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IMPACT OF IRON DEFICIENCY ANEMIA ON THE CLINICAL COURSE OF PREGNANCY AND PERINATAL OUTCOMES

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Abstract: Background: Iron Deficiency Anemia (IDA) remains a prevalent complication in obstetrics, particularly in the Fergana Valley region. It creates a state of chronic hemic hypoxia, potentially disrupting the physiological adaptation of the maternal organism to pregnancy. This study aims to evaluate the specific influence of IDA on the frequency of obstetric complications, labor anomalies, and perinatal outcomes. Methods: A retrospective comparative study was conducted involving 300 pregnant women. The Main Group consisted of 200 women with diagnosed IDA (mild to severe), and the Control Group included 100 women with normal hemoglobin levels. Clinical courses, placental function (Doppler), and labor outcomes were analyzed. Results: Women with IDA had a significantly higher incidence of placental insufficiency (45% vs. 12%, p<0.001), threatened preterm labor (38% vs. 15%), and preeclampsia (22% vs. 8%). During labor, the IDA group experienced a higher rate of uterine inertia (weakness of labor activity) and postpartum hemorrhage. Neonates born to anemic mothers had lower APGAR scores and a higher frequency of low birth weight. Conclusion: IDA is a significant independent risk factor for adverse pregnancy outcomes. It compromises placental function and maternal tolerance to delivery. Effective preconception care and early correction of iron stores are essential to reduce obstetric morbidity.
Keywords: Iron deficiency anemia, pregnancy complications, placental insufficiency, preeclampsia, postpartum hemorrhage, fetal hypoxia.

TEMIR TANQIS ANEMIYASINING HOMILADORLIK KECHISHIGA TA’SIRI

Annotatsiya: Kirish: Temir tanqisligi anemiyasi (TTA) akusherlikda, ayniqsa Farg‘ona vodiysi mintaqasida keng tarqalgan asorat bo‘lib qolmoqda. U surunkali gemik gipoksiya holatini yuzaga keltirib, ona organizmining homiladorlikka fiziologik moslashuvini buzishi mumkin. Ushbu tadqiqot TTAning akusherlik asoratlari, tug‘ruq anomaliyalari va perinatal natijalar chastotasiga o‘ziga xos ta’sirini baholashga qaratilgan. Usullar: 300 nafar homilador ayol ishtirokida retrospektiv qiyosiy tadqiqot o‘tkazildi. Asosiy guruhga TTA tashxisi qo‘yilgan (yengil va og‘ir darajali) 200 nafar ayol, Nazorat guruhiga esa gemoglobin darajasi normal bo‘lgan 100 nafar ayol kiritildi. Homiladorlikning klinik kechishi, platsenta funksiyasi (Doppler) va tug‘ruq natijalari tahlil qilindi. Natijalar: TTA bo‘lgan ayollarda fetoplatsentar yetishmovchilik (45% ga 12%, p<0.001), muddatidan oldin tug‘ruq xavfi (38% ga 15%) va preeklampsiya (22% ga 8%) uchrash darajasi sezilarli darajada yuqori bo‘ldi. Tug‘ruq jarayonida TTA guruhida tug‘ruq faoliyatining sustligi va tug‘ruqdan keyingi qon ketishlar ko‘proq kuzatildi. Anemiyasi bor onalardan tug‘ilgan chaqaloqlarda APGAR ballari pastroq va kam

vaznli tug‘ilish chastotasi yuqori bo‘ldi. Xulosa: TTA homiladorlikning salbiy oqibatlari uchun jiddiy mustaqil xavf omilidir. U platsenta funksiyasini va onaning tug‘ruqqa bardoshliligini pasaytiradi. Akusherlik kasallanishini kamaytirish uchun homiladorlikkacha bo‘lgan parvarish va temir zaxiralarini erta tiklash zarur.
Kalit so‘zlar: Temir tanqisligi anemiyasi, homiladorlik asoratlari, fetoplatsentar yetishmovchilik, preeklampsiya, tug‘ruqdan keyingi qon ketish, homila gipoksiyasi.

