WEB OF SCIENTIST: INTERNATIONAL SCIENTIFIC RESEARCH JOURNAL
ISSN: 2776-0979, Volume 5, Issue 01, January, 2024

INFLUENCE OF ENDEMIC GOITTER ON THE COURSE OF PREGNANCY

Kamilova Iroda Abdurasulovna
Doctor of Medical Sciences, Assistant at the Department of Obstetrics and Gynecology in Family Medicine, Tashkent Medical Academy, Tashkent, Uzbekistan

Abstract
Endemic goiter (EG) and its prevention during pregnancy is the most pressing problem in the Republic of Uzbekistan, due to its high prevalence. Natural iodine deficiency, characteristic of local conditions, and the observed defects in systematic iodine prophylaxis maintain the intensity of goiter endemicity in certain regions, especially noticeable in high-risk groups, such as pregnant women and their newborns [1-5].

Keywords: Course of pregnancy, endemic goiter, iodine deficiency, thyroid status.

Introduction:

Endemic goiter refers to iodine deficiency diseases, which are one of the most common non-infectious human pathologies. According to WHO, more than 2 billion inhabitants of the planet Earth live in areas depleted of iodine, about 700 million people have endemic goiter, which allowed WHO experts to call this disease “one of the most common human scourges.” The importance of iodine deficiency diseases is great for many countries, since iodine deficiency, of varying severity, is observed almost throughout the entire country. According to the results of epidemiological studies, the actual iodine consumption by the population averages from 40 to 100 mcg/day, with the norm determined by WHO experts being 150 mcg/day. Diseases caused by iodine deficiency, primarily endemic goiter, are most widespread in foothill and mountainous areas. The spectrum of iodine deficiency diseases is wide and depends on the period of life in which the body experiences iodine deficiency. Iodine is a micronutrient; the daily requirement for it is 100-200 mcg; over the course of a person’s life, they consume about 3-5 g (about a teaspoon) of iodine. Iodine is necessary for the biosynthesis of thyroid hormones - thyroxine (T4) and triiodothyronine (T3); it enters the human body with food, water, and air. Up to 90% of the daily iodine requirement is provided by food, 4-5% by water, and about 4-5% by air. With insufficient intake of iodine into the body, the thyroid gland is exposed to a whole complex of stimulating factors that cause shifts in the synthesis and metabolism of thyroid hormones.

The active uptake of iodine by the thyroid gland increases, the synthesis of triiodothyronine (T3) - a biologically more active hormone that requires less iodine for its synthesis - increases, the excretion of iodide by the kidneys decreases, with the subsequent reuse of endogenous iodine in biosynthetic thyroid hormones.
Currently, the leading role in the pathogenesis of diffuse euthyroid goiter has been established for autocrine growth factors - insulin-like growth factor type 1, epidermal growth factor, fibroblast growth factor, etc., which, under conditions of iodine deficiency, have a pronounced proliferative effect on thyrocytes. In addition, under conditions of iodine deficiency, the sensitivity of thyroid cells to the pituitary thyroid-stimulating hormone (TSH), which stimulates trophic processes in the gland, increases, which is also mediated by autocrine growth factors.

Purpose of the Study:
To assess the impact of iodine prevention and treatment of endemic goiter on the course of pregnancy and perinatal outcomes in women in the region of natural iodine deficiency.

Materials and Methods:
The studies were conducted from 2017 to 2022, in maternity complex No. 6. The effectiveness of individual iodine prophylaxis was assessed during observation of 20 pregnant women without goiter (group 1). The method of prevention consisted of using Yodamarin 200 mg, 1 tablet per day, in the morning after meals, for 3 months with breaks, while using iodized salt. The results were compared with those obtained during observation of 10 pregnant women who received only mass prophylaxis with iodized salt (group 2). The effectiveness of treatment with iodine preparations was assessed in 40 pregnant women with EZ (group 3). All pregnant women with stage I EZ from the first trimester of pregnancy were prescribed the drug “Yodamarin 200”, the dosage of which was 200 mcg per day against the background of daily intake of high-quality iodized salt in the amount of 5-6 g per day, prepared in accordance with new standards at the rate of 45 ± 15 g/day. t, during the entire period of pregnancy and lactation. Pregnant women with stages II–III EZ and diffuse nodose forms of goiter were prescribed combination therapy with Iodide-200 and L-thyroxine at a dose of 50 mcg. Pregnant women who received L-thyroxine 100–150 mcg before pregnancy were also transferred to combination therapy with iodides and L-thyroxine.

The data obtained in the 3rd group of patients were compared with indicators characterizing the general condition, thyroid status, pregnancy and childbirth outcomes of 30 pregnant women with EZ who did not receive treatment with iodine drugs (group 4).

The groups of patients were comparable in age, social status, somatic and obstetric-gynecological history.

Research Methods:

Data from examination, palpation and ultrasound of the thyroid gland (TG), determination of levels of thyroid-stimulating hormone (TSH), free thyroxine (free T4), excretion of iodine in urine (determination of iodine content in urine using the colorimetric method)

Statistical processing of the obtained results was carried out using the statistical analysis program Microsoft Excel 2007.

Results and its Discussion:

In the course of the studies, it was revealed that in the group of pregnant women who received individual prophylaxis (group 1) with iodine preparations ("Iodide-200"), the volume of the thyroid gland during pregnancy varied from 8.9 ml to 13.5 ml, respectively, in the first trimester the average value was 10.05±0.2 ml, in II – 10.84±0.2 ml and in III – 12±0.5 ml, in contrast to the group of pregnant women who received only iodized salt as a method of mass prevention (group 2), where the thyroid volume was 11.17±0.5 ml in the first trimester, 11.10±0.30 ml in the second trimester, and 15.79±0.2 ml in the third trimester. The significance of these differences was high and amounted to $p<0.001$.

Consequently, the thyroid volume in pregnant women under conditions of adequate iodine supply through individual iodine prophylaxis corresponds to a slight physiological increase, in contrast to women who received only iodized salt as mass prophylaxis.

More pronounced values of thyroid volume were noted in the group of pregnant women with developed ES who did not receive prenatal prevention and treatment of this pathology in relation to gestational age. The results obtained by assessing the volume of the thyroid gland in the examined women during pregnancy indicate an increase in the volume of the thyroid gland with increasing gestational age.

Conclusions:

Pregnant women in regions with iodine deficiency need preconceptional, gestational and lactation individual iodine prophylaxis, and, if necessary, differentiated monotherapy with iodine drugs or its combined use with L-thyroxine in the presence of EZ for the health of both the mother and her child.

To improve obstetric and perinatal outcomes in a region with natural iodine deficiency, it is necessary to carry out mandatory iodine prophylaxis and differentiated treatment both in the pregravid period and throughout pregnancy and lactation. Individual iodine prophylaxis should be a mandatory component against the background of population iodine prophylaxis in pregnant women in conditions of iodine deficiency. During pregnancy, the greatest effectiveness in the treatment of EZ was obtained with treatment with iodide drugs in the form of monotherapy, and in the presence of diffuse enlargement of the thyroid gland - combination therapy with iodides and L-thyroxine.

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