EXCELLENCIA
INTERNATIONAL MULTI-DISCIPLINARY JOURNAL OF EDUCATION
Volume 02, Issue 05, 2024 ISSN (E): 2994-9521

Analysis of Current Research on the Occurrence, Development and Complications of Preeclampsia

Adizova Sarvinoz Rizokulovna [1]

[1] Bukhara State Medical Institute named after Abu Ali ibn Sina, Department of Obstetrics and Gynecology No. 2, Republic of Uzbekistan, Bukhara region

Abstract:

In this article, the occurrence of preeclampsia, which is one of the leading causes of illness and death of mothers and children, and the impact of neurogenic, hormonal, immunological, placental, genetic factors on the pregnant organism, perinatal complications and perinatal death as a cause of premature birth and blood circulation disorders in the fetoplacental system are discussed. An analysis of modern studies on the complications of preeclampsia is presented.

Keywords: preeclampsia, endothelial dysfunction, perinatal complications, trophoblast.

Preeclampsia, which is one of the leading causes of morbidity and mortality of mothers and children, remains an urgent problem even in the 21st century. In different literature, the complication of pregnancy with preeclampsia is given in different percentages, it is 8-26% [2,15,21]. In developed countries, 6-10% of all pregnancies are complicated by preeclampsia, while in developing countries, the disease has even higher rates. According to the data provided by the World Health Organization (WHO), preeclampsia ranks second among the causes of maternal death and accounts for 11.8-14.8%, while preeclampsia is the main cause of perinatal morbidity and mortality [4,17,24]. Every year, 300-500 thousand babies born to pregnant women with preeclampsia die.

Preeclampsia is a multifactorial pathology with polyorgan disorders that is very dangerous for the life of the mother and the fetus, which in turn is an inducer of premature birth, one of the reasons for antenatal fetal death and fetal growth retardation, and in many cases it is a pathology that causes massive bleeding, purulent-septic complications. [13,22, 31].

As a cause of perinatal complications and perinatal death in pregnant women with preeclampsia (PE), premature birth and blood circulation disorders in the fetoplacental system take the main place [17,19].

According to many researchers, endothelial dysfunction is the main factor in the development of preeclampsia. It can be concluded that the development of PE is the result of the combined effect of a number of factors (neurogenic, hormonal, immunological, placental, genetic) on the pregnant organism. In the development of preeclampsia, the disruption of adaptive mechanisms in the body associated with changes in the reactivity of the central nervous system is also important [11,16,28].

Preeclampsia is a clinical manifestation of the inability of the mother's body's adaptation mechanisms to adequately meet the needs of the developing fetus. This pathological condition is etiologically characterized by immunological and autoimmune disorders. The pathogenesis of preeclampsia occurs as a systemic inflammatory response syndrome, and oxidative stress and endothelial dysfunction are of particular importance [10,14,25].

Pregnant women with preeclampsia have disorders in the immune system that prevents the mother's body from recognizing the fetoplacental unit. Immune antibodies produced in large quantities cause the secretion of tumor necrosis factor, which in turn causes apoptosis of cytotrophoblast cells. PE, in turn, decreases the amount of HLA-G and HLA-E. In physiological pregnancy, this cell-trophoblast interaction is associated with natural killer cell secretion of vascular endothelial growth factor and placental growth factor [9,34].

At 8 weeks of pregnancy, trophoblast cells migrate from the placenta to the maternal tissue and the uterine artery. In physiological pregnancy, endovascular cells of the trophoblast contribute to the remodeling of the spiral arteries. For normal placentation, trophoblast cells must be activated by complement and protected from immune responses [12;23].

Immunogenetic predisposition to the development of PE is manifested in the early stages of pregnancy, the loss of tolerance of the mother to the fetus disrupts placental processes. Immunogenetic disorders deepen aseptic inflammatory processes and this, in turn, causes worsening of endothelial dysfunction in all blood vessels of the mother and aggravation of preeclampsia [7,26,30]. As a result of endothelial dysfunction developed in preeclampsia due to various pathogenetic mechanisms, the balance of control of angiogenesis is disturbed. As a result, metabolic hypoxia develops and leads to the initiation of a complex of molecular changes, which causes the active release of oxygen by special organelles of mitochondrial cells. However, the mechanism of development of endothelial dysfunction is still not fully understood. Direct contact of trophoblast cells with maternal blood, as well as its transformation into the form of endothelial cells, is an important feature of human placentation.

Endothelial transformation of pre-programmed non-endothelial cells is essential for successful trophoblast engraftment into the uterine vasculature [1,14,23].

