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Forecasting And Prevention of Fetoplacentar Insufficiency in Pregnant Women with Preterm Obstetric Care

Jalilova Dildora Murot kizi

Samarkand State Medical University. Independent researcher, Samarkand, Uzbekistan.
Orcid: 0009-0007-0174-1416, E mail: jalilovad7@gmail.com

ABSTRACT: Preterm births are one of the most pressing problems in obstetric practice, negatively impacting high perinatal morbidity and mortality rates and the health of newborns. In recent years, special attention has been paid to the significance of oxidative stress and ferroptosis mechanisms in the placental tissue. In this direction, a comprehensive study of the clinical, echographic, microbiological, biochemical, and morphological characteristics of the fetoplacental complex is of great scientific and practical importance for improving early diagnosis, prognosis, and preventive measures.

1. INTRODUCTION

Fetoplacental dysfunction, also known as placental insufficiency, is a pathological condition that develops as a result of changes in the structure and function of the placenta, coupled with a decline in its adaptive and compensatory capabilities. Although the placenta is one of the primary organs that ensures the normal growth and development of the fetus, its functional capacity can become limited in certain situations. In particular, the risk of preterm labor combined with fetoplacental insufficiency elevates the risk of chronic fetal hypoxia, growth restriction, and neonatal complications. An important role in the development of fetoplacental insufficiency is played by infectious and inflammatory processes, changes in the vaginal microbiota, and hemodynamic, metabolic, and hormonal disorders. According to the International Classification of Diseases (ICD-10), this condition is included in the group of pathologies characteristic of the perinatal period. Placental insufficiency often develops against the background of various maternal extragenital and obstetric diseases and is characterized by disruptions in the hormonal, nutritional, metabolic, and transport functions of the placenta. As a result, severe complications such as intrauterine fetal growth retardation, preeclampsia, chronic hypoxia, and antenatal fetal death may develop. However, the contribution of this syndrome to the formation of perinatal mortality is often insufficiently recognized. Today, placental insufficiency is considered not only a factor that negatively affects the outcomes of pregnancy and childbirth but also an important biological mechanism that creates a prerequisite for the development of certain somatic and metabolic diseases in the child's later life. Therefore, the study of its etiopathogenesis, early diagnosis, and prevention is one of the priority areas of modern perinatology and obstetrics. Your opinion can be expressed scientifically as follows: Fetoplacental insufficiency (FPI) is divided into primary and secondary forms. Primary FPY develops in the early stages of pregnancy, and metabolic disorders, genetic factors, infections, and endocrine system diseases play an important role in its formation. These factors disrupt the processes of placentation, angiogenesis, vascularization, and the differentiation of chorionic villi. Secondary or late FPY occurs after 16 weeks of pregnancy and is associated with the influence of exogenous factors, insufficient functioning of compensatory-adaptive mechanisms, and involuntary-dystrophic changes occurring in the placenta [5]. In recent years, as a result of studying the molecular mechanisms of placental damage, the significance of ferroptosis and lipid peroxidation processes has been increasingly recognized. In this regard, determining the level of Glutathione Peroxidase 4 (GPX4) in the blood of pregnant women may allow for an assessment of the level of placental tissue resistance to oxidative stress and lipid peroxidation. Iron deficiency anemia (IDA) is the most common extragenital pathology in pregnancy. According to the World Health Organization (WHO) (2011), 38% (32.4 million women) of pregnant women aged 15–49 suffered from TMA. TTA in pregnancy occurs as a result of insufficient provision of iron during hematopoiesis, which is necessary to meet the increased demand of the mother's and fetal bodies. During pregnancy, iron

