ASPECTS OF THYROID DEVELOPMENT IN EMBRYOGENESIS

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The thyroid gland (glandula thyroidea) is an unpaired endocrine organ whose function is regulated by the central nervous system and the thyroid-stimulating hormone of the anterior pituitary gland.

The gland is located in the region of the anterior triangle of the neck (trigonum cervicale anterius), bounded from above by the base of the lower jaw, from below by the jugular notch of the sternum, on the sides by the anterior edges of the right and left sternocleidomastoid muscles.

The thyroid gland consists of two lobes (lobus dexter et sinister) and an isthmus (isthmus glandulae thyroidei) between them.

In 30-35% of cases, there is an additional pyramidal lobe, lobus pyramidalis. In an adult, the thyroid gland is adjacent to the larynx and the upper part of the trachea in such a way that the isthmus is located at the level of II-IV tracheal semirings, leaving, as a rule, the first semiring free. The lateral lobes of the gland cover the plates of the thyroid cartilage. Each lobe has an anterior and a posterior surface; top, bottom and outer edges. The outer edges end at the top and bottom poles.

As noted in the literature, the thyroid gland is both the largest endocrine organ in terms of its mass and size, and, perhaps, one of the most significant from the standpoint of evaluating the critical role for the body of hormones secreted by it into the systemic circulation - iodothyronines.

The gland is usually located on the anterior surface of the neck, being fixed to the anterior and lateral surfaces of the trachea and larynx by connective tissue, and consists of two lobes connected by an isthmus. When viewed from the front, it rather has the shape of a butterfly, however, in all European languages \u200b\u200b(German Schilddruse, English Thyroid, etc.) it received the not entirely correct name "thyroid", etymologically dating back to the Greek

word thyreos.

Probably, the iron owes its name to a large extent to the topographic proximity with the thyroid cartilage of the larynx, which, with its outlines, really resembles the Greek "oblong shield". The average weight of a normal thyroid gland ranges from 15-30 g, but the variations in size, shape, and topography are limitless.

During intrauterine development, the complete formation of the histological and cytological structure of the thyroid gland occurs. Moreover, a functionally active thyroid gland is the first of the endocrine glands to appear in the process of embryogenesis.

Physiologists noted the fact that in higher vertebrates and humans, the endocrine cells of the thyroid gland have a dualistic origin in embryogenesis from different areas of the primary endodermal pharynx.

The medial rudiment of the gland, from which the thyrocytes of the follicles are subsequently formed, is formed from the median protrusion of the ventral wall of the pharynx between the first and second pairs of pharyngeal pockets and is closely adjacent to the myoblasts of the heart that are laid down here.

The two lateral rudiments (ultimobranchial bodies) have a more complex origin and, according to modern views, are derivatives of the fourth pair of pharyngeal pouches and the neural crest. The laying of the thyroid gland is formed in the human fetus on the 16-17th day of intrauterine development in the form of an accumulation of endodermal cells at the root of the tongue. The group of cells that gives rise to the thyroid gland grows into the underlying mesenchyme along the pharyngeal intestine to the level of the third or fourth pair of gill pockets and then migrates to the neck area ventral to the cartilages of the larynx.

Contact with the mesenchyme provides a constant inductive interaction of tissues, and in its absence, anaplasia of the thyroid gland occurs. By the end of the 4th week, the thyroid rudiment takes the form of a cavity connected to the pharynx only by a narrow opening at the root of the tongue, known as the thyroid-lingual duct. The germ - the epithelial cord - soon descends to the place of the final localization of the gland and, at the same time, pulls the thyroid-lingual duct along with it.

Then the distal end of the cord bifurcates, and the right and left lobes of the thyroid gland subsequently develop from it, connected by an isthmus. Normally, the proximal end of the epithelial cord (thyroid-lingual duct) atrophies and completely disappears by the 8th week of intrauterine life, and a rudimentary remnant remains in its place - the blind opening of the tongue (foramen caecum linguae). The distal part of the epithelial cord is often (in about 50% of cases) preserved as a pyramidal lobe of the thyroid gland.

At the level of the 4th pair of gill pockets, the rudiment of the thyroid gland meets and merges into a single mass with the paired gill bodies that form here (lateral thyroid or ultimobranchial glands). The latter are formed as caudal protrusions of 4 gill pockets, gradually separate from them and are represented by proliferating cells.

The value of gill bodies in the formation of the thyroid gland is insignificant. They make up only 1/6-1/8 of the mass of its parenchyma. The mass of the gland increases in parallel with the increase in the body weight of the fetus, and its right lobe is significantly larger than the left. At the early stages of development (6-8 weeks), the primordium is a layer of epithelial cells surrounded by a mesenchymal capsule with primitive capillaries. Epithelial cells are cuboidal and rapidly proliferate. At 8 weeks, the mesenchyme grows into the epithelial layer

and divides it into separate fragments.

