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Case Report

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# Placental chorioangioma with Dysgenesis of corpus callosum: A case report

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**Abstract**

Chorioangioma is the most common benign tumor of the placenta which results from vascular malformations. Their clinical significance changes according to their associated complications such as preeclampsia, premature placenta release, polyhydramnios, intrauterine growth restriction, fetal anemia, fetal cardiomegaly and fetomaternal hemorrhage. This case is about a 32-year-old patient whose pregnancy was complicated by a large placental chorioangioma in the third trimester.

**Keywords:** placental disease, chorioangioma, preeclampsia, vascular malformations

## 1. Introduction

Chorioangioma is the most common benign tumor of the placenta, which is non-trophoblastic in origin and believed to result from differentiation and proliferation defects of vascular structures (1). The incidence of placental chorioangioma in microscopically examined placentas is approximately 1% (2). These vascular malformations, so-called hamartomas, are of clinical importance when associated with various gestational complications such as preeclampsia, premature placenta release, polyhydramnios, intrauterine growth restriction, fetal anemia, fetomaternal hemorrhage and congestive heart failure of the fetus (3, 4). We report a pregnancy complicated by preeclampsia associated with a large placental chorioangioma, emphasizing the importance of closely monitoring both fetus and mother throughout pregnancy, both at antenatal and postnatal periods, to prevent potential complications.

## 2. Case Report

A 32-year-old primigravida consulted our obstetrics and gynecology service at ten weeks gestation. History revealed no significance for any disease. She did not smoke, and her blood group was A Rhesus+. She had a singleton pregnancy. The Combined test was below the cut-off value. She did not have any complaints in the previous half of the pregnancy. After referral for a detailed ultrasound in the second trimester, an ultrasound scan at 20+6 weeks gestation showed corpus callosum dysgenesis without additional major anomalies. We informed the patient about the potential neurological problems and accompanying genetic diseases. Fetal Doppler parameters did not demonstrate evidence of either anemia or cardiac dysfunction. An oral glucose tolerance test excluded gestational diabetes. Maternal serology for TORCH infections revealed no abnormality. We performed amniocentesis during the 21st gestational week to exclude any genetic or metabolic diseases. The test result did not demonstrate any quantitative or gross anomalies. Furthermore, we detected no clinically significant chromosomal copy number changes. Karyotype and array-CHG analysis results were normal.

At the 27$^{th}$ gestational week, Doppler US revealed a mass with significant vascularization, a flow rate of 33 cm/sec, 37x28mm, located near the caudal placental pole, away from the umbilical cord insertion area. We recommended weekly MCA follow-up.

We performed a fetal cranial MR imaging (Fig. 1a, 1b) at 28+6 weeks gestation to evaluate the fetal neuronal development, and the results confirmed ‘corpus callosum dysgeneses’.

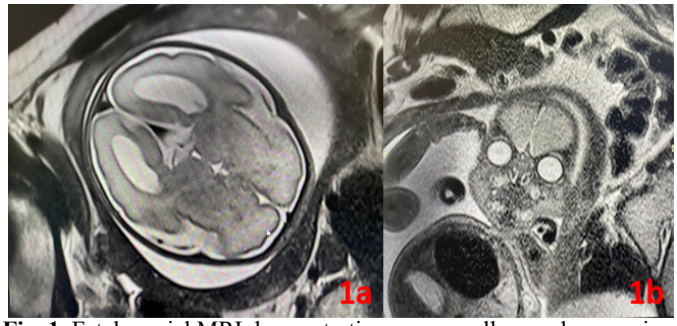


**Fig. 1.** Fetal cranial MRI demonstrating corpus callosum dysgenesis

Although the mass remained asymptomatic for a while, she complained of nausea, vomiting and pregnancy-induced hypertension accompanied by significant proteinuria towards the end of the pregnancy. At the 36$^{th}$ week of gestation, fetal heart rate was 140 bpm rhythmic and regular, with no evidence of cardiomegaly or fetal anemia. The vascular mass,

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considered placental chorioangioma, measured 55x49x51mm, with a 33cm/sec flow rate. MCA PSV:0.95 MoM. At the 37th week of gestation, MCA PSV:1.11MoM. Non-stress Tests were reactive. We recommended NST once in 3 days.

Caesarean delivery was performed at the 39th week without intervening complications. The newborn was male with an Apgar score of 8 for five minutes. During the postpartum period, both mother and neonatal were healthy. We obtained informed consent from the patient during the postpartum period to publish her data and images. They were discharged from the hospital on the third day.

## 3. Discussion

Placental chorioangiomas are vascular masses originating from placental tissue, resulting from development defects of vascular structures and associated with increased angiogenic growth factors as stated by Guschmann et al. (3) by immune-histochemical investigation of 136 cases. Placental chorioangioma is associated with severe pregnancy outcomes related to its mass size and the degree of hydrops fetalis (1). They could be associated with significant problems, including polyhydramnios, a tendency to preterm labour due to increased uterine distention, fetal anemia due to hemorrhage, fetal hydrops, fetal cardiomegaly, fetal growth restriction and fetal demise related to decompensated heart failure (2). For those complicated cases, pregnancy evaluation should be individualized to interfere in time and manage with appropriate modalities (5). In this case, we report a pregnancy complicated by a large placental chorioangioma in the third trimester. Histopathological examinations of the delivered placenta confirmed the diagnosis. (Fig. 2, 3)

