

The tragic complications of preterm birth

Temirova Dilnoza Olimjonovna

Asian International University

Abstract: This article discusses the causes of preterm labor, the causes that lead to premature labor, the complications that result from it, and measures to treat them. Preterm labor is currently one of the most important and in many respects unresolved problems of obstetrics and gynecology. Although the mechanisms of development of preterm labor have been studied in depth for several years, little attention has been paid to studying the mechanisms of development of complications arising from them.

Keywords: Premature birth, resuscitation, neonatal jaundice, pregnancy, blood loss.

Relevance: Every year, more than 15 million babies are born very prematurely and very small worldwide, which corresponds to one baby being born prematurely every 116 minutes.

Complications related to preterm birth are the leading cause of death in children under five years of age, with approximately 900,000 global deaths observed in 2019. In our republic, the number of preterm births has increased by 15.4% in the last 3 years, with a total of 6,840 preterm births registered in 2019 and 10,873 in 2023, according to SIAT.

Termination of pregnancy between 22 and 37 weeks is called premature labor. Babies born at this time are considered premature. •15-16 weeks - early miscarriage •16-22 weeks - late miscarriage •22-37 weeks - preterm labor •37-42 weeks - full-term labor •After 42 weeks - postterm labor Many healthy babies are born prematurely.

However, in general, the earlier a baby is born, the more health problems they will have and the longer they will need to stay in the hospital. Recently, vascular and hemodynamic disorders in the mother, which are observed in various somatic diseases, have traditionally been associated with premature birth it depends on the risk factors for. There is a general dysfunction of the endothelium in the uteroplacental pool, which develops with hemodynamic and microcirculatory disorders, including preeclampsia and various somatic pathologies. It is necessary to determine the indicators of the anticoagulant potential of the blood of pregnant women who have given birth prematurely, in particular, the main It is very important to study the composition of the anticoagulant, antithrombin III. Women with a physiological course of pregnancy, which indicates the important role of antithrombin III deficiency in the development of severe pregnancy complications. Main part: Failure to carry a pregnancy to term - a gradual expulsion of the fetal egg from the uterus as a result of increased uterine contraction activity The fetus moves from its wall and exits its cavity. Taking into account the gestation period for the fetus, the specifics of obstetric tactics and consequences in premature birth, it is appropriate to distinguish 3 periods of such births: 1. Premature birth at 22-27 weeks (fetal weight 500-1000 gr.) accounts for 5% of the total number of births. They are mostly associated with isthmic-cervical insufficiency, occurs due to infection of the lower pole of the placenta and its premature rupture. In this group, the consequences of childbirth for the fetus are very bad, and perinatal morbidity and mortality are very high. 2. The causes of premature births occurring at 28-33 weeks (fetal weight 1000-1800g) are different from those of very premature births. Despite the immature fetal lungs, Their maturation can be achieved by using glucocorticoids or other drugs.

Etiology: 1. Genital infantilism 2. 3. 4. 5. 6. 7. 8. 9. Endocrine diseases (thyrotoxicosis, diabetes mellitus) Kidney, heart diseases Isthmic-cervical insufficiency Multiple gestation Hypertensive syndromes Maternal age ($x \leq 18$, $35 \geq x$) Placenta previa.

Incorrect fetal position 10. Intrauterine infections

Signs of premature birth in a baby: 1. 2. 3. 4. 5. 6. 7. 8. Weight 500-2500 g and height 25-45 cm Body hair is abundant (hair is short, sparse) Skin color is red (epidermis is not developed) The frenulum and sagittal slit are large Reflexes are weak (sucking, squeezing) Crying and screaming are weak Testicles are not descended in boys, labia are open in girls Umbilical cord is below normal

Complications. 1. Premature rupture of the fetal bladder. 2. 3. 4. Infection of the birth canal. Pain relief and fetal hypoxia. Rapid labor - less than 6 hours in primiparous women (trauma of the fetal head and birth canal), therefore, it is always interrupted.

Premature babies have a long-term severe

has a significant risk of adverse effects, resulting in a decrease in human resources worldwide. Long-term consequences include: Pathology of the visual organ: → Premature birth causes blindness or high myopia → Progressive hyperopia and myopia. Pathology of the auditory organ: → 5-10% of very premature infants have auditory pathology Chronic lung disease in premature infants: → Manifested in varying degrees is: when exercise tolerance decreases, supplemental oxygen is needed at home. Cardiovascular diseases and non-communicable diseases: } Hypertensive disease } Decreased lung function } Increased incidence of bronchial asthma } Growth failure in childhood } Excessive weight gain in adolescence.

