

CASE REPORT:

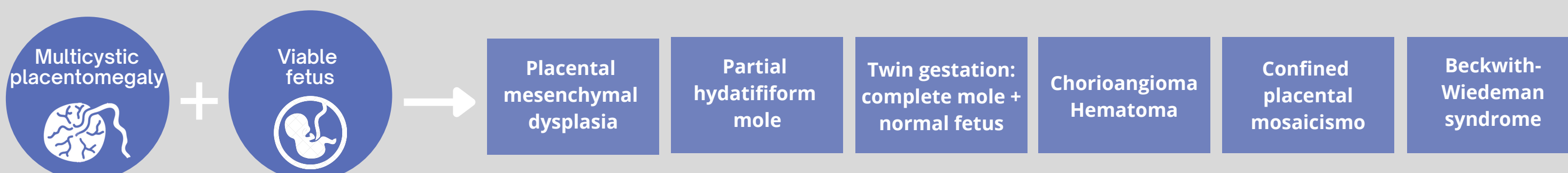
PLACENTAL MESENCHYMAL DYSPLASIA

AN UNDERDIAGNOSED PATHOLOGY OF THE PLACENTA

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INTRODUCTION

We present the case of a primigravida with a past history of curettage for complete hydatidiform mole that presented in her first trimester ultrasound scan at 12+1 gestational weeks an **hypertrophic placenta with multiple anecogenic areas that showed no color Doppler signal giving a “Swiss cheese” appearance**. She had a low risk result for both the chromosomopathy and early preeclampsia screenings. Free β hCG serum level was 118.9 UI/L (1.48 MoM) and serum PAPP-A was 713.6 mIU/L (0.97 MoM). The scan at 15+6 weeks showed a persistent atypical placenta with a 44 mm thickness along with a viable fetus. The differential diagnoses that were first considered were incomplete hydatidiform mole, a triploidy, or a normal variant. A recommendation of strict control to rule out signs of pregnancy induced-hypertension, intrauterine growth restriction or hydrops was made.

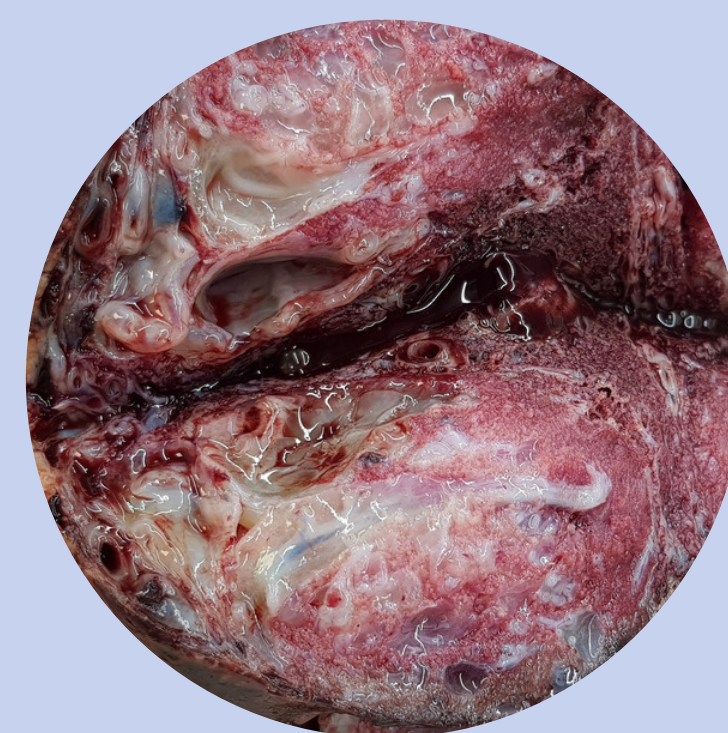
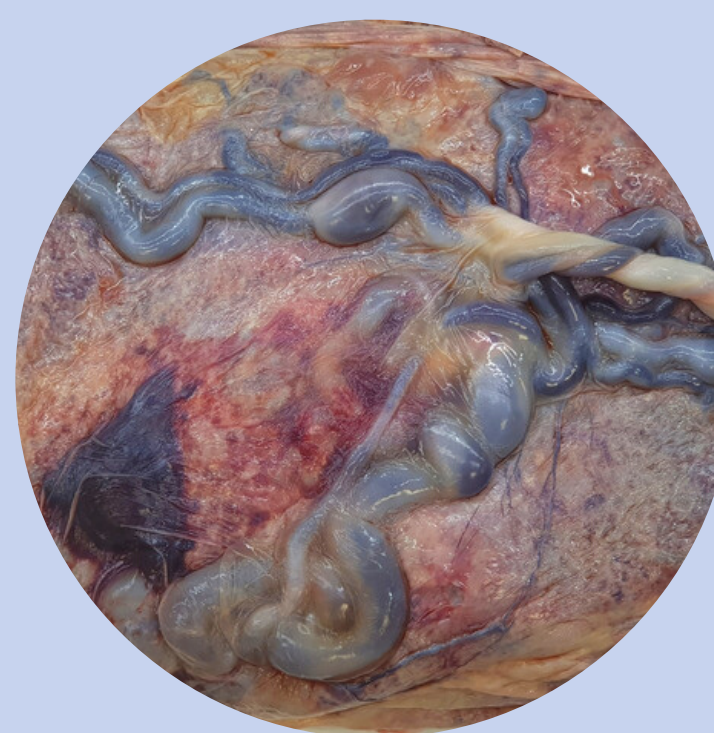
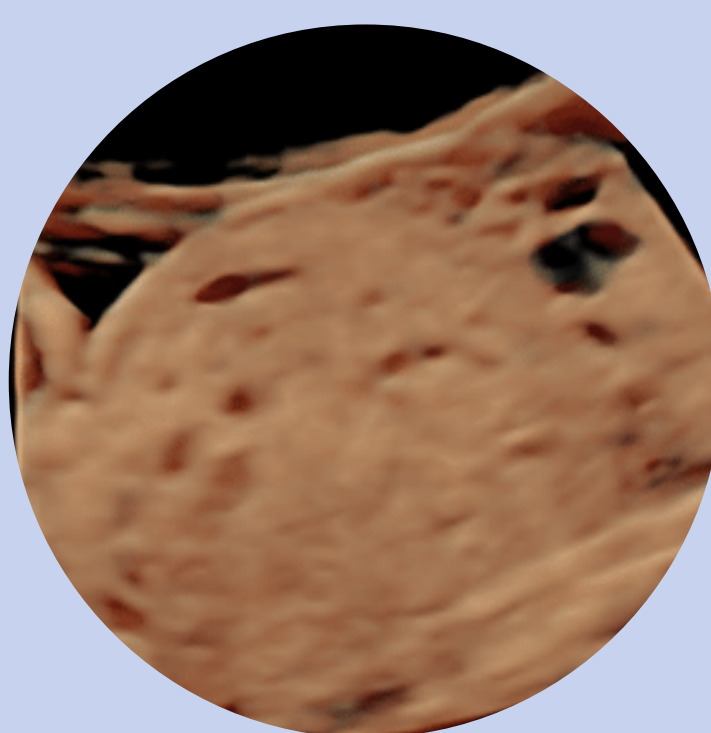
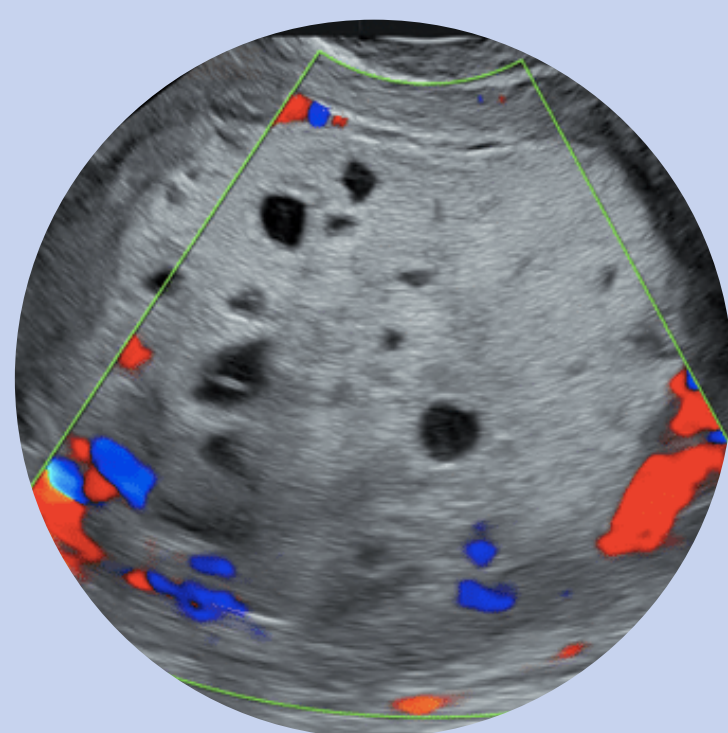


