CLINICAL AND ANATOMICAL ASPECTS OF PREECLAMPSIA: MODERN FEATURES OF THE COURSE

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Abstract: Preeclampsia (PE) is the most serious complication of pregnancy and childbirth worldwide and remains one of the leading causes of maternal and perinatal morbidity and mortality. It is characterized by a complex etiopathogenesis with specific clinical and laboratory changes (arterial hypertension, proteinuria and edema), as well as a morphological picture of damage to target organs and blood vessels of the uteroplacental site. However, at present, the triad of clinical signs of PE does not occur in all cases of the disease. The goal is to study the clinical and anatomical aspects of PE at the present stage.
Keywords:preeclampsia, clinical picture, etiopathogenesis, pathological anatomy.

INTRODUCTION: No problem in obstetrics attracts as much attention as the development of preeclampsia (PE) and eclampsia. PE remains one of the leading causes of maternal and perinatal morbidity and mortality, reduces the quality of subsequent life of women and disrupts the psychosomatic development of their children [1, 2]. According to WHO data [3], PE is registered in 28% of pregnant women, making up the bulk of all hypertensive conditions during pregnancy.

It is currently accepted that PE is a complex dysfunction of the endothelium (endotheliosis), in which there is a disturbance in the growth, differentiation and functioning of placental vessels associated with inadequate production of vascular endothelial growth factor, as well as an imbalance of coagulation potential blood with the development of DIC syndrome. It has been established that the placental growth factor system regulates the growth and function of the placenta. One of the fundamental reasons leading to the development of PE/eclampsia is a disruption of cellular regulation processes associated with changes in the production of growth factors that ensure the development of the placenta and the formation of its vascular systems [4]. On the one hand, they are stimulators of angiogenesis (vascular endothelial growth factor, placental growth factor), on the other hand, they regulate the metabolic activity of the trophoblast through an autocrine mechanism [3].

MAIN PART: It is known that homocysteine has a direct cytotoxic effect on the endothelium, causing damage to the vascular wall, increases the consumption of nitric oxide, causes platelet hyperaggregation and acts as a procoagulant agent, activating factors XII, V and tissue factor. At later stages of pregnancy, homocysteinemia becomes the cause of the development of chronic placental insufficiency and fetal hypoxia, as well as generalized microangiopathy, clinically manifested as PE [2].

The criteria for PE, including severe PE, have been developed and are known [1, 2], and include primarily a triad of signs: arterial hypertension (AH), proteinuria and edema. However, according to domestic and foreign literature data, the triad of clinical signs of PE is not observed in all cases of the disease. There are also other important symptoms of PE: headache, nausea, vomiting, epigastric pain; visual impairment [3].

A severe form of PE can manifest itself in the form of HELLP syndrome, which has its own specific symptoms, but it is not listed in ICD-10. This syndrome is characterized by symptoms caused by liver failure (intoxication, pain in the right hypochondrium, epigastric pain, thrombocytopenia, edema, ascites, hydrothorax, etc.). However, 20% to 25% of patients with HELLP syndrome may not have hypertension or proteinuria.

The pathological anatomy of PE has characteristic aspects. As a rule, attention is paid to the pathology of three target organs: kidneys, liver and brain [1].

We studied 17 cases of maternal death from PE/eclampsia in medical institutions.

The classic triad of clinical and laboratory signs of PE (hypertension, proteinuria and edema) at the time of the onset of the development of this disease was noted only in 35% of patients, 2 signs - also in 35% of patients, in 30% of patients only one sign was identified, mainly swelling. In addition, in 35% of patients with a full-blown picture of the disease, PE was preceded by headache and abdominal pain, in 29% by convulsions, in 65% by thrombocypenia (6% mild severity, 41% moderate severity).

High blood pressure (BP) was noted only in 35.5% of cases: stage II hypertension (140–159 mm Hg) in 23.5%, grade I hypertension (160–179 mm Hg) in 12% . In 65.5% of cases, hypertension was within normal limits: less than 120 mmHg. in 6%, 120-129 mm Hg. in 23.5%, but among them 35% had so-called high normal blood pressure (130-139 mm Hg).

The second significant clinical sign of PE, proteinuria, was also not found in all cases. Thus, 29% of patients who died from PE had a trace amount of protein in the urine, 29% had less than 0.5 g/l, 24% had 0.5-3 g/l, 18% had high proteinuria, the level of protein in the urine exceeded 3 g/l.

CONCLUSION:PE/eclampsia is the most serious complication of pregnancy and childbirth and often leads to death in both mother and fetus. It is characterized by a complex etiopathogenesis with specific clinical and laboratory changes, as well as a morphological picture of damage to target organs and blood vessels of the uteroplacental site. An analysis of medical histories of deceased pregnant and postpartum women who suffered from PE/eclampsia showed an atypical course of this complication in the majority of women (primarily without a significant increase in blood pressure, with weak/moderate proteinuria). However, many observations revealed a characteristic laboratory sign—thrombocytopenia and enzymatic dysfunction of the liver.

REFERENCES:

1. Savelyeva G.M., Krasnopolsky V.I., Strizhakov A.N., Kurtser M.A., Radzinsky V.E., Shalina R.I. What classification of gestosis (preeclampsia) should a doctor adhere to in everyday work? Journal of Obstetrics and Women's Diseases. 2013;62(1):5-9.
2. Sidorova I.S. Causes of maternal mortality from preeclampsia and eclampsia in 2013. Obstetrics and gynecology. 2015;1:14-18.
3. Prevention of reproductive losses: strategy and tactics. Featured Media Education Seminar “Innovation sinobstetric sandgyn ecology from the stand point of evidence-based medicine.” Radzinskii V.E., ed. Moscow: Editorial Board Status Praesens; 2014.
4. Dobrokhotova Yu.E., Makarov O.V., Lysyuk E.Yu., Olenev A.S., Kuznetsov P.A., Dzhokhadze L.S., Osmanova A.P. Preeclampsia. Choice of obstetric management tactics. Journal of Obstetrics and Women's Diseases. 2016;65(2):16-23.