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"Nephrosis" is a word coined by Friedrich Müller in 1905 to distinguish fatty degeneration of tubules with normal glomeruli from "nephrosis": a term that denotes an inflammatory lesion of glomeruli.(Martin and Chester,1992).

The nephrotic syndrome is characterized by proteinuria, hypoproteinemia, edema and hyperlipidemia. Most children (90%) with nephrosis have some from of idiopathic nephrotic syndrome (INS). The underlying pathogenetic abnormality in nephrotic syndrome is proteinuria, which results from an increase in glomerular capillary wall permeability. The mechanisms of the increase in permeability is unknown but may be related, at least in part, to loss of negatively charged glycoprotein within capillary wall. The cause of the syndrome remains unknown. Early success in controlling nephrosis with "immunosuppressive" drugs suggested that the disease was mediated by immunologic mechanisms. Increasing evidence suggest that the syndrome may result from abnormality in Thymus-derived (T-cell) lymphocyte function, perhaps through the production of a factor that increases vascular permeability(Bergstein, 2000).

T-cells are known to be made up of two well defined functional populations; cytotoxic ($CD8^+$) and helper ($CD4^+$) cells. Effector T-cell (helper) signaling leads to induction of high level transcription of interleukin-2 (IL-2) gene. IL-2 is an autocrine and paracrine cytokine secreted by activated T lymphocytes. It plays a povital role in proliferation of cognate activated helper and cytotoxic T-cells and

also participates in the expansion and differentiation of activated natural killer (NK) cells and B-lymphocytes (Oppenhein et al , 1994)

The high affinity IL-2 receptor (IL-2R) is not expresed on resting T-cells but is induced to maximal levels 2-3 days after the cells become activated. When exposed to appropriate activating stimuli, resting CD4 T-lymphocytes begin to express both IL-2R and shortly thereafter begin to proliferate.(Bergstein ,2000 ; Oppenheim et al , 1994)

It has been noticed that immunosuppressive drugs, as cyclosporin A, act in part by blocking the induction of IL-2 and IL-2R to helper T-lymphocytes. Abnormal T-lymphocyte function and reduced IL-2 production have been implicted in the pathogenesis of the nephrotic syndrome (Hulton et al , 1994)

So, the present work was designed to investigate the peripheral blood T-cell subset (CD4 and CD8), IL-2R expression and IL-2 production as T-cell activation criteria in patients with INS.