

# The Relationship Between Thyroid Disorder and Serum Levels of Uric Acid In Normal Renal Physiology

Shabir Ud Din Lone<sup>1</sup>, Sheikh Junaid Aziz<sup>2\*</sup>, Hilal Ahmad Wani<sup>3</sup>, Sheikh Imran Sayeed<sup>4</sup>

<sup>1</sup>Associate Professor, Department of Physiology, Government Medical College, Srinagar, J & K, India.
<sup>2</sup>Lecturer, Department of Physiology, Government Medical College, Srinagar, J & K, India.
<sup>3</sup>Assistant Professor, Department of Biochemistry, Government Degree College, Handwara, J & K, India.
<sup>4</sup>Professor & Head, Department of Physiology, Government Medical College, Srinagar, J & K, India.

#### ABSTRACT

**Introduction:** Thyroid disorder can affect renal physiology. The changes in renal hemodynamics in hypothyroidism are decrement in renal blood flow, renal plasma flow, glomerular filtration rate (GFR) and single nephron GFR. Till date a number of studies have confirmed association, but the data on relationship between thyroid disorders viz. the highly prevalent Subclinical hypothyroidism, in our society is very limited, especially in college going students.

**Materials and Methods:** The study was conducted on the college going students. In total 842 subjects were screened, among both genders, 100 newly detected subclinical hypothyroid cases were selected and equal numbers of students of same age group with normal thyroid profile were randomly selected in control group. Hypothyroidism leads to hemodynamic changes especially affects the renal blood flow, with effect also on autonomic activity results in reduced GFR. Both GFR and RBF due to thyroid disorder influence the renal function. The serum uric acid levels in hypothyroid and euthyroid controls was analysed using unpaired student 't' test. **Results and Observations:** Correlation between thyroid profile T3, T4 and TSH and serum uric acid was analysed using Pearson's correlation coefficient tests and prevalence of hyperuricemia among subclinical hypothyroid was statistically

## INTRODUCTION

Thyroid disorder can affect renal physiology. Hypothyroidism slows down all metabolic activities and one of the pathways is the purine metabolism. Hypothyroidism can influence this pathway and causes changes in uric acid levels.<sup>1-3</sup> Hypothyroidism leads to hemodynamic changes, decreased cardiac output, increased vasoconstriction in the renal vasculature, decreased expression of vascular endothelial growth factor and IGF-I, which leads to reduced renal vasculature response to vasodilators. Thus, renal blood flow is reduced.<sup>1.4</sup> In hypothyroidism sensitivity to beta adrenergic stimuli is also reduced with the result glomerular filtration rate (GFR) also diminishes. Both GFR and RBF are reduced due to hypothyroid state that influences the biomarker of renal functions.<sup>5-7</sup> In kidneys the deficiency of thyroid hormone results in renal parenchymal growth retardation which causes

significant. The student showed that serum uric acid level was significantly increased in hypothyroid individuals, even in college going students.

**Conclusion:** Study shows that there is an increased uric acid level in the study group and the prevalence of hyperuricemia is high in subjects with high TSH. The study suggested that hyperuricemia is secondary to decreased renal plasma flow.

Key words: Hypothyroidism, GFR, RBF, Uric Acid, Sub Clinical, Renal.

*Correspondence to:	
Dr Sheikh Junaid Aziz,	
Lecturer,	
Department of Physiology,	
Government Medical College, S	Srinagar, J & K, India.
Article History:	
Received: 24-01-2021, Revised:	19-02-2021, Accepted: 21-03-2021
Access th	is article online
Website:	Quick Response code

www.ijmrp.com	
DOI:	
10.21276/ijmrp.2021.7.2.012	

reduction in surface area for filtration.<sup>8</sup> The reduction in renal plasma flow is because of the indirect effect on CVS leading to generalised hypodynamic circulatory state and pathologic changes in the glomerular structure such as thickening of glomerular basement membrane and expansion of mesangial matrix leading to decrease in renal blood flow.<sup>9</sup> There is also reduced sodium reabsorption in both proximal and distal tubular segments because of reduced activity of Na/K ATPase initially in the proximal tubules and later in almost all segments of the nephron. Thus, reduced sodium chloride reabsorption increases the distal sodium and chloride delivery, triggering the macula densa mediated tubuloglomerular feedback which reduces the RAAS activity. Consequently, the GFR falls.<sup>10</sup> Thus the changes in renal hemodynamics in hypothyroidism are decrement in renal

