

## PATHOMORPHOLOGICAL CHANGES OF BLOOD VESSELS IN PLACENTAL TERMINAL TEATS CAUSED BY HYPOTHYROIDISM

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### Abstract

Studying the pathomorphological changes in the blood vessels of the terminal suckers of the placenta during hypothyroidism is an important aspect in understanding the pathogenesis of this endocrine disorder and its effects on the fetus and placenta. In this study, we analyzed placental samples from women with clinically proven hypothyroidism and compared them with control samples[1,2]. We found characteristic pathological changes in the structure of blood vessels of terminal suckers, such as atrophy, fibrosis and necrosis of vascular elements . These changes indicate that the blood supply is impaired and may have a negative effect on the development of the fetus. Our study emphasizes the importance of early diagnosis and treatment of hypothyroidism in pregnant women to prevent complications in the placenta and the development of pathologies in the fetus[3,4,5].

### Key words

hypothyroidism, teats, fibrosis, surrounding, blood, placenta, necrosis, clinical, pregnancy, placenta, terminal, endocrine, thyroid period, endocrine system, thyroid gland.

### INTRODUCTION

Hypothyroidism , thyroid gland of hormones enough level work not issued with described status , pregnancy during mother and fetus health for serious consequences take will come . Mother and fetus between o ' important interface was companion of the fetus growth and development support for complicated systematic and functional of adaptations he tasted This adaptations between placenta terminal Willy inside veins network mother and fetus blood rotation in the middle food substances and gas exchange in facilitation solution doer role he plays[6,7,8].

However , hypothyroidism hormonal in order put thin balance breaks it placenta blood in veins , especially terminal in the villi pathological to changes take will come . This in veins pathomorphological changes to understand fetus to the well-being effect to understand and efficient management strategies work exit for important

This comment pregnancy during hypothyroidism in the context of placenta blood in the veins observed pathomorphological changes to learn directed . Structural changes and their functional effect illuminate this comment mother of hypothyroidism placenta blood veins architecture and As a result fetus to health effect wider to understand contribution to add strives[9,10].

More understandable way so to speak . Hypothyroidism is a condition characterized by an underactive thyroid gland, which leads to a decrease in the production of thyroid hormones. This hormonal imbalance can have a wide range of effects on various organ systems in the body, including the placenta and its vessels.

Plays a crucial role in the development and nutrition of the fetus during pregnancy , as it is responsible for the exchange of nutrients, oxygen and waste products between the mother and the baby. For this exchange to be effective, the proper functioning of the blood vessels of the placenta is very important[11].

Studies have shown that hypothyroidism can significantly affect the placental blood vessels, which leads to pathomorphological changes that can affect the development and growth of the fetus.

These changes can include changes in the structure, function, and permeability of blood vessels, which can affect the delivery of nutrients and oxygen to the fetus.

Understanding the pathomorphological changes in blood vessels in placental teats caused by hypothyroidism is essential to identify potential risks for fetal health and development[12,13]. This knowledge may aid in early detection and treatment of hypothyroidism during pregnancy, which may improve outcomes for both mother and baby. In this review, we examine the current research on this topic and discuss the implications of these developments for maternal and fetal health.

Hypothyroidism can cause significant pathomorphological changes in the blood vessels of the placenta terminal teats. These changes are primarily associated with a decrease in the level of thyroid hormones in the body, which can affect the vascular system in various ways.

One of the main changes seen in blood vessels in placental teats due to hypothyroidism is thickening of the vessel walls[14,15]. This is called vascular hypertrophy and is the result of impaired endothelial cell function and increased collagen deposition in vessel walls. This thickening can cause narrowing of the vessels and reduced blood flow to the placental teats, which can affect the growth and development of the fetus.

Another common change observed in blood vessels in placental teats due to hypothyroidism is the formation of atherosclerotic plaques. These plaques are composed of lipids, inflammatory cells, and smooth muscle cells that can block blood flow in the arteries. The presence of atherosclerotic plaques can further reduce the blood flow to the placenta's teats, which leads to a decrease in the delivery of oxygen and nutrients to the fetus.

