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Current Status of Iodine Deficiency Disorders Control Program

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ABSTRACT

The need of the study is because we all know very well that Iodine plays a very important role in our endocrine gland thyroid due to deficiency and sufficiency of iodine in global level or our country . our society suffers various disorders. Government and other health organisations try to control iodine deficiency disorders. So, in this study we will see iodine deficiency disorders control programme implemented by National /International level.

Key Words : Iodine, Deficiency disorders, Thyroid

INTRODUCTION

Iodine has been considered as an essential nutrient throughout different span of life especially developmental stages (Bleochradt and Born, 1994). If a pregnant woman is starved of iodine, the fetus cannot produce enough thyroxine with consequent retardation of physical and mental growth. Hypothyroid fetuses often perish in the womb and many affected infants die within a week of birth. Hypothyroid children are intellectually subnormal may also suffer physical impairment. Studies have documented that in an areas with and incidence of mild to moderate disorder due to iodine deficiency, IQs of school children are ,on an average, 13 points below those of children living in areas where there is no iodine deficiency. On the other hand, iodine deficiency has been considered as the most common preventable cause of mental deficiency in the world including India where iodine deficiency disorders constitute a major health problem. Out of 457 districts in the country 275 districts have been surveyed for IDD out of those, 235 districts have been found to be endemic. These districts cover all the States and UTs of India (WHO, 1997). Following the successful trial of iodised salt in Kangr valley of Himachal Pradesh, a national goitre program was launched by the government of India in 1962 with the objectives (i) to survey the problem of iodine deficiency in the country, (ii) produce and supply iodizes salt, and the (iii) resurvey the area after five years to access the impact of iodized salt program. The objective of universal iodization of salt for human and animal consumption was added to the national program in 1983 (Sudersan, 1998).

Until 1990, only a few countries Switzerland, some of the Scandinavian countries Australia the US and Canada were completely iodine sufficient. Since then, globally the number of household using iodized salt has risen from <20% to >70%, dramatically reducing iodine deficiency. This effort has been achieved by a coalition of international organisations including ICCIDD (now IGN), WHO, MI and UNICEF, working closely with national IDD control committees and the salt industry; this informal partnership was established after the world summit for children in 1990 (Pearce *et al.,* 2013).

Zimmermann and Anderson (2012) revealed that the two most commonly used approaches to assessing iodine nutrition on the population level are estimation of the household penetration of adeqately iodized salt (HHIS) and measurement of urinary iodine cocentrations (UICs). UIC surveys are mainly done in school aged children (SACs), because they are convenient population easy to reach through school based surveys and usually representative of general population. There fore, WHO

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use UICs fron 6-12-y- old children in nationally representative surveys, expressed as median in ug/L, to classify a population iodine status more countries are beginning to carry out studies in high risk population groups *i.e.* women of reproductive age, pregnant women and younger children , however data is limited and majority of countries still conduct routine iodine monitoring in SACs (Iodine global network, 2016). In 2017, representative UIC surveys are available for 139 countries. There are no up -to-date UIC data available for 55 countries. Available UIC data now cover > 98% of world's population of SAC.

UNICEF (2015) reported that approximately 75% of households worldwide have access to iodized salt. Those with greatest access are living in the WHO regions of eastern Mediterranean region. The international child development steering group identified iodine deficiency are one of the four key global risk factors for impaired child development where the need for intervention is urgent (Walker *et al.,* 2007) but controlling IDD in the remaining 1/3rd of the global population at risk will not be easy. Although the key contributors to successful national programs have been identified, reaching economically disadvantage groups living in remote areas and convincing small scale salt producer to iodize their salt are major challenges. An important strategy will be to strengthen national coalitions that include government partners national and international agencies, the health care sectors and salt producers. In the countries that have begun iodized salt programs, sustainability will become a major focus. These programs are fragile and require long term commitment from governments. In several countries where iodine deficiency have been eliminated , salt iodization program fell apart and Iodine deficiency recurred (Dunn, 2006). Children in Iodine deficient areas are vulnerable to even short term lapses in iodized salt programs.

