

CLINICAL CASE

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Prenatal management of placental mesenchymal dysplasia

Manejo prenatal de la displasia mesenquimal placentaria

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ABSTRACT

Introduction: Placental mesenchymal dysplasia (PMD) is a vascular condition associated with preeclampsia and intrauterine growth restriction (IUGR) due to placental thrombosis. **Case Report:** A pregnant woman diagnosed with PMD and a healthy fetus. After evaluating the thrombotic pathogenesis, it was decided to implement high-risk obstetric management, initiating prophylaxis with 150 mg of acetylsalicylic acid daily starting at 13 weeks. At 35 weeks, a cesarean section was performed due to premature rupture of membranes and oligohydramnios. **Results:** The fetal growth curve remained normal (40th–50th percentile). The mother did not develop preeclampsia during pregnancy or the postpartum period. The infant is developing normally at 14 months.

Keywords: placental mesenchymal dysplasia, aspirin, case report.

RESUMEN

Introducción: La displasia mesenquimal placentaria (DMP) es una patología vascular asociada a preeclampsia y restricción del crecimiento intrauterino (RCIU) por trombosis placentaria. **Caso clínico:** Gestante con diagnóstico de DMP y feto sano. Tras evaluar la patogenia trombótica, se decidió extrapolar el manejo de alto riesgo obstétrico, iniciando profilaxis con 150 mg diarios de ácido acetilsalicílico desde la semana 13. A la semana 35 se realizó cesárea por rotura prematura de membranas y oligohidramnios. **Resultados:** La curva de crecimiento fetal se mantuvo normal (percentil 40-50). La madre no desarrolló preeclampsia en el embarazo ni puerperio. El lactante cursa con desarrollo normal a los 14 meses.

Palabras clave: displasia mesenquimal placentaria, aspirina, reporte de caso.

INTRODUCTION

Placental mesenchymal dysplasia (PMD) is a placental vascular anomaly with an estimated incidence of between 0.02% and 0.03%⁽¹⁾. It was first described in 1982 and associated with Beckwith-Wiedemann syndrome⁽²⁾; in 1991, Moscoso et al. proposed the term placental mesenchymal dysplasia. Macroscopically, PMD is characterized by placentomegaly with multiple vesicles appearing as a cluster of grapes on ultrasound⁽³⁾.

The etiology of this dysplasia is multifactorial. One theory proposes that hypoxia and hypoperfusion stimulate fibroblasts to produce connective tissue fibers, leading to increased production of vascular endothelial growth factor by the villi, which induces angiogenesis⁽⁴⁾. Another theory suggests that its origin is biparental androgenic mosaicism confined to the placenta. Clinically, MPD is associated with intrauterine growth restriction (IUGR)⁽¹⁾, hypertensive disorders of pregnancy, placenta previa, preeclampsia, gestational hypertension, and HELLP syndrome, fetal death⁽¹⁾, and, in 30% of cases, Beckwith-Wiedemann syndrome.

A presumptive diagnosis of PMD is made by ultrasound; however, a differential diagnosis with partial hydatidiform mole, twin pregnancy with complete mole, and mosaicism confined to the placenta must be considered^(5, 6). A definitive diagnosis is provided by histopathological examination^(6, 7).

The purpose of this case report is to describe the prenatal management of placental mesenchymal dysplasia.

CASE PRESENTATION

A 41-year-old woman, experiencing her second vaginal delivery, with no relevant medical or surgical history. She underwent a routine ultrasound at 13 weeks of gestation; the placenta was noted to be enlarged, with a cystic appearance, hypoechoic areas, and dilated chorionic vessels. No abnormal findings were observed on the first-trimester fetal ultrasound (Figure 1), and uterine artery Doppler imaging showed no abnormalities; a quantitative test for beta-human chorionic gonadotropin (β -hCG) was ordered. The result was 9,139 IU, a normal value for the gestational age, which ruled out the possibility of gestational trophoblastic disease. Starting at this week (13 weeks), treatment with acetylsalicylic acid (aspirin) was initiated at a daily dose of 150 mg taken orally each night.

