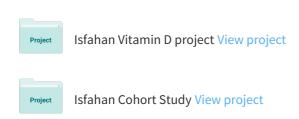
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Residual Goiter in Semirom; Iodine Status and Thiocyanate Overload Do Not Play a Role

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Discussion

It is rare to find research which deals with the incidence of infection by this parasite. Unlike prevalence which describes the status of a disease, incidence studies the intensity of how a disease attacks a studied community. In this study, we found that for every 100 children who live in the studied region, 7.49 are infected after 1 year and that the sorology remains positive for 4 years. All children infected with *T. canis* present with the asymptomatic form as per the Pawlowsky classification [2], but if we consider that in 3 years nearly a quarter of these children will be infected, this may increase the risk that one of these children will present with one of serious forms of infection by this parasite [3].

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Residual Goiter in Semirom; Iodine Status and Thiocyanate Overload Do Not Play a Role

Summary

This study was performed to investigate the role of thiocyanate overload in the etiology of endemic goiter in schoolchildren of Semirom. Iran. A total of 1828 schoolchildren were selected by multi-stage random sampling. Urinary iodine concentration (UIC) and urinary thiocyanate (USCN) were measured in a group of these children. The median UIC was 18.5 µg/dl. The mean \pm SD of USCN in goitrous and nongoitrous subjects did not differ significantly $(0.75 \pm 0.78 \,\text{mg/dl})$ vs. $0.63 \pm 0.40 \,\text{mg/dl}$; p = 0.30). Finally, we concluded that neither iodine deficiency nor thiocyanate overload contributed to the high prevalence of goiter in Semirom. The role of other goitrogenic factors should be investigated in this region.

Key words: goiter, iodine deficiency, goitrogens, thiocyanate, Iran

The relationship between goiter and iodine deficiency is well established. However, a number of genetic and environmental factors also exist, which can contribute to the risk of goiter. Naturally occurring goitrogens are among the environmental contributors to goiter [1, 2]. Thiocyanate (SCN) is one of the best known and most widely studied goitrogens responsible for causation or aggravation of endemic goiter especially in a relatively or severely iodine-deficient region [3]. SCN affects thyroid function depending on its concentration. At low concentrations, stimulation of thyroid function was found [4], whereas a pathologically elevated concentration of SCN acts as a competitive inhibitor of I⁻ transport in the thyrocyte [5].

Semirom is a mountainous region in central area of Iran. Because of the increased goiter prevalence, all citizens of Semirom were given a single dose injection of 480 mg iodized oil intramuscularly in 1993. In 1994, universal mandatory salt iodization program was started in Iran. The present study aimed to determine the role of SCN overload in the etiology of endemic goiter in Semirom schoolchildren.

The methodological protocol of the study was described in detail elsewhere [6]. In summary, 1828 schoolchildren with mean age of 9.33 ± 1.03 years were selected by multi-stage cluster random sampling in 2003. Goiter staging was performed by inspection and palpation. Urinary iodine concentration (UIC) was measured in 132 children by the digestion method based on a modification of Sandell–Kolthoff reaction [7]. Urinary SCN (USCN) was determined in 144 children by the method of

Aldridge as modified by Michajlovskij and Langer [8]. Mann–Whitney U-test was used to compare measurements in different groups. Prevalence of high USCN between goitrous and normal children was compared by chi-square test. Written consent was obtained from all children's parents who were informed about the study. The study was approved by the ethics committee of the Isfahan Endocrine and Metabolism Research Center affiliated to Isfahan University of Medical Sciences.

Overall, 36.7% of subjects were classified as goitrous. Goiter prevalence among girls and boys was 38.6 and 35.0%, respectively (p = 0.06).

The mean \pm SD and median UIC was 19.3 ± 9.1 and $18.5 \,\mu\text{g/dl}$, respectively. Mild $(5 \leq \text{UIC} < 10 \,\mu\text{g/dl})$ and moderate $(2 \leq \text{UIC} < 5 \,\mu\text{g/dl})$ iodine deficiency was detected in 6.4 and 3.2% of cases, respectively. Only 1.8% of children were severely iodine deficient $(\text{UIC} < 2 \,\mu\text{g/dl})$.

Although the mean \pm SD of USCN in goitrous children (N=59) was higher than USCN in nongoitrous ones (N=88), the difference was not statistically significant ($0.75\pm0.78\,\mathrm{mg/dl}$ vs. $0.63\pm0.40\,\mathrm{mg/dl}$; p=0.30). The prevalence of high USCN (USCN > $0.9\,\mathrm{mg/dl}$) in goitrous and nongoitrous children was 25.4 and 18.2%, respectively (p=0.29).

The results of the present study indicate that goiter is still endemic in this region, and there is no biochemical iodine deficiency or no inadequacy in iodine intake in Semirom population. We finally concluded that SCN overload might not contribute to the residual goiter in the studied children. Further investigations are required to find the role of other possible goitrogens in the etiology of goiter in Semirom schoolchildren.

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