PREDICTION AND PREVENTION OF FETOPLACENTAL INSUFFICIENCY IN THREATENING PREMATURE BIRTH

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Annotation: The insufficiency of the feto-placental system to date remains one of the pressing problems of modern obstetrics. In the structure of perinatal morbidity and mortality, a significant proportion belongs to complications induced by acute fetoplacental insufficiency. These include fetal delay syndrome, acute and chronic fetal hypoxia, premature aging of the placenta. Early fetoplacental insufficiency, which occurs with disorders of the placentation process and leads to immaturity of the placenta, is one of the causes of premature birth. Our study of the causes of this problem will reduce obstetric and perinatal problems.

Relevance: the frequency of pathology is directly proportional to the age of pregnant women: if on average in the population it reaches 20-30%, then in the older age group the violation is much wider [1.3].

All causes of fetoplacental insufficiency can be divided into three groups: placental formation disorders, pregnancy complications and extragenital pathologies (EGP).

As you know, the main manifestations of placental insufficiency are hypoxia and intrauterine growth retardation of the fetus, which lead to developmental disorders, accompanied by a decrease in adaptation in the early neonatal period [5.7]

Therapy of placental insufficiency is a difficult task. Taking into account the variety of factors leading to the development of FPN, the therapy of this complication should be complex and pathogenetic orientation: 1) improvement of uterine-placental blood flow, microcirculation, rheological and coagulation properties of blood; 2) normalization of gas exchange between the mother and fetus; 3) improvement of the metabolic function of the placenta; 4) restoration of impaired functions of cell membranes[9, 12]

Therefore, at present, it is relevant to develop and introduce into clinical practice modern diagnostic methods that allow to detect pathological changes in the fetoplacental complex at the early, preclinical stage of the disease. Of undoubted interest is the concept of the paramount importance of neurogenic regulation of protective and adaptive mechanisms that provide homeostasis in the mother-placentafetal system.

Goal: To reduce the number of premature births by developing optimal methods of prediction and prevention of fetoplacental insufficiency.

The implantation process is considered inflammatory, during which cytokines, chemokines and prostaglandins are produced, and leukocytes are infiltrated into the uterus [20.25]. Approximately during implantation, inflammation is inhibited and a tolerogenic environment is created. Numerous mechanisms are involved in the induction of such an environment, including the release of anti-inflammatory molecules such as TGFβ [8,15] and the induction of specialized anti-inflammatory T-cells known as CD4+FOXP3+ regulatory T-cells (Treg), which inhibit the anti-

inflammatory immune responses of the fetus [9,11]. Tolerogenic dendritic cells (DCs), uniquely present in the decidum, cross-present fetal antigens to maternal CD4+ T-cells, thereby also activating the generation of Treg cells [15,17]. Treg cells interact with DCs and macrophages, changing their phenotypic states so that they are proto-tolerogenic [11, 14]. It is important to note that Treg cells reduce the degree of anti-fetal immune reactions, limit the influence of T-effector cells (Teff) and maintain energy in the T-cell population (Tcon), which are otherwise able to differentiate in Teff [8,13]. Placenta cells also contribute to the creation of a tolerogenic environment, with the help of a colony of stimulating factor (CSF)1 secreted from trophoblasts and interleukin (IL)10, which induces decidual macrophages with the regulatory phenotype "M2", expands the functionally suppressing cells CD4+FOXP3+ Treg., and limits the activation of T-helper (Th) 1-, Th17- and Th2-cytokine-producing cells Teff [12,20].

In normal pregnancy, there are significant cross-interference between the maternal and fetal systems due to the joint circulatory system through the placenta. Nutrients and metabolic products are transmitted from mother to fetus, along with cells, signaling molecules, extracellular vesicles (ECVs) and nucleic acids. Cell exchange leads to microchimerism in the mother or fetus, and the maternal cells transmitted to the fetus are called maternal microchimerism (MMC).

