Section: Biochemistry

Original Research Article

STUDY OF SERUM ELECTROLYTES (NA+AND K+) IN PATIENTS WITH THYROID DYSFUNCTION

Jigar A Parmar[1], Pratik Raghavani[2], Margit Gajjar[3], Dipti Gajjar[4]

[1]*Associate Professor, Department of Biochemistry, GMERS Medical College, Gotri, Vadodara, Gujarat, India.*
[2]*Associate Professor, Department of Biochemistry, B.J.Medical College, Ahmedabad, Gujarat, India.*
[3]*Associate Professor, Department of Biochemistry, GMERS Medical College, Rajpipla, Gujarat, India.*
[4]*Assistant Professor, Department of Pathology, GMERS Medical College, Rajpipla, Gujarat, India.*

Received *: 27/12/2023*
Received in revised form *: 20/02/2024*
Accepted *: 07/03/2024*

Corresponding Author:
Dr. Dipti Gajjar
Department of Pathology, GMERS Medical College, Rajpipla, Gujarat, India.
Email: diptikrs@gmail.com

DOI: 10.5530/ijmedph.2024.1.95

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2024; 14 (1); 518-521

ABSTRACT

Background: Thyroid dysfunction is linked to a variety of electrolyte issues in a lot of literature. Hyponatraemia is said to result from increased renal water retention mediated by vasopressin in severe hypothyroidism and myxoedema. On the other hand, patients with thyrotoxicosis were noted to have hypokalaemia, hypomagnesaemia, and hypercalcaemia. Therefore, the current study was conducted to evaluate the changes in serum electrolyte levels in hyperthyroid and hypothyroid conditions.

Materials and Methods: The present cross-sectional investigation was conducted in the Clinical Chemistry laboratory, namely in the Department of Biochemistry, at GMERS Medical College and General Hospital in Vadnagar, Gujarat. 120 patients made up the study group. Based on their thyroid hormone profile, the patients were classified into two groups: those who were hyperthyroid and those who were hypothyroid. In groups A and B, respectively, there were 60 individuals with hypothyroidism and 60 with hyperthyroidism who had received a diagnosis. After being separated, the serum was immediately used for analysis. Using a Easylyte Electrolyte analyzer, serum electrolyte (Na+, K+) was analyzed.

Results: The mean TSH levels for patients with hypothyroidism were 50.10 ± 40.41 and for those with hyperthyroidism were 0.15 ± 0.16, with a statistically significant difference between the two groups. Sodium levels were noticeably greater in patients with hyperthyroidism, and the difference demonstrated a significant statistical difference. Although patients with hyperthyroidism had lower potassium values than those with hypothyroidism, There was no statistically significant disparity observed between the two groups.

Conclusion: This means that subclinical hypothyroid and hyperthyroid individuals will likely have electrolyte abnormalities and should have their serum electrolytes evaluated frequently. Additionally, electrolyte abnormalities must be observed.

Keywords: Hypothyroidism, Myxoedema, Serum Electrolytes, Thyroid Dysfunction.

INTRODUCTION

Normal skeletal system development and maturation depend on thyroid hormones. Disturbances in calcium and phosphorus homeostasis are typically linked to thyroid dysfunction. Secondary osteoporosis has a major role in thyroid diseases. In some research, serum calcium and phosphorus levels are normal, but in other studies, hypothyroidism is associated with lower levels. Even though the alterations in calcium and magnesium that accompany thyroid diseases may be minor, the patient will ultimately benefit from these disruptions.[1,2]

Thyroid hormones regulate renal hemodynamics, glomerular filtration rate (GFR), and electrolyte homeostasis as part of a fundamental regulatory mechanism that controls all bodily processes. The Na+-K+ ATPase enzyme, located on the cell membrane, facilitates the transport of nutrients and

water through the membrane. It consists of sodium and potassium. Thyroid hormones regulate the majority of sodium-potassium pumps in tissues.[3,4]
Subclinical hypothyroidism affects around 10% of adults, presenting with elevated levels of thyroid-stimulating hormones (TSH) and normal levels of free thyroxine (FT4) in the bloodstream. The recommended reference range for T4 is 77–155 nmol/l, T3 is 1.2–2.8 nmol/L, and TSH is 0.3–4 mU/l.3 Deviation of hormone levels from the usual range indicates either hyperthyroidism or hypothyroidism.
Hypothyroidism is one of the most widespread endocrine disorders globally. In India, the incidence of hypothyroidism is approximately 10-11%. Hypothyroidism arises from a reduction in the amounts of thyroid hormones and is one of the prevalent endocrine illnesses. Hypothyroidism, characterised by reduced thyroid gland function, causes cognitive and physical deceleration due to a decline in the basal metabolic rate. The incidence of spontaneous hypothyroidism ranges from 1% to 2% and is higher in older women. Additionally, it is ten times more prevalent in women compared to men.[5,6]
Thyroid dysfunction is linked to a variety of electrolyte issues in a lot of literature. Hyponatraemia is said to result from increased renal water retention mediated by vasopressin in severe hypothyroidism and myxoedema. On the other hand, patients with thyrotoxicosis were noted to have hypokalaemia, hypomagnesaemia, and hypercalcaemia.[7] Therefore, the current study was conducted to evaluate the changes in serum electrolyte levels in hyperthyroid and hypothyroid conditions.