The rapid growth of the organ occurs due to the growth of both the epithelium and the mesenchyme with numerous blood vessels. Negative reactions to thyroglobulin and thyroperoxidase during this period indicate the absence of a secretory process in the gland.

And also our literary analysis showed that the differentiation of the epithelium is better expressed on the periphery of the lobe. By the 10th - 11th weeks (in fetuses about 7 cm in size), the first signs of secretion appear, which is manifested by the ability to absorb iodine, form a colloid, and synthesize thyroxine.

Single follicles appear under the capsule. In follicles and epithelial strands, type A cells or follicular cells are dominant. Significantly less in the composition of the follicles is laid B-cells. There is an opinion that these types of cells have common stem elements or can transform into each other. Parafollicular (near-follicular) or C-cells grow into the thyroid rudiment from the 5th pair of gill pockets.

By the 12th - 14th weeks of fetal development, the entire right lobe of the thyroid gland acquires a follicular structure, and the left one - 2 weeks later. At this time, the follicles are small, rounded, contain a colloid stained with eosin. Between the follicles are clusters of interfollicular epithelium.

By 16-17 weeks, the fetal thyroid gland is already fully differentiated. At the stage of 18-20 weeks, medium-sized follicles predominate in the gland; separate large follicles appear, containing a dense homogeneous colloid, which stains more intensely than in the early stages of fetal development. The thyroid glands of fetuses aged 21-32 weeks are characterized by high functional activity: there are signs of epithelial exfoliation, colloid resorption; the follicular epithelium in the right lobe reaches its highest dimensions. The activity of the gland increases up to 33-35 weeks.

At 36-40 weeks, there are signs of gland hypofunction. Its parenchyma is represented by large follicles overstretched by a dense homogeneous colloid. The height of the follicular epithelium decreases; thyrocyte nuclei are dark, compact. The interfollicular epithelium is located in small clusters; the connective tissue stroma is moderately developed. Violation of the process of lowering the thyroid gland is the cause of numerous anomalies. The germ of the gland or part of it can stop at any point on the way from the tongue to the level of 2-6 tracheal rings.

If the process of lowering is not stopped in a timely manner, then the thyroid gland can move below the level of its usual location, for example, into the upper mediastinum. Sometimes additional thyroid glands are formed as a result of separation from the main rudiment of any part of the gland. In this case, the position of the accessory glands can be very unusual, for example, in the wall of the heart. These and other positions of the accessory thyroid glands (in the pericardium, goiter) are explained by the close contact of the rudiment of this organ with other organs in the early stages of the development of the human embryo.

Violation of the development of the thyroid gland explains the cause of congenital cysts and fistulas, which are the result of the preservation of the thyroid-lingual duct.

The authors also stated that the anterior pituitary gland (adenohypophysis) is also a derivative of the primary endodermal pharynx (Rathke's pouch) and during embryogenesis is formed parallel to the thyroid gland. At the 5th week of intrauterine development, a communication of Rathke's pouch with a tubular diverticulum of the third ventricle of the brain

is established.

During the 12th week of gestation, communication with the pharyngeal cavity is obliterated due to the development of the sphenoid bone, and the pituitary gland is enclosed in a bone cavity - the Turkish saddle. Between 9 and 12 weeks, most types of pituitary cell elements appear, secretory granules become visible at 10-12 weeks. At the same time, the production of thyroid-stimulating hormone in the pituitary gland is determined by immunohistochemical methods.

From the fibrous capsule covering the thyroid gland, connective tissue partitions extend into the depths of the organ, which form the stroma of the organ, contain vessels and nerves. The division of the parenchyma into lobules is incomplete and the gland is therefore pseudolobular. The structural unit of the thyroid gland is the follicle - a closed vesicle, the wall of which is lined with a single-layer (follicular) epithelium. Parenchyma cells. There are three types of cells: A, B and C. The bulk of the parenchyma cells are thyrocytes (follicular or A-cells).

They line the wall of the follicles, in the cavities of which the colloid is located. Each follicle is surrounded by a dense network of capillaries, into the lumen of which the thyroid hormones thyroxine and triiodothyronine are secreted.

In cells, apical, lateral and basal surfaces are distinguished. The basal surface of the cells is in close contact with the blood capillaries, here in the plasma membrane there are receptors for thyrotropin; on the lateral surfaces of thyrocytes there are girdle closing contacts, on the apical surface of the cells there are many microvilli, in the apical part of the cells there is the Golgi apparatus, different types of vesicles (secretory, bordered, endocytic with immature and mature thyroglobulin), the membrane has receptors for binding immature thyroglobulin, thyroperoxidase.