The placenta releases exosomes into the maternal bloodstream during pregnancy [16,19]. The functional significance of various ECVs during pregnancy is largely unknown, but it is assumed that they mediate the relationship between the fetus and the mother at key stages of pregnancy, including implantation and childbirth [6, 14]. Trophoblasts and villi secrete exosomes containing placenta-specific microRNAs into the maternal bloodstream, which play a key role in the regulation of immune signaling [19,23]. The exosomes obtained from the mother are also enriched during pregnancy, and the transmission of exosomes between the fetus and the mother is bidirectional [10, 18].

Apoptosis is a natural form of programmable cell death (PCD), along with autophagy, on-goat, mitotic catastrophe, etc. [1, 7]. With regard to reproductive issues, this phenomenon has been studied fragmentarily. Nevertheless, at present there is every reason to talk about the participation of PCD processes in all stages of placenta development, from implantation to rejection.

The role of apoptosis inductors is assigned to ligands from the superfamily of tumor necrosis factor (TNF, Fas-L, TRAIL, TWEAK, LTa, LTp, 4-1BBL, LIGHT) [9, 17], expressed in the human placenta [5, 11]. Fas-L containing cells are able to cause apoptosis of cells on the surface of which there is a corresponding receptor - Fas. Fas-L is also present on the surface of the trophoblast [13,24]. The initiation of apoptosis of T-lymphocytes sensitized to the fetus through FasL-Fas interaction protects it from aggression from the mother's immune system. In a small amount, Fas is also expressed on the surface of the trophoblast itself, but this path of apoptosis activation in it is blocked [4, 15]. This block is not absolute: in the presence of IF and FNor, the sensitivity of the trophoblast to the initiation of apoptosis through the FasL-Fas system increases [21,23].

Trophoblastic expression of Fas-L decreases during labor activity, which may be evidence of the participation of apoptosis processes in placenta rejection [8,15]. Perhaps the FasL-Fas mechanism is involved in the violation of the formation of the placenta in gestosis, since in this pathology excessive expression of Fas-L and Fas was found in decidual tissue [17, 19].

Unlike Fas, TNFA-induced apoptosis in the villi trophoblast is not blocked [20,22]. The biological feasibility of this phenomenon is unclear. It is possible that the restriction of apoptosis in the trophoblast is carried out through other regulatory systems.

The study of cytokine regulation of apoptosis showed that TNF and y-interferon (IFN) synergistically induce apoptosis in trophoblast culture, and the level of expression of the Bcl-2 protein in the cytotrophoblast modulates the degree of apoptosis. TNOa is involved in the activation of placenta apoptosis in intrauterine fetal development delay [12,23]. It has been shown that spontaneous abortions are accompanied by an increase in Fas-L expression in decidual lymphocytes and Fas in extra-villi trophoblast [11.18].

Overexpression Nodal, one of the members of the transforming growth factor-ß superfamily, activates apoptosis and inhibits the proliferative activity of the trophoblast by means of the p27-cyclin E/Cdk2 mechanism [19, 20].Studies on another cytokine, placental growth factor (PlGF), which was first isolated in 1991, have shown that PlGF protects the trophoblast from apoptosis caused by growth factor deficiency, but does not have a similar protective effect against TNOa-induced apoptosis. In contrast, the epidermal growth factor blocks TNOa-induced apoptosis, prevents alcohol induced apoptosis in the placenta [3, 8], but does not protect the trophoblast in case of growth factors deficiency [7.19].

Apoptosis in the placenta is affected by the level of glu-cocorticoids. 11ß-hydroxysteroid dehydrogenase-2 is localized in the placenta, the expression of which increases with an increase in the gestational age in parallel with the increase in apoptosis in the placenta [13,17].

Hepatocyte growth factor inhibits apoptostrophoblast by phosphorylation of serine-threonine protein kinesis (Akt), which leads to inhibition of kinesis glycogensin-thetase (GSK-3beta). This, in turn, causes the activation of the transcription factor ß-catenin and inducible NO; inthase [16, 17].

