

CHANGES IN ELECTROLYTE IN HYPOTHYROIDISM

GULAB KANWAR, KUSUM BALA JAIN, JITENDRA JAIN, RAHUL KABRA
KSHETRAPAL SINGH SHEKHAWAT & ROHIT JAIN

Department of Biochemistry, Government Medical College, Kota, Rajasthan, India

ABSTRACT

Introduction

Thyroid hormone is a central regulator of body function. Thyroid hormone perform a wide array of metabolic functions including regulation of lipid, carbohydrates, protein and electrolyte metabolism. Disorders of the thyroid functions are considered to be a cause of electrolyte disorders. So our aim is to assess the levels of serum electrolytes in patients with thyroid disorders.

Material and Method

This study was conducted in Department of Biochemistry, GMC kota. 50 patient samples and 50 control samples were taken. Serum was separated and serum electrolyte levels were estimated by fully Automated Analyzer ERBA EM 360. Thyroid estimation was done on Roche Chemiluminescence Analyzer. Data was analyzed using Mann-Whitney U test.

Results

Disorders of thyroid hormone increases with age and females are predominantly affected. All the variables are significantly decreased in cases than controls except TSH and chloride which are significantly increased in cases.

Conclusions

Hypothyroidism is associated with electrolyte disturbances, so need to be monitored and treated appropriately in hypothyroid patients.

KEYWORDS: Hypothyroidism, Electrolytes, Sodium, Potassium, Chloride

INTRODUCTION

Electrolytes play an important role in body processes, such as controlling fluid levels, acid base balance (pH), nerve conduction, blood clotting and muscle contraction.¹ Thyroid disease is common in general population, and the prevalence increases with age. In India 42 million people are suffering from thyroid disease; Hypothyroidism being the commonest thyroid disorder.²

Thyroid hormone is a central regulator of body hemodynamics, thermoregulation and metabolism. It therefore has an influence on renal hemodynamics, glomerular filtration as well as the renin-angiotensin-aldosterone system and renal electrolyte handling. Thyroid hormones perform a wide array of metabolic functions including regulation of lipid, carbohydrates, protein and electrolyte metabolism. While the effect of thyroid hormones on lipid metabolism is well

known, the effect of electrolytes are not been well established.³

Sodium and potassium are important components of enzymes. Na⁺-K⁺-ATPase, which is an enzyme present on cell membrane that helps in transport of water and nutrients across the cell membrane.⁴ Thyroid hormones regulate the activity of Na⁺-K⁺ pumps inmost of the tissues.⁵

We therefore want to investigate the effects of thyroid stimulating hormone(TSH) on serum electrolytes in a broad spectrum of patients admitted to hospitals.

MATERIAL AND METHODS

This study was performed in the Department of Biochemistry, Government Medical College, Central Laboratory NMCH and MBS Hospital Kota from period of September 2014 to August 2015.50 patient samples and 50 control samples were taken.

Inclusion Criteria

- Patients with newly diagnosed hypothyroidism.
- Age group between 10 years to 70 years

Exclusion Criteria

Patients with the following diseases were excluded from the study

- Patients on thyroxine treatment
- Patients on hypolipidemic drugs, antihypertensives, steroids.
- Patients with renal disease, liver disease, cardiovascular disease and diabetes mellitus.
- Pregnancy and patients on drugs.
- Patients with gout
- Patients not willing to participate in study.

After explaining the type of study, written consent was taken from all the subjects. A 12-hour fasting period, venous blood samples were collected from all the cases and controls. Serum was separated and serum sodium and potassium levels were estimated by fully Automated Analyzer ERBA EM 360. Thyroid estimation was done on Roche Chemiluminescence Analyzer.

Statistical Analysis

Statistical analysis was done using suitable statistical tool. Data was estimated on excel sheet and analysed statistically. Quantitative data was summarized in the form of MEAN \pm SD and differences in means of both the groups were analyzed using Mann-Whitney U test. The P value $<.05$ was taken as significant.

