Guest Editorial

Goiter Prevalence and Salt Iodization—Pitfalls and Caveats

The etiology of goiter remains a topic of debate. The main etiological factor for endemic goiter worldwide is deficiency of iodine. Since, the seminal work of Sir Robert McCarrison in the western Himalayas in 1902, the large number of studies has been done on iodine deficiency and prevalence of goiters.

Abnormalities caused by deficiency of iodine are termed iodine deficiency disorders (IDD). Zimmermann M et al (2008) claim that, currently in various regions of the world, two billion people have insufficient iodine intake with those in South Asia and sub-Saharan Africa particularly affected. WHO estimates the global goiter prevalence to be 15.8% in the general population (bulletin of World Health Organization 2005). This means that around one billion people in the world have goiters. It is indeed a formidable health care issue especially for the health care of the developing world with limited health budgets as more than 65% of people with goiters live in Asia and other developing countries in the world. Close monitoring of IDD and goiters have to be undertaken to minimize the disease burden in these countries.

The 39th World Health Assembly, in 1986, called for prevention and control of IDD. The 43rd World Health Assembly, in 1990, adopted a resolution to eliminate iodine deficiency as a public health problem. Soon after, 70 heads of state gathered at World Summit for children and pledged to make elimination of IDD as one of the health and social development goals to be achieved by 2000. Universal salt iodization (USI) was identified as the main intervention for achieving this goal. Universal iodization has undoubted beneficial effects. The undisputed benefits of universal iodization, such as prevention of iodine-deficiency goiter, mental and growth retardation, poor productivity and cretinism, have been achieved through joint efforts of international, national and local agencies. Great strides have been made in many countries.

However, caution is needed as over iodization has definite deleterious effects. There is good evidence from many parts of the world that excess iodine will cause autoimmune thyroiditis, hyperthyroidism and probably an increase in papillary carcinoma of thyroid. There is sufficient evidence to suggest that there is an increase in prevalence of autoimmune thyroiditis especially in countries with a policy of universal iodization. The progression of the autoimmune thyroiditis in these countries and morbidity caused by it needs close scrutiny and further study.

It must be emphasized that the eradication of iodine deficiency far outweighs these risks which are almost always self-limiting and disappears over many years as the iodine-deficient population achieves iodine repletion.

Careful monitoring of population given iodine supplementation is essential to be sure that adequate but not excessive iodine intake is being maintained. There is also sufficient data to show that people with excess urinary iodine continue to use iodized salts. It has several health implications. Secondly, a poorly organized and monitored iodization program may infuse a false sense of security regarding the iodine status and endemicity of goiters.

The iodization policy and process need close monitoring. The undeniable benefits of iodization may be negated by lack of monitoring and regular revisions of the policy of iodization, when irrefutable evidence of morbidity due to iodization is presented. Goiters due to iodine deficiency may be supplanted by goiter due to autoimmune thyroiditis resulting from excess iodine.

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