

Research Journal of Pharmaceutical, Biological and Chemical Sciences

Serum Uric Acid Level in Thyroid Disorders.

Ravisekar P*, Saranya R, Manjuladevi AJ, Shanthi B and Kalaiselvi VS.

Sree Balaji Medical College and Hospital, Bharath University, Chrompet, Chennai – 44, Tamil Nadu, India.

ABSTRACT

There are studies recognising a positive correlation between thyroid function and purine nucleotide metabolism has been accepted in hypothyroidism. One such parameter serum uric acid, the concentration of which is often increased in primary hypothyroidism. On the conflicting, the relationship between hyperthyroidism and purine metabolism is more difficult. Hence, This study is taken up to identify the same in south Chennai population attending Sree Balaji Medical College & Hospital. To study the concentration of serum uric acid in thyroid disorders and to analyze their association in comparison with control. A total of 50 subjects including 30 patients, with thyroid disorders (20 hypothyroid and 10 hyperthyroid) attending the O.P.D. of Sree Balaji Medical College & Hospital and 20 healthy age and sex matched controls were studied. The Serum T3, T4 and TSH were measured to identify the thyroid disorders. Serum uric acid was measured to correlate the association with the thyroid function .Data collected was analysed using SPSS package. Hypothyroidism is a clinical syndrome resulting from a deficiency of thyroid hormones which, in turn, results in a generalized slowing down of metabolic processes. The present study evaluates the prevalence of hyperuricemia in patients affected by thyroid disorder and to investigate the concentration ofserum uric acid level with thyroid disorder patients.

Keywords: Hyperthyroidism, hypothyroidism, serum uric acid.



*Corresponding author



INTRODUCTION

Hypothyroidism is a clinical syndrome resulting from a deficiency of thyroid hormones which, in turn, results in a generalized slowing down of metabolic processes, On the conflicting, the association between hyperthyroidism and hyperuricemia has always been more difficult. In 1989 Ford etal. [1], in contrast with previous reports [2, 3], demonstrated that hyperthyroidism can cause hyperuricemia through the increase of purine nucleotide turn ove rand the decrease of renal urate excretion. Uric acid is non nitrogenous substance produce from purine metabolism, either from break down of ingested purine nucleic acid, or from tissue destruction. There are physiological variations between thyroid hormones and uric acid synthesis and excretion. As these hormones affect most of the metabolic pathways in the body, purine metabolism is one of these metabolic pathways can be affected by disturbance in thyroid hormones, that can change the uric acid level, which may lead to hyperuricemia [4]. Here we report our experience regarding the relationship between uric acid metabolism and thyroid disorders, presenting data obtained from patients suffering from either hypothyroidism or hyperthyroidism. Hypothyroidism is associated with hyperuricemia. In comparison to the prevalence reported in the general population, there is elevation of hyperuricemia was found in the hypothyroid patients9. Many studies were done regarding the biochemical status of hypothyroid patients, including uric acid levels. We have a review for evaluation of serum uric acid levels in hypothyroid patients and that might be helpful for clinical management of hypothyroid patients with hyperuricemia.

MATERIALS AND METHODS

The present study was conducted on 50 subjects including 30 patients, out of which 20 were hypothyroid and 10 were hyperthyroid attending the O.P.D. of Sree Balaji Medical college and Hospital & 20 healthy age & sex matched control. After obtaining the informed consent from the subjects, the fasting venous blood samples were collected by standard aseptic techniques. Serum was separated for the various assays. Serum T3, T4 and TSH were measured by chemiluminescence Immuno sorbent assay(CLIA) using Siemes kit . Serum uric acid was measured by kinetic method using Mindray kit in autoanalyzer. Data collected were analysed using SPSS package.

Inclusion Criteria: Age group- 20-60 yrs of age Exclusion Criteria: Patients with muscular disorders, those on drugs that could alter the serum uric acid.

RESULTS

The mean values of T3 and T4 in hypothyroid patients were 32.25 ± 7.15 ng/dl and 5.47 ± 0.74 mg/dl respectively. TSH levels were 20.11 ± 2.27 mIU/l. The mean values of T3 and T4 in hyperthyroid patients were 190.52 ± 8.60 ng/dl and 15.19 ± 1.32 mg/dl respectively. Their TSH level was 0.31 ± 0.13 mIU/l. The mean value of T3 and T4 in control patients was 60.46 ± 13.20 ng/dl and 7.44 ± 1.28 mg/dl, respectively. TSH level was 2.52 ± 0.84 mIU/l.the serum uric acid values in the three groups were 4.8 ± 0.90 , 6.50 ± 1.70 , 6.70 ± 1.80 in hypothyroid, hyperthyroid and normal individuals, respectively as shown in the table 1. On comparison with control group , theserum uric acid levels show significant elevations in both hypothyroid and hyperthyroid patients.

