Ultrasound in the management of Chorioangioma

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ABSTRACT

Objective: To evaluate the role of ultrasound in the diagnosis and prediction of the clinical course of a large chorioangioma.

Methods: Six women with a large chorioangioma were consecutively detected at 26–28 weeks gestation by ultrasound. At initial examination, the tumors were evaluated for size, vascularity, location, and echogenecity (relative to placenta). Fetuses were assessed anatomically and for early signs of hydrops. Sonographic fetal and tumor examination were repeated weekly until delivery, and associated maternal complications were recorded.

Results: The sonographic diagnosis of chorioangiomas was subsequently confirmed pathologically in all cases. The maximum diameter of the tumors varied between 6 and 12 cm. Chorioangioma was hyperechogenic, avascular and complicated by intrauterine fetal growth restriction and oligohydramnios in one woman; a healthy but small for date infant was delivered at 38 weeks gestation. Two tumors were partially vascular and hyperechogenic, these tumors were associated with moderate polyhydramnios and preterm delivery of normal infants at 33 and 36 weeks. The remaining 3 tumors were hypoechogenic, diffusely vascular and were associated with severe polyhydramnios. In one case, the tumor was complicated by abrupt generalized non immune fetal hydrops and stillbirth; in the 2nd case, there was acute fetal distress and delivery of a premature anemic infant at 28 weeks; however, in the 3rd case, while the polyhydramnios and tumor vascularization decreased, its echogenecity increased and delivery of normal infant at full term was achieved.

Conclusion: Sonographic assessment of echogenicity and vascularity of the large chorioangioma appears to be detrimental in predicting the clinical outcome of pregnancy. Spontaneous regression of tumor vascularity with subsequent resolution of hydramnios may occur. While vascular and hypoechogenic tumors are associated with higher incidence of pregnancy complications, favorable outcome is expected in avascular and hyperechogenic tumors.

Keywords: Placental tumors, ultrasound diagnosis, chorioangioma.

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Large chorioangiomas of over 5 cm in diameter are easily diagnosed by conventional ultrasound examination; most of these tumors protrude from the fetal surface of the placenta, near the insertion of umbilical cord.[1] They appear by ultrasound as well-delineated and circumscribed masses with different echogenecity from the placenta.[1,2] Large chorioangiomas are rare and their incidence varies between 1 in 5000 to 1 in 50,000.[1,2] Large chorioangiomas are associated with increased risk of fetal and maternal complications such as polyhydramnious, preterm delivery, antepartum hemorrhage, intrauterine fetal growth restriction, fetal anemia and thrombocytopenia, non-immune fetal hydrops, fetal cardiomegaly, hepatomegaly, and congenital malformations.[1-3] The present study describes the ultrasonographic features of large chorioangioma and the role of ultrasound in the diagnosis and management of this rare placental tumor.

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Methods. Between November 1993 and March 2000, a total of 6 consecutive women with chorioangioma were detected by abdominal ultrasound scan. These women were referred because of suspected fetal complications. The same operator scanned all patients using a Kretz Combison 310 Real time scanner (Kretztechnik GmbH, Zipf, Austria), or Aloka SSD-2000, (Alcoa Co, Japan) which is equipped for pulsed Doppler and color Doppler imaging (CDI). The diagnosis of large chorioangioma was made sonographically when a well-circumscribed tumor of more than 5 cm, having different echogenicity from the rest of placenta was documented. The vascularity of the tumors was assessed with CDI; the size and distribution of tumor blood vessels were recorded at initial examination. When the depth of the largest pocket of amniotic fluid measured 8-14 cm, it was defined as moderate hydramnios, while a pocket depth of more than 15 cm was considered as severe hydramnios. Foetuses and placental tumors were assessed in detail at initial ultrasound examination; Serial ultrasound and Doppler examination at weekly intervals were performed to detect early signs of fetal distress and hydrops. All women were given a one-day course of dexamethasone at 24 weeks gestation.

