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When benign turns fatal: a sonographic case of giant placental chorioangioma causing non-immune fetal hydrops and intrauterine death

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Abstract

Placental chorioangioma is the most common benign vascular tumour of the placenta, occurring in approximately 1% of pregnancies, although clinically significant giant lesions larger than 4–5 cm are rare. Large chorioangiomas can behave as arteriovenous shunts, producing high-output fetal cardiac failure, anaemia, polyhydramnios and, ultimately, non-immune fetal hydrops. We report a 26-year-old woman who presented at 27 weeks and 4 days of gestation with decreased fetal movements. Grayscale ultrasonography revealed a well-circumscribed, heterogeneously hypoechoic mass along the anterior surface of the placenta measuring approximately 9.2 × 11.9 cm, and colour Doppler demonstrated prominent internal vascular channels, consistent with a giant placental chorioangioma. The fetus showed features of non-immune hydrops, including scalp oedema, bilateral pleural effusion, ascites and cardiomegaly, with no maternal risk factors or serological evidence of immune hydrops. Despite supportive monitoring, intrauterine fetal demise occurred shortly after presentation, attributed to high-output cardiac failure from the vascular tumour. This case highlights that early antenatal detection with ultrasound and colour Doppler is crucial, as complications correlate strongly with tumour size and vascularity, and that timely diagnosis is essential for counselling and management.

Keywords: Placental chorioangioma, non-immune fetal hydrops, high-output cardiac failure, antenatal ultrasonography, colour Doppler, intrauterine fetal death, placental vascular tumour

Introduction

Chorioangioma is the most common benign, non-trophoblastic tumour of the placenta, arising from primitive chorionic mesenchyme. Small lesions occur in roughly 1% of pregnancies, are usually microscopic or incidental, and carry no clinical significance [1, 2]. Giant chorioangiomas—generally defined as those larger than 4–5 cm—are far less frequent, with an estimated incidence of approximately 1 in 9,000 to 1 in 50,000 pregnancies, and it is these large tumours that account for the clinically important complications [3, 4].

The adverse effects of a giant chorioangioma are largely haemodynamic. The richly vascular tumour can act as a peripheral arteriovenous shunt, diverting blood through a low-resistance circuit and progressively increasing fetal cardiac output. The resulting chain of events may include fetal anaemia, thrombocytopenia, cardiomegaly, high-output congestive cardiac failure, polyhydramnios and non-immune hydrops, with intrauterine fetal death as the most severe outcome [3, 5, 6]. Maternal complications such as mirror syndrome and preterm labour may also occur [4].

Antenatal ultrasonography, supplemented by colour Doppler, is the principal modality for the diagnosis and surveillance of these tumours. On grayscale imaging the lesion typically appears as a well-circumscribed, rounded, predominantly hypoechoic mass on the fetal surface of the placenta, often near the cord insertion, while colour Doppler characteristically demonstrates abundant intratumoral vascularity or a prominent feeding vessel—the feature that distinguishes a chorioangioma from a placental haematoma, venous lake or other placental masses [1, 2, 7]. Because the prognosis correlates strongly with tumour size and vascularity, early recognition allows close surveillance, timely counselling and, in selected cases, antenatal intervention [3, 5]. We report a case of a giant placental chorioangioma presenting with non-immune fetal hydrops and intrauterine fetal demise, and discuss the sonographic features and clinical implications of this rare but potentially fatal entity.

Case Presentation

A 26-year-old woman, gravida [G] para [P], presented at 27 weeks and 4 days of gestation with a complaint of decreased fetal movements. She had no significant past medical history, no maternal risk factors, and no serological evidence of an immune cause of hydrops. A focused antenatal ultrasound with grayscale and colour Doppler was performed at presentation, including assessment of the placenta, characterisation of the mass, fetal biometry, and evaluation for hydrops.

Grayscale ultrasonography revealed a well-circumscribed, heterogeneously hypoechoic mass along the anterior surface of the placenta, measuring approximately 9.2 × 11.9 cm. On colour Doppler interrogation, the lesion demonstrated prominent internal vascular channels, a finding consistent with a giant placental chorioangioma (Figure 1).