blood flow, renal plasma flow, glomerular filtration rate (GFR) and single nephron GFR. Hypothyroidism is also associated with hyperuricemia and increased serum creatinine and decreased creatinine clearance. This fact suggests that hypothyroid hyperuricemia is secondary to a reduction in renal plasma flow and glomerular filtration leading to decrease in urate excretion.<sup>11</sup>

Hypothyroidism is a clinical syndrome that has been divided into Overt and Subclinical forms.<sup>12</sup> Till date a number of studies have confirmed association, but the data on relationship between thyroid disorders viz. the highly prevalent Subclinical hypothyroidism, in our society is very limited, especially in college going students. Thus, this study was planned to evaluate the changes in biochemical parameters of normal renal function in subclinical hypothyroid among college going students.

## AIMS AND OBJECTIVES

To find relationship between Thyroid profile (T3, T4, TSH) and Serum Uric acid in subjects of normal renal function.

## MATERIAL AND METHODS

The study was conducted on 1<sup>st</sup> year to final year MBBS students of our college, in the Department of Physiology, GMC Srinagar, for a period of one year. This Prospective cross-sectional study was conducted in collaboration with Department of Biochemistry, GMC Srinagar. The total of about 842 subjects were screened based on the history and physical examination and the first 100 students among both genders, who were newly detected for sub-clinical hypothyroidism were selected for the study group and the 100 randomly selected students of the same age group with normal thyroid profile were selected for the control group. The study was initiated with the approval of Institutional ethical committee, GMC Srinagar and was carried out after getting written informed and written consent from the subjects. The students who had any of the trait from the below mentioned list of exclusion criteria were not selected.

## **Exclusion Criteria**

- 1. Person on thyroxin treatment.
- 2. Subjects with history of renal disease, liver disease, cardiovascular disease, hypertension, diabetes mellitus, gout, muscular disorders, or malignancy.
- 3. History of smoking, or alcoholism.
- **4.** Subjects on drugs (Hypolipidemic, antihypertensive, steroids, probenacid, allopurinol etc.)

## Equipment's Used in the Study

- 1. Proforma to record the anthropometric measurements and the clinical findings of the subjects.
- 2. Portable weighing machine to record the body weight in kilograms.
- 3. Stadiometer to measure the standing height in cms.
- 4. Standardized mercury sphygmomanometer to record the Blood Pressure in mm of Hg.

## Methodology

After the written and informed consent, the experimental protocol included:

- 1. Recording of a Detailed Medical History including surgical, drug and family history.
- 2. Measurement of Anthropometric Indices: The subjects were asked to stand erect, with their arms relaxed at their side and feet together. The following were measured:

- Weight (in kgs) was recorded using a portable standard weighing machine.
- Height (in cms) was measured to the nearest 0.5 cm using a stadiometer.
- Body Mass Index (BMI) was calculated using Quetelet's Index. BMI = Weight (Kg)/ Height (m<sup>2</sup>).
- 3. Recording of Vital Signs viz. pulse rate, respiratory rate and BP were done and documented.
- 4. Blood Investigations: After overnight fasting, 4ml venous blood sample was collected from cubital vein. Serum obtained after centrifugation was divided into 2 aliquots- one for Thyroid profile and second for serum uric acid. Serum T3 and T4 were performed using competitive ELISA technique<sup>13</sup> and serum TSH was performed using sandwich ELISA technique.<sup>14</sup>

## **REFERENCE VALUES**

Serum/plasma	Normal range		
Т3	0.69 - 2.15 ng/ml		
T4	5.2 - 12.7 μg/dL		
TSH	0.3 - 5.5 μIU/ml		

