Rapid Evolution of Placental Chorioangioma: Natural Progression and Outcome

Placental chorioangioma is a benign vascular tumor most frequently diagnosed in the second trimester of pregnancy by sonographic and Doppler evaluation. The course of the tumor may be totally indolent, with minimal to no maternal and fetal consequences, or it may also lead to premature labor, intrat uterine growth restriction, hydrops fetalis, and high-output fetal heart failure, which may be due to arteriovenous anastomoses between the tumor and the placenta. We present the case of a rapidly growing chorioangioma, which culminated in premature labor, neonatal anemia, and heart failure, eventually leading to neonatal death. The uniqueness of our case resides in its sudden and brisk temporal evolution, which led to critical consequences.

The patient was a 32-year-old woman, gravida 3, para 2, with a history of 2 cesarean deliveries, 1 of which resulted in a live birth and the other in neonatal death due to sepsis. First-trimester screening for major abnormalities and aneuploidy yielded negative findings.

On a second trimester scan at 18 weeks, a chorioangioma was discovered, and close surveillance was initiated. At 22 weeks, the tumor measured 58 × 43 mm (Figure 1A). No single vascular pedicle was identified, and the fetus was growing appropriately without any signs of impairment. At 27 weeks 3 days, the tumor nearly doubled in size and measured 96.7 mm; this rapid increase motivated close surveillance, despite the normal fetal growth, the normal amniotic fluid index, and the normal umbilical and middle cerebral artery Doppler findings (1.29 multiples of the median). Prenatal treatment was discussed at that time. Eight days later, the sonographic evaluation showed further tumor growth, which now measured 15 cm. In addition, the fetus now manifested signs of hydrops, with scalp edema, ascites, and polyhydramnios. She was thus admitted at 28 weeks 4 days for close surveillance, and cortisone was administered in anticipation of imminent delivery. Blood work values were within the normal range except for maternal anemia. She received 12 mg of betamethasone twice over 24 hours. Nonstress fetal heart testing maintained reactivity and was performed repeatedly. She underwent a cesarean delivery 24 hours after admission because of the fetal hydrops. We noticed an excess of fluid, and the placenta and cord were hydropic. Surgery was complicated by transient uterine atony, which responded to medical treatment.

The female neonate had considerable edema, weighed 2100 g, and had an Apgar score of 7 at 1 minute but rapidly became cyanotic. No heart murmur was heard. She had substantial ascites, and the presence of ecchymosis was noted on several areas of her body. She later had spontaneous bleeding from her mouth. The neonate’s hemodynamic status was critical, with substantial desaturation, which necessitated mechanical ventilation. Fetal bradycardia ensued as well and partially responded to dobutamine, a continuous adrenaline infusion, as well as cardiac massage. Neonatal echocardiography revealed a structurally normal heart that was, however, in severe heart failure. The neonate then went into a cardiac arrest, and resuscitation for 20 minutes was unsuccessful, leading to neonatal death. Postmortem blood work revealed severe anemia (hemoglobin level, 5.6 g/dL) and disseminated intravascular coagulation.

The mother had an uncomplicated postoperative medical course and was discharged home on postoperative day 2 in good physical condition and without complications. Our medical team provided her with ongoing psychological support and follow-up. The pathology report showed a hypertrophic 1130-g placenta with a 15-cm chorioangioma. Vascularization of the chorioangioma was seen (Figure 1B).

Placental chorioangioma is the most common placental tumor, with an estimated prevalence of 1% in systematic placental histologic studies. The first case of a prenatal sonographically diagnosed chorioangioma was reported by Asokan et al in 1978. Small chorioangiomas tend to remain asymptomatic during pregnancy. Large or giant chorioangiomas (>4–5 cm in diameter) are more often diagnosed prenatally by sonography during the second trimester.

Chorioangioma presents as a well-circumscribed hypoechoic placental tumor located in most cases at the chorionic plate, adjacent to the cord insertion. The vascular nature of the tumor can be confirmed by a Doppler study. Signs of shunting with the umbilical vein may also be shown. In our case, after the rapid growth of the tumor, several abnormal flow velocity signals were shown, thus possibly indicating the formation of a vascular communication between the fetal circulation and the tumor.

Complications are due to anastomosis of the arteriovenous system within the placenta, by means of a “stealing” phenomenon, which short circuits blood from a high-to a low-resistance vascular bed, leading to heart failure; this condition can be suspected by the presence of hydrops, serous effusions, subcutaneous edema, cardiomegaly and increased tricuspid regurgitation, an enlarged wave in the inferior vena cava, and pulsations in the umbilical vein, with reduced or no diastolic flow.
On the other hand, polyhydramnios by itself is associated with a worse prognosis and a 6-fold increase in the fetal mortality risk. Anemia and thrombocytopenia are frequently reported complications. These can be explained by tumor cell sequestration as well as by thrombotic microangiopathy.

Spontaneous regression of chorioangiomas as a result of infarction, with resolution of the associated hydrops fetalis, has been reported. In most cases, the size of the tumor stabilizes, and thus it stays asymptomatic, or else it grows and causes maternal and fetal consequences. Maternal complications include bleeding, preeclampsia, retroplacental hematoma, hemolytic anemia, and disseminated intravascular coagulation, but the main complications are mostly fetal and usually manifest as heart failure, polyhydramnios (14%–28%), intrauterine growth restriction (30%), and prematurity (40%), in addition to the risks of anemia, thrombocytopenia, and in utero death.

Several novel interventions have been proposed to block the vascular supply to the tumor as part of a curative approach and include endoscopic laser coagulation, fetoscopic ligation of the vascular pedicle of the tumor with bipolar electrocautery, and microcoil embolization. The use of interstitial laser therapy to devascularize the tumor has been reported in 3 cases, and they all resulted in a successful pregnancy outcome.

Prenatal management of chorioangioma cases has to be tailored to each case depending on the extent of tumor involvement with the fetal circulation and relies primarily on Doppler examination of the umbilical circulation. If the umbilical flow velocity waveform is abnormal (eg, reduced or no diastolic flow), a thorough evaluation of the functional state of the fetal circulation by means of evaluating fetal cardiac function and flow indices in the aorta, middle cerebral artery, and vena cava is warranted. Hydrops and fetal heart failure are medical emergencies. A rapid increase

Figure 1. Placental chorioangioma. A, Vascularization of the chorioangioma at 22 weeks. B, Macroscopic view of the vascular pedicle of the chorioangioma after delivery.
in the size of the chorioangioma may be a critical sign signaling the need for aggressive therapy and close surveillance to properly time fetal delivery. These cases should be managed in tertiary referral centers.

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References