Case Report

IODINE DEFICIENCY–INDUCED GOITER IN CENTRAL NEW JERSEY: A CASE SERIES

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ABSTRACT

Objective: We report 4 cases of iodine deficiency–induced goiter in minority women living in central New Jersey.

Methods: Clinical presentations, laboratory data, thyroid ^{123}I uptake, and thyroid sonograms were obtained from 4 minority women with iodine deficiency–induced goiter. The recent literature on the topic was also reviewed.

Results: Four patients were diagnosed with iodine deficiency–induced goiter during a 4-year period in an endocrine clinic in central New Jersey. The diagnosis of iodine deficiency–induced goiter was made if the patient met the following criteria: (*1*) a diet history that indicated avoidance of iodized salt or seafood; (*2*) a 24-hour urinary iodine concentration of <100 μg; and (*3*) a decrease in the size of the goiter determined by ultrasonography of the thyroid after iodine replacement. Thyroid volume was calculated as width × length × height × π/6 cm^3. Three of the four patients had an increase in radioactive iodine uptake.

Conclusion: Iodine deficiency–induced goiter still occurs in New Jersey, the residents of which are supposedly iodine replete. It is important to obtain dietary history during the evaluation of patients with euthyroid goiter, especially in minority women. Measurement of urinary iodine excretion is warranted in suspicious cases. Iodine deficiency–induced goiter can be treated by adding iodized salt to the diet. **(AACE Clinical Case Rep. 2015;1:e40-e44)**

Abbreviations:
T_4 = thyroxine; **TSH** = thyroid-stimulating hormone

INTRODUCTION

Since the introduction of iodine supplementation of salt and other foods starting in the 1920s, iodine deficiency has been rare in the United States (1). It can still be observed in Africa, Europe, and Southeast Asia and in Western Pacific and Eastern Mediterranean regions (2). Progress has been achieved in eliminating iodine deficiency worldwide through programs of universal salt iodization as recommended by the World Health Organization and the International Council for the Control of Iodine Deficiency Disorders (3). Iodine supplementation in iodine-deficient regions has been shown to decrease thyroid volume (4-6). Based on the findings of the U.S. Food and Drug Administration's Total Diet Study for 2003-2004, the estimated average daily iodine intake ranges from 138 to 353 μg/person (7). However, sporadic cases of euthyroid goiter related to iodine deficiency are reported (8), even in New Jersey (9), the residents of which are supposedly iodine replete. Here, we report the cases of 4 minority women with iodine deficiency–induced euthyroid goiters.

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METHODS

Four patients were diagnosed with iodine deficiency–induced goiter since June 2006 in an Endocrinology Clinic in central New Jersey. Recorded clinical data included patient age, ethnicity, body mass index (BMI), systolic blood pressure, diastolic blood pressure, dietary history,

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goiter duration, levels of thyroid-stimulating hormone (TSH), free thyroxine (T_4), total T_4 and total triiodothyronine, thyroid ^{123}I-uptake scan, and 24-hour urine iodine level. Goiter sizes determined by physical exam and ultrasound were documented at the initial visit and after iodine treatment. Patients' past medical history and family history were also documented.

The 24-hour urinary iodine excretion was measured by a commercial laboratory, with a sensitivity of 10 μg/L. In our study, iodine deficiency–induced goiter was diagnosed by: (*1*) a full diet history of avoidance of dairy foods, seafood, and iodized salt, indicating possible iodine deficiency; (*2*) a 24-hour urine iodine level of <100 μg, suggesting possible iodine deficiency; and (*3*) shrinkage in the size of goiter determined by ultrasonography of the thyroid repeated after iodine treatment. The same radiology group obtained the thyroid sonograms before and after iodine treatment. Thyroid gland volume was calculated as width × length × height × π/6 cm^3 (10).

