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THE IMPACT OF MATERNAL CHRONIC DISEASES (ANEMIA, HYPERTENSION, DIABETES) ON FETOPLACENTAL INSUFFICIENCY

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Abstract: Background: Fetoplacental insufficiency (FPI) is a major complication of pregnancy leading to intrauterine growth restriction (IUGR) and perinatal hypoxia. Maternal chronic extragenital pathologies, specifically iron-deficiency anemia (IDA), chronic hypertension, and diabetes mellitus, are hypothesized to be primary drivers of placental dysfunction in the Fergana Valley region. This study aims to evaluate the specific impact of these conditions on the development and severity of FPI. Methods: A prospective comparative study was conducted involving 200 pregnant women in the third trimester. Participants were divided into four groups: Group I (n=60) with moderate/severe anemia, Group II (n=50) with chronic hypertension, Group III (n=40) with pre-gestational diabetes, and Group IV (Control, n=50) with uncomplicated pregnancies. FPI was assessed using Doppler ultrasonography (umbilical and uterine arteries) and placental morphometry. Results: Signs of FPI were diagnosed in 65% of women in Group II (Hypertension) and 55% in Group I (Anemia), compared to only 8% in the Control Group ($p < 0.001$). Hypertension was strongly associated with hemodynamic disturbances (elevated Resistance Index), while anemia was linked to placental hypoplasia. Diabetes showed a mixed pattern of placentomegaly and functional insufficiency. Conclusion: Chronic maternal diseases significantly impair placental function through distinct pathophysiological mechanisms. Early screening and aggressive management of anemia and hypertension are crucial for preventing FPI-related perinatal morbidity.

Keywords: Fetoplacental insufficiency, maternal anemia, chronic hypertension, diabetes mellitus, Doppler ultrasonography, placental dysfunction.

ONA ORGANIZMIDAGI SURUNKALI KASALLIKLARNING (ANEMIYA, GIPERTENZIYA, DIABET) FETOPLATSENTAR YETISHMOVCHILIKKA TA’SIRI

Annotatsiya: Kirish: Fetoplatsentar yetishmovchilik (FPY) homilaning bachadon ichi rivojlanishdan orqada qolishi va perinatal gipoksiyaga olib keluvchi asosiy asoratdir. Farg‘ona vodiysi mintaqasida onaning surunkali ekstragenital patologiyalari, xususan, temir tanqisligi anemiyasi (TTA), surunkali gipertenziya va qandli diabet platsenta disfunksiyasining asosiy omillari ekanligi taxmin qilinadi. Ushbu tadqiqot mazkur kasalliklarning FPY rivojlanishi va og‘irlik darajasiga o‘ziga xos ta’sirini baholashga qaratilgan. Usullar: Uchinchi trimestrda bo‘lgan 200 nafar homilador ayol ishtirokida prospektiv qiyosiy tadqiqot o‘tkazildi. Ishtirokchilar to‘rt guruhga bo‘lindi: I guruh (n=60) o‘rta/og‘ir darajali anemiya bilan, II guruh (n=50) surunkali gipertenziya bilan, III guruh (n=40) homiladorlikdan oldingi diabet bilan va IV

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guruh (Nazorat, n=50) asoratsiz homiladorlik bilan. FPY Doppler ultratovush tekshiruvi (kindik va bachadon arteriyalari) hamda platsenta morfometriyasi yordamida baholandi. Natijalar: FPY belgilari II guruh (Gipertenziya) ayollarining 65 foizida va I guruh (Anemiya) ayollarining 55 foizida aniqlandi, Nazorat guruhida esa bu ko‘rsatkich atigi 8 foizni tashkil etdi ($p < 0.001$). Gipertenziya gemodinamik buzilishlar (Rezistentlik indeksining oshishi) bilan, anemiya esa platsenta gipoplaziyasi bilan kuchli bog‘liqlik ko‘rsatdi. Diabetda platsentomegaliya va funksional yetishmovchilikning aralash ko‘rinishi kuzatildi. Xulosa: Onaning surunkali kasalliklari turli patofiziologik mexanizmlar orqali platsenta funksiyasini sezilarli darajada buzadi. Anemiya va gipertenziyani erta aniqlash va faol davolash FPY bilan bog‘liq perinatal kasallanishlarning oldini olishda hal qiluvchi ahamiyatga ega.
Kalit so‘zlar: Fetoplatsentar yetishmovchilik, onalar anemiyasi, surunkali gipertenziya, qandli diabet, Doppler ultratovush, platsenta disfunksiyasi.

