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Analysis of Influencing Factors of Preeclampsia on Postpartum Hemorrhage

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Abstract: Postpartum hemorrhage is a serious complication of preeclampsia. The main influencing factors of Preeclampsia on postpartum hemorrhage include vascular disease, trophoblast differentiation and infiltration disorder, the influence of antispasmodic, sedative and antihypertensive drugs, the incidence of hypertensive disorder complicating pregnancy related to calcium deficiency, coagulation dysfunction, and decreased blood volume during pregnancy. In recent years, postpartum hemorrhage has been the leading cause of maternal death in the world. Therefore, it is critical to understand the influencing factors of preeclampsia on postpartum hemorrhage. Based on the research and guidelines of preeclampsia and postpartum hemorrhage in recent years, this paper analyzes the influencing factors of preeclampsia on postpartum hemorrhage, and provides new ideas for clinical reduction of postpartum hemorrhage.

Keywords: Postpartum hemorrhage in preeclampsia

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1. Introduction

Preeclampsia (PE) is a disease that occurs only during pregnancy, characterized by changes in blood pressure, which is caused by multiple factors, mechanisms, and pathways. Its basic pathophysiological changes are systemic small blood vessel spasm and vascular endothelial injury, which leads to multiple organ and system damage of parturient, seriously affecting the health of mother and baby, and is the main reason for the increase of maternal and perinatal mortality [1-2]. Postpartum hemorrhage is a serious complication of PE. Postpartum hemorrhage in severe preeclampsia may accelerate or aggravate the risk of heart failure, DIC, acute renal injury syndrome, cerebral ischemia, pulmonary edema, multiple organ injury and liver diseases. Therefore, it is crucial to study the influencing factors of preeclampsia on postpartum hemorrhage, in order to provide new ideas for clinical diagnosis, treatment, and prevention.

2. The influence of preeclampsia on postpartum hemorrhage

Postpartum hemorrhage refers to the amount of bleeding exceeding 500 ml within 24 hours after delivery of the fetus in vaginal delivery, or exceeding 1000 ml within 24 hours after cesarean section. Uterine atony, soft birth canal injury, placental factors, and coagulation dysfunction are the four major causes of postpartum hemorrhage. Among the postpartum hemorrhage factors related to preeclampsia, uterine atony and coagulation dysfunction are the most common [3]. This paper discusses the influencing factors of preeclampsia on postpartum hemorrhage from six aspects: vascular disease, trophoblast differentiation and infiltration disorder, the influence of antispasmodic sedative and antihypertensive drugs, the incidence of

hypertensive disorder complicating pregnancy related to calcium deficiency, coagulation dysfunction, and the decrease of blood volume during pregnancy.

2.1. Vascular disease

During normal pregnancy, in order to ensure the material exchange between the mother and the fetus, the uterine spiral arteries are remodeled and transformed from high-resistance and low-volume vessels in the non-pregnant period to low-resistance and high-volume vessels. In preeclampsia, the extravillous trophoblast infiltration capacity is insufficient, only the decidual blood vessels are remodeled, and the diameter of the uterine spiral artery is 1/2 of that in normal pregnancy. Besides, the oxidation rate of the body exceeds the removal rate of oxides in the body, the balance of the oxidation system/antioxidant system is destroyed, the placenta is damaged by oxidative stress, a variety of placental factors are released, the vascular endothelial function is disordered, and the relaxation and contraction of blood vessels are affected [4]. Moreover, vascular endothelial injury, increased vascular permeability, and edema of uterine smooth muscle occur, affecting uterine contraction.

2.2. Trophoblast differentiation and infiltration disorder

Studies have shown that in normal early pregnancy, trophoblast cells infiltrate the decidual surface into the placental bed tissue, and gradually reach within 5.5 mm of the superficial myometrium [5-6]. In the process of placenta formation, trophoblast cells infiltrate the spiral arteries, resulting in a series of pregnancy-specific morphological and functional changes in the uteroplacental vessels. These changes increase the uteroplacental blood flow during pregnancy, and retrogradely transform the uterine spiral arterioles, so that the uterine spiral arterioles rich in muscle elastic fibers in non-pregnancy gradually lose their normal myometrial structure and are replaced by amorphous fibrous substances. Subsequently, the trophoblasts in the eroded blood vessels replace the vascular endothelial cells and are buried in this layer of amorphous material, so that the vascular lumen expands. In normal pregnancy, there is trophoblast infiltration in the decidua, myometrium and spiral arteries of the placental bed. Hence, trophoblast differentiation and infiltration disorder lead to superficial placental implantation [7].

2.3. Effects of antispasmodic and sedative antihypertensive drugs

Magnesium sulfate is a commonly used drug for the clinical treatment of early-onset severe preeclampsia. Magnesium ions act on motor nerve endings, inhibit the synthesis and secretion of acetylcholine, change the conduction between nerves and muscles, and improve skeletal muscle tension [8]. By inhibiting the vascular neuromuscular, the release of acetylcholine from the motor nerve terminals is reduced, so that the skeletal muscle relaxes, the blood vessels expand, the tension of the uterine muscle fibers is reduced, and the resistance of the uterine muscle fibers to the uterine blood vessels is reduced [9]. The first generation of calcium anticoagulant drugs have inhibitory effects on the release of calcium ions in cells, inhibit the entry of calcium ions into cardiomyocytes, reduce myocardial energy and oxygen consumption, and have inhibitory effects on bronchial excitation, vasoconstriction, and uterine smooth muscle contraction, which easily lead to postpartum hemorrhage [10].

