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Case Report

Large placental chorioangioma, cause for gross polyhydramnios; experience of two cases and review of literature

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ABSTRACT

Placental chorioangioma is a benign vascular tumor most frequently diagnosed by sonographic and Doppler evaluation. Although it is not uncommon (1% incidence) cases which measure more than 4 cm in diameter are rare. Large chorioangioma can be associated with pregnancy complications. These include hydrops fetalis, growth restriction, fetal or neonatal demise, polyhydramnios and preterm birth. We report here 2 cases of large placental chorioangioma resulting in polyhydramnios, intrauterine growth restriction and preterm birth. Both the tumors were more than 5 cm and on ultrasound were seen as a hypoechoic lesion in continuation with upper pole of placenta projecting into the amniotic cavity .Gross examination showed a globular mass arising from the fetal surface of placenta and in both the cases histopathological examination was suggestive of placental chorioangioma along with areas of calcification. The maternal outcome was good but one of the babies died due to very low birth weight.

Keywords: Chorioangioma, Placenta, Polyhydramnios, Preterm labour

INTRODUCTION

Chorioangioma is a benign vascular tumor of the placenta consisting of a vascular mass arising from the primitive chorionic mesenchyme with a reported prevalence of 1%. It is one of the rare cause of polyhydramnios. Tumors measuring > 5cm are rare and are called giant chorioangioma. Although small tumors are asymptomatic giant chorioangiomas are responsible for maternal and fetal complications which include intrauterine fetal growth retardation, hydrops fetals, fetal anemia, polyhydramnios, pregnancy induced hypertension, preeclampsia, antepartum haemorrhage and preterm labour.[1]

CASE REPORT

First case is a 23 years female second gravida with previous one normal vaginal delivery and with no significant past medical or surgical history. She was referred from a district hospital as a case of subchorionic haematoma at a gestational age of 28 weeks. She had no complaints on admission .On examination she had mild pallor, edema feet, pulse rate of 82/min and BP of 120/70 mmHg. Systemic examination was normal and on per abdomen height of uterus was more than the period of ammenorrhoea that is 32 weeks, fetal heart could not be located with stethoscope and there was no uterine activity. An ultrasound revealed a normal fetal growth for gestational age of 29 weeks 1 day with estimated fetal weight of 1.4 kg and no signs of fetal hydrops and adequate liquor of 17.6 cm with posterior placenta involving a well-defined round to oval heterogenous lesion predominantly hypoechoic lesion of approximate size 8.6 X 7.0 X 7.5 cm noted involving the fundal end of placental margin. Lesion showing nodular echogenicity within and mild vascularity within. Internal vessels showed an MCA PSV of 25.0 cm/s, RI 1.0, PI 1.39 s/o

neoplastic etiology. Color Doppler USG was normal. Her investigations were normal.

After a week patient started developing over distension of uterus and a repeat scan showed polyhydramnios with AFI of 35 cm and increase in the size of lesion which measured around 10.3 x 9.2 x 9.8 cm. The lesion showed mild vascularity within internal vessels. Patient was treated with indomethacin, tocolytics and steroids for lung maturity. There was progressive increase in polyhydramnios with the patient experiencing shortness of breath and respiratory embarrassment and AFI was 43 cm and tumor size being 11.0 x 10.9 x8.4 cm with few areas of calcification seen but color Doppler was normal. Hence amnioreduction was done and about 500 ml of Amniotic fluid was removed. Her Amniotic fluid index dropped to 31 cm but the size of tumour increased to 11.3 X 10.6 x8.4 cm. 15 days after amnioreduction again there was increase in polyhydramnios with largest pocket measuring 13 cm .The patient had premature rupture of membranes and had a preterm delivery at 32 weeks of gestation . A female baby was born vaginally with a birth weight of 2 Kg. Baby cried immediately after birth .Baby was admitted in PBU for prematurity and postnatal period was uneventful.

On gross examination of the placenta, placenta weighed 1155 gm and fetal surface showed globular solid mass of size 12x11 x10 cm arising from fetal surface with reddish brown color. On maternal surface mass showed congested vessels. Section from solid nodular mass showed proliferation of capillaries with myxoid stroma. Tumor mass was well encapsulated with areas of calcification seen and histological features suggestive of chorioangioma.

