Available online at www.joac.info

ISSN: 2278-1862

Journal of Applicable Chemistry
2016, 5 (4): 851-855
(International Peer Reviewed Journal)

Urinary Iodine Status of Adolescent Children in Baramati, Pune District, Maharashtra

V.B. Mandhare, N.S.Rajurkar* and N.H.Zatakia

*Department of chemistry, Savitribai Phule Pune University, Pune, **INDIA**

Email: nsraj@chem.unipune.ac.in

Accepted on 14th July 2016

ABSTRACT

Estimation of urinary iodine is a surrogate measure for iodine intake as almost all ingested iodine excreted in the urine. Measurement of urinary iodine is considered a sensitive marker of current iodine intake and can reflect recent changes in iodine status. Estimation of urinary iodine is cost-efficient and easily obtainable indicator for iodine status. Therefore, urinary iodine measurement affords a biological indication of iodine deficiency disorders (IDD). The present cross-sectional study assessed the iodine status of adolescent (N=82), by estimating urinary iodine using Sandell-Kolthoff reaction. The results revealed that 32.93% of school-children (12-16 year old) have optimum iodine intake, 26.83% have risk of iodine induced hyperthyroidism, 24.40% have risk of hyperthyroidism and autoimmune thyroid disease, 8.54% have mild deficiency, 4.87% have moderate deficiency and 2.43% have severe deficiency respectively.

Keywords: Iodine Deficiency Disorder (IDD), Urinary Iodine.

INTRODUCTION

Urinary iodine is considered a sensitive marker of current iodine intake and can reflect recent changes in iodine status. Individual's urinary iodine concentrations can vary daily, or even within the same day [1, 2]. Iodine is an essential micronutrient for humans; the recommended intake of iodine is 100-150µg day^{-1}. The majority of dietary iodine (>90%) is excreted in the urine [3]. Hence, estimation of urinary iodine is considered as a surrogate measure for iodine intake. Urinary iodine analysis is most common biochemical method used for assessing the iodine status of population and therefore plays an important role in public health surveillance in many countries. Iodine deficiency develops due to imbalance between dietary iodine intake and body requirements. If an environment is adequate in iodine content, humans have no difficulty in obtaining the required small amount of iodine through food and water [4].

Iodine deficiency disorder (IDD) is a significant public Health problem and is the most common cause of brain damage throughout the world (WHO/UNICEF/ICCIDD, 1992). Iodine deficiency disorders (IDD) are linked to iodine deficient soil. Due to glaciations, flooding, rivers changing course and deforestation in the iodine present in top soil is constantly leached. This in turn leads to deficiency of iodine in crops grown on iodine deficient soil with consequently low iodine in the diet for livestock and humans. In the past, iodine deficiency was thought to cause only goiter and cretinism. However, over the past quarter of the century, it has become increasingly clear that iodine deficiency leads to a much wider spectrum of disorders commencing with the intrauterine life and extending through childhood into adult life with serious health and social problems. The spectrum of disease includes goiter, cretinism, hypothyroidism, brain damage, abortion, still birth, mental retardation, psychomotor defects and hearing and speech impairment [5]. Globally 2 billion people are at risk of iodine deficiency disorders due to insufficient iodine intake. Nearly 266 million school-aged children worldwide have insufficient iodine intake. Of the 130 countries which reported data for IDD in 2006, IDD was a public health problem in 47 countries [6]. Globally, India has the largest number of children born vulnerable to iodine deficiency disorder [7]. In India the entire population is prone to iodine deficiency disorder (IDD) due to deficiency of iodine in the soil of the subcontinent and consequently the food derived from it. Of these, an estimated 350 million people are at risk of iodine deficiency disorder (IDD) as they consume salt with inadequate iodine. Every year nine million pregnant women and newborns are at risk of IDD in India [8]. The present work is undertaken to estimate the urinary iodine concentration in school going children from Baramati dist. Pune, India.

MATERIALS AND METHODS

In this cross-sectional study, 82 urine samples were collected randomly from a mixed population of school children. These children were belonging to lower socio economic-strata from Baramati, district Pune, India. Informed written consent was taken from each subject and from their guardian prior to the study. Information of age, height, education, food habits, and family income etc. was collected by using semi-structured questionnaires. Collected urine samples in clean sterile bottles were coded and placed in a deep freezer at -20^0 C until further analysis. These urine samples were then analyzed using Sandell-Kolthoff reaction.