It has been established that leptin is synthesized in the placenta - a polypeptide hormone that has the features of a long-chain cytokine (like IL-2, IL-12), as evidenced by the high homology of its receptor with representatives of the I class of cytokine receptors [15,21]. The stimulating effect of this cytokine on the proliferation of trophoblast cells has been revealed, as evidenced by the activation of the inclusion of 3H-thymidine, the movement of the cell to the G2/M phase of the cell cycle, the increase in the expression of cyclin D1. In addition, leptin inhibits the processes of apoptosis in tropho-blast [13,14].

In the extravorsintrophoblast, progesterone significantly reduces the activity of apoptosis, reducing the number of TUNEL-positive cells, the expression of Fas, Fas-L, caspaz 3, 8 and PARP (poly(ADP)ribozopolymerase) and increasing the expression of Bcl-2 [18,22]. Endothelin-1 also suppresses apoptosis in the

trophoblast, which probably suggests its protective role in damage to the trophoblast [4.7].

The system of myeloid cell leukemoid factor-1 (Mcl-1) plays an important role in the regulation of PCG in the placenta. This system is represented by the apoptosis inhibitory factor Mcl-1L and the pro-apoptotic component Mcl-1S, as well as Mtd-L and Mtd-P - isoformsMtd/Bok (Matador/ Bcl-2-related ovariankiller) - a member of the Bcl-2 family, which induce the development of mitochondrial-dependent mechanisms of apoptosis [4, 20]. The pro-apoptotic activity of Mcl-1S, Mtd-L molecules is neutralized by its binding to Mcl-1L, which also blocks the apoptogenic protein BIM and other members of the Bak family [21, 22].

Oxygen is a potential regulator of apoptic cell death. Some members of the Bcl-2 family, including the Nix and Nip H3 ligands, as well as Mcl-1 and Mtd, are directly regulated by oxygen through the transcription factor HIF-1 [4,6,13]. Mcl-1L expression is activated when oxygenation decreases, which indicates the activating regulatory effect of HIF-1 [41, p.144;]. In addition, the level of proapoptotic Mcl-1c and Mcl-1S in the placenta increases at 10-13 weeks, when pO2 increases significantly.

Nitric oxide in the placenta not only affects the vascular tone, but also acts as a factor affecting cell apoptosis. It also regulates implantation of blastocyst [15,19], differentiation of trophoblast [17,23], its mobility and invasion [17,22;]. The dominant effect of nitric oxide during pregnancy is the modulation of the formation of new vessels of placental villi [14,18,22;]. It is assumed that nitric oxide by increasing the sensitivity of decidual membrane cells to proliferative stimuli affects the processes of decidualization [16,20;].

In vitro studies have shown that extravorsin trophoblast cells are more susceptible to apoptosis during cultivation, and the addition of NO to the donor medium reduces the sensitivity of extravorsinthrophoblast cells to apoptogenic stimuli in preeclampsia, but activates the NO-induced path of apoptosis in normal cells [14,19].

In the cells of the trophoblast, genes APG9L1 and APG9L2 were found, which carry out post-transcriptional regulation of endothelial NO; intases are necessary for the formation of such a phenomenon as autophagy - one of the types of programmed cell death - the intracellular system of degradation of most proteins and some cell organelles [17,19.23;].

An analogue of nitric oxide is carbon monoxide, formed in the process of hemoxygenase reaction, which is also involved in the regulation of PCG. Carbon oxide protects the trophoblast from apoptosis induced by episodes of hypoxia/reoxygenation. This probably explains the paradoxical fact of the lower prevalence of preeclampsia in smoking pregnant women [16,18,22;].

