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Thyroid diseases

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Abstract: This article analyzes the function of the thyroid gland, types of thyroid gland diseases, shows the causes of thyroid gland diseases, and briefly describes the methods of its treatment.

Key words: Thyroid gland, hypothyroidism, hormones, nervous system, sympathetic and parasympathetic nerve fibers.

Thyroid gland is an endocrine gland of humans and animals. The development of the thyroid gland begins during pregnancy, and when the child reaches the age of 1, its weight is 1-2 g. During growth, it increases to 15-20 g. The thyroid gland develops from the epithelium of the embryonic sac. The thyroid gland is fully formed and secretes hormones in the 8-9 months of human embryo development, it is located in the neck, in the area of the larynx; It consists of 2 panels and a neck. The thyroid gland is supplied with blood by a pair of superior and a pair of inferior arteries, innervated by sympathetic and parasympathetic nerve fibers. It produces the iodine hormone thyroxine (T4), triiodothyronine (TZ) and thyrocalcitonin, which are involved in the regulation of substance and energy metabolism in the body. The function of the thyroid gland is controlled by the central nervous system, and its activity is controlled by the pituitary gland. The thyroid gland. The Thyroid gland is very important in the body, it provides brain activity, metabolism, bone growth, immune systems, physical and mental development, sexual maturation processes, adaptation and other reactions. Dysfunction of this gland leads to goiter, hypothyroidism, hyperthyroidism and other diseases.

The activity of internal secretion glands is important in the formation and development of the fetus and healthy birth. Unfortunately, now endocrine diseases (diseases of internal secretion glands) are widespread and often have a negative effect on pregnancy. Thyroid gland diseases make up a large part of endocrine diseases, and iodine deficiency is the leading one. In areas with moderate and severe iodine deficiency (our country is one of such areas), the consumption of less than normal amounts of iodine leads to the enlargement of the thyroid gland in pregnant women and



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the development of diffuse non-toxic goiter depends on the amount of passing iodine. Lack of enough iodine in the body of the pregnant mother causes iodine deficiency in the body of the fetus. This leads to the appearance of goitre in the fetus later. Due to the occurrence of hypothyroidism in the fetus and in the neonatal period, the child born may be mentally and physically defective (endemic cretinism). For the prevention and treatment of widespread non-toxic goiter in iodinedeficient areas, taking iodine at the rate of 150-200 µg per night is recommended. That is, it is recommended to take one tablet of "Iodomarin-200" or "Iodbalans-200" every day. These drugs usually stop the enlargement of the goiter, and in some cases make it smaller. If a woman has a large goiter before she becomes pregnant, and if the goiter is growing rapidly in the early stages of pregnancy, it is appropriate to take iodine together with thyroid hormones. For this purpose, it is recommended to take 50-100 mcg of L-thyroxine daily and use "Iodomarin-200" or "Yodbalans-200" in one tablet. This procedure allows to restore the normal activity of the thyroid gland in a pregnant woman. In diseases of the thyroid gland, its dysfunction is expressed by the production of thyroid hormones than (hyperthyroidism) less more the norm or (hypothyroidism).Hyperthyroidism is one of the metabolic diseases. Hyperthyroidism is a disease characterized by excessive production of thyroid hormones and uniform enlargement of the thyroid gland. Genetic factors and autoimmune processes are involved in the origin of the disease. The disease occurs more often in women aged 20-50. Physical and mental stress also create conditions for the occurrence of the disease. The main symptoms of the disease (the patient's weight loss, mental restlessness, loss of calmness, nervousness, constant rapid heartbeat, moist and hot skin It is not difficult to make a diagnosis of poison ivy in the cases where there are obvious symptoms such as small tremors in the fingers of the outstretched hand, twitching of the evelids (exophthalmos). diagnosis can be difficult. To confirm the presence of the disease, the amount of thyroid hormones - free thyroxine (er T4) and free triiodothyronine (er T3) and thyroid stimulating hormone (TTG) is determined. The amount of hormones (er T3, er T4) is higher than the norm in a toxic goiter, and the amount of TTG is reduced. One of the most common conditions in determining the hormonal activity of the thyroid gland in pregnant women is the total thyroxine (um T4) and total triiodothyronine (um T3) is to determine the amount in the blood. The increase in the binding properties of proteins in the blood of pregnant women with thyroid hormones increases the amount of um T₃ and um T₄ in the blood of a pregnant woman. However, since the protein-bound forms of thyroid hormones do not have a hormonal effect, an increase in their total amount is not the basis for the presence of thyrotoxicosis in pregnant women. For this, the effective forms of thyroid hormones (i.e. T3 and T4) should be increased.

About 3 percent of women with mild to moderate goiter can become pregnant. When the disease is severe, infertility is observed. Women with metabolic hyperthyroidism find it difficult to conceive, and toxic goiter is more common during pregnancy.



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If not properly treated, the spread of poison ivy worsens the course of pregnancy, especially has a negative effect on the pregnancy process, especially early toxicosis is severe, the probability of miscarriage or premature birth increases, and the risk of thyrotoxicosis crisis increases. Congenital developmental defects (hydro- and microcephaly, hypospadias, cryptorchidism, umbilical hernia, soft palate defect, Down's disease) may occur in the fetus and in the newborn. As a result of the lack of thyroid hormones, hypothyroidism occurs. Most hypothyroidism (90-95%) is primary hypothyroidism and can be caused by thyroiditis, thyroid resection, radioactive iodine treatment, tumor, etc. Pregnancy in hypothyroidism is rare, because the lack of thyroid hormones has a negative effect on the functioning of the internal organs responsible for reproduction. Maturation of primordial follicles slows down in the ovaries, the ovulation process is disturbed, corpus luteum development slows down. Memory loss, loss of interest in surrounding events, weakness, dry and pale yellow skin, weight gain, swelling of the face and eyelids, enlargement of the tongue, slurred speech, menstrual irregularity are the main symptoms of hypothyroidism. Primary hypothyroidism in pregnant women often occurs after surgical removal of chronic autoimmune thyroiditis or diffuse toxic goiter and other goiters. Hypothyroidism in chronic autoimmune thyroiditis is usually diagnosed before pregnancy. But the onset of autoimmune thyroiditis in some cases coincides with pregnancy. In order to detect autoimmune thyroiditis in the early stages of pregnancy, it is recommended to detect autoimmune bodies in the blood of pregnant women who are suspected of having a disorder in the functioning of the thyroid gland.

To confirm the diagnosis of hypothyroidism, the amount of TTG, T3 and free T4 in the blood serum is determined. In primary hypothyroidism, the amount of TTG in blood serum increases above the norm (1-3 μ g/l), T4 and T3 are low. Pregnancy is difficult in women with hypothyroidism. They often have toxicosis of pregnancy, anemia, and miscarriages. Also, the possibility of slow labor movements and bleeding increases. Hypothyroidism has a negative effect on the structure of the fetus and often causes pathological changes in the development of its brain and skeleton. If a woman becomes pregnant during the period of hypothyroidism without good treatment, the possibility of giving birth to children with congenital defects in skeletal development increases

Conclusion:

Hypothyroidism is treated by external administration of deficient thyroid hormones. The patient is prescribed to take 25 μ g of thyroxine once a day, in the morning. The amount of thyroxine is increased every week, and depending on the severity of the disease, it is brought to 150-200 μ g overnight. Thyroxine is given in this amount until the end of pregnancy. After a woman's eye is opened, the amount (dose) of thyroxine is changed depending on her condition. Treatment with thyroxine is continued even during breastfeeding.



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