Journal of Toxicology and Environmental Health Sciences Vol. 2(7), pp. 93-96, December 2010
Available online at http://www.academicjournals.org/JTEHS
ISSN 2006-9820 ©2010 Academic Journals

Full Length Research Paper

High urinary iodine content (UIC) among primary school children in Ibadan, Nigeria, a public health concern

Onyeaghala A. A.[1*], Anetor J. I.[1], Nurudeen A.[1] and Oyewole O. E.[2]

[1]Department of Chemical pathology, University College Hospital, Ibadan. Nigeria.
[2]Department of Health Promotion and Education, Faculty of Public Health, University of Ibadan.

Accepted 9 August 2010

Urinary iodine excretion is a good marker for the dietary intake of iodine, and is the index for evaluating the degree of iodine deficiency, correction and toxicity. A study, investigating the random urinary iodine level in school children in Ibadan, a South-Western cosmopolitan city of Nigeria, has not been evaluated, thus the emanation of this study. Random urinary iodine was measured in 300 primary school children in Ibadan after obtaining their consent. The urinary iodine level was measured using the standard method of ammonium persulphate reaction. Classifying the urinary iodine level obtained based on World Health Organization (WHO), United Nation's International Children Emergency Fund (UNICEF) and International Council for the Control of Iodine Deficiency Disorders (ICCIDD) recommendation, it was found that 15 (5%) had moderate iodine deficiency, 15 (5%) had mild iodine deficiency, 69 (23%) fell into the sufficient group and 201 (67%) fell into the excess group, with urinary iodine level greater than 300 µg/L. This study infers that if this trend continues unmonitored, the entire population could be prone to developing iodine induced hyperthyroidism (IIH) with the associated toxicity.

Key words: Iodine, hyperthyroidism, iodine deficiency disorders, Iodine induced hyperthyroidism.

INTRODUCTION

Iodine Deficiency Disorder (IDD) is a major global cause of morbidity, mortality and impaired develop-ment[1]. Universal salt iodisation has been extremely effective in reducing the burden of IDD and represents a major global public health success (Dunn et al., 1998). In Africa, great progress has been made towards the elimination of iodine deficiency, saving millions of children from its adverse affects, largely due to the increased household availability of iodized salt.

The daily recommended intake of iodine: 150 µg for adults, 200 µg during pregnancy, 50 µg for the first year in life, 90 µg for ages 1 to 6, and 120 µg for ages 7 to 12 (Dunn et al., 1998). The trace element iodine is an essential nutrient for human growth and development.

The thyroid gland depends on iodine for production of thyroid hormone and this is one of the physiological functions of iodine. The association between iodine deficiency and endemic goiter has been known for centuries. These disorders are collectively described as iodine deficiency disorders (IDD) and it includes: Endemic goiter, hypo-thyroidism, cretinism and congenital abnormalities (Dunn et al., 1998). While endemic goiter is the most visible consequence of iodine deficiency, the most significant and profound effects are on the developing brain. The potential impact of iodine deficiency on the intellectual development of large segments of the populations in underdeveloped countries is of particular concern, especially when all of the adverse effects of iodine deficiency can be prevented by long-term, sustainable iodine prophylaxis.

*Corresponding author. E-mail: vip4162003@yahoo.co.uk, oaugustine@in.com.

In most countries of the world, universal salt iodization has been employed as a means of eliminating disorders secondary to iodine deficiency. WHO, UNICEF and ICCIDD has brought iodine sufficiency within reach of about 1.5 billion people of the world who were deficient decades ago; and now rely on the urinary iodine concentration as the primary indicator of effectiveness (WHO, ICCIDD, 1999).

In Africa and indeed Nigeria, great progress has been made towards the elimination of iodine deficiency, saving millions of children from its adverse effects, largely due to the increased household availability of iodized salt (International Council for the Control of Iodine Deficiency Disorders, 2003; World Health Organization, 2007; WHO, UNICEF, ICCIDD, 2001; Lantum, 2009). However, the relationship between iodine intake and the risk of thyroid disease is U-shaped, with both low and high iodine intake being associated with thyroid disease (Laurberg et al., 2001). The effect of the consumption of additional iodine is also dependent on the initial status of the population. In populations that are mildly deficient or replete, increases in dietary iodine may induce hypothyroidism, while in populations that were previously severely deficient, increased dietary iodine is associated with hyper-thyroidism (Markou et al., 2001; Stanbury et al., 1998).

In Nigeria, the National Agency for Food and Drug Administration and Control (NAFDAC) has greatly promoted salt iodization using public campaigns (Lantum, 2009). This, coupled with the influx of Western diets into the country has exposed lots of families to consume high levels of iodized salts and iodine containing food. Currently, the programme is now in place to monitor the level of high iodine intake in the population. This study was therefore, carried out to investigate the levels of exposure to iodine in the study population.