Serum uric acid analysed immediately using Uricase-Trinder-Enzymatic and colorimetric method in fully auto chemistry analyser. Uricase converts uric acid to allantoin and hydrogen peroxide. The hydrogen peroxide formed further reacts with 4aminoantipyrine and TOOS by the catalytic action of peroxidase to form a quinoneimine dye complex. Intensity of the colour formed is directly proportional to the amount of uric acid present in the sample.<sup>15,16</sup>

## **REFERENCE VALUES**

Serum/plasma	mg/dl
Women	2.5-6.8
Men	3.6-7.7

## **RESULTS AND OBSERVATIONS**

The serum uric acid levels in hypothyroid and euthyroid controls was analysed using unpaired student 't' test, correlation between thyroid profile (T3, T4, TSH) and serum uric acid in thyroid patients was analysed using Pearson's correlation coefficient test and prevalence of hyperuricemia among study group and control group was found. This was done by means of SPSS software version 16 and statistical significance was observed 'P' value <0.05. The mean age of control and cases is 21.16 and 21.44 years respectively which is almost a match.

The mean height in control and case group is 160.09 and 159.44cms respectively. This means only 0.75 cm difference between two groups.

The mean weight between control and case group is 60.09 and 60.65kgs respectively, with a difference of -0.56kgs.

The mean respiratory rate in control and case group is 18.55 and 18.68 respectively. The difference is only -0.12 which came to be non-significant.

The Table E and Table F shows mean diastolic and systolic blood pressure between the groups and the difference in the means between control and case group was not significant.

The mean BMI in control and case group is 23.43 and 23.89 respectively and the difference is non-significant.

There are overall 170 females and 30 males which constitute about 85% and 15% of the total subjects respectively. In case group about 81 are females and 19 are males. Whereas in case group 89 are females and 11 are males. So here we can see the females are more at risk of having hypothyroidism. In control group mean T3 was 1.50, in case group 0.854, with a difference of 0.647 even with a degree of freedom 198, not significant.

The mean T4 level in control was 9.5 and in cases was 6.0, with a difference of 3.5 which is statistically not significant.

In control group TSH mean value was 2.2 and in cases the mean value of TSH was 9.63. The difference of -7.38 was found significant.

When compared serum uric acid level between cases and controls it was found that there was a Significant increase in uric acid value in the study group than in the control group. Results analysed using Student 't' test disclosed a statistically significant 'p' value.

Table A: Shows Age distribution between control and case group					
Group	Obs	Mean	Std. Err.	Std. Dev.	[95% Conf. Interval]
Control	100	21.16	0.1968027	1.968027	20.7695
Case	100	21.44	0.2138252	2.138252	21.01572
Combined	200	21.3	0.1452774	2.054533	21.01352
Diff		-0.28	0.2906072		-0.8530824

diff = mean (Control) – mean (Case); t = -0.9635; Ho: diff = 0; degrees of freedom = 198;

Ha: diff < 0 Pr(T < t) = 0.1682; Ha: diff != 0; Pr(|T| > |t|) = 0.3365; Ha: diff > 0; Pr(T > t) = 0.8318;

Table B: Shows distribution of height in cms					
Group	Obs	Mean	Std. Err.	Std. Dev.	[95% Conf. Interval]
Control	100	160.19	0.9386962	9.386962	158.3274 162.0526
Case	100	159.44	0.9483979	9.483979	157.5582 161.3218
Combined	200	159.815	0.6660495	9.419363	158.5016 161.1284
Diff		0.75	1.334395		-1.88145 3.38145

diff = mean (Control) – mean (Case); t = 0.5621; Ho: diff = 0; degrees of freedom = 198;

Ha: diff < 0; Pr(T < t) = 0.7126; Ha: diff != 0; Pr(|T| > |t|) = 0.5747; Ha: diff > 0; Pr(T > t) = 0.2874;

#### Table C: Shows weight distribution in kgs

			-	-	
Group	Obs	Mean	Std. Err.	Std. Dev.	[95% Conf. Interval]
Control	100	60.09	0.6613569	6.613569	58.77772 61.40228
Case	100	60.65	0.7481121	7.481121	59.16558 62.13442
Combined	200	60.37	0.498405	7.048511	59.38717 61.35283
Diff		-0.56	0.9985312		-2.529121 1.409121

diff = mean (Control) – mean (Case); t = -0.5608; Ho: diff = 0; degrees of freedom = 198; Ha: diff < 0; Pr(T < t) = 0.2878; Ha: diff != 0; Pr(|T| > |t|) = 0.5756; Ha: diff > 0; Pr(T > t) = 0.7122;

Table D: Shows respiratory rate distribution.

