Placental Chorioangioma Diagnosis and Management

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ABSTRACT
Placental chorioangioma constitutes major importance in perinatology practice, as being the most common type of placental tumors, and having the potential of devastating perinatal outcomes. In this review, we report the symptoms, diagnostic findings and recent temporary and definitive treatment choices in patients with chorioangioma.

Keywords: Placenta, Chorioangioma, Diagnosis, Management

Introduction

Chorioangioma, a benign placenta mass, is the most common type of placental tumors.1 The term ‘chorioangioma’ was first used in 1978 by John Clark.2 It is a non-trophoblastic tumor and the consequence of the abnormal proliferation of vessels arising from chorionic tissue.3 The overall reported incidence is about 1% however, clinically significant chorioangioma incidence ranges from 1 in 3500 to 1 in 9000 births.4

Symptomatology
In majority of the cases, chorioangioma does not present with any symptoms. However, the size and vascularization of the tumor seem as the most significant factors associated with the presence of maternal or fetal complications. Generally, the tumors >4-5 cm (giant tumors) with extensive vascularization are reported to exhibit relatively higher rates of drawbacks.5,6 The most common complications are severe polyhydramnios7 and mirror syndrome, fetal anemia, cardiac heart failure and non-immune hydrops fetalis,8 intrauterine growth restriction and fetal death9,10 and also preterm labor.11 Spontaneous evolution in the prenatal period with the resolution of polyhydramnios during the course of pregnancy, resulting in a birth of a full-term newborn without any complications may also appear even in a case of giant chorioangioma.12

Diagnosis and differential diagnosis
Ultrasound is the most commonly used tool in the prenatal diagnosis of placental abnormalities,13 and chorioangioma as well. Hypoechoic, well-circumscribed, ovoid or round intraplacental mass different from the rest of placenta containing small anechoic spaces that protruded into the amniotic cavity are the usual gray scale findings.14,15 In addition, low-resistance flow within the anechoic cystic areas and a central delivering arterial structure also with a low resistant waveform are the characteristics of chorioangioma in Doppler ultrasound14,16 (Figure 1).

Figure 1: An example of chorioangioma with its arterial supply

Teratoma of the placenta, degenerated myoma and blood clots are the lesions that should be differentiated from chorioangioma. Doppler is used to show the feeding vessel of chorioangioma and also the potential arteriovenous shunt with low resistance.17 The change in the echo pattern of blood clots with time and the localization of the myoma on maternal surface are the clues for the differential diagnosis.16

Magnetic resonance imaging (MRI) may also used in diagnosis and differential diagnosis.14 The reported MRI findings of the chorioangioma are; mass arising from the fetal surface of the placenta, demonstrating a signal intensity greater than that of the placenta both on T1- and T2-weighted images, relatively high signal intensity on proton density and T2-weighted images, and also rim of increased signal intensity on T1-weighted images.18,19
**Management Strategies**

Once the diagnosis of chorioangioma is established, optimal management strategy should be administered because of the potential serious prenatal complications and adverse pregnancy outcome. The management of giant choriangiomas predominantly depends on the symptoms and gestational age. However, with the perspective of evidence based medicine, there is no guideline or reported clinical advice. This circumstance represents chorioangioma management as a great challenge.

**Amnioreduction**

Serial amnioreduction is usually performed in case of chorioangioma and co-existent severe polyhydramnios. This procedure is generally performed to release maternal abdominal discomfort and dyspnea, and also allowing prolongation of pregnancy. However, severe polyhydramnios in chorioangioma cases is due to the hypercirculatory state, and not surprisingly, amnioreduction does not reveal a total recovery. Interestingly, it was also suspected that the decrease in intrauterine pressure after the decompression of amnion might result in elevated perfusion of chorioangioma, in other words, a 'steal' phenomenon and fetal deterioration may occur. Preterm premature rupture of membranes, placental abruption and preterm delivery are the other serious complications. Although amnioreduction has some success in favorable postnatal outcome, it should be performed in selected cases by taking in to account of the benefits and detriments.

**Intrauterine blood transfusion**

Placental chorioangioma may present with fetal anemia. Middle cerebral artery Doppler and cordocentesis in cases with >1.5 MoM of peak systolic velocity, should be performed in cases with chorioangioma to evaluate the presence of fetal anemia. The mechanism of the anemia was reported as the potential result of feto-maternal hemorrhage due to the shunting of large volumes of blood to the tumor and/or hemolysis because of the destruction of fetal erythrocytes in the exaggerated vasculature of chorioangioma. Treatment with intrauterine blood transfusion may advance fetal status and perinatal outcome.

**Intra-tumoral alcohol injection**

Successful management of pregnancies complicated by chorioangioma via alcohol injection was formerly described. The injection site may be either the major feeding artery of the chorioangioma or the sluggish circulation in the center of the tumor. The goal of this procedure is to block the vascular flow of the tumor so that to finalize the relief in the symptoms, which is also used as a therapeutic practice in arteriovenous malformations of the brain and acardiac twins. The theoretical consideration about the transition of alcohol to fetus was excluded by the confirmation of the undetectable levels of alcohol in cord blood (cordocentesis) just after the procedure. This technique is particularly suitable for the perinatology clinics in which the more complex procedures like radiofrequency or laser ablation is not performed and also no opportunity for further referral is obtained.

**Minimally invasive devascularization with laser, bipolar and radiofrequency ablation**

The disruption of the vascular supply of the chorioangioma should be the primary target for the absolute cure of this entity. In line with this attitude, Quintero et al. first advanced the fetoscopic ligation of the arterial supply with success in 1996. Today, with advancement in technology and interventional skills, intrauterine laser implementation seems to be the favorable modality in management. There are a number of reports which the invasive devascularization was used with acceptable success. Table 1 demonstrates the summary of the reported cases of chorioangioma between 2005-2015.

