WEB OF SCIENTIST: INTERNATIONAL SCIENTIFIC RESEARCH JOURNAL
ISSN: 2776-0979, Volume 3, Issue 7, July, 2022

FEATURES OF MATERNAL AND FETAL CIRCULATORY SYSTEM AND THEIR ROLE IN THE DEVELOPMENT OF PREECLAMPSIA (OVERVIEW)

Akhmedov F. K.
Bukhara State Medical Institute, Uzbekistan

Resume
In preeclampsia, blood flow disorders in the spiral arteries occur earlier than in the uterine arteries. It should be noted that the uterine vascular pool is an integral part of the blood circulation system of the whole body, and the disruption of blood flow in the uterine vessels during pregnancy leads to systemic circulatory disorders. Thus, the results of the analysis of modern research data showed that, despite the many diagnostic methods available today, the problem of determining the most effective method that allows reliable assessment of the severity and prognosis of the development of preeclampsia remains relevant.

Keywords: preeclampsia, placentation, mother-placental-fetal system, hemodynamics

Introduction

Hypertensive disorders during pregnancy - chronic hypertension, gestational hypertension, and preeclampsia - present a unique challenge because pathology and its therapeutic treatment simultaneously affect the mother and fetus, sometimes conflicting their well-being. In particular, preeclampsia is one of the worst complications of pregnancy. Preeclampsia, often presenting with first-onset hypertension and proteinuria in the third trimester, can rapidly progress to serious complications, including death of both the mother and the fetus [1,3,7].

Although the exact mechanisms remain unclear, placental ischemia and hypoxia have been implicated as the main causative factor in the pathophysiology of PE [16]. For PE, a two-stage disorder model was proposed [17]. The first stage is characterized by decreased placental perfusion secondary to abnormal implantation and development of the placental vasculature. The second stage is described by the release of harmful factors from the placenta into the mother's bloodstream, which causes widespread endothelial dysfunction, which, in turn, leads to the clinical symptoms of PE (preeclampsia). For PE to appear, both stages must occur. In addition, they interact with maternal constitutional factors (genetic, behavioral, or environmental), which

leads to different subtypes of PE [9]. Recent research has revealed glimpses of pathways that play a role in one or both stages.

The problem of studying the genetic mechanisms of susceptibility to multifactorial diseases (MDD) remains one of the most urgent in human genetics. The importance of this issue is determined by the enormous importance of polygenic diseases for modern medicine. One of the directions for understanding the role of genetic markers in the development of MDs is an evolutionary approach to the study of their genetic architecture [18, 26].

Risk factors for preeclampsia include obesity, insulin resistance, and hyperlipidemia, which promote the release of inflammatory cytokines and oxidative stress that cause endothelial dysfunction [7,13]. However, all the clinical manifestations that correspond to the development of preeclampsia are still not clear. Impaired trophoblast invasion and remodeling of uteroplacental arteries in preeclampsia exacerbates reactive oxygen species, hypoxia, and endothelial dysfunction in PE [15,21].

Over the past few decades, extensive research has been conducted to determine the etiology of preeclampsia as a pregnancy pathology. However, this syndrome remains as hypothesized fifty years ago. Even now, the etiology and true origin of the syndrome leading to clinical symptoms of preeclampsia are not clear.

With the beginning of pregnancy, the hemodynamics of the mother and its structural systems undergo a number of physiological changes, which help the development of the fetus and the preparation of the body for childbirth. From 6-8 weeks of pregnancy to 30 weeks of pregnancy, the total volume of blood circulation increases, which is directly related to the size of the fetus. At the same time, plasma volume increases to a maximum (up to 40-50 percent), compared to the same elements (up to 20-30 percent), that is, with a relative decrease in hemoglobin and hematocrit content, hypervolemic hemodulation occurs, which improves metabolism and gas exchange between the mother and the fetus. Improves[1,3,11]. The total volume of erythrocyte cells circulating in the body of a pregnant woman increases by an average of 16%, the volume of blood for oxygen increases, as a result of which the need for oxygen is not felt in the mother and the fetus[22,26].

