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MORPHOLOGICAL AND HISTOMETRIC CHARACTERISTICS OF THE PLACENTA COMPONENTS IN WOMEN WITH CONGENITAL HEART DEFECTS

Isroilova Gulsanam Muxtorjonovna
Assistant of the department of obesterics and
gynecology of Ferghana Medical Institute of Public Health

Annotation

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 innova tion has shifted from increasing survival rates to improvement of long-term (neuro) developmental outcomes.

Keywords: placental weight, Tetralogy Fallot, placental biomarkers, angiogenic biomarkers, hypoplasia, CHD.

Placental weight In Tetralogy of Fallot (ToF), double-outlet right ventricle (double-outlet RV), major ventricular septal defects (VSDs) and hypoplastic left heart syndrome (HLHS), a reduced absolute placental weight and placenta weight percentiles were found at birth. Placenta volumes measured by MRI between 18 and 39 weeks of gestation, tended to be smaller in a study with mixed CHD cases, but the found differences did not reach statistical significance. As placenta weight does not differentiate between cases with and without IUGR, it is important to identify placenta weight in relation to the birthweight. Five studies reported on placenta-to-birthweight ra tio's, a measure to assess placental weight by correcting for birthweight, and described conflicting results. Four studies, of which three with a retrospective design, found no significant difference in placenta-tobirthweight ratio in CHD cases, suggesting that smaller placentas in CHD are only present in cases with a low birthweight. Three prospective studies did find a significant lower

placenta-to-birthweight ratio in CHD cases, which means that the placentas were even smaller than expected, based on the birthweight of these cases. If CHD placentas are smaller in comparison to birthweight, fetus were able to reach their growth potential, indicating preservation of the placental function.

Histological findings indicating impaired fetal-maternal exchange are more frequently found in pregnancies with CHD. It was first reported in HLHS cases, where fibrin deposition, distal villous hypoplasia, lower vascular area and lower vasculo-syncytial membrane counts were described. Later this was confirmed in placentas of a group of mixed CHD cases, as fetal placental thrombosis, placental infarction, choriangiosis, infarction and delayed villous maturation were frequently reported, whilst these are phenomena that are not found in placentas of healthy newborns. There was no significant difference in incidence of microscopic placenta abnormalities in cases with and without PE, PIH and IUGR.

Angiogenic biomarkers in pregnancy, such as Placental Growth Factor (PlGF) and pregnancy-associated plasma protein A (PAPP-A) are mainly produced in the placental trophoblasts. Altered expres sion of angiogenic biomarkers in CHD cases may modify vascular pathways in the placenta, as concentrations of the angiogenic bio markers Placental Growth Factor (PlGF) and pregnancy-associated plasma protein A (PAPP-A) were significantly lower in maternal serum of CHD cases. Soluble fms-like tyrosine kinase (sFlt-1) and soluble endoglin (sEng) are anti-angiogenic biomarkers and were significantly higher in maternal serum of CHD cases. sFlt-1 and sEng levels were higher in umbilical cord blood of CHD cases. None of the studies describing angiogenic biomarkers corrected for placenta weight. Alterations in expression of these (anti-)angiogenic biomarkers suggest placental dysfunction and impaired placental perfusion. These findings may explain the decreased birthweight and placenta weight in isolated CHD cases.

All in all, CHD is associated with lower placental weight, macroscopic and microscopic placental abnormalities, altered angiogenic biomarkers in placental tissue and maternal serum. These findings suggest impaired vascular development of both the placenta and fetal organs.

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