General spasm of vessels, hypovolemia, and microcirculation disturbances, which cause organ and tissue hypoperfusion, play a decisive role in the pathogenesis of the early preclinical stage of PE. Disturbances of blood flow in the renal vessels and its branches lead to ischemia of the renal cortex. Disorders of spiral artery remodeling lead to fetal death, preeclampsia, and fetal growth retardation. Severe preeclampsia is characterized by endothelial dysfunction, impaired oxygen supply as a result of impaired placental blood supply [21; 32;].

Women with mild preeclampsia have a 3.6 times higher risk of developing arterial hypertension in the future, and 6 times more in women with severe preeclampsia [6;18]. If a woman had two previous pregnancies on the background of preeclampsia, then this woman's risk of developing eclampsia in the future is 6 times higher. In women with preeclampsia, the risk of thromboembolism is also 1.5-1.9 times higher according to the severity of preeclampsia. Women with preeclampsia have a higher risk of cardiovascular disease, metabolic disorders, acute cerebrovascular accident, and sudden death than other women.

Endothelial dysfunction of pregnant women complicated by preeclampsia persists in macrocirculation vessels for 2 years and in microcirculation vessels for 5 years after delivery [87; pp. 409-419]. Today, the only treatment for preeclampsia is delivery of the fetus and placenta. Taking into account the severity of preeclampsia, premature birth is performed in order to reduce maternal morbidity and mortality [10,16;27].

The goal of the entire Health Care Community is to deliver a mature child who does not need intensive care and can adapt to the environment. Usually, low birth weight babies are born due to preeclampsia. Perinatal mortality rates are always higher in preterm infants compared to term infants [1,12;26].

A population-based study of the WHO Perinatal Database Registry examined mortality statistics for preterm infants (33–36 weeks, n=6391). The authors found that neonatal mortality (deaths among children chronologically 0–27 days old) and infant mortality (deaths among children chronologically 0–3It shows that among infants born prematurely and whose mothers had preeclampsia, the rate of neonatal mortality specific to the period of pregnancy increased by 6-8.5 times compared to infants born on time. Babies born between 34 and 36 weeks are considered to be at low risk of serious illness [3;18,35]. But when births after 34 weeks of gestation were studied, there was a lack of care in terms of neonatal outcomes. It was found that babies born in these periods are expected to have a lot of respiratory diseases. For example, premature infants have a higher incidence of respiratory distress syndrome, transient tachypnea of the newborn, persistent pulmonary hypertension, and respiratory failure than term infants [24;31,].

According to data, respiratory distress syndrome is nine times more common in premature babies than in full-term babies. Severe preeclampsia is a significant risk factor for fetal death, with a stillbirth rate of 21 per 1,000. But in cases of mild preeclampsia, the risk of perinatal death is less than 50% (stillbirth rate 9 per 1000) than in pregnancies with severe preeclampsia. The growth of the fetus in accordance with the period of gestation is a sign of the satisfactory condition of the fetus. In children of pregnant women with preeclampsia, fetal growth restriction in the uterus is one of the factors of perinatal death [5; 20, 33].

In preeclampsia, the blood circulation in the fetoplacental system is disturbed, the blood flow decreases and ischemia is observed, as a result of which the growth of the fetus in the uterus is limited. Data show that at any gestational age, an infant weighing less than 10 percent of body weight at birth significantly increases the risk of death. The body weight of a baby born to a mother with severe preeclampsia is 12% lower than expected, and the body weight in a pregnancy with mild preeclampsia does not differ significantly from the norm.

However, some evidence suggests that children born to mothers with preeclampsia have lower growth rates at 24 months than children born to mothers without preeclampsia (r=0.04) [13;22]. Children with preeclampsia during pregnancy also have delayed development in the nervous system.

These studies emphasize the notion that the physiologically immature fetus is highly susceptible to intrauterine and placental blood flow abnormalities and may cause significant changes in fetal development that may increase the risk of maternal and fetal morbidity in the postpartum period. Based on the results of scientific research, preeclampsia observed in the mother is a risk factor for kidney diseases [12,23,29].

Several years of research in Norway have shown that low birth weight children born to women with preeclampsia during pregnancy have a significantly higher risk of cardiovascular disease and kidney disease. In addition, the literature shows that children born to mothers with preeclampsia during pregnancy have a higher risk of developing diabetes [17;22,35].

In the prevention and treatment of preeclampsia, it is necessary to take into account the presence of two phenotypes, which are early and late preeclampsia. Early preeclampsia is caused by a high degree of violation of trophoblast invasion into the spiral arteries of the uterus, deficiency of invasion waves, changes in the development of angiogenesis and placentation in the early stages of pregnancy. Complications of early preeclampsia include fetal growth restriction syndrome, premature birth, and antenatal death of the fetus.

Perinatal complications are common in early PE. Late PE is characterized by partial disruption of trophoblast development processes, insufficient improvement of spiral arteries, but compensation of morphological changes [13,18;24]. Often, placental insufficiency, metabolic diseases, and excessive activation of hemostasis are accompanied by a decrease in blood cells, i.e. platelets.

