



# International Journal of Pathology Sciences

ISSN Print: 2664-9063  
ISSN Online: 2664-9071  
IJPS 2024; 6(1): 01-03  
[www.pathologyjournal.net](http://www.pathologyjournal.net)  
Received: 02-11-2023  
Accepted: 03-12-2023

**Vasundhara Kamineni**  
Department of Obstetrics and  
Gynecology, KAMSRC,  
Hyderabad, Telangana, India

**Haripriya V**  
Department of Obstetrics and  
Gynecology, KAMSRC,  
Hyderabad, Telangana, India

**Shailaja Prabhala**  
Department of Pathology and  
Lab Medicine, AIIMS  
Bibinagar, Hyderabad,  
Telangana, India

**Padmaja Korti**  
Department of Pathology,  
KAMSRC, Hyderabad,  
Telangana, India

**Corresponding Author:**  
**Shailaja Prabhala**  
Department of Pathology and  
Lab Medicine, AIIMS  
Bibinagar, Hyderabad,  
Telangana, India

## Placental mesenchymal dysplasia associated with severe fetal growth restriction: A case report

**Vasundhara Kamineni, Haripriya V, Shailaja Prabhala and Padmaja Korti**

DOI: <https://doi.org/10.33545/26649063.2024.v6.i1a.11>

### Abstract

Placental mesenchymal dysplasia is an uncommon condition attributed to abnormal mesenchyme leading to stem villous hyperplasia and vesicle formation in the placenta. On antenatal ultrasound it is easily confused with a molar pregnancy and hence, there is always a possibility of a medical termination of pregnancy. Here, we present a case report where the ultrasound scan was suggestive of a molar pregnancy but with no obvious abnormalities in the fetus. The pregnancy was monitored closely with regular ultrasound scans and was continued to almost full term. The patient underwent an elective Caesarean section in view of severe intrauterine growth restriction. A small for gestational age female baby was delivered with no apparent congenital malformations. Placental mesenchymal dysplasia was ascertained by the pathological examination of the placenta.

**Keywords:** Placental mesenchymal dysplasia, hydropic change of placenta, intrauterine growth restriction

### Introduction

Placental Mesenchymal Dysplasia (PMD) is an uncommon benign placental abnormality, about 100 cases being reported so far in literature <sup>[1]</sup>. The true incidence is not known as most pregnancies may be terminated early in gestation, or this distinct clinical entity may not be recognised because the condition closely mimics a partial molar pregnancy <sup>[2]</sup>. It is recognised as a placental vascular malformation, characterised by a large placenta with villous edema, cystic degeneration, and abnormal blood vessels. The fetus is however morphologically normal. Most cases are identified during routine antenatal sonographic evaluation. The classical feature being increased placental thickness with hypoechoic spaces on 2D imaging. Maternal and fetal complications reported are preeclampsia, preterm labour (33%), fetal growth restriction (FGR), intrauterine fetal death (IUID) (13%), neonatal death (NND), association with Beckwith-Weidman Syndrome (30%), Karyotype abnormalities (Trisomy13). Reasons for early pregnancy termination include mistaken diagnosis of partial mole, and maternal or fetal complications. Normal neonatal outcomes in spite of PMD have been reported in about 9% of the cases in one literature review.

We report here a case of PMD with severe fetal growth restriction where the pregnancy continued till 36weeks with no associated maternal complications.