deficiency can lead to a number of complications: miscarriage, preeclampsia, premature detachment of the normally located placenta, and primary and secondary placental insufficiency[1]. When studying the placentas of women with iron deficiency anemia under a light microscope, Sharmistha Biswas et al. observed that the villi lacked complete trophoblast cover, increased intravillous and perivillous fibrin accumulation, an increase in syncytial nodules, and a decrease in the number of blood vessels compared to the control group. It was also found that in pregnant women with iron deficiency anemia, there was a decrease in the birth weight of the fetus, an increase in the weight and volume of the placenta[2]. Iron is present in the body in the form of Fe3+, and its transport and storage are largely controlled by transferrin receptors (TFR), ferritin (FTH), and ferroportin (FPN). Iron is transported from the maternal circulation to the placenta primarily in a transferrin-bound form (transferrin-bound iron, Tf-Fe^{3+}). Upon reaching the maternal-facing surface of the placenta, iron is taken up by trophoblast cells through transferrin receptor 1 (TfR1)-mediated endocytosis at the apical membrane. Within the endosome formed during endocytosis, Fe^{3+} is reduced to Fe^{2+} through a reduction process. Subsequently, Fe^{2+} is transported into the cytoplasm via divalent metal transporter 1 (DMT1), where it can either be stored or transferred for further utilization. Iron is then transported from trophoblast cells to the fetoplacental circulation. This process is mediated primarily by ferroportin (FPN), the major cellular iron export protein, which facilitates the efflux of Fe^{2+} from the cell. In the extracellular environment, Fe^{2+} is oxidized back to Fe^{3+} by ferroxidases and subsequently binds to fetal transferrin. Endothelial cells of the fetoplacental vasculature also express TfR1, enabling the uptake of iron and its delivery into the fetal bloodstream. TfR1 is mainly responsible for the cellular uptake of circulating Fe^{3+}, whereas ferroportin regulates the export of intracellular iron. In addition, when intracellular iron concentrations increase, ferritin heavy chain (FTH) can sequester excess iron and thereby limit its toxicity. Conversely, degradation of ferritin contributes to an increase in the intracellular labile iron pool. Elevated intracellular iron levels promote single-electron transfer reactions, in which Fe^{3+} is readily converted to Fe^{2+}. This process generates highly reactive and toxic hydroxyl radicals through Fenton-type reactions. The accumulation of reactive oxygen species (ROS) subsequently induces lipid peroxidation, ultimately leading to the initiation and progression of ferroptosis [3,6].

Ferroptosis is a form of regulated cell death characterized by iron-dependent lipid peroxidation within tissues[7]. As a result, impairment of cellular protein metabolism leads to insufficient production of energy and inadequate availability of essential micronutrients required for cell proliferation and differentiation. Consequently, the development of chorionic villi and angiogenic processes become impaired. Disruption of normal blood circulation within the trophoblast cells of the villi adversely affects placental maturation and contributes to premature placental aging. Such pathological alterations in placental tissue ultimately lead to the development of placental insufficiency [4]. Glutathione peroxidase 4 (GPX4) is one of the key antioxidant enzymes responsible for neutralizing lipid hydroperoxides. A decrease in GPX4 activity may promote the activation of ferroptotic pathways and result in placental cell injury. Therefore, reduced GPX4 levels may serve as an early biomarker preceding the development of primary fetoplacental insufficiency and may facilitate early risk prediction of this condition in pregnant women belonging to high-risk groups. However, the prognostic value of GPX4 requires further validation through large-scale clinical and experimental studies before its routine implementation in obstetric practice. In the present study, we developed an algorithm aimed at the early prevention of fetoplacental insufficiency during pregnancy. By determining serum ferritin and GPX4 levels in pregnant women, we sought to assess the severity of iron deficiency and evaluate the likelihood of iron-related metabolic disturbances occurring during placental development. In particular, these biomarkers may help predict whether disturbances in iron homeostasis could lead to the accumulation of free iron and the initiation of iron-dependent lipid peroxidation processes. Currently, laboratory testing combined with Doppler ultrasonography is considered one of the safest, non-invasive, and highly informative methods for evaluating the functional status and compensatory capacity of the fetoplacental complex. Doppler assessment allows detailed investigation of blood flow characteristics within the mother–placenta–fetus circulation system and enables the early detection of uteroplacental and fetoplacental hemodynamic disturbances. This technique can be applied from 14–16 weeks of gestation to predict the risk of placental insufficiency and serves as an important diagnostic tool for identifying pregnant women at high risk of adverse pregnancy outcomes associated with impaired placental blood flow. In addition, laboratory measurement of GPX4 and ferritin concentrations provides valuable information regarding the antioxidant defense capacity of placental cells during early pregnancy. Specifically, GPX4 reflects the resistance of placental tissues to lipid peroxidation, whereas ferritin serves as an indicator of systemic iron stores and iron homeostasis. The combined assessment of these biomarkers may therefore facilitate the early identification of oxidative stress, ferroptosis-related placental injury, and iron deficiency–associated abnormalities, thereby improving the prediction and prevention of fetoplacental insufficiency.