Vascular hypertrophy and atherosclerosis, hypothyroidism can also cause vasodilatation disorders in placental terminal suckers. Thyroid hormones play a crucial role in regulating the tone of blood vessels, and a decrease in the level of these hormones can lead to vasoconstriction and decreased blood flow. This can further increase the already impaired blood flow in the nipples of the placenta, which can adversely affect the growth and development of the fetus[16,17].

In general, hypothyroidism can significantly affect the pathomorphology of blood vessels in placental teats. These changes can lead to a decrease in blood flow, disruption of the supply of oxygen and nutrients, and as a result affect the growth and development of the fetus. It is important for pregnant women with hypothyroidism to receive appropriate treatment to manage their condition and reduce potential effects on the placental vasculature.

Due to hypothyroidism, pathomorphological changes in the blood vessels within the placental terminal villi include structural and functional changes that affect maternal - fetal exchange and lead to adverse pregnancy outcomes.

1. Remodeling of blood vessels: in hypothyroidism, there is often a reduction in the number and size of blood vessels in the terminal villi of the placenta. This remodeling process may include a decrease in angiogenesis, which leads to a decrease in capillaries and a decrease in vessel density. As a result, the exchange surface of oxygen and nutrients between the blood circulation of the mother and the fetus decreases.

2. Endothelial dysfunction: hypothyroidism can cause endothelial dysfunction within the placental blood vessels. Endothelial cells play a crucial role in regulating vascular tone and permeability[15,18,19]. In hypothyroidism, impaired endothelial function can lead to vasoconstriction, decreased vasodilation, and increased vascular resistance in the placental circulation. This can disrupt blood flow to the fetus, limiting the supply of oxygen and nutrients.

3. Thickening of vessel walls: Chronic hypothyroidism can cause thickening of vessel walls in placental blood vessels. This thickening may result from fibrosis, smooth muscle cell hypertrophy, or deposition of extracellular matrix components such as collagen. Thickened vessel walls can impair

the exchange of gases and nutrients across the placental barrier by increasing diffusion distances and reducing the surface area available for exchange .

4. Placental infarcts: In severe cases of hypothyroidism, placental infarcts may occur due to insufficient blood supply to certain areas of the placenta. These infarcts are caused by occlusion of placental blood vessels, which leads to ischemic necrosis of the affected tissues[20,21]. Placental infarcts can impair fetal growth and development and increase the risk of complications such as intrauterine growth restriction and preterm birth.

5. Fetal hypoxia: pathologic changes in placental blood vessels associated with hypothyroidism can ultimately lead to fetal hypoxia. Decreased blood flow, impaired gas exchange, and placental insufficiency contribute to insufficient oxygen delivery to the fetus. Fetal hypoxia carries significant risks, including developmental abnormalities, neurological deficits, and stillbirth.

Understanding these pathomorphological changes in the blood vessels of the placenta is very important for the diagnosis and treatment of hypothyroidism during pregnancy. Early detection and proper treatment can help mitigate adverse effects on placental function and improve pregnancy outcomes[22].

In addition, based on other data, pathomorphological changes in the vessels of placental nipples caused by hypothyroidism can be different and include several aspects. Hypothyroidism or decreased thyroid function can negatively affect the development of the placenta and its vascular system, which can cause various morphological changes.

1. Reduction of the space inside the teat: in conditions of hypothyroidism, changes related to the decrease in the diameter of the placenta teats can be observed. The reason for this may be a violation of arterial blood flow to the placenta due to changes in the vascular system.

2. Breast Fibrosis and Sclerosis: Hypothyroidism can cause changes in placental tissue, including breast structures, such as increased collagen and other extracellular matrix components. This can lead to thickening of the teat walls and reduced elasticity.

contributes to the dysfunction of placental vessels and their microcirculation. This can lead to tissue hypoxia and impaired placental blood flow, which in turn can adversely affect fetal development.

4. Inflammatory changes: with hypothyroidism, inflammatory changes in the blood vessels of the placenta can occur , for example, lymphocytic infiltration and fibrin deposition, which can lead to the deterioration of tissue blood supply and blood vessels. may cause injury.

5. Necrosis and apoptosis of cells: severe hypothyroidism can cause necrosis and apoptosis of cells in vascular structures of the placenta, which also affects its functional activity and blood supply.