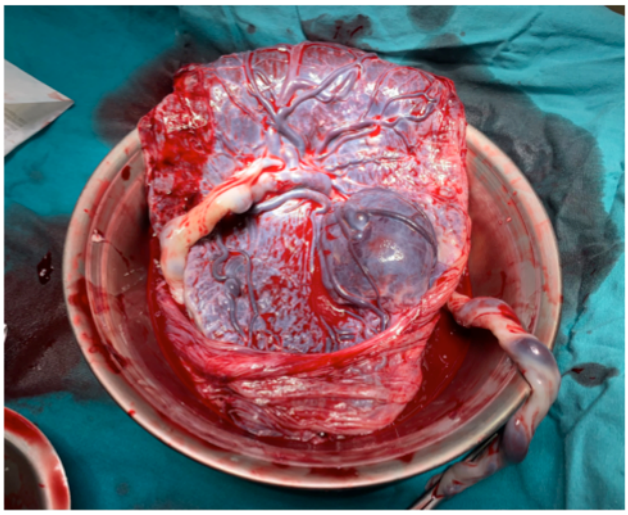

**Fig. 2.** Fetal surface of expelled placenta demonstrating placental chorioangioma

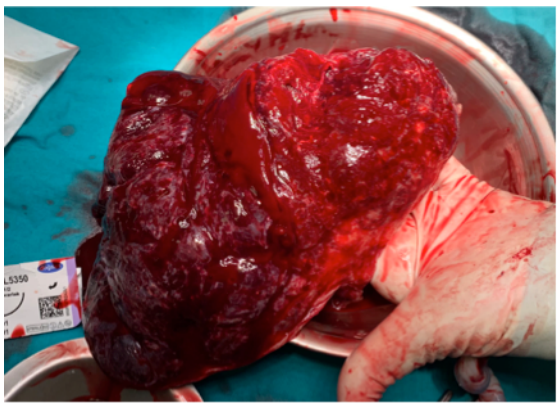

**Fig. 3.** Maternal surface of expelled placenta demonstrating placental chorioangioma

Placental chorioangioma can be demonstrated by various imaging techniques such as grey-scale ultrasonography, CDI, three-dimensional (3D) and four-dimensional (4D) ultrasound. Grey-scale ultrasound remains the primary diagnostic tool (2). We determined the placental mass by CDI, which is also beneficial in demonstrating the vascular nature of the mass. Besides the complications caused by placental chorioangioma, the fetal development defect causing corpus callosum dysgenesis was demonstrated by a detailed ultrasound examination in an earlier period, and genetic analysis was performed to evaluate the risks. The case demonstrates the critical role of close surveillance by imaging modalities such as Doppler ultrasound and Gray-scale ultrasound at regular intervals. Detailed ultrasound is also indispensable in evaluating fetal development and prepares us for associated risks. By the end of the second-trimester mother had signs of preeclampsia. It is shown that even preeclampsia symptoms could be a clue for us to suspect a background pathology. We managed the late pregnancy period with frequent follow-ups to protect maternal and fetal health.

In conclusion, as revealed in this case report and many other studies, placental chorioangioma is associated with vast clinical significance ranging from pregnancy-induced hypertension to fetal demise. It is crucial to remember that there could be an underlying pathology such as placental chorioangioma in patients suffering from irrelevant symptoms. Approaching suspiciously is substantial in diagnosis. Close surveillance of both fetal and maternal status with serial imaging modalities should be performed to recognize the possible complications earlier and interfere on time. Even though it is conducted with expectancy management in the reported case, close monitoring is essential in management.

**Conflict of interest**

The authors declared no conflict of interest.

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None to declare.

**Authors' contributions**

Concept: N.D.G., E.D, Design: E.D, N.D.G., Data Collection or Processing: N.D.G., Analysis or Interpretation: E.D., Literature Search: E.D., Writing: E.D.

**References**

**1.** Buca D, Iacovella C, Khalil A, Rizzo G, Sirotkina M, Makatsariya A, Liberati M, Silvi C, Acharya G, D'Antonio F. Perinatal outcome of pregnancies complicated by placental chorioangioma: systematic review and meta-analysis. Ultrasound Obstet Gynecol. 2020 Apr;55(4):441-449. doi: 10.1002/uog.20304. PMID: 31034661.

**2.** Fan M, Skupski DW. Placental chorioangioma: literature review. J Perinat Med. 2014 May;42(3):273-9. doi: 10.1515/jpm-2013-0170. PMID: 24334427.

**3.** Guschmann M, Henrich W, Dudenhausen JW. Chorioangiomas--new insights into a well-known problem. II. An immunohistochemical investigation of 136 cases. J Perinat Med. 2003;31(2):170-5. doi: 10.1515/JPM.2003.023. PMID: 12747234.

**4.** Haak MC, Oosterhof H, Mouw RJ, Oepkes D, Vandenbussche FP. Pathophysiology and treatment of fetal anemia due to placental chorioangioma. Ultrasound Obstet Gynecol. 1999 Jul;14(1):68-70. doi: 10.1046/j.1469-0705.1999.14010068.x. PMID: 10461342.

**5.** Al Wattar BH, Hillman SC, Marton T, Foster K, Kilby MD. Placenta chorioangioma: a rare case and systematic review of literature. J Matern Fetal Neonatal Med. 2014 Jul;27(10):1055-63. doi: 10.3109/14767058.2013.847424. Epub 2013 Oct 17. PMID: 24460422.