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RESEARCH MATERIALS AND METHODS:

A prospective study was conducted involving 141 pregnant women between 22 and 32 weeks of gestation who were at risk of preterm birth associated with fetoplacental insufficiency (FPI). The participants were divided into three groups. Group I (main group, n = 50) consisted of pregnant women at 22–32 weeks of gestation with a risk of preterm birth in the presence of FPI. Group II (comparison group, n = 51) included pregnant women of the same gestational age with a risk of preterm birth associated with FPI who received standard management. Group III (control group, n = 40) consisted of conditionally healthy pregnant women with uncomplicated pregnancies. Baseline clinical characteristics, the course of pregnancy, and obstetric outcomes were comprehensively analyzed in all study participants. The study protocol included clinical, laboratory, instrumental, morphological, and statistical assessment methods. Laboratory investigations comprised complete blood count analysis, coagulation profile testing, and determination of plasma levels of glutathione peroxidase-4 (GPX4) and ferritin. Instrumental evaluation was performed using ultrasonography (US) with Doppler assessment of the uteroplacental and fetoplacental circulation. In addition, placental tissue samples obtained after delivery were subjected to morphological examination. All collected data were analyzed using contemporary statistical methods to evaluate differences between the study groups and determine the clinical significance of the investigated parameters.

Results: When examining pregnant women at risk of preterm labor against the background of fetoplacental insufficiency (FPI) at 22–32 weeks of gestation, the average age of the women in the study groups ranged from 19 to 41 years. According to the analysis of the age composition of the examined pregnant women, the largest share was women aged 19–24, accounting for 45% of the total observation group. The proportion of pregnant women aged 25–29 was 26%, and those aged 30–34 was 16%. Women aged 35–41 were relatively rare, accounting for 13% of those examined. Thus, the majority of the women included in the study were young women of reproductive age. Analysis of the primary cohort of pregnant women by place of residence (n=141): rural residents accounted for 55%, while urban residents accounted for 45%. The gynecological history according to the data obtained during the anamnesis collection process is presented in Table 1.

Table 1. Distribution of examined women by reproductive history, n (%).

Indicators	Study groups, n (%)		
	1st main group (n=50).	2nd comparison group (n=51);	Control group 3 (n=40).
History of medical abortion	5 (10%)	4 (7,8)	-
Preterm labor	10 (20%)	9 (17,6 %)	20 (50%)
Induced labour	2 (4%)	1(2%)	-
History of preterm birth	13 (26%)	9 (37,2%)	-
History of FPY	16 (32%)	18 (35,2%)	-

- significance difference P < 0,05.

