Case Report

Giant Placental Chorioangioma: An Unusual Cause of Adverse Foetal and Neonatal Outcome

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Abstract

Chorioangioma is the most common benign tumour of the placenta, accounting for 1% of the pregnant cases. Giant chorioangiomas are known to be correlated with a higher rate of adverse foetal and neonatal complications, including foetal cardiac failure, foetal anemia, hydrops, intrauterine death and neonatal demise. Early detection and serial ultrasound monitoring are essential for timely intervention to ensure a favourable pregnancy outcome. We reported a case of giant chorioangioma complicated with polyhydramnios with poor foetal and neonatal outcome.

Keywords: Chorioangioma; doppler ultrasound; foetal hydrops; neonatal death; polyhydramnios

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Introduction

Chorioangioma is the most common non-trophoblastic tumour of the placenta characterised by vascular overgrowth within chorionic villi with a benign course, encountered in approximately 1% of all pregnancies (1,2). Being first discovered by John Clarke in 1798, the pathogenesis of this neoplasm is multifactorial, with associations with inadequate oxygen tension, cytokines and steal phenomenon (3-5). A higher incidence of chorioangioma is found in women with advanced maternal age, hypertension, diabetes, female babies, first delivery and multiple pregnancies (2). Most of the chorioangiomas are small; hence, they are mainly discovered incidentally and remain asymptomatic. Nevertheless, giant placental chorioangioma (≥4cm) is associated with a wide spectrum of adverse maternal, foetal and neonatal complications, such as polyhydramnios, placental abruption, preterm labour, foetal anemia, high-output heart failure, non-immune foetal hydrops, intrauterine growth restrictions and stillbirths (3,6,7). Ultrasound complemented by colour Doppler is commonly used prenatally to diagnose chorioangioma and characterise its vascularity (3). Complicated chorioangiomas usually require supportive and definitive intervention to prolong the pregnancy and retard the progression of complications. Amnioreduction may help to reduce discomfort in severe polyhydramnios (8). Endolaser coagulation or embolisation with the aim of blocking the arteriovenous shunt demonstrated favourable

pregnancy outcomes in selected cases (3,9). Herein, we presented a case of giant placental chorioangioma with failed amnioreduction complicated with neonatal demise due to foetal hydrops.

Case report

A 44-year-old, Gravida 5 Para 4 with previously uncomplicated pregnancies presented with symptoms of labour at 29 weeks of gestation. Her current pregnancy was unplanned. She was booked and dated at 11 weeks at a private hospital. At booking, she was overweight with a body mass index of 25.6 kg/m^2. Modified glucose tolerance test was unremarkable. At 22 weeks, a well circumscribed, hyperechoic mass with significant vascularity at its base was noted at the placental cord insertion site (Fig. 1). Amniocentesis with chromosomal study was done and no abnormality was detected. She then developed polyhydramnios 4 weeks later. Ultrasound revealed a 10 cm mass at the placenta cord insertion.

At 29 weeks, she developed progressively worsening breathlessness and reduced foetal movements. Upon examination, an increasing polyhydramnios was found. Amnioreduction was attempted but failed. She was then referred to our centre for further management. Upon arrival, she complained of preterm contractions and was found to be in latent phase of labour. Abdominal examination revealed a tense abdomen with moderate contraction felt (1 in 10 minutes). Uterus was larger than date at 40 weeks with clinically excessive liquor and positive fluid thrill. Foetal parts were unable to be felt. Pelvic examination was also done and showed dilated os at 3 cm with soft axial cervix measuring 0.5 cm. The membranes remained intact and presenting part was not felt. In the anticipation of preterm delivery, antenatal corticosteroids and magnesium sulphate for neuroprotection were given immediately. Tocolysis was attempted but she eventually progressed to active labour within 4 hours.

