Editorial

Thyroidology and public health: The challenges ahead

Sanjay Kalra, Ambika Gopalakrishnan Unnikrishnan[1], Rakesh Sahay[2]

Department of Endocrinology, Bharti Hospital and BRIDE, Karnal, Haryana, [1]AIMS, Kochi, Kerala, [2]Osmania Medical College, Hyderabad, Andhra Pradesh, India

Among the sub-specialties of endocrinology, perhaps, thyroidology has had the longest association with public health.

In the nineteenth century, various European physicians noted the endemic distribution of goitre and cretinism in specific areas of central Europe and England. The use of seaweed, rich in iodine, was postulated as a treatment for goitre. Soon, salt iodization and iodine supplementation of drinking water were recommended by Boussingault (1833) and Chatin (1850), respectively.[1] Thus, the public health implications of clinical medicine were understood quite early in the evolution of thyroidology.

Much progress was made in this field all over the world. Iodine deficiency disorders (IDDs) were characterized and prevented using salt iodization or iodized oil in various countries.

In some countries, such as the United Kingdom, the control of IDD was serendipitous. Endemic goitre disappeared due to iodine supplementation in diary cattle, which resulted in high iodine levels in milk as well as other dairy products.[2] In other nations, such as India, planned interventions were carried out with advice from expert thyroidologists. The National Goitre Control Programme, launched in 1962, and later renamed the National Iodine Deficiency Disorder Control Programme in 1992, relied on universal salt iodization as a means of eliminating IDD. This importance of iodine in the control of IDD is justified,[3] and has shown good results worldwide. While mixed results have been reported from various IDD control programmes, there is no doubt that the global control of IDD is a major public health achievement of thyroidology.[3,4]

Can we rest on our laurels, through? Can we, as endocrinologists, afford to relax after having contributed to the control of the most common endocrinopathy and the leading cause of mental retardation?

The answer is a definite "no." IDD may reappear if control programmes are not sustained.[4] There is a need to conduct periodic surveys to assess iodization and to ensure physician and public awareness to create a demand for iodized salt.[4]

Equally important, however, are the newer challenges that thyroidology and public health have to meet together.

While the central role of iodine in thyroid function is undisputed, several other minerals and trace elements are also involved in thyroid metabolism. These include iron, selenium and zinc. Coexisting deficiencies of these micronutrients can interfere with thyroid function. Iron deficiency reduces the activity of heme-dependent thyroid peroxidase, and iron supplementation improves the efficacy of iodine supplementation.[5] This is of immense public health importance in India, where the prevalence of iron-deficiency anemia is very high.[6]

Selenium deficiency also contributes to myxedematous cretinism. The thyroid gland expresses many selenocysteine-containing proteins, including glutathione peroxidase, deiodinase and thioredoxine reductase, which are essential for thyroid function. In combined iodine and selenium deficiency, which may occur in cystic fibrosis, malnutrition and in patients on total parenteral nutrition

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Corresponding Author: Dr. Sanjay Kalra, Department of Endocrinology, Bharti Hospital and B.R.I.D.E, Karnal, India. E-mail: brideknl@gmail.com

or phenylketonuria diet, it is essential to achieve an iodine-replete status first before correcting selenium deficiency.

This too is of public health importance. Grains grown in areas irrigated by flood waters have less selenium content than those originating from areas with comparatively less ground water supply.[6] This may explain varying responses to IDD control programmes in different areas of the country. Because of this, there may be a role for micronutrient supplementation in patients with hypothyroidism as well.

In spite of achieving better iodine status in the population, maternal hypothyroidism continues to remain a major public health problem. Sub-optimal control of maternal thyroid status in pregnancy leads to impaired cognitive function in children[7] thus jeopardizing the health of an entire unborn generation. It is imperative, therefore, for thyroidologists to ensure aggressive control of hypothyroidism in antenatal women. The importance of this simple medical intervention, which has the potential to make sweeping changes in improving neonatal brain development, must be explained to gynecologists, other medical personnel and the general public.

In a large study performed on stored thyrotropin samples from 25,216 pregnant women, children aged 7–9 years were found to have intelligence quotients (IQs) that varied with maternal TSH levels. Children of hypothyroid mothers had IQs 7 points lower than those of matched controls, with 19% scoring 85 or less.[7] While the association has been recognized by earlier authors as well,[8,9] the increasing incidence of hypothyroidism in pregnancy, and a greater understanding of the need to achieve " low normal" TSH levels make this respect of thyroidology more important.

