Introduction and aim of the work.

Introduction :

Anti-neutrophil cytoplasmic antibodies (ANCA) are heterogeneous group of autoantibodies with a wide and diverse range of clinical associations. In vasculitis the diagnostic utility of proteinase 3 (PR3)-ANCA and myeloperoxidase-ANCA for Wegener's granulomatosis and microscopic polyangiitis, respectively, is now well established. Because of their significance as tools for diagnosis and prognosis, these autoantibodies have been analyzed extensively as markers for underlying immunopathologenic disturbances (Gross and Csernok, 1995).

Renal vasculitis frequently presents itself as rapidly progressive glomerulonephritis (GN), but its diagnosis may be hampered by the difficulty in demonstrating classic vasculitic lesions in renal biopsy specimens.

Early diagnosis of renal vasculitis has been greatly enhanced by the advent of antineutrophil cytoplasmic autoantibodies (ANCA).(Wen,1994). ANCA are not only the markers for vasculitis but may also play a role in the pathogenesis by activating the neutrophils to attack target blood vessels (Wen, 1994). ANCA-associated vasculitis responds well to steroid and / or cyclophosphamide therapy. Renal failure in these patients is frequently reversible if treated early. Long term patient and kidney survival rate are good with proper treatment and far better than of the other causes of rapidly progressive glomerulonephritis (Wen, 1994).

De Oliviera et al, 1995, had demonstrated that changes in ANCA concentration may reflect changes in disease activity as patients whom ANCA were detectable one year or more after treatment, were at particular risk of clinical relapse. Also the temporal relationship between changes in ANCA concentration and clinical relapse varied considerably between patients.

Aim of Work :

This work aimd at examining the prevalence and clinical association of ANCA in patients with glomerulonephritis to detect the risk of relapse and to determine the need for frequent clinical review besides the possible use of ANCA as a marker for continuing maintenance immunosuppression therapy.