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Myths and fallacies in thyroid disease

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Introduction

Thyroid disease is the commonest endocrine disorder encountered by medical practitioners world over. There are several myths and fallacies that continue to influence the management of this common disease. Adherence to these myths and fallacies mar treatment practices, even today, despite good evidence against such practices.

There are several areas in aetiology, diagnosis and management of thyroid disease which are based on myths and fallacies rather than scientific evidence. These aspects need to be highlighted, discussed and reappraised.

Goitrogens

Goitrogens interfere with production of thyroxine in the thyrocytes and eventually cause enlargement of the thyroid. There are several so called "well-known goitrogens". The finding of Chesney, Clawson and Webster that cabbage feeding produced significant thyroid hyperplasia in rabbits was the start of the notion that cabbage was goitrogenic [1].

Subsequently a large number of food like cauliflower, rape, mustard and cabbage seeds, turnip, rutabagas, Brussels sprouts, etc., were found to possess goitrogenic properties [2,3,4]. Most of the workers have shown that, cruciferous vegetables belonging to the Brassica family possessed goitrogenic effects in experimental animals. The isolation of 1, 5-vinyl 2thio-oxazolidone from ground rutabaga (yellow turnip) was of considerable significance and gave a great impetus to the search for similar goitrogenic agents in foods commonly used in various diets [3].

The experimental rabbits in the study by Chesney et al may have been deficient of other nutrients and micronutrients because they were fed mostly on cabbage. The deficiency of many nutrients is more likely have been cause of goitre in rabbits rather than cabbage itself. The theory that cabbage produced goitres in human beings has now been repudiated by several authors [5, 6]. Besides, no human being consumes cabbage exclusively like rabbits. Cooking destroys most goitrogens. It is time that the cabbage and goitre story is debunked and excluded from medical text books. No controlled studies have been conducted on humans on goitrogenesis of these foods.

In modern medical practice, the relevance of goitrogens must be considered carefully lest we perpetuate myths and fallacies. A food item may exert a goitrogenic effect if it is consumed in large amounts as part of the staple diet and not if a vegetable like cabbage is consumed in small amounts. The consumption of a well-balanced high fibre diet with vegetables is far more beneficial than omitting vegetables based on erroneous belief of a possible infinitesimal risk of goitrogenesis.

In a population based descriptive study done in Sri Lanka we were unable to show any association between common food items considered to be goitrogenic and prevalence of goitre [7]. It is time that we advised patients correctly that vegetables including cabbage are not goitrogenic in the manner it is consumed by most people.

Examination of the thyroid gland

In modern high tech medical practice there is a tendency not to emphasize the importance of clinical examination. A medical practitioner, especially in a developing country, must learn the technique of examining the thyroid gland properly. The technique of thyroid examination needs reappraisal. The landmark for the thyroid gland is the cricoid cartilage and not the thyroid cartilage. The whole gland must be observed and palpated in a methodical manner.

The traditional method of palpating from behind has been taught and practiced for a long time. The disadvantages of this method are that many features which can be best observed from the front, such as the obvious nodularity, symmetry evidence of compression of the gland and the position of the trachea etc. are missed. If the examiner proceeds to examine from behind prematurely (as most people do). Going behind and palpating is only one method of palpation and NOT examination of the thyroid gland as a whole. There is a need to emphasize that the thyroid must be examined both from the front and behind.

Other aspects of thyroid examination like percussion for retrosternal extension and palpating for the trachea are done in the traditional manner, and these techniques are erroneous. The principle of percussion is to obtain a normal 'note' of the area percussed and look for an abnormality thereafter. One cannot obtain a note by percussing over the manubrium as most people do and are taught to do. Percussion must start from the apex area of lung i.e. upper chest and then percussion directed towards the goitre.

It is a large goitre which will exert a mass effect and cause deviation and compression of the trachea, which is important to determine in the examination. The standard method taught for palpation of the trachea, even in text books, is to feel for it at the supra sternal notch. If a large goitre, especially with sub or retrosternal extension, is present the fallacy of this teaching is obvious as the trachea is not palpable in the suprasternal area in such a scenario as it is covered by the enlargement of the thyroid. The only place where trachea is always palpable (however big the goitre) is just below the thyroid cartilage. The palpation of trachea must begin from the thyroid cartilage and path of the trachea followed downwards. This can easily be achieved.

