



## Urinary Iodide Excretion Studies in Thyroid Patients in North Kerala

Authors

Minikumari Amma.V.<sup>1</sup>, Anu.E.B.<sup>2</sup>, P.S.Harikumar<sup>3</sup>, Indu Sasidharan<sup>4</sup>

<sup>1,2</sup>Department of Biochemistry, Government Medical College, Thiruvananthapuram

<sup>3</sup>Centre for Water Resources Development & Management, Kunnammangalam, Kozhikode

<sup>4</sup>Department of Pharmacology, Government Medical College, Thiruvananthapuram

Corresponding Author

Dr Minikumari Amma.V.

Department of Biochemistry, Government Medical College, Thiruvananthapuram

Email: [manusanthan@gmail.com](mailto:manusanthan@gmail.com), Phone: 9496910448

### Abstract

**Background:** Iodine is a micronutrient for the synthesis of thyroid hormones. The decreased production of thyroid hormone results in hypothyroidism. One of the reasons for hypothyroidism is iodine deficiency. The prevalence of thyroid disease is very high in Kerala even after implementing universal salt iodization. The aim of this study was to find out whether the iodine intake is normal in thyroid patients consuming only non-iodized salt.

**Methods:** The study was conducted among the thyroid patients attending the Out Patient Departments of Kozhikode Medical College. Through thyroid hormonal assay, well established cases of hypo and hyper thyroid patients were taken under study. 24hr urine samples from 500 thyroid patients and 500 normal controls were collected. 24 hr urinary iodide estimation was done in the CWAL of CWRDM, Kunnammangalam.

**Result:** The average urinary iodide excretion was found to be 190, 188.2, 187.9 and 190 µg/day in hypo, their control, hyper and their control respectively.

**Conclusion:** Nutritional iodine deficiency is not the major problem as judged by urinary iodide excretion study.

**Keywords:** Iodine deficiency, urinary iodide, hypothyroidism.

### Introduction

Iodine, one of the essential trace elements, occupies a prominent place in human health. Iodine in adequate amount is a pre requisite for the biosynthesis of thyroid hormones by the thyroid gland. The average iodine requirement for an adult is estimated as 100 µg/day<sup>1</sup>. It is met through sea foods, vegetables, milk, meat and drinking water. Decreased synthesis of thyroid hormone is called hypothyroidism. Iodine deficiency can leads to

hypothyroidism. Hypothyroidism is manifested by sluggishness, sleepiness, dry skin, cold intolerance, constipation and the gland swells leading to goitre. The severe consequences of hypothyroidism during foetal and neonatal life is cretinism that causes irreversible mental retardation including deaf mutism, short-stature and under development of musculoskeletal system. Besides, lack of iodine results in

reproductive impairment in women and increased infant mortality

Excess iodine intake is associated with iodine auto immunity and iodine induced hyperthyroidism<sup>2</sup>. The number of thyroid patients is increasing every year. A large number of thyroid patients are coming in the medicine and surgery outpatient (OP) departments of Kozhikode Medical College. Assessment of the urinary iodine excretion is believed to give the best index of the prevalence of Iodine Deficiency Disorders (IDD). The present study is an attempt to evaluate the incidence of IDD using urinary iodide excretion as the best biomedical indicator.

### Materials and Methods

The thyroid patients who attended the Surgery and Medicine outpatient Department of Kozhikode Medical College for a period of 2 years were taken up for the study. Based on the assays of T<sub>3</sub>, T<sub>4</sub> and TSH, 250 patients with hypothyroidism and 250 patients with hyperthyroidism were identified. For each test group, 250 normal age matched subjects from the same house hold and eating same food items were used as controls.

In order to assess the load of hypo and hyper thyroid patients getting treated at the Medical College, a pilot study was conducted for one month to assess the sample population. Using this data, the prevalence was calculated (18.75%) This data was used to estimate the sample size for the present study. Sample size calculation is by  $4pq/I^2$  where  $p$  = prevalence,  $q = 100 - P$ ,  $I = P/5$ . Substituting the values to above formula, sample size was found to be 433 and so taken 500 patients.

