ORIGINAL ARTICLE

Serum Electrolytes in Subclinical Hypothyroidism and Subclinical Hyperthyroidism

Shraddha B Pattanashetti[1], Pratibha Krishnappa[2]

Abstract

Background: Thyroid hormones have a central regulatory role in body hemodynamics, thermoregulation, and metabolism. The profound influence of thyroid hormones is observed on renal hemodynamics, glomerular filtration, renin-angiotensin-aldosterone system, and electrolyte balance. Thus, our main aim was to find out the electrolytes imbalance between subclinical hypothyroidism (SHO) and subclinical hyperthyroidism (SHE) and their correlation.

Materials and methods: In our study, newly diagnosed 50 SHO and 35 SHE cases were selected. Blood samples were collected to analyze serum electrolytes (sodium, potassium, and chloride) by Electrolyte Analyzer from Roche. Mean, median, standard deviation, minimum value, maximum value, standard error of mean, and values at 95% confidence interval are calculated for the parameters. And the correlation between serum electrolytes with serum TSH was assessed.

Results: There were no significant changes in levels of serum electrolytes in SHO and SHE, but the correlation between the levels of serum sodium and potassium with TSH showed little negativity or no changes in SHO and SHE, whereas the levels of serum chloride showed little positivity or no changes with TSH in SHO and little negativity or no changes with TSH in SHE.

Conclusion: Hypothyroid and hyperthyroid patients in subclinical conditions will be having electrolyte imbalances and should be regularly checked for serum electrolytes. Also, electrolyte disturbances need to be monitored and treated appropriately to prevent further complications.

Keywords: Chloride, Potassium, Subclinical hyperthyroidism, Subclinical hypothyroidism, Sodium.

Indian Journal of Medical Biochemistry (2021): 10.5005/jp-journals-10054-0167

Introduction

Thyroid hormones have a central regulatory mechanism on overall body functions, it affects renal hemodynamics, glomerular filtration rate (GFR) and electrolyte homeostasis. Sodium and potassium are components of the enzyme Na^+-K^+ ATPase, this enzyme is present on the cell membrane which helps in the transport of water and nutrients across the cell membrane.[1] Thyroid hormones regulate sodium-potassium pumps in most of the tissues.[2] Abnormalities in thyroid hormone synthesis, secretion, or actions are usually categorized as hypothyroidism, and hyperthyroidism, depending on the concentration of TSH, T3, and T4 in circulation. Hypothyroidism is a biochemical disorder characterized by a deficiency of thyroid hormones that results in a generalized slowing down of metabolic processes, which includes reduced renal blood flow, GFR, and absorption of sodium, potassium, chloride, and water are seen. Whereas hyperthyroidism is a biochemical entity resulting from the hypersecretion of thyroid hormones which, in turn, results in a generalized hyperactivation of metabolic processes.[3] The effects of thyroid hormones on renal function and electrolyte balances have not been well established.

The literature search has demonstrated variations in levels of serum electrolytes in overt cases of hypothyroidism and hyperthyroidism. Very few studies have been done in subclinical stages. Therefore, this study is taken up to assess and compare the alterations in the levels of serum electrolytes in subclinical hypothyroidism (SHO) and subclinical hyperthyroidism (SHE). We also assessed the correlation between TSH and serum electrolyte levels.

[1]Shree Veera Brahmendra Swamy Clinic, Kampli, Ballary, Karnataka, India

[2]Department of Biochemistry, ESIC Medical College and Post Graduate Institute of Medical Science and Research, Rajajinagar, Bengaluru, Karnataka, India

Corresponding Author: Shraddha B Pattanashetti, Shree Veera Brahmendra Swamy Clinic, Kampli, Ballary, Karnataka, India, Phone: +91 9742797553, e-mail: shraddhapattanashetti@gmail.com

How to cite this article: Pattanashetti SB, Krishnappa P. Serum Electrolytes in Subclinical Hypothyroidism and Subclinical Hyperthyroidism. Indian J Med Biochem 2021;25(1):1–4.