According to the table data, the severity of the reproductive history in Groups 1 and 2 was higher than in the control group. Particularly frequent were cases of preterm labor and premature labor in the anamnesis. It can be concluded that pregnant women with a history of preterm labor, fetoplacental insufficiency, and medical abortion require special attention, and it is advisable to conduct preventive treatment for them at an early stage. The proportion of preterm births was higher in the control group (50%) and amounted to 20% and 17.6% in the study and comparison groups, respectively. Medical abortion and induced delivery were recorded exclusively in the main and comparison groups, whereas no such cases were observed in the control group. Serum analysis of glutathione peroxidase-4 (GPX4) demonstrated a marked reduction in enzyme levels in both study groups compared to the control group. Mean GPX4 concentrations were 28.67±0.91 μmol/L in Group I and 30.28±0.90 μmol/L in Group II, whereas in the control group the corresponding value was 52.10±2.66 μmol/L. The observed differences were statistically significant ($p<0.01$; $p<0.05$), indicating a pronounced decrease in antioxidant defense capacity and enhanced oxidative stress in pregnant women with fetoplacental insufficiency complicated by the risk of preterm birth. Complete blood count analysis revealed lower hemoglobin and erythrocyte levels in the study groups compared to controls. Hemoglobin concentrations were 110±8 g/L in Group I and 112±7 g/L in Group II, whereas in the control group it reached 120±6 g/L. In contrast, leukocyte counts were elevated in both study groups ($12.5\pm2.0\times10^9$/L and $11.8\pm2.1\times10^9$/L) compared to the control group ($9.5\pm1.2\times10^9$/L), suggesting the presence of mild anemia and a systemic

inflammatory response in pregnancies complicated by fetoplacental dysfunction. The obtained results indicate the presence of mild anemia, inflammatory processes, and alterations in the hemostatic system among pregnant women with a risk of preterm birth. Analysis of serum ferritin levels revealed that the values in Group I and Group II were nearly identical, amounting to 46.03±0.19 ng/mL and 47.04±0.17 ng/mL, respectively. These values were significantly lower than those observed in the control group (116.17±6.01 ng/mL). The reduction in ferritin levels reflects depleted iron stores and impaired iron metabolism in the maternal organism. Such a condition represents an important pathogenic factor contributing to oxidative stress and metabolic disturbances in pregnancies complicated by fetoplacental insufficiency and risk of preterm delivery. A more than 2.5-fold decrease in ferritin levels compared to the control group was recorded in both study groups. Iron deficiency–associated anemia leads to impaired cellular metabolism and reduced oxygen delivery to fetal tissues. As a result, insufficient oxygenation and nutrient supply may negatively affect fetal development. This highlights the importance of optimizing uteroplacental circulation through the improvement of blood flow within the uterine spiral arteries and ensuring adequate oxygen delivery to all fetal and placental structures. From a therapeutic perspective, the selection of pharmacological agents with vasodilatory properties that are safe for use during pregnancy plays a crucial role. Such agents may contribute to the improvement of uteroplacental perfusion, reduction of hypoxic injury, and support of normal fetal development under conditions of fetoplacental insufficiency. When a pregnant woman presents, therapeutic and symptomatic management is initiated based on her clinical complaints, which include persistent lower abdominal pain, a dull cramping or pressure sensation, tachycardia, and increased uterine tone.

Therapeutic Approach and Outcomes

The proposed complex treatment protocol included the following components: timely hospitalization and clinical-anamnestic evaluation, diagnosis based on comprehensive clinical and laboratory investigations, and standard therapy in accordance with clinical guidelines. Standard treatment consisted of progesterone vaginal tablets (200 mg twice daily until 36 weeks of gestation), nifedipine (10 mg orally every 30 minutes up to a maximum single dose of 40 mg, followed by 10 mg every 8 hours within 48 hours under strict blood pressure monitoring), indomethacin (100 mg rectal suppository once daily for 5 days), iodine preparations (200 mg once daily), and Fersinol containing iron and ascorbic acid (1 capsule twice daily for 60 days). In addition to standard therapy, pathogenetically oriented treatment was administered, including selenium tablets (200 mg daily for 14–21 days) and L-arginine L-aspartate. In moderate and severe cases, L-arginine L-aspartate was administered intravenously (100 mL once daily for 5–7 days), followed by oral syrup administration (10 mL three times daily for 14–30 days). Patients were also managed under dynamic clinical observation. The addition of selenium and L-arginine L-aspartate to standard therapy contributed to an increase in glutathione peroxidase-4 (GPX4) levels, restoration of antioxidant defense mechanisms, and a reduction in ferroptotic damage. These effects were associated with improved functional status of the fetoplacental complex, stabilization of placental metabolic activity, prolongation of pregnancy, reduced incidence of preterm birth, and prevention of perinatal and neonatal complications. The duration of pregnancy prolongation in the main group averaged 28.4±6.2 days, which was significantly higher than in the comparison group (19.7±5.4 days; $p<0.05$). Following the implemented therapeutic interventions, delivery outcomes were evaluated as term and preterm births across the study groups.