Pregnancy increases the demands on the circulatory system, but at the same time it also includes the mechanisms to meet them. During the physiological period of pregnancy, total peripheral vascular resistance (UPTQ) gradually decreases and reaches 979-987 dyn.sec.cm-5 at 14-24 weeks of pregnancy, with the additional opening of capillaries that did not work before and a decrease in the tone of other

peripheral vessels, which increases the blood vessel volume in them. increases[6,12,16,20].

With an increase in the volume of circulating blood, an increase in the contractility of the myocardium is observed. An increase in the ejection fraction of the heart (by 8-10 weeks of pregnancy it increases by 30-40% due to stroke volume and the number of heart contractions also accelerates), left ventricular myocardial mass, left heart lobe and other parameters also increase slightly. Thus, it is noted in the studies that left ventricular enlargement without hypertrophy of the myocardium can be considered as a comparative diagnostic criterion between chronic arterial hypertension and arterial hypertension caused by pregnancy [4,24].

Hemodynamic processes in the mother-placental-fetus system are one of the important factors in ensuring the physiological course of pregnancy, the growth and development of the fetus[10,21].

Complications with preeclampsia during pregnancy lead to a decrease in the volume fraction, as a result of which the functional reserve of blood circulation, blood flow of the uterine arteries, systolic blood pressure and hypofunction of the myocardium increase, pulmonary vascular resistance increases, and in general, the body's adaptation to the environment and pregnancy is disturbed[2,8] .

The blood supply of the uterus through the maternal artery is one of the most important factors, contributing to the maintenance of the intrauterine environment and allowing the growth and development of the fetus[5].

Inadequate development of blood vessels of the uterus for a long time can lead to placental defects, which in turn leads to the development of preeclampsia, retardation of fetal growth[9,23].

Uterine artery Doppler measurements, body weight gain, body mass index, placental growth factor (PLGF) placenta protein13 (PP13) have a better predictive value for predicting early preeclampsia than other tests with moderate predictive value.

A decrease in the resistance of peripheral blood vessels is an integral part of the normal functioning of the maternal-placental-fetal system. Decreased uterine artery resistance is important for trophoblast invasion. In this context, high resistance of uterine blood flow leads to incomplete invasion of trophoblast, which in turn can serve as the most important predictor. Currently, the study of blood flow indicators of the uterine arteries at 11-14 weeks is used as a marker to predict preeclampsia, delayed fetal development, premature birth [7,25].

During placentation, cytotrophoblast invasion is regulated by the oxygen concentration gradient between the placenta and maternal arteries. Therefore, the

hypoxic environment encountered by cytotrophoblast at the beginning of placentation gradually turns into a nonoxic environment during invasion [27].

In pathologies with anomalous placenta, trophoblastic invasion is weak and limited to the spiral arteries present in the superficial decidual layer. Placental maldevelopment and its mechanisms are one of the problems that still need to be studied[13].

Thus, the first stage of the mechanisms leading to the development of preeclampsia can be explained by the defective trophoblast invasion and the high resistance of the uterine-placental blood circulation during pregnancy, the resistance of the uterine arteries.

Inadequate placental blood supply causes placental hypoxia and local oxidative stress, leading to a systemic inflammatory response, endothelial dysfunction, and clinical signs of preeclampsia.

Assessment of placental blood flow is important in determining clinical signs of preeclampsia. At such a time, the most complete information can be evaluated simultaneously by looking at the blood flow in both uterine arteries, umbilical arteries, internal carotid arteries, and main cerebral arteries[14,18].

Studies have shown that in women at low risk for hypertensive conditions, dopplerometric examinations are not clinically relevant, but in high-risk situations and when there is a risk, it is necessary to evaluate the blood flow of the uterine arteries, which can lead to preeclampsia and delayed fetal development is especially important in determining their initial forms[15,27].

The hemodynamic state of the umbilical arteries evaluates the fetoplacental and microcirculation on the fetal side of the placenta. In fetoplacental insufficiency, the resistance of peripheral blood vessels increases and the diastolic blood flow in the umbilical artery decreases due to a decrease in vascularization of terminal vessels[17,19]. Due to fetal growth retardation, the blood flow rate in the fetal aorta decreases. An increase in the resistance of the fetal blood vessels and their spasm is one of the compensatory mechanisms, which leads to hypoxia and metabolic disorders, which are important for life, due to the violation of the centralization of the blood circulation.