Evaluation of the fetus demonstrated features of non-immune hydrops, including scalp oedema and bilateral pleural effusion, together with ascites and cardiomegaly (Figure 2). The constellation of a vascular placental mass

with fetal hydrops, in the absence of maternal risk factors or serological evidence of an immune aetiology, indicated high-output cardiac failure secondary to the vascular tumour as the underlying mechanism.

Despite supportive monitoring, intrauterine fetal demise occurred shortly after presentation. Examination of the delivered placenta confirmed a well-circumscribed vascular tumour corresponding to the antenatally identified mass (Figure 3), in keeping with a giant placental chorioangioma as the cause of the non-immune hydrops and fetal death.

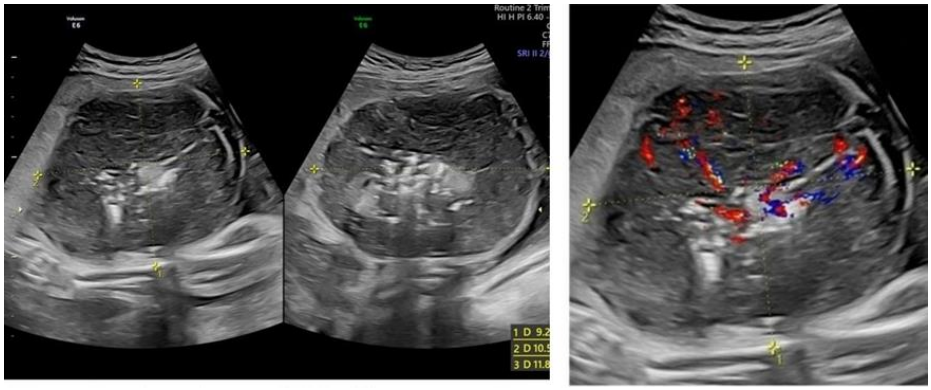

Fig 1: Antenatal ultrasonography. (a) Grayscale images demonstrate a well-circumscribed, heterogeneously hypoechoic mass along the anterior surface of the placenta measuring approximately 9.2 × 11.9 cm. (b) Colour Doppler image shows abundant internal vascular channels throughout the lesion, consistent with a giant placental chorioangioma.

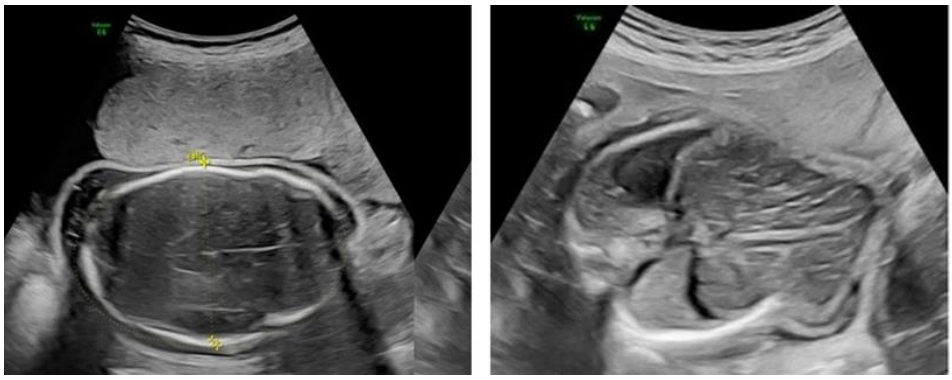

Fig 2: Ultrasonography of the fetus shows features of non-immune hydrops. (a) Scalp oedema is seen as a double-contour halo around the fetal cranium. (b) Axial image of the fetal thorax demonstrates bilateral pleural effusion.

Fig 3: Gross specimen of the delivered placenta shows a well-circumscribed, dark, vascular tumour corresponding to the antenatally identified mass, in keeping with a giant placental chorioangioma.

