Case Report

Placental mesenchymal dysplasia and intrauterine fetal growth restriction with doppler velocimetry alterations: a case report

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ABSTRACT

Placental mesenchymal dysplasia (PMD) is a rare placental abnormality. We report a case of PMD associated with intrauterine growth restriction (IUGR), which was diagnosed by an ultrasound scan during the second trimester of pregnancy. A 36-year-old primiparous woman with signs of placental chorioangioma was referred to our hospital at the 23th gestational week. An ultrasonography revealed a small-for-gestational-age fetus with a large multicystic placenta. A serial Doppler sonographic assessment of umbilical and uterine artery blood flow showed a compromised fetus. A female, small-for-gestational-age baby was delivered by c-section at 28 weeks, and PMD was histopathologically confirmed.

Keywords: *Fetal growth; doppler; velocimetry*

Placental mesenchymal dysplasia (PMD) is a rare type of placental lesion, often associated with severe intrauterine growth restriction (IUGR) and fetal death. Our objective was to report a case of PMD involving a serial Doppler velocimetry study followed by a live birth. A 36-year-old primiparous woman with chronic hypertension and signs of placental chorioangioma confirmed by imaging was referred to our hospital at the 23th gestational week. An ultrasonography revealed a large multicystic placenta and fetal growth at the 10th percentile. Color Doppler imaging showed an absence of end-diastolic frequency in the umbilical artery (UA). The differential diagnosis included either molar pregnancy or PMD (Figure 1). Fetal karyotype was

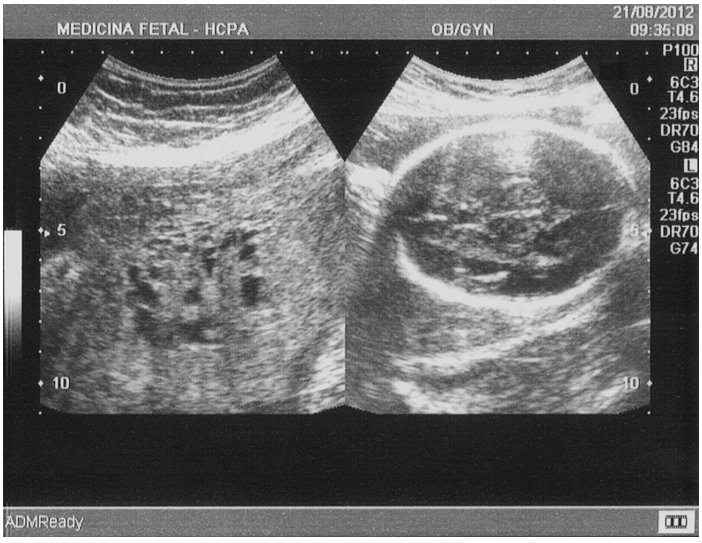

Figure 1: Ultrasound at 26 weeks of gestation. The image shows placental mesechymal dysplasia, with hypo echoic areas that can resemble a molar pregnancy.

performed in amniotic fluid, and the result was 46,XX. Fetal echocardiography revealed right ventricular hypertrophy. A fetal ultrasound examination at 27 weeks of pregnancy revealed oligohydramnios, multicystic placenta, and fetal growth below the 5th percentile. Doppler imaging showed an absennce of end-diastolic frequency in the UA and a middle cerebral artery (MCA) resistance index (RI) of 0.7. At 27 weeks and 6 days of pregnancy, imaging showed a fetal growth below the 5th percentile, an MCA IR of 0.95, and an UA IR of 1.53 with absent end-diastolic frequency, uterine arteries with early diastolic notching, and a reduction in the A component of the flow velocity waveform in the ductus venosus (Figure 2), indicating time of delivery. The patient was administered two doses of betamethasone and magnesium sulfate for neuroprotection. A 655 g female baby was delivered via c-section at 28 weeks of pregnancy with Apgar scores of 6 and 8, at 1 and 5 minutes, respectively, and was immediately transferred to the neonatal intensive care unit. The graph with the estimated fetal body weight curve is shown in Figure 3 and revealed a small-for-gestational-age newborn. A gross examination showed that the placenta was monochorionic and monoamniotic, weighed 255 g, and had velamentous umbilical cord insertion. Vesicles were found in 80% of the parenchymas, which were hemorrhagic and had lacerations (Figure 4). Microscopic examination showed large hydropic stem villi and vessels with thick walls lacking trophoblast proliferation. PMD was histopathologically confirmed (Figure 5).

PMD was first described in 1991 by Moscoso. It is a rare benign condition of placentomegaly and abnormal chorionic villi, often clinically mistaken for a partial hydatidiform mole, which is clinicopathologically distinct and has a high incidence of IUGR and fetal death[1,2]. The exact incidence of PMD is unknown, but it is generally assumed to be about 0.02% and is associated with Beckwith-Wiedemann syndrome (macrosomia, exomphalos, macroglossia, omphalocele, craniofacial features, and ear anomalies) in about 20% of cases. A high rate of IUGR is observed in about 50% of cases[3-6]. Sonographs show enlarged placentas with multicystic anechoic regions and widely distributed, large, edematous villi, which can be seen under gross examination[7-9]. Microscopically, these vesicles correspond to dilated stem vessels, with thickened vasculature surrounded by normal villi[1]. These placental histopathological characteristics seem to be responsible for changes in the Doppler-velocimetric waveforms and may result in IUGR and fetal death. This is explained by a potentially chronic hypoxia secondary to obstructive fetal vascular thrombosis and decreased maternal-fetal gas exchange due to an insufficient amount of normal chorionic villi and shunting of blood from the exchange surface in the dysplastic villi[5]. Doppler evaluation may play a role in these cases[10].

