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Original Article

Evaluation Of Thyroid Functions In Children On Hemodialysis In Sohag University Hospital

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Abstract

Background: End-stage renal disease (ESRD) and hemodialysis (HD) can cause thyroid dysfunction. The earliest and most common thyroid function abnormality in ESRD patients on HD is low triiodothyronine (T3) level, low T3 syndrome. Moreover the prevalence of subclinical hypothyroidism is significantly higher in ESRD patients on HD compared to the general population. Detailed knowledge of all these changes is extremely important to plan the good management of the patient.

Aim To evaluate thyroid functions in patients undergoing chronic HD in the pediatric nephrology unit at Sohag University Hospital.

Methods The study was a prospective study. The age of the studied cases ranged from 6 to 23 years. Overall, 19 male and 22 female patients were subjected to detailed history taking and full clinical examination. All patients were evaluated for complete blood count (CBC), kidney function tests ,liver function tests, arterial blood gases (ABG), parathyroid hormone level (PTH) , vitamin D level and thyroid function tests

Results: The study conducted at the Pediatric Nephrology Unit of Sohag University Hospital investigated thyroid function in 41 pediatric patients with ESRD undergoing HD. Thyroid function tests revealed that there were 6 patients with subclinical hypothyroidism and 6 cases of low T3 syndrome. Clinical examination revealed no abnormalities in thyroid gland palpation among all participants.

Conclusion: The low T3 syndrome and subclinical hypothyroidism are relatively common thyroid disorders among children with ESRD. We recommend regular monitoring of thyroid functions in children with ESRD for early detection of any disturbance to reduce the morbidity and mortality.

Key words: thyroid functions - hemodialysis – children

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Introduction:

Thyroid dysfunction is a prevalent comorbidity in children with end-stage renal disease (ESRD) undergoing hemodialysis (HD). Chronic kidney disease (CKD) and ESRD can significantly affect thyroid function, necessitating careful assessment and management. CKD involves progressive kidney function loss, leading to metabolic waste accumulation, electrolyte imbalances, and fluid overload, which impact the endocrine system, including the thyroid gland. The thyroid gland plays a vital role in producing THS, namely T4 and T3. [1]

In children with CKD and ESRD, various factors contribute to thyroid function disruption. Alterations in the hypothalamic-pituitary-thyroid (HPT) axis can occur, affecting thyroid hormones synthesis and secretion. Reduced renal function impairs thyroid hormones clearance, leading to elevated circulating levels. Changes in binding proteins and enzyme activities further complicate thyroid hormones metabolism. [2]

Beyond the HPT axis, CKD and ESRD affect fluid, electrolyte balance, and immune system function, exacerbating thyroid dysfunction. Disturbances like hyperphosphatemia and hypocalcemia interfere with thyroid hormones synthesis and action, while immune system alterations contribute to autoimmune thyroid diseases like Graves' disease and Hashimoto's thyroiditis. [3]

Thyroid dysfunction in HD children has signifycant consequences, impacting growth, developent, bone health, cardiovascular function, cognitive function, and metabolic balance.[4]

Given this complex interplay, evaluating thyroid function in HD children is critical. Monitoring thyroid hormones levels, including TSH, T3, and T4, is essential for identifying and managing thyroid disorders effectively. Understanding the effects of CKD and ESRD on the thyroid and other systems enables comprehensive management strategies tailored to optimize the health of HD children. [5]

Patients and Methods

Study Design:

This prospective hospital-based study extended from April 2022 to October 2022. The study was conducted on 41 children who initiated HD for ESRD. Participants were recruited from the Pediatric Nephrology Unit at Sohag University Hospital.

Ethical Considerations:

Approval for this study was obtained from the Ethical Committee at Sohag Faculty of Medicine, Sohag University, before its initiation. Informed written consent was obtained from all legal guardians of participating patients. Confidentiality and personal privacy were maintained throughout the study, and collected data were utilized solely for scientific purposes.