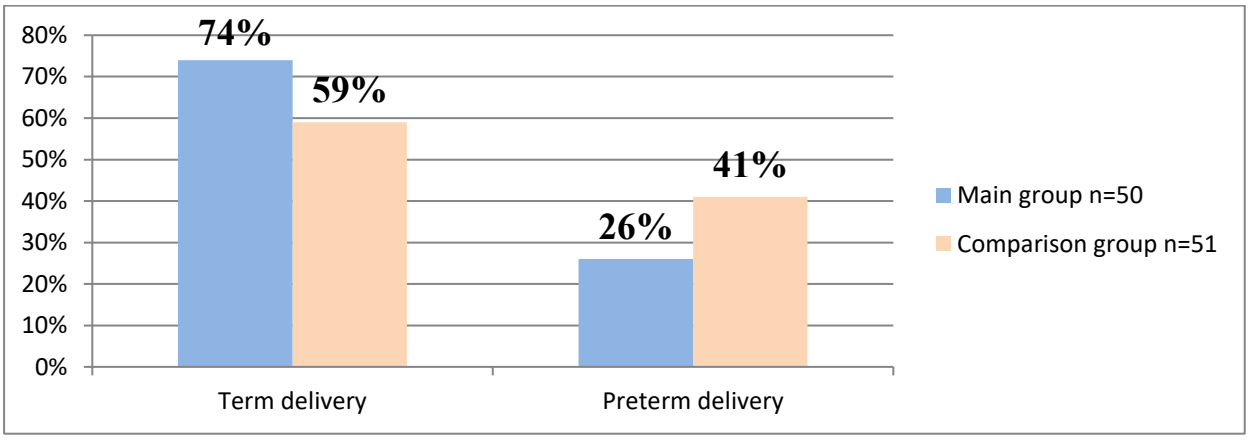

Figure 1. Distribution of deliveries according to the course of labour.

In Group I, the proportion of spontaneous vaginal deliveries was higher compared to Group II. In contrast, the rate of caesarean section in Group II was approximately 2.2 times higher than in Group I. These findings indicate a more favorable course of labor in Group I and a reduced need for operative delivery interventions.

