INTRODUCTION AND AIM OF THE WORK

Acute postinfection glomerulonephritis (AGN) is the most common nonsuppurative renal disease of childhood (Cameron and Ogg, 1973). Several bacterial and viral infections have been incriminated in its causation, but the most commonly recognized clinical picture follows infection with group A, β hemolytic streptococci (Dillon, 1972; Dodge *et al.*, 1972; Dillon, 1979; Kaplan, 1980 and Ginsburg, 1988).

Glomerulonephritis in man is mediated by immunological mechanisms. Two major forms of immunologically induced glomerular diseases are recognized, namely immune complex glomerulonephritis and antiglomerular basement membrane antibody (anti-GBM) disease. Immune complex glomerulonephritis may be defined as a disease that results from deposition within a glomeruli of antigen-antibody complexes formed in the circulation (Wilson, 1982 and Couser *et al.*, 1985).

Acute glomerulonephritis is a common complication of streptococcal infection particularly in young age groups. Acute poststreptococcal glomerulonephritis (APSGN) is exceedingly rare before the age of 2 years. Thereafter, it may occur at any age but most often in school-age children. The mean age of onset in many cases of sporadic form is approxi-

mately 7 years (Boswell and Oknoyan, 1968; Abrass, 1985; Clark, *et al.*, 1988 and Boineau and Lewy, 1989).

Poststreptococcal glomerulonephritis was the first renal disease in which immunological mechanisms were implicated, because of the findings of hypocomplementemia, a serum sickness-like latent period, and the later demonstration of immune deposits in the glomeruli (Villareal *et al.*, 1982). However, many aspects of its immunopathology remain uncertain. The characteristic clinical history is of the development of acute nephritis 2 to 3 weeks after a group A β hemolytic streptococcal infection of the respiratory tract or skin, and certain M serotypes have been identified that are particularly associated with development of the disease (Rodriguez-Iturbe *et al.*, 1979). It is unusual to develop a second attack, suggesting the development of long-term immunity to the antigen(s) involved. Poststreptococcal nephritis remains an important cause of glomerular disease in underdeveloped countries but is rare in Western Europe and North America (Arieff, 1971; McCarty, 1972 and Arant, 1987).

Assay for circulating immune complexes are positive in the majority of cases (Rodriguez-Iturbe *et al.*, 1980). Hyperglobulinemia (suggesting polyclonal B cell activation) and cryoglobulins are frequently detected, and circulating levels

of complement are reduced in the majority of patients (Rodriguez-Iturbe , 1984).

Some cases show reduction in classical pathway components, whereas others have a reduced C3 with normal C1 and C4; these generally return to normal within 2 months. Poorly characterized complement activators have been identified in sera from some patients. Immunofluorescence of renal biopsies reveals discrete deposits of IgG and C3 along the glomerular basement membrane (GBM), and electron microscopy shows the presence of electron-dense "humps" in a subepithelial position (Hinglais *et al.*, 1974).

A genetic susceptibility to poststreptococcal nephritis has been suggested from family studies (Rodriguez Iturbe *et al.*, 1981). and major histocompatibility complex (MHC) associations also have been reported: A South American study shows an increased incidence of HLA-DR4 in unrelated patients (Layrisse *et al.*, 1983). and an association with an HLA-D antigen (termed DEn) has been found in Japan (Sazauki *et al.*, 1979).

In most instances however the glomerulonephritis clears out relatively rapidly leaving an almost completely healthy kidney. In few cases the nephritis gradually turns to chronicity (Kurtzman, 1978).