SUMMARY

Objective: A follow-up study to determine the prevalence, incidence, and natural course of positive antithyroperoxidase antibodies (TPO-Ab) and antithyroglobulin antibodies (Tg-Ab) in the general population and examine the influences of different levels of iodine intake.

Design: Study conducted in Panshan, Zhangwu, & Huanghua, 3 regions with mildly deficient, more than adequate, and excessive iodine intake, respectively. Of 3,761 unselected subjects enrolled at baseline, 3,018 participated in the 5-year follow-up study. TSH, TPO-Ab, & Tg-Ab were measured.

Results: Among subjects in Panshan, Zhangwu, & Huanghua, the prevalence of positive TPO-Ab was 11.2, 11.8 & 12.0%, respectively, whereas 11.2, 11.2, & 11.3% of subjects were Tg-Ab positive, respectively. In the older population (≥ 45 years), Tg-Ab positive individuals were more frequent in Huanghua than Panshan and Zhangwu (P <0.05). The 5-year cumulative incidence of positive TPO-Ab was 2.1, 3.8, & 2.8% in Panshan, Zhangwu, & Huanghua, respectively, whereas 2.9, 3.6, & 5.1% of subjects were Tg-Ab positive, respectively (P<0.05), corresponding to the increase in iodine intake. Subjects who were TPO-Ab and/or Tg-Ab positive at baseline developed thyroid dysfunction more frequently than those without antibodies (14.4 vs. 3.3%; P <0.01). Their incidence of elevated TSH levels was 1.3, 8.5, and 15.4% in Panshan, Zhangwu, and Huanghua, respectively (P < 0.05).

Conclusions: Subjects who were TPO-Ab and Tg-Ab positive at baseline developed thyroid dysfunction more frequently than antibody-negative subjects. High iodine intake was a risk factor for developing hypothyroidism in antibody-positive subjects. A constant exposure to excessive iodine intake increased the incidence of positive Tg-Ab.

COMMENT

Implementation of iodine fortification in populations where iodine deficiency (ID) prevails raises potential concern of triggering (or aggravating) thyroid autoimmune features, especially when the iodine prophylaxis overcorrects the deficiency. This is precisely the issue discussed in the present article.

In three regions in China, a programme to correct ID was implemented by use of iodized salt in households. In the first area (Panshan), mild ID was present, as attested by the median urinary iodine concentration (UIC) in schoolchildren of 84 µg/L. The second region (Zhangwu) was considered ‘more than adequate’, with a mean UIC of 243 µg/L (clearly too high according to WHO guidelines). Finally in the third region (Huanghua), the mean UIC reached 651 µg/L, a clear-cut indication of major iodine excess.

Present study comprised 3,761 adults (934 men; 2,827 women) and started in 1999. Five years later, 3,018 subjects (80% of the original cohort) underwent follow-up studies. At the beginning of the study among adults aged 45 yrs or more, thyroid antibodies were more prevalent in the
region with iodine excess compared to the other 2 regions (16% vs 10-11%), confirming the correlation between excessive iodine intake and thyroid autoimmunity in older subjects. After 5 years of follow-up, thyroid dysfunction was significantly more frequent in those subjects who were already antibody-positive initially. The rate of subjects with an abnormally elevated serum TSH (>4.8 µU/ml) who tested positive for TPO-Ab and/or Tg-Ab increased from 5.3% in the mildly iodine deficient region, to 14.3% in the more than iodine-adequate region, and finally to 23.4% in the region with iodine excess.

In summary, thyroid failure was more likely to develop among thyroid antibody-positive individuals submitted chronically to environmental iodine excess. Such findings point to the absolute need to monitor closely the degree of salt iodization in areas where ID prophylaxis is introduced and avoid overcorrection of ID that may increase the risk of secondary thyroid disorders. *(Daniel Glinoer, M.D.; Ph.D.)*

**See Figure below**

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**FIG. 3.** Five-year cumulative incidences of abnormal TSH levels in euthyroid subjects that were TPOAb and/or TgAb positive at the baseline study in three cohorts. Panshan, Zhangwu, and Huanghua were areas with mild iodine deficiency, more than adequate iodine intake, and excessive iodine intake, respectively. The cumulative incidence of supranormal TSH increased with greater iodine intake (*P* = 0.010 among the three cohorts). Although the incidence of subnormal TSH decreased with increasing iodine intake, no significant difference was observed among the three cohorts.