MORPHOFUNCTIONAL CHANGES IN DAMAGED PLACENTA DURING PREGNANCY

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Abstract. *Placental integrity plays a decisive role in maintaining normal intrauterine development of the fetus. Structural and functional alterations of the placenta represent one of the most significant causes of chronic fetal hypoxia, intrauterine growth restriction, premature birth, and increased perinatal mortality. Despite advancements in prenatal diagnostics, placental pathology remains insufficiently diagnosed during early stages, leading to delayed therapeutic interventions. This study focuses on the comprehensive evaluation of morphofunctional changes in damaged placentas obtained from complicated pregnancies. Histological, morphometric, and immunohistochemical investigations revealed pronounced degenerative and ischemic processes within placental villi, including stromal fibrosis, fibrinoid necrosis, endothelial swelling, trophoblastic thinning, and increased apoptotic activity. These pathological alterations significantly impair transplacental oxygen and nutrient diffusion, leading to progressive fetal compromise. Furthermore, a marked decrease in angiogenic factors and microvascular density was observed, reflecting placental vascular insufficiency. The severity of morphological changes correlated with adverse fetal outcomes such as low birth weight, low Apgar scores, and increased need for neonatal intensive care. The findings emphasize the importance of early detection of placental dysfunction through Doppler ultrasonography and biochemical markers, as well as the need for timely preventive and therapeutic strategies aimed at preserving placental perfusion and reducing fetal morbidity and mortality.*

Key words: *Placental insufficiency, morphofunctional changes, chronic fetal hypoxia, villous fibrosis, trophoblastic apoptosis, impaired fetoplacental circulation, intrauterine growth restriction, angiogenesis deficiency.*

Annotatsiya. *Plasenta homila rivojlanishini ta'minlovchi asosiy vaqtinchalik organ bo'lib, uning strukturaviy va funksional yaxlitligi homilaning sog'lom*

rivojlanishi uchun hal qiluvchi ahamiyatga ega. Plasentaning zararlanishi surunkali homila gipoksiyasi, intrauterin rivojlanishning sekinlashuvi, muddatidan oldin tug‘ilish va perinatal o‘lim ko‘rsatkichlarining oshishiga olib keluvchi muhim patologik omillardan biridir. Zamonaviy prenatal diagnostika usullariga qaramay, plasenta patologiyalari ko‘pincha kech aniqlanadi va bu o‘z vaqtida davolash choralarini ko‘rishga to‘sqinlik qiladi. Mazkur tadqiqot zararlangan plasentalarda yuzaga keladigan morfologik va funksional o‘zgarishlarni har tomonlama baholashga qaratilgan. Gistologik, morfometrik va immunogistokimyoviy tahlillar villus stromasining fibrozlashuvi, fibrinoid nekroz, endoteliy shishishi, trofoblast qavatining yupqalashuvi hamda apoptoz jarayonlarining kuchayganini aniqladi. Ushbu patologik jarayonlar transplasentar kislorod va oziqa moddalari almashinuvini sezilarli darajada buzadi va homila yetishmovchiligiga olib keladi. Bundan tashqari, angiogen omillar miqdorining kamayishi va mikroqon tomirlar zichligining pasayishi plasentar qon aylanishining yetishmovchiligini ko‘rsatadi. Aniqlangan o‘zgarishlarning og‘irlik darajasi homilaning kam vazn bilan tug‘ilishi, Apgar ko‘rsatkichlarining pastligi va yangi tug‘ilgan chaqaloqlarni reanimatsiya bo‘limiga yotqizish ehtiyojining ortishi bilan chambarchas bog‘liq. Tadqiqot natijalari plasentar disfunksiyani Doppler ultratovush va biokimyoviy markerlar yordamida erta aniqlash, shuningdek, homila morbidligi va mortalitetini kamaytirishga qaratilgan profilaktik va davolovchi choralarni vaqtida qo‘llash zarurligini ta’kidlaydi.

***Kalit so‘zlar**: Plasentar yetishmovchilik, morfofunksional o‘zgarishlar, surunkali homila gipoksiyasi, villus fibrozlanishi, trofoblast apoptozi, fetoplasentar qon aylanishi buzilishi, intrauterin rivojlanishning sekinlashuvi, angiogenez yetishmovchiligi.*

***Аннотация.** Плацента является ключевым временным органом, обеспечивающим нормальное внутриутробное развитие плода, и её структурная и функциональная целостность имеет решающее значение для благоприятного исхода беременности. Повреждение плаценты относится к числу ведущих причин хронической гипоксии плода, задержки внутриутробного развития, преждевременных родов и повышения показателей перинатальной смертности. Несмотря на развитие современных методов пренатальной диагностики, плацентарная патология нередко выявляется на поздних стадиях, что ограничивает возможности своевременного лечения. Настоящее исследование направлено на комплексную оценку морфофункциональных изменений в повреждённых плацентах при осложнённой беременности. Гистологические, морфометрические и иммуногистохимические исследования выявили выраженные дегенеративно-ишемические изменения ворсин хориона,*

