

Giant Placental Chorioangioma Causing Mirror Syndrome, Maternal Sepsis and Extreme Preterm Birth: A Case Report

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Introduction

Placental chorioangioma is a benign vascular tumour (1% of placentas). Giant lesions (>4–5 cm) predispose to high-output cardiac failure, anaemia, polyhydramnios, hydrops and mirror syndrome.

Case summary:

27-year-old primigravida at 27+1 weeks with severe polyhydramnios (AFI 35 cm) and 6 cm chorioangioma. Developed PPRM complicated by acute kidney injury, elevated liver enzymes, PPRM and maternal sepsis. Emergency caesarean at 27+2 weeks. Neonate (1580 g) had severe anaemia requiring transfusion and NICU admission. Histopathology confirmed 6.5 cm chorioangioma. Mother required ICU care and antibiotics with full recovery.

Discussion:

Placental chorioangioma is a benign vascular placental tumour that is usually small and clinically insignificant. However, large or giant chorioangiomas ($\geq 4\text{--}5$ cm) are associated with significant fetal and maternal morbidity, including polyhydramnios, fetal anaemia, fetal growth restriction, cardiomegaly, high-output cardiac failure, hydrops fetalis, preterm birth and perinatal death. The risk of adverse outcomes increases with tumour size, particularly in the presence of fetal hydrops or hyperdynamic circulation. The tumour may act as a low-resistance vascular shunt, increasing fetal cardiac output and potentially leading to cardiac decompensation. Sequestration or haemorrhage of fetal red blood cells within the highly vascular tumour may also contribute to fetal anaemia. Polyhydramnios may occur as a consequence of increased fetal cardiac output and altered fetal fluid dynamics. Serial ultrasound and colour Doppler surveillance are therefore important to assess tumour size and vascularity, fetal growth, amniotic fluid volume, cardiac function and MCA-PSV as a marker of fetal anaemia.

Management should be individualised according to tumour size, vascularity, gestational age and evidence of fetal or maternal compromise. Small, asymptomatic lesions can generally be managed conservatively with close surveillance, while large or complicated lesions require multidisciplinary assessment and careful consideration of fetal intervention or delivery.

In our case, the 6.5 cm highly vascular chorioangioma was associated with severe polyhydramnios and subsequently complicated by PPRM and maternal sepsis, necessitating emergency delivery at 27+2 weeks. The neonate had severe anaemia requiring immediate transfusion and intensive neonatal care, while the mother developed significant systemic complications requiring intensive care and intravenous antibiotics. Histopathology confirmed a 6.5 cm chorioangioma.

This case highlights that giant chorioangiomas can cause severe and rapidly progressive maternal and fetal complications, even in the absence of classical hydrops or mirror syndrome. Early recognition, close ultrasound and Doppler surveillance, multidisciplinary management and timely escalation are essential when maternal or fetal deterioration occurs.

Conclusion

Giant placental chorioangiomas are rare but potentially life-threatening placental tumours. Tumour size and vascularity are important predictors of adverse outcome. Close antenatal surveillance can facilitate early detection of fetal anaemia, hyperdynamic circulation and polyhydramnios. Individualised multidisciplinary management and timely intervention or delivery are essential to optimise maternal and neonatal outcomes.



Figure 1. Fetal medicine ultrasound demonstrating severe polyhydramnios and a placental mass.



Figure 2. Placental ultrasound with color Doppler showing the 6 cm highly vascular chorioangioma with significant blood flow.