Group	Obs	Mean	Std. Err.	Std. Dev.	[95% Conf. Interval]
Control	100	18.55556	0.1916749	1.907141	18.17518 18.93593
Case	100	18.68	0.1710809	1.710809	18.34054 19.01946
Combined	200	18.61809	0.12814	1.807637	18.3654 18.87078
Diff		-0.124444	0.2567798		6308344 .3819455

diff = mean (Control) – mean (Case); t = -0.4846; Ho: diff = 0; degrees of freedom = 197; Ha: diff < 0; Pr(T < t) = 0.3142; diff != 0; Pr(|T| > |t|) = 0.6285; Ha: diff > 0; Pr(T > t) = 0.6858;

Table E: Distribution of Diastolic BP					
Group	Obs	Mean	Std. Err.	Std. Dev.	[95% Conf. Interval]
Control	100	75.6	0.4988877	4.988877	74.6101 76.5899
Case	100	75.6	0.4988877	4.988877	74.6101 76.5899
Combined	200	75.6	0.3518794	4.976326	74.90611 76.29389
Diff		0	0.7055337		-1.391325 1.391325

diff = mean (Control) – mean (Case); t = 0.0000; Ho: diff = 0; degrees of freedom = 198; Ha: diff < 0; Pr(T < t) = 0.5000; Ha: diff != 0; Pr(|T| > |t|) = 1.0000; Ha: diff > 0; Pr(T > t) = 0.5000;

Table F: Distribution of Systolic BP					
Group	Obs	Mean	Std. Err.	Std. Dev.	[95% Conf. Interval]
Control	100	110.6	0.708106	7.08106	109.195 112.005
Case	100	111.4	0.6966007	6.966007	110.0178 112.7822
Combined	200	111	0.4962168	7.017566	110.0215 111.9785
Diff		-0.8	0.993311		-2.758827 1.158827

Table F: Distribution of Systolic BP

diff = mean (Control) – mean (Case); t = -0.8054; Ho: diff = 0; degrees of freedom = 198; Ha: diff < 0; Pr(T < t) = 0.2108; Ha: diff != 0; Pr(|T| > |t|) = 0.4216; Ha: diff > 0; Pr(T > t) = 0.7892;

Table G: Distribution of BMI					
Group	Obs	Mean	Std. Err.	Std. Dev.	[95% Conf. Interval]
Control	100	23.439	0.2026984	2.026984	23.0368 23.8412
Case	100	23.895	0.2565481	2.565481	23.38595 24.40405
Combined	200	23.667	0.1638683	2.317448	23.34386 23.99014
Diff		-0.456	0.3269611		-1.100773 .188773

diff = mean (Control) – mean (Case); t = -1.3947; Ho: diff = 0; degrees of freedom = 198; Ha: diff < 0' Pr(T < t) = 0.0823; Ha: diff != 0; Pr(|T| > |t|) = 0.1647; Ha: diff > 0; Pr(T > t) = 0.9177;

Table H:	Shows	Gender	distribution:
----------	-------	--------	---------------

Sex	Gro	Total	
	Control	Case	_
F	81	89	170
	81	89	85
Μ	19	11	30
	19	11	15
Total	100	100	200
	100	100	100

Pearson chi2(1) = 2.5098 Pr = 0.113

#### Table I: T3 levels in the control and case group

Group	Obs	Mean	Std. Err.	Std. Dev.	[95% Conf. Interval]
Control	100	1.5012	0.0349953	0.3499532	1.431762 1.570638
Case	100	0.854	0.0375578	0.3755777	.7794772 .9285228
Combined	200	1.1776	0.0343762	0.4861522	1.109812 1.245388
Diff		0.6472	0.0513348		.5459669 .7484331

diff = mean (Control) – mean (Case); t = 12.6074; Ho: diff = 0; degrees of freedom =198;