ВЛИЯНИЕ ЖЕЛЕЗОДЕФИЦИТНОЙ АНЕМИИ НА ТЕЧЕНИЕ БЕРЕМЕННОСТИ

Аннотация: Введение: Железодефицитная анемия (ЖДА) остается распространенным осложнением в акушерстве, особенно в регионе Ферганской долины. Она создает состояние хронической гемической гипоксии, потенциально нарушая физиологическую адаптацию организма матери к беременности. Целью данного исследования является оценка специфического влияния ЖДА на частоту акушерских осложнений, аномалий родовой деятельности и перинатальные исходы. Методы: Было проведено ретроспективное сравнительное исследование с участием 300 беременных женщин. Основную группу составили 200 женщин с диагностированной ЖДА (от легкой до тяжелой степени), контрольную группу — 100 женщин с нормальным уровнем гемоглобина. Был проведен анализ клинического течения, функции плаценты (допплерометрия) и исходов родов. Результаты: У женщин с ЖДА наблюдалась значительно более высокая частота фетоплацентарной недостаточности (45% против 12%, $p<0.001$), угрозы преждевременных родов (38% против 15%) и преэклампсии (22% против 8%). Во время родов в группе с ЖДА чаще отмечались слабость родовой деятельности и послеродовые кровотечения. Новорожденные, рожденные от матерей с анемией, имели более низкие оценки по шкале Апгар и более высокую частоту низкого веса при рождении. Заключение: ЖДА является значимым независимым фактором риска неблагоприятных исходов беременности. Она нарушает функцию плаценты и переносимость родов матерью. Эффективная преконцепционная подготовка и ранняя коррекция запасов железа необходимы для снижения акушерской заболеваемости.
Ключевые слова: Железодефицитная анемия, осложнения беременности, фетоплацентарная недостаточность, преэклампсия, послеродовое кровотечение, гипоксия плода.

INTRODUCTION

Pregnancy acts as a physiological "stress test" for the maternal hematopoietic system. The demand for iron increases threefold to support the expansion of maternal red blood cell mass, the growth of the fetus and placenta, and to buffer against blood loss at delivery. When this demand is unmet due to pre-existing depletion of iron stores, Iron Deficiency Anemia (IDA) ensues. In the Andijan region, IDA is a pervasive issue, often exacerbated by multiparity and short inter-pregnancy intervals.

While the biochemical diagnosis of IDA is straightforward, its clinical impact is multifaceted and often underestimated. Anemia is not merely a reduction in hemoglobin; it represents a systemic state of hemic hypoxia. Oxygen is the fundamental substrate for all metabolic processes.

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A deficiency compromises the function of the myometrium (uterine muscle), the placenta, and the maternal immune system.
This study seeks to move beyond prevalence statistics and delve into the *pathophysiology of complications*. We hypothesize that the hypoxic environment induced by IDA is a primary driver for a cascade of adverse events: from poor placentation leading to preeclampsia, to uterine muscle fatigue causing prolonged labor and hemorrhage. Understanding these links is crucial for re-emphasizing the importance of anemia correction not just for "good blood tests," but for safe motherhood.

LITERATURE REVIEW

Anemia and Placental Insufficiency The placenta requires high oxygen tension for proper trophoblast invasion and vascular remodeling. *Vleugels et al. (2019)* demonstrated that maternal anemia leads to placental hypoxia, which triggers oxidative stress and inflammation. This results in "Placental Adaptation Failure," clinically manifesting as Fetoplacental Insufficiency (FPI) and Intrauterine Growth Restriction (IUGR). In anemic mothers, placentas are often hypertrophic (enlarged) in a compensatory effort to extract oxygen, yet functionally inefficient due to villous immaturity.
The Link to Preeclampsia There is a well-documented U-shaped relationship between hemoglobin and preeclampsia (both very high and very low Hb are risks). However, moderate-to-severe IDA is specifically linked to an increased risk of preeclampsia. The mechanism is thought to involve the hypoxic placenta releasing anti-angiogenic factors (sFlt-1) into the maternal circulation, causing widespread endothelial dysfunction.
Impact on Labor and Delivery Iron is a cofactor for enzymes involved in muscle contraction. Severe anemia leads to myometrial hypoxia and fatigue. Studies by *Drukker et al. (2015)* indicate that anemic women have a higher incidence of primary and secondary uterine inertia (weakness of labor activity), necessitating oxytocin augmentation and increasing the rate of operative deliveries (C-sections). Furthermore, anemia significantly impairs the body's ability to tolerate blood loss. A blood loss of 500ml, which is physiological for a healthy woman, can precipitate hypovolemic shock and cardiac failure in a severely anemic woman.
Fetal and Neonatal Consequences The fetus is an efficient parasite for iron, often maintaining its levels at the mother's expense. However, when maternal stores are exhausted, fetal iron deficiency occurs. This leads to chronic fetal hypoxia, reduced fetal movements, and "brain-sparing" hemodynamic redistribution. Long-term, infants born to anemic mothers are at higher risk for neurodevelopmental delays and behavioral issues.