RESULTS

Table 1: Distribution of Age Groups

Age in years	Cases		Controls	
	No.	%	No	%
11-20	7	14	3	6
21-30	5	10	13	26
31-40	16	32	13	26
41-50	11	22	7	14
51-60	9	18	11	22
61-70	1	2	3	6
71-80	1	2	0	0
Total	50	100.0	50	100.0
Mean ± SD	39.84 ± 14.16		39.94 ± 15.25	

Table 2: Table of Gender Distributions

Gender	Cases		Controls	
	No	%	No	%
Male	8	16	9	18
Female	42	84	41	82
Total	50	100.0	50	100.0

Table 3: Comparison of Variables in Cases and Controls

Lab Variables	Cases (Mean ± SD)	Control (Mean ± SD)	P-Value
T3	0.9761± 0.43	1.37±0.25	<0.001
T4	6.37± 2.65	8.95±1.64	<0.001
TSH	51.27±110.4	2.48±1.11	<0.001
Sodium	129.66±11.83	139.86±3.23	<0.001
Potassium	3.93±0.31	5.92±17.34	<0.05
Chloride	105.98±1.36	99.12±1.62	<0.001

Table no.1 depicts the distribution of age (years) group between cases and controls. Shows mean age 39.84 ± 14.16 (years) in cases and 39.94 ± 15.25 (years) in controls.

Table no.2 depicts the gender distribution. Shows 8(16%) males and 42(84%) females in 50 cases and 9(18%) males and 41(82%) females in 50 controls

Table no.3 depicts the comparison of variables in cases and controls, showing all the variables are significantly decreased in cases than controls except TSH and chloride which are significantly increased in cases.

DISCUSSIONS

Hypothyroidism is a condition in which the body suffers from insufficient thyroid hormone. It is one of the most prevalent endocrine disorder and seen more in women than men. We also observed more female cases than males. In our study 84% were females and 16% were males in cases, which is in accordance with the previous studies.^{6,7} The higher prevalence of thyroid disease in women suggests that estrogen might be involved in pathophysiology of thyroid dysfunction. Estradiol has an antagonistic effect on the hormones T3 and T4. The reason is estradiol competes with T3 and

T4 for binding sites on the receptor proteins.⁸ Moreover, estradiol also limits the thermogenic action of T4 and promotes storage of fat.

Disorders of the thyroid glands are considered to be a cause of electrolyte disturbances. Hyponatremia is the most common electrolyte abnormality encountered in clinical practice.⁹

From our study it was also observed that serum sodium as well as the potassium levels were markedly decreased in cases as compared to healthy controls that was in accordance to the study done by Murgod R et al.⁴ Sodium and potassium are important components of the enzyme Na-K-ATPase, which is a cell membrane enzyme that helps in transport of water and nutrients across the cell membrane. Thyroid hormone regulates the activity of this pump in most of tissues.⁵ In hypothyroidism, because of low potassium levels, and because of deficiency of thyroid hormones, this enzyme is affected, resulting in accumulation of water inside the cells and causing edema.

According to Stura T et al plasma renin activity (PRA) and plasma aldosterone (PA) may be suppressed in hypothyroidism probably due to dysfunction of juxtaglomerular cells and glomerulosa cells respectively and the possibility that suppression of PRA and PA in patients with hypothyroidism is related to exaggerated sodium excretion and decreased potassium excretion cannot be ruled out.¹⁰ Study done by Christoph Schwarz et al observed association between thyroid hormones and electrolyte disorders. In his study there was decreased sodium and increased potassium levels in hypothyroidism. According to him electrolyte abnormalities are relevant only in marked hyper and hypothyroidism.¹¹ though our study is not in conformity with the study done by Christoph where he observed decreased sodium and increased potassium in hypothyroidism.

