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METABOLIC SHIFTS AS AN INDICATOR OF POST-HYPOXIC COMPLICATIONS IN NEWBORN

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Annotation
From this article we see what is metabolic shifts as an indicator of post-hypoxic complications in newborn and an associated, related in meaning to the noun metabolism; related to the metabolism and energy, to the processes of chemical transformations of substances and energy in the body

Keywords: hyperleptinemia, gestational complications, leptin, post-hypoxic.

Introduction

Maternal and child health protection is a priority of the socio-economic policy of the Republic of Uzbekistan. One of the urgent problems of perinatology in such conditions is the adequate assessment of the condition of the fetus during pregnancy and childbirth. During childbirth, the baby experiences increased hypoxia during contractions, with great physical force ("painful stress") as it passes through the birth canal. After birth, the child's living conditions change radically, he immediately finds himself in a completely different environment ("environmental-psycho-physiological stress"), where the temperature is much lower than in intrauterine ("temperature stress"), gravity ("gravitational stress"), mass visual tactile, sound, vestibular and other stimuli ("sensory stress"), other types of respiration ("oxidative stress"), and nutrient uptake ("food stress") appears. is accompanied by changes in almost all functional systems of the body.

Metabolic disease is a condition in which normal metabolic processes are disrupted, most often due to the absence or insufficiency of a particular enzyme.

Hypoxia occurs when a baby receives inadequate oxygen to its brain before, during, or after delivery. The condition can lead to brain injury and, if improperly treated, may progress into a permanent disorder, such as cerebral palsy, cognitive deficiencies, or hypoxic-ischemic encephalopathy (HIE).

Pathologies, also known as congenital metabolic disorders, are diseases that are caused by a genetic change in a protein or enzyme, as a result of which a certain metabolic process is blocked. This blockage affects the normal functioning of certain cells and organs and is manifested by a number of symptoms that are different for

each patient. Among these symptoms, different types of neurological syndromes can occur. This group of pathologies is very extensive, but it can be systematized using the current classification, which is currently undergoing significant changes due to the fact that today we have much greater knowledge about the basic mechanisms of development of such pathologies. Below are the main groups of pathologies, compiled on the basis of the type of damage to the body in each of them. They affect the intermediate metabolism. These include aminoacidopathy (phenylketonuria and propionic aciduria). It also includes disorders in the metabolism of carbohydrates or neurotransmitters and neuromodulators. Metabolism is a set of chemical reactions that occur in a living organism to sustain life. These processes that allow the body to live and develop are called metabolism. There are about a hundred different diseases associated with hereditary metabolic disorders. Untimely detection of such diseases leads to severe disability or death.

Adipose tissue, being an endocrine organ, synthesizes a large number of biologically active substances, adipocytokines, which have both local and systemic effects, affecting the vascular wall, tissue sensitivity to insulin, glucose metabolism, and systemic inflammation. The data of clinical and experimental studies prove a close relationship between the imbalance of adipocytokines and pregnancy complications such as insulin resistance, gestational diabetes mellitus, and preeclampsia. In this regard, the close attention of obstetricians-gynecologists and endocrinologists is directed to the etiopathogenetic aspects of the formation of gestational complications in metabolic disorders caused by an imbalance of adipocytokines in maternal obesity, and the search for markers of these disorders. The review presents current literature data on adipose tissue hormones and their influence on the course of the gestational process.

Leptin (from the Greek "leptos" - "thin"), a peptide hormone that regulates energy metabolism, was identified in 1994. Leptin production is encoded by the obesity gene (ob gene). The gene encoding the leptin receptor is called the diabetes gene (db gene). The interaction of leptin with specific receptors located in the hypothalamic region activates the production of nerve impulses directed to the areas of the brain responsible for the regulation of appetite. Counts,

that it acts on the hypothalamus, blocking the synthesis and release of neuropeptide Y, which causes hunger. During pregnancy, the placenta is an additional source of leptin and leptin receptors. In terms of its biological properties and structure, placental leptin is identical to adipose tissue leptin. A positive correlation of serum leptin levels with body mass index (BMI) was shown both in the absence of pregnancy and in pregnant women. During pregnancy, an important role of leptin is in the

regulation of maternal energy metabolism. The placenta produces a large amount of leptin mRNA throughout the entire gestation period. Obese pregnant women have elevated levels leptin can lead to metabolic disorders caused by dysfunction of adipocytes in adipose tissue. The concentration of leptin in the serum of pregnant women is proportional to weight gain from the very beginning of pregnancy. A significant increase in the concentration of leptin in the early stages of pregnancy and a decrease to the pregestational level in the early postpartum period were shown. An increase in the concentration of leptin simultaneously with an increase in the duration of pregnancy, both in women with obesity and with a normal BMI, is more pronounced than its increase corresponding to an increase in body weight. At the same time, if a pregnant woman is overweight and obese, the concentration of serum leptin is significantly higher than in normal weight. It is assumed that such changes contribute to the mobilization of maternal fat depots to ensure the availability of substrates necessary for fetal growth. The sharp decline in leptin levels after childbirth may reflect the energy costs of the lactation process. Leptin receptors are found in the integumentary and glandular epithelium of the endometrium; their interaction with leptin activates the proliferation of stromal and endometrial epithelium cells, which ensures successful implantation of the egg. During pregnancy, the fetal membranes and uterine tissues are an additional source of leptin production, which contributes to a decrease in the contractile activity of the smooth muscles of the uterus and indicates the participation of leptin in the regulation of the implantation process. This is confirmed by studies showing that leptin production in the placenta is regulated by 17β-estradiol, which, in turn, plays a key role in the process of blastocyst implantation, in the differentiation and invasion of the trophoblast, in the regulation of the growth of the uterine vasculature and in the activation of protein kinase signaling pathways. In a culture of luteinized granulosa cells obtained from women participating in the IVF program, an excess concentration of leptin suppressed the expression of estradiol by these cells. It has been shown that in violation of the cyclic fluctuation of the level of leptin due to ovarian dysfunction, the secretion of estradiol in granulosa cells decreases.

Hyperleptinemia and gestational complications

With obesity and an abnormal concentration of leptin in the serum, as a rule, pathological changes in the endometrium, defects in the implantation of a fertilized egg in the uterus and early embryogenesis, as well as habitual miscarriage, are observed. High levels of leptin are associated with the development of oxidative stress, accompanied by the release of proinflammatory cytokines, and the launch of

proangiogenic processes that enhance the activity of vascular endothelial growth factor. In in vitro studies, leptin promotes trophoblast invasion by modulating various trophoblast growth factors, including IL-1 and 17β-estradiol. When studying the effect of leptin on angiogenesis, its role in the formation of new placental vessels was shown. Leptin receptors were found in the endothelial cells of the blood vessels of the chorionic villus in the first trimester of pregnancy, which indicates the protective role of leptin in relation to the development and compensation of gestational complications.

Since diseases associated with metabolic disorders are hereditary, this means that they are transmitted to the child from the parents. The father and mother of an infant may be healthy but carry defective genes. If a child inherits such an affected gene from each of the parents, this will lead to the development of the disease. Very often, the birth of a sick child with hereditary metabolic disorders may be the first case of a hereditary disease in the family. At the time of birth, a child with such a hereditary disease looks absolutely healthy, and only after a few days or months do the first signs of the disease appear. Many of these diseases are treatable. To avoid complications of the disease and severe consequences, the child must be promptly prescribed treatment. The effectiveness of therapy depends, among other things, on how timely the examination of the infant was carried out and the diagnosis was accurately established.

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