

Case Report

CHORIOANGIOMA OF THE PLACENTA – A RARE CAUSE OF POLYHYDRAMNIOS

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ABSTRACT

Polyhydramnios complicates about 1% of all pregnancies. Chorioangioma of the placenta is a rare cause of polyhydramnios which may be associated with adverse foetal outcome. A careful antenatal ultrasound with colour Doppler can easily diagnose the condition. Presenting a case of acute polyhydramnios with a large chorioangioma diagnosed antenatally on ultrasound. Patient was kept under close surveillance and had a favourable maternal and foetal outcome.

Key words: Chorioangioma, Placenta, Polyhydramnios, Ultrasound, Doppler

INTRODUCTION

Polyhydramnios is defined as amniotic fluid in excess of 2000ml. An Amniotic fluid index (AFI) of greater than 24 cm or deepest vertical pocket of fluid of 16 cm or greater on ultrasonography denotes polyhydramnios (Williams Obstetrics, 22nd edition). It is identified in about 1 percent of pregnancies. It is easily diagnosed clinically and confirmed on USG. Once detected, a thorough work up for the cause begins which includes evaluation of foetus especially for CNS and gastrointestinal tract anomalies and maternal evaluation for diabetes mellitus. Because of the tendency to focus on the foetal and the maternal causes, one may miss another rare cause of polyhydramnios i.e. placental tumours. Presenting a case where chorioangioma was the cause of polyhydramnios and maternal respiratory embarrassment.

CASES

A 25year old primigravida was admitted to labour room at 32 weeks gestation with history of dull aching continuous pain right hypochondrium, respiratory embarrassment and bilateral lower limb oedema since one week. She also complained of abdominal distension which progressed rapidly over one week. The accompanying respiratory embarrassment was so severe that she had been sleeping in a chair for the past one week. She also developed oedema in bilateral lower limbs which did not subside on rest. . There was no history of increased BP recording, visual disturbances or seizures. Patient was non-smoker, non-alcoholic and vegetarian. She had a strong family history of diabetes mellitus as her father and grandfather were diabetic. Patient was booked with a senior obstetrician in a private nursing home and her 1st and 2nd trimester had been uneventful. USG examination done at 18 weeks was normal with singleton pregnancy, AFI of 14, no congenital malformations in foetus and a fundal placenta. USG done at another hospital 1 day prior to admission revealed a single foetus in cephalic presentation. The foetal parameters corresponded to 31 weeks 5 days. No foetal congenital malformations were noted. Amniotic fluid volume was markedly increased with largest vertical pocket measuring 15 cm and AFI of 28 cm. Placenta was fundal with Grade 2 maturity. There was a well-defined heterogeneous hypo echoic mass measuring 5x4 cm arising from the foetal surface of the placenta with extensive vascularity on colour Doppler suggestive of chorioangioma of placenta. There was no evidence of foetal hydrops and foetal Doppler flow study was normal (Figure1).

At admission, on general physical examination, patient was moderately built and nourished, afebrile, pulse rate was 96/min, regular and good volume. JVP was not raised. Her respiratory rate was 32/min and her dyspnoea was worse on lying down. Her blood pressure was 130/80 mmHg in right arm in sitting

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position. She had bilateral pedal and pre tibial pitting oedema. There was no pallor, cyanosis or clubbing. Cardiovascular System examination was normal. Respiratory System examination showed normal vesicular breathing with no adventitious sounds. Abdominal examination revealed an over distended and tense uterus corresponding to 36 weeks size. There was mild tenderness over the fundus of the uterus on the right side. Foetus was in longitudinal lie but the presentation could not be appreciated. Liquor was increased. The uterus was relaxed ie.no contractions were felt. There was no other palpable mass. Abdominal girth was 48 inches. Foetal heart rate was heard with hand held foetal Doppler and was 150 beats per minute, regular. Non Stress Test (NST) was reactive.

