CASE REPORT

PMD Initially Diagnosed as Partial Mole

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Introduction

Placental mesenchymal dysplasia (PMD) is a rare condition of placentomegaly and abnormal chorionic villi often clinically mistaken as partial hydatidiform mole. However, it is clinico-pathologically distinct with high incidence of IUGR and fetal death [1]. We present a case of PMD which was initially suspected as a partial mole.

Case Report

A 23-year-old third gravida reported to the Department of Obstetrics and Gynecology, Govt. Medical College, Calicut, Kerala, as a referred case of suspected partial mole. She was at 34 weeks of gestation and had regular antenatal checkups from a private hospital. She had no previous medical/surgical problems. Her previous deliveries were normal, but the second baby died on the 15th postnatal day due to congenital heart disease.

On examination, the vitals were stable and systems were within normal limits. Uterine size corresponded to 32 weeks of gestation with cephalic presentation and good fetal heart sounds.

All the investigations were normal, except for an ultrasound taken at 21 weeks 4 days of gestation showing a live fetus of gestational age 20 weeks + 5 days with the placenta showing multiple anechoic areas. The pending diagnosis was hydropic degeneration of placenta, partial mole or extension of fibrin deposition.

A repeat ultrasound 2 weeks later showed an active fetus with the placenta showing increased thickness with multiple sonolucent areas of 2–5 mm with the possibility of a partial mole and/or PMD. Serial USGs showed a fetus with no detectable anomalies and placental findings consistent with a partial mole.

The patient was counseled regarding the possible diagnosis and the pregnancy was continued with her consent as she had no complication and the fetus had no other problems. At 38 weeks of gestation, labor was induced with prostaglandin E₂. She had a normal delivery and gave birth to a 2.18-kg female baby with Apgar '19. The baby had no external anomalies, but the placenta showed remarkably dilated vessels (Figs. 1, 2). The placenta was sent for histopathologic examination.

The mother and baby were well and were discharged on the third postnatal day. They were advised to review with β -hCG and fetal karyotyping after 6 weeks. Histopathology examination of the placenta showed hydropic villi with thick muscular vessels separated by edema fluid, myxoid

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stroma, and foci of chorangiosis, all of which were consistent with the diagnosis of PMD (Fig. 3).

Six weeks later, the patient came for review with the baby. Her serum β -hCG volume was then <1.2 μ IU/ml

(normal) and the karyotyping of the baby showed a normal 46 xx pattern.

Discussion

There are only 82 reported cases of PMD [1]. It is a benign stromal tumor considered to be a malformation of the placenta. Macroscopically, there is placentomegaly with markedly hydropic stem villi that resemble a partial mole, but there is no trophoblastic proliferation or trophoblastic inclusion [2].

The proposed etiology is androgenetic mosaicism. The same haploid paternal complement found in the androgenetic cells was present in coexisting biparental cells, suggesting origin from a single fertilization event [3]. Placental anomaly appears to be a limited malformation of extraembryonic mesoderm involving the mesenchyme and vessels of the stem villi of several cotyledons [1].

On gross appearance, the placenta shows aneurysmal and varicose dilatation of chorionic vessels. Microscopy shows areas of enlarged stem villi with cistern-like malformation intermixed with normal placental tissue, convoluted myxoid vessels, and chorioangiomatoid changes [4].

Ultrasound features resemble a partial mole with the placenta exhibiting multiple cystic spaces [5]. Increased MSAFP is a serum marker. Abnormal vascular permeability in mesenchymal dysplasia explains the transfer of large quantities of AFP from fetal to maternal circulation [6]. PAPP-A was also found to be raised in one case of mesenchymal dysplasia [6].

Karyotyping shows 88 % normal fetuses. A majority of the fetuses are female (78 %). Abnormal fetuses have associated conditions like trisomy 13, Klinefelter syndrome, Beckwith Wiedmann syndrome, and triploidy [7].

Fetal complications usually seen are IUGR fetal anemia and thrombocytopenia and intrauterine fetal demise [1].

Fig. 1 Baby with the placenta

Fig. 2 Placenta showing dilated vessels

Fig. 3 Histopathology showing myxoid stroma and aneurysmal dilatation

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