In preeclampsia, blood flow disorders in the spiral arteries occur earlier than in the uterine arteries. It should be noted that the uterine vascular pool is an integral part of the blood circulation system of the whole body, and the disruption of blood flow in the uterine vessels during pregnancy leads to systemic circulatory disorders.

Thus, the results of the analysis of modern research data showed that, despite the many diagnostic methods available today, the problem of determining the most

effective method that allows reliable assessment of the severity and prognosis of the development of preeclampsia remains relevant.

The aforementioned are early diagnosis and predictors of preeclampsia, which provide an opportunity for timely prevention and adequate therapeutic measures.

Literature

1. Akhmedov F.K. Peculiarities of cardiac hemodynamic in pregnant women with mild preeclampsia// Europen Science Review. - 2015. - №4-5. - C. 56 -58.
2. Akhmedov F.K. Features of renal function and some indicators of homeostasis in women with mild preeclampsia// Europen Science Review. - 2015. - №4-5. - C. 58 - 60.
3. Akhmedov F.K., Negmatullaeva M.N., Kurbanova Z.Sh. Modern views on the problem of preeclampsia // A new day in medicine. - 2018. - № 1 (21). - S. 180-185.
4. Akhmedov F.K., Negmatullaeva M.N., Features of the state of central hemodynamics and hemostasis in pregnant women with preeclampsia of varying degrees and severity // New Day of Medicine. - 2020. - No. 1 (29) - S. 147-150.
5. Akhmedov F.K., Negmatullaeva M.N. Modern views on the role of the immune system in the development of preeclampsia// ACADEMICIA: An International Multidisciplinary Research Journal https://saarj.com/ ISSN: 2249-7137 Vol. 11, Issue 5, May 2021 Impact Factor: SJIF 2021 = 7.492. – P. 555-562.
6. Akhmedov Farhod Kahramonovich. Role of study renal blood flow and concentration of uric acid in blood and urine in the diagnosis of preeclampsia// Biology and integrative medicine. 2 (42) 2020. – P. 86-94.
7. Bargetzi, M. J. Interleukin-1 beta induces unterleukin-1 receptor antagonist and tumor necrosis factor binding protein in humans / M. J. Bargetzi, M. Lantz, C. G. Smith [et al.] // Cancer. Res. – 2017. – Vol. 53, № 17. – P. 4010-4013.
8. Bowen, R. S. Hypoxia promotes interleukin-10 production by placental trophoblast cells from preeclamptic pregnancies / R. S. Bowen, Y. Gu, Y. Zhang [et al.] // J .Soc. Gynecol. Investig. – 2015. – Vol. 12, № 6. – P. 428-439.
9. Carl, V. S. Role of endogenous IL-10 in LPS-induced STAT3 activation and IL-1 receptor antagonist gene expression / V. S. Carl, J. K. Gautam, L. D. Comeau [et al.] // J. Leukol. Biol. – 2014. – Vol. 76, № 3. – P. 735-742
10. Cheranov, S. Y. TNF-α dilates cerebral arteries via NADPH oxidasedependent CA2+ spark activation / S. Y. Cheranov, J. H. Jaggar // Am. J. Physiol. Cell Physiol. – 2016. – Vol. 290. – № 4. – P. c964-c971.