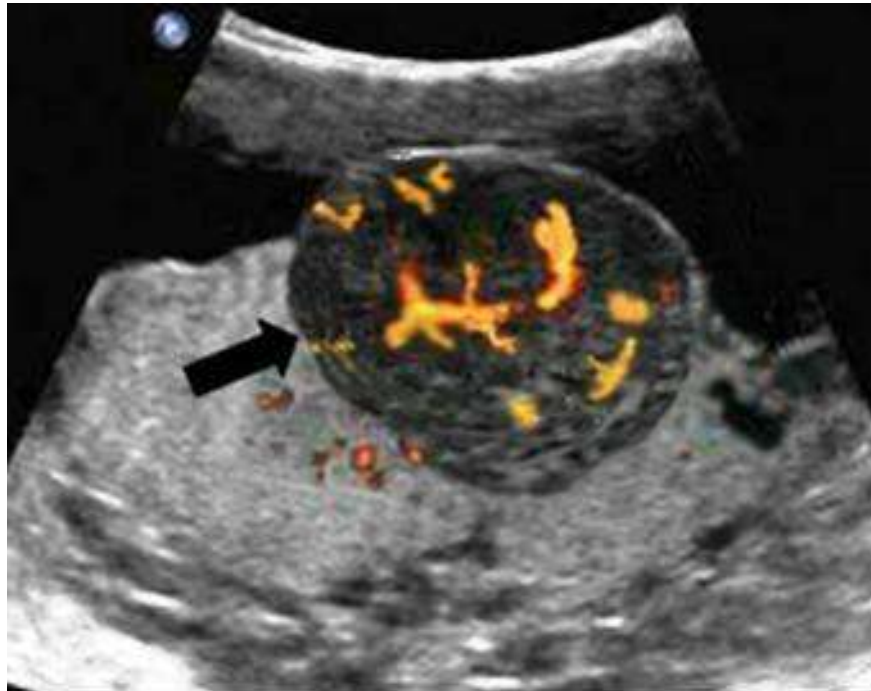


Figure1: Ultrasound showing a 5x4cm mass arising from placental surface with extensive vascularity on colour Doppler

Her routine blood and urine investigations were normal. Glucose tolerance test using 100gm glucose was also normal. USG guided amnioreduction was done using 18 gauge needle under local anaesthesia. One litre of liquor was drained gradually over a period of 2 hours and the patient was relieved of respiratory distress. She was managed conservatively with complete bed rest and tocolysis (Tab. Isoxuprine hydrochloride 40mg BD). 2 doses of injection betamethasone 12 mg I/M were given 24 hours apart. Patient was monitored with biweekly NST and weekly USG and Doppler. There was no further increase in amniotic fluid volume on serial ultrasounds and the AFI remained 11- 12 cm. There was no evidence of foetal growth restriction. Middle cerebral artery blood flow studies on Doppler did not show any evidence of foetal anaemia. There was no evidence of foetal hydrops on repeated scans.

USG done at 36 weeks and 3 days revealed a slight increase in tumour size to 5.8 x 4.8cm with AFI of 12 cm. Estimated foetal weight was 2.45 kg. Per vaginal examination revealed a soft, posterior, uneffaced cervix with os closed. Vertex was at -3 station. Decision was taken to terminate the pregnancy in view of increase in the size of the tumour. Patient refused to give consent for induction of labour and requested to be delivered by caesarean section. She was taken up for elective LSCS at 36 weeks and 5 days and a female baby weighing 2.45 Kg was delivered. Placenta weighed 700gms and its foetal surface showed a nodular growth measuring 6 x 5 cm with large vessels running over its surface. Rest of the placenta and umbilical cord was normal (Figure2).

