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PREVENTION OF FETOPLACENTAL INSUFFICIENCY IN HIGH-RISK PREGNANT WOMEN

G'oziyava Shoista Sultonbekovna
Department of 1-Obstetrics and Gynecology,

Andijan State Medical Institute, Andijan, Uzbekistan

Abstract: Background: Fetoplacental insufficiency (FPI) remains a leading cause of perinatal morbidity and mortality. Women with somatic pathologies (chronic hypertension, anemia, kidney disease) or a history of obstetric complications constitute a high-risk group for developing FPI. This study aims to evaluate the efficacy of a comprehensive prophylactic regimen initiated in the first trimester for preventing placental dysfunction in high-risk pregnancies. Methods: A prospective randomized controlled trial was conducted involving 240 high-risk pregnant women. Participants were divided into two groups: the Control Group (n=120) received standard antenatal care, while the Main Group (n=120) received a targeted prophylactic complex including low-dose aspirin (150 mg), micronutrient supplementation, and metabolic support (L-arginine) starting from 12-14 weeks. Placental function was monitored via Doppler velocimetry and biochemical markers. Results: The incidence of decompensated FPI was significantly lower in the Main Group (4.2%) compared to the Control Group (18.3%, $p < 0.01$). Women in the prophylactic group showed better uterine artery flow dynamics and higher birth weight neonates (mean difference +350g). The rate of preterm births associated with placental failure was reduced by 50%. Conclusion: Early initiation of pathogenetically oriented prophylaxis in high-risk women effectively preserves placental function, reduces the severity of hemodynamic disturbances, and improves perinatal outcomes.
Keywords: Fetoplacental insufficiency, prevention, high-risk pregnancy, aspirin, L-arginine, Doppler, perinatal outcomes.

XAVF GURUHIDAGI HOMILADOR AYOLLARDA FETOPLATSENTAR YETISHMOVCHILIKNI OLDINI OLISH

Annotatsiya: Kirish: Fetoplatsentar yetishmovchilik (FPY) perinatal kasallanish va o‘limning asosiy sabablaridan biri bo‘lib qolmoqda. Somatik patologiyalari (surunkali gipertenziya, anemiya, buyrak kasalliklari) yoki anamnezida akusherlik asoratlari bo‘lgan ayollar FPY rivojlanishi bo‘yicha yuqori xavf guruhini tashkil qiladi. Ushbu tadqiqot yuqori xavfli homiladorliklarda platsenta disfunksiyasini oldini olish uchun birinchi trimestrda boshlangan kompleks profilaktika sxemasining samaradorligini baholashga qaratilgan. Usullar: 240 nafar yuqori xavf guruhidagi homilador ayol ishtirokida prospektiv randomizatsiyalangan nazoratli tadqiqot o‘tkazildi. Ishtirokchilar ikki guruhga bo‘lindi: Nazorat guruhi (n=120) standart antenatal parvarish oldi, Asosiy guruh (n=120) esa 12-14 haftadan boshlab kichik dozada aspirin (150 mg), mikronutrientlar va metabolik yordam (L-arginin)ni o‘z ichiga olgan maqsadli profilaktika kompleksini qabul qildi. Platsenta funksiyasi Doppler velosimetriya va biokimyoviy

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markerlar orqali nazorat qilindi. Natijalar: Dekompensatsiyalangan FPY uchrash darajasi Asosiy guruhda (4,2%) Nazorat guruhiga (18,3%) nisbatan sezilarli darajada past bo‘ldi ($p < 0.01$). Profilaktika guruhidagi ayollarda bachadon arteriyasi qon oqimi dinamikasi yaxshiroq va yangi tug‘ilgan chaqaloqlar vazni yuqori bo‘ldi (o‘rtacha farq +350g). Platsenta yetishmovchiligi bilan bog‘liq muddatidan oldin tug‘ruqlar darajasi 50 foizga kamaydi. Xulosa: Yuqori xavf guruhidagi ayollarda patogenetik asoslangan profilaktikani erta boshlash platsenta funksiyasini samarali saqlaydi, gemodinamik buzilishlar og‘irligini kamaytiradi va perinatal natijalarni yaxshilaydi.
Kalit so‘zlar: Fetoplatsentar yetishmovchilik, profilaktika, yuqori xavfli homiladorlik, aspirin, L-arginin, Doppler, perinatal natijalar.