An emergency lower segment caesarean section had to be performed due to abnormal foetal lie. Intraoperatively, excessive liquor of 600cc was noted but no postpartum haemorrhage occurred. A baby girl was delivered. She had dysmorphic appearance and generalised oedema, and weighed 1.52 kg, heavy for the gestational age of 29 weeks, likely due to the accumulation of fluid in hydrops fetalis. She was limp and cyanosed at delivery with a heart rate of 58 bpm. Cardiopulmonary resuscitation was commenced but unfortunately, the baby passed away within 1 hour of life. Placenta was inspected and weighed 1021 g, which was huge considering the birthweight of 1520 g. It looked unhealthy with umbilical cord inserted marginally (Fig. 2). A lobulated mass measuring 100 x 75 x 70 mm was seen attached to the chorionic plate near the umbilical cord insertion site (Fig. 3). The placenta was sent for histopathological examination.

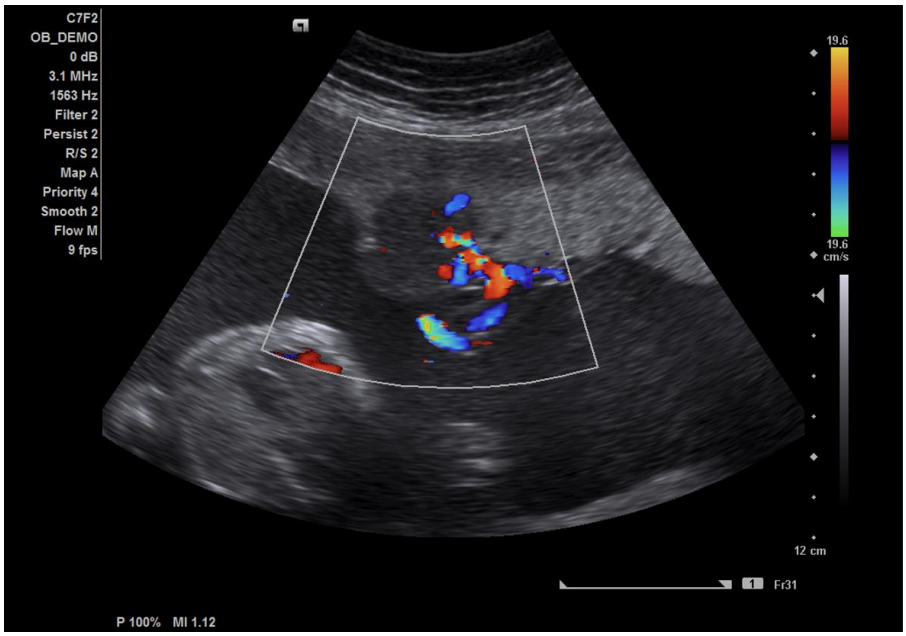

FIGURE 1: Colour Doppler ultrasound revealed a hyperechoic mass at the placental cord insertion site with vascularity at its base