11. Deshpande, S. S. Rac1 inhibits TNF-α-induced endothelial cells apoptosis: dual regulation by rective oxygen species / S. S. Deshpande, P. Angkeow, J. Huang [et al.] // FASEB J. – 2019. – Vol. 14, № 12. – P. 1705-1714.
12. Dickensheets, H. L. Interleukin-10 upregulates tumor necrosis factor receptor type-II (p75) gene expression in endotoxine-stimulated human monocytes / H. L. Dickensheets, S. L. Freeman, M. F. Smith [et al.] // Blood. – 2017. – Vol. 90, № 10. – P. 4162-4171.
13. Girard, S. IL-1 receptor antagonist protects against placental and neurodevelopmental detects induced by maternal inflammation / S. Girard, L. Tremblay, M. Lepage [et al.] // J. Immunol. – 2015. – Vol. 184, № 7. – P. 3997-4005.
14. Granowitz, E. V. Effect of interleukin-1 (IL-1) blockade on cytokine synthesis: IL-1 receptor antagonist inhibits lipopolysackharide-induced cytokine synthesis by human monocytes / E. V. Granowitz, E. Vannier, D. D. Poutsiaka [et al.] // Blood. – 2017. – Vol. 79, № 9. – P. 2364-2369.
15. Hashii, K. Peripheral blood mononuclear cells stimulate progesterone production by luteal cells derived from pregnant and non-pregnant women: possible involvement of interleukin-4 and interleukin-10 in corpus luteum function and differentiation / K. Hashii, H. Fujiwara, S. Yoshioka [et al.] // Human. Reprod. – 2018. – Vol. 13, № 10. – P. 2738-2744.
16. Hennessy, A. A deficiency of placental IL-10 in preeclampsia / A. Hennessy, H. L. Pilmore, L. A. Simmons [et al.] // J. Immunol. – 2019. – Vol. 163, № 6. – P. 3491-3495.
17. Huang, H. Y. Interleukin (IL)-1β regulation of IL-1β and IL-1 receptor antagonist expression in cultured human endometrial stromal cells / H. Y. Huang, Y. Wen, J. S. Kruessel [et al.] // J. Clin. Endocrinol. Metab. – 2011. – Vol. 86, № 3. – P. 1387-1393.
18. Juge-Aubry, C. E. Adipose tissue is a major source of interleukin-1 receptor antagonist upregulation in obesity and inflammation / C. E. Juge-Aubry, E. Somm, V. Giusti [et al.] // Diabetes. – 2013. – Vol. 52, № 5. – P. 1104-1110.
19. Negmatullaeva M.N., Akhmedov F.Q., Tuksanova D.I. Modern diagnostics of markers of preeclampsia // Vestnik Tashkentskoy meditsinskoy akademii. - 2020. -№2 (94). - S. 145 - 147.
20. Negmatulleva M.N., Tuksanova D.I., Nosirova M.Sh., Akhmedov F.K. Features of the state of the circulatory system mother and fetus in the second trimester ofpregnancy in women with mitral stenosis ofrheumatic etiology// European

Journal of Biomedical and Pharmaceutical sciences. - 2020. - №7(6). - P. 100 - 103.

21. Maniyozova G.M., Akhmedov F.K., Shamsieva T.T. Indicators of cytokines in pregnant women with antiphospholipid syndrome who received pregravadar preparation // New Day of Medicine. - 2020. - No. 2 (30/2). - S. 194-197.
22. Medvedev, BI Clinical and biochemical predictors of the development of preeclampsia / BI Medvedev, EG Syundyukova, SL Sashenkov // Obstetrics and gynecology. - 2013. - No. 5. - S. 30-35.
23. Maedler, K. Leptin modulates β-cell expression of IL-1 receptor antagonist and release of IL-1β in human islets / K. Maedler, J. Oberholzer, P. Bucher [et al.] // PNAS. – 2014. – Vol. 101, № 21. – P. 8138-8143.
24. Matsuki, T. IL-1 plays an important role in lipid metabolism by regulating insulin levels under physiological conditions / T. Matsuki, R. Horai, K. Sudo [et al.] // J. Experim. Med. – 2013. – Vol. 198, № 6. – P. 877-888.
25. Solieva N.K., Negmatullaeva M.N, Sultonova N.A. Features Of The Anamnesis Of Women With The Threat Of Miscarriage And Their Role In Determining The Risk Group// The American Journal of Medical Sciences and Pharmaceutical Research. - 2020. - № 2(09). - P. 32-34.
26. Tuksonova D.I., Solieva N.K., Negmatullaeva M.N., Shamsieva T.I. The importance of immune status in miscarriage // News of dermatovenerology and reproductive health. - 2020. - No. 1. - S. 52 -54.
27. Tuksanova Dilbar Ismatovna and Solieva Nozima Karimovna. The value of the immune status in miscarriage // Evropean Journal of Pharmaceutical and Medical Research. - 2020. - №7 (6). - P. 117- 119.