

## **INTRODUCTION**

Nephrotic syndrome is one of the commonest renal diseases in childhood. It is characterized by proteinuria, hypoalbuminemia, hyperlipidemia, and edema. An increased glomerular permeability leading to proteinuria is the primary renal abnormality in the nephrotic syndrome, the other clinical and laboratory findings being considered as secondary manifestations (*Kher, 1992*).

Albumin is the predominant plasma protein lost in urine in nephrotic patients, but other plasma proteins such as immunoglobulins, various coagulation factors, vitamin D-binding protein, and metalloproteins are also excreted in significant amounts in urine (*Kher et al, 1988*).

Abnormalities in calcium and vitamin D metabolism in patients with nephrotic syndrome are not uncommon. Patients with nephrotic syndrome and heavy proteinuria excrete a substantial amount of vitamin D-binding protein in the urine. Consequently, they have low serum levels of vitamin D metabolites (*Sato et al, 1982*).

Hypocalcemia has long been recognized in nephrotic patients, but it was simply attributed to hypoalbuminemia and the consequent decrease in protein-bound calcium (*Lim et al, 1977*). However, serum ionized calcium may be also reduced, even in nephrotic patients with normal renal function; this may result in symptomatic hypocalcemia (*Goldstein et al, 1981*).

Recent studies have shown a multifactorial origin for the hypocalcemia including low serum levels of vitamin D metabolites, reduced intestinal absorption of calcium, and skeletal resistance to the calcemic action of parathyroid hormone (*Freundlich et al, 1985*).