Reagents used for Urinary iodine Analysis: The following regents were used in Sandell-Kolthoff reaction for urinary iodine measurement. All the reagents were obtained from Merck (AR)
1] Ammonium persulfate- 1 mol L^{-1}
2] Conc. H_2SO_4 (98%)
4] Arsenious acid solution (As_2O_3) - 0.0253mol L^{-1}
5] Ceric ammonium sulfate solution - 0.0158mol L^{-1}
5] Standard iodine solution (KIO_3)
All the solutions including calibrators was prepared in deionized water and stored in amber coloured bottle

Biochemical assessments: Urine samples were collected in a clean sterile bottle and stored in a deep freezer for further analysis. Iodine in the urine sample was determined by following the recommended improved ammonium persulfate method of ICCIDD/WHO/UNICEF (1990). It works under the principal of oxidation reduction of ceric ammonium sulfate solution and arsenious acid solution. In this method, urine samples are digested initially with ammonium persulfate solution. After the digestion, iodide in urine reacts with ceric ammonium sulfate solution and reduces ceric ion (Ce^{+4}) to cerous (Ce^{+3}) ion and oxidation of arsenic ion from (As^{+3}) to As^{+5}. The chemical representation of Sandell-Kolthoff reaction is described as follows:

$$2Ce^{+4} + 2I^- \longrightarrow 2Ce^{+3} + I_2$$
$$I_2 + As^{+3} \longrightarrow As^{+5} + 2I^-$$

The ceric ion (Ce^{+4}) has yellow color, while the cerous ion (Ce^{+3}) is colorless. Thus, the course of the reaction can be followed by the disappearance of yellow color as the ceric ion is reduced [9].

RESULTS AND DISCUSSION

Total 82 urine samples were examined and obtained data was analyzed with the help of WHO/UNICEF/ICCIDD guideline [10] as given in table 1.

Table1. Epidemiological criteria based on the WHO/UNICEFF/ICCIDD guidelines

Urine iodine in children (μg L^{-1})	Iodine intake	Iodine nutritional status
< 20	Insufficient	Severe deficiency
20-49	Insufficient	Moderate deficiency
50-99	Insufficient	Mild deficiency
100-199	Adequate	Optimal
200-299	More than adequate	Risk of iodine induced hyperthyroidism
≥ 300	Excessive	Risk of hyperthyroidism and auto immune thyroid disease

Status of urinary concentration level in relation to deficiency or otherwise is presented in table-2.

Table 2. Urinary iodine status of adolescent (N=82)

Urinary iodine status	**N (%)**
Severe deficiency (< 20 μg/L)	2.43 (2)
Moderate deficiency (20-49 μg/L)	4.87 (4)
Mild deficiency (59-99 μg/L)	8.54 (7)
Optimal (100-199 μg/L)	32.93 (27)
Risk of iodine induced hyperthyroidism (200-299 μg/L)	26.83 (22)
Risk of hyperthyroidism & autoimmune thyroid disease (≥300 μg/L)	24.40 (20)

An examination of table 2 reveals that 32.93% of school-children have optimum iodine intake, 26.83% have risk of iodine induced hyperthyroidism, 24.40% have risk of hyperthyroidism and autoimmune thyroid disease, 8.54% have mild deficiency, 4.87% have moderate deficiency and 2.43% have severe-deficiency.

Table 3 depicts the urinary iodine concentrations in girls by age group.

Table 3. Urinary iodine status in girls by age group (N=42)

	Urinary iodine concentration μg/L					
Age (Years)	**Severe deficiency** (< 20 μg/L)	**Moderate deficiency** (20-49 μg/L)	**Mild deficiency** (59-99 μg/L)	**Optimal** (100-199 μg/L)	**Risk of iodine induced hyperthyroidism** (200-299 μg/L)	**Risk of hyperthyroidism & autoimmune thyroid disease** (≥300 μg/L)
	N (%)	**N (%)**	**N (%)**	**N (%)**	**N (%)**	**N (%)**
12 (N=1)	-	-	1 (100)	-	-	-
13 (N=15)	5 (33.33)	1 (6.67)	1 (6.67)	1 (6.67)	-	7 (46.66)
14 (N=20)	2 (10)	-	1 (5)	7 (35)	6 (30)	4 (20)
15 (N=5)	-	1 (20)	2 (40)	1 (20)	-	1(20)
16 (N=1)	-	-	-	1 (100)	-	-
Total (N=42)	7 (16.66)	2 (4.76)	5 (11.90)	10 (23.81)	6 (14.29)	12 (28.58)

The table shows that amongst 12-16 years old girls, 14 year old girls had 10 % low iodine intake and 46.66 % had the highest urinary iodine value. The highest prevalence of iodine deficiency disorder (33.33%) was found in 13 year old girls. Optimal urinary iodine value (35 %) was found in 14 year old girls only. Further 28.58% girls show high urinary iodine level while 23.81% show optimal urinary iodine value and 16.66% had very low iodine level. Thus they are prone to hypothyroidism induced by iodine deficiency.