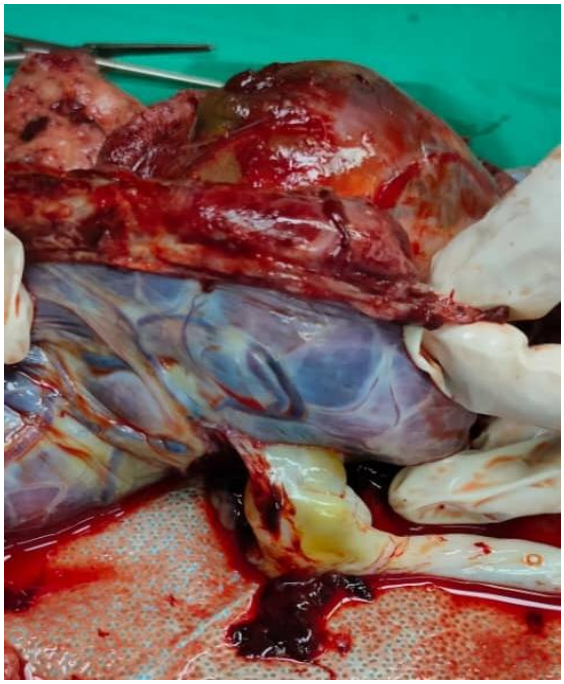

FIGURE 2: The umbilical cord was inserted marginally

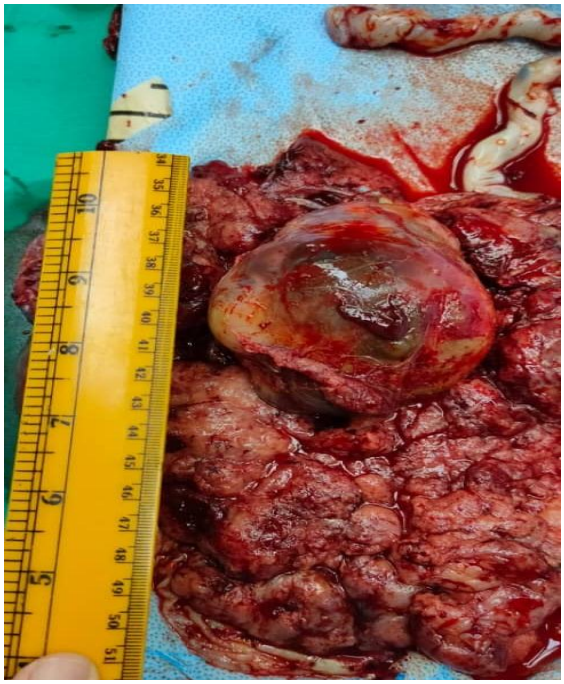

FIGURE 3: A lobulated mass measuring 100 x 75 x 70 mm was seen attached to the chorionic plate near the umbilical cord insertion site

Cut section of the mass showed tan solid cut surfaces with areas of haemorrhage. The umbilical cord insertion site appeared oedematous. Microscopic examination of the diseased placenta revealed a circumscribed lesion arising from a stem villous composed of proliferative capillaries and thin-walled vessels separated by intervening fibrous stroma forming lobular architecture, which was consistent with chorioangioma of the placenta (Fig. 4). Extensive haemorrhage and necrosis were seen in some areas with foci of calcification noted. In a focal area, there was dense neutrophilic infiltration at the chorionic portion of the foetal membrane with mild decidual neutrophilic infiltration, suggestive of acute or sub-acute chorioamniotic infection that may have been predisposed by the amnioreduction procedure contributing to the onset of preterm labour. Areas of villous infarction and perivillous fibrin depositions were seen. However, no atherosis or increase in syncytial knots was noted, indicating that foetal growth restriction not yet developed for this patient. No evidence of funisitis was present. No cellular atypia or evidence of malignancy.

Discussion

Placental chorioangioma is the most frequent benign neoplasm of the placenta, with an estimated incidence of 0.17-1.40% in the microscopically examined placenta (2). The pathogenesis is multifactorial, including lower oxygen tensions and overexpression of angiogenic cytokines, such as vascular endothelial growth factor (3). Another concept proposed is the steal phenomenon in which blood flows preferentially to the chorioangioma and away from the intervillous space, causing reduced blood supply to the developing fetus (5). Several risk factors have been associated with development of placental chorioangioma including advanced maternal age, maternal hypertension and diabetes, primiparity, female fetuses and twin pregnancies (3). Similarly, our patient had an older age during her pregnancy, and conceived a female fetus. Most of the chorioangiomas are small (<4 cm in diameter) and not of great clinical significance throughout the course of pregnancy, compared to large chorioangiomas (≥4 cm in diameter) which are rare, with an incidence rate of 0.11-0.29% of the affected cases (7).

In suspected cases of placental chorioangioma, gray-scale ultrasound is commonly performed and shows a hypo- or hyperechoic well-circumscribed placental mass (7). Prenatal diagnosis of chorioangioma could be confirmed by colour flow Doppler ultrasonography or magnetic resonance imaging as they provide a better characterisation of the vascularised placental