Results. During the study period there were a total of 31670 deliveries in the Department of Obstetrics. Of these, 6 cases (1 in 5300) of large chorioangioma were diagnosed sonographically and confirmed by the subsequent pathological examination. They were referred for sonographic examination because of associated fetal complications, suspected intrauterine fetal growth restriction in one case and polyhydramnios in the remaining 5 cases. Table 1 summarizes the clinical and sonographic findings at initial presentation. The tumors were protruding from the chorionic plate into the amniotic cavity in all cases. They had well-defined circumscription and complex echogenecity, and were centrally located near the insertion of umbilical cord in 4 cases and marginally located in 2 cases. Arterial flow with an identical pulsation rate to that in the umbilical arteries was demonstrated in all cases except case one. The tumors size had not changed throughout pregnancy in all the cases. At presentation, the tumor in case one appeared avascular at CDI and hyperechogenic (Figure 1), and the depth of the large amniotic fluid pocket (AFD) was 2 cms. Repeated fetal assessment showed normal biophysical profiles and the growth pattern was constant at 10th centile line. A normal but small infant weighing 2.2 kg was delivered at 38 weeks. About half of the tumor in cases 2 and 3 was avascular and hyperechogenic (Figure 2) while the remaining parts were hypoechogenic and vascular. The AFD were 9 and 12, and the fetuses were normal. Hydramnios, echogenicity and vascularity did not change during pregnancy, and spontaneous preterm labor at 33 and 36 weeks resulted in delivery of normal infants with good Apgar score, weighing 1.9 Kg and 2.4 Kg. The 3 pregnancies presenting with vascular and hypoechogenic chorioangiomas were complicated by severe hydramnios (AFD >15 cms). The amniotic fluid index remained almost constant during pregnancy in cases 4 and 5. In case 4, the fetus developed abrupt and generalized non immune fetal hydrops (NIFH) at 26 weeks gestation; it had ascites, bilateral pleural effusions, gross skin edema (Figure 3) and a fetal heart rate of over 160

Table 1 - Clinical and sonographic findings at initial presentation in 6 cases of chorioangioma.

Case	Gestational age (weeks)	Tumor's size (cm)	Tumor's location	Echogenecity	Vascularity	Maternal and Fetal complications
1	28	7x4x6	Marginal	Hyperechoic	Avascular	Oligohydramnios, IUGR
2	22	7x5x4	Marginal	Hyperechoic center, hypoechoic margins	Peripheral vascularization	Moderate hydramnios
3	24	6x4x4	Central	Hypoechoic center, hyperechoic margins	Central vascularization	Moderate hydramnios
4	22	12x8x8	Central	Hypoechoic	Diffuse vascularization	Severe hydramnios
5	23	6x4x3	Central	Hypoechoic	Diffuse vascularization	Severe hydramnios
6	25	6x5x5	Central	Hypoechoic	Diffuse vascularization	Severe hydramnios

IUGR=Intra uterine growth restriction

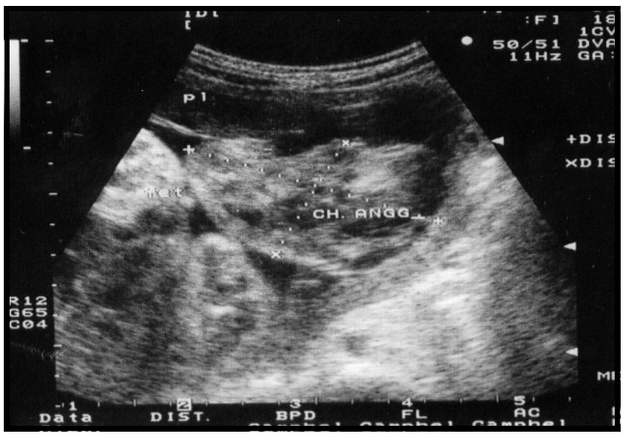

Figure 1 - Ultrasound showing hyperechogenic chorioangioma located at placental margin, the tumour was avascular on color doppler imaging. PL=placenta, CH ANGO=chorioangioma (case 1).