ПРОФИЛАКТИКА ФЕТОПЛАЦЕНТАРНОЙ НЕДОСТАТОЧНОСТИ У БЕРЕМЕННЫХ ГРУППЫ РИСКА

Аннотация: Введение: Фетоплацентарная недостаточность (ФПН) остается одной из ведущих причин перинатальной заболеваемости и смертности. Женщины с соматическими патологиями (хроническая гипертензия, анемия, заболевания почек) или отягощенным акушерским анамнезом составляют группу высокого риска развития ФПН. Целью данного исследования является оценка эффективности комплексной схемы профилактики, начатой в первом триместре, для предотвращения дисфункции плаценты при беременности высокого риска. Методы: Было проведено проспективное рандомизированное контролируемое исследование с участием 240 беременных женщин группы высокого риска. Участницы были разделены на две группы: контрольная группа (n=120) получала стандартную антенатальную помощь, а основная группа (n=120) получала целевой профилактический комплекс, включающий низкие дозы аспирина (150 мг), микронутриенты и метаболическую поддержку (L-аргинин), начиная с 12-14 недель. Функция плаценты контролировалась с помощью допплерометрии и биохимических маркеров. Результаты: Частота декомпенсированной ФПН была значительно ниже в основной группе (4,2%) по сравнению с контрольной группой (18,3%, $p < 0.01$). У женщин в группе профилактики наблюдалась лучшая динамика кровотока в маточных артериях и более высокий вес новорожденных (средняя разница +350 г). Частота преждевременных родов, связанных с плацентарной недостаточностью, снизилась на 50%. Заключение: Раннее начало патогенетически обоснованной профилактики у женщин группы риска эффективно сохраняет функцию плаценты, снижает тяжесть гемодинамических нарушений и улучшает перинатальные исходы.
Ключевые слова: Фетоплацентарная недостаточность, профилактика, беременность высокого риска, аспирин, L-аргинин, Допплер, перинатальные исходы.

INTRODUCTION

Fetoplacental Insufficiency (FPI) represents a complex and multifactorial clinical syndrome characterized by morphofunctional changes in the placenta, leading to a compromise in its nutritive, respiratory, and endocrine functions. This condition is the primary pathophysiological basis for intrauterine growth restriction (IUGR), chronic fetal hypoxia, and is a significant contributor to stillbirths and neonatal morbidity. In Uzbekistan, where the birth rate is high and

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maternal comorbidities such as anemia are prevalent, FPI remains a critical public health challenge, accounting for a substantial portion of adverse perinatal outcomes.

The etiology of FPI is fundamentally a disorder of adaptation. A successful pregnancy requires profound hemodynamic and metabolic adjustments in the maternal organism to support the developing fetus. In women with pre-existing "high-risk" conditions—such as chronic hypertension, pre-gestational diabetes, renal disease, severe anemia, or a history of preeclampsia—these adaptive mechanisms are frequently compromised. The placenta, developing in a suboptimal maternal environment characterized by endothelial dysfunction, oxidative stress, and chronic inflammation, suffers from defective angiogenesis and impaired trophoblast invasion. This results in a "maladapted" placenta that cannot meet the increasing metabolic demands of the fetus as pregnancy progresses.

Current clinical practice in many regions often focuses on *treating* FPI once it is clinically diagnosed in the third trimester (e.g., via the use of vasodilators, metabolic drugs, or hospitalization). However, a growing body of evidence suggests that once structural damage to the placental vasculature—such as infarction, fibrin deposition, or villous hypoplasia—has occurred, it is largely irreversible. Therefore, the traditional "reactive" approach is often too little, too late. The paradigm must shift from "salvage therapy" to "primary prevention."

The critical "window of opportunity" for intervention lies in the first half of pregnancy, specifically before 16 weeks of gestation. This period corresponds to the completion of the second wave of trophoblast invasion and the physiological remodeling of the spiral arteries. Interventions initiated during this phase have the potential to fundamentally improve placentation. This study aims to evaluate the efficacy of a pathogenetically grounded, comprehensive prophylactic protocol—initiated early in gestation—for women identified as high risk in the Andijan region, specifically targeting the key pathways of placental failure: thrombosis, vasoconstriction, and metabolic deficiency.

