

Giant placental chorioangioma complicated by fetal hyperdynamic heart strain

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Description of the outcome of pregnancy complicated by large placental chorioangioma

Case report: 44 years old woman (III/I, spontaneous conception, gestational diabetes mellitus diagnosed in 28th week of pregnancy, treated only by diet, otherwise no remarkable personal or family history) was presented to routine third trimester ultrasound scan (gestational age 30+3). She went through standard antenatal care with first and second trimester ultrasound screenings, where no anomalies were detected. Ultrasound examination showed female fetus in breech position, estimated fetal weight on 68. centile (1630g), normal amniotic fluid index. In the fetus was found mild cardiomegaly with normal cardiac anatomy and no regurgitation on atrioventricular (AV) valves were detected. There was small pericardial effusion, otherwise no signs of hydrops were seen. At the cranial edge of placenta was found well circumscribed large tumor (113x90x83mm) with a supply vessel directly from the umbilical cord. Doppler (pulsatility indices - PI) findings were: umbilical artery (UA) 0.82, middle cerebral artery (MCA) 2.0, ductus venosus (DV) 1.1. Peak systolic velocity (PSV) in MCA was 79cm/s (1.93MoM). The finding was closed as a placental chorioangioma, hyperdynamic circulation, anemia cannot be excluded.



Picture 1: Chorioangioma



Picture 2: Cardiomegaly

The patient was admitted to hospital, corticoid therapy (2 doses of betamethasone i.m.) was administered. Doppler parameters were checked daily, because of no knowledge of speed of growth. PSV MCA was in the range 1.6-1.9MoM, DV PI was progressively worsening to 1.25.

Fourth day after diagnosis were noted prefrontal subcutaneous edema, slight progression of pericardial effusion and tricuspid valve regurgitation. It was decided to terminate the pregnancy by caesarean section.

The birth weight of the female newborn was 1660g, Apgar score 6-8-9. The newborn had mild generalized edema, particularly in the lower limbs. Echocardiographic examination 30minutes after delivery showed: ventricular hypertrophy with reduced function bilaterally, patent foramen ovale with bidirectional shunt, pulmonary hypertension and small amount of fluid in the pericardium. Circulation support was not required, ventilation was supported by nasal CPAP. In blood count, there was thrombocytopenia (77x10⁹/l), no anemia was found. Platelet count decreased within three days to 36x10⁹/l and dexamethasone i.v. was applied. The newborn had no bleeding symptoms. Platelet count then gradually normalized. The mother was tested on anti-thrombocyte antibodies with a negative result. The third day, ventricular function was assessed as normal and the ninth day, the heart size was normal too and the edema resolved.

The histological examination of the placenta: the total size of placenta 160x125x30mm, the tumor size 130x95x65mm. It was closed as atypical cellular chorangioma of the placenta, a variant of chorioangioma with higher mitotic activity and mild cytological irregularities. This variant does not have a different biological behavior from the classical variant, it is a benign form.



Picture 3: Placenta

Discussion: Placental chorioangioma is the most common benign tumor of the placenta with reported prevalence of approximately 0,5-1% . It is reported that female fetuses have a higher occurrence of this tumor. Most chorioangiomas are small and asymptomatic. Large tumors (> 4-5cm) are associated with maternal and fetal complications. The pathophysiology of the complications is not fully understood. The main hypotheses are: chronic arteriovenous shunting with a development of a high output fetal cardiac failure and the sequestration of red blood cells and platelets within the tumor. Antenatally, it is diagnosed by ultrasound as a hypo- or hyperechoic rounded mass with anechoic spaces, which protrude into the amniotic cavity. Most of them are located near the umbilical cord insertion. Color Doppler displays a presence of vascular channels in the tumor contiguous with the fetal circulation.

The fetal complications include polyhydramnios (most common, 14-28%), preterm delivery, non-immune fetal hydrops, cardiomegaly, fetal heart failure, fetal anemia and thrombocytopenia, fetal growth restriction and intrauterine fetal death. The maternal complications include preeclampsia and maternal mirror syndrome (maternal edema associated to fetal hydrops, unclear etiology).

The chorioangioma is usually managed conservatively. Patients are monitored by ultrasound, where fetal growth, dopplerometry, signs of hydrops and tumor size are assessed. The most common invasive therapeutic procedures are amniocentesis and intrauterine transfusions. Other invasive interventions to block AV shunting include alcohol injection, micro coil embolisation and endoscopic laser coagulation. These techniques are more successful when tumor is located away from umbilical cord site and its circulation is not directly depended on the umbilical cord.

Conclusion: The Placental chorioangioma is a benign vascular malformation of a placenta. The crucial role in detection and outcome prediction plays ultrasound examination (gray scale and color Doppler). Most chorioangiomas are small and asymptomatic with good prognosis. When vascularisation is poor, the prognosis is favorable too. Large tumors are associated with fetal complications. Perinatal and fetal mortality was reported of 28%. Overall prognosis depends on the presence or development of fetal hydrops. The important finding of our case is, that PSV MCA is not specific for fetal anemia – even big velocity (1.9MoM) measurement could be associated only with hyperdynamic fetal circulation and normal hematocrit.