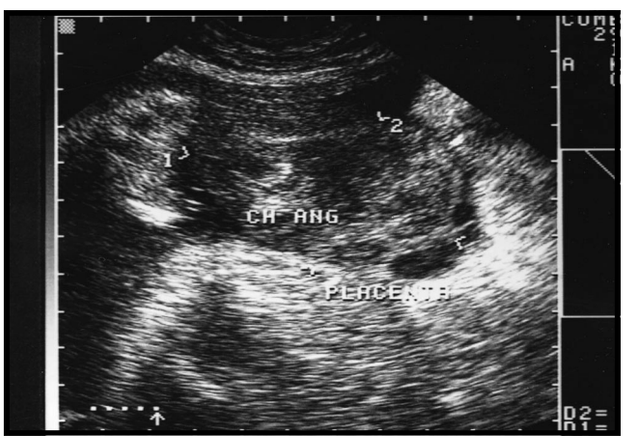

Figure 2 - Abdominal ultrasound at 24 weeks, showing large chorioangioma located centrally. Note the hyperechogenic periphery and hypoechogenic center, CH ANG= chorioangioma, (case 3).

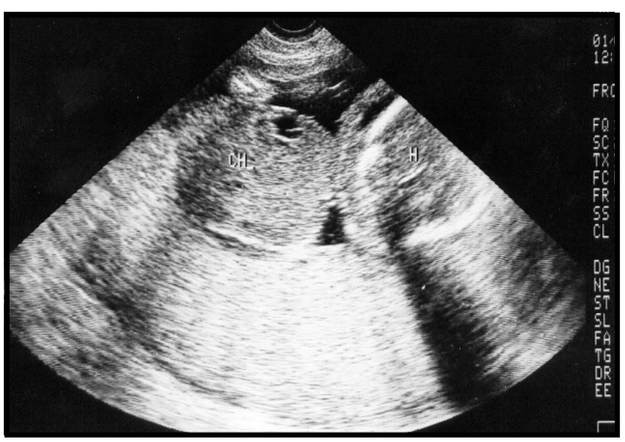

Figure 3 - Large hypoechogenic, centrally located chorioangioma. The tumor was very vascular on color doppler imaging. There is evidence of gross scalp edema, CH=chorioangioma, H=fetal head, (case 4).

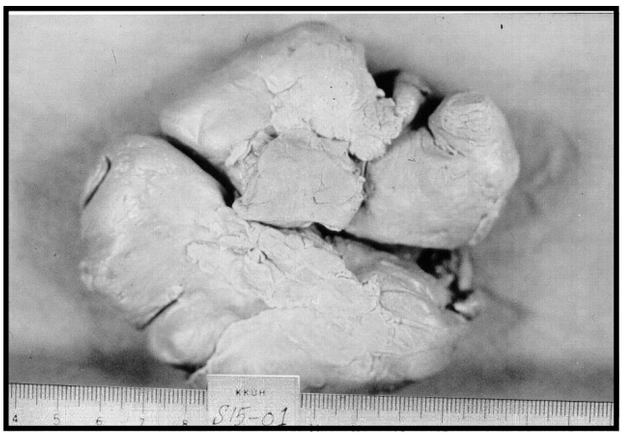

Figure 4 - Gross appearance of chorioangioma, measuring 11x9x4 cm, (case 4).

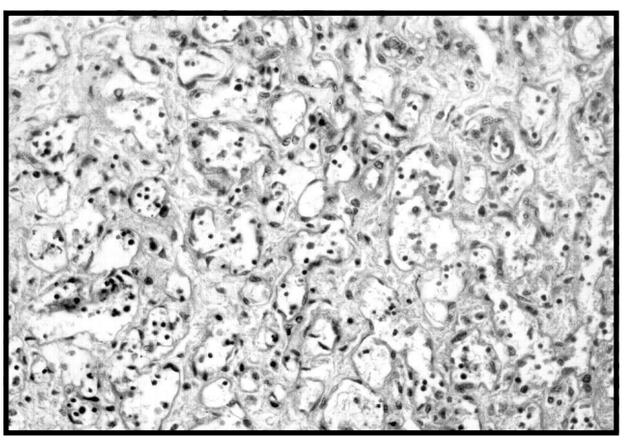

Figure 5 - Histology of case number 4 tumor; chorioangioma with numerous proliferating blood vessels separated by loose connective tissue.