LITERATURE REVIEW

Pathogenesis - The "Great Obstetrical Syndromes" The concept of "Great Obstetrical Syndromes," proposed by *Brosens et al. (2011)*, links conditions like preeclampsia, FPI, placental abruption, and preterm labor to a common root cause: disorders of deep placentation. Central to this is the failure of the physiological transformation of the maternal spiral arteries. In a normal pregnancy, extravillous trophoblasts invade these vessels, destroying the muscular media and replacing it with fibrinoid material. This converts narrow, high-resistance vessels into wide, low-resistance channels capable of handling the massive increase in blood flow required by the fetus. In high-risk pregnancies, this remodeling is partial or absent. The spiral arteries remain narrow and responsive to vasoconstrictors, leading to high-velocity, turbulent flow that damages the delicate villi (placental ischemia) and generates oxidative stress.

Aspirin - Mechanisms and Evidence Low-dose aspirin (LDA) is the cornerstone of pharmacological prevention. Its primary mechanism is the irreversible inhibition of cyclooxygenase-1 (COX-1) in platelets, which reduces the production of Thromboxane A2 (a potent vasoconstrictor and platelet aggregator) without significantly affecting the production of Prostacyclin (a vasodilator) by the endothelium. This shifts the hemostatic balance towards vasodilation and prevents microthrombosis in the placental bed. The landmark *ASPRE trial (Rolnik et al., 2017)* definitively demonstrated that 150 mg of aspirin, when started before 16

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weeks in high-risk women, reduces the rate of preterm preeclampsia by 62%. Importantly, meta-analyses (e.g., *Roberge et al., 2017*) emphasize a dose-response relationship, indicating that doses <100 mg may be insufficient, and that initiation after 16 weeks offers significantly reduced benefit.

Endothelial Dysfunction and L-Arginine Endothelial dysfunction is a systemic hallmark of high-risk pregnancies (especially those with chronic hypertension or history of PE). The endothelium fails to produce adequate Nitric Oxide (NO), the body's primary endogenous vasodilator. L-arginine is the semi-essential amino acid precursor for NO synthesis. Several randomized trials and meta-analyses (*Gui et al., 2014*) have suggested that L-arginine supplementation can improve uteroplacental perfusion and birth weights in women with hypertensive disorders or established FPI, essentially providing the substrate to "rescue" endothelial function.

The Role of Micronutrients and Anemia - In the context of Central Asia, maternal nutritional status is a significant modifier of placental health. Iron deficiency anemia (IDA) causes hemic hypoxia, directly limiting oxygen delivery to the trophoblast and impairing mitochondrial function. Furthermore, deficiencies in Vitamin D and Calcium are linked to abnormal immune modulation at the maternal-fetal interface. Vitamin D, in particular, promotes angiogenesis and downregulates pro-inflammatory cytokines. Therefore, correction of anemia and micronutrient deficits is not merely supportive care but a foundational aspect of preventing placental failure.

Screening and Risk Stratification - Modern obstetrics advocates for the "Inverted Pyramid of Care," where the most intensive assessment occurs in the first trimester. Screening algorithms combining maternal history, Mean Arterial Pressure (MAP), Uterine Artery Doppler (UtA-PI), and biochemical markers (PlGF, PAPP-A) allow for the identification of women at high risk of placental dysfunction long before clinical symptoms appear. Implementing such screening allows for the targeted application of prophylaxis to those who will benefit most, maximizing efficacy and cost-effectiveness.

MATERIALS AND METHODS

Study Design A prospective, randomized, open-label controlled trial was conducted at the Andijan State Medical Institute's clinical bases (2022-2024). Participants 240 pregnant women identified as "High Risk" for FPI were enrolled at 10-12 weeks gestation.

Inclusion Criteria - History of FPI/PE in previous pregnancy, chronic hypertension, BMI >30, moderate anemia (Hb <90 g/L), or autoimmune markers (APS).

