INTRODUCTION

THE GLOMERULAR DISEASES

Disorders of glomerular structure and function are encountered frequently in clinical medicine. Many arise as a part of a well-defined multisystem or multi-organ disease, while in others the clinical and laboratory manifestations are consequent to the sole or predominant involvement of the glomeruli. The latter are known as the primary glomerulopathies [1].

The primary glomerular diseases are a category of glomerulonephritis in which clinical and biochemical abnormalities are the sole or predominant consequences of damage to the integrity of glomerular structure and/or function. Clinically, several syndromes are found (Table I). These syndromes arise as a result of the admixture of hematuria, proteinuria, reduced glomerular filtration rate, and sodium chloride and water retention-all these are cardinal manifestations of glomerular disease [1].

The primary glomerular diseases are best classified according to the principal clinical and laboratory manifestations of the disease. The findings in urine analyses, specifically the extent and nature of hematuria and proteinuria are extremely useful in defining glomerular diseases [2].

Many glomerulopathies are immunologically mediated. With the use of immuno-histologic techniques (immunofluorescence or immunoperoxidase), the presence or absence of antibody, antibody class, site of deposition (capillary wall or mesangium), and patterns of deposition (granular or linear) can be determined [2].

Linear capillary wall deposition is usually indicative of the presence of an autoantibody (most often IgG) directed against intrinsic glomerular

basement membrane antigens. In most cases, the antigenic site lies within a non-collagenous domain of the α3 domain of type IV collagen [1].

Granular patterns of immunoglobulin deposition and electron dense deposits are indicative of immune complex presence in the glomeruli. The antigen may be endogenous (e.g. nuclear antigens or histones), or exogenous (e.g. viral or bacterial antigens). The antigen may also be intrinsic or extrinsic to the kidney (renal or non renal endogenous antigens). Circulating immune complexes composed of extrinsic (non-renal) antigens and antibody may be deposited in the glomeruli. Some antigens normally extrinsic to the glomeruli may localize within glomerular structures and form immune complexes *in situ* by reacting with circulating antibodies [2].

Polyangiitis is a situation in which autoantibodies against neutrophil cytoplasmic antigens are formed [1].