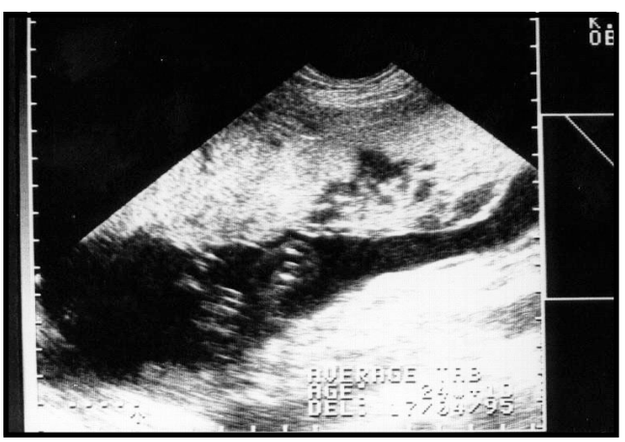

Figure 6 - Focal aneurysmal dilatation of subchorionic blood vessels simulating chorioangioma. Note the ill-defined border of the vascular mass, and the associated hydramnios.

with recurrent episodes of bradycardia. After counselling, the parents opted for cesarean section, however fetal heart stopped beating within an hour of presentation and a stillborn infant was delivered vaginally. Placenta and its tumor were manually removed. The tumor was solid and vascular, measuring 11 x 9 x 4 cms (Figure 4). It had a vascular stalk containing 2 big arteries that connected the chorioangioma to the umbilical cord. Histopathological sections of the tumor showed numerous capillaries of various sizes, which were separated by rather loose connective tissue (Figure 5). The fetus in case 5 had reduced fetal activity at 28 weeks gestation, and fetal heart tracing showed significant reduced variability with late deceleration. An emergency cesarean section was performed and an anatomically normal infant weighing 1.3 kg was delivered. The infant was found anemic (Hemoglobin = 11g/dL) but the baby made a complete recovery after blood transfusion. The severe hydramnious in case 6 subsided gradually, so that the amniotic fluid volume was normal at 32 weeks gestation. The reduction of amniotic fluid was associated with increased echogenecity and decreased vascularity of the tumor. A normal fetus weighing 3.6kg was delivered at full term. Histopathological examination of the tumor showed small but multiple areas of degeneration with micro-calcification. There was no evidence of arterio-venous shunt in any of the 6 chorioangiomas.

