INTRODUCTION AND AIM OF THE WORK

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There are various immunological abnormalities described in chronic renal failure patients (CRF). The marked depression of the immune function of these patients is evidenced by change in the T-cell and its subpopulations, lymphocytes of phenotype CD_3 (pan T cell), CD_8 (suppressor, cytotoxic cells) were decreased, the (helper inducer) CD_4 cell showed tendency to decrement and CD_4/CD_8 ratio negatively correlates with serum creatinine level ***(Cheng et al., 1991)***. The chronic renal failure patients present with decreased T-lymphocytes, increased CD_4/ CD_8 ratio with depressed immune system ***(Lee et al., 1991)***. Renal failure changes the ratio of subpopulation of lymphocytes by decline in the helper (CD_4) cells and raise of cytotoxic suppressor cells CD_8 ***(Bartunkova et al., 1990)***.

Depression of immunological responsiveness was observed in CRF patients and manifested by anergy to intradermal antigen, delayed graft rejection, and impaired ability to produce high antibody titer to variety of antigens, the most important clinical consequences of this impaired immune state is enhanced susceptibility to infection and noeplasm ***(Zmonarski et al., 1995)***.

Administration of 1,25 dihydroxy claciferol in CRF patients reduces the infection complications and improves the clinical condition of patients ***(Bartunkova et al., 1990)***.

T-lymphocyte have receptors for parathyroid hormone (PTH), patients with chronic renal failure have high level of PTH and impaired

lymphocyte function but not altered CD4/CD8 ratio ***Klinger et al., (1990)***. The basal level of cytosolic calcium Ca^{2+} in uremic patients were elevated in many cells including brain synaptosomes, pancreatic islets, polymorphnuclear leukocytes, platelets, and B & T cells. The raise in Ca^{2+} has been attributed to the state of secondary hyperparathyroidism (2HPT) in chronic renal failure patients, these observations led to proposition that chronic renal failure is a state of cellular calcium intoxication mediated by excess parathyroid hormone ***(Stajeceva – Taneva et al., 1993).***

The prime object of this work is to evaluate the specific role of T-lymphocyte subpopulation in immunoregulatory mechanisms in chronic renal failure patients under hemodialysis and those under conservative treatment, by utilization of highly sensitive immunofluorescence technique using most recently developed monoclonal antibodies to estimate the T-lymphocytes and its subpopulations. Also to estimate the level of urea, creatinine calcium, phosphrus and parathyroid hormone (PTH) with the relation to T-lymphocyte subpopulation.