The data were expressed as mean and SD, median and number of patients in each category, and percentage of total patients in that group. Normal distributions between groups were compared using a 2-tailed paired t-test. $P<.05$ was selected as the level of significance.

CASE REPORTS

Since June 2006, 28 patients with a euthyroid goiter who had a dietary history of avoidance of dairy foods, seafood, or iodized salt had a 24-hour urine iodine level measured. A total of 8 patients had a 24-hour urine iodine level <100 μg, among which 4 had repeated thyroid sonograms available before and after iodine replacement.

Case 1

A 24-year-old woman originally from Brazil presented with goiter. She was allergic to shellfish and did not use iodized salt by choice. Her family history showed her aunt had hyperthyroidism. A physical exam showed a BMI of 27 kg/m^2 and a palpable thyroid. She had a normal thyroid-function test (TSH, 0.78 μU/mL) and was negative for thyroid peroxidase antibody. An iodine-uptake scan showed a 24-hour uptake of 37%, and a thyroid sonogram showed a diffuse goiter (Table 1). These findings, together with a history of avoidance of iodized salt and seafood, raised the suspicion of iodine deficiency. Her 24-hour urine iodine level was <10 μg. The patient was advised to take iodized salt and a multivitamin containing iodine. A thyroid sonogram obtained 11 months later showed that the thyroid volume decreased by 34.5%, from 26.41 to 17.29 cm^3 (Fig. 1).

Case 2

A 40-year-old Asian American woman who immigrated to New Jersey from Hong Kong 10 years previously presented with a goiter of 10-years duration. She had 2 fine-needle biopsies done in 2000 and 2004, which were both negative for malignant cells. Her family history was negative for thyroid disease. Physical examination was notable only for a large, uneven, soft bilateral goiter.

Table 1
Basic Characteristics of 4 Patients With Iodine Deficiency–Induced Goiter[a]

Patient No.	Age (years)	Gender	Ethnic Group	TSH (μU/mL)	Free T_4 (ng/dL)	TPO Ab (IU/mL)	Neck Exam	Thyroid Sonogram	^{123}I Uptake Scan (24-h)	24-h Urine Iodine (μg)
1	24	F	Hispanic	0.78	1.2	<10	Diffuse goiter	Diffuse, prominent	Diffuse increase 37%	<10
2	39	F	Asian	0.67	1.21	<10	Diffuse goiter	MNG	Diffuse increase 68.7%	30
3	38	F	Asian	0.54	NA	<10	Diffuse goiter	Diffusely enlarged	Diffuse increase 37%	90
4	83	F	Asian	0.97	1.97	40	Diffuse goiter	Diffusely enlarged	NA	64
Mean (SD)	53 (26)			0.73 (0.22)	1.59 (0.54)					<48.5 (17.8)

Abbreviations: F = female; MNG = multinodular goiter; NA = not available; T_4 = thyroxine; TPO Ab = thyroid peroxidase antibody; TSH = thyroid-stimulating hormone.
[a] Normal ranges: TSH, 0.35-5.5 μU/mL; Free T_4, 0.9-1.8 ng/dL; TPO Ab, <30 IU/mL; ^{123}I uptake, 15-35% at 24-hours; 24-hour urine iodine, 100-460 μg/L.