Parameter	Control	Hypothyroid	Hyperthyroid
	N=20	N=20	N=10
T3(ng/dl)	60.46± 13.20	32.25 ± 7.15	190.52±8.60
T4(mg/dl)	7.44 ± 1.28	5.47 ± 0.74	15.19± 1.32
TSH(mIU/L)	2.52 ± 0.84	20.11 ± 2.27	0.31 ± 0.13
Serum uric acid(mg/dl)	4.8±0.90	6.50 ±1.70	6.70 ±1.80

Table 1: Thyroid function tests and serum uric acid levels in patients and controls

Table 2: serum uric acid in control, hypothyroid

CONTROL	HYPOTHYROID	P Value
4.8±0.90	6.50 ±1.70	<0.0001*

*Highly significant

July-August

2015

6(4)



DISCUSSION

The findings of this study confirm that the possible inter-relationship between purine nucleotide metabolism and thyroid disorders, in particular primary hypothyroidism and hyperthyroidism, In this study, the correlation between hypothyroidism and hyperuricemia is well established [5-15], Many biochemical pathways in the body can be affected by disturbance of thyroid hormones level, uric acid is one of this biochemical pathways. The hypodynamic state of the circulatory system in hypothyroidism that causes the elevation of uric acid level as in this study [16].

In the present study serum uric acid is significantly increased in test group with hyperthyroidism, which agree with that obtained by Jeff 2008, hypersecretion attribute to increased level of thyroid hormones (T3 and T4), cause increased in the metabolic rate of the metabolites such as purine, which result in increased production of uric acid in the blood, that exceed the renal capacity to excrete uric acid, which may accumulate in joints causing gout, or deposited in the renal causing renal stone [17].hypothyroid hyperuricemia is due to a reduction in renal plasma flow and glomerular filtration secondary to thyroid hormone deficiency. The increase in serum uric acid levels in our hyperthyroid patients seems to be due to accelerated purine nucleotide turnover. This hypothesis is shared by Sato and colleagues [18].

CONCLUSIONS

The present study shows, the elevation of serum uric acid is known as hyperuricemia. It is a common feature not only of hypothyroidism, but also of hyperthyroidism. Therefore, we would give special importance of the routine evaluation of serum uric acid levels, both in patients affected by hypothyroidism and in patients with hyperthyroidism. In this evaluation we will be able to correct the possibly altered purine nucleotide metabolism and to prevent the onset of gout, which can worsen thyroid endocrine disorders. This finding can be explained by the high prevalence of hyperthyroidism and the increase inpurine nucleotide turnover caused by thyroid hyperfunction.

REFERENCES

- [1] Fordh C, Limw C, Chisnall WN, Pearce JM. Clin Endocrinol 1989; 30: 293- 301.
- [2] Yokogoshi Y, Saito S. Nippon Rinsho 1996; 54:3360-3.
- [3] Smyth CJ. Arthritis Rheum 1975; 18: 713-9.
- [4] Bishop ML, Engel Kivk DJL, Fody EP. Clinical Chemistry principles, procedures, correlations 4th ed. Lippinicott Williams & Wilkins California USA 2000; 345-354.)
- [5] Giordano N, Santacroce C, Mattii G, Geraci S, Amendola A, Gennari C. Clin Exp Rheumatol 2001; 19: 661-665.
- [6] Kuzzel W C, Schaffarzick RW, Naugler WE et al. J Chronic Dis 1955; 2: 64-8.
- [7] Kuhlback B. Acta Med Scand 1957; 159: 1-70.
- [8] Leeper RD, Benua RS, Brener JL, Rawson RW. J Clin Endocrinol Metab 1960; 20: 1457-66.
- [9] Boyle JA, Greig W, Duncan AM et al. Acta Rheum Scand 1996; 12: 204-9.
- [10] Ryckewaert A, Masse C, Jurmand SH et al. N Engl J Med 1970; 282: 1171-4.
- [11] Bland JH, Frymoyer JW. N Engl J Med 1970; 282: 1171-4.
- [12] Erickson A R, Enzenauer RJ, Nordstrom DM, Merenich JA. Is J Med 1994; 97: 231-4?
- [13] Montenegroj, Gonzaleso, Sarachor et al. Am J Kidney Dis 1996; 27: 195-8.
- [14] Mooraki A, Bastani B. Clin Nephrol 1998; 49: 59-61.
- [15] Makino Y, Fujii T, Kuroda S et al. Nephron 2000; 84: 267-9.
- [16] Katza L, Emmanouel DS, Lindheimermd. Nephron 1975;15: 223-9.
- [17] Ladensonpw. Am J Med 1990; 88: 638-41.
- [18] Sato A, Shirota T, Shinoda T et al. Metabolism 1995; 44: 207-11.

6(4)