Inclusion Criteria:

Children who commenced chronic HD for ESRD at age ranging from 2 to 16 years **in** both sexes

Exclusion Criteria:

Children with known thyroid disease

Children with a history of autoimmune disease

Methods:

A. Clinical History:

Socio-demographic data were collected, including age, sex, and residence.

Information regarding the cause of ESRD, onset of hemodialysis, and its duration was obtained.

Symptoms suggestive of thyroid hormone deficiency were noted, such as fatigue, sluggishness, weight gain, hair and skin changes, cold intolerance, and constipation.

History of previous thyroid disease, thyroid surgery, radioactive

iodine intake, or autoimmune diseases was recorded. _Family history of goiter or altered thyroid functions was also documented.

B. Clinical Examination:

a. General examination encompassed vital signs, anthropometric measurements, and examination of various body systems.

b. Local examination included palpation of the thyroid gland to assess size, consistency, nodules, and tenderness.

c. Laboratory Investigations:

Basic investigations comprised:

Complete blood count, blood urea, serum creatinine, electrolytes (sodium, potassium), serum calcium, phosphorus, PTH level , liver function tests ,blood gases, complete urine analysis for patients who still pass urine and thyroid function tests were conducted, including: TSH , T3 , FT3 ,T4 and FT4.

Table (1): Normal reference ranges of thyroid hormones [6]

Thyroid-stimulating hormone (TSH	6 mo-18 yr	0.5-4.5 µ IU/L
Thyroxine (T4), total	1-5 yr 6-18 yr	4.5-11.0 µ g/dL 4.5-10.0 µ g/dL
Thyroxine (T4), free	31 days-18 yr	0.7-2.00 ng/dL
Triiodothyronine (T3), free	6 wk Adult (20-50 yr)	240-560 pg/dL- 3.7-8.6 pmol/L 230-660 pg/dL- 3.5-10.0 pmol/L
Triiodothyronine (T3), total	1-6 yr 7-11 yr 12-18 yr	90-240 ng/dL-1.4-3.7 nmol/L 90-230 ng/dL- 1.4-3.6 nmol/L 100-210 /dL- 1.5-3.3 nmol/L

.Abdominal ultrasonography with stress on kidneys and urinary tracts.

.Neck ultrasound was done in children with goiter.

Statistical analysis :

Statistical analysis was done by SPSS version 28 (IBM Co., Armonk, NY, USA). Quantitative parametric data were presented as mean and standard deviation (SD). Quantitative non-parametric data were presented as median and interquartile range (IQR), analyzed by Mann Whitney-test. Categorical data were presented as frequency and percentage, analyzed using the Chi-square test. Spearman's rank correlation coefficient was calculated to estimate the degree of correlation between two quantitative variables. A two tailed P value < 0.05 was considered statistically significant.

Results:

A total of 19 (46.3%) patients were males and 22 (53.7%) patients were females. The mean age of the cases was 15.17 ± 4.69 years. The median duration of dialysis was 47.67 (IQR 17.27 – 106.59) months with 3 sessions per week. Overall, 27 (65.9%) cases had short stature and 14 (34.1%) cases were underweight as shown in **(Table 2)**

Table 2: Baseline characteristics of the studied patients

Variables	Total patients (n=41)
Age (years)	
Mean ± SD	15.17 ± 4.69
Range	6 – 23
Gender	
Male	19 (46.3%)
Female	22 (53.7%)
Male/female ratio	0.9
Height (cm)	
Mean ± SD	134.68 ± 16.12
Range	100 – 165
< 3rd percentile (short stature)	27 (65.9%)
Weight (kg)	
Mean ± SD	30.46 ± 10.28
Range	12 – 50
< 3rd percentile (under weight)	14 (34.1%)
Duration of dialysis (months)	
Median (IQR)	47.67 (17.27 – 106.59)
Range	4.07 - 189.77
Number of sessions/week	3 ± 0