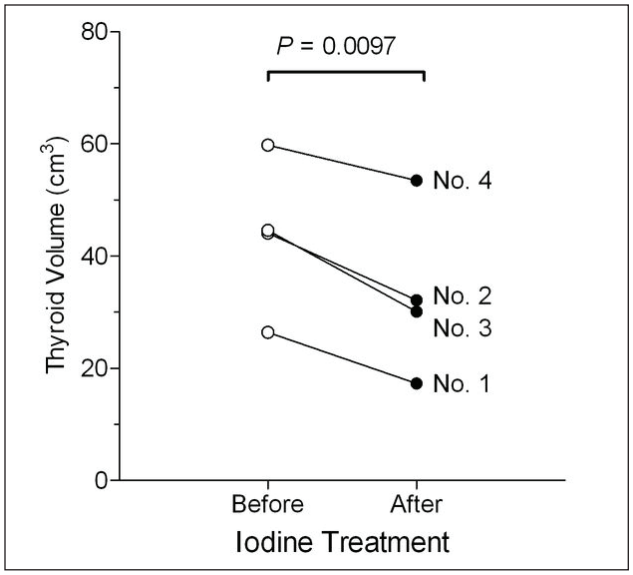

Fig. 1. Thyroid volumes before and after iodine supplementation. Thyroid volumes were measured using ultrasound imaging and were compared before and after iodine supplementation. *P* value was calculated using a 2-tailed paired t-test.

Values from thyroid laboratory tests were all within normal limits (TSH, 0.67 μU/mL). The thyroid peroxidase antibody test was negative. A scan for ^{123}I uptake showed a 24-hour uptake of 69% and a diffusely enlarged gland. The 24-hour urine iodine level was 30 μg. Further discussion with the patient revealed that she habitually refrained from consuming iodized salt and typically did not consume iodine-supplemented foods. She was diagnosed with goiter secondary to iodine deficiency and started on iodized salt therapy. A repeat thyroid sonogram obtained 9 months later showed that the thyroid volume decreased by 27.1%, from 44.12 to 32.16 cm^3 (Fig. 1). We continued to follow up with the patient for possible side effects of increased iodine intake. She continued to do well and remained euthyroid, with goiter size getting smaller upon examination.

Case 3

A 38-year-old woman originally from Lebanon who had thyroid goiter was referred by her primary care physician for evaluation. The patient recalled having a goiter for at least 1.5 years and underwent biopsy of a left thyroid nodule, which was negative for malignancy. Her dietary history indicated avoidance of iodized salt and seafood by choice. Her sister also had a thyroid disorder of an unknown type. Diffuse goiter was palpable upon exam. Values from laboratory tests showed normal thyroid function (TSH, 0.54 μU/mL), and her 24-hour urine iodine level was 90 μg. Ultrasonography of the thyroid showed a diffuse goiter (Table 1). She was diagnosed as having iodine deficiency–induced goiter and was instructed to take iodized salt. A thyroid sonogram obtained 3 years later showed that the thyroid volume decreased by 32.5%, from 44.6 to 30.1 cm^3 (Fig. 1).

Case 4

An 83-year-old Asian female with a prior medical history of hypertension, osteoporosis, and multinodular goiter of 4-years duration was referred by her primary care physician for evaluation. The patient had avoided consuming iodized salt ever since a diagnosis of hypertension 5 years previously and did not eat seafood by choice. The patient had no family history of any thyroid disorder. Physical exam showed bilateral thyroid nodules. Her thyroid function was normal (TSH, 0.97 μU/mL), and her 24-hour urine iodine level was 64 μg (Table 1). The patient was suspected to have iodine deficiency–induced goiter and was instructed to take a multivitamin containing 150 μg of iodine daily. Repeat thyroid sonogram 5 months later showed that the thyroid volume decreased by 10.5%, from 59.8 to 53.5 cm^3 (Fig. 1).

On average, the thyroid volume for the 4 patients reported in this series was decreased by 26.1%, from 43.73 (13.65) cm^3 to 33.26 (15.01) cm^3, after iodine treatment (*P* = .0097).

DISCUSSION

Even though iodine deficiency is not a major public health problem in the United States, our study shows that it is found among minority women in central New Jersey. For most individuals, the thyroid gland adapts to low iodine intake by marked modification of its activity if the intake falls to below 100 μg/day (11). Goiters are initially diffuse but eventually become nodular because the cells in some thyroid follicles proliferate faster than others (11), as shown in the 4 women in our study. Our data suggest that minority women in the United States are prone to develop iodine deficiency–induced goiter, consistent with a previous report indicating that non-Hispanic black women of reproductive age have a lower urinary iodine level (35.5 ± 10% with urinary concentration <100 μg/L) than non-Hispanic whites and Mexican Americans (12,13).