Source of support: Nil

Conflict of interest: None

Materials and Methods

The study was carried out in ESICMC-PGIMSR, Rajajinagar, Bengaluru. After taking approval of the Institutional Ethics Committee and written consent from all subjects, newly diagnosed 50 SHO and 35 SHE cases were selected from the OPD of the Department of Medicine. Among 50 SHO cases, 40 (80%) are females and 45 (90%) patients are in the age-group of 21–60 years and among 35 SHE cases 28 (80%) are females, 33 (94.3%) patients are in the age-group of 21–60 years.

Criteria for the selection of subclinical cases are based on laboratory investigations as follows:

Cases	*Criteria for selection of cases*[4]
SHO	Patients with TSH >10 mIU/mL and with normal FT_3 and normal FT_4.
SHE	Patients with TSH <0.1 mIU/mL and with normal FT_3 and normal FT_4.

Inclusion Criteria

Newly diagnosed cases of SHO and SHE based on laboratory investigations, in the age-group of 18–65 years, are included in the study.

Exclusion Criteria

Patients on mineral supplementation, antithyroid drugs, diuretics, pregnancy, renal disease, hepatic disease, cardiovascular disease, stroke, or other neurological disorders or depressions, critically ill patients admitted in intensive care units, any other medications like amiodarone, lithium, statins, corticosteroids, beta-blockers, ACE inhibitors cyclosporine, NSAIDs are known to affect the lipid and mineral profile are excluded from the study.

Statistical Analyzis

Mean, median, standard deviation, minimum value, maximum value, standard error of mean, and values at 95% confidence interval are calculated for the parameters. The parameters are compared for subclinical cases of thyroid disorders by using unpaired Student's "*t*" test. The relationship between the parameters is assessed by Pearson's coefficient. p value < 0.001 will be considered as statistically significant. The data are analyzed using the statistical package SPSS 20.

Collection of Blood Samples

Under aseptic precautions, 5 mL fasting venous blood is collected from the cases and transferred into plain sterile vacationer tubes, allowed to clot at 37°C, and then centrifuged at 3,000 rpm for 10 minutes to separate the serum. The serum was separated and immediately used for analyzis.

Analyzis of Samples

A separated serum will be fed into fully/semi-automated analyzers (Access II from Beckman Coulter, Electrolyte Analyzer from Roche for which Internal and External Quality Assurance Programs are maintained) are used for the analyzis of thyroid profile (serum TSH, fT_3, fT_4), and serum electrolytes (sodium, potassium, chloride) where serum TSH, fT_3, fT_4 measured by Chemiluminescence Immunoassay method by access II auto analyzer—Beckman Coulter[4,5] and serum sodium, potassium, chloride measured by ion-selective electrode by electrolyte semi auto analyzer.

Results

Figures 1 and 2 show that, in SHO cases, there is little negative or no correlation between serum TSH levels and serum sodium and potassium levels, and there is little positive or no correlation between serum TSH levels and serum chloride levels.

Figure 3 shows that, in SHE cases, there is little negative or no correlation between serum TSH levels and serum sodium, potassium, and chloride levels (Tables 1 and 2).

Discussion

The thyroid hormone acts as a central regulator of body functions. Disorders of thyroid function lead to electrolyte disturbances. Sodium and potassium are important components of the enzyme Na-K ATPase, an enzyme on the cell membrane which helps in the transport of water and nutrients through the cell membrane. In hypothyroidism, there will be a deficiency of the enzyme Na-K ATPase because of suppressed plasma renin activity (PRA) and plasma aldosterone (PA) due to dysfunction of juxtaglomerular cells and glomerulosa cells which leads to exaggerated sodium and potassium excretion. In an international study done in hypothyroid cases, the serum sodium and potassium levels were markedly decreased, they also correlated the levels of serum sodium, potassium, and chloride with TSH where serum sodium and potassium showed negative correlation with TSH but serum chloride was positively correlated. In our study, we observed no significant changes in the levels of serum sodium, potassium, and chloride in SHO, and the correlation between the levels of serum sodium, potassium, and chloride and TSH showed little negativity or no correlation between serum sodium and potassium with TSH and little positivity or no changes between serum chloride with TSH in SHO.