The causes of CKD included bilateral atrophic kidneys in more than half of them (51.2%), 5 (12.2%) had single atrophic kidney, 3 (7.3%) had focal segmental glomerulosclerosis FSGN, 2 (4.9%) had Nephronophthisis, 2 (4.9%) had Tubulo- interstitial nephritis, 1 (2.4%) for each of the following cases with cystic kidney, Infantile nephrosis, Obstructive uropathy, Rapidly progressive GN, Bilateral hypoplastic dysplastic kidneys, Chronic pyelonephritis, chemotherapy induced ESRD and multi drug resistant NS as shown in (**figure 1)**

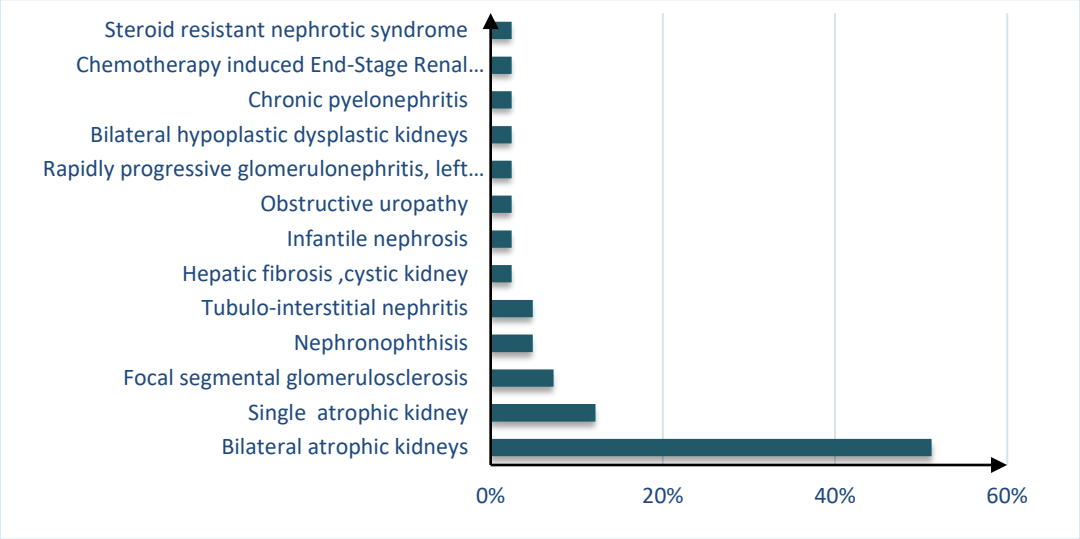

Figure 1: Cause of dialysis among the studied patients

Thyroid function was assessed, revealing a median TSH level of 2.31 µIU/L (IQR 1.8 - 3.2), with 6 patients exceeding the upper normal limit 4.5 µIU/L. The median T3 level was 130 ng/dL (IQR 112.55 - 153.5), with 6 patients below the lower normal limit 90 ng/dL. The median free T3 level was 2.5 pg/mL (IQR 2.25 - 2.75), with 12 patients below the lower normal limit 2.4 pg/mL. The median T4 level was 8.6 µg/dL (IQR 5.6 - 9.55), with 3 patients above the upper normal limit 11 µg/dL. The median free T4 level was 1 ng/dL (IQR 0.9 - 1.2), with all patients within the normal range (0.7-2.00 ng/dL as showed in (T**able 3)**