Advocay should focus on damage to reproduction and cognitive development. Governments need to understand the serious impact of iodine deficiency; many still equate iodine deficiency with goitre, a mostly cometic and thus a low priority. IDD is one of the most important cause of preventable neurocognitive impairment worldwide, and elimination of IDD can contribute to at least five of millennium development goals (The united nations, 2007); 1) Eradicate extreme poverty and hunger, 2) Achieve universal primary education; 3) Reduce child mortality; 4) Improve maternal health; and 5) develop a global partnership for development. The world bank (182) strongly recommends that government invest in micronutrient programs, including salt iodization , to promote development, concluding; "Probably no other technology offers as large an opportunity to improve lives at such low cost and in such a short time" (Mc Guire and Galloway, 1994).

Monitoring the impact of programs of salt iodization:

The social process for successful implementation of national IDD control program includes the following components :

– Situation assessment

– Communication of results to health professionals, political authorities and the public

– Development of an action plan

– Implementation of the plan

– Evaluation of its impact at population level

The last phase monitoring is often neglected not only because it is the last phase in the process but because it may be over shadowed by other components of program such as implementation. In addition, many countries affected by IDD are low -income countries without the financial or technical resources to support a laboratory needed to properly monitor salt quality and iodine status.

The most effective way to achieve the virtual elimination of IDD through universal salt iodization ,USI. The indicators used in monitoring and evaluating IDD control programs include both indicators to monitor and evaluate the salt iodizataion process as well as indicators to monitor the impact of salt iodization on the target populations (these have been discussed previously).

It is considered that iodine deficiency has been eliminated from the country when

– access to iodized salt at household level is at least 90%

– the median urinary Iodine concentration is at least 100ug/L and with less than 20% of the samples below 50ug/L

– when at least 8 of 10 program indicators are implemented, these indicators are

(i) An effective, functional national body (council or committee) responsible to the government for national program for elimination if IDD (this body should be multidisciplinary, involving the relevant fields of nutrition, medicine, education, the salt industry, the media and consumers with chairman appointed by the ministry of health.

(ii) Evidence of political commitment to universal salt iodization and elimination program.

(iii) Appointment of a responsible executive officer for IDD elimination program.

(iv) Legislation or regulation on universal salt iodization (While ideally regulations should cover both human and agricultural salt. If latter is not covered this does not necessarily preclude a country from being certified as IDD-free).

(v) Commitment to assessment and reassessment of progress in the elimination of IDD, with access to laboratories able to provide accurate data to provide accurate data on salt and urinary iodine.

(vi) A program of public education and social mobilization on the importance of IDD and the consumption of iodized salt.

(vii) Regular data on salt iodine at the factory, retail and at household level.

(viii) Regular laboratory data on urinary iodine in school aged children with appropriate sampling for higher risk areas

(ix) Cooperation from the salt industry in maintenances of quality control.

(x) A database for recording of results or regular monitoring procedures particularly for salt iodine , urinary iodine and if available neonatal TSH, with mandatory public reporting.

Currently, there is much less information available on the impact of salt iodization programs that on the implementation of programs.

Also surprisingly, few longitudinal or case control studies have addressed the influence of USI on disorders induced by iodine deficiency, such as impairment of thyroid function, low birth weight, perinatal mortality and morbidity and the prevention of mental retardation. The oft-quoted statement that correction of deficiency protects 50-100 million neonates from brain damage and mental retardation annually is politically attractive, but scientifically questionable. It results simply from a multiplication of the birth rate of affected countries by the percentage of access to iodized salt at household level. Both figures lack precision. Moreover, this calculation implies that 100% of neonates born in iodine deficient areas before the implementation of programs of iodine supplementation suffered intellectual impairment, which is a gross overestimation.