Hypokalemia seen in hyperthyroidism is because of potassium shift in the cell and enhanced renal potassium excretion. Mechanism of serum sodium and chloride in hyperthyroidism is

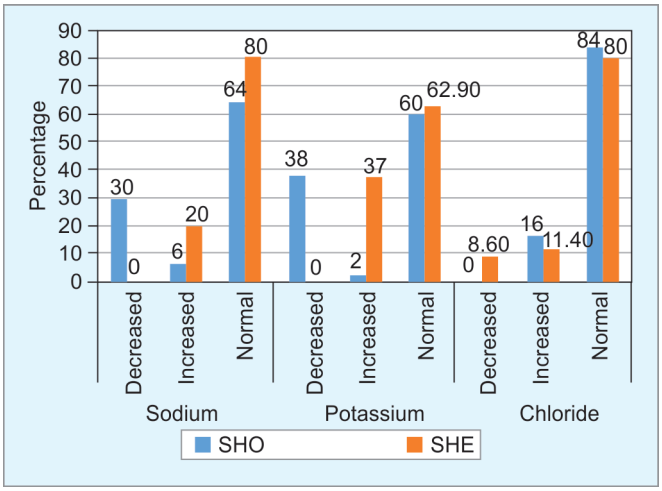

Fig. 1: Serum electrolytes levels in SHO and SHE

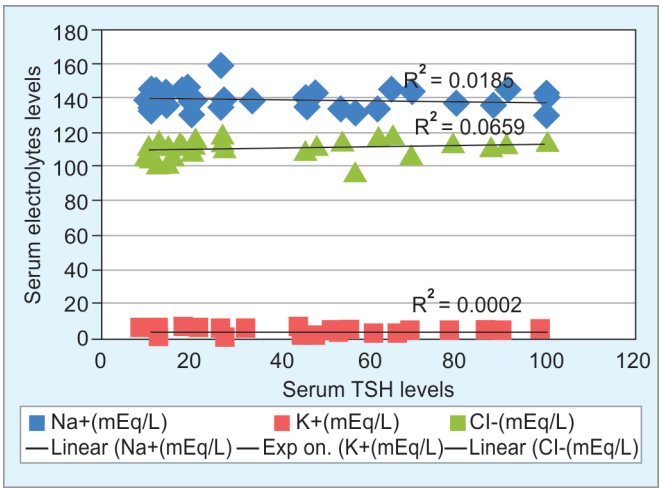

Fig. 2: Correlation between serum TSH and serum electrolytes levels in SHO

not well established. Bharti et al.[6] in his study done on subclinical hyperthyroid patients show that there were no significant changes in the levels of serum sodium, potassium, and chloride which is similar to my study. Along with this in our study, the correlation between the levels of serum sodium, potassium, and chloride with TSH showed little negativity or no changes in SHE. Prospective studies with large sample size and long-term follow-up in patients with newly diagnosed subclinical thyroid disorders are necessary which may help to determine whether the electrolyte disorder really exists with thyroid disorders and will it resolves itself after starting supplementary substitution.

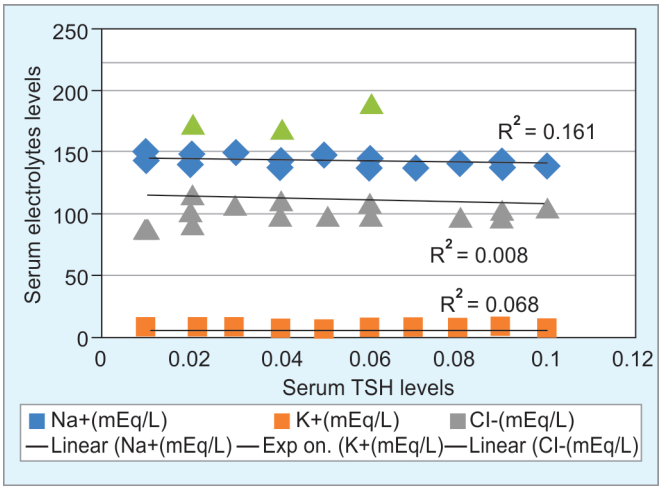

Fig. 3: Correlation between serum TSH and serum electrolytes levels in SHE

Conclusion

It has been shown in our study that there is no significant changes in the serum sodium, potassium, and chloride levels in SHO and SHE and no correlation between the levels of serum sodium, potassium, and chloride with TSH which showed little negativity or no correlation between serum sodium and potassium with TSH in SHO and SHE, respectively, little positivity or no changes between serum chloride with TSH in SHO and little negativity or no changes in SHE. This suggests that hypothyroid and hyperthyroid patients in subclinical conditions will be having electrolyte imbalances and should be regularly checked for serum electrolytes. Also, electrolyte disturbances need to be monitored and treated appropriately to prevent further complications.

