Iodine Deficiency: An under Recognized Problem

Sir,

We read with interest the original article by Palaniappan et al. on iodine excess and Hashimoto’s thyroiditis in children in which authors have reported a possible link between excess iodine intake by children and increasing the prevalence of autoimmune thyroiditis and eventually thyroid hypofunction.\[1\]

In this regard, we would like to highlight recently published data on iodine status in several studies carried out in our country highlighting that iodine deficiency still continues to be endemic throughout India.\[2,3\] In the state of Tamil Nadu, the overall utility of iodine-rich salt among households and 6–12 years children has been extensively evaluated by Pandav et al. with estimations of urinary iodine excretion (UIE) and goiter indices, respectively. His study among school children aged between 6 and 12 years age has reported consumption of iodized salt at 18%, total goiter index of 13.5%, median UIE <100 mcg/L in 56%, and below 50 mcg/L in 22% of the children.\[4\]

As most reference ranges of TSH, free T4 and UIE levels are strongly determined by diurnal and circadian variations, quality control standards for all biological samples in particular for UIE status become important. Several studies carried out in state of Tamil Nadu and Chhattisgarh have utilized stringent external and internal quality standards greatly adding to the quality of data presented.\[5\] By not including iodine deficient children and the prevalence of autoimmune thyroid disease in the iodine deficient cohort, it is difficult to accept a causal link between the iodine excess and autoimmune thyroid disease. We would also like to know about the laboratory details where the urine iodine was performed and standardization procedure undertaken in this aspect. The mean and standard deviation of the urine iodine excretion between the two groups are presented with a P value, but the confidence intervals of UIE between the two groups could better highlight the overlap.

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Sudha Rathna Prabhu, Subramanian Kannan, Shriraam Mahadevan
Department of Genetcs, MEDISCAN, Chennai, Tamil Nadu,
Department of Endocrinology, Diabetology and Bariatric Medicine, Narayana Health City, Bengaluru, Karnataka, Department of Endocrinology, Diabetes and Metabolism, Sri Ramachandra Medical College and Research Institute, Chennai, Tamil Nadu, India

Address for correspondence:
Dr. Shriraam Mahadevan,
Department of Endocrinology, Diabetes and Metabolism,
Sri Ramachandra Medical College Medical, Porur,
Chennai, Tamil Nadu, India.
E-mail: mshriraam@gmail.com

REFERENCES
1. Palaniappan S, Shanmughavelu L, Prasad HK, Subramaniam S, Krishnamoorthy N, Lakkappa L. Improving iodine nutritional status and increasing prevalence of autoimmune thyroiditis in children. Indian J Endocrinol Metab 2017;21:85-9.
2. Shinde M, Joshi A, Naik G, Trivedi A. Prevalence of goiter and the status of iodized salt among the primary school children of a rural district in central India. Natl J Community Med 2015;6:51-5.
3. Pandav CS, Krishnamurthy P, Sankar R, Yadav K, Palanivel C, Karmarkar MG. A review of tracking progress towards elimination of iodine deficiency disorders in Tamil Nadu, India. Indian J Public Health 2010;54:120-5.
4. Pandav CS, Yadav K, Srivastava R, Pandav R, Karmarkar MG.
Parathyromatosis Following Endoscopic Parathyroid Surgery: A Rare Occurrence

Sir,

Parathyromatosis, a cause for recurrent hyperparathyroidism, is common in middle-aged women and chronic kidney disease patients. It involves multiple nodules of benign hyperfunctioning parathyroid tissue scattered throughout the neck and mediastinum.

Primary or Type 1 parathyromatosis is the result of hyperplasia of parathyroid rests from embryologic development.

Secondary or Type 2 parathyromatosis, a rare complication of parathyroidectomy, first described in 1975 by Palmer et al., is more common which arises due to seeding of parathyroid tissue during surgery. It has been also considered as a low-grade malignancy.

Thirty-five cases have been reported so far. Its preoperative diagnosis is rare due to the lack of awareness of this entity. Sonographic imaging provides clues to the diagnosis.

Medical and surgical interventions carry high failure rates. Cinacalcet and bisphosphonates are main-stays of medical therapy. Alcohol ablation and several novel calcimimetics have been used in these patients.

Repeated neck explorations to remove parathyroid implants are often unsuccessful.

We came across a 55-year-old male with bone pains, pruritus, polyuria, difficulty in getting up from the chair for the last 1 year. He was operated in the past for renal stones. He was diagnosed with primary hyperparathyroidism (PHPT) due to left superior parathyroid adenoma and got operated endoscopically for the same. Postsurgery, discharged medications included oral calcium and Vitamin D supplements. Two years after the surgery, he presented with bone pains and increasing fatigue.

Physical examination was unremarkable. Serum calcium was 14 mg/dl, intact parathyroid hormone (PTH) was 1400 pg/ml, and 24-h urinary calcium was 649.28 mg/dl, suggestive of recurrent hyperparathyroidism. Ultrasonography, sestamibi and positron emission tomography scans failed to localize lesion.

Differential diagnosis of incomplete removal of adenoma, hyperplasia, multiple/ectopic adenoma, malignancy, and parathyromatosis were considered. On exploration, multiple nodules (<5 mm) were evident in the left-side neck compartment embedded in strap muscles, sternocleidomastoid, on thyroid surface, and left central compartment. Right parathyroid glands were normal. The patient underwent left hemithyroidectomy, removal of ipsilateral straps, parts of sternocleidomastoid, berry picking of superficial nodules, and clearance of tissue close to the entry of ports. Postoperatively, serum calcium was 9.7 mg/dl and PTH (<2.5 pg/ml) was undetectable. Histopathology revealed multiple, small, hypercellular parathyroid glands along with normal looking thyroid follicles with diagnosis of parathyromatosis.

On 1-year follow-up, serum calcium was normal.

Parathyromatosis is a rare but clinically relevant disease. It is characterized by ectopic hormone secreting parathyroid tissue scattered throughout the neck and mediastinum. It may be considered a benign malignancy with locally invasive behavior.

In our opinion, the cause of secondary hyperparathyroidism, in this case, was most likely due to rupture of capsule leading to spillage of tumor cells during removal of parathyroid adenoma by endoscopic parathyroid surgery. Secondary parathyromatosis following endoscopic parathyroid surgery has not been reported so far. The preoperative diagnosis of parathyromatosis poses great challenges and needle aspiration may be helpful in selected patients.