Table 3: Thyroid function tests of the studied patients

Variables	**Total patients (n=41)**
TSH (µIU/L)	
Median (IQR)	2.31 (1.8 - 3.2)
Range	0.5 – 4.5
Sub-clinical hypothyroidism (TSH>4.5 µIU/L)	6 (14.6%)
Normal TSH (0.5 – 4.5)	35 (85.4%)
T3 (ng/dL)	
Median (IQR)	130 (112.55 - 153.5)
Range	90 -240
FT3 (pg/ml)	
Median (IQR)	2.5 (2.25 - 2.75)
Range	2.4 - 7
T4 (µg/dL)	
Median (IQR)	8.6 (5.6 - 9.55)
Range	4.5 - 11
FT4 (ng/dL)	
Median (IQR)	1 (0.9 - 1.2)
Range	0.7 - 2

The study identified 6 cases of subclinical hypothyroidism and 6 cases of low T3 syndrome. Palpation of the thyroid gland was normal in all patients. .

Patients with subclinical hypothyroidism elicited significantly decreased levels of potassium, total bilirubin and PCO2 compared to those with normal thyroid function (P=0.002, 0.02, 0.006 respectively). On the other hand, pH level was significantly increased in patients with sub-clinical hypothyroidism than others with normal thyroid function (P=0.02) as show in (**Table 4)**.

Table 4: Association between thyroid function of the studied patients and different laboratory investigations

Variables	Normal (n=35)	Subclinical hypothyroidism (n=6)	P value
CBC			
Hb (g/dL)	11.3 (8.75 - 12.4)	11.4 (9.55 - 11.7)	0.9
Anemia	12 (34.3%)	2 (33.3%)	>0.999
WBCs ($x10^3$cells/µl)	5.8 (5.18 - 7.9)	7.7 (5.58 - 10.02)	0.148
PLT ($x10^3$cells/µl)	188 (163 - 228)	226.5 (186 - 288.75)	0.148
HCT (%)	34.3 (28.7 - 37.3)	34.4 (30.58 - 35.7)	0.815
MCV (fL)	88.5 (85 - 95.2)	88.6 (86.28 - 93.85)	0.706
Kidney function			
BUN (mg/dL)	114 (105 - 127)	114 (97.25 - 171.25)	
Serum creatinine (mg/dL)	7.4 (6.09 - 9.44)	6.55 (5.08 - 8.7)	0.376
Sodium (mmol/L)	133.2 (133 - 135.1)	133.5 (132.75 - 135)	0.815
Potassium (mmol/L)	4.6 (3.84 - 4.9)	3.6 (3.55 - 3.76)	**0.002***
Total calcium (mg/dl)	8.9 (8 - 9.8)	8.95 (8.53 - 10.35)	0.505
Ionized calcium (mg/dl)	1.02 (0.9 - 1.2)	1.16 (1.03 - 1.47)	0.102
Liver function			
ALT (IU/L)	12 (10 - 16)	16.5 (9 - 89)	0.552
AST(IU/L)	18 (16 - 24)	30.5 (15 - 75.25)	0.252
Total protein (g/dL)	7.2 (6.6 - 7.4)	6.75 (5.9 - 7.65)	0.577
Albumin (g/dL)	3.7 (3.5 - 4.2)	3.7 (3.35 - 4.13)	0.872
Total bilirubin (mg/dL)	0.5 (0.4 - 0.7)	0.3 (0.28 - 0.38)	**0.02***
ABG			
pH	7.3 (7.2 - 7.3)	7.32 (7.3 - 7.37)	**0.02***
PCO_2 (mmHg)	33.1 (30.6 - 40.5)	29.6 (28.5 - 30.13)	**0.006***
PO_2 (mmHg)	127 (112 - 148)	120.5 (109.48 - 125)	0.459
Base Deficit (mmol/L)	-8.8 (-10.9 to -7.1)	-7.7 (-8.7 to-7.23)	0.237
HCO_3 (mmol/L)	17.7 (15.6 - 20.6)	19.2 (17.6 - 66.78)	0.209
PTH (pg/mL)	392 (166.3 - 526)	450.7 (12.33 - 1369)	0.76
25-OH (vitamin D) (nmol/L)	22.7 (11.57 - 40.17)	30.82 (14.4 - 50.86)	0.416
Alkaline phosphatase (IU/L)	350 (172 - 624)	358 (114.5 - 880.5)	0.986
Phosphorus (mg/dL)	4.9 (3.2 - 6.1)	4.65 (3.38 - 5.8)	0.787

There is no Association between T3 Syndrome of the studied patients and different laboratory investigations as show in **(Table 5**).