Second case is a 20 years old primigravida who was admitted at a gestational age of 32 weeks with polyhydramnios and pain in abdomen. On examination she was mildly pale with a pulse rate of 86/min and BP of 130/80 mmHg. Her systemic examination was normal. Per abdomen uterus was over distended upto xiphisternum and fetal heart were not audible with the stethoscope and uterine contractions were not observed. On per vaginal examination cervical os was admitting tip with 25% effacement and with a adequate pelvis. Her USG was done which revealed a single live intrauterine fetus of average gestational age of 26 weeks 5 days with fetal growth restriction and with AFI of 38 cms and estimated fetal weight of 968 grams. Placenta was fundoposterior with a presence of well-defined rounded heterogenous complex cystic lesion noted in fundal region attached to placenta of size 8.6 x 7.0 x 6.2 cm showing anechoic areas s/o benign placental lesion most likely chorioangioma. There was no gross anomaly. On color Doppler lesion showed mild to moderate vascularity in lesion and also few linear oblique serpengineous hyperechoic areas within suggestive of calcifications. Her investigations were within normal limits. She was started on antibiotics, steroids and tocolytics. Patient had premature rupture of membranes at 34 weeks of gestation and went in spontaneous labour. A preterm female baby was born vaginally with a birth weight of 1 kg .The baby died after 6 hours of birth due to very low birth weight with pulmonary haemorrhage and respiratory failure. On gross examination there was a globular nodular mass of size 8x7x5 cm arising from fetal surface and weight of the placenta was 1000 gm. The postnatal period was uneventful and histopathology confirmed the diagnosis of placental chorioangioma.

DISCUSSION

Placental chorioangioma is the most common placental tumor with an estimated prevalence of 1% in systematic placental histologic studies. Clarke in 1978 described the first case of chorioangioma. The first case of prenatal sonographically diagnosed chorioangioma was reported by Asokan et al.[2] It is a benign vascular tumor most frequently diagnosed in the second trimester of pregnancy by sonographic and Doppler evaluation. Chorioangiomas are believed to originate at about the 16th day after fertilization although there is no documentation of chorioangioma during the first trimester.[3] The description of sonographic diagnosis have been limited only to a few cases is due to the fact that only tumours more than 5 cm are associated with clinical manifestations and the prevalence of these large tumors is smaller varying from 1 per 500 to 1 per 16000 placentas examined.[4] Increased maternal serum alphafetoprotein and B-HCG can arouse the suspicion which could not be done in our cases due to limited resources. Color Doppler imaging not only helps to differentiate chorioangioma from other lesions like degenerating fibroid, placental teratoma , deceased twin , placental haematoma, partial hydatiform mole but also helps in prenatal follow up of these cases.[5]

Sepulveda W et al concluded that placental chorioangioma is associated with an increased risk of pregnancy complications, the most common being Polyhydramnios and preterm delivery which were similar to that found in our patients.[1] Both of our cases had gross polyhydramnios with AFI>35 cms and ultimately had spontaneous preterm delivery. The pathophysiology of maternal and fetal complications is not well understood. Theories for polyhydramnios include a) transudation of fluid caused by a mechanical obstruction of blood flow by the tumour near the cord insertion, b) increased transudation of fluid through a large vascular surface area, c) functional insufficiency of the placenta secondary to by passing foetal circulation via shunt mechanism into the tumor vascular bed.[6] Polyhydramnios <14-33%> independent of tumor size and is probably related to the vascularity of the tumour and fluid leakage. Polyhydramnios and fetal hydrops may spontaneously regress when the chorioangioma degenerates.[7,8]

Sepulveda W et al reported that pregnancy with large chorioangioma more than 4 cm in diameter are rare and

large chorioangioma can be associated with pregnancy complications which include hydrops fetalis, (heart failure), growth restriction (poor growth of the baby), fetal or neonatal demise and preterm birth.[1]

Chorioangioma is usually treated with expectant management as majority of the tumors are asymptomatic. Small tumors are usually monitored with USG every 6-8 weeks, whereas large highly vascular tumors require serial USG examination with Doppler flow studies every 1-2 weeks as in such instances, the foetus is at risk of high output cardiac failure, hydrops and premature delivery due to polyhydramnios. Jauniaux et al reported that polyhydramnios can be directly treated by repeated amnioreduction thus reducing the maternal discomfort and the risk for preterm delivery or spontaneous rupture for membranes.[7] Sreelakshmi K et al reported the treatment of polyhydramnios with therapeutic amniocentesis, maternal indomethacin therapy and steroid administration for acceleration of fetal lung maturity before 34 weeks which was the treatment , also given in one of our case.[9] Quintero et al has advocated in utero interventions to improve the perinatal outcome such as ultrasound guided or endoscopic laser devascularisation in fetus with early features of congestive cardiac failure and or hydrops.[10] Nicolini recommended injection of thrombogenic materials and Lau et al recommmended microcoil embolization.[11,12] Kung FT et al showed calcifications, reduction of the tumour size and improvement in clinical outcome.[13] Similar was the finding in our cases where we found calcification in the tumor with reduced flow of blood within the lesion. One of the case had a favourable outcome and pathological examination also confirmed the presence of calcifications.

CONCLUSION

Careful visualisation of the placenta on ultrasonography in cases of polyhydramnios is required as chorioangioma is one of the rare cause of polyhydramnios. Giant chorioangioma is associated with adverse fetal outcome and hence requires timely detection. Regular follow up of the patients with placental chorioangiomas with serial USG and color Doppler is required as they can increase in size and cause maternal discomfort and large chorioangiomas are associated with high perinatal morbidity and mortality as in one of our case. Also timely referral of such cases to a tertiary care center to prevent complications is warranted.

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