Original Article

EFFECTS OF IODIZED SALT CONSUMPTION ON GOITER PREVALENCE IN ISFAHAN: THE POSSIBLE ROLE OF GOITROGENS

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ABSTRACT

Objective: To evaluate the success of the Iranian Iodine Deficiency Disorders Committee in achieving World Health Organization (WHO) goals for reducing the prevalence of goiter in children by adding iodine to table salt beginning in 1989.

Methods: In 1997, 8,000 male and female 6- to 18-year-old students were selected by cluster sampling in schools of Isfahan. Their thyroids were examined by four endocrinologists, and goiter was staged on the basis of the WHO classification. As an index of iodine consumption, urinary iodine concentrations were measured in 3,000 students.

Results: Goiter was observed in 62% of the students. Of the overall study group, 94% had sufficient iodine consumption (urinary iodine concentration of 10.0 µg/dL or more). Of those students who had sufficient iodine intake, 63.2% had goiter. Of the 6% of students with iodine deficiency, 5% had mild, 0.9% had moderate, and only 0.1% had severe iodine deficiency. Goiter was absent in half of the students with severe iodine deficiency. The prevalence of goiter in 6- to 10-year-old children was 65%.

Conclusion: Despite sufficient iodine intake, the prevalence of goiter is still high in Isfahan City. Apparently, either this high prevalence has no relationship to iodine deficiency and possibly other unknown goitrogens are involved in the pathogenesis of goiter in Isfahan or the period of iodine intake has been too brief to affect thyroid sizes. Inasmuch as goiter prevalence is also high in the 6- to 10-year-old children, who have had iodized salt available for most of their lives, the second option is less probable. Another possibility is an increased rate of autoimmune thyroid diseases (because of iodine repletion) that resemble goiter during their early stages. **(Endocr Pract. 2001;7:95-98)**

Abbreviations:
CI = confidence interval; **IDD** = Iodine Deficiency Disorders; **WHO** = World Health Organization

INTRODUCTION

Endemic goiter is present throughout most of Iran (1). For several years, iodine deficiency has been considered a contributing factor for endemic goiter in this country (1,2). The prevalence of goiter had been estimated as more than 70% in Isfahan Province and as 92% in girls and 85% in boys in its capital (Isfahan City) in 1989 (3). World Health Organization (WHO) programs have been designed to reduce the prevalence of goiter to less than 5 to 10% in 6- to 12-year-old children throughout the world by the year 2000 (4). In 1989 in Iran, the Iodine Deficiency Disorders (IDD) Committee had iodine added to table salt in an attempt to eradicate endemic goiter in 6- to 18-year-old residents of this country. The goals of iodine supplementation were intake of iodine of at least 150 mg daily by each resident by the year 1996 and reduction in prevalence of goiter to less than 10% of the population by the year 2000 (3). This study, which was undertaken in 1997, was designed to evaluate the success of the IDD Committee in achieving WHO goals, 8 years after iodide supplementation was initiated in Isfahan.

METHODS

Isfahan Province, located in the central part of Iran, has a total population of approximately 4,000,000, and its capital city has a total population of approximately 1,000,000. From 400,000 male and female 6- to 18-year-old Isfahani students in 1997, we selected 8,000 (an equal number of boys and girls) by cluster sampling. Thyroid examination and staging were done by four endocrinologists according to the WHO classification (4). For

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evaluation of the iodine deficiency rate, urine samples were collected in 3,000 randomly selected students. Urine samples were sent to the laboratory in less than 1 hour, and their iodine concentrations were measured by the digestion method (5,6). Urinary iodine concentrations of 10.0 μg/dL or more were considered indicative of sufficient iodine intake; from 5.0 to 9.9 μg/dL signified mild iodine deficiency, from 2.0 to 4.9 μg/dL indicated moderate deficiency, and less than 2.0 μg/dL suggested severe deficiency (4).

For determining the relationship between the severity of iodine deficiency and the goiter stage, χ^2 statistical analysis and the Spearman correlation coefficient were used. *P* values less than 0.05 were considered significant. For assessment of the prevalence of goiter in different age-groups and in the study year (1997) versus 8 years earlier (1989), a comparison of proportions in two independent test groups was applied.

RESULTS

The overall prevalence of goiter in all 6- to 18-year-old students was 62% (95% confidence interval [CI], 61 to 62%). The frequency of goiter stages was as follows: Ib = 33%, Ia = 15%, II = 14%, and III = 0.1%. The prevalence of goiter was 68% in girls and 55% in boys. Goiter prevalence in 6- to 10-year-old students was 65%, in 11- to 14-year-old students was 64%, and in 15- to 18-year-old students was 58% (95% CI, 56 to 58%) (Table 1). The prevalence of goiter in 8- to 10-year-old students was 66% (95% CI, 63 to 66%), and the most common goiter stages were Ib = 35%, II = 20%, and Ia = 11%. The prevalence of goiter in the 6- to 12-year-old female and male Isfahani students was 66.7% and 61.7%, respectively. On the basis of urinary iodine concentrations, 94% of students (95% CI, 93 to 95%) had sufficient iodine intake, 5% had mild iodine deficiency, about 1% had moderate deficiency, and 0.1% had severe iodine deficiency. Urinary iodine concentrations were the same in both girls and boys.