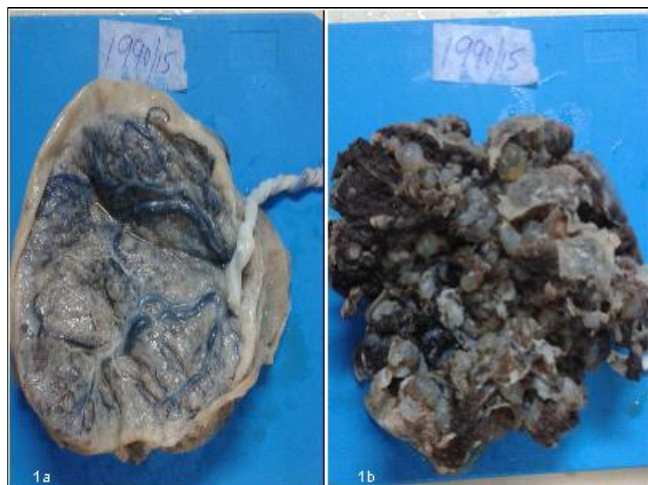
### Case Report

A 20 year old primigravida, booked elsewhere, first presented to our antenatal clinic at Kamineni Academy of Medical Sciences, Hyderabad, at 26 weeks of gestation. It was a spontaneous conception. History of hyperemesis in 1<sup>st</sup> trimester was present. TIFFA scan at 20 weeks was suggestive of normal fetal survey, cystic spaces within the placenta consistent with a partial molar pregnancy and she was advised to undergo termination of pregnancy. The couple however refused termination and presented at our centre for further care. Amniocentesis and FISH analysis revealed a diploid karyotype and the couple were counseled about the same. Pregnancy was monitored with serial ultrasonography. There was evidence of mild oligohydramnios and severe fetal growth restriction at 35 weeks. Antenatal corticosteroids were administered.

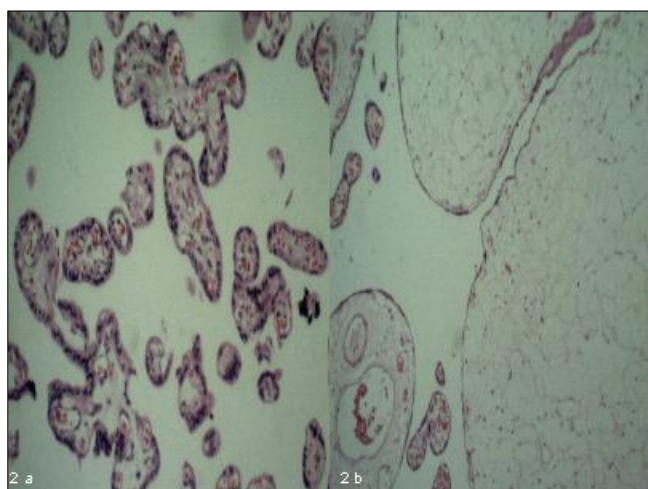
A doppler study at 36 weeks was suggestive of uteroplacental insufficiency and NST was non-reassuring. Elective caesarean section was performed and a live late preterm female baby was delivered with birth weight of 1.26 kg, APGAR score of 6 and 8, without any obvious congenital anomalies. The baby was admitted to NICU for respiratory distress and low birth weight and was later discharged on 5<sup>th</sup> day of life.

On pathological examination, the placenta weighed 450 gm, and measured 14 x 13 x 6 cm. The fetal surface showed thick, engorged, tortuous vessels. The membranes appeared normal. The maternal surface showed ill formed cotyledons and many variable sized cysts filled with clear fluid. The umbilical cord was 25 cm in length, marginally inserted and its cut surface showed three vessels, i.e. two arteries and one vein. (Figure 1)

The histopathology sections showed admixture of unremarkable third trimester chorionic villi and villous structures showing marked hydropic change, absence of vessels within these villi and having a lining of single layer of attenuated trophoblastic cells. Many vessels with thick walls/fibromuscular dysplasia were seen. None of these villi revealed trophoblastic proliferation. It was reported as Placental Mesenchymal Dysplasia. (Figure 2).



**Fig 1:** Gross appearance of placenta with multiple fluid filled vesicles



**Fig 2:** Microscopy of placenta showing normal villi (2a) and hydropic villi with dilated vessels (2b), Hand E staining, 40 X and 100X.