Discussion

Placental chorioangioma occupies an instructive place in obstetric imaging: although it is the most common benign tumour of the placenta, the great majority of lesions are small and clinically silent, and it is only the giant variant that carries a substantial risk to the fetus [1, 2]. The present case illustrates the dramatic end of this spectrum, in which a large, highly vascular tumour produced non-immune hydrops and intrauterine death within a short interval of presentation.

The pathophysiological link between a giant chorioangioma and fetal compromise is haemodynamic. The tumour's extensive vascular bed behaves as an arteriovenous shunt, lowering peripheral resistance and increasing venous return, which drives a progressive rise in fetal cardiac output. Sustained hyperdynamic circulation leads to cardiac hypertrophy and dilatation, high-output congestive cardiac failure and, eventually, hydrops [3, 5, 6]. Concurrent mechanical fragmentation of red cells and platelets within the abnormal vascular channels may produce microangiopathic anaemia and thrombocytopenia, further compromising the fetus, while increased transudation contributes to polyhydramnios [4, 6]. In the present case the combination of scalp oedema, pleural effusion, ascites and cardiomegaly, with no immune basis, is characteristic of this high-output mechanism.

Ultrasonography is the cornerstone of diagnosis. The typical grayscale appearance is that of a well-circumscribed, rounded, predominantly hypoechoic mass arising from the fetal surface of the placenta, frequently close to the cord insertion and sometimes protruding into the amniotic cavity [2, 7]. The heterogeneous echotexture reflects internal areas of degeneration, haemorrhage or infarction. The single most useful diagnostic feature is the demonstration of abundant intratumoral vascularity, or a prominent pulsatile feeding vessel, on colour Doppler imaging; this finding reliably distinguishes a chorioangioma from avascular placental lesions such as a haematoma, venous lake or partial mole, which may otherwise appear similar on grayscale imaging [1, 7]. In the present case the prominent internal vascular channels on colour Doppler were central to the diagnosis. Magnetic resonance imaging may serve as a complementary problem-solving tool in equivocal cases, but is rarely required when the sonographic and Doppler features are characteristic [4].

Once a giant chorioangioma is identified, the demonstrated correlation between tumour size, vascularity and adverse outcome mandates close surveillance for the early features of fetal decompensation—rising middle cerebral artery peak systolic velocity as a marker of anaemia, increasing cardiac size, developing effusions and polyhydramnios [3, 5]. A range of antenatal interventions has been described for tumours that cause fetal compromise before viability, including amnioreduction, intrauterine transfusion, and fetoscopic or interventional devascularisation of the feeding vessel by laser, embolisation or chemical ablation, with variable success [5, 6]. Where complications develop late in pregnancy, timely delivery is generally preferred [3]. In the present case the rapid deterioration and early gestational age limited the available options, and fetal demise occurred before intervention could be undertaken, underscoring how swiftly a "benign" tumour can prove fatal.

The differential diagnosis of a vascular placental mass includes placental haematoma or subchorionic haemorrhage, venous lake, partial hydatidiform mole, placental teratoma and, rarely, placental metastasis. The well-defined margins, solid vascular architecture and abundant Doppler flow of a chorioangioma, together with the absence of the cystic "snowstorm" pattern of molar disease and the avascularity of a haematoma, generally allow confident differentiation [1, 2, 7]. Recognition is important because, unlike most of these mimics, a giant chorioangioma carries a high perinatal mortality, reported in some series at around 30–40% [5, 6].

Conclusion

Giant placental chorioangioma is a rare but potentially fatal cause of non-immune fetal hydrops. This case demonstrates how a benign vascular tumour can, through high-output cardiac failure, lead to rapid fetal deterioration and intrauterine death. Antenatal ultrasonography with colour Doppler is the key to early diagnosis, characteristically revealing a well-circumscribed, heterogeneously hypoechoic placental mass with abundant internal vascularity, and to the detection of the hydropic complications that herald fetal compromise. Because outcome correlates closely with tumour size and vascularity, prompt sonographic recognition is essential to allow close surveillance, appropriate counselling and, where feasible, timely intervention. Radiologists and obstetricians should remain alert to placental chorioangioma as a cause of otherwise unexplained non-immune fetal hydrops.

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