включая фиброз стромы, фибриноидный некроз, отёк эндотелия, истончение трофобластического слоя и усиление апоптоза. Эти изменения значительно нарушают процессы транс-плацентарного транспорта кислорода и питательных веществ, что приводит к прогрессирующей фетоплацентарной недостаточности. Кроме того, отмечено существенное снижение экспрессии ангиогенных факторов и плотности микрососудистой сети, что отражает плацентарную сосудистую недостаточность. Степень морфологических нарушений коррелировала с неблагоприятными перинатальными исходами, включая низкую массу тела при рождении, снижение оценки по шкале Апгар и увеличение потребности в интенсивной терапии новорождённых. Полученные данные подчёркивают необходимость раннего выявления плацентарной дисфункции с использованием допплерографии и биохимических маркеров, а также внедрения своевременных профилактических и лечебных мероприятий, направленных на снижение перинатальной заболеваемости и смертности.

***Ключевые слова**: Плацентарная недостаточность, морфофункциональные изменения, хроническая гипоксия плода, фиброз ворсин, апоптоз трофобласта, нарушение фетоплацентарного кровообращения, задержка внутриутробного развития, дефицит ангиогенеза.*

INTRODUCTION

The placenta is a unique and highly specialized temporary organ that plays a central role in maintaining normal fetal development throughout pregnancy. Acting as the interface between maternal and fetal circulations, it ensures efficient gas exchange, nutrient and waste transport, endocrine regulation, and immunological protection of the fetus. Proper placental structure and function are therefore essential for sustaining an optimal intrauterine environment and for securing favorable pregnancy outcomes. Any structural or functional impairment of the placenta may result in placental insufficiency, a pathological condition characterized by reduced uteroplacental blood flow and compromised transplacental exchange. This insufficiency often leads to chronic fetal hypoxia, intrauterine growth restriction (IUGR), preterm delivery, and increased perinatal morbidity and mortality. Clinical and experimental evidence indicates that placental damage is a major contributing factor to low birth weight, neurodevelopmental disorders, and long-term metabolic diseases in offspring. Placental injury can be triggered by a wide range of maternal, fetal, and environmental factors, including hypertensive disorders of pregnancy, diabetes mellitus, infectious diseases, autoimmune conditions, smoking, and exposure to environmental pollutants. These factors initiate ischemic, inflammatory, and oxidative stress-related processes

that disrupt trophoblastic integrity, alter villous architecture, and impair placental angiogenesis. As a result, the diffusion capacity of oxygen and nutrients decreases, directly affecting fetal growth and survival. Despite the progress in prenatal screening and imaging technologies, early-stage placental dysfunction remains difficult to diagnose, and its morphological correlates are often detected only after delivery. Therefore, a comprehensive understanding of morphofunctional alterations in damaged placentas is essential for improving diagnostic accuracy, developing targeted preventive strategies, and reducing adverse perinatal outcomes. This study aims to analyze the key morphological and functional changes occurring in damaged placentas and to evaluate their clinical significance in complicated pregnancies.

LITERATURE REVIEW

Placental pathology has been widely studied as one of the central mechanisms underlying adverse pregnancy outcomes. Numerous clinical and experimental investigations have demonstrated that maternal systemic disorders significantly influence placental morphology and function. Among these, hypertensive disorders of pregnancy, gestational diabetes mellitus, chronic infections, autoimmune diseases, and exposure to environmental pollutants are recognized as major contributors to placental ischemic and inflammatory injury. Several authors have emphasized that chronic uteroplacental hypoperfusion results in progressive structural remodeling of placental villi. Smith et al. reported that villous fibrosis, increased deposition of fibrinoid material, and the formation of syncytial knots represent key histopathological hallmarks of chronic placental insufficiency. These changes are associated with reduced capillary density and thickening of the trophoblastic membrane, which directly compromise the efficiency of maternal–fetal exchange. Burton and colleagues highlighted the role of oxidative stress in placental damage, noting that excessive production of reactive oxygen species leads to endothelial dysfunction, trophoblastic apoptosis, and impaired angiogenesis. Similarly, Redline demonstrated that inflammatory-mediated placental lesions, including villitis and intervillositis, are closely related to fetal growth restriction and increased perinatal mortality. Recent studies have also explored the impact of environmental toxins such as heavy metals, particulate air pollutants, and cigarette smoke on placental development. These agents induce microvascular injury, alter cytokine expression, and disrupt placental endocrine activity, further aggravating fetoplacental insufficiency. Moreover, alterations in angiogenic and anti-angiogenic factor balance, including decreased vascular endothelial growth factor (VEGF) expression, have been implicated in the progression of placental dysfunction.

Although substantial data exist regarding the morphological features of damaged placentas, many studies focus on isolated pathological conditions, leaving a need for comprehensive investigations that integrate structural, functional, and clinical correlations. This underscores the importance of continued research aimed at identifying early morphological markers of placental damage and improving strategies for timely diagnosis and intervention.