Activation of apoptosis of trophoblastic cells can be caused by an increase in the production of free radicals, accompanying the disruption of blood supply to the placenta [12.16;]. With inadequate restructuring of the spiral uterine-placental arteries, the blood flow in them is variable, as they continue to respond to vasoconstrictive effects. As a result, there is an alternation of episodes of hypoxia and hyperoxia, which leads to excessive production of free radicals. Activation of

apoptosis in the placenta during hypoxia-reoxygenation is carried out through the OT-kV system, p38, stress-activated PC, mitogen-activated PC [13,18;], accompanied by an increase in the level of TUNEL-positive nuclei, 4-hydroxynonenal, nitrosylation products, activation of caspase-3 and polyADP-ribozopolymerase and prevented by vitamins *C* and E [7,9,15;.], defer-roxamine and superoxide dismutase [17, 19.22;] and low concentrations of glycyrine trinitrate [3,9;].

It has been established that in case of hypoxia in the placenta, a caspase-dependent modification of myeloid cell leukemoid factor-1 (Mcl-1) occurs. In particular, in preeclampsia, the apoptosis inhibitory factor Ms1-^ is broken down by caspases-3 and -7 and the inclusion of apoptogenic Ms1-^. Episodes of hypoxia-reoxygenation in preeclampsia are not only accompanied by cleavage of Ms1-1, but also activate its expression, along with Mtd-L and Mtd-P. In conditions of chronic hypoxia in both normal and pre-eclamptic placenta, the expression of syncytine, a specific protein, a marker of syncytial fusion of trophoblastic cells, decreases, which indicates a decrease in the rate of differentiation of the trophoblast [4, 10.19;].

Caspase-14 [4,8;] was found in the placenta, which is characteristic of the epidermis and plays an important role in the process of keratinization. Features of the functioning of caspases in the placenta are a little-explied issue. Initiator caspases-8 and -10 are activated in the part of the differentiated cytotrophoblast that is intended for syncytial fusion [6]. Effector caspases are expressed in the cytotrophoblast only in inactive form [5.9,14;].

An increase in the content of caspase cleavage products during bladder drift was revealed, which is difficult to explain at this stage, since in other tissues tumor growth is usually associated with apoptosis suppression [9.13;].

The cytotrophoblast, which is preparing for synthialization, initiates the cellular program of apoptosis and at the same time produces a significant number of apoptosis inhibitors, including proteins of the Bcl-2 family [18.20;]. Bcl-2 is a family of proteins that control the process of programmed cell death. They participate in the mito-chondrial regulation of apoptosis.

In the placenta, bcl-2 is expressed in the villi and extravorsin trophoblast, mesenchyma villi, in placental macrophages. Maximum expression was detected in trophoblast cells in the first trimester of pregnancy [10.20;]. The degree of expression is significantly reduced in the placenta after 32 weeks of gestation, which occurs simultaneously with the slowdown in the growth of the placenta. Perhaps this is one of the mechanisms of the so-called "aging" of the placenta [7,18;]. During labor activity, bcl-2 expression does not change [17,21;].

Regulation of a number of caspases is carried out by a flice-like inhibitor protein (Flip). Flip is expressed in the placenta and competes with caspase-8 for binding to death receptors such as TNF-R and Fas [12,16;], thereby reducing the destructive activity of caspase-8 in the villi cytotrophoblast.

In the early stages, apoptosis of the subject endometrial cells makes room for the growing fetal egg [7, 11,19]. It has been found that endometrial stromal cells (ESCs) express Fas, while the cells of the implanted trophoblast secrete Fas-L. However, it has been shown that regardless of hormonal differentiation, ESCs are

primarily resistant to Fas-dependent apoptosis. At the same time, y-interferon and TNF are able to unblock this path, which is accompanied by the activation of caspase-3, -8 and -9 [11,15;].

Later, the endo- and myometria extravorinal tropho-blast migrating through the blood vessels attaches to the vascular wall, causing its transformation. Fibrinoid changes in the wall of the spiral uterine-placental arteries lead to the expansion of these vessels, providing sufficient blood flow to the placenta, regardless of the influence of vasoconstrictive factors [10,14;]. Trophoblast cells, taking part in this extremely complex dosed invasive process, show some functional similarity to malignant cells [5.13;].