Table 5 Association between T3 Syndrome of the studied patients and different laboratory investigations

Variables	Normal (n=35)	Low T3 Syndrome (n=6)	P value
CBC			
Hb (g/dL)	11.4 (9.3 - 12.4)	8.58 (7.2 - 10.9)	0.058
Anemia	4 (66.7%)	10 (28.6%)	0.069
WBCs ($x10^3$cells/µl)	5.8 (5.19 – 6.95)	8.44 (5.2 - 10.33)	0.155
PLT ($x10^3$cells/µl)	188 (172 - 238)	178.5 (98 - 346)	0.618
HCT (%)	34.4 (29.2 - 37.3)	26.85 (21.1 - 34.6)	0.050
MCV (fL)	88.5 (85 - 92.1)	92.35 (87.9 - 97.6)	0.210
Kidney function			
BUN (mg/dL)	114 (98 - 133)	114 (113 - 118)	0.726
Serum creatinine (mg/dL)	7.26 (5.2 - 9.44)	6.23 (5.9 - 7.4)	0.438
Sodium (mmol/L)	133 (133 - 135)	134.5 (133 - 136)	0.489
Potassium (mmol/L)	4.6 (3.7 - 4.9)	4.15 (3.38 - 4.66)	0.209
Total calcium (mg/dl)	8.9 (8 - 9.6)	9.75 (8.6 - 11.6)	0.337
Ionized calcium (mg/dl)	1.04 (0.9 - 1.2)	1.14 (0.98 - 1.6)	0.300
Liver function			
ALT (IU/L)	12 (10 - 27)	12.5 (9 - 13)	0.460
AST(IU/L)	19 (15 - 30)	17.5 (16 - 18)	0.395
Total protein (g/dL)	7.2 (6.4 - 7.5)	6.95 (6.6 - 7.1)	0.222
Albumin (g/dL)	3.6 (3.5 - 4.2)	3.9 (3.5 - 4)	0.956
Total bilirubin (mg/dL)	0.45 (0.3 - 0.6)	0.55 (0.3 - 0.8)	0.492
ABG			
pH	7.3 (7.2 - 7.3)	7.3 (7.3 - 7.3)	0.656
PCO_2 (mmHg)	32 (29.6 - 40.2)	34.9 (30.5 - 42.9)	0.618
PO_2 (mmHg)	127 (112 - 141)	114 (113 - 146)	0.726
Base Deficit (mmol/L)	-8.4 (-10.3 - -7.1)	-9.2 (-10.1 - -7.7)	0.726
HCO_3 (mmol/L)	18.25 (15.8 - 20.6)	18.1 (17 - 19.7)	0.850
PTH (pg/mL)	410.8 (169.1 - 553.4)	246.65 (37.9 - 881)	0.740
25-OH (vitamin D) (nmol/L)	24.9 (14.13 - 40.17)	9.72 (8.47 - 61.16)	0.197
Alkaline phosphatase (IU/L)	350 (123 - 624)	398 (172 - 775)	0.543
Phosphorus (mg/dL)	5.2 (3.5 - 6.4)	4.45 (3.2 - 5.7)	0.460

Discussion

The aim of the study was to evaluate thyroid functions in patients undergoing chronic HD in a specific dialysis unit. The study included 41 cases with a mean age of 15.17 ± 4.69 years, ranging from 6 to 23 years. The gender distribution showed a slightly higher percentage of females, with 46.3% males and 53.7% females, consistent with findings from similar studies by Kumar PR &Lim HI.[7,8]

However, other studies by Shaik L. & Sharma A., reported that higher male proportions, indicating variability across populations.[9,10]

In the current study, we observed that 65.9% of patients exhibited short stature, while 34.1% were underweight. Various factors contribute to longitudinal growth delay in CKD, including poor appetite, severe anorexia, anemia, imbalanced diet, recurrent hospital admissions, uremic toxins, and hyperparathyroidism, all of which impact bone mineralization.