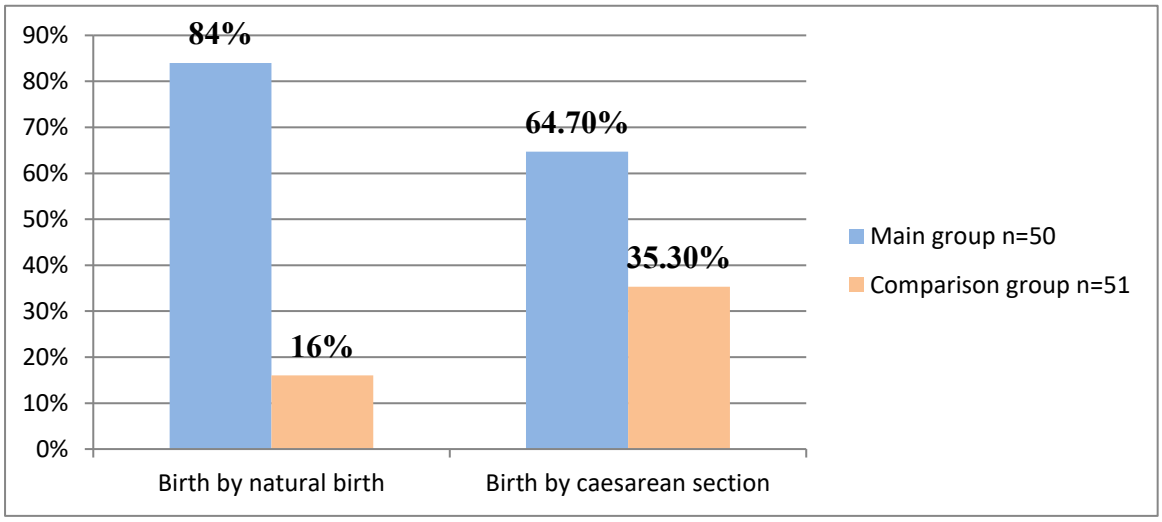

Figure 2. Distribution of delivery management methods

According to the analysis, neonatal anthropometric parameters in the main group were relatively satisfactory at birth. The mean birth weight in the main group was 3090±420 g, whereas in the comparison group it was 2820±390 g ($p<0.05$). The mean body length of newborns was 50.2±2.1 cm in the main group and 48.6±2.3 cm in the comparison group ($p<0.05$). These findings indicate more favorable perinatal outcomes in the main group. Morphological examination of placental tissues obtained after delivery revealed changes associated with impaired cytotrophoblast invasion in the early stages of pregnancy. This process is characterized by insufficient trophoblastic differentiation and proliferation, as well as disorders in the remodeling of the uterine spiral arteries. As a result, hemodynamic disturbances in uteroplacental circulation develop, leading to impaired microcirculation. These changes are manifested by the presence of ischemic foci, fibrinoid deposition, and thrombus formation within the placental tissue. Disruption of blood flow results in reduced perfusion of certain villi and contributes to hypoxic injury at the tissue level. Such pathological alterations are particularly common in placentas obtained from cases of preterm birth at 22–27 weeks of gestation and are closely associated with the development of retrochorial hematoma.

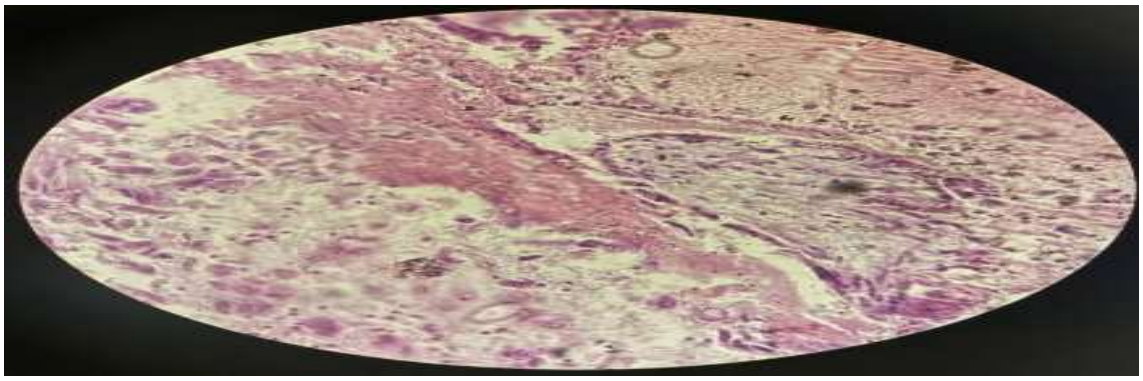

Figure 3. Dostiyorova M., 25 years old, № 11552, Group II. Diagnosis: Pregnancy V, 34 weeks; Delivery V, late preterm birth. Hematoxylin and eosin (H&E) staining, ×200 magnification.

The ferroptosis-associated morphological features of placental insufficiency are characterized by cytoplasmic eosinophilia, disruption of cell membrane integrity, and hemorrhagic extravasation within the intervillous spaces. These morphological alterations are closely associated with metabolic disturbances during angiogenesis and reflect impaired cellular and vascular

homeostasis within the placenta.