Ha: diff < 0; Pr(T < t) = 1.0000; Ha: diff != 0; Pr(|T| > |t|) = 0.0000; Ha: diff > 0; Pr(T > t) = 0.0000;

Table J: T4 levels in control and case group

Group	Obs	Mean	Std. Err.	Std. Dev.	[95% Conf. Interval]	
Control	100	9.5746	0.1832368	1.832368	9.211018 9.938182	
Case	100	6.008	0.2651117	2.651117	5.481961 6.534039	
Combined	200	7.7913	0.2044877	2.891892	7.388059 8.194541	
Diff		3.5666	0.3222731		2.931072 4.202128	

diff = mean (Control) – mean (Case); t = 11.0670; Ho: diff = 0; degrees of freedom = 198;

Ha: diff < 0; Pr(T < t) = 1.0000; Ha: diff != 0; Pr(|T| > |t|) = 0.0000; Ha: diff > 0; Pr(T > t) = 0.0000;

## Table K: TSH levels in control and case group

Group	Obs	Mean	Std. Err.	Std. Dev.	[95% Conf. Interval]	
Control	100	2.2524	0.103568	1.03568	2.046899 2.457901	
Case	100	9.6359	0.2889086	2.889086	9.062643 10.20916	
Combined	200	5.94415	0.3031795	4.287606	5.346293 6.542007	
Diff		-7.3835	0.3069113		-14.767	

diff = mean (Control) – mean (Case); t = -24.0574; Ho: diff = 0; degrees of freedom = 198; Ha: diff < 0; Pr(T < t) = 0.0000; Ha: diff != 0; Pr(|T| > |t|) = 0.0000; Ha: diff > 0; Pr(T > t) = 1.0000;

Table L. Seruin und acid ieveis of control and case group						
Group	Obs	Mean	Std. Err.	Std. Dev.	[95% Con	f. Interval]
Control	100	5.412	0.1064133	1.064133	5.200853	5.623147
Case	100	6.876	0.1159338	1.159338	6.645962	7.106038
Combined	200	6.144	0.0940882	1.330608	5.958462	6.329538
Diff		-1.464	0.1573672	-2.928		928
$diff = magn (Cantrol)$ , magn (Canch) $t = -0.2021$ , $U_{0}$ , $diff = 0.4$ degrees of freedom = 100.					NO.	

Table L: Serum uric acid levels of control and case group

diff = mean (Control) – mean (Case); t = -9.3031; Ho: diff = 0; degrees of freedom = 198;

Ha: diff < 0; Pr(T < t) = 0.0000; Ha: diff != 0; Pr(|T| > |t|) = 0.0000; Ha: diff > 0; Pr(T > t) = 1.0000;