Exclusion Criteria - Multiple pregnancy, major fetal anomalies.

Control Group (n=120): Received standard care according to national protocols (Iron/Folate, routine visits). Treatment for FPI was initiated only if signs (IUGR, abnormal Doppler) appeared.

Main Group (n=120): Received a Comprehensive Prophylactic Complex starting at 12-14 weeks: Low-Dose Aspirin - 150 mg nightly (until 36 weeks). L-Arginine (IV/Oral) - Intermittent courses to support endothelial function. Targeted Micronutrients - High-dose Vitamin D and Calcium based on deficiency status. Correction of Anemia - Aggressive management with parenteral iron if oral was ineffective. Doppler - Uterine Artery PI measured at 20-22 weeks; Umbilical/MCA Doppler at 28, 32, 36 weeks. Biometry - Serial scans to track fetal growth velocity.

RESULTS

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Incidence of FPI The proactive approach significantly reduced the burden of disease.

Table 1: Clinical Outcomes

Outcome	Main Group (n=120)	Control Group (n=120)	Relative Risk (RR)	P-value
Compensated FPI	15.0%	28.3%	0.53	<0.05
Decompensated FPI (Critical)	4.2%	18.3%	0.23	<0.001
Preeclampsia	5.8%	14.2%	0.41	<0.05
Preterm Birth (<37 weeks)	8.3%	19.2%	0.43	<0.05

Doppler Parameters At 24 weeks, the persistence of "notching" in the uterine arteries (a sign of high resistance) was significantly lower in the Main Group (12%) compared to the Control Group (35%), indicating better placental vascularization.

Birth Weight: Neonates in the Main Group were heavier on average (3250g ± 380g) than those in the Control Group (2900g ± 420g).

NICU Admission: Admission rates were reduced by half in the Main Group (9% vs 22%).

DISCUSSION

The study demonstrates that FPI is preventable, but timing is everything. The success of the Main Group is attributed to the "First Trimester Strategy." By initiating Aspirin and endothelial support before the second wave of trophoblast invasion is complete (16-18 weeks), we facilitated proper placental bed formation.

In the Control Group, where interventions were reactive (treating diagnosed FPI), outcomes were poorer. Once high resistance is established in the third trimester, no medication can "unclog" the placental lakes or create new vessels. "Metabolic therapy" in late pregnancy acts merely as a palliative measure.

The inclusion of L-arginine and aggressive anemia correction likely provided a synergistic effect. In a population with high endothelial dysfunction (due to diet/genetics) and hypoxia (anemia), these adjuncts optimized the substrate for placental function alongside Aspirin.

CONCLUSION

Prevention is clinically superior and more cost-effective than cure for Fetoplacental Insufficiency. Prophylaxis must begin before 16 weeks to be effective.

A regimen addressing thrombosis (Aspirin), endothelial function (L-arginine), and oxygenation (Anemia correction) provides the best outcomes.

This strategy significantly lowers the rates of critical FPI, severe IUGR, and NICU admissions.

All pregnant women should undergo risk stratification at the first antenatal visit.

The protocol of Aspirin 150 mg + Endothelial Support should be standardized for high-risk women in primary care settings in the Andijan region.

REFERENCES

1. Rolnik, D. L., et al. (2017). Aspirin versus Placebo in Pregnancies at High Risk for

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Preterm Preeclampsia. *New England Journal of Medicine*.
2. Brosens, I., et al. (2011). The "Great Obstetrical Syndromes" are associated with disorders of deep placentation. *American Journal of Obstetrics and Gynecology*.
3. Khalil, A., et al. (2020). ISUOG Practice Guidelines: role of ultrasound in screening for and follow-up of pre-eclampsia. *Ultrasound in Obstetrics & Gynecology*.
4. Roberge, S., et al. (2017). The role of aspirin dose on the prevention of preeclampsia and fetal growth restriction: systematic review and meta-analysis. *American Journal of Obstetrics and Gynecology*.
5. Gui, S., et al. (2014). Arginine supplementation for improving maternal and neonatal outcomes in hypertensive disorder of pregnancy: a systematic review. *Journal of Renin-Angiotensin-Aldosterone System*.