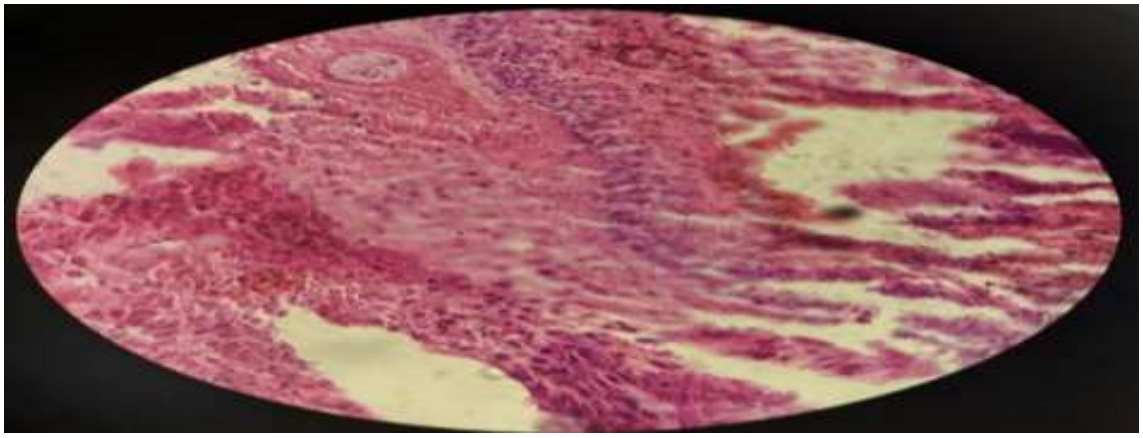

Figure 4. G'afurova G., 23 years old, No. 12609, Group I. Diagnosis: Pregnancy II, 34 weeks; Delivery II, late preterm birth. Hematoxylin and eosin (H&E) staining, ×200 magnification.

Histological examination of the placenta revealed structural alterations in chorionic villi. The villous stroma demonstrated cellular infiltration, interstitial edema, and vascular congestion. Accumulation of erythrocytes within the intervillous spaces and signs of microcirculatory disturbances were also observed. In some villi, degenerative changes of the trophoblastic layer and disruption of tissue architecture were identified. These morphological alterations indicate increased oxidative stress and enhanced cellular injury within the placental tissue. According to the study results, a decreased GPX4 marker level was observed in 74% of pregnant women in Group I and in 80% of those in Group II. In addition, fetoplacental insufficiency (FPI) was detected in 46% of cases in Group I (n=23) and in 51% of cases in Group II (n=26). After implementation of preventive and therapeutic interventions, a significant increase in GPX4 levels was recorded: 47.77±1.59 μmol/L in Group I and 34.74±0.76 μmol/L in Group II ($p<0.001$). These findings indicate that the proposed treatment approach is based on principles of early diagnosis, risk stratification, and timely correction of identified disturbances. The introduction of this complex therapeutic strategy into clinical practice may optimize patient management, improve the accuracy of perinatal outcome prediction, and reduce the incidence of preterm birth and related complications. The obtained results indicate that a decrease in GPX4 levels occurs significantly earlier than the clinical and ultrasonographic signs of fetoplacental insufficiency. Specifically, in 26% of pregnant women in Group I and 29% in Group II, GPX4 levels were below reference values, however, no clinical or ultrasound signs of FPI were detected at the time of examination. Further dynamic follow-up of pregnant women revealed that, although clinical and ultrasonographic signs of FPI were absent at initial presentation, fetoplacental insufficiency subsequently developed in 36% of 14 pregnant women with decreased GPX4 levels in Group I and in 60% of 15 pregnant women with decreased GPX4 levels in Group II during the course of complex therapy. These results confirm the high prognostic significance of the GPX4 marker. The frequency of fetoplacental insufficiency in the main group undergoing complex treatment was 24.3% lower than in the comparison group. Thus, a decrease in GPX4 levels is an important early and prognostic criterion for the development of ferroptosis and can be used as a promising biomarker for detecting fetoplacental insufficiency at the preclinical stage. A decrease in GPX4 levels reflects the development of ferroptosis through the weakening of antioxidant protection and increased lipid peroxidation.