MATERIAL AND METHODS

The present cross-sectional investigation was conducted in the Clinical Chemistry laboratory, situated in the Department of Biochemistry at GMERS Medical College and General Hospital in Vadnagar, Gujarat. The six-month trial was conducted from February-22 to July-22. 120 patients made up the study group. Based on their thyroid hormone profile, the patients were classified into two groups: those who were hyperthyroid and those who were hypothyroid. In groups A and B, respectively, there were 60 individuals with hypothyroidism and 60 with hyperthyroidism who had received a diagnosis. Patients who fulfilled the specified criteria were chosen from the inpatient and outpatient clinics of the Medicine department. The study's objective was conveyed to the patients who fulfilled the eligibility criteria, and written informed consent was acquired.
Of the total 120 patients, there were 73 males and 47 females. The age range was taken as 20 – 60 years. Criteria for the selection of subclinical cases are based on laboratory investigations as follows:
The study's inclusion criteria called for patients of hypothyroidism and hyperthyroidism that had just received a lab-based diagnosis and were between the ages of 20 and 60. Patients who had not had any treatment for a thyroid disease or a related ailment were particularly chosen for our study.
Patients receiving mineral supplements, taking antithyroid medication, using diuretics, being pregnant, suffering from renal disease, hepatic disease, cardiovascular disease, stroke, or other neurological disorders or depressions, critically ill patients admitted to intensive care units, and taking any other medications known to affect the lipid and mineral profile, such as amiodarone, lithium, statins, corticosteroids, beta-blockers, ACE inhibitors, cyclosporine, and NSAIDs were excluded from the study.
During patient interviews, demographic information such age, sex, and occupation were recorded. Records of similar complaints treated previously and currently were noted. Patients had comprehensive medical evaluations, during which their vital signs and other clinical symptoms indicative of thyroid dysfunction were documented. A symptom assessment was conducted.
Following aseptic protocols, a volume of 5 mL of blood was collected from the subjects after fasting. The blood was then transferred into clean and sterile tubes and left to coagulate at room temperature. Afterward, the tubes were centrifuged at a speed of 3,000 rpm for a duration of 10 minutes to separate the serum. Following the separation process, the serum was promptly utilised for examination. An Easylyte Electrolyte analyzer was utilised to analyse the serum electrolyte levels of sodium (Na+) and potassium (K+).
The data analysis was conducted using the statistical software SPSS 15.0. The current study has conducted a descriptive statistical analysis. The significance of study parameters on a continuous scale between two groups was determined using a Student t-test.

RESULTS

A cohort of 120 patients diagnosed with both hypo- and hyperthyroidism were selected for the investigation. The clinical data was analyzed to ascertain the age and sex distribution of hypothyroidism and hyperthyroidism among the selected patients. Patients with hyperthyroidism had a mean age of 42.40 years, compared to 49.09 years for those with hypothyroidism. From a statistical perspective, there was no discernible disparity in age between the two groups. (p value > 0.05). As a result of the analysis of both sexes, it was established that males were more likely than females to have the disorders.
According to the data in the table, the mean TSH levels for patients with hypothyroidism were 50.10 ± 40.41 and for those with hyperthyroidism were

0.15 ± 0.16, with a statistically significant difference between the two groups. Additionally, the mean T3 levels in patients with hypothyroidism were 55.12 ± 33.54 and those in patients with hyperthyroidism were 130.10 ± 70.31, with a statistically significant difference between the two groups. Additionally, the mean T4 levels in patients with hypothyroidism were 2.50 ± 0.14 and those with hyperthyroidism were 3.16 ± 3.98, with a statistically significant difference between the two groups.

The electrolyte analysis demonstrated a substantial statistical difference in sodium levels between patients with hyperthyroidism, whose sodium levels were notably higher. Although patients with hyperthyroidism had lower potassium values than those with hypothyroidism, There was no discernible disparity of statistical significance between the two groups.

Table 1: Thyroid profile and serum electrolytes levels in Serum hypothyroidism and Serum hyperthyroidism

	Group	Mean ± Std. deviation
TSH (ng/dl)	Group A	50.10 ± 40.41
	Group B	0.15 ± 0.16
T3 (ng/dl)	Group A	55.12 ± 33.54
	Group B	130.10 ± 70.31
T4 (ng/dl)	Group A	2.50 ± 0.14
	Group B	3.16 ± 3.98
Sodium	Group A	142.65 ± 6.95
	Group B	145.33 ± 6.11
Potassium	Group A	6.69 ± 0.77
	Group B	5.65 ± 0.22

DISCUSSION

The central regulator of several bodily processes, including metabolism and hemodynamics, is the thyroid hormone. As a result, it affects the handling of electrolytes, glomerular filteration, and renal hemodynamics. When a person has hypothyroidism, their body produces inadequate thyroid hormones, which causes their metabolism to slow down and their electrolyte balance to become disturbed.[8,9]