In the scientific literature, it is possible to find information that endothelial dysfunction, which leads to a violation of vascular permeability and a systemic inflammatory response, serves as the most important component of the pathogenesis of PE. As a result of the formation of a systemic inflammatory response, the functional activity of leukocytes changes, the migration of cells to the inflammatory zone is stimulated. As a result of the above mechanisms, the most severe complications of preeclampsia can be eclampsia, HELLP syndrome, and premature placental abruption.

In addition, preeclampsia has been shown to cause progressive placental insufficiency, fetal distress, fetal growth restriction syndrome, and in severe cases, antenatal fetal death [8,18,29].

It should be noted that the presence of preeclampsia does not always cause the development of complications, a relatively mild course of the disease is also observed. Therefore, the presence of factors or their combination that cause obstetric and perinatal complications can be estimated through the analysis of certain indicators [10;20,27].

Thus, PE occupies an important place among the problems in the health care system due to one or more of the various pathogenetic processes and causes specific, diverse and at the same time severe complications. The prevalence of the problem and the analysis of the scientific literature specific to the pathogenesis show that it is still one of the urgent issues that is waiting for its solution. Timely diagnosis of PE, prediction of its complications and development of measures aimed at preventing its aggravation is considered one of the important tasks of modern science and researchers.

References:

1. Абдурахманова Д. Н., Мадаминова М. Ш., Садуллаева А. Ф. Преэклампсия - актуальная проблема в современном акушерстве// International scientific review. 2016. - № 5 (15). – С. 111-113.
2. Абдурахманова Р.А., Омаров Н.С.М., Омарова П.М. Сравнительная характеристика течения и ведения беременности, осложненной гестозом по разным классификациям//Амбулаторно-поликлиническая помощь – в эпицентре женского здоровья Тез. докладов Юбилейного Всерос. Конгресса с межд. участием. Под ред. Г.Т. Сухих, В.Н. Прилепской. 2017. - С. 3-5.
3. Абдуллаева Дилноза Кузибаевна. "Допплерометрические показатели маточно-плацентарно-плодового кровотока у беременных с преэклампсией"// Вестник ТМА, №. 7 (10). 2016. – С. 49-51.
4. Айламазян Э.К., Репина М.А. Материнская смертность вследствие эклампсии - чему можно научиться? //Журнал акушерства и женских болезней. - 2013. - Т. 62. № 3. - С.3-8.
5. Адизова С. Р., Ихтиярова Г. А. Морфологическая характеристика плаценты у женщин с преэклампсией //Новый день в медицине. – 2020. – №. 2. – С. 26-30.