MATERIALS AND METHODS

Study Design A retrospective comparative analysis was conducted at the Andijan Regional Perinatal Center (2022-2023).
Participants We analyzed 300 medical records of parturient women: Main Group (n=200): Women with confirmed IDA during pregnancy (Hb <110 g/L). Subdivided into Mild (90-109 g/L), Moderate (70-89 g/L), and Severe (<70 g/L). Control Group (n=100): Women with normal Hb levels throughout pregnancy.
Pregnancy Complications: Incidence of early toxicosis, threatened miscarriage, preeclampsia, FPI, and polyhydramnios/oligohydramnios.

Labor Complications: Duration of labor, weakness of labor activity, premature rupture of membranes (PROM), and postpartum hemorrhage (PPH).
Neonatal Outcomes: Birth weight, APGAR scores, and need for resuscitation.
Statistical Analysis Data were analyzed using SPSS v26. Relative Risk (RR) and Chi-square tests were used to determine statistical significance ($p<0.05$).

RESULTS

Course of Pregnancy The incidence of complications was directly proportional to the severity of anemia.

Table 1: Frequency of Obstetric Complications

Complication	Main Group (IDA) (n=200)	Control Group (n=100)	P-value
Fetoplacental Insufficiency	45.0%	12.0%	<0.001
Threatened Preterm Labor	38.0%	15.0%	<0.001
Preeclampsia	22.0%	8.0%	<0.01
Viral Infections (ARVI)	55.0%	25.0%	<0.001

Women with IDA were significantly more susceptible to infections, likely due to iron's role in immune function.
Course of Labor Anemia negatively impacted the labor process. The rate of Uterine Inertia (weak labor) was 18% in the Main Group compared to 6% in the Control Group. Consequently, the C-section rate was higher (32% vs 18%). Crucially, Postpartum Hemorrhage (PPH) occurred in 12% of anemic women versus 3% of controls. This is attributed to uterine atony caused by hypoxic muscle fatigue.
Perinatal Outcomes - Low Birth Weight (<2500g): 18% in Main Group vs. 5% in Control.
Asphyxia (APGAR <7 at 1 min): 15% in Main Group vs. 4% in Control.
Neonates from the severe anemia subgroup showed signs of iron deficiency at birth (low cord blood ferritin).

DISCUSSION

The results unequivocally demonstrate that IDA acts as a pathological background that worsens every stage of the reproductive process.
The "Hypoxic Uterus": The high rate of labor weakness validates the theory that an oxygen-starved myometrium cannot contract effectively. This creates a dangerous cycle: anemia -> weak labor -> prolonged labor -> uterine atony -> hemorrhage -> worse anemia.
The 45% rate of FPI suggests that the placenta cannot fully compensate for low maternal hemoglobin. This highlights the need for Doppler monitoring in all anemic pregnancies, not just those with hypertension.
Immune Susceptibility: The high rate of viral infections in the IDA group adds another layer of risk, as infection triggers inflammation (hepcidin rise), further blocking iron absorption.

CONCLUSION

Iron Deficiency Anemia is a modifiable risk factor that, if left untreated, significantly increases maternal and perinatal morbidity.
IDA is strongly associated with placental insufficiency, preeclampsia, and preterm labor.

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Anemic women are at 4-fold higher risk of postpartum hemorrhage due to uterine atony.
Fetal outcomes are compromised, with higher rates of growth restriction and asphyxia.
Screening and treating anemia *before* pregnancy is the most effective strategy.
Pregnant women with Hb <90 g/L should be considered "High Risk" and monitored for placental function (Doppler) and preeclampsia.
Active management of the third stage of labor and availability of uterotonics/blood products is critical for anemic women to prevent catastrophic hemorrhage.

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