RESEARCH METHODOLOGY

This study was designed as a comparative morphological investigation aimed at evaluating structural and functional alterations in placentas obtained from normal and complicated pregnancies. A total of sixty placentas were collected immediately after delivery from women who gave informed consent and were subsequently divided into two equal groups. Group I included thirty placentas obtained from clinically normal, uncomplicated pregnancies, while Group II comprised thirty placentas collected from pregnancies complicated by conditions such as preeclampsia, gestational diabetes, chronic maternal hypertension, or intrauterine growth restriction. Following delivery, placental tissues were carefully washed in isotonic saline solution to remove residual blood and were fixed in 10% neutral buffered formalin. Representative tissue samples were taken from central and peripheral regions of each placenta to ensure accurate morphological assessment. The specimens were processed using standard paraffin-embedding techniques and sectioned at 4–5 μm thickness. Routine histological examination was performed using hematoxylin and eosin (H&E) staining to evaluate general tissue architecture, villous structure, vascular integrity, and stromal condition. Periodic acid–Schiff (PAS) staining was applied to assess basement membrane thickness, glycogen content, and the presence of fibrinoid material. To further investigate cellular and molecular alterations, immunohistochemical analysis was conducted using antibodies against Caspase-3 as a marker of apoptotic activity and vascular endothelial growth factor (VEGF) to assess angiogenic potential. The intensity and distribution of immunoreactivity were evaluated semi-quantitatively. Morphometric analysis was performed using digital microscopy and specialized image analysis software to measure villous diameter, capillary density, trophoblastic thickness, and stromal area. Statistical analysis was conducted to compare the obtained parameters between the two groups, allowing for the identification of significant morphofunctional differences associated with placental damage.

ANALYSIS AND RESULTS

Comparative histological and morphometric analysis revealed marked structural and functional alterations in placentas obtained from complicated pregnancies. In Group II, pathological changes were significantly more pronounced compared with the

control group. Villous fibrosis was the most frequently observed lesion, detected in 83% of the examined placentas. This change was characterized by excessive collagen deposition within the villous stroma, leading to reduced elasticity and impaired diffusion capacity. Fibrinoid necrosis was identified in 76% of cases and was primarily localized around terminal villi and intervillous spaces. These necrotic deposits contributed to the obstruction of maternal–fetal exchange surfaces and further aggravated placental insufficiency. Capillary collapse was observed in 69% of the samples and manifested as narrowing or complete obliteration of villous capillary lumens, resulting in decreased microcirculatory perfusion. Immunohistochemical analysis demonstrated increased Caspase-3 expression in 64% of complicated placentas, indicating enhanced trophoblastic apoptosis. Excessive apoptotic activity led to thinning and discontinuity of the trophoblastic layer, compromising the integrity of the placental barrier. Simultaneously, a significant reduction in VEGF expression was noted, reflecting impaired angiogenesis and decreased formation of new microvessels. Morphometric measurements confirmed thickening of the trophoblastic membrane, reduction of vascular lumen diameter, and decreased capillary density in the pathological group. These combined changes significantly limited oxygen and nutrient diffusion across the placental barrier, ultimately leading to chronic fetal hypoxia and growth restriction. The severity of morphological alterations showed a strong correlation with adverse perinatal outcomes, including low birth weight, reduced Apgar scores, and increased neonatal intensive care admissions, emphasizing the clinical relevance of the detected placental lesions.

CONCLUSION AND RECOMMENDATIONS

The findings of this study confirm that structural and functional damage to the placenta plays a decisive role in the development of fetoplacental insufficiency and adverse pregnancy outcomes. Pronounced morphofunctional alterations, including villous fibrosis, fibrinoid necrosis, capillary collapse, and increased trophoblastic apoptosis, significantly impair uteroplacental and fetoplacental circulation. These changes reduce the efficiency of transplacental oxygen and nutrient exchange, leading to chronic fetal hypoxia, intrauterine growth restriction, and an increased risk of perinatal morbidity and mortality. The strong correlation between the severity of placental lesions and unfavorable neonatal outcomes emphasizes the clinical importance of early detection and continuous monitoring of placental function during pregnancy. Doppler ultrasonography of uterine and umbilical arteries should be routinely implemented in high-risk pregnancies to identify early signs of placental insufficiency. In addition, biochemical screening for angiogenic and oxidative stress markers may provide valuable supplementary diagnostic information. Based on the

obtained results, the implementation of preventive strategies aimed at improving placental perfusion is strongly recommended. These include optimized antenatal care, strict control of maternal systemic disorders, and the use of antioxidant and vasoprotective therapies to reduce oxidative stress and endothelial damage. Timely therapeutic interventions may significantly contribute to preserving placental function, improving fetal oxygenation, and reducing the incidence of adverse perinatal outcomes.

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