PMD should be included in the differential diagnosis for ultrasound findings that reveal a normal-looking fetus with cystic lesions of the placenta because management and outcomes differ. PMD is probably under-diagnosed and under-reported due to sonologists, pathologists and other healthcare providers being unfamiliar with this obstetric condition. Additionally, patients should be advised of the increased risk of preeclampsia, fetal demise, and maternal morbidity. Prenatal care involving Doppler velocimetry studies seems to be essential for increased fetal survival in cases of placental abnormalities. Further research is necessary on the true incidence of complications and on the types of complications associated with PMD.

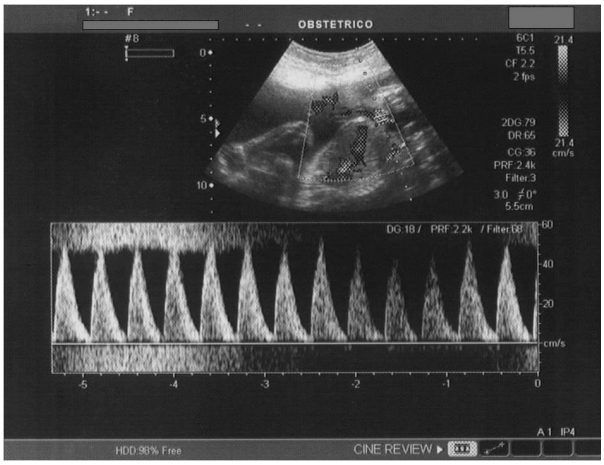

Figure 2: Doppler ultrasound findings.

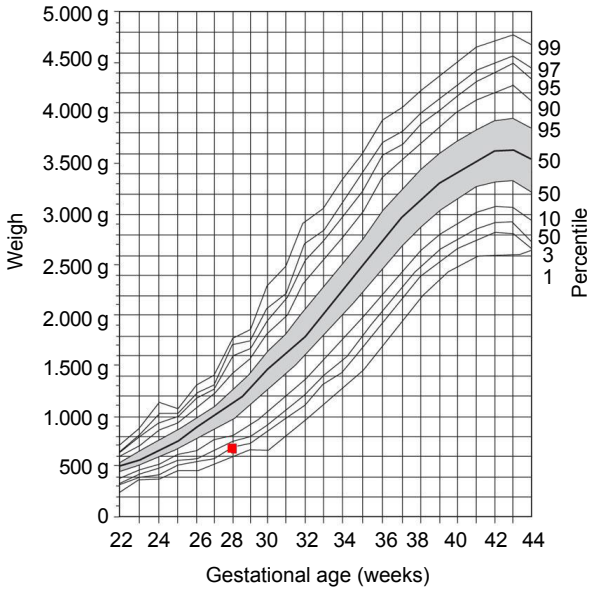

Figure 3: Graph of estimated fetal body weight (case in red).

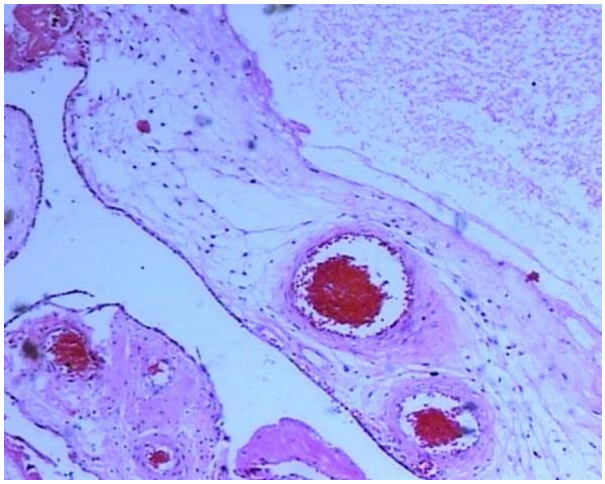

Figure 4: Macroscopic aspect of the placenta.

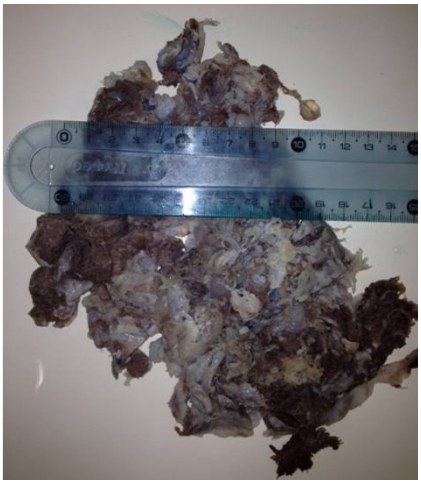

Figure 5: Central vessels with fibromuscular sclerosis increased in villous stromal fibrosis with degeneration (HE, 50 X).

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