Comparatively, the Alexandria study by Garbadi et al, reported that 38.8% of cases had short stature, with 29.4% being underweight. [11]

Another study by Garrido-Magaña et al. found that 66% of cases experienced growth problems, affecting height for age. [12]

Smith et al, showed that one-third of study patients had height below the 3rd percentile. [13]

In the current study, the most frequently detected cause of dialysis was bilateral atrophic kidneys in more than half of the cases (51.2%). This suggests a delay in diagnosing CKD until the stage of

bilateral atrophic kidneys in many diseases, without knowing the initial underlying cause. Other identified causes included single atrophic kidney (12.2%), focal segmental glomeruloscle-rosis (FSGS) (7.3%), nephronophthisis (4.9%), chronic tubulo-interstitial nephritis (4.9%), and various other conditions each accounting for smaller percentages.

The Alexandria study by Garbadi et al. identified congenital anomalies of the kidney and urinary tract (CAKUT) as the most prevalent cause of CKD, affecting 44.7% of cases, followed by obstructive uropathy (28.2%).[11]

Similar findings were reported by Safouh et al**.** where CAKUT was the leading cause (31.5%), along with other significant contributors such as obstructive uropathy, primary glomerulonephritis, reflux/urinary tract infection, familial/metabolic disorders, and unexplained causes. [14]

Regarding CBC results of the studied patients, the median hemoglobin (Hb) level was 11.3, with 18 cases exhibiting anemia defined as Hb levels below 11 g/dL.

The study by Hasan et al**.** supported our findings, reporting a high prevalence of anemia among CKD patients. They observed hemoglobin levels below 12 g/dL in a significant percentage of patients, with values ranging from 5.8 to 11.6 g/dL and a mean ± standard deviation of 9.2 ± 1.6 g/dL among all included patients. [15]

In the present study, we observed that 27 cases exhibited low sodium levels, potentially attributed to factors such as excessive inter dialytic water intake, inadequate water removal during HD, imbalanced diet, and dysregulation of thirst. Additionally, our study reported 2 cases with low potassium levels, which could be due to rapid shifts of potassium from the extracellular to the intracellular space secondary to correction of acidosis and increased PTH levels facilitating calcium entry into cells, thus affecting cellular permeability to potassium. Furthermore, 7 cases were found to have low calcium levels, possibly due to the kidneys' inability to produce active vitamin D.

Contrary to expectations, a study by Pańczyk-Tomaszewska et al**.** reported normal serum calcium levels in children under study and explained his results that may be due to increased PTH that reported in their investigation. [16]

In the present study concerning thyroid function tests, elevated TSH levels above 4 µIU/L were observed in 6 cases. Additionally, FT3 levels were noted in 12 cases, and low T3 levels were observed in 6 cases. Furthermore, 3 cases exhibited elevated T4 levels. These abnormalities may be attributed to It is noteworthy that all patients showed no abnormalities upon thyroid gland palpation.

The prevalence of THS abnormalities in patients with ESRD exhibits variability across studies. Garbadi et al, documented a prevalence of 14.1% for high TSH levels and 21.2% for low FT3 levels. [11] . Conversely, Toda et al. observed a lower prevalence of high TSH levels at 4%.[17]

Conversely, Kaptein et al. reported a higher prevalence of increased TSH levels, with 10.5% of cases falling within the range of five to ten mUl/l and 1% exceeding 10 mUl/l. [18]

Pan et al**.** noted a substantial prevalence of low T3 levels in 69.1% of patients, [19] while Kamal et al. found low T3 syndrome in 42% of individuals. [20]

Subclinical hypothyroidism, characterized by an elevated TSH level above 4mU/L with normal levels of T4 and T3, was observed in 6 cases in our study. The increased TSH levels in our patients may be attributed to the direct effect on the pituitary–thyroid axis and ineffective clearance of abnormal serum constituents, which affects TSH levels. Additionally, a decrease in clearance and an increase in the half-life of TSH may contribute to elevated levels.