The impact of thyroid disease is not limited to expectant mothers and their fetuses. The general population too is exposed to various thyroid disruptor chemicals[10] in the environment. These include chemicals produced as byproducts from various industries. Increasingly, concern has been voiced over the contribution of chemicals such as perchlorate,[11] nitrate,[12] organochlorine insecticides[13] and fungicides[13] to the pathogenesis of thyroid diseases.

Nitrate is a contaminant of drinking water in agricultural areas,[12] and competes with iodide uptake by the thyroid. Perchlorate is an industrial effluent that has similar effects on thyroid physiology. As nitrate and perchlorate are present in high levels in Indian waters,[14,15] it becomes a challenge for endocrinologists to spread awareness about the role of these thyroid disruptors.

An increased risk of thyroid cancer has been noted with increased dietary nitrate intake, longer duration of consumption of nitrate-rich water and higher average nitrate levels in public water supplies (relative risk [RR] = 2.6 [1.1–6.2]).[12] High dietary intake is also associated with higher prevalence of hypothyroidism (RR 1.2 [1.1–1.4]).

Hypothyroidism is also linked with the use of organochlorine insecticides (adjusted odds ratio ORadj = 1.2 [1.0–1.6]) and fungicides (ORadj = 1.4 [1.1–1.8]). However, no association is found with use of herbicides, fumigants, organophosphates, pyrethroids or carbonates. Use of chlordane, an organochlorine (ORadj = 1.3 [0.99–1.7]), bonomyl, a fungicide (ORadj = 3.1 [1.9–5.1]), maneb/mancozeb, another fungicide (ORadj = 2.2 [1.5–3.3]) and paraquat, a herbicide (ORadj = 1.8 [1.1–2.8]) was significantly associated with hypothyroidism among female spouses (n = 16,529) enrolled in the Agricultural Health Study, carried out in the Iowa and North Carolina, USA. Maneb/mancozeb use was linked to both hypothyroidism and hyperthyroidism (ORadj = 2.3 [1.2–2.4]).[13]

India being an agricultural country that extensively uses insecticides and pesticides, the public health implications of these associations are not difficult to understand.

All external influences on the thyroid need not be negative, however. Studies suggest that thyroid cancer risk decreases with greater alcohol consumption (two drinks per day vs none, RR = 0.57 [0.36–0.89]). This analysis was carried out in a cohort of 490,159 men and women enrolled in a prospective study and followed-up over a median 7.5 years duration.[16] Given the ill-effects of alcohol intake, this is obviously not a strategy that has public health importance.

Another public health debate that has stirred up following the recent, tragic nuclear incident at Fukushima, relates to potassium iodide prophylaxis for people exposed to radioactive iodine.[17,18]

While potassium iodine is certainly effective in preventing the uptake of radioactive iodine, the prophylactic dose should be administered within 3 h of exposure to nuclear radiation.[18] The American Thyroid Association suggests making available potassium iodine to all individuals living within 200 miles of nuclear reactors.[19]

A simpler, more ubiquitous, source of ionizing radiation, which is proven to be a risk factor for thyroid cancer, is iatrogenic. It is important to standardize and implement safety instructions related to I-131 therapy, which is used for the management of various thyroid disorders. Safety instructions provided to patients may vary markedly.[20] This too may become a cause for concern as public awareness about the endocrine effects of exposure to radiation grows.

As awareness about thyroid diseases increases among the patients, and as thyroid disease increases among patients, thyroidologists will be frequently called upon to play a role in advocacy and public health. While we should be aware of, and advocate, relevant measures for all the issues listed above, one simple aspect of current medical practice should not be forgotten.

With easy and widespread availability of thyroid-function testing, people often self-prescribe thyroid tests and present to their physician, demanding treatment for "disordered" thyroid function, assuming this to be the cause of all symptoms. Such "direct access testing" (DAT) should be discouraged.[21]

DAT, without guidance of physicians, may lead to erroneous interpretation of thyroid function reports and lead to wrong diagnosis and management. This may lead to worsening of clinical status, and unwanted adverse effects, in some patients.

Thyroidology and public health thus have to deal with a multitude of challenges, ranging from patient awareness about the perils of DAT to advocacy regarding environmental thyroid disruptors.

Through this supplement and through other contributions, Indian journals like the IJEM and the Journal of Thyroid Research and Practice are emerging as strong platforms that will strengthen the public health advocacy efforts of thyroidologists and related medical personnel in India as well as across the globe.

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