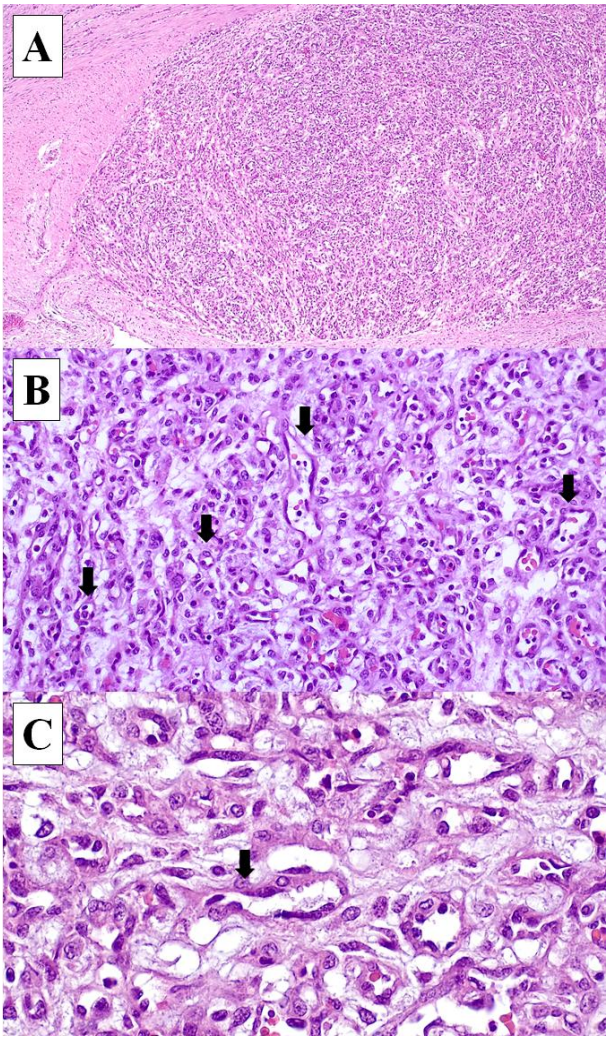

FIGURE 4: The tumour composed of proliferation of small capillaries (black arrow) contained red blood cells and were lined by bland-looking endothelial cells. These cells demonstrated fairly uniform nuclei. Cellular atypia was not identified (H&E, A x4, B x20 and C x40)

mass, distinguishing it from other common differential diagnoses, such as placental teratoma and subamniotic hematoma (3,10). Ideally, foetal ultrasound examinations with colour Doppler should be conducted weekly for early detection of hepatosplenomegaly and increased blood flow velocities (10).

Of note, large chorioangiomas tend to be associated with severe pregnancy-related complications, such as polyhydramnios, placental abruption, preterm delivery, foetal anaemia, foetal growth restriction, congestive cardiac failure, hydrops and foetal death, which was demonstrated in our patient (3,6,7). Polyhydramnios is a frequent manifestation of chorioangioma, thought to be due to the fluid transudation from the umbilical venous obstruction by the mass effect of the tumour or transudation through the diseased vessel walls. Excessive urination and cardiac failure of the fetus further aggravate polyhydramnios, leading to preterm labour (3,11). Additionally, in giant chorioangioma as seen in this case, a large arteriovenous shunt exists between the foetal circulation and the rapidly enlarging tumour, resulting in foetal hydrops and anaemia (12). Fetuses complicated with hydrops had the highest risk for perinatal death (13).

The management of complicated chorioangioma is challenging as it relies on the gestational age and maternal or foetal complications. Delivery is recommended for late onset cases, while prenatal intervention is possible should the complications develop early in pregnancy. Supportive interventions such as amnioreduction for polyhydramnios with respiratory compromise and blood transfusions for anemic foetuses are commonly practiced (8). On the other hand, definitive treatment including alcohol injection, endolaser coagulation, embolisation and radiofrequency ablation aim to occlude the arteriovenous shunting (8,14). In this case, amnioreduction was attempted but unfortunately failed, ultimately resulting in adverse neonatal outcome. However, the ideal treatment modality for a specific complication of chorioangioma remains controversial due to insufficient evidence.

Conclusion

Chorioangioma is the most common benign tumour of the placenta and is associated with unfavourable maternal, foetal and neonatal outcomes particularly in the large chorioangiomas. Routine ultrasound scans with colour Doppler are reliable for timely diagnosis and early detection of complications to suggest when intervention may be required. In the event of development of complications, it is recommended for early referral to healthcare facilities with foetal medicine specialties for close monitoring and further management to minimise the adverse pregnancy outcome.

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Conflict of Interest: The authors declare that there was no conflict of interest.

Ethics Statement: Informed consent was obtained by participant in this study.

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