MATERIALS AND METHODS

Selection of patients and sample collection

This study was a public health study. The subjects were 300; apparently healthy primary school children, with no history or biochemical marker suggestive of renal failure, whose mean age were 9.45 ± 1.26 years, and comprised both males and females. However, they had been resident in Ibadan for at least five years. Primary school children are appropriate population group for the assessment of iodine status because of their physiological vulnerability.

Furthermore, measurement of urinary iodine levels in schoolchildren is important for public health considerations, as this group effectively reflects the current status of IDD in the general population, as well as the extent to which IDD control measures have had an impact on the population.

All the pupils were in their early primary school years. Prior to the study, consent was received from the school authorities, parents and pupils. Anthropometric measurements of the study population were taken. 10 ml random urine sample was collected into clean and sterile universal bottle from all the pupils who were chosen at random. Since the samples were not analysed immediately, they were stored and frozen at -20°C until they were ready for analysis.

Table 1. Measured parameters in the study population.

Variable	Mean ± SD
Age (yrs)	9.45 ± 1.26
Height (m)	1.31 ± 0.11
Weight (kg)	23.71 ± 5.28
Waist circumference (m)	0.48 ± 0.17
Body mass index (kg/m^2)	14.03 ± 1.87
Mean urinary iodine (µg/l)	317.13 ± 118.29

Table 2. Classification of iodine nutrition of the studied population based on the epidemiological criteria for assessing Iodine nutrition using joint criteria of WHO, UNICEF and ICCIDD (2001).

Range (µg/L)	Distribution (%)
Severe (< 20)	0
Moderate (20 - 49)	15 (5)
Mild (50 - 99)	15 (5)
Sufficient (100 - 199)	69 (23)
Excess (> 300)	201 (67)

Analytical method

The standard method (the ammonium persulphate technique) was used for estimating the level of iodine in the urine (Dunn et al., 1993). Urine is digested with ammonium persulphate. Iodine present in the urine acts like a catalyst in the reduction of ceric ammonium sulphate (yellow) to cerous ammonium sulphate (colourless). The degree of disappearance of the yellow colour is a measure of iodine content in the urine. A standard curve plotted during the analysis was used to extrapolate the concentration of iodine in the urine samples.

RESULTS

Table 1 shows the measured parameters in the studied population. The urinary iodine obtained from the subjects was classified into various degrees of iodine deficiency using the criteria stipulated by the World Health Organi-zation. The results are shown in Table 2. From the table, it was evident that none of the pupils had any degree of iodine deficiency. However, 5% (15) fell into moderate and mild levels of iodine deficiency respectively. Further-more, 23% (69) had optimal level and 67% (201) were excessively sufficient with iodine.

DISCUSSION

Urinary iodine excretion is a good marker of the dietary intake of iodine, and is the index for evaluating the degree of iodine deficiency, correction and toxicity. Many countries have adopted massive salt iodisation as a means of correcting IDD in countries where they were

prevalent. The results from this study show that none of the pupils had any degree of severe iodine deficiency. This is a welcome development in terms of public health and could attest that lots of families have massively embraced salt iodisation. However, that there were still mild to moderate iodine deficiency as shown from our study could imply that not all families have adopted the massive iodization process. This finding is consistent with the findings of Mu et al. (2001), who reported mild to moderate iodine deficiency across the populations they studied including school children. Iodized salt is widely available commercially, but it may not be impossible that only a hand full of all households' purchase iodized salt for domestic use. This however, needs to be evaluated through further study.

The contribution of iodized table salt in the production of iodine nutrition is probably insignificant. Most of the salt in our diet comes from salt added during preparation and processing of food. The assessment, monitoring and evaluation of the iodine content of salts imported and those produced within the country are currently poor. It is not unlikely that household salt may not contain the recommended level of iodine. The World Health Organization has recently recommended that iodization of salts should be maintained at concentration of 20 to 40 ppms (World Health Organization (WHO)/United Nations Children's Fund/International Council for Control of Iodine Deficiency Disorders, 1996).This is in contrast to the present legislation on salt iodization in the country which recommended salt iodization at 50 ppm/kg salt.

The excretion of high urinary iodine found in the studied population is of great public health concern. Our studies showed that 201 (67%) of the pupils had excess urinary iodine. Our finding was consistent with the report of Delange et al. (1999), who reported high concentrations of urinary iodine in some African countries few years after the introduction of massive iodization programme. They concluded that the risk of Iodine Induced Hyperthyroidism (IIH) after correction of iodine deficiency is closely related to a recent excessive increment of iodine supply. Ibadan, a South –Western state of Nigeria is not included in the goiter belt region of the country; and has been assumed to be sufficient in iodine even prior to salt iodization campaign. It is therefore, not impossible that the massive iodization programme embarked by the government has exposed residents to higher concentrations of iodine.

Furthermore, the daily recommended allowance of iodine for children is about 150 µg. Large quantities of iodide are present in drugs, antiseptics, bread, food preservatives and some fast food products. Considering the flair that school children have for bread and fast foods, it is likely that these food products could contribute to the increased level of iodine observed. Further reasons that could be responsible for the high urinary iodine level observed could be due to poor monitoring of the production, quantity and quality of iodine use for food production and preservation. In most instances, the monitoring of iodised salt focuses mainly on whether iodine is present or not but the quantity is not always considered.