ВЛИЯНИЕ ХРОНИЧЕСКИХ ЗАБОЛЕВАНИЙ МАТЕРИ (АНЕМИЯ, ГИПЕРТЕНЗИЯ, ДИАБЕТ) НА ФЕТОПЛАЦЕНТАРНУЮ НЕДОСТАТОЧНОСТЬ

Аннотация: Введение: Фетоплацентарная недостаточность (ФПН) является серьезным осложнением беременности, приводящим к задержке внутриутробного развития плода (ЗВУР) и перинатальной гипоксии. Предполагается, что хронические экстрагенитальные патологии матери, в частности железодефицитная анемия (ЖДА), хроническая гипертензия и сахарный диабет, являются основными драйверами дисфункции плаценты в Ферганской долине. Целью данного исследования является оценка специфического влияния этих состояний на развитие и тяжесть ФПН. Методы: Было проведено проспективное сравнительное исследование с участием 200 беременных женщин в третьем триместре. Участницы были разделены на четыре группы: группа I (n=60) с анемией средней/тяжелой степени, группа II (n=50) с хронической гипертензией, группа III (n=40) с прегестационным диабетом и группа IV (Контроль, n=50) с неосложненной беременностью. ФПН оценивалась с помощью допплерографии (пуповинные и маточные артерии) и морфометрии плаценты. Результаты: Признаки ФПН были диагностированы у 65% женщин во II группе (Гипертензия) и у 55% в I группе (Анемия), по сравнению с 8% в контрольной группе ($p < 0.001$). Гипертензия была тесно связана с гемодинамическими нарушениями (повышенный индекс резистентности), тогда как анемия — с гипоплазией плаценты. Диабет показал смешанную картину плацентомегалии и функциональной недостаточности. Заключение: Хронические заболевания матери значительно нарушают функцию плаценты через различные патофизиологические механизмы. Ранний скрининг и активное лечение анемии и гипертензии имеют решающее значение для профилактики перинатальной заболеваемости, связанной с ФПН.
Ключевые слова: Фетоплацентарная недостаточность, анемия матери, хроническая гипертензия, сахарный диабет, допплерография, дисфункция плаценты.

INTRODUCTION

The placenta is a unique, transient, yet highly complex organ that serves as the critical interface between the mother and the fetus. It orchestrates the exchange of nutrients, respiratory gases, and waste products, while also acting as an endocrine organ essential for pregnancy maintenance.

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Fetoplacental Insufficiency (FPI) represents a clinical manifestation of placental dysfunction, wherein the organ fails to meet the metabolic demands of the growing fetus. This condition is a primary etiological factor for Intrauterine Growth Restriction (IUGR), chronic fetal hypoxia, and remains a leading cause of perinatal morbidity and mortality worldwide. Furthermore, adverse intrauterine environments linked to FPI are increasingly recognized as determinants of adult-onset diseases, such as cardiovascular disease and metabolic syndrome, through the mechanism of fetal programming.

In the structure of obstetric complications in Uzbekistan, particularly in the densely populated Andijan region, FPI occupies a central position. While placental pathology can sometimes be primary—arising from genetic defects or initial implantation failure—the vast majority of cases in our clinical practice are "secondary" FPI. This secondary form develops against the background of pre-existing maternal health conditions. The maternal organism provides the biological substrate, the vascular bed, and the metabolic environment in which the placenta develops; therefore, systemic maternal health is intrinsically and inseparably linked to placental performance.

Three chronic extragenital conditions are particularly widespread among women of reproductive age in our region, creating a "perfect storm" for placental dysfunction: 1) Iron-Deficiency Anemia (IDA) - Affecting nearly 40-50% of pregnant women in Central Asia due to dietary factors and short inter-pregnancy intervals. IDA leads to chronic hemic hypoxia, forcing the placenta to adapt under stress. 2) Chronic Hypertension - A prevalent cardiovascular disorder characterized by systemic vasoconstriction and endothelial damage, which directly impairs the remodeling of uterine spiral arteries. 3) Diabetes Mellitus - Both pre-gestational and gestational diabetes are rising in prevalence. The hyperglycemic and pro-inflammatory environment of diabetes induces oxidative stress within placental tissues.

