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Iodine Deficiency in Rajasthan: Causes and Challenges

Dr. Priti Mathur

Associate Professor, Department of Zoology, Government Bangur PG College, Pali, Rajasthan, India

ABSTRACT: Iodine deficiency is a lack of the trace element iodine, an essential nutrient in the diet. It may result in metabolic problems such as goiter, sometimes as an endemic goiter as well as congenital iodine deficiency syndrome due to untreated congenital hypothyroidism, which results in developmental delays and other health problems. Iodine deficiency is an important global health issue, especially for fertile and pregnant women. It is also a preventable cause of intellectual disability.

Iodine is an essential dietary mineral for neurodevelopment among children.[1] The thyroid hormones thyroxine and triiodothyronine contain iodine. In areas where there is little iodine in the diet, typically remote inland areas where no marine foods are eaten, iodine deficiency is common. It is also common in mountainous regions of the world where food is grown in iodine-poor soil.

Prevention includes adding small amounts of iodine to table salt, a product known as iodized salt. Iodine compounds have also been added to other foodstuffs, such as flour, water and milk, in areas of deficiency.[2] Seafood is also a well known source of iodine.[3]

KEYWORDS-iodine,thyroid,goiter,deficiency,Rajasthan

I. INTRODUCTION

A low amount of thyroxine (one of the two thyroid hormones) in the blood, due to lack of dietary iodine to make it, gives rise to high levels of thyroid stimulating hormone (TSH), which stimulates the thyroid gland to increase many biochemical processes; the cellular growth and proliferation can result in the characteristic swelling or hyperplasia of the thyroid gland, or goiter. In mild iodine deficiency, levels of triiodothyronine (T_3) may be elevated in the presence of low levels of levothyroxine, as the body converts more of the levothyroxine to triiodothyronine as a compensation. Some such patients may have a goiter, without an elevated TSH. Goiter is said to be endemic when the prevalence in a population is > 5%, and in most cases goiter can be treated with iodine supplementation. If goiter is untreated for around five years, however, iodine supplementation or thyroxine treatment may not reduce the size of the thyroid gland because the thyroid is permanently damaged.[1,2,3]

Congenital iodine deficiency syndrome, previously known as cretinism, is a condition associated with iodine deficiency and goiter, commonly characterised by mental deficiency, deafness, squint, disorders of stance and gait and stunted growth due to hypothyroidism. Paracelsus was the first to point out the relation between goitrous parents and their mentally disabled children.[9]

As a result of restricted diet, isolation, intermarriage, etc., as well as low iodine content in their food, children often had peculiar stunted bodies and retarded mental faculties, a condition later known to be associated with thyroid hormone deficiency.

There is preliminary evidence that iodine deficiency enhances the sensitivity of breast tissue to estrogen.[11][12] In rats treated with estradiol, iodine deficiency has been shown to lead to changes similar to benign breast changes that are reversible by increased iodine in the diet.[11][12] In a few studies, iodine supplementation had beneficial effects (such as reducing the presence of breast cyst, fibrous tissue plaques and breast pain) in women with fibrocystic breast changes.

Following is a list of potential risk factors that may lead to iodine deficiency:[17]

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1. Low dietary iodine
2. Selenium deficiency
3. Pregnancy[18]
4. Exposure to radiation
5. Increased intake/plasma levels of goitrogens, such as calcium
6. Sex (higher occurrence in women)
7. Smoking tobacco
8. Alcohol (reduced prevalence in users)
9. Oral contraceptives (reduced prevalence in users)
10. Perchlorates
11. Thiocyanates[5,7,8]
12. Age (for different types of iodine deficiency at different ages)

Iodine accounts for 65% of the molecular weight of T_4 and 59% of T_3. There is a total of 15–20 mg of iodine in the human body, primarily concentrated in thyroid tissue and hormones. Thirty percent of iodine is distributed in other tissues, including the mammary glands, eyes, gastric mucosa, choroid plexus, arterial walls, the cervix, and salivary glands. In the cells of these tissues, iodide enters directly by sodium-iodide symporter (NIS).

Iodine deficiency disorders (IDD) is a public health problem in India. A ban on the sale of uniodised salt for household consumption has been introduced in Rajasthan State since 1992. The present study was conducted in the district of Bharatpur, Rajasthan with the objective to assess the prevalence of iodine disorders in school children as no data is available on this aspect. A total of 3072 children in the age group of 6-12 years were included in the study and were clinically examined. On the spot urine samples were collected randomly from 450 children. A total of 1064 salt samples were collected randomly from the families of the children. The total goiter prevalence was found to be 7.2% in the subjects studied. It was found that the percentage of children with urinary iodine excretion <20.0; 20.0-49.9, 50.0-99.9 and 100 mcg/L and above was 1.1, 1.1, 7.8 and 90.0% respectively. The assessment of iodine content of salt revealed that 56% of the families were consuming iodised salt. The findings indicated that the population is in a transition phase from iodine deficient (as revealed by the TGR) to iodine sufficient (as revealed by the medium UIE of 200.0 mcg/L) nutriture.[9,10,11]

II. DISCUSSION

The diagnostic workup of a suspected iodine deficiency includes signs and symptoms as well as possible risk factors mentioned above. A 24-hour urine iodine collection is a useful medical test, as approximately 90% of ingested iodine is excreted in the urine.[20] For the standardized 24-hour test, a 50 mg iodine load is given first, and 90% of this load is expected to be recovered in the urine of the following 24 hours. Recovery of less than 90% is taken to mean high retention, that is, iodine deficiency. The recovery may, however, be well less than 90% during pregnancy, and an intake of goitrogens can alter the test results.[21]