Discussion. Chorioangioma is the most common benign placental tumor, with an incidence of 0.5-1% of placenta examined pathologically.[2] Most of these tumors are small without any clinical significance and are unlikely to be detected during gross pathological examination, unless the placenta is systematically sliced.[2] Feto-maternal complications are only found in association with large tumors measuring over 5 cm in diameter.[2] Asokan et al in 1978 was the first to diagnose chorioangioma by the ultrasound.[4] Since then, several papers have described the sonographic features of large placental tumors. With the advent of routine use of antenatal ultrasonography; the majority of large chorioangiomas are diagnosed during the 2nd trimester, particularly when they are associated with hydramnious or other fetal complications. Most of the large chorioangiomas appear sonographically as single, encapsulated, intraplacental but mainly subchorionic tumors. They protrude into amniotic cavity, and are located centrally near the insertion of the umbilical cord or at the placental margin and have a different echogenecity from the rest of the placenta. Two rare placental lesions may present confusion with chorioangioma. The first lesion is the focal mesenchymal dysplasia where part of the placenta is hypertrophic but the amniotic fluid is not changed and the fetus is structurally normal.[5] The other lesion is the focal aneurysmal dilatation of the subchorionic vessel (Figure 6); it is a vascular mass with irregular shape and ill-defined borders, and associated with moderate hydramnious but normal fetal outcome.[6] Color Doppler imaging and Doppler velocimetry have been used recently to assess the vascular dynamic of large chorioangiomas. Isolated case reports have investigated the role of CDI in the antenatal diagnosis and management of this rare tumor.[7-10] In this study, the correlation between gray scale echogenecity and tumor's vascularity was evident. The hyperechogenic tumor was avascular on CDI (case one), and in cases 2 and 3, only the hyperechogenic parts of the tumors were avascular, on the other hand, the 3 hypoechogenic tumors were hypervascularized on CDI at initial presentation. Gradual decrease of vascularity with parallel increase in tumor's echogenicity was observed in case 6. The regression of severe hydramnios in this case and the normal fetal outcome can be explained by the possible development of thrombosis in feeding blood vessels. This possibility is supported by the subsequent postpartum pathological examination of chorioangioma in case 6, which revealed numerous areas of necrosis, calcification and fibrous degeneration. Spontaneous regression of fetal hydrops and polyhydramnios has been previously reported.[11-12] In addition to case one in this paper, several other case reports have shown that large avascular or cellular chorioangoma are not associated with feto-maternal complications.[9-12] The associated moderate IUGR in case one cannot be linked directly to the avascular nature of the tumor, as the fetus was structurally normal and showed constant growth pattern after presentation at 28 weeks. The patient was seen too late to evaluate the tumor initial vascularity. It is reasonable to assume that such a large tumor was originally vascular and spontaneous thrombosis and necrosis may have occurred at any stage of pregnancy prior to presentation. This assumption is based on the pathological findings of widespread degenerative changes in the tumor, the documented vascular changes of chorioangioma in case 6, and literature review.[8-12] In the present study, amniotic fluid volume seems to be directly proportional to tumor vascularity in all cases. Moderate hydramnios had contributed to preterm labor in case 2 and 3, however treatment with amnioreduction was not attempted in both cases as repeated amniocentesis has only a short term benefit in a pregnancy of less than 32 weeks, and in the absence of early signs of fetal hydrops.[9] Three main theories have been postulated to explain the occurrence of hydramnious in association with chorioangioma[2] and these are: Mechanical obstruction of the umbilical vessels by the tumor, cardiac failure with its accompanying fetal fluid imbalance and excessive transudation of fluids from the wall of abnormal vessels of chorioangioms into

amniotic cavity, through the tumor and fetal placental plate.[13] Only the last theory could explain the pathophysiology of hydramnious in chorioangioma. In the majority of the reported cases of vascular chorioangioma, alpha-fetoprotein and amniotic fluid volume are increased while in the avascular and degenerated tumor amniotic fluid is normal.[7,10-12] These findings support the concept of excessive fluid transfer from the vascular tumor to the amniotic cavity. Nonimmune fetal hydrops may develop in vascular chorioangioma as in case no. 4. Literature review revealed that only in 2 cases of chorioangioma, the anatomical arteriovenous shunts were demonstrated pathologically.[14,15] In fact none of the cases presented in this study have pathological findings of anatomical arteriovenous shunting. The cause of generalized fetal hydrops in vascular chorioangioma could be either due to vascular stasis causing chronic fetal hypoxia and loss of plasma proteins to the serous cavities or interstitial space,[17] or it could be secondary to either microangiopathic hemolytic anemia resulting in fetal hydrops,[16] or to functional shunting of large volume of fetal blood through the tumor with consequences of increased venous return and congestive heart failure.[16] Hypervascularization of chorioangioma is a common abnormality in all of the 3 theories of pathogenesis of fetal hydrops; therefore successful fetal outcome should be expected if the tumor is devascularized. Alcohol has been used recently, with variable success, to obliterate the large veins at the centre of placental chorioangiomas.[10,18] The widespread use of ultrasound plays an important role in early detection, differential diagnosis and management of chorioangiomas. Fetal and maternal outcome depend on echogenicity and vascularity of the tumor. Avascular tumors are not expected to be associated with fetal or maternal complications. Serial and frequent ultrasound examinations are required for hypoechogenic vascular tumors in order to detect changes in tumor vascularity, fetal anaemia, and early signs of congestive heart failure or fetal hydrops. Invasive therapeutic procedures should be considered in complicated chorioangiomas before 28-32 weeks gestation, however termination of pregnancy is an appropriate management for pregnancies of over 32 weeks gestation, and in the event of development of early signs of hydrops or fetal distress.

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