MATERIALS & METHODS

All ultrasound scans were normal until week 22+6 when the control scan showed signs of moderate anemia and a mildly thickened myocardium. An amniocentesis was offered. **QF-PCR showed a mosaicism of 2 cell lines, a normal 46 XX biparental line and another 46 XX possibly caused by a paternal uniparental disomy**, present in all the markers analyzed and that probably affected all chromosomes. Taking into account both the ultrasound and genetic findings a placental mesenchymal dysplasia was then suspected. Further ultrasound controls showed normal fetal growth and normal cerebroplacental Dopplers as well as no changes in the placenta, which had a 83 mm thickness at 28+6 weeks.

RESULTS

At week 30+4 the patient presented abdominal pain and hydrorrhea. The ultrasound showed no fetal heart rate and anhydramnios. The women gave birth to a female stillborn of 1675 g (p72). The placenta weighed 1256.6 g and presented markedly dilated tortuous chorionic vessels and subchorionic hematomas. **The histological examination confirmed the diagnosis of placental mesenchymal dysplasia.** Serial cuts showed a thickened, fleshy parenchyma, with the presence of numerous millimetric cystic cavities, the largest measuring 28x10mm, and highly branched and thickened blood vessels with signs of focal thrombosis.



CONCLUSIONS

Placental mesenchymal dysplasia (PMD) is a rare vascular anomaly of the placenta that is misdiagnosed as a partial hydatidiform mole. Both entities are characterized by placental enlargement and cystic structures that seem to be intermixed with normal-appearing placenta. Although these two conditions appear similar on imaging, they have vastly different maternal and fetal implications. Unlike molar pregnancies, which rarely result in viable fetuses, pregnancies with PMD usually result in normal births, but some cases present with complications such as intrauterine growth restriction, fetal anemia or intrauterine death. Thus it is essential to differentiate them in the antenatal period to avoid unnecessary abortions and to assess the potential risk of persistent gestational trophoblast disease in this women. In patients with PMD early hospitalization is recommended to monitor fetal well-being closely in order to reduce the fetal mortality rate.