After calculating the gestational age, the head circumference, length, and body weight of the preterm infant are measured. WHO recommendations for improving outcomes of preterm birth: The World Health Organization has recommended a number of measures to improve outcomes in preterm infants.

developed recommendations. In addition, there is a description of a number of therapeutic interventions that are used in pregnant women at risk of preterm birth. For newborns: → Kangaroo care → Surfactant administration → Oxygen therapy For women at risk of preterm birth: → Antenatal glucocorticoid therapy to improve neonatal outcomes → Tocolysis → Magnesium sulfate for fetal neuroprotection → Antibiotic therapy (with or without premature rupture of membranes) → Optimal method of preterm delivery

Obstetrics and gynecological history (very young mother, not physically ready for motherhood or older mothers, history of premature births, frequent spontaneous abortions, multiple pregnancies, complicated obstetric history), extragenital diseases (diabetes mellitus, pyelonephritis, myopia, varicose veins of peripheral blood vessels, obesity, bronchial asthma, pneumonia, urogenital diseases, viral diseases), pregnancy complications (gestational hypertension, eclampsia, preeclampsia, placenta previa, placental abruption, fetoplacental insufficiency, excessive or excessive amniotic fluid)

(excessive prematurity, breech presentation, cervical insufficiency, uterine malformation). Fetoplacental insufficiency is a complex of morphofunctional diseases of the placenta that develop from the fetus and placenta due to various extragenital and gynecological pathologies, as well as complications of pregnancy. Primary insufficiency occurs in the early stages of pregnancy (16-18 weeks), during the formation of the placenta and during organogenesis under the influence of infectious viral, bacterial diseases, endocrine, iatrogenic factors. Secondary fetoplacental insufficiency occurs when the placenta is initially formed normally with, develops under the influence of maternal factors or pregnancy complications. Perinatal morbidity and perinatal fetal death Complications caused by fetoplacental insufficiency include intrauterine development delay syndrome, acute and chronic fetal hypoxia, premature aging of the placenta, and underdeveloped pregnancy. Early FPY, which occurs with a violation of the placentation process and leads to underdevelopment of the placenta, is one of the causes of premature birth. Therefore, it is currently urgent to develop modern diagnostic methods that allow detecting pathological changes in the fetoplacental complex at the initial, preclinical stage of the disease and introduce them into clinical practice.

The basic concept of neurogenic regulation of protective-adaptive mechanisms that ensure homeostasis in the maternal-placental system is of undoubted interest. Fetoplacental insufficiency remains one of the urgent problems of modern obstetrics to this day. These include intrauterine growth retardation and

acute and chronic fetal hypoxia. One of the main methods for diagnosing fetoplacental insufficiency is ultrasound Dopplerometry, which determines the parameters of blood flow in the uterine arteries, umbilical artery and middle cerebral artery of the fetus. Indicators of dysfunction in the maternal-placental-fetal system are also: a decrease in the size of the fetal head, abdomen, limbs, according to ultrasound fetometry, the thickness and degree of maturity of the placenta do not correspond to the gestational age.

Despite the existence of various treatment regimens for FPY at different stages of pregnancy, the search for more effective methods of treating and preventing this pathology continues. Pharmacotherapy for FPY includes the following groups of drugs: drugs that promote relaxation of the uterine muscles (β -adrenomimetics, spasmolytics, calcium channel blockers); drugs that improve microcirculation and rheological properties of blood (platelet aggregation inhibitors, angioprotectors, anticoagulants); drugs that correct metabolic disorders (amino acids, protein compounds); drugs that increase the resistance of brain and fetal tissues to hypoxia (antihypoxants, nootropics). It is known that periodic or prolonged increases in myometrial tone lead to impaired blood circulation in the fetoplacental space due to a decrease in venous outflow.