6. Адизова С. (2023). Biokimyoviy markyorlarning preeklampsiya istiqbolini belgilashdagi o‘rni. Journal of Experimental Studies, 1(11), 34–41.
7. Адизова, С., Иброхимова, Д., & Ихтиярова, Г. (2023). Peculiarities of hemostasis in pregnant women with preeclampsia. Перспективы развития медицины, 1(1), 25.
8. Буштырева И.О., Курочка М.П., Гайда О.В. Прогностические критерии преэклампсии.// Российский вестник акушера-гинеколога 2017; 17(2). – С. 59–63
9. Говсеев Д.А. Акушерские и перинатальные аспекты повторной преэклампсии//Здоровье женщины. - 2015. - № 10 (106). - С. 163.
10. Гурьев Д.Л., Охапкин М.Б., Карпов Н.Ю., Блинов Д.В. Течение и исходы беременности, осложненной преэклампсией, в зависимости от типа центральной материнской гемодинамики//Акушерство и гинекология.- 2011. - № 7-2. - С. 14-19.
11. Дубровина, С. О. Раннее прогнозирование преэклампсии (обзор литературы) // Проблемы репродукции. – 2018. - №24(3). – С. 67-73.
12. Камилова М. Я, Рахматуллоева Д.М., Давлятова Г.К., Мулкамонова Л.Н., Ишан-Ходжаева Ф.Р. Ранние и поздние преэклампсии: течение беременности, родов и перинатальные исходы//Вестник Авиценны. - 2021. - № 4 (69). – С. 34-38.
13. Малдыбаева Э.К., Долгая Г.В., Турдиева А.С., Сарымсакова Т.А. Ретроспективный анализ перинатальных исходов у женщин с преждевременными родами //Вестник КРСУ. - 2015. – Т. 15. № 4. – С.67-69.
14. Меркушкина Т.И., Тюрина Е.П. Течение и исходы беременности у женщин с преэклампсией//Научная мысль.- 2018. - № 2-4. - С. 46-53.
15. Сексенова А.Б., Нургалиева Л.И., Кистаубаева Л.Т., Мырзабаева Н.Н. Преэклампсия тяжелой степени: возможности ранней диагностики в амбулаторных условиях // Вестник КазНМУ. – 2022. – №1. – С.56-59
16. Слободчикова Т.С., Тайжанова Д.Ж., Амирбекова Ж.Т. Биохимические маркеры ранней диагностики преэклампсии // Вестник КазНМУ. 2019. №4. - С. 9-13.
17. Туксанова Д.И., Шарипова М. Анализ комплексного обследования женщин с преэклампсией и сочетанной анемией// Проблемы медицины и биологии. – Кемерово 2018. - №4. - С. 195-198
18. Шериф В., Бабенко О.М. Сравнительная характеристика анамнеза, течения беременности и родов у женщин с преэклампсией и гестационной гипертензией//Новости медицины и фармации. - 2021. - № 2 (381). - С. 34-35.
19. Agarwal S, Kovilam O, Agrawal DK. Vitamin D and its impact on maternal-fetal outcomes in pregnancy: A critical review.// Crit Rev Food Sci Nutr. 2018 Mar 24;58(5):-P. 755-769
20. Aita K. et al. Acute and transient podocyte loss and proteinuria in preeclampsia // Nephron. Clin. Pract, 2009; Vol. 112, N 2. -P. 65-70.
21. Adizova S.R., Ashurova N.G. IZUChENIE EFFEKTIVNOSTI I PRIEMLEMOSTI KONTRATsEPTIVNYKh SREDSTV U MNOGOROZhAVShIKh ZhENShchIN // Молодежный инновационный вестник. - 2016. - Vol. 5. - N. 1. - P. 193-194.
22. Adizova S.R., Ixtiyarova G.A. Prichini endotelial'noy disfunksii pri gipertenzivnix rasstroystv u beremennix. // Tibbietda yangi kun, 3(35/1)2021 S. S.214-217
23. Adizova S.R. Diagnosing the severity of preeclampsia // New day in Medicine 12 (62) 2023; P 28-33.

24. Adizova Sarvinoz Rizokulovna. (2023). MODERN ASPECTS OF DIAGNOSING THE SEVERITY AND COMPLICATIONS OF PREECLAMPSIA. Oriental Journal of Academic and Multidisciplinary Research , 1(3), 4-10.
25. Barton J. R., Brown M.A., Magee M.A. et al. Hypertensive Disorders of Pregnancy ISSHP Classification, Diagnosis, and Management Recommendations for International Practice. // Hypertension. 2018; -P. 72:24–43.
26. Dиaz Serrano NC, Gamboa-Delgado EM, Domнnguez-Urrego CL, et al .Vitamina D y riesgo de preeclampsia: revisión sistemática y metaanálisis // Biomédica 2018;-P. 43-53
27. Gerhard I. Prognostik value of hormonedetermination in the first trimester of preg_nancy //Acta endocrinol. –2013. – Vol. 103, № 256. – P. 158–160.
28. Grimes S., Ombay K., Lanes A., Walker M., Corsi D. J. Potential biological therapies for severe preeclampsia: A systematic review and meta-analysis //BMC pregnancy and childbirth. – 2019. – V. 19. – №. 1. – P. 1-12.
29. Heikura U, Hartikainen A.L., Nordström T. Maternal hypertensive disorders during pregnancy and mild cognitive limitations in the offspring.// Paediatr Perinat Epidemiol. 2013 Mar; 27 (2): 188-98.
30. Huppertz B. Maternal and fetal factors and placentation: implications for pre-eclampsia.//Pregnancy Hypertens. 2014 Jul;4(3).- P. 244-249.
31. Khalil A., Cowans N.J., Spencer K., Goichman S., Meiri H., Harrington K. First trimester maternal serum placental protein 13 for the prediction of preeclampsia in women with a priori high risk. //Prenat. Diagn. 2019; 29(8): -P. 781–9.
32. Ree P.H., Hahn W.B., Chang S.W., Jung S.H., Kang J.H., Cha D.H. et al. Early detection of preeclampsia using inhibin and other second-trimester serum markers. //Fetal. Diagn. Ther. 2018; -P. 29:
33. Rizokulovna , A. S. (2022). Blood Homocysteine Level and its Prognostic Value in Pregnant Women with Preeclampsia. Central Asian Journal of Medical and Natural Science, 3(5), 51-57.
34. Rizokulovna, A. S. (2023). Pathogenic Aspects of the Development of Preeclampsia. Miasto Przyszłości, 38, 1–7.
35. Roberts D.J. The placenta in pre–eclamp_sia and intrauterine growth restriction /M.D.Post //J. Clin. Pathol.– 2018. – V. 61 (12).– P. 1254–60.