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MORPHOFUNCTIONAL CONDITION OF THE PLACENTA AND FEMALE WITH MULTIPLE PREGNANCIES

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Abstract:

Impaired placentation is an important contributing factor to intra-uterine growth restriction and pre-eclampsia in fetuses with congenital heart defects (CHD). These pregnancy complications occur more frequently in pregnancies with fetal CHD. One of the most important factors influencing the life of children with CHD is neurodevelopmental delay, which seems to start already in utero**.**Gestational diabetes mellitus (GDM) and preeclampsia (PE) are common pregnancy complications with similar risk factors. Although GDM is associated with PE, the exact mechanism underlying the association is unclear.

Keywords: Pregnancy, preeclampsia, gestational diabetes mellitus, placental proteins, inflammation

INTRODUCTION

Gestational diabetes mellitus (GDM) and preeclampsia (PE) are common pregnancy complications with similar risk factors (obesity, age over 35, multiparity, etc.). GDM is defined as glucose intolerance first diagnosed during pregnancy. GDM is associated with obstetric and neonatal complications and is recognized as a risk factor for cardiometabolic disorders in the mother and offspring in the future. The PE incidence has also been shown to substantially increase in GDM. PE is characterized by de novo hypertension (a systolic or diastolic blood pressure ≥140 or ≥90 mm Hg, respectively) diagnosed after 20 weeks of gestation with proteinuria and/or dysfunction of at least one organ (the kidney, liver, nervous system, etc.) and is a main cause of maternal and fetal morbidity and mortality. GDM complicated by PE further increases perinatal adverse events and future maternal risk of hypertension, cardiovascular disorders, and diabetes mellitus. Although GDM is associated with PE, the exact mechanism underlying the association is unclear. The PE pathophysiological process includes early insufficient trophoblast invasion, which leads to incomplete spiral artery remodeling and eventually causes placental ischemia and oxidative stress. Hyperglycemia can cause inflammation and autophagy of trophoblast cells and inhibit their migration and invasion. Neutrophils are overactive in GDM and PE and release excessive amounts of neutrophil extracellular traps (NETs), including DNA fibers and serine proteases (cathepsin G, neutrophil elastase (NE), and proteinase 3 (PR3)), in inflammation sites. The proteases have recently been shown to activate proinflammatory cytokines, including interleukin (IL) 1β and TNF-α. Excessive NET production hinders circulation in the intervillous space, leading to placental ischemia.

RESEARCH METHODS.

With an incidence of five to eight per thousand newborns, congenital heart defects (CHD) are the most common congenital anomalies. As approximately half of the CHDs are severe and treatment carries risks, CHDs are a large contributor to infant mortality worldwide. Due to continuous improvement of ICU-care and cardiothoracic surgery, together with increased prenatal detection, survival rates of affected newborns have increased greatly. For that reason, the focus of innovation has shifted from increasing survival rates to improvement of long-term (neuro)developmental outcomes. An important part of the morbidity in children and adolescents with CHD is neurodevelopmental delay, which is reported in a significant amount of cases. In earlier literature, this impairment was attributed to the adverse effects of complex cardiothoracic surgery in early life. More recently, a decreased prenatal and postnatal head circumference and delayed cortical maturation on prenatal ultrasound and MRI were found in CHD fetuses and newborns before surgery. This raises the question whether the hemodynamic effects of the CHD induce intrauterine exposure to decreased flow or low saturation in early neonatal life.

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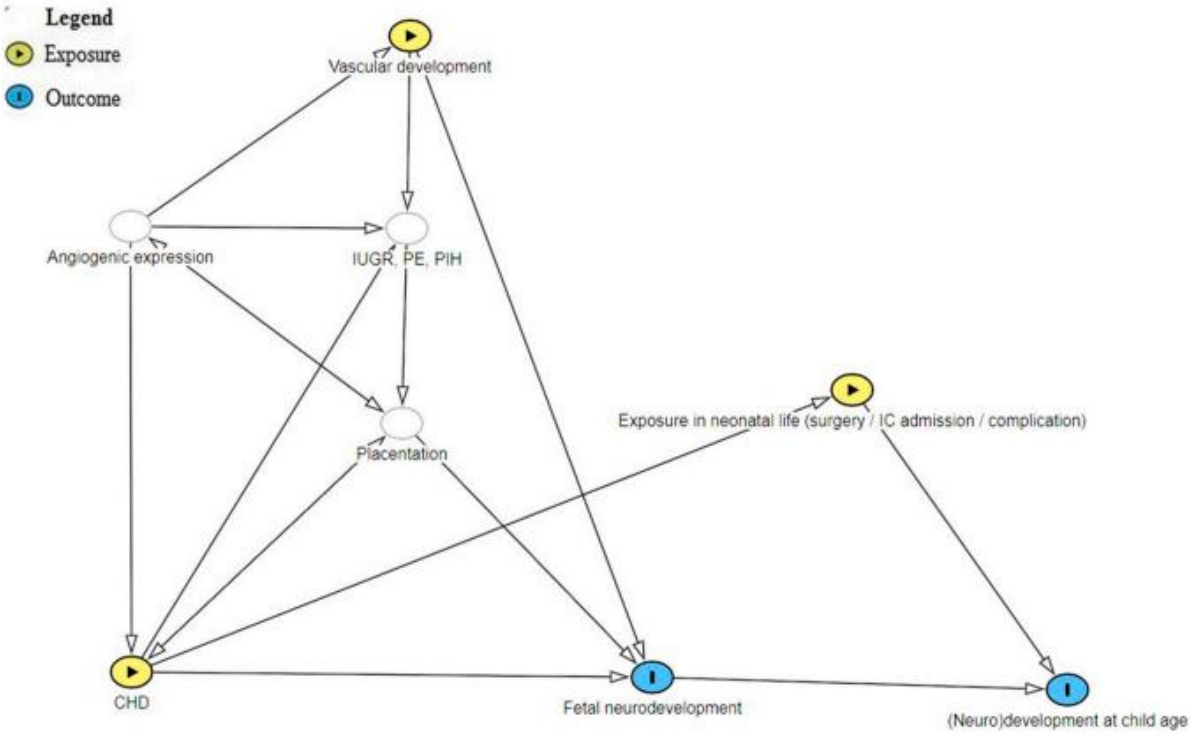