There are several other aspects in thyroid examination such as technique of eliciting eye signs for detecting early exophthalmos and eliciting lid lag which needs reappraisal and rationalization. Medical practitioners especially the doctors of the future must learn to do these assessments properly instead of blindly adhering to the traditional methods, which in some instances are vestiges of the colonial past, as repetition of the technique was required to pass examinations.

Size of the thyroid

One of the commonest disorders of the thyroid is enlargement of the thyroid gland or goitre. The first issue that a doctor has to resolve is, whether there is true enlargement of the thyroid gland. It is generally accepted that the thyroid should be enlarged at least three times before it is clinically palpable.

Clinical assessment of the size of the thyroid is not the most reliable method of assessing the size of the thyroid. This is more so in the examination of small goitres which even experienced examiners find difficult. Yet the size of the gland needs to be determined before clinical decisions are made. The 'old' method of comparing the normal size of a lobe as equivalent to the size of the patients thumb has some merit. More reliance is placed currently on ultrasound scan examination to assess the size. Several authors state that significant inter- and intra-observer variation occur in sonographic measurements of thyroid volume [8-10]. Proper training and experience is needed before reliance can be placed only on ultrasonography to assess thyroid size. Delange concluded that "the suitability of the concept of universal normative values for thyroid volume measured by ultrasonography can be questioned" [8].

A large study on ultrasound scans assessment of goitres, state that "thyroid ultrasound is subjective because finding and measuring the maximum diameters require judgment and experience" [11]. In addition thyroid size differs in different populations. The size of the thyroid is affected by several factors including the iodine status of a population. Many regions of the world have not developed the reference range for thyroid volumes based on ultrasonography yet. Most ultrasound scan based assessments of thyroid are based on figures from Western literature, hence sometimes when goitre is visible, the ultrasound scan reports the gland as normal. One reason for this is that most estimates of size are not based on three dimensional assessments. The best method of assessing size of thyroid ultrasonically is debatable too.

The well-known method is based on the three axes of each lobe and a new principle based on planimetry in two planes is used commonly [12].

Volumetric evaluation of the thyroid gland is based on the use of an ellipsoid model. The value obtained thus, replaces clinical evaluation of volume. With the ellipsoid model, the height, width, and depth of each lobe are measured and multiplied. The obtained result is then multiplied by a correction factor, which is $\pi / 6$, or 0.524 [13].

In a small study done using the ellipsoid model at Teaching Hospital Ragama, it was found that the mean thyroid volume was 8.918ml. The reference values of thyroid volume (TV) were 8.919 ±5.168 ml and 6.62 ± 3.09 ml for males and females respectively. There was a significant difference in TV between genders (p<0.001).The TV was closer to values given in Asian studies than the standard Western figures used commonly by radiologists.

A large scale population based study must be undertaken in conjunction with the radiologists to obtain the references range for thyroid size and volumes for Sri Lanka, yet there may be regional variation which needs to be determined subsequently and factored in.

Empirical use of thyroxine in benign goitres

Thyroxine is empirically used by many doctors to treat benign goitres irrespective of the cause of the goitre and its nodularity. This is done for endemic as well as sporadic goitre with the aim of suppressing TSH. This mode of treatment was popularized by the findings of Astwood and his colleagues in 1960 [14]. In this series of 230 patients, 24% with nodular goitre had a complete response after administration of thyroid extract. This was not a controlled trial. Due to the reasonable response obtained, many people started adopting this therapeutic option without good evidence to support it.

In the last decade, this therapeutic concept has been cautioned against and the role of TSH in goitre development questioned. This is because TSH mainly regulates thyroid function, induces hypertrophy of the follicular cells and increases the blood flow within the thyroid gland. The intrathyroidal mechanisms and the increased activity of local growth factors may however be the main causes of initiation, promotion and maintenance of hyperplasia hence suppression of TSH has little effect in suppressing the growth of the gland. There is a reduction in goitre size around 30-40% in some patients. This effect does not continue and the size remains reduced by 30-40% only. Stopping treatment results in an increase of goitre volume within a few weeks [15]. Studies and data on long term use of thyroxine are not available and caution is needed in the use of thyroxine suppressive therapy in benign disease.