Table 1: Thyroid profile and serum electrolytes levels in SHO and SHE

	SHO (n = 50)							
	Mean	*SD*	*Minimum*	*Maximum*	*Median*	*SE*		*95% CI*
TSH (μIU/mL)	32.1	27.65	10.6	100.0	18.9	3.911	24.31	39.95
FT3 (pg/mL)	3.3	0.53	2.2	4.5	3.4	0.075	3.17	3.47
FT4 (ng/dL)	0.7	0.19	0.2	1.1	0.7	0.027	0.61	0.71
Na^+ (mEq/L)	139.9	5.83	130	160	141	0.824	138.2	141.53
K^+ (mEq/L)	4.04	0.99	1.1	5.2	4.2	0.140	3.76	4.32
Cl^- (mEq/L)	110.9	4.61	97	120	111	0.652	109.7	112.26
	SHE (n = 35)							
	Mean	*SD*	*Minimum*	*Maximum*	*Median*	*SE*		*95% CI*
TSH (μIU/mL)	0.05	0.03	0.01	0.10	0.04	0.005	0.037	0.056
FT3 (pg/mL)	3.90	0.72	2.53	5.91	3.78	0.122	3.659	4.148
FT4 (ng/dL)	1.37	0.57	0.54	3.20	1.32	0.096	1.182	1.565
Na^+ (mEq/L)	142.9	3.53	137	152	142	0.597	141.7	144.19
K^+ (mEq/L)	4.93	0.46	3.9	5.9	4.9	0.078	4.775	5.088
Cl^- (mEq/L)	112.7	24.17	90	191	106	4.085	104.5	120.88

Table 2: Serum electrolytes levels in SHO and SHE

		SHO		*SHE*		
Serum electrolytes	*Levels*	*n*	*%*	*n*	*%*	*p value*
Serum sodium	Decreased	15	30.00	0	0.00	$p < 0.001$
	Increased	3	6.00	7	20.00	
	Normal	32	64.00	28	80.00	
Serum potassium	Decreased	19	38.00	0	0.00	$p < 0.001$
	Increased	1	2.00	13	37.10	
	Normal	30	60.00	22	62.90	
Serum chloride	Decreased	0	0.00	3	8.60	$p = 0.098$
	Increased	8	16.00	4	11.40	
	Normal	42	84.00	28	80.00	

References

1. Murgod R, Amdsoans G. Changes in electrolyte and lipid profile in hypothyroidism. Int J Life Sci Pharma Res 2012;2(3):185–194. Rao GM. 1992. Serum electrolytes and osmolality in diabetes.
2. Ismail BF, Edelman IS. The mechanism of the calorigenic effect of thyroid hormone stimulation of Na+ + K+ activated adenosinetri phosphatase activity. J Gen Physiol 1971;57(6):710. DOI: 10.1085/jgp.57.6.710.
3. Abebe N, Kebede T, Wolde M. Assessment of renal function and electrolytes in patients with thyroid dysfunction in Addis Ababa, Ethiopia: a cross sectional study. Pan Afr Med J 2016;24:338. DOI: 10.11604/pamj.2016.24.338.8455.
4. Abdel-Gayoum AA. Dyslipidemia and serum mineral profiles in patients with thyroid disorders. Saudi Med J 2014;35(12): 1469–1472.
5. Access 2 manual for Hybritech TSH from 2006 Beckman Coulter, Inc.
6. Bharti A, Shrestha S, Rai R, et al. Assessment of serum minerals and electrolytes in thyroid patients. Int J Adv Sci Res 2015;1(06):259–263. DOI: 10.7439/ijasr.v1i6.2189.