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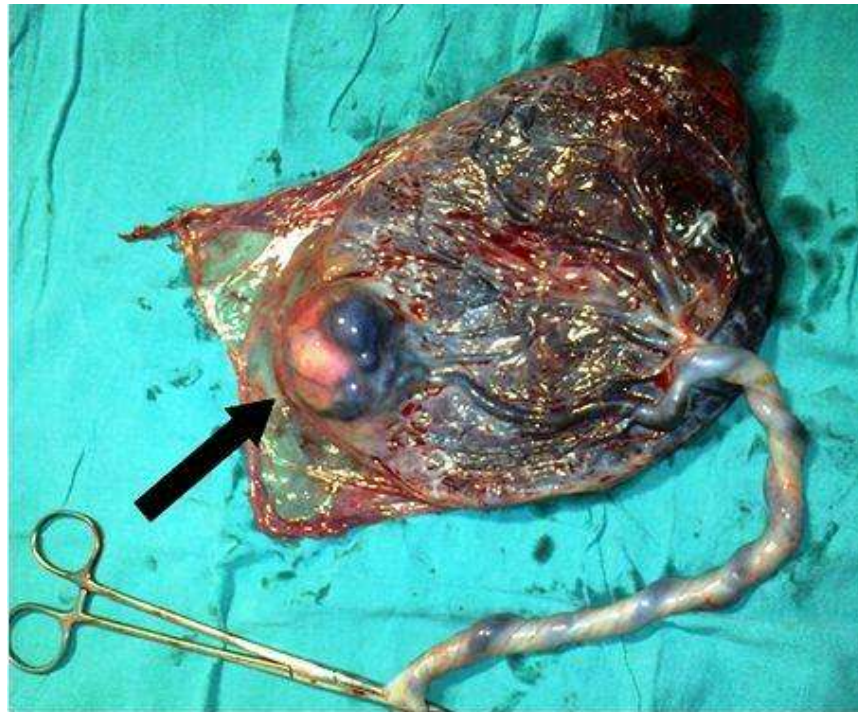


Figure2: Placenta showing a 5 x 6cm nodular growth with large vessels on the surface

Histopathology of the placenta showed a nodular lesion measuring 6x5x4 cm composed of capillary sized blood vessel supported by a loose stroma. The adjacent placenta showed features of chorioangioma with prominence of syncytial knots and micro calcifications (Figures 3A and 3B).

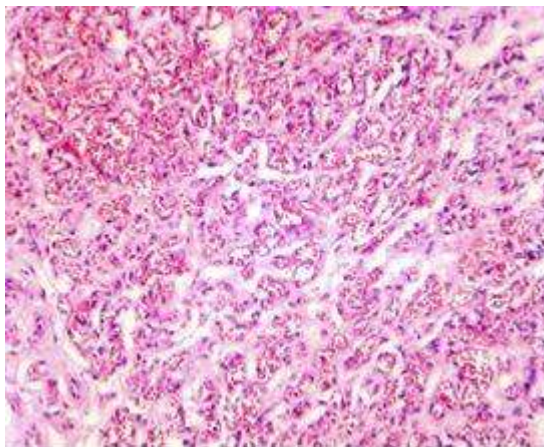


Figure 3A

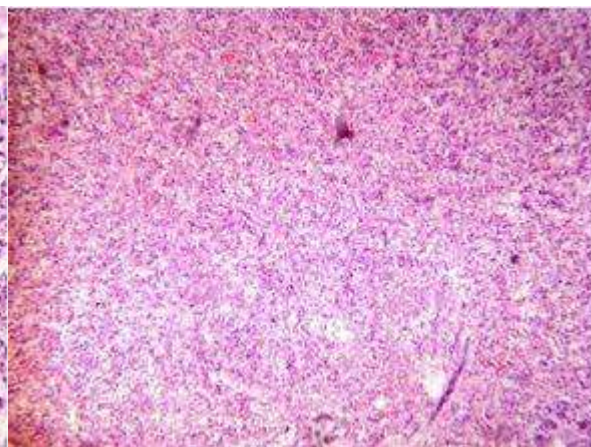


Figure 3B

Figure 3 –Microscopic examination showed a cellular tumour composed of endothelial cells and blood vessels separated by loose placental stroma, Low power (A) and High power (B) view on haematoxylin and eosin stain

Baby cried immediately at birth but had excessive secretions and was intubated and endotracheal suction was done. Chest X-ray was normal and there was no evidence of tracheo esophageal fistula. She was extubated the same day. Hemogram of the baby at birth was Hb- 15.2 gm%, PCV – 44.1, TLC- 14,400/cu mm, platelets- 1.94L/cu mm; CRP was 0.2mg/dL, Serum Calcium – 9.6mg/dL, blood group – B+. Baby developed mild physiological jaundice and was discharged on day 5 of birth after phototherapy.