If a 24-hour urine collection is not practical, a random urine iodine-to-creatinine ratio can alternatively be used.[20] However, the 24-hour test is found to be more reliable.[22]

A general idea of whether a deficiency exists can be determined through a functional iodine test in the form of an iodine skin test. In this test, the skin is painted with an iodine solution: if the iodine patch disappears quickly, this is taken as a sign of iodine deficiency. However, no accepted norms exist on the expected time interval for the patch to disappear, and in persons with dark skin color the disappearance of the patch may be difficult to assess. If a urine test is taken shortly after, the results may be altered due to the iodine absorbed previously in a skin test.[21]

The prevalence of iodine deficiency in the Bikaner district of Rajasthan, India, was estimated in a 1995 pilot project involving 527 children 6-12 years of age recruited from three high schools. Goiter, as detected by palpation, was present in 20.5% of these children. A goiter prevalence above 5% is considered indicative of endemic iodine deficiency. According to urine analyses, 3% of children had severe iodine deficiency (urinary excretion levels under

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2 mcg/ml), 9% had moderate deficiency (2.0-4.9 mcg/ml), 18% had mild deficiency (5.0-9.9 mcg/ml), and the remaining 70% had adequate iodine levels (10 mcg/ml and above). 32% of families were using salt with no iodine content; another 8% consumed salt with less than 15 ppm of iodine. These findings indicate that iodine deficiency is a serious public health problem in this district and suggest a need to strengthen monitoring of the quality of salt procuredin Rajasthan.

III. RESULTS

Treatment


Iodine supplements

Iodine deficiency is treated by ingestion of iodine salts, such as found in food supplements. [12,13,15]Mild cases may be treated by using iodized salt in daily food consumption, or drinking more milk, or eating egg yolks, and saltwater fish. For a salt and/or animal product restricted diet, sea vegetables (kelp, hijiki, dulse, nori (found in sushi)) may be incorporated regularly into a diet as a good source of iodine.[20]

The recommended daily intake of iodine for adult women is 150–300 µg for maintenance of normal thyroid function; for men, it is somewhat less at 150 µg.[20]

Prognosis

With iodine supplementation, goiters caused by iodine deficiency decrease in size in very young children and pregnant women. Generally, however, long-standing goiters caused by iodine deficiency respond with only small amounts of shrinkage after iodine supplementation, and patients are at risk for developing hyperthyroidism.[20]

Being pregnant while iodine-deficient additionally carries the risk of causing congenital iodine deficiency syndrome in the newborn. This disease can only be managed by lifelong administration of thyroxine (T_4).[23]

In areas where there is little iodine in the diet, typically remote inland areas and semi-arid equatorial climates where no marine foods are eaten, iodine deficiency gives rise to hypothyroidism, symptoms of which are extreme fatigue, goiter, mental slowing, depression, weight gain, and low basal body temperatures.[33]

Iodine deficiency is the leading cause of preventable mental retardation, a result which occurs primarily when babies or small children are rendered hypothyroidic by a lack of the element. The addition of iodine to table salt has largely eliminated this problem in the wealthier nations, but as of March 2006, iodine deficiency remained a serious public health problem[17,18,19]

The sample of 105 school children (12-15 years) of Jodhpur district were assessed by clinical examination of thyroid gland using the standard method as recommended by the joint WHO / UNICEF / ICCIDD consultation, morbidities, Iodine in urine and salt using standard laboratory technique. Total goiter rate was 11.4 %, all of them had grade I goiter. The goiter prevalence in male and female was found to be 7.4 % and 15.7% respectively. The median urinary iodine excretion was 166.2 µg/ l. Proportion of the children in severe grade (<20 µg/l) was 5.0 %. Overall high proportion of school children (81.5%) consumed salt having inadequate iodine content and proportion was significantly higher in females than males ($P<0.05$).The proportion of households consuming adequate iodized salt increased with decline of income.[31,32,33] The proportion of households consuming adequate iodized salt increased with increase of housing conditions grades, found statistically significant. Sickness at the time of survey

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was 16.2 % and the overall morbidities observed were significantly higher in females (19.6 %) than males (P<0.05). Total goiter rate was high and only 18.5 percent of salt had adequate iodine content indicating that the consumption of iodized salt in desert area is extremely low in spite of the national programs in operation and needs more attention. In addition to iodization of salt, there is a strong need of formulating nutritional intervention packages for this region of Rajasthan[20,21,22]

IV. CONCLUSION

Iodine deficiency disorders (IDDs) have been recognized as one of the major nutritional disorders throughout the world affecting 200 million people who are at risk and another 71 million suffering from goiter and other IDDs. These groups of disorders can affect every stage of life, but most vulnerable age group is between 6 and 12 years and these disorders together constitute the single largest preventable cause of brain damage leading to learning disabilities and psychomotor impairment. [28,29,30]The existence of endemic goiter in Rajasthan has long been described, but consistently high prevalence of IDDs outside the endemic zones and failure to attain goals set by the National Iodine Deficiency Disorder Control Program questions the strategy and achievements till date. Therefore, the present article is an attempt to critically examine the program since inception in Rajasthan[23,25,27]

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