# DISCUSSION

Present study evaluated the relationship between the effects of hypothyroidism on renal function and to study the correlation of TSH, T3 with serum uric acid levels. The subjects with age group of 19-24 years were included in the study. Mean age of the study and control group were 21.4 years and 21.16 years respectively. A study conducted by Chandhury H.S et al in 2013, the common age of hyperthyroidism was found to be between 25-35 years.<sup>17</sup> Mean BMI of the cases and control group were 23.89 kg/m2 and 23.43 kg/m2 respectively. There is no significant difference between cases and control group in contrast to other studies having significant difference in BMI between hypothyroid and euthyroid group. The mean weight and height of cases and control group were (60.65, 60.09) and (159.44, 160.19) respectively. No significant difference was seen. Among cases 11 were males and 89 females and in control group 81 were females and 19 were males. In our study percentage of females were more and the results of this study were similar to Chandhury H.S et al; 2013, Tejomani M et al; 2013 also observed and recorded hypothyroidism was more frequent in females than males.<sup>11,18</sup> The mean respiratory rates per min in cases were 18.68 and in control were 18.55. No significant difference was found. The mean systolic and diastolic blood pressure in cases and control group were between (111.4 and 110.6) mmHg and (75.6 and 75.6) mmHg respectively. The recorded blood pressure between the group and of male and female were of no significance. In our study the mean TSH value among case group and control group were 9.63 µIU/mI and 2.25µIU/mI respectively. There is a significant and statistically conducted difference in TSH value among study group. Also the mean value of T3 in cases and control group is 6µg/dL and of control group 9.57µg/dL. It is observed that the levels of T3 and T4 in cases are decreased when compared to control. The mean serum uric acid values of case and control group are 6.87mg/dL and 5.41mg/dL respectively and the difference is statistically highly significant. In a similar study done by Tayal D et al 2005 showed a significant increase in serum uric acid level in hypothyroid patients.<sup>19</sup> A study done by Ajay kumar et al 2013, showed increase in serum uric acid levels in a newly diagnosed hypothyroid patients, which got decreased within 6 months of thyroxine replacement therapy.<sup>20</sup> Gulab Kanwar et al 2015 and Vijaypriya I indraruth 2016, there study showed serum uric acid level was significantly increased in hypothyroid patients and suggested hyperuricemia is secondary to decreased renal plasma flow and since 75% of uric acid is eliminated through kidneys and in hypothyroid patients there is impaired renal function.<sup>21,22</sup> A study done by Giordano et al 2001 showed 33.3% prevalence in hyperuricemia in patients with hypothyroidism in compared to 10% prevalence in general population.<sup>23</sup> Tayal D et al 2009, also showed that no case of gout was reported despite the presence of hyperuricemia in overtly hypothyroid cases.<sup>19</sup>

## CONCLUSION

Our study shows that there is an increased uric acid level in the study group and the prevalence of hyperuricemia is high in subjects with high TSH.

These biochemical changes happen because of the changes in renal function. These findings are suggestive of an interaction between thyroid gland and kidney and the effect of hypothyroid state on renal functions. So, it is suggested that at the level of diagnosis of hypothyroid disorder we have to assess the renal function also.

## ACKNOWLEGEMENT

The authors would like to thank all the persons who participated in this study. Without their cooperation this work would not have been possible. Authors are also grateful to Dr. Inam-UI-Haq, Associate Professor SPM Department, who helped with the statistical analysis. Lastly authors are thankful to all the staff members of the Department of Physiology for their help in the data collection and compilation and provision of some valuable suggestions.

## REFERENCES

1. Khalid AS, Ahmed MI, Elfaki HM, Hassan N, Suliman SM. Renal Function in Hypothyroidism Eighth Arab Conference on the Peaceful uses of Atomic Energy Amman,3-7 December 2006.

2. Nagarajappa K, Sushma BJ, Shweta R. Study of Thyroid stimulating hormone, Serum creatinine, Serum uric acid levels in patients with Hypothyroidism. Int. J. Pure App. Biosci. 2014; 2(2): 187-90.

3. Capasso G, De Santo NG, Kinne R. Thyroid hormones and renal transport Cellular and biochemical aspects. Kidney int 1987 Oct;32(4):443-51.

4. Rasheed AM, Al- Khayat TH. The effect of thyroid hormone levels on different kidney function tests International Journal of Scientific & Engineering Research, Volume 5, Issue 10, October-2014; 983-91.

5. Woodward A, McCann S and Al-Jubouri M. The relationship between estimated glomerular filtration rate and thyroid function: An observational study Ann Clin Biochem 2008; 45: 515–7.

6. Arora S, Chawla R, Tayal D et al. Biochemical markers of liver and kidney function are influenced by thyroid function- A case controlled follow up study in Indian hypothyroid subjects. Indian journal of clinical biochemistry 2009;24(4):370-74.

7. Basu G, Mohapatra A. Interactions between thyroid disorders and kidney disease. Indian J Endocrinol Metab 2012 Mar;16(2):204-13.

8. Vargas F, Moreno JM, Rodriguez-Gomez I, Wangensteen R, Osuna A, Alvarez-Guerra M, et al. Vascular and renal function in experimental thyroid disorders. Eur J Endocrinol. 2006; 154: 197–212.