2.4. There is a certain correlation between the incidence of hypertensive disease in pregnancy and calcium deficiency

During pregnancy, both pregnant women and fetuses need adequate nutrition, especially calcium. At 30 weeks pregnant, women need 7 times as much calcium as before. In addition, the glomerular filtration rate increases, and the calcium content in the maternal urine is also increasing. Therefore, pregnant women are prone to calcium deficiency during pregnancy. Studies have shown that the serum calcium content of

women with gestational hypertension and normal pregnancy is lower, and the content of preeclampsia is lower. Calcium is an important component of human survival. It can not only release the activity of enzymes in the human body, but also stabilize the excitability of human nerves and muscles. Calcium ions, as coagulation factor IV, participates in the coagulation process in multiple links. Calcium ions can also increase the sensitivity of uterine smooth muscle to oxytocin. Calcium ions are a cell excitatory coupling agent, and its balanced rate of exchange can ensure the regularity of vascular smooth muscle cells, thus regulating systolic and diastolic blood pressures. Therefore, women with gestational hypertension are more likely to have postpartum hemorrhage [11-12].

2.5. Coagulation disorders

Studies found that due to the influence of the high level of progesterone during pregnancy, vasodilation and the increase of vascular volume combined with the influence of progesterone will lead to coagulation factors I, II, VII, VIII, XI, XII, and fibrinogen at the end of the third month of pregnancy. At this time, fibrinopeptide A, thrombin and antithrombin III will also increase, and the patient's blood will appear hypercoagulable. At the same time, the increase of blood volume and venous dilatation will lead to the compression of the inferior vena cava by the uterus, which will obstruct the blood flow, varicose veins, and slow down the blood flow of patients. Therefore, the body will initiate a self-protection mechanism, the intrinsic and extrinsic coagulation mechanisms will be enhanced, and the content of coagulation factors in the blood will decrease [13]. Vascular endothelial dysfunction, platelets and coagulation factors are activated, and the body is in a hypercoagulable state. If thrombosis occurs, uterine microcirculation will be blocked, placental ischemia and hypoxia, and vascular endothelial cell damage will aggravate. In this case, disseminated intravascular coagulation (DIC) is prone to occur, causing maternal hemorrhage.

2.6. Hypovolemia

During pregnancy, the main factors that affect the blood volume of pregnant women are as below.

(1) Weakened gastrointestinal motility, decreased gastric acid secretion, and partial ingestion of foods with low iron content, which can affect iron intake.
(2) Placenta formation and postpartum 300-400 mg of iron is required for the blood loss of the fetus.
(3) The increases in hemoglobin and red blood cells by 1/3 requires 500-600 mg of iron.
(4) The iron requirement for fetal growth and development is 300-400 mg, and the total iron demand in the late pregnancy is about 900 mg. The total iron requirement of pregnant women throughout pregnancy is 670-1650 mg, an average of 3.5–7.5 mg per day, and 1 mg per day during postpartum lactation. The daily diet cannot meet the needs of the mother and the fetus, and the absorption rate of the human body is only 10%. Therefore, in the second half of pregnancy, pregnant women should supplement at least 4 mg of iron daily to effectively reduce the occurrence of iron deficiency anemia. The amount and red blood cells will increase with the increase of gestational age, but the total amount of plasma increases more than the amount of red blood cells, and the increase ratio is not synchronized. In normal pregnancy, the blood is diluted, the blood viscosity decreases, and the hematocrit decreases [14]. Studies have confirmed that iron during pregnancy is unidirectionally transported through the placenta to the fetus. If iron cannot be supplemented in time, pregnant women will gradually consume the iron reserves in their bodies as the gestational age increases.

2.7. Overall effects of preeclampsia

Preeclampsia will cause systemic vasospasm, thick blood, slow blood supply to organs, resulting in ischemia and hypoxia of organs and tissues, especially ischemia of heart, brain, liver, kidney and placenta, which is easy to cause pathological changes of organs. Symptoms such as dizziness, nausea, vomiting, or

convulsions in parturient women are caused by cerebral ischemia. In severe cases, cerebrovascular embolism symptoms such as coma will occur; placenta ischemia will lead to necrosis and infarction of placental tissue, which will lead to rupture of placental blood vessels and placental abruption. Placenta abruption will do harm to both parturient and fetus by increasing postpartum hemorrhage, causing hemorrhagic shock, and affecting the delivery of fetus. Fetus may suffocate due to abruption hemorrhage, resulting in adverse pregnancy outcomes. Therefore, it is very important to find out the influencing factors of placental abruption in patients with severe preeclampsia to save the life safety of patients and fetuses.

3. Conclusion

At present, postpartum hemorrhage still ranks first among the causes of maternal deaths, with uterine atony being the primary cause of postpartum hemorrhage. Pregnancy-induced hypertension patients are more likely to cause postpartum atony. The occurrence of postpartum hemorrhage too abrupt for temporary medication to take effect. Therefore, preventing postpartum uterine atony is the key to reducing postpartum hemorrhage and maternal mortality. From the above analysis of the six causes of postpartum hemorrhage due to preeclampsia, we can see that the main factors affecting uterine contraction are vascular disease, the use of antispasmodic and sedative antihypertensive drugs, and calcium deficiency in pregnancy-induced hypertension. In case of postpartum hemorrhage, the calcium requirement of pregnant women during pregnancy should be well understood, and adequate calcium should be supplemented in a timely and effective manner. The dosage and duration of medication should be strictly controlled to achieve a better curative effect while minimizing the impact on postpartum hemorrhage [15].

Disclosure statement

The author declares no conflict of interest.

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