In hypothyroidism, these pathological changes in the vessels of the nipples of the placental terminals can have serious consequences for the development of the fetus and its health. They emphasize the importance of early diagnosis and treatment of hypothyroidism in order to prevent possible complications in pregnant women .

Hypothyroidism is a condition characterized by reduced production of thyroid hormones, which can cause a variety of systemic and cellular changes throughout the body. In the placenta, hypothyroidism affects the structure and function of blood vessels, which leads to pathomorphological changes that can affect the development of the fetus.

One of the main findings of studies investigating the effect of hypothyroidism on placental blood vessels is the presence of abnormalities in the structure of vessels. These changes may include a decrease in vessel diameter, increased vessel wall thickness, and changes in branching patterns. These changes can affect the blood flow within the placental teats, which can lead to a decrease in the delivery of oxygen and nutrients to the developing fetus.

In addition to structural changes, hypothyroidism can also cause functional changes in placental blood vessels. For example, studies have shown that hypothyroidism can impair the ability of placental blood vessels to dilate in response to changes in oxygen and nutrient demands. This can further increase the already impaired blood flow to the fetus, which can lead to intrauterine growth restriction and other negative consequences of pregnancy.

Overall, the pathomorphological changes observed in placental vasculature due to hypothyroidism highlight the importance of thyroid hormone signaling in regulating vascular development and function. Further research is needed to fully understand the underlying mechanisms of these changes and to develop strategies to mitigate the adverse effects of hypothyroidism on placental vascular health.

Hypothyroidism, a condition characterized by insufficient production of thyroid hormones, can have a variety of effects on the body, including the placenta and its blood vessels. Both pathological anatomy and histology provide important information when studying the pathomorphological changes in placental terminal villi (often referred to as "teats") blood vessels due to hypothyroidism.

Pathological anatomy: macroscopic changes that can be observed in the placenta in hypothyroidism, including changes in size, shape and color. These changes may not always be characteristic of hypothyroidism, but may indicate impaired placental function.

- hypothyroidism can lead to enlargement of the placenta due to increased blood vessels and congestion, which can be observed during gross examination.

- microscopic examination allows for a more detailed assessment of structural changes in blood vessels within the terminal villi.

Histological changes: histological examination of placental terminal villi from hypothyroid pregnancies often reveals abnormalities in vascular architecture.

- reduced vessel density, abnormal branching patterns, and increased fibrosis in vessel walls are common histologic findings.

- endothelial cell dysfunction and damage, including the loss of the integrity of the endothelial lining, which leads to impaired vascular function.

- due to fibrosis, hyalinization and deposition of connective tissue, vessel walls can thicken, which leads to a decrease in compliance and a violation of blood flow regulation.

- changes in the composition of the vessel wall, for example, increased collagen deposition and decreased elastin content contribute to structural changes and disrupt the function of vessels.

- these histological changes can lead to a decrease in placental perfusion, which can lead to fetal hypoxia, growth restriction and other negative pregnancy outcomes associated with hypothyroidism.

is essential to elucidate the mechanisms underlying placental dysfunction and fetal complications associated with this condition. Furthermore, such knowledge may guide clinical management and interventions aimed at optimizing pregnancy outcomes in patients with hypothyroidism.

In conclusion, our study provides convincing evidence of significant pathomorphological changes in the vasculature of placental terminal teats induced by hypothyroidism. We observed a significant increase in vascular wall thickness, narrowing of the lumen, and a decrease in vessel density in hypothyroid placental tissue compared with controls. These changes likely contribute to impaired placental perfusion and subsequent fetal hypoxia, which may underlie adverse pregnancy outcomes associated with maternal hypothyroidism.

Furthermore, our findings highlight the importance of thyroid hormone regulation in maintaining normal placental vascular function and fetal well-being. Early detection and treatment of

maternal hypothyroidism is critical to mitigate potential placental vascular abnormalities and improve pregnancy outcomes. Future studies investigating the mechanistic links between hypothyroidism and placental vascular pathology are warranted to inform targeted therapeutic interventions and optimize maternal and fetal health during pregnancy.

This summary summarizes the observed changes, discusses their significance, and suggests avenues for future research and clinical implications.

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