There is a hypothesis that assumes the activation of apoptosis of smooth muscle cells (MMC) in the invasion of the intra-vorsinated trophoblast into the muscle layer of the spiral arteries of the uterus, which is a fundamental process in their gestational transformation. A key role in this process is assigned to the apoptotic tokins of the TNF family, in particular, the Fas-Fas-L system, TRAIL and metalloproteinases, in particular MPP-12 [11, 15,18;]. This is evidenced by TRAIL products with both vorinous and extravilli trophoblast [6.15], the expression of TRAIL-R1 and -R2 receptors on the MMC of spiral arteries, the ability of TRAIL to cause apoptosis of GMC, induction of apoptosis of GMC by trophoblast by a TRAIL-dependent mechanism. At the same time, by activating the Akt-, ERK-dependent pathway, TRAIL promotes the proliferation and viability of endothelial cells [18.22;].

IFN is also involved in the apoptotic transformation of spiral arteries, which increases the sensitivity of MMC to Fas-induced apoptosis [4.19;]. A similar permissive effect in IFN is also observed in the case of stromal cells of the endometrium, trophoblast, etc. It has been established that yIFN, one of the main sources of which in the trophoblast is natural killers (NK), stimulates apoptosis and reduces the secretion of metalloproteinases (MMP2) [13,16;], which leads to the suppression of intra-vorsinal-gotrophoblast invasion [17.20;].

Thus, apoptosis is both a mechanism for the transformation of spiral arteries and a mechanism that limits and localizes this process. If for one reason or another the process is not localized due to the apoptosis of the invasing trophoblast cells, then, depending on the degree of the violation, there is a bladder drift or chorioncarcinoma [9, 13;].

On the contrary, early spontaneous abortion, gestosis and fetal delay syndrome have common pathogenetic points associated with insufficient trophoblast invasion [2, 19;]. Incomplete restructuring of the spiral arteries of the uterus causes compensatory reactions in the form of increased apoptosis in the placenta, leading to an increase in the permeability of the fetoplacental barrier to improve fetal nutrition [2; 5, 13;].

Apoptosin vasing trophoblast may be associated with factors that cause its premature differentiation [4, 12;]. It is shown that in gestosis there is excessive macro-phagal infiltration of the decidual membrane. It is suggested that macrophages can affect the invasion of the trophoblast, possibly initiating the

latter's apoptosis. The specific mechanisms of this process are not yet clear, but they seem to be central to the pathogenesis of gestosis [13,19;].

Apoptosis is of great importance in the formation of fetomaterin tolerance. The participation of HLA-G, Fas-FasL, and TRAIL-TRAIL-R in the apoptosis of maternal leukocytes during pregnancy is shown [18, 23;]. The ability of syncytiotrophoblast and placental macrophages to secrete the soluble form of Fas-L involved in the formation of tolerance was discovered [19, 22;]. Also in syncytiotrophoblast and cytotrophoblastexpressed the ligand of programmable cell death (PDL1) [11.18;], which, by activating T-cell apoptosis, limits the expansion of T-lymphocytes, suppresses the production of IFN and contributes to the formation of T-cell tolerance [3, 8;].

It was found that the granule inhibitor B-P1-9 is synthesized in the extra-villi and villi trophoblast, the maximum level of expression of which was recorded in the second trimester. It is believed that this protein allows the trophoblast to block the aggression of natural killers of the maternal body [14.19].

Thus, apoptosis is involved in the formation of fetomaternal tolerance. The cytokins produced by the placenta cause apoptosis in the mother's immunocompetent cells and block their apoptosis-mediated cytotoxicity in relation to trophoblast cells.

Thus: a significant amount of research will allow us to speak with confidence about the important role of the process of programmable cell death in the decisive periods of placenta development. In the process of implantation, thanks to apoptosis, space is released for the blastocyst infiltrating the endometrium. During the invasion of the extravilliar trophoblast, apoptosis plays a leading role in the gestational restructuring of spiral arteries, localization of the invasion. The processes of programmed cell death are involved in the formation of an immunological partnership between the immune systems of the mother and the fetus necessary for pregnancy.

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