Table 4 shows the urinary iodine concentration of boys by age group. As indicated in the table 4, 27.27% boys show the highest urinary iodine level and 36.38% show the adequate urinary iodine level. 12-16 year age group show high iodine level i.e. 27.50% it may be due to the high consumption of iodinated food or iodized salt. These children are prone to have hyperthyroidism induced by excessive amount of iodine, similarly for the same age group, 42.50% show the optimal urinary iodine concentrations.

Table 4. Urinary iodine Status in boys by age group

Urinary iodine concentration µg/L						
Age (Years)	**Severe deficiency** (< 20 µg/L)	**Moderate deficiency** (20-49 µg/L)	**Mild deficiency** (59-99 µg/L)	**Optimal** (100-199 µg/L)	**Risk of iodine induced hyperthyroidism** (200-299 µg/L)	**Risk of hyperthyroidism & autoimmune thyroid disease** (≥300 µg/L)
	N (%)	**N (%)**	**N (%)**	**N (%)**	**N (%)**	**N (%)**
12 (N=2)	-	-	-	1 (50)	1 (50)	-
13 (N=9)	-	1 (11.11)	1 (11.11)	2 (22.22)	3 (33.34)	2 (22.22)
14 (N=22)	-	1 (4.54)	1 (4.54)	8 (36.38)	6 (27.27)	6 (27.27)
15 (N=4)	-	-	-	3 (75)	1 (25)	-
16 (N=3)	-	-	-	3 (100)	-	-
Total (N= 40)	-	2 (5)	2 (5)	17 (42.50)	11 (27.50)	8 (20)

APPLICATIONS

Sandell-Kolthoff reaction provides cost effective method estimation of urinary iodine which helps in preliminary diagnosis of iodine disorder. The method can be applied routinely for such studies.

CONCLUSIONS

- Urinary iodine estimation provides a simple method for preliminary estimation of iodine disorders.
- Among 82 children, 2% have severe iodine deficiency, 4% have moderate deficiency, 7% have mild deficiency, 27% have optimal iodine intake, 22% have risk of iodine induced hyperthyroidism and 20% have risk of hyperthyroidism & autoimmune thyroid disease respectively.

ACKNOWLEDGEMENTS

Authors are thankful to BCUD, Savitribai Phule Pune University for financial support under UGC-UPE phase II

REFERENCES

[1] Em. DeMaeyer, F.W. Lowenstein, C.W. Thilly, ‘The control of endemic goiter’, Geneva: World Health Organization, **1979**.

[2] L.B. Rasmussen, L. Ovesen ‘Day-to-day and within day variation in urinary iodine excretion’, *Eur. J. Clin. Nutr.*, **1999**, 53, 401-407.

[3] F. Delange ‘Optimal Iodine Nutrition during pregnancy, Lactation and the Neonatal period’, *Int J Endocrinol Metab*, **2004**, 2, 1-12.

[4] Report to the 43rd World Health Assembly. World Health Organization, Geneva., **1990**

[5] B.S. Hetzel, F. Delange, J.T. Dunn, J. Ling, Mannar Venkatesh, C.S. Pandav, Editors, ‘Towards the global elimination of brain damage due to iodine deficiency’**,** International Council for the

Control of Iodine Deficiency Disorders. New Delhi: Oxford University Press; **2004**.
[6] B de Benoist, E. McLean, M. Anderson, L. Rogers, ‘Iodine deficiency in 2007’: Food Nutr Bull, **2008**, 29, 195-202.
[7] http://www.iodinenetwork.net/documents/scorecard-2010
[8] http://www.unicef.org/india/health.html
[9] A Muhammad, A. B. Javed , F. Khan, ‘Iodine status of children (8-11 years) in district Peshawar, Pakistan’, ARPN Journal of Agriculture and Biological Science, **2011**, 6, 1-6.
[10] WHO, UNICEF, ICCIDD ‘progress towards the elimination of IDD Geneva: WHO’, 1999 **2001.**

AUTHORS’ ADDRESSES

1. **Prof. (Mrs.) N.S. Rajurkar**
Head, Department of Chemistry,
Savitribai Phule Pune University,
Ganeshkhind Road, Pune- 411 007
Mobile: 09822599531, E-mail: nsraj@chem.unipune.ac.in
2. **Viju B. Mandhare**
Department of Chemistry,
Savitribai Phule Pune University,
Ganeshkhind Road,Pune- 411 007
Mobile: 09764557744, E-mail: vbmandhare007@gmail.com
3. **Dr. Neeta H. Zatakia**
Department of Chemistry,
Savitribai Phule Pune University,
Ganeshkhind Road, Pune- 411 007
Mobile: 09860182881, E-mail: neeta3210@gmail.com