The study was conducted after getting ethical clearance. A written consent was obtained from all the participants. All the patients and controls were given non-iodized salt for consumption. They were advised to use only this salt for one week before taking urine, not to eat items containing antithyroid agents (cabbage, tapioca etc.) and stay away from medicines for one day

Details about the patients were collected from the OP using direct questionnaire. The details included geographic nature of place of residence, age, sex, family history, type of thyroid abnormality, symptoms and food habits. Blood samples were collected and serum separated. Thyroid hormones were estimated in serum by Enzyme Linked Immunosorbent Assay (ELISA). 24 hr urine samples were collected after adding thymol as preservative and were analyzed for iodine content. Catalytic reduction method was used to estimate the level of iodine in urine<sup>3</sup>. Quality assurance was done by electrometric method using iodide selective electrode. The analysis was done in the Central Water Analysis Laboratory (CWAL) of Centre For water Resources Development and Management (CWRDM), Kunnammangalam, Kozhikode. Statistical analysis was done and  $p$ -value less than 0.05 was considered significant.

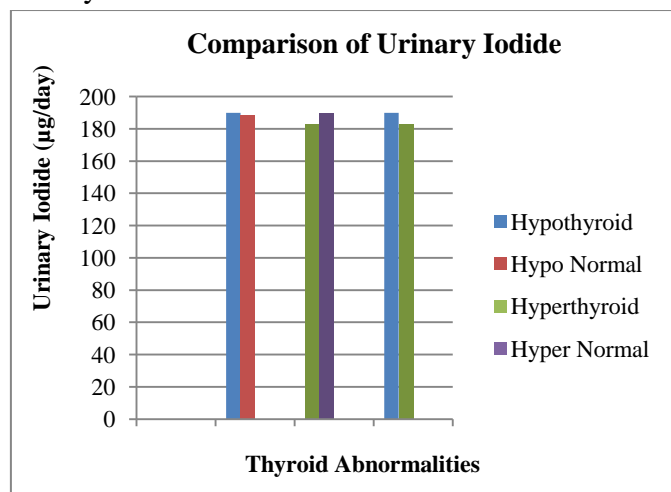
### Results

**Table 1.** Comparison of Urinary Iodide Excretion

Group	Urinary iodide( $\mu$ g/Day)	$p$ -Value
Hypo	190 $\pm$ 16.6	0.90
Hypo normal	188.2 $\pm$ 17.5	
Hyper	182.9 $\pm$ 17.4	0.68
Hyper normal	190 $\pm$ 21.6	
Hypo	190 $\pm$ 16.6	0.64
Hyper	182.9 $\pm$ 17.4	

Among the study group, 96% are non-vegetarian consuming sea fish. More than 87% of the study population is females. Irrespective of the type of thyroid complaint, 17.22% of the patients are having family history of thyroid disease. In the study groups, there is no significant variation in the number of hypo and hyperthyroid patients coming from different land areas. In all groups of study the median 24hr urinary iodide excretion was more than 180  $\mu$ g/day (Table 1, Fig 1). There is no significant variation in the urinary iodide excretion between the groups ( $p$  value >0.05).

**Fig 1.** Graphical representation of comparison of urinary iodide excretion



### Discussion

Iodide deficiency occurs when iodine intake falls below the recommended levels. If excess of iodine is consumed, it is eliminated through urine. Urinary iodide excretion is a good marker of very recent dietary iodine intake. Since there is significant variation in the volume of daily urinary excretion, it was found better to estimate the total iodide in 24 hr urine.

In all the study population, the median urinary iodide excretion was more than 180µg/day. This value is well above the recommended levels (100µg/day). That means the iodide intake is sufficient without consuming iodized salt. So the intake becomes excess if using iodized salt.