The World Health Organization has reported that population that exposed to excess concentrations of urinary iodine could be prone to developing Iodine induced Hyperthyroidism (IIH) and autoimmune thyroid disorders (Dunn et al., 1998). The thyroid gland has intrinsic mechanisms that maintain normal thyroid function even in the presence of iodine excess; however, this mechanism can be depleted at increased iodine levels (Roti and Uberti, 2001). It has widely been reported that administration of iodine in any chemical form could induce the development of IIH. In iodine-sufficient areas, iodine-induced hyperthy-roidism has been reported in euthyroid patients with or without previous thyroid diseases (Stanbury et al., 1998).

IIH is most commonly encountered in older persons with long standing nodular goiter and in regions of chronic iodine deficiency, but instances in the young have been abound (Stanbury et al., 1998). It generally occurs after an incremental rise in mean iodine intake in the course of programmes for the prevention of iodine deficiency. There is evidence, particularly in animals, that high levels of iodine can be a contributing factor in the development of autoimmune thyroid disease. The production of excess iodine–rich thyroglobulins, which may possess some immunogenic properties, has been incriminated to contribute to the development of IIH. Some, but not all, studies on humans have shown that the presence of serum anti-microsomal antibodies is more prevalent in areas having adequate iodine than in areas of mild iodine deficiency. Mutational events in thyroid cells have also been incriminated to be responsible for the development of IIH. These events lead to autonomy of function. When the mass of cells with such an event becomes sufficient and iodine supply is increased, the subject may become thyrotoxic. These changes may occur in localized foci within the gland or in the process of nodule formation.

In conclusion, this study showed that high urinary iodine level is present in the studied population and it has public health implication. Despite remarkable progress in the control of the iodine deficiency disorders (IDD), they remain a significant global public health problem. Assessing the control of IDD and preventing the development of IIH, and monitoring the progress of salt iodisation programmes are cornerstones of a control strategy. Thyroglobulin has become a promising new biochemical marker for the diagnosis of iodine induced hyperthyroidism.

Adequate monitoring using this marker is highly recommended if the development of IIH is to be prevented.

REFERENCES

Delange F, de Benoist B, Alnwick D (1999) Risks of iodine-induced hyperthyroidism after correction of iodine deficiency by iodized salt. Thyroid Jun., 9(6): 545-556.

Dunn JT, Semigran MJ, Delange F (1993). Methods for measuring Iodine in urine. International Council for the Control of Iodine Deficiency Disorders.
Dunn JT, Semigran MJ, Delange F (1998). The prevention and management of iodine-induced hyperthyroidism and its cardiac features. Thyroid, Jan., 8(1): 101-6
International Council for the Control of Iodine Deficiency Disorders. IDD Newsletter (2003). Iodine Nutrition in Africa. IDD Newsletter 19: 1-6.
Lantum DN (2009). Did universal salt iodization help reduce the infant mortality rate in Nigeria? IDD Newslett., May, 32 (2): 6-7.
Laurberg P, Bulow Pedersen I, Knudsen N, Ovesen L, Andersen S (2001). Environmental iodine intake affects the type of nonmalignant thyroid disease. Thyroid, 11: 457-469.
Markou K, Georgopoulos N, Kyriazopoulou V, Vagenakis AG (2001). Iodine-induced hypothyroidism. Thyroid, 11: 501-510.
Mu Li, Gary Ma, Karmala Guttikonda , Steven C Boyages ,Creswell J Eastman (2001). Re-emergence of iodine deficiency in Australia. Asia Pacific J. Clin. Nutr., 10(3): 200-203.
Roti E, Uberti ED (2001). Iodine excess and hyperthyroidism. Thyroid, 11(5): 493-450.
Stanbury JB, Ermans AE, Bourdoux P, Todd C, Oken E, Tonglet R, Vidor G, Braverman LE, Medeiros-Neto G (1998). Iodine-induced hyperthyroidism: occurrence and epidemiology. Thyroid, 8: 83-100.
WHO, UNICEF, ICCIDD (2001). Assessment of the Iodine Deficiency Disorders and monitoring their elimination. Geneva: World Health Organization; 2001. WHO Document WHO/NHD/01.1.
WHO, ICCIDD (1999). Progress towards the elimination of iodine deficiency disorders (IDD) Geneva: WHO, publisher,pp1-13
World Health Organization (2007). Assessment of iodine deficiency disorders and monitoring their elimination a guide for programme managers. – 3rd ed.
World Health Organization (WHO)/United Nations Children's Fund/International Council for Control of Iodine Deficiency Disorders (1996). Recommended Iodine Levels in Salt and Guidelines for Monitoring their Adequacy and Effectiveness. WHO/NUT/96.13. Geneva: WHO.