DISCUSSION.

In recent years, the roles of oxidative stress, disturbances in iron metabolism, and ferroptosis mechanisms in the pathogenesis of fetoplacental insufficiency (FPI) and preterm birth have been increasingly recognized. The placenta is the principal organ responsible for fetal development, and its structural and functional impairment leads to intrauterine growth restriction, chronic hypoxia, and adverse perinatal outcomes. The results of the present study demonstrated a significant reduction in GPX4 levels in pregnancies complicated by the risk of preterm birth and fetoplacental insufficiency. GPX4 is one of the key antioxidant enzymes responsible for protecting cells against lipid peroxidation. A decrease in its activity promotes the accumulation of lipid hydroperoxides in cell membranes, thereby creating favorable conditions for the development of ferroptosis. In our study, significantly lower GPX4 levels in the main and comparison groups compared to

the control group indicate enhanced oxidative stress in placental tissues. One of the most important findings of this study is that reduced GPX4 levels were detected in many pregnant women prior to the clinical and ultrasonographic manifestations of fetoplacental insufficiency. Specifically, in 26% of women in Group I and 29% of women in Group II, GPX4 levels were below reference values, despite the absence of clinical or ultrasound signs of FPI at the time of examination. Further dynamic follow-up revealed that fetoplacental insufficiency subsequently developed in a proportion of these women, confirming the significance of GPX4 as an early prognostic biomarker. This finding suggests that molecular and metabolic alterations in the placenta begin significantly earlier than their clinical manifestations. During the study, it was established that ferritin levels were significantly reduced in both the study and comparison groups compared to the control group. Ferritin is a key indicator of iron reserves in the body, and its decrease indicates iron deficiency and impaired iron metabolism. Under conditions of iron deficiency, cellular energy metabolism is disrupted, tissue hypoxia develops, and oxidative stress intensifies. At the same time, impaired iron homeostasis can lead to the formation of free iron ions and activate lipid peroxidation reactions. As a result, the process of ferroptosis intensifies, and structural damage to trophoblast cells and chorionic villi occurs.

Results of morphological studies of the placenta also confirmed biochemical changes. In histological specimens, edema, cellular infiltration, microcirculatory disorders, hemorrhagic foci, degenerative changes in the trophoblast layer, and fibrinoid deposits were observed in the stroma of the chorionic villi. These changes may be associated with impaired blood circulation in the placenta, increased oxidative stress, and processes of ferroptotic damage. The results of the complex treatment are also noteworthy. The addition of selenium and L-arginine L-aspartate to standard therapy led to a significant increase in GPX4 levels. Selenium is an important component of the GPX4 enzyme, and its adequate supply enhances antioxidant protection. L-arginine stimulates nitrogen oxide synthesis, improves uteroplacental blood flow, and increases the oxygen supply to tissues. As a result, the duration of pregnancy extension increased in the main group, the frequency of preterm and surgical births decreased, and the anthropometric indicators of newborns were better. The results of the study show that determining GPX4 and ferritin levels at early stages of pregnancy allows for the assessment of the functional state of the fetoplacental complex, the selection of pregnant women in high-risk groups, and the timely initiation of individual preventive measures. In particular, a decrease in GPX4 levels can be considered an early sign of the development of ferroptosis. Therefore, the use of this biomarker in combination with Doppler and ultrasound studies can increase the effectiveness of detecting fetoplacental insufficiency at the preclinical stage. The results obtained show that iron metabolism disorders, oxidative stress, and ferroptosis play an important role in the pathogenesis of fetoplacental insufficiency. A decrease in GPX4 levels reflects a weakened antioxidant defense of placental cells and serves as an early and prognostically significant sign of the development of fetoplacental insufficiency. This makes it possible to use GPX4 in practical obstetrics as a promising biomarker for predicting the risk of preterm labor and fetoplacental insufficiency. The developed approach is recommended for implementation into the practical healthcare system to increase the effectiveness of preventive and therapeutic-diagnostic measures for pregnant women in high-risk groups.

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