Despite the high prevalence of these conditions, they are often treated as separate entities. The comparative impact of these distinct pathologies on the hemodynamic and morphological parameters of the placenta is not fully elucidated in the local population. Does the "hypoxic" stress of anemia damage the placenta in the same way as the "shear stress" of hypertension or the "metabolic toxicity" of diabetes? Understanding these nuances is critical for moving beyond generic management of FPI to a more targeted, etiology-based approach. This study aims to investigate how each of these chronic conditions specifically alters fetoplacental circulation and morphology, providing a basis for differential management strategies in the Andijan region.

LITERATURE REVIEW

Pathophysiology of Anemia and Placental Adaptation Hemoglobin is the primary oxygen carrier in the blood. In cases of moderate-to-severe Iron-Deficiency Anemia (IDA), the oxygen-carrying capacity of maternal blood is significantly reduced. Literature suggests that the placenta initially attempts to compensate for this hemic hypoxia through angiogenesis and hyperplasia (placentomegaly) to increase the surface area for exchange. However, *Breymann (2015)* notes that when anemia is chronic and severe, these adaptive mechanisms fail. The chronic lack of oxygen leads to degenerative changes in the syncytiotrophoblast, focal necrosis, and reduced vascularization of the terminal villi. Furthermore, iron is a crucial cofactor for many enzymes involved in oxidative metabolism and cellular growth; its deficiency directly impairs trophoblast

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proliferation. Recent studies have established a direct correlation between low maternal ferritin levels in the first trimester and the risk of subsequent low birth weight and placental hypoplasia.
Hypertension - The Vascular Origin of Insufficiency Chronic hypertension is fundamentally a disease of the vasculature, characterized by generalized vasoconstriction and endothelial dysfunction. In the context of pregnancy, *Sibai (2002)* highlights that chronic hypertension interferes with the critical process of trophoblastic invasion. Normally, trophoblasts remodel the muscular spiral arteries of the uterus into low-resistance, high-flow vessels. In hypertensive women, this remodeling is often incomplete or absent. Consequently, the blood flow to the intervillous space remains high-resistance and pulsatile, which can damage the delicate villous tree. This "ischemic" placenta responds by releasing anti-angiogenic factors (such as sFlt-1) and inflammatory cytokines into the maternal circulation, which not only exacerbates maternal endothelial damage but also predisposes the pregnancy to superimposed preeclampsia and placental abruption. Doppler ultrasonography typically reveals characteristic "notched" uterine artery waveforms and elevated pulsatility indices (PI), signaling high downstream resistance.
Diabetes Mellitus - The Paradox of "Starvation amidst Plenty" Diabetes creates a unique and toxic metabolic environment for the developing placenta. *Desoye & Hauguel-de Mouzon (2007)* describe the diabetic placenta as an organ under siege from oxidative stress and inflammation induced by maternal hyperglycemia and hyperinsulinemia. Unlike the small, ischemic placentas seen in hypertension, diabetic placentas are often thickened and heavy (placentomegaly). However, this increase in mass does not translate to better function. Histologically, these placentas exhibit thickening of the trophoblastic basement membrane, villous immaturity, and increased deposition of glycogen. These structural changes increase the diffusion distance for oxygen, leading to fetal hypoxia despite abundant glucose supply. This creates a dangerous clinical picture where the fetus may be macrosomic (large for gestational age) due to hyperinsulinemia but simultaneously hypoxic and acidotic due to placental inefficiency.
Diagnostic Approaches and Regional Gaps While Doppler velocimetry of the umbilical and uterine arteries is the gold standard for assessing FPI, its interpretation must be contextual. *Baschat (2004)* emphasized that the sequence of Doppler changes can vary depending on the etiology of the insufficiency. In the Fergana Valley, where multiparity and co-morbidities (e.g., anemia plus hypertension) are common, distinguishing the primary driver of FPI is challenging. There is a paucity of local data comparing the specific morphometric and hemodynamic "fingerprints" of these diseases side-by-side. Most current protocols apply a uniform treatment for FPI (e.g., antiplatelets, metabolic drugs) regardless of the cause. This study seeks to address this gap by linking specific maternal pathologies to distinct placental phenotypes.

