Chorioangioma is a benign vascular malformation of the placenta and represents the most common primary tumour of the placenta. Presence of this vascular malformation can result in significant maternal and fetal morbidity depending on the size of the lesion. This case study involves a 30-year-old G1 P0 who was referred by her obstetrician to a tertiary referral centre due to her history of antepartum haemorrhage (APH), threatened premature labour and an ultrasound finding of a possible chorioangioma.

The patient was transferred to a tertiary referral centre at 26 + 5 weeks gestation. The patient had been admitted to a private hospital the day prior to transfer after she had experienced a small APH which subsequently became complicated by the onset of premature labour. The patient was rhesus negative and red cell antibodies were not detected. All other routine antenatal blood screening was normal. The 19-week morphology scan was unremarkable. There was no significant medical history, and the patient was a non-smoker and did not consume alcohol. Multivitamins was the only regular medication.

An ultrasound scan was performed at the private hospital prior to the patient’s transfer after her obstetrician noted an increased symphyseal fundal height and to investigate the APH. The important findings from this ultrasound scan included the following:
1. Estimated fetal weight 1333 g (> 95th percentile)
2. No obvious fetal anomalies
3. Posterior placenta 33 mm from internal cervical os
4. Polyhydramnios (AFI 28 cm with deepest pool of 9 cm)
5. Visible fetal bladder and stomach
6. No features of fetal hydrops
7. Normal umbilical artery Doppler study
8. Elevated middle cerebral artery (MCA) Peak systolic velocity at 56 cm/sec
9. Well-circumscribed, homogeneous and highly vascular mass measuring 4.4 x 4.2 x 5.3 cm situated adjacent to the cord insertion possibly representing a chorioangioma
10. Cervical length 0.5 cm.

On admission to the tertiary referral unit, the patient was scanned by a member of the maternal fetal medicine team. The scan confirmed the findings of a placental mass situated adjacent to the cord insertion measuring 6.4 x 5.9 x 4.4 cm. The mass was well-circumscribed, homogenous and demonstrated only mild vascularity on colour Doppler assessment. The scan also confirmed normal fetal anatomy and polyhydramnios. The fetus was not hydropic. Umbilical artery and MCA Doppler studies were within normal limits. To exclude other common causes of polyhydramnios, testing was performed for viral studies, isoimmunisation and diabetes. Subsequently, TORCH, red cell antibody and diabetes screening were all found to be normal. A plan was made to serially scan the patient and a member of the neonatology team had been asked to consult in the event of preterm delivery. Amnioreduction was offered as a management option which the patient declined. A course of Betamethasone had been prescribed.

Within three days of admission, the patient went into premature labour, which further became complicated by a placental abruption. An emergency lower segment caesarean section was performed. A live male infant was delivered weighing 1278 g with Apgars of 3 and 8 at 1 and 5 minutes respectively. The baby was admitted to the Level 3 NICU and had an uncomplicated neonatal period. Placental histopathology was inconclusive for a chorioangioma but diagnostic of a retroplacental clot.

As stated previously, chorioangioma is a benign vascular malformation of the placenta composed of tissue normally present in the placenta. The majority of cases are typical capillary haemangiomas arising from just beneath the...
chorionic plate. It is the most common primary tumour of the placenta, with an incidence of about 1%, but these are usually microscopic in size. Small chorioangiomas usually do not cause any physiologic complications and may be managed expectantly. Sonographically visible tumours that are large enough to produce clinical symptoms are uncommon, occurring in approximately one in 3500–9000 births. It is the larger chorioangiomas, especially those greater than 4–5 cm, that are reported to have anywhere between a 30–50% rate of maternal and fetal complications1–5.

On sonography, most chorioangiomas are located underneath the chorionic plate near the insertion of the umbilical cord and often protruding into the amniotic cavity. Chorioangiomas are typically well-circumscribed, solid lesions that have different echogenicity to the rest of the placental tissue. By colour Doppler imaging, a chorioangioma can be differentiated from an avascular tumour (incomplete hydatidiform mole, placental teratoma, cytotrophoblastic cysts, degenerated myoma) or a haematoma, on the basis of its flow pattern, which in some cases, is pulsatile1,5,6,7.

The clinically significant chorioangiomas are associated with a number of well recognised pregnancy complications including polyhydramnios, nonimmune fetal hydrops, fetal heart failure/cardiomegaly, fetal anaemia and thrombocytopenia, fetal growth restriction, preterm delivery, perinatal death, placental abruption and maternal preeclampsia. Although the underlying pathophysiology for these complications has not been fully elucidated, a prominent role for arteriovenous (AV) shunting and sequestration of red blood cells and platelets by the chorioangioma has been postulated5,8. It is thought that chorioangiomas may act as peripheral AV shunts leading to an increase in fetal cardiac output, cardiac hypertrophy, high output congestive cardiac failure and fetal hydrops1,5,6,7. Fetal anaemia is thought to be a consequence of either fetomaternal haemorrhage, microangiopathic haemolysis occurring in the winning chorioangioma blood vessels and/or blood sequestration in the intravascular space of the tumour1,6,7,8,9. Lastly, three hypotheses exist for the cause of polyhydramnios: (a) accumulation of fluid secondary to compression of the umbilical vein by the mass; (b) fluid imbalance caused by an increased production of fetal urine or by congestive heart failure; (c) excess amniotic fluid resulting from a transudate through the wall of the abnormal tumour vessels and subsequently through the fetal plate of the placenta2,6,12–14.

To prevent fetal loss from the haemodynamic complications of a large chorioangioma, several in-utero interventions have been proposed, and, if gestational age precludes early delivery, intratrauterine therapy should be considered. For direct tumour management, a variety of methods have been reported including fetoscopic ligation and bipolar electrosurgery, injection of absolute alcohol, microcoil embolisation and interstitial laser treatment2,4,6,8,9,12,15. However, there is limited experience and no consensus as to the true efficacy of any of these methods. Therefore, amniodrainage for alleviating the polyhydramnios and intratrauterine transfusions in the presence of fetal anaemia are the two most common conservative therapeutic procedures utilised in the management of such complications.

In conclusion, chorioangioma is a benign vascular malformation of the placenta and, depending on the size of the lesion, can be associated with significant maternal and fetal morbidity. Any placental mass detected on ultrasound, which is suspicious for choioangioma requires close ultrasound surveillance for the early detection of fetal complications such as the development of non-immune hydrops.

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