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DISCUSSION

Vascular tumours comprise the most common group of benign placental growths with an incidence of 1% (Reshetnikova *et al.*, 1996). Chorioangioma is a benign vascular tumour arising from the primitive chorionic mesenchymal tissue. According to Guschmann *et al.*, (2003), the true prevalence of this tumour is likely unknown because many are thought to be undetectable without careful sectioning of the placenta as most chorioangiomas are minute and singular. In fact, in one clinical series of 136 chorioangiomas, more than half of all tumours were discovered by using only histologic techniques. They are unlikely to be recognised on ultrasound when less than 3cm in size (Text Book of Diagnostic Ultrasound, 2005). Large chorioangiomas ≥ 5 cm or multiple chorioangiomas have been reported to occur at a rate of 1:3500 to 1:16,000 births (Esen *et al.*, 1997).

Ultrasound can easily detect placental tumours larger than 5cm in size. Gray scale findings include intraplacental subchorionic location, well defined circumscribed growth, with complex echogenicity different from the rest of the placenta and protrusion into the amniotic cavity near the insertion of the umbilical cord. Colour Doppler imaging shows vascular channels in the tumour which show pulsatile flow at the same pulsation rate as the umbilical cord. MRI is the diagnostic modality of choice in patients where USG is equivocal.

The differential diagnosis of placental tumors includes partial hydatidiform mole, placental hematoma (intraplacental or subchorionic), teratoma, metastases, and leiomyoma. They are easily distinguished from fibroids because they are contained within the substance of the placenta and contain foetal vascular flow patterns. The use of colour Doppler imaging confirming the presence of vascular channels in the tumour contiguous with the foetal circulation helps exclude teratomas, and incomplete hydatidiform mole (Zalel *et al.*, 2002). According to Sepulveda *et al.*, (2000) feeding vessels may have arterio venous shunt causing low resistance flow.

According to Demiriz *et al.*, (1997), Zoppini *et al.*, (1997) and Batukan *et al.*, (2001) most small chorioangiomas are asymptomatic but large or multiple chorioangiomas probably act as arteriovenous shunts and may cause maternal and foetal complications which include polyhydramnios, preterm labour, foetal hemolytic anemia, foetal thrombocytopenia, cardiomegaly, hydrops fetalis, intrauterine growth restriction, placental abruption, preeclampsia, and congenital abnormalities. Of the various reported clinical complications, the correlation of chorioangioma with hydramnios and preterm delivery is significant, latter being a sequelae of the hydramnios (Bashiri *et al.*, 2002).

The pathophysiology of maternal and foetal 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, and (c) functional insufficiency of the placenta secondary to bypassing foetal circulation via shunt mechanism into the tumour vascular bed. Foetal cardiomegaly is thought to occur secondary to high cardiac output from arteriovenous shunting, with resultant left-to-right shunt or chronic hypoxia from unoxygenated blood that bypasses maternal circulation through the tumour vascular bed.

Chorioangioma is usually treated with expectant management, as the majority of tumours are asymptomatic. Small tumours are usually monitored with USG every 6–8 weeks, whereas large, highly vascular tumours require serial USG examinations 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 (Zalel *et al.*, 2002). In utero interventions to improve perinatal outcome in the most severe cases has been reported. Quintero *et al.*, (1996) advocated ultrasound guided or endoscopic laser devascularisation, Nicolini (1999) recommended injection of thrombogenic materials and Lau *et al.*, (2003) recommended micro coil embolization.

The patient presented in this case report had a large, highly vascular tumour associated with polyhydramnios. Ultrasound examination along with Doppler flow study by an experienced radiologist helped diagnose the condition. She was kept under close surveillance and did not develop any other maternal or foetal complications. Decision to terminate pregnancy was taken as soon as an increase in the size of the tumour was noted as chorioangiomas are known to increase suddenly leading to foetal

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complications including intra uterine death (Zalel et al., 2002).The pregnancy had a favourable outcome and both the mother and the baby were discharged in satisfactory condition.

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