Sodium and chloride are interdependent and changes in sodium ions will also be reflected in the chloride ions. It is postulated that hormones which are involved in ECFV (Extracellular Fluid Volume) regulation act on renal sodium transporters may also modulate the renal chloride transporters.¹² Besides the classic hormones, such as aldosterone, that are known to be involved in the regulation of NaCl transport by the kidney other hormones, such as thyroid hormone, are also capable of regulating the ECFV via modulation of nephron ion and fluid transport.¹³ Importantly, thyroid hormone modulates the expression of Na⁺/K⁺ ATPase mRNA and protein and hence regulates the activity of this critical component of renal sodium transport.¹⁴ In proximal tubule, thyroid hormone acts on the Na⁺/H⁺ exchanger to change intratubular acidification dynamics. Thus, the thyroid hormones act by regulating the expression of different sodium and chloride transporters in the kidney is plausible in the light of its involvement in increasing renal fluid reabsorption.¹⁵ Therefore there is increased chloride level in hypothyroidism patients.

CONCLUSIONS

The present study was designed to evaluate the changes of serum electrolytes in hypothyroid patients. In this study we conclude that there was a significant decrease in serum electrolytes except serum chloride which was significantly increased in hypothyroid patients. This suggests that hypothyroid patients should be regularly checked for electrolytes. Early detection and treatment can prevent further complications.

REFERENCES

1. Rao GM. Serum electrolyte and osmolality in diabetes mellitus. *Indian J Med Sci* 1992; 46(10): 301-303

2. Unnikrishanan AG, Menon UV. Thyroid disorder in india: An epidemiological perspective. *Indian J endocrinal metab* 2011; 15: 78-81.
3. Mariani LH, Berns JS. The renal manifestations of thyroid disease. *J Am Soc Nephrol* 2012;23(1);22-6
4. Murgod R, Soans G. Changes in electrolyte and lipid profile in hypothyroidism. *International Journal of life science and pharma research* 2012; 2(3): 185-194
5. Ismail BF, Edelman IS. The mechanism of the calorogenic effect of thyroid stimulation of Na⁺-K⁺ activated adenosine triphosphatase activity. *J gen physiol* 1971; 57:710
6. Shaikh BA. Clinical features of primary hypothyroidism: a year experience at Chandka medical college, Larkana. *Medical Channel*. 2008; 14 (1): 72-75.
7. Shilpashree MK, Ravi BV, Vedavathi. Serum Lipoprotein (a) and lipid profile in hypothyroidism, *J Clin Biomed Sci* 2014;4(1):235-239
8. Vasudevan DM, Shreekumari S, Vaidyanathan K. *Textbook of Biochemistry* .Jaypee Brothers Medical publisher, 7th edition 2013;664-671
9. Kargili A, Turgut FH, Karakurt F, Kasapoglu B, Kanbay M, Akcay A.A forgotten but important risk factor for severe hyponatremia : Myxoedema coma, *clinics(Sao-Paulo)*2010; 65:447-448
10. Satura T, Kitajima W, Hayashi M, Kato E, Matsuki S. Renin and aldosterone in hypothyroidism: Relation to excretion of sodium and potassium. *Clin endocrinol*.1980; 12:483-489
11. Schwarz C, Leichtle AB, Arampatzis S, Fiedler GM, Zimmermann H, Exadaktylos AK, Lindner G. Thyroid function and serum electrolytes: does an association really exist?. *The European Journal of Medical Science*. 2012; 142: w13669.
12. Santos Ornellas D, Grozovsky R, Goldenberg RC, Carvalho DP, Fongi P, Gugginoi WP, Morales M. Thyroid hormone modulates ClC-2 chloride channel gene expression in rat renal proximal tubules. *Journal of Endocrinology* 2003; 178: 503–511.
13. Masilamani S, Kim GH, Mitchell C, Wade JB & Knepper MA. Aldosterone-mediated regulation of EnaC alpha, beta, and gamma subunit proteins in rat kidney. *Journal of Clinical Investigation* 1999; 104: 19–23.
14. McDonough AA, Brown TA, Horowitz B, Chiu R, Schlotterbeck, Bowen J & Schmitt. Thyroid hormone coordinately regulates Na⁺-K⁺ATPase α - and β -subunit mRNA levels in kidney. *American Journal of Physiology. Cell Physiology* 1988; 254: 323–329.
15. Edelman IS. Thyroidal regulation of renal energy metabolism and (Na-K)-activated adenosine triphosphatase activity. *Medical Clinics of North America* 1975; 59: 605–614.0

