deposited in the glomerular basement membrane. The glomeruli become edematous and infiltrated with polymorphonuclear leukocytes, which occlude the capillary lumen. The resulting decrease in plasma filtration results in an excessive accumulation of water and retention of sodium that expands plasma and interstitial fluid volumes, leading to circulatory congestion and edema. The cause of the hypertension associated with AGN cannot be completely explained by fluid retention. Excess renin may also be produced.