**Conclusion**

Despite all these reported management strategies of the chorioangioma, the benefit of treatment over conservative management in improving the fetal and neonatal mortality has not been conclusively demonstrated.

It should be emphasized that the placental chorioangioma management should be individualized. The size and vascularization of the tumor seem as the most significant factors associated with the presence of maternal or fetal complications. Generally, the tumors >4-5 cm (giant tumors) with extensive vascularization are reported to exhibit relatively higher chance of drawbacks.

Temporary solutions (i.e. intrauterine transfusion, amnioreduction, transplacental pharmacotherapy) or definitive treatment choices (i.e. surgical ligation/clipping, fetoscopic laser ablation, embolization, alcohol injection and radiofrequency ablation) or the combinations, which might exhibit satisfactory perinatal outcomes, should be performed for the proper perinatal practice.

**Table 1: Summary of the reported cases between 2005-2015**

| Author | Year | Tumor size | Prenatal complications | Treatment | Outcome |
|--------|------|------------|------------------------|-----------|---------|
| Cقدماس | 2015 | 85x47 mm | Polyhydramnios at 24 weeks of gestation | None (Spontaneous follow-up) | Spontaneous reduction of the amniotic volume, C/S due to cord prolapse at 38 weeks. 3250 gr, healthy newborn. |
| Author | Year | Tumor Size | Description | Treatment | Outcome |
|--------|------|------------|-------------|-----------|---------|
| Padys | 2015 | 60x50 mm | At 19 weeks of gestation, the tumor diameter was 40 mm. At 29 weeks, the chorioangioma had grown (60x50 mm). | Propranolol (oral) was given at a dose of 40 mg three times per day, in order to obtain the similar stabilizing effect on infantile hemangiomas, and maintained until birth. Unremarkable pregnancy follow-up with no change in size of the tumor. | Birth at 39 weeks, 3650 gr, healthy newborn |
| Jhun  | 2015 | 156 mm (diameter) | Polyhydramnios, severe heart failure | At 29 weeks of gestation, partial devascularization of the dominant feeding vessel with fetoscopic laser | Birth at 33 weeks, to persistent signs of fetal cardiac failure. After birth, the infant developed multifocal infantile hemangiomas with extracutaneous involvement. |
| Yen Lim | 2012 | Mean of 83 mm (A total of 8 cases) | 6 case had polyhydramnios, 3 had hydrops, 5 had high cardiac output | 5 case had intervention. (1 case had bipolar coagulation, 2 had bipolar + diode laser, 1 had bipolar and radiofrequency ablation, 1 had surgical clip application.) | 80% survival rate of intervention, hydrops disappeared in 2 / 2 and cardiac output normalized in 4 / 4. All were live born at mean of 35.4 weeks. |
| Ercan | 2012 | 55x51x49 mm | Polyhydramnios, threatened preterm labor | Amnioreduction + alcohol injection + intrauterine transfusion at 25 weeks’ gestation. | C/S at 29 weeks’ gestation, healthy 1510 new born. Discharged 2 weeks later |
| Babic | 2012 | 42x56x58 mm | Polyhydramnios and related maternal symptoms at 22 weeks. MCA PSV was 1.97 MoM for gestational age, with the development of mild pericardial effusion at 29 weeks’ gestation. Poor fetal right ventricular contractility with enlarged thick ventricular walls and mild pericardial effusion at 30 weeks and 2 days of gestation. | At 22 weeks amnioreduction and percutaneous injection of 1.5 mL of enbucrilate (liquid adhesive glue) in to the feeding vessel of the tumor + 50 mL blood transfusion (Hemoglobin 10g/dL → 14g/dL). At 29 weeks, 50 mL blood transfusion. At 30 weeks, betamethasone and elective C/S. | C/S at 30 weeks, live female baby 1.6kg, with Apgar score 5, 7 and 8. Discharged 6 weeks later in good condition. |
| Sepulveda | 2009 | 67 mm | Polyhydramnios, mild congestive heart failure | Endoscopic laser coagulation + amnioreduction at 26 weeks’ gestation | C/S at 37 weeks’ gestation, 3460 gr, good outcome |
| Sepulveda | 2009 | 58 mm | Polyhydramnios, mild congestive heart failure, short cervix (22 mm at 20 weeks) | Endoscopic laser coagulation + intrauterine transfusion at 27 weeks’ gestation | Preterm C/S at 28 weeks, chronic renal insufficiency, died at 1 year of age. |
| Sepulveda | 2009 | 85 mm | Polyhydramnios, congestive heart failure, fetal hydrops | Endoscopic laser coagulation | Intrauterine fetal death at 29 weeks’ gestation. |
| Bermudez | 2007 | 53x48x61mm | Polyhydramnios, congestive heart failure, fetal hydrops and anemia | Amnioreduction, laser ablation and intrauterine transfusion at 24 weeks. Repeated intrauterine transfusion at 25 weeks. | Intrauterine fetal death at 26 weeks’ gestation. |
| Deren | 2007 | 72x83 mm | Polyhydramnios, congestive heart failure, fetal hydrops at 16 weeks’ gestation. | Intrauterine transfusion at 16 weeks’ gestation. Alcohol injection into the tumor twice at 24 and 25th weeks gestation. | Preterm labour at 28 weeks, 1330 gr. Discharged 2 weeks later in good condition. |
Intrauterine transfusion at 25 weeks’ gestation

Spontaneous thrombosis of the main vessel of the tumor. C/S due to breech presentation, 3270 gr healthy fetus.

C/S due to breech presentation at 39 weeks, good outcome.

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