The National Academy of Sciences recommends a daily iodine intake of 150 μg for adults, 220 μg for pregnant women, and 290 μg for lactating women to ensure normal thyroid function (14). Major dietary sources of iodine in the U.S. are iodized salt, bread, milk, and iodine supplements (1). Based on National Health and Nutrition Examination Surveys, the median urine iodine concentration in adults in the U.S. decreased by >50% between the early 1970s and the late 1990s (15). Of particular concern, the prevalence of urine iodine concentrations <50 μg/L among women of childbearing age increased by almost 4-fold, from 4 to 15% over this period. Our study shows that iodine deficiency still exists in New Jersey and that this may be due

to several factors. First, the avoidance of iodized salt, seafood, and dairy foods and other iodine-supplemented foods (commercially processed food) due to seafood allergy, hypertension, and cultural or economic reasons. It has been reported that millions of American are reducing salt consumption to protect against hypertension and coronary artery disease, and there is potential for decreased in population iodine intakes as sodium intake decreases (16). In addition, all 4 patients in this report avoided dairy foods, the most important source of iodine in the United States, possibly due to culture or other unknown reasons. Second, recent pregnancy and breast-feeding can quickly deplete iodine stores. Healthy women maintain iodine stores of 15 to 20 mg in the thyroid. During pregnancy, in order to help meet the approximately 50% increase in maternal iodine requirements, women may draw on this significant iodine store (17). Third, our patients also had strong family histories of thyroid disease. It is possible that in genetically susceptible patients, mild iodine deficiency may lead to the development of thyroid goiter.

Because our cases of iodine deficiency occurred in an iodine-abundant environment, they highlight the importance of obtaining specific dietary information (including information regarding vegan [18] and dairy foods) during the routine evaluation of some subgroups of patients with goiter (19). Our data indicate that iodine deficiency–induced goiter occurs in subgroups of women in the United States, including individuals of Hispanic, African, and Asian ethnicities and pregnant and lactating women, particularly when those patients are also allergic to seafood or have hypertension.

Once a patient is suspected to have iodine deficiency–induced goiter, the diagnosis should be evaluated using a combination of physical exam, laboratory studies, and possible multiple 24-hour urine iodine measurements (20). If the patient's 24-hour urine iodine levels are <100 μg on multiple measurements (indicating iodine deficiency) and the patient has a goiter on physical exam or by ultrasound, a suggestive diagnosis of iodine deficiency–induced goiter can be made, which can be confirmed by reduction of goiter size after iodine therapy. An increase in radioactive iodine uptake supports the diagnosis of iodine deficiency or insufficiency. The iodine supplement therapy needs regular follow-up for potential deleterious effects of increased iodine intake. Current American Thyroid Association guidelines for the management of thyroid nodules advocate against suppressive levothyroxine therapy for patients with euthyroid nodular goiter. Furthermore, regardless of the patient's iodine status, fine-needle aspiration biopsy is required if nodules meet standard criteria for biopsy (21).

CONCLUSION

Iodine deficiency can be a cause of goiter among minority women in New Jersey. A full dietary history, multiple 24-hour urinary iodine level measurements, and reduction of goiter size after iodine treatment are necessary for the diagnosis, especially in minority patients with seafood allergy or hypertension and avoidance of dairy foods. An elevated thyroid ^{123}I uptake supports the diagnosis. Treatment should include the addition of iodized salt to the diet or an iodine-containing prenatal multivitamin for patients with hypertension who refrain from salt. Dietary iodine supplementation may have an antigoitrogenic effect, leading to the resolution of thyroid goiter. Excessive iodine supplementation is not recommended.

DISCLOSURE

The authors have no multiplicity of interest to disclose.

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