Half of the students with severe iodine deficiency, 32% of those with moderate deficiency, and 35% of students with mild iodine deficiency did not have goiter. The prevalence of goiter in students who consumed sufficient iodine—94% of all students—was 63.2%. No significant relationship was found between iodine deficiency and various goiter stages (Table 2).

DISCUSSION

In this study, the prevalence of goiter and the daily iodine intake of 6- to 18-year-old Isfahani students were evaluated in 1997. Attempts of the IDD Committee in providing table salt supplemented with iodine and distributing it to all parts of Iran by quantitative salt control resulted in considerable success (94%) in achieving the necessary daily iodine in students in Isfahan. Only 0.1% of students had severe iodine deficiency. Thus, after 8 years, the effort to increase iodine intake was highly successful (3).

Another goal of the IDD Committee was the reduction of goiter prevalence to less than 5 to 10% by the year 2000 (3,4). Because of the increased rate of goiter in this 1997 study, this goal is not expected to be achieved. The most important finding in this study is the hyperendemic prevalence of goiter despite sufficient iodine intake (Table 1). Although in 94% of 6- to 18-year-old students urinary iodine concentrations were more than 10 μg/dL, the prevalence of goiter was 62%. Among students with urinary iodine concentrations indicative of sufficient iodine intake, 63.2% had goiter of various stages. No statistical relationship was detected between goiter stages and urinary iodine concentrations. In addition, half of the students who had severe iodine deficiency (urinary iodine <2.0 μg/dL) did not have goiter (Table 2). This finding suggests that other goitrogens may have a role in the pathogenesis of goiter in Isfahan.

Another possibility is that some families have only recently used iodized salt and the period of consumption has been too brief to decrease thyroid size. The validity of this explanation is questionable because goiter prevalence in the 8- to 10-year-old students was higher than that in the 15- to 18-year-old students ($P<0.01$). Because iodized salt was produced and distributed for 8 years before the current

Table 1
Prevalence of Goiter
in 6- to 18-Year-Old Students in Isfahan,
Stratified by Age-Group and Goiter Stage

Age-group (yr)	Stage of goiter (%)				
	0	Ia	Ib	II	III
6-10	35	12	35	18	0
11-14	35	12	37	15	1
15-18	42	20	27	10	1

Table 2
Prevalence of Various Goiter Stages and Urinary Iodine Concentrations in 6- to 18-Year-Old Isfahani Students

Urinary iodine concentration (μg/dL)	Stage of goiter (%)				
	0	Ia	Ib	II	III
<2.0	50	0	50	0	0
2.0-4.9	32	4	52	12	0
5.0-9.9	35	10	42	13	0
≥10.0	36	13	36	15	0

study, the 15- to 18-year-old students, who had iodine deficiency during about 10 years of their childhood, would be expected to have a higher frequency of goiter than the 6- to 10-year-old group. One study in 1996 in Italy showed that iodized salt can prevent goiter in children who were born after iodized salt was available for consumption and can prevent the increase in goiter size in older children, but it has been less successful in decreasing goiter size in children who lived the early years of their lives in iodine deficiency (7).

Seven years after initial iodine supplementation of table salt in Tehran, the capital of Iran, the prevalences of stage I and II goiter were 44% and 44% in females and 49% and 33% in males, respectively, while the median urinary iodine concentration was 17.5 μg/dL (8). That study found a high prevalence of goiter despite the adequacy of iodine intake in Tehran. Therefore, other goitrogens, in addition to iodide, seem likely to contribute to the high frequency of goiter in Iran.

In comparison with a study in Isfahan City before the availability of iodized salt, the prevalence of goiter has decreased about 30% (P<0.0001) and its size has also decreased (3). At that time, the prevalences of stage III and II goiters were 7% and 32%, respectively, but 8 years later, they were only 0.1% and 14%, respectively (3). This point suggests that iodine deficiency is an important goitrogen and that the IDD Committee achieved a decrease in goiter size and prevalence by iodine supplementation. Nevertheless, goiter is still hyperendemic; thus, a focus on other goitrogens is necessary. Because iodine sufficiency increases the probability of autoimmune thyroid diseases in a repleted iodine area, perhaps one pathogenesis of goiter in Isfahan is increased autoimmune thyroid diseases that can appear as goiter during their early stages, even in endemic prevalence (9,10).

CONCLUSION

By the availability of iodized salt for consumption, daily iodine intake in Isfahan has been increased and generally seems sufficient. Nevertheless, goiter was still hyperendemic in this city when this study was performed in 1997, and the prevalence of goiter is unlikely to decrease to 5 to 10% with analysis of data through the year 2000. Accordingly, production, distribution, and control of iodized salt should be continued, and consumption of iodized salt should be encouraged in Iran. A search for other goitrogens, such as thiocyanate and perchlorate in trace amounts in water or food, and for selenium deficiency should also be conducted.

For assessment of the hypothesis that perhaps the period of consumption of iodized salt was too brief to decrease the prevalence of goiter, a similar study should be repeated in 5 to 10 years. For evaluation of the possibility of autoimmune thyroid disease as a cause of goiter in Isfahan, serum antithyroid antibody titers in patients with goiters should be measured.

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