Nephrotic syndrome (NS) is one of the most frequent glomerular pathological conditions seen in children.1 Hypocalemia is a common feature in NS patients. It was initially attributed to hypoalbuminemia leading to reduction of protein-bound calcium.2,5 It may also relate to low ionized calcium levels due to loss of vitamin D-binding protein and 25-hydroxyvitamin D3 (25-OHD). Steroid-sensitive nephrotic syndrome (SSNS) are only intermittently proteinuric. Consequently, concern regarding vitamin D nutritional status in NS has focused on treatment of steroid-resistant nephrotic syndrome (SRNS), with its persistent proteinuria, rather than SSNS.3,9 Before children with NS can be considered candidates for routine 25-OHD screening, the prevalence of low 25-OHD levels in this population should be confirmed.4

Patients & Methods

A case-control study conducted on 20 children with first episode of SSNS attending Alexandria University Children’s Hospital (AUCH) compared to 20 healthy children as a control group.

Age of the patients included in the study ranged between 2.0 and 5.90 years (mean 3.60 ± 1.54 years). There were 15 (75%) males and 5 (25%) females.

Serum ionized calcium, total calcium, serum phosphorus, alkaline phosphatase (ALP), serum albumin, total protein, parathormone (PTH), 25-OHD, spot urine protein/creatinine (Pr/Cr) ratio were measured during the active stage of the disease and serum ionized calcium was repeated after remission.

Vitamin D deficiency (VDD) was defined as 25-OHD level ≤ 20 ng/ml, severe VDD ≤ 5ng/ml, vitamin D insufficiency 21-29 ng/ml, and vitamin D sufficiency ≥ 30 ng/ml.

The study was approved by the Research Ethics committee in Alexandria University and informed consent was obtained from enrolled patients.

Objective

To study the level of 25-OHD during the active stage of the disease and serum ionized calcium during the active stage and after remission in SSNS.

Results

Children with active SSNS had low ionized calcium, low serum 25-OHD levels, high phosphorus and low ALP levels versus controls.

All of NS patients in the present study had VDD, 80% of which had severe degree (Fig.1).

Eighteen out of the 20 SSNS patients (90%) had low serum ionized calcium levels during the active stage of the disease. After remission, ionized calcium level increased and only 8 patients (40%) were still hypocalcemic with the lowest level being 4.3 mg/dl (Fig.2).

However, both were significantly lower than the control group.

Two patients had history of tetany during the active stage of the disease with serum ionized calcium levels 4.1 and 4.5 mg/dl (although their ionized calcium was not the least value among the patients).

The mean level of serum PTH was higher in SSNS during the active stage of the disease compared to the controls but only 30% of the patients had secondary hyperparathyroidism with PTH levels > 65 pg/ml.

Serum ionized calcium was negatively correlated to spot Pr/Cr ratio in urine (r = − 0.565, p = 0.009)(Fig.3).

Conclusions

Children with SSNS are at risk of VDD and hypocalemia, therefore further research will be needed to prove the need of vitamin D supplementation to reach normal levels of 25-OHD and to prevent the occurrence of possible complications.

References