Introduction

Although the exact etiology of primary nephrotic syndrome (PNS) is still unclear, an immune mechanism is generally accepted as a cause . The importance of the immune system in the pathogenesis of nephrotic syndrome (NS)was first suggested in 1974 *(Shalhoub, 1974)*.The disorder was believed to be a consequence of T lymphocyte dysfunction *(Nash et al., 1992 b)* . This was supported by finding that relapses are often triggered by viral upper respiratory tract infection . Furthermore, measles infection which is known to inhibit T lymphocyte function is associated with induction of remission *(Nash et al., 1992a)* .

Recently, leukotrienes (LTs) of the 5-lipoxygenase pathway have been implicated in glomerular injury *(Lewis et al., 1990)*. They are produced mainly by inflammatory cells, such as monocytes, macrophages, neutrophils, eosinophils, mast cells and also by intrinsic glomerular cells and other cells. They have been detected in serum, urine and renal tissue during glomerular inflammation *(Yard et al., 1991 and Lefkowith et al., 1992)*.

Menegatti et al . (1999) found that there is co-expression of both 5-LO and LTA4 hydrolase mRNA in renal tissue of PNS patients strongly suggesting that LTB4 is produced in renal tissue of some nephrotic patients. Few studies have been done to clear out the role of LTs involving renal tissue extraction, treatment with specific LTs antagonist or dietary manipulations aimed at reducing LTs biosynthesis *(Badr, 1991)*. However, their results are conflicting. Moreover these studies doesn't consider the different clinical stages of the disease.

Recent cloning of and characterization of cDNA sequence of 5-LO and LTA4 hydrolase have been directed our attention to examine the gene expression of 5-LO and LTA4 hydrolase in PNS patients considering their different clinical states.