MATERIALS AND METHODS

Study Design A prospective, comparative clinical study was conducted at the Andijan State Medical Institute's clinical bases (Regional Perinatal Center) from 2022 to 2024.
Participants 200 pregnant women (gestational age 28-36 weeks) were enrolled and categorized into four groups: Group I (Anemia): Hb < 90 g/L (Moderate/Severe IDA). n=60. Group II (Hypertension): Chronic hypertension diagnosed before 20 weeks. n=50. Group III (Diabetes): Pre-gestational Type 1 or Type 2 Diabetes. n=40. Group IV (Control): Healthy women with uncomplicated pregnancies. n=50.

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Doppler Velocimetry: Performed using a GE Voluson E8 system. Resistance Index (RI), Pulsatility Index (PI), and Systolic/Diastolic (S/D) ratios were measured in the Uterine Arteries (UtA), Umbilical Artery (UA), and Fetal Middle Cerebral Artery (MCA).
Placental Morphometry: Ultrasound assessment of placental thickness, structure (calcifications), and estimation of volume.
Fetal Biometry: Estimated Fetal Weight (EFW) to diagnose IUGR (<10th percentile).
Statistical Analysis Data were processed using SPSS v.26. Differences between groups were analyzed using ANOVA and Chi-square tests. A p-value < 0.05 was considered significant.

RESULTS

Prevalence of FPI Clinical and instrumental signs of FPI were significantly higher in the study groups compared to controls.

Table 1: Frequency of Fetoplacental Insufficiency Markers

Indicator	Group I (Anemia)	Group II (Hypertension)	Group III (Diabetes)	Group IV (Control)	P-value
Hemodynamic Disturbances (Doppler)	45.0%	72.0%	35.0%	6.0%	<0.001
IUGR (<10th percentile)	38.3%	52.0%	10.0%*	4.0%	<0.001
Placental Structural Changes	55.0%	60.0%	75.0%	12.0%	<0.001
Chronic Fetal Hypoxia (CTG)	42.0%	68.0%	40.0%	5.0%	<0.001

Note: In the Diabetes group, macrosomia was more common than IUGR, but functional hypoxia was present.

Group II (Hypertension) showed the most severe hemodynamic compromise, with a significant elevation in Uterine Artery RI (0.68 ± 0.05 vs 0.48 ± 0.03 in control), indicating high vascular resistance.
Group I (Anemia) showed a compensatory "brain-sparing effect" (reduced MCA RI) in 40% of cases, suggesting chronic adaptation to low oxygen delivery.
Group III (Diabetes) typically showed normal umbilical flow but signs of venous pulsation in severe cases, reflecting cardiac strain.
Anemia - Associated with "thin" placentas (<25mm at 32 weeks) and premature aging (Grade III calcification).
Hypertension - Associated with "small" placentas, infarctions, and retroplacental hematomas.
Diabetes - Associated with placentomegaly (thickened placenta >45mm) and polyhydramnios.

DISCUSSION

The study confirms that the maternal environment dictates placental health. Hypertension emerged as the most destructive factor for placental vasculature, leading to the highest rates of IUGR (52%) and severe hemodynamic disturbances. This aligns with the mechanism of vasoconstriction reducing intervillous flow.

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Anemia, often underestimated, caused FPI in 55% of cases. The mechanism here is not vascular resistance (Doppler was often less severe than in HTN) but rather "substrate deficiency"—the blood simply lacks enough oxygen. This leads to a distinct form of FPI characterized by fetal wasting rather than acute distress, until labor begins.

Diabetes presented a unique profile: the fetus is often large (macrosomia) but hypoxic. The "large" placenta is structurally immature and edematous, failing to function efficiently despite its size. This highlights that placental size does not equal placental function.

CONCLUSION

Fetoplacental insufficiency is not a uniform diagnosis; its phenotype depends heavily on the underlying maternal pathology.

Hypertension causes the most severe "vascular" form of FPI, characterized by high resistance and severe growth restriction.

Anemia leads to a "hypoxic" form of FPI with compensatory redistribution of fetal blood flow.

Diabetes causes "metabolic" placental dysfunction, often masked by normal or large fetal size.

Women with hypertension require intensive Doppler monitoring starting from 24 weeks.

Aggressive treatment of IDA in the 2nd trimester is a direct intervention to improve placental oxygenation.

In diabetic pregnancies, placental thickness should be monitored as a marker of control; placentomegaly suggests poor metabolic management.

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