Vol.7 No.9:159

Critical Complications and Symptoms of Preeclampsia

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Received: September 06, 2021; Accepted: September 20, 2021; Published: September 27, 2021

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Citation: Graham J (2021) Critical

Complications and Symptoms of Preeclampsia. Gynecol Obstet Case Rep Vol.7 No.9:159

Myometrium segments of these arteries remain anatomically intact and undiluted, and adrenergic nerve supply to the spiral arteries is not affected. The mean external diameters of the uterine spiral arteries in women with preeclampsia are less than one half of the diameters of similar vessels from uncomplicated pregnancies. This failure of vascular remodelling prevents an adequate response to increased fetal demands for blood flow that occur as gestation progresses. Inappropriate integrin expression by the extravillous cytotrophoblast may explain the shallow pattern of invasion and lack of arterial remodelling that occurs in preeclampsia.

Patients with chronic hypertension tend to have a higher risk of developing preeclampsia. The risk of developing preeclampsia is highest during the first pregnancy. Each pregnancy with a different partner increases the risk of preeclampsia more than does a second or third pregnancy with the same partner. Age factor also has an effect as the risk of preeclampsia is higher for very young pregnant women or minors and also for pregnant women older than 35. Obesity can be considered as a risk factor for preeclampsia. Preeclampsia is more common in women who are carrying twins, triplets or other multiples.

Gap between two pregnancies also plays a role, having babies less than two years or more than 10 years apart leads to a higher risk of preeclampsia. Previous health complications such as lung diseases, heart malfunctioning etc., also play a role in increasing chances of preeclampsia. Risk of preeclampsia is more in the case of in vitro fertilization than that of normal.

Perspective

Preeclampsia is a pregnancy complication characterized by increased blood pressure and may cause damage to another organ system, most often the liver and kidneys. Preeclampsia usually begins after 20 weeks of pregnancy in women whose blood pressure had been normal. If this condition is left untreated it may lead to severe sometimes even fatal complications for both the mother and the baby. If a patient is diagnosed with preeclampsia, the most effective treatment is the delivery of baby. Even after delivering the baby, it can still take a while for the pregnant mother to get better.

If a patient is diagnosed with preeclampsia in the early stages of pregnancy, to deliver baby the patient and doctor face the challenge. A baby needs more time to mature, but the patient need to avoid putting herself or baby at risk of serious complications. Rarely, preeclampsia develops after delivery of a baby, a condition known as postpartum preeclampsia. Preeclampsia sometimes develops without any symptoms. High blood pressure may develop slowly, or it may have a sudden onset. Monitoring your blood pressure is an important part of prenatal care because the first sign of preeclampsia is commonly a rise in blood pressure. Blood pressure that exceeds 140/90 mm of mercury (mm Hg) or greater documented on two occasions, at least four hours apart is abnormal. Several other complications may include increased protein in urine, severe headaches, blurred vision, abdominal pain, nausea or vomiting sensation, decreased platelet levels, shortness of breath etc.

Placental ischemia is stated as a key factor for preeclampsia. During early human pregnancy, cytotrophoblast cells attack the uterine spiral arteries, replacing the endothelial layers of these vessels with the subsequent destruction of the medial elastic, muscular, and neural tissue. By the end of the second trimester of pregnancy, the uterine spiral arteries are lined exclusively by cytotrophoblast, and endothelial cells are no longer present in the endometrial or superficial myometrium regions. This remodelling of the uterine spiral arteries results in the formation of a low resistance arteriolar system with a dramatic increase in blood supply to the growing foetus. In preeclampsia, invasion of the uterine spiral arteries is limited to the proximal decidua, with 30% to 50% of the spiral arteries of the placental bed escaping endovascular trophoblast remodelling.