Thus, summing up the literature review, we can say about the value of studying the anatomy and physiology of the thyroid gland.

Literature

1. Ageikin, V.A. Thyroid dysfunction in newborns and infants born to mothers with thyroid diseases / V.A. Ageikin, R.G. Artamonov // Ros. ped. magazine. - 2000. - No. 5. - S.61-63.

2. Ailamazyan, E.K. Thyroid gland and reproduction / E.K. Aylamazyan, V.V. Potin, I.O. Krikheli et al. // Medical academic journal. - 2008 T. 8, No. 2. - S.22-28

3. Baev, O.R. Prevention of iodine deficiency in pregnant women / O.R. Baev, M.A. Strizhakova // Questions of gynecology, obstetrics and perinatology. - 2004. - V. 3, No. 3. - S. 52-54.

4. Butova, E.A. Perinatal aspects of iodine deficiency states / E.A. Butova, A.A. Golovin, E.A. Kochergina // Obstetrics and Gynecology. - 2004.-№3.-S. 9-12.

5. Byshevsky, A.Sh. Thyroid gland, hemostasis and lipid peroxidation /A.Sh. Byshevsky and others // Transbaikal Medical Bulletin. -2004. - S. 24-28.

6. Dedov, I.I. Endocrinology / I.I. Dedov, G.A. Melnichenko, V.V. Fadeev // M.: "Medicine". - 2000. - 632 p.

7. Kamyshnikov, B.C. Handbook of Clinical Biochemical Laboratory Diagnostics / B.C. Kamyshnikov. - Minsk: Belarus, 2002. - 495 p. 9. Kasatkina, E.P. The role of the thyroid status

of a pregnant woman in the formation of the child's intelligence / E.P. Kasatkina // Gynecology. 2002. - V. 4. - No. 3. - S.132-133.

8. Melnichenko, G.A. Features of the functioning of the thyroid gland during pregnancy / G.A. Melnichenko, S.V. Lesnikova // Gynecology. 2003. - No. 1 (2). - From 14-17.

9. Perminova, S.G. Reproductive function of women with thyroid pathology / S.G. Perminova, V.V. Fadeev, I.E. Korneeva and others // Probl. Reproductions. - 2006, - No. 1. pp.70-77

10. Sandukhadze, I.N. Pregnancy and childbirth in women with hypothyroidism / Sandukhadze I.N. // midwife. gin. - 2005. No. 5. -p.42-44

11. Poppe, K. Thyroid disease female reproduction / K. Poppe, D. Velkiniers, D. Glinoer // Clinical Endocrinology.-2007. - Vol.66. - P.309-321.

12. Lavado-Autric, R. Early maternal hypothyroxinemia alters histogenesis and cerebral cortex cytoarchitecture of the progeny/ R. Lavado-Autric et all// J. Clin. Invest.
- 2003. - Vol. 111(7). - R. 1073-1082.

13. Shin, D.J. Thyroid hormone regulation and cholesterol metabolism are connected through Sterol Regulatory ElementBinding Protein (SREBP-2)/ D.J.Shin, T.F.Osborne//J. Biol. Chem. - 2003 - Vol. 278. - P. 34114 - 34118.

14. Vanderpump, M.P. Epidemiology and prevention of clinical and subclinical hypothyroidism/ M.P.Vanderpump, W.M. Tunbridge // Thyroid - 2002 -Vol. 12. -P. 839 - 847.

15. Woollett, L.A. Maternal cholesterol in fetal development: transport of cholesterol from the maternal to the fetal circulation/ LA.Woollett // Am. J. Clin. Nutr. 2005. - Vol. 82(6). - P.1155-1161.

16. Зияева, Ш. Т., et al. "Антиоксидантная активность гиполипидемического препарата фирутас." Вестник Казахского Национального медицинского университета 5 (2014): 183-187.

17. Tulyaganov, T. S., et al. "SYNTHESIS AND PHARMACOLOGICAL PROPERTIES OF KOMAROVININE ALKALOID AND ITS TETRAHYDRODERIVATIVE." KHIMIKO-FARMATSEVTICHESKII ZHURNAL 21.3 (1987): 295-297.

18. Каримов, Рустам Наимович, Мадорбек Омонов, and Севара Камилова. "РОЛЬ МУТАЦИИ В ГЕНЕ MTHFR ПОЛИМОРФИЗМА 677 С/Т У БОЛЬНЫХ С СЕРДЕЧНО-СОСУДИСТОЙ НЕДОСТАТОЧНОСТЬЮ." Journal of new century innovations 11.3 (2022): 120-124.