Low T3 syndrome or non-thyroidal illness syndrome (NTIS), in ESRD is characterized by reduced levels of T3 despite normal T4 and TSH levels, as observed in 6 cases. This condition arises due to decreased synthesis of T3 from T4, influenced by chronic recurrent metabolic acidosis and reduced clearance of inflammatory cytokines, such as tumor necrosis factor, which inhibit the conversion of T4 to T3. Nutritional deficiencies common in ESRD also contribute to this syndrome. Additionally, dialysis procedures, medications such as steroids and beta-blockers, uremic toxin accumulation, and reduced renal clearance of thyroid hormones further disrupt thyroid hormone metabolism, collectively leading to low T3 levels.

Several studies have investigated the prevalence of thyroid dysfunction in patients with CKD, particularly those undergoing HD. Choncholet et

al. identified subclinical hypothyroidism in 9.5% of cases, [21] while Chaker et al. reported it in 9.2%, and Lo et al found a higher prevalence of 56% in an American study. [22] . Kamal et al. reported a prevalence of 46% for subclinical hypothyroidism in their study. [20]

Kaptein's studies found significant thyroid hormone abnormalities, including low T3, in his patients.[23] Bakkaloglu et al. emphasized the importance of thyroid function monitoring in children with ESRD due to the high incidence of low T3 levels. [24]

Similarly, Pan et al. reported that 626 (69.1%) of their cases had low T3 levels. Kamal et al. also found that 42% of their patients exhibited low T3 syndrome. Furthermore, an Italian study by Enia et al. conducted on 41 patients, demonstrated lower FT3 levels in their patients. [25]

In this study, concerning metabolic bone diseases in our patients, we found that 30 cases had PTH levels above normal (3 to 5 times the normal range) due to disturbances in the homeostasis of calcium, phosphate, and vitamin D. Additionally, 22 cases had alkaline phosphatase (ALP) levels above the normal range due to disturbances in bone mineral disease. Furthermore, 3 cases had phosphate levels above the normal range due to reduced renal phosphate excretion and inadequate treatment with phosphate binder agents.

This study reported that the median 25-hydroxy vitamin D (25OH VITD) was 23.74 (interquartile range between 12.66 - 42.81) nmol/L, with 15 cases exhibiting deficient 25OH VITD (<20), 9 cases insufficient 25OH VITD (20 - <30), and 17 cases sufficient 25OH VITD (30 - 100).These results align with the study by Sharaf et al. which focused on measuring serum PTH levels in children with ESRD undergoing regular HD , revealing elevated PTH and ALP levels. [26]

Similar findings were reported by Shroff et al. which noted low levels of 1,25-dihydroxy vitamin D (1,25(OH)2D) and vitamin D deficiency in children on dialysis, along with a high prevalence of 25OH VITD deficiency. [27]

These findings collectively underscore the complex interplay between PTH regulation, vitamin D metabolism, and bone health in pediatric patients with CKD undergoing dialysis.

Conclusion:

Patients with ESRD on chronic HD may experience alternation in thyroid function. The study reported 6 cases with subclinical hypothyroidism and 6 cases with low T3 syndrome. There were no other abnormalities in thyroid functions in our study. We recommend for regular monitoring of thyroid function in patient with CKD for early detection of any abnorm-alities. We need further researches on lager numb-ers in HD patient to know more about thyroid dysfunction in HD children.

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