Thyrotropin-releasing hormone (TRH) triggers the synthesis of thyroid stimulating hormone (TSH) in the anterior pituitary. This hormone, in turn, binds to the TSH receptor in the thyroid gland, leading to the production of thyroglobulin, thyroid peroxidase, sodium-iodide symporter (NIS) protein, and thyroxin. Additionally, TRH inhibits TSH synthesis when thyroid hormone is present, creating a classic endocrine negative feedback loop. The thyroid gland regulates numerous metabolic processes by releasing the hormones thyroxine (T4) and triiodothyronine (T3), which determine the body's metabolic rate and the rate of energy production.[10,11]

A worldwide investigation revealed that individuals with hypothyroidism exhibited notably reduced levels of blood salt and potassium. There were negative relationships between the levels of serum sodium and potassium and TSH.[12] Our study discovered that the levels of blood sodium and potassium in individuals with serum hypothyroidism (SHO) did not experience significant changes. Furthermore, the correlation analysis between the levels of serum sodium, potassium, and thyroid-stimulating hormone (TSH) indicated a weak negative connection or no correlation at all between serum sodium and potassium with TSH in cases of serum hypothyroidism.

Plasma renin, angiotensin II, and serum angiotensin converting enzyme levels decrease concomitantly with the decline in thyroid hormone levels. Additionally, the RAAS activity has decreased overall. Afferent arteriolar vasoconstriction and efferent arteriolar vasodilatation are the effects of this. This may lead to proximal convoluted tubule (PCT) hypoperfusion and tepid Na reabsorption as a result. The apical Na-H exchanger (NHE), the Na-Pi co-transporter, and the basolateral NA/K ATPase all display diminished activity. Deactivating these transporters reduces the proximal reabsorption.

CONCLUSION

Our investigation has demonstrated that there are changes in the blood sodium, potassium levels in subclinical hypothyroidism (SHO) and subclinical hyperthyroidism (SHE) and that there is no relationship between these levels and the TSH, which exhibited only mild negative. This means that subclinical hypothyroid and hyperthyroid individuals will likely have electrolyte abnormalities and should have their serum electrolytes evaluated frequently. Additionally, electrolyte abnormalities must be observed.

REFERENCES

1. Cardoso, L. F.; Maciel, L. M.; de Paula, F. J. J. A. B. d. E.; Metabologia. The multiple effects of thyroid disorders on bone and mineral metabolism. 2014, 58, 452-463.
2. Ramón, A. M. In Tilte2020; Elsevier.
3. Schliebe, N.; Strotmann, R.; Busse, K.; Mitschke, D.; Biebermann, H.; Schomburg, L.; Köhrle, J.; Bär, J. r.; Römpler, H.; Wess, J. r. J. A. J. o. P.-R. P. V2 vasopressin receptor deficiency causes changes in expression and function of renal and hypothalamic components involved in electrolyte and water homeostasis. 2008, 295, F1177-F1190.

4. Suhail, M. J. J. o. c. m. r. Na+, K+-ATPase: ubiquitous multifunctional transmembrane protein and its relevance to various pathophysiological conditions. 2010, 2, 1.
5. Razvi, S.; Bhana, S.; Mrabeti, S. J. J. o. t. r. Challenges in interpreting thyroid stimulating hormone results in the diagnosis of thyroid dysfunction. 2019, 2019.
6. Pediatrics, A. A. o.; Rose, S. R.; Endocrinology, S. o.; Genetics, C. o.; Association, A. T.; Brown, R. S.; Committee, P. H.; Pediatrics, L. W. P. E. S. J. Update of newborn screening and therapy for congenital hypothyroidism. 2006, 117, 2290-2303.
7. Basu, G.; Mohapatra, A. J. I. j. o. e.; metabolism. Interactions between thyroid disorders and kidney disease. 2012, 16, 204.
8. Capasso, G.; De Tommaso, G.; Pica, A.; Anastasio, P.; Capasso, J.; Kinne, R.; De Santo, N. J. M.; Metabolism, E. Effects of thyroid hormones on heart and kidney functions. 1999, 25, 56-64.
9. Van Hoek, I.; Daminet, S. J. G.; endocrinology, c. Interactions between thyroid and kidney function in pathological conditions of these organ systems: a review. 2009, 160, 205-215.
10. Neumann, S.; Raaka, B. M.; Gershengorn, M. C.: Constitutively active thyrotropin and thyrotropin-releasing hormone receptors and their inverse agonists. In Methods in enzymology; Elsevier, 2010; Vol. 485; pp 147-160.
11. Fekete, C.; Lechan, R. M. J. F. i. n. Negative feedback regulation of hypophysiotropic thyrotropin-releasing hormone (TRH) synthesizing neurons: role of neuronal afferents and type 2 deiodinase. 2007, 28, 97-114.
12. Santos, N. C.; Costa, P.; Ruano, D.; Macedo, A.; Soares, M. J.; Valente, J.; Pereira, A. T.; Azevedo, M. H.; Palha, J. A. J. J. o. t. r. Revisiting thyroid hormones in schizophrenia. 2012, 2012.