The risk of excess iodine intake

A discussed so far in this chapter, iodine deficiency impairs thyroid function. Similarly, iodine excess including overcorrection of a previous state of iodine deficiency, can also impair thyroid function. The effort of iodine on thyroid gland is complex with a "U- shaped " relation between iodine intake and risk of thyroid diseases. Both low and high iodine intake and risk of thyroid disorders. Healthy adults with normal thyroid glands can tolerate up to 600-1000 ug iodine per day without any side effects (WHO, 1994). However , this upper limit is much lower in a population which has been exposed to iodine deficiency for a prolonged period in the past. The optimal level of iodine intake to prevent the disease may be relatively narrow range around the recommended daily intake of 150ug (Kudsen *et al.*, 2000).

When iodine intake chronically high as in coastal areas of Japan deuce to daily intake of seaweeds rich in iodine or in Eastern China , because of high iodine content of drinking water from the shallow wells , the prevalence of thyroid enlargement and goiter is high as compared to populations with normal iodine intake. Also, the prevalence of subclinical hypothyroidism is elevated. The mechanism behind this impairment of thyroid function are probably both iodine enhancement of thyroid autoimmunity and reversible inhibition of thyroid function by excess iodine (the wolf checkoff effect) in susceptible subjects (Roti *et al*., 2000). However, this type of thyroid failure has not been observed in neonates after the administration of huge doses of iodized oil to their mothers during pregnancy. Increased thyroid volume in children due to iodine excess has been observed when median urinary iodine is > 5000ug/L (Zimmermann *et al.,* 2005).

Iodine induced hypothyroidism (IIH) is the main complication of iodine prophylaxis. Stantury *et al.* (1998) reported in the most iodine supplementation programs. But it is rare following as well executed program of iodine supplementation. The outbreak of IIH most extensively investigated occurred in Tasmania in late 1960s. This followed iodine supplementation simultaneously by tablets of iodine, iodized bread and the use of iodophors by the milk industry (Conolly *et al.,* 1970). The incidence of hypothyroidism increased from 24/100000 in 1963 to 125/100000 in 1967. The disease occurred most frequently in individuals over 40 years of age with multinodular goitre and preexisting heart diseases. The most severe manifestations were cardiovascular and were occasionally fatal. The epidemic lasted about 10 to 12 years, but it was followed by an incidence of hypothyroidism somewhat less than that existing prior to epidemic.

Todd *et al.* (1995) reported that the introduction of iodized salt in Zimbawe resulted in sharp increase in the incidence of IIH from 3/100000 to 7/100000 over 18 months. A high risk of IIH was also reported from eastern Congo following the introduction of iodized salt (Bourdoux *et al.,* 1996). A multicentre study conducted in seven African countries including zimbawe and Congo showed that occurrence if IIH in last two countries was due to sudden introduction of poorly monitored and excessively iodized salt in populations which had been severely iodine deficient for very long periods in the past. The conclusion of the study was that the risk of IIH was related to a rapid increment of iodine intake resulting in state of acute iodine overload (Delange *et al.*, 1999).

Baltisberger *et al.* (1995) reported that IIH following iodine fortification of salt cannot be entirely avoided even when fortification provides only physiological amounts of iodine. In a well controlled longitudinal study in Switzerland, the incidence of hypothyroidism transiently increased by 27% during one year after iodine supply was increased from 90 ug/day to 150 ug/day.

The reason for the development of IIH after iodine fortification and /or supplementation is thought to be that iodine deficiency increases thyrocite proliferation and mutation rates which in turn , trigger the development of multifocus autonomal growth and scattered cell clones harboring activation mutations of the TSH receptors (Dremier *et al.,* 1996). Measurement of total intrathyroidal iodine by means of X-ray flourescence scanning shows that only some nodules keep their capacity to store iodine, become autonomous and cause hypothyroidism. It should be noted that there is considerable anecdotal evidence that increase iodine intake in patients with Graves disease my exacerbate hypothyroidism in susceptible patients (Joncheer *et al.,* 1992).

It thus appears that IIH can be considered one of the iodine deficiency disorders, and it may be largely unavoidable in the early phase of iodine repletion in iodine deficient populations, particularly in those with moderate to severe iodine deficiency. Its incidence reverts to normal or even below normal after one to ten years of iodine supplementation (Delange and Lecomate, 2000).

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