The decision to start aspirin at 13 weeks was based on an early suspected diagnosis of DMP; no software was used to calculate the

risk of PE. During the ultrasound follow-up performed at 23 weeks, a male fetus was identified with no pathological fetal findings. Placentomegaly (8 cm thick) and a placenta with multiple subamniotic cysts were observed (Figures 2). Doppler ultrasound of the uterine arteries continued to show no abnormalities.

Starting at week 28, a maternal-fetal Doppler ultrasound was performed monthly. The results remained stable and showed no abnormalities. The fetal growth curve remained within normal parameters throughout the pregnancy. The patient continued taking aspirin until 35 weeks. She experienced premature rupture of membranes (PROM) with severe oligohydramnios; a cesarean section was performed 48 hours after fetal maturity at the ES-SALUD hospital. The histological diagnosis was confirmed; the fetus was born weighing 2,500 grams (50th percentile), with an Apgar score of 8–9. His growth and development to date—now at one year and two months of age—are normal; he currently weighs 10 kilograms. As for the mother, she did not develop preeclampsia during pregnancy or the postpartum period; she currently has normal blood pressure and no cardiovascular sequelae.



FIGURE 1. FIRST-TRIMESTER OBSTETRIC ULTRASOUND (13 WEEKS' GESTATION). IN THE UPPER REGION, A MORPHOLOGICALLY NORMAL FETUS IS SEEN IN A SAGITTAL VIEW. IN THE LOWER REGION, THERE IS MARKED PLACENTAL THICKENING (PLACENTOMEGALY) ASSOCIATED WITH MULTIPLE ANECHOIC CYSTIC LESIONS ARRANGED IN "CLUSTERS," FINDINGS CHARACTERISTIC OF AND SUGGESTIVE OF PLACENTAL MESENCHYMAL DYSPLASIA (PMD).