Earlier studies showed the existence of iodine deficiency in most parts of world including India<sup>4-10</sup>. Because of the increased incidence of Iodine Deficiency Disorders, universal iodination was implemented. After several years of this programme, studies were carried out in North India showing deficient, adequate or even excess urinary iodide excretion<sup>11-14</sup>. Most of the recent research works in Kerala showed excess intake of iodine instead of iodine deficiency. A study in the surgery department of Medical College Trivandrum Kerala, showed the high prevalence of multinodular goiter cannot be attributed to iodine deficiency as only 2% of the total number of cases studied had low urine iodide level<sup>15</sup>.

The increased excretion may be due to the fact that there may be adequate iodine intake through iodine rich food materials like marine fish, dairy products, onion, egg etc. which are commonly consumed even by low income group. If iodine deficiency is the major reason for thyroid complaint, the number of hypothyroid patients should increase. But in the present study it was noted that the number of hypo and hyperthyroid patients are almost equal.

### Conclusion

The number of thyroid patients are increasing day by day. The present study shows that the increased thyroid complaints are not due to nutritional iodine deficiency as judged by urinary iodide. The iodide uptake by the thyroid gland and utilization is also important. Some antithyroid agents like thiocyanate, perchlorate etc will inhibit the uptake of iodide.

The consumption of iodized salt along with iodine rich food materials could enhance iodine intake to toxic levels. Prolonged intake of iodide at or near toxic level causes hyperthyroidism and autoimmune thyroid diseases and even thyroid carcinoma. So periodical and epidemiological studies should be conducted to detect high iodine intake. A final word on the compelled intake of iodized salt can be said only after several of such studies

### References

1. Matovinovic J, Ramalingaswami V Therapy and Prophylaxis in endemic goitre.1960
2. Hye Rim Chung, Ann. Paediatric Endocrinal metabolism. 2014 ;March 19(1)
3. Rogina B, Dubravcic M, Microdetermination of iodides by arresting the catalytic reduction of ceric ions. Analyst. 1953; 78:38
4. M.C.Carrison, R., Observations on endemic cretinism in the chittal and Gilgit valleysLancet.1908;2:1275.
5. Ramalingaswamy V, Subramanian T A .The etiology of Himalayan Goitre. Lancet.1961; 1:791

6. Ramalingaswamy V, Endemic goitre in South East Asia. Ann. Int. Med. 1973;78;277
7. Das.S.C, Isichei.U.P. West Afr. J. Medicine. 1998; April-June 17(2): 113-120.
8. Pangpaelv.P. J. Medi. Asso. Thailand. 1999; March 82(3): 284-9.
9. Andersen.S, Pedersen.K.M. Eur. J. Endocrinol. 2001; 144(5) : 461-5.
10. Thomson.C.D,Colls.A.J. Br. J. Nutrition. 1997; Dec 78(6): 901-12.
11. Dr.Tapaskumar Sen. (Dissertation Project). Assessment of iodine deficiency disorders in North 24 Parganas District, West Bengal, India.
12. Marwaha.R.K,Tandon.N, Gupta.N, Karak. A.K, Verma.K,KochuPillai.N. Residual goitre in the post iodization phase. Iodine status, thiocyanate exposure and Autoimmunity. Clin. Endocrinol. 2003; 59: 672-81.
13. Kapil. U. M,Padak.P. Assessment of iodine deficiency in Ernakulam District, Kerala state. Indian Paediatrics. 1999; 36: 178-80.
14. Chandra. A.K,Singh .L.H, Tripathy.S, Dehra. A, Khanam. J. Iodine Nutritional status of children in North East India. Indian .J.of Paediatrics.2006; 73: 795-8
15. Nishad Kerakada, Ganesh Manikantan, Meer.M. Chisthi. International Surgery Journal. 2017;Feb 4(2):680-684.