## Discussion

PMD, also sometimes referred to as placental mesenchymal hyperplasia, pseudo partial mole is a rare entity first described by Moscoso *et al.* in 1991<sup>[3]</sup> and about 100 cases reported since then. Arizawa and Nakayama reported its incidence around 0.02%<sup>[4]</sup>. It is characterized by placental engorgement with multiple grape-like vesicles seen on 2D USG. Grossly, PMD placentas are large for gestational age with aneurysmally dilated chorionic plate vessels with fibromuscular hyperplasia. Cystically dilated vesicles are present, which are similar to those seen in molar pregnancies. Microscopically, these vesicles correspond to dilated stem vessels with thickened vasculature surrounded by normal villi. Pathologically, PMD can be distinguished by the presence of dilated stem vessels and lack of trophoblastic proliferation. The differential diagnosis includes partial molar pregnancy, complete mole with a co-twin, and chorioangioma. Karyotype is however normal and there is a female preponderance (46XX) as was seen in our case. Differentiation is important for subsequent management and offering prognosis. The risk of persistent gestational trophoblastic disease to the mother does not exist unlike with a complete mole with a co-twin.

Nayeri *et al.*<sup>[5]</sup> in their review of 61 cases reported pregnancy complications IUGR, IUFD, PTB. Association with BW syndrome (Macrosomia, exomphalos, macroglossia, omphalocele, visceromegaly, placentomegaly and childhood tumors) was seen in 23% of cases. Chan *et al.*<sup>[6]</sup> in their case series reported poor obstetric outcome in all the four cases. The index case was associated with severe fetal growth restriction but had no other anomalies and there were no maternal complications. Cohen *et al.*<sup>[7]</sup> described 3 cases of PMD associated with fetal aneuploidy. Truc *et al.*<sup>[8]</sup> in their analysis of 11 cases reported the incidence of IUGR to be 50%, IUFD and NND to be 43%. They proposed that obstructive fetal vascular thrombosis and the consequent reduction in maternal-fetal gas exchange to be responsible for IUFD. Poor oxygenation at the dysplastic villi makes the fetus susceptible for IUGR.

## Conclusion

PMD, though a rare entity, probably goes unrecognized and hence under-reported because it closely mimics a partial molar pregnancy. Favourable pregnancy outcome is possible with close monitoring. There is a high incidence of preterm births, IUGR, IUFD, neonatal death and association with Beckwith-Weidmann syndrome. PMD should always be considered in the differential diagnosis in women presenting with typical sonographic abnormalities and fetal karyotype determination is recommended. The woman and partner have to be properly counseled regarding the possible outcomes and adequate support provided.

## References

1. Ohira S, Ookubo N, Tanaka K, Takatsu A, Kobara H, Kikuchi N, *et al.* Placental mesenchymal dysplasia: chronological observation of placental images during gestation and review of the literature. *Gynecol Obstet Invest.* 2013;75(4):217-23.
2. Parveen Z, Tongson-Ignacio EJ, Fraser CR, Killeen JL, and Thompson KS. Placental Mesenchymal Dysplasia. *Archives of Pathology & Laboratory Medicine.* 2007;131(1)131-137.

3. Moscoso G, Jauniaux E, Hustin J. Placental vascular anomaly with diffuse mesenchymal stem villous hyperplasia. A new clinico-pathological entity? *Pathology, Research and Practice*. 1991;187:324-328.
4. Arisawa M, Nakayama M. Suspected involvement of the X chromosome in placental mesenchymal dysplasia. *Congenital Anomalies (Kyoto)*. 2002;42:309-317.
5. Nayeri UA, West AB, Nardini HAG, Copel JA, Sfakianaki AK. Systematic review of sonographic findings of placental mesenchymal dysplasia and subsequent pregnancy outcome. *Ultrasound Obstetrics and Gynecology*. 2012;41(4):366-374.
6. Chan YF, Sampson A. Placental mesenchymal dysplasia: a report of four cases with differentiation from partial hydatidiform mole. *Australian and New Zealand Journal of Obstetrics and Gynaecology* 2003;443:475-479.
7. Cohen MC, Roper EC, Sebire NJ, Stanek J, Anumba DOC. Placental mesenchymal dysplasia associated with fetal aneuploidy. *Prenat Diagn*. 2005;25:187-192.
8. Truc P, Julie S, Carla S, Chan L. Placental mesenchymal dysplasia is associated with high rates of intrauterine growth restriction and fetal demise. A report of 11 new cases and a review of the literature. *American Journal of Clinical Pathology*. 2006;126:67-78.