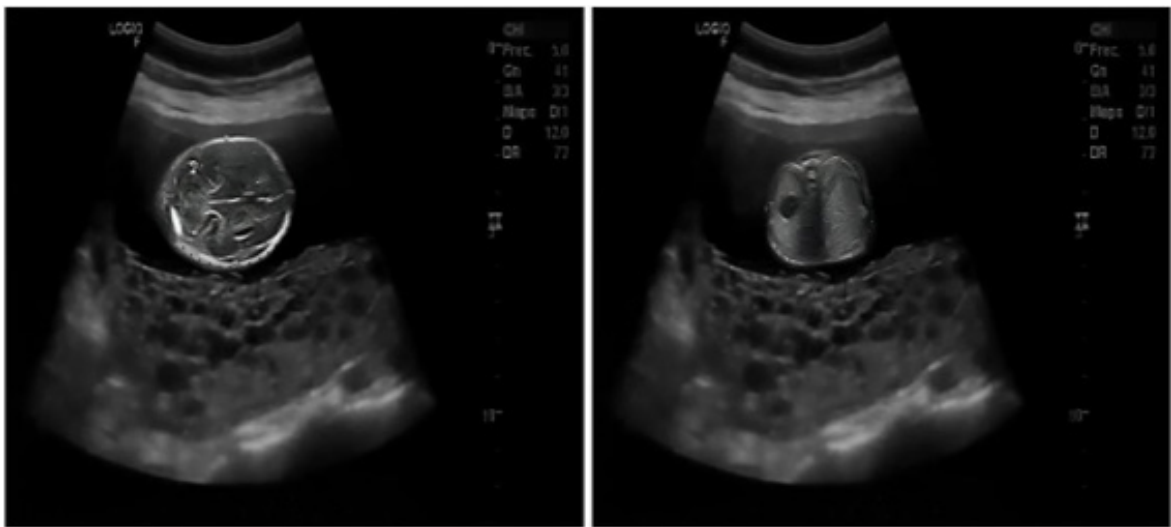


FIGURE 2. OBSTETRIC ULTRASOUND AT 23 WEEKS' GESTATION. THE IMAGE SHOWS FETAL CROSS-SECTIONS AT THE CEPHALIC (LEFT) AND THORACOABDOMINAL (RIGHT) LEVELS, DEMONSTRATING NORMAL FETAL ANATOMY FOR THE GESTATIONAL AGE. IN THE LOWER PORTION, SEVERE HETEROGENEOUS PLACENTOMEGALY (8 CM THICK) IS OBSERVED, WITH MULTIPLE SUBAMNIOTIC CYSTS AND VESICULAR ANECHOIC AREAS—FINDINGS CHARACTERISTIC OF THE PROGRESSION OF PLACENTAL MESENCHYMAL DYSPLASIA.

DISCUSSION

Pregnancies with placental mesenchymal dysplasia (PMD) are associated with intrauterine growth restriction (IUGR) and hypertensive disorders such as preeclampsia (PE). Its incidence ranges from 0.002% to 0.02% of analyzed placentas, leading to diagnostic confusion with partial hydatidiform mole on initial ultrasounds⁽⁸⁾. This condition results in rates of IUGR and preterm births of approximately 33%⁽⁹⁾.

In Peru, a case report study found that 100% (n=4) of affected pregnancies resulted in perinatal death⁽¹⁰⁾. The main distinction of our article is that a completely favorable perinatal outcome was achieved without the development of metabolic complications or fetal growth restriction.

The pathogenesis of PMD causes chronic hypoxia and a circulatory imbalance resulting from fetal vascular obstruction due to microthrombosis, diverting blood flow toward diseased villi. These abnormalities lead to marked fibromuscular hyperplasia⁽¹¹⁾. Concurrently, national histopathological evidence demonstrates the consistent presence of hydropic trunk villi with irregularly thick-walled, thrombosed vessels⁽¹⁰⁾. In this patient, ultrasound revealed severe heterogeneous placentomegaly measuring 8 cm with subamniotic cysts, a

morphological finding that aligns directly with the obstructive progression described in the national and international literature^(8,10,11).

To assess the impact of therapy, it has been reported that untreated uteroplacental insufficiency triggers severe IUGR detected at 30 weeks, with critical resistance indices on Doppler ultrasound⁽⁹⁾. In contrast, in this case, it was decided to initiate active pharmacological intervention by administering 150 mg of acetylsalicylic acid at night starting at 13 weeks' gestation. Conventional management of PMD is usually limited to expectant monitoring due to the lack of standardized guidelines, which may predispose patients to fatal outcomes⁽¹¹⁾. Early introduction of aspirin may represent a successful preventive strategy aimed at mitigating placental endothelial dysfunction.

The perinatal outcomes obtained strongly support the appropriateness of this active medical approach. The fetal growth curve remained stable between the 40th and 50th percentiles, resulting in a successful birth at 35 weeks. In contrast to the study of four cases, in which a pregnant woman developed severe preeclampsia at 25 weeks and her newborn died within one hour of birth⁽¹⁰⁾, the patient in this study had a pregnancy free of hypertensive events. This suggests that early antiplatelet therapy modulated vascular resistance in the malformed vessels of the placen-



ta, effectively counteracting the natural and adverse course of this condition.

In conclusion, this clinical case report provides valuable scientific evidence regarding the clinical management of a rare obstetric condition. In line with international recommendations^(8, 9) and the pathological analysis⁽¹⁰⁾ regarding the need for accurate prenatal screening, it is suggested that PMD be classified as an independent risk factor. This would justify the systematic initiation of prophylactic aspirin starting in the first trimester. Publishing these findings is essential for expanding knowledge and positively transforming the maternal-fetal prognosis in the country⁽¹⁰⁾.

ARTICLE INFORMATION

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