9. Bradley SE, Coelho JB, Sealey JE, Edwards KD, Stephan F. Changes in glomerulotubular dimensions, single nephron glomerular filtration rates and the renin-angiotensin system in hypothyroid rats. Life Sci. 1982;30:633–9.

10. Basu G and Mohapatra A. Interactions between thyroid disorders and kidney disease Indian J Endocrinol Metab. 2012 MarApr; 16(2): 204–13.

11. Mariani LH, Berns JS. The renal manifestations of thyroid disease. J Am Soc Nephrol. 2011;23(1):22–6.

12. Boelart, Franklyn JA. Thyroid hormone in health and disease. J Endocrinol 2005; 187:1-15.

13. Sterling L. Diagnosis and treatment of thyroid disease. Cleveland CRC Press, 1975: 9- 51. 9.

14. Caldwell G, Kellett HA, Gow SM, Beckett GJ, Sweeting VM, Seth J, Toft AD. A new strategy for thyroid function testing. Lancet 1985; 1: 1117-9.

15. Bishop ML, Duben-Engelkirk JL, Fody EP. Clinical Chemistry: Principles, Procedures, Correlations. 4th edn, California: Lippincott Williams & Wilkins, 2000. pp. 270–71.

16. Burtis CA, Ashwood ER & Bruns DE, editors. Tietz Textbook of clinical chemistry and molecular diagnostics.4th ed. Missouri: Elsevier Saunders; 2006.

17. Chaudhury HS, Raihan KK, Uddin MN, Ansari SM, Hasan M, Ahmed M, et al. Renal function impairment in hypothyroidism. Bangladesh. J. Med. Biochem. 6(1):19-25, 2013.

18. Tejomani M, Meera KS, Vasudha KC. Relevance of Creatine Kinase Activity and Serum Creatinine Levels in Hypothyroidism International Journal of Recent Trends in Science And Technology, Volume 8, Issue 3, 2013 pp 263-9.

19. Tayal D, Chawla R, Arora S, Gupta VK, Sohi JS, Mallika V. Dynamic Changes in Biochemical markers of Renal Function with Thyroid Status – A Study in Indian Population. Internet journal of medical update. 2009 July; 4 (2): 36-41.

20. Ajaykumar N, Shanthi M, Parameshwari R. The Effect of L-Thyroxine on Metabolic Parameters in Newly Diagnosed Primary Hypothyroidism. International Journal of Pharmaceutical Science Invention. 2013Aug; 2(8): 8-14.

21. Kanwar G, Jain KB, Jain J, Shekhawat KS, Kabra R, Jain R. Association of serum uric acid and creatinine levels with hypothyroidism. International Journal of Scientific Research and Engineering Studies (IJSRES) Volume 2 Issue 9, September 2015 ISSN: 2349-8862.

22. Indirajith VI. Serum uric acid level in primary hypothyroidism. The Tamil Nadu Dr.M.G.R Medical University Journal of Pre and Para Clinical Sciences Volume 2 Issue 4 2016.

23. Giordano N, Santacroce C, Mattii G, Geraci S, Amendola A, Gennari C. Hyperuricemia and gout in thyroid endocrine disorder. Clin Exp Rheumatol. 2001;19:661–5.

Source of Support: Nil.

Conflict of Interest: None Declared.

**Copyright:** © the author(s) and publisher. IJMRP is an official publication of Ibn Sina Academy of Medieval Medicine & Sciences, registered in 2001 under Indian Trusts Act, 1882.

This is an open access article distributed under the terms of the Creative Commons Attribution Non-commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

**Cite this article as:** Shabir Ud Din Lone, Sheikh Junaid Aziz, Hilal Ahmad Wani, Sheikh Imran Sayeed. The Relationship Between Thyroid Disorder and Serum Levels of Uric Acid In Normal Renal Physiology. Int J Med Res Prof. 2021 Mar; 7(2): 47-52. DOI:10.21276/ijmrp.2021.7.2.012