Fig. 1. The directed acyclic graph of hypothetical associating factors, available on http://www.dagitty.net

In our study, the histological study of the placental structures and umbilical cord in women with multiple pregnancies was used to analyze and evaluate perinatal complications that occur depending on the type of placentation and the state of attachment of the umbilical cord to the placenta. In the first trimester of pregnancy, the gestational age was determined using ultrasound. The number of fetal eggs and amnions was examined, and the type of chorion and amniotic fluid was studied. After delivery, the placenta was examined to confirm the type of chorion, amniotic fluid, and to identify pathology in the placenta and umbilical cord. Also, the place of attachment of the umbilical cord, the distance between them, and the angioarchitectonics of the placenta were visually examined with a visual macroscopic examination of each placenta. The placenta and umbilical cord were fixed in 10% formalin at room temperature and sent to the Republican Center for Pathology for histological examination.

RESULTS

The placenta is an important organ that ensures the balance between the mother and the fetus. One of the most common problems of perinatal pathology is placental insufficiency, which has become an urgent problem in obstetrics. From an evolutionary point of view, the female body is suitable for the development of one fetus, and multiple pregnancy is a classic example of placental insufficiency. Placental insufficiency is complicated by dissociated fetal development, fetal hypoxia, and fetal injury during childbirth. Morphological changes occurring in the placenta, in turn, trigger mechanisms of impaired uteroplacental and fetal-placental blood circulation in the form of a "domino effect". Histological examinations of the placenta of pregnant women with multiple pregnancies diagnosed with placental insufficiency revealed that this is associated with the activation of biosynthetic processes. Placental insufficiency is based on the violation of biological amines, heparin metabolism, changes in placental microcirculation, and morphological disorders of the placenta. Currently, placental insufficiency is considered a syndrome and is understood to occur as a result of morphofunctional changes in the placenta that support adequate exchange between the mother and fetus. The placenta (Latin: placenta - a ball), the place of the child - an organ formed during pregnancy and connecting the fetus and the mother's body with each other, ensuring the exchange of substances between them. The fetus receives oxygen and nutrients from the mother's blood through the placenta, and releases decomposition products and carbon dioxide into it. The placenta also acts as a barrier, regulating the flow of various substances to the fetus. The placenta contains enzymes and vitamins; hormones (gonadotropin, estrogen,

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progesterone) and other substances that affect the mother's body are synthesized in it. The placenta is fully formed by the 4th month of pregnancy: the placenta of a normally developed fetus is round, thick, soft, and resembles a ball in shape. The fetus is attached to the placenta through the umbilical cord or umbilical system. The placenta has two surfaces: the maternal surface facing the uterine wall (basal plate) and the fetal surface facing inward - into the amniotic (fetal sac) cavity. The umbilical cord is connected to the surface of the fetus by blood vessels. Placental dysfunction can cause various complications during pregnancy, such as premature birth, placental abruption, pregnancy toxemia, and others (see also Placenta).

CONCLUSION

Placentas of dichorionic and monochorionic twins were analyzed. Macroscopically, we observed the presence of calcifications, infarcts, fibrinoid tissue, and vascularization in the placentas of dichorionic and monochorionic twins. The distance between the umbilical cord and the umbilical cord was studied. Edge fusion (46.8%) and body fusion (14%) were detected. Abnormal fusion of the umbilical cord was significantly higher in monochorionic type C compared with bichorionic type C pregnancies. Edge fusion of the umbilical cord was noted in 41.2% of monochorionic and 38.1% of bichorionic pregnancies. Fusion of the umbilical cord to the placental body was observed in 20.6% and 10.2%, respectively. Thus, twin pregnancy causes placental pathologies, which may be specific. The identification of chorionic, amniotic, and placental abnormalities is essential for the adequate management of multiple pregnancy. Postpartum pathological examination of the placenta helps to assess the presence of abnormalities in the placental and umbilical system, as well as providing information about the chorion and helping to understand potential disease mechanisms affecting twin pregnancy.

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