Unfortunately some clinicians start patients on thyroxine even before a hormone profile is obtained. This clearly has negative consequences including iatrogenic hyperthyroidism. A proper assessment of morphology, function and histology must be ascertained before considering any empirical therapy. There is very little or no value in suppressing TSH if the initial value is within the normal range or low. There are several unanswered questions in this empirical method of treatment such as; What is the correct dose and what is the appropriate duration?[16].

It must be emphasized that suppressive thyroxine therapy for benign disease has very limited usefulness and is contraindicated in patients with suppressed TSH. A recent meta-analysis has cautioned about the doubtful benefits of long-term suppressive therapy [17]. At the recommended dose thyroxine may cause cardiac and bone side effects [18]. The empirical use of thyroxine must be done with a great deal of care. It must only be used for a short period of time.

The management of thyroid disorders is a common clinical problem. Outlined above are the some fallacies myths and false beliefs that hamper proper treatment and of this condition. Awareness of these issues will enable the clinician to give proper advice and make appropriate assessments and decisions regarding treatment.

References

1. Chesney AM, Clawson TA,Webster B. *Bull Johns Hopkins Hosp* 1928; **431**: 261.
2. Clements FW. Naturally occurring Goitrogens. *Br Med Bull* 1960; **16**: 133-7.
3. Gaitan E. Goitrogens in Food and Water. *Annu Rev Nutr* 1990; **10**: 21-39.
4. Kennedy T H, Purves H D. Studies on experimental Goitre. The effect of Brassica seed diet on rats. *Br J Exp Pathol* 1941; **22**: 241.
5. Astwood, EB, Greer MA, Ettlinger MG, The antithyroid factor of yellow turnip. *Science* 1949; **109**: 631.
6. Greer MA. Goitrogenic Substances in Food. *Am J Clin Nutr* 1957; **5**: 440-4.
7. Fernando R, Pinto MDP, Pathmeswaran A. Goitrogenic Food and Prevalence of Goitre in Sri Lanka. *International Journal of Internal Medicine* 2012; **1**: 17-20.
8. Delange F. What do we call a goiter? *Eur J Endocrinol* 1999; **140**: 486-8.
9. Peterson S, Sanga A, Erlof, et al. Classification of thyroid size by palpation and ultrasonography in field surveys. *Lancet* 2000; **355**: 106-10.
10. Jarløv AE, Nygard B, Hegedus L, Karstrup S, Hansen JM. Observer variation in ultrasound assessment of the thyroid gland. *Br J Radiol* 1993; **66**: 625-7.
11. Zimmermann MB1, Hess SY, Molinari L, New reference values for thyroid volume by ultrasound in iodine-sufficient

schoolchildren: a World Health Organization/Nutrition for Health and Development Iodine Deficiency Study Group Report. *Am J Clin Nutr* 2004; **79**: 231-7.

12. Knudsen N, BuÈlow I, Jùrgensen T, et al. Goitre prevalence and thyroid abnormalities at ultrasonography: A comparative epidemiological study in two regions with slightly different iodine status. *Clin Endocrinol (Oxf)* 2000; **53**: 479-85.

13. Shabana W, Peeters E, De Maeseneer M. Measuring Thyroid Gland Volume: Should we change the correction factor? *AJR Am J Roentgenol* 2006: **186**; 234-6.

14. Astwood EB, Cassidy C E, Aurbach GD. Treatment of goiter and thyroid nodules with thyroid. *JAMA* 1960 Oct 1; **174**: 459-64.

15. Gärtner R .Thyroxine treatment of benign goiter. *Acta Medica Austriaca* 1994; **21**: 44-7.

16. Koutras DA. The medical treatment of non-toxic goiter: several questions remain. *Thyroidology* 1993; **5**: 49-55.

17. Bandeira-Echtler E, Bergerhoff K, Richter B. Levothyroxine or minimally invasive therapies for benign thyroid nodules. *Cochrane Database Syst Rev* 2014; **6**: CD004098.

18. Premawardhana LDKE, Lazarus JH. Management of thyroid disorders. *Postgrad Med J* 2006; **971**: 552-8.

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