In this regard, in patients with increased uterine contractile activity, FPY complex therapy should include β -adrenomimetics, which have a tocolytic effect and improve uteroplacental blood flow in small doses by reducing vascular resistance at the arteriolar level. In order to avoid the negative effects of β -adrenomimetics on the cardiovascular system, it is advisable to combine their administration with cardiotonic drugs. At the same time, there is evidence that the use of low-dose aspirin is effective in preventing FPY and preeclampsia in women with impaired uteroplacental blood flow. Therefore, in combination with long-term antithrombotic therapy (Clexane), it often leads to an improvement in the performance of the fetoplacental system. It is possible to use Piracetam from nootropics, Riboxin, Dipyridamole from metabolic enhancers, Sermin, Tivortin, Actovegin from amino acids, B-group vitamin complexes, Nifedipine, Verapamil from tocolytics, Magnesium sulfate, papaverine from spasmolytics.

Summary

A person can take the following steps to reduce the risk of other risk factors: - be aware of the signs of preterm labor and plan to contact a doctor or go to the hospital immediately if symptoms appear; - attend all prenatal appointments, check-ups and scans, even if your pregnancy is going well; - maintain a healthy weight before and during pregnancy; - try to reduce stress during pregnancy, for example by using relaxation techniques; - leave at least 18 months between pregnancies.

References:

1. Temirova, D. O. (2024). Diagnosis of Cervical Erosion. *American Journal of Bioscience and Clinical Integrity*, 1(11), 84-89.
2. Темирова, Д. А. (2024). СОВРЕМЕННЫЕ МЕТОДЫ ЛЕЧЕНИЯ СИНДРОМА АШЕРМАНА. *Modern education and development*, 16(10), 132-142.
3. Темирова, Д. О. (2024). КЛИНИЧЕСКОЕ ЗНАЧЕНИЕ МИОМЫ МАТКИ В ГИНЕКОЛОГИИ. *Modern education and development*, 16(10), 116-131.
4. Olimjonovna, T. D. (2024). THE SYNDROME OF UNFORTUNATE CONSEQUENCES HELPPA. *Modern education and development*, 16(10), 156-166.
5. Olimjonovna, T. D. (2024). UTERINE PROLAPSE IS A DELICATE PROBLEM FOR WOMEN. *Modern education and development*, 16(10), 167-176.
6. Olimjonovna, T. D. (2024). BACTERIAL VAGINOSIS IS A DANGEROUS DISEASE. *Modern education and development*, 16(10), 143-155.
7. Temirova, D. (2024). ADENOMYOSIS AND DISORDERS OF REPRODUCTIVE FUNCTION. *European Journal of Modern Medicine and Practice*, 4(10), 195-199.

8. Темирова, Д. О., & Мухитдинова, Х. С. (2025). РАЗРЫВ МАТКИ–СЕРЬЕЗНОЕ ОСЛОЖНЕНИЕ В АКУШЕРСТВЕ. *Modern education and development*, 19(2), 365-374.
9. Мухитдинова, Х. С., & Темирова, Д. О. (2025). КЛИНИЧЕСКОЕ ФАКТОРЫ СТРОЕНИЕ СПЕРМАТОЗОИДОВ ПРИ МУЖСКОГО БЕСПЛОДИЯ. *Modern education and development*, 19(2), 416-426.
10. Мухитдинова, Х. С., & Темирова, Д. О. (2025). ОСОБЕННОСТИ ПАТОЛОГИЯ ЯИЧНИКОВ В СТРУКТУРЕ ГИНЕКОЛОГИЧЕСКОЙ ЗАБОЛЕВАЕМОСТИ. *Modern education and development*, 19(2), 450-463.
11. Темирова, Д. О., & Мухитдинова, Х. С. (2025). ВНЕМАТОЧНАЯ БЕРЕМЕННОСТЬ–ЗАБОЛЕВАНИЕ, ТРЕБУЮЩЕЕ НЕОТЛОЖНОЙ ПОМОЩИ. *Modern education and development*, 19(2), 342-354.
12. Темирова, Д. О., & Мухитдинова, Х. С. (2025). МОРФОФУНКЦИОНАЛЬНЫЕ ОСОБЕННОСТИ ТРИХОМОНИАЗА. *Modern education and development*, 19(2), 355-364.
13. Темирова, Д. О., & Мухитдинова, Х. С. (2025). ПРЕЖДЕВРЕМЕННАЯ ОТСЛОЙКА ПЛАЦЕНТЫ. *Modern education and development*, 19(2), 316-327.
14. Темирова, Д. О., & Мухитдинова, Х. С. (2025). СПКЯ-ОДНА ИЗ ПРИЧИН БЕСПЛОДИЯ. *Modern education and development*, 19(2), 328-341.