e-ISSN: 0975-1556, p-ISSN:2820-2643

Available online on www.ijpcr.com

International Journal of Pharmaceutical and Clinical Research 2025; 17(1); 915-920

Case Report

A Case Report of Intrauterine Abrupt Fetal Death at 24 Weeks Due to Placental Mesenchymal Dysplasia

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Received: 25-10-2024 / Revised: 23-11-2024 / Accepted: 26-12-2024

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Conflict of interest: Nil

Abstract:

Introduction: An uncommon placental defect called placental mesenchymal dysplasia (PMD) is typified by placentomegaly and grapelike vesicles that resemble molar pregnancy. According to reports, the prevalence of PMD is 0.02%, with a clear female preponderance. To avoid needless pregnancy termination, it's critical to distinguish PMD from its mimics, particularly molar pregnancy. Unlike molar pregnancies, PMD usually features a normal fetus. However, PMD is distinct in that it has a high incidence of fetal growth restriction (FGR) and intrauterine fetal death (IUFD).

Case report: A 22-year-old primigravida at 24 weeks and 5 days of gestation, with sudden intrauterine fetal demise. She presented to this hospital with a complaint of not perceiving fetal movements for the last 3 to 4 days. Ultrasonographic evaluation showed a single dead fetus with marked tissue edema, ascites, and pleural effusion and a large hyperechoic mass suggestive of hydrops fetalis with placental SOL with multiple loculated cysts. MRI revealed a single intrauterine gestation of average gestational age 21 weeks 4 days with mesenchymal dysplasia of the placenta and hydrops fetalis.

Conclusion: The differential diagnosis of placental cystic lesions by sonography should include PMD if a fetus with a phenotypically normal appearance can be identified. The fetal death in this case might be due to the marked vascular changes in the placenta leading to cardiac decompensation, hydrops fetalis, and intrauterine death.

Keywords: PMD: Placental Mesenchymal Dysplasia.

Introduction

Placental mesenchymal dysplasia (PMD) is a rare placental anomaly characterized by placentomegaly and grapelike vesicles resembling molar pregnancy. The incidence of PMD is reportedly 0.02%1, with a definite preponderance of females [2].

Clinically and pathologically, PMD has a differential diagnosis with partial hydatidiform mole, twin pregnancy with complete mole, and spontaneous mosaicism. [2] On pathologic examination, the placenta is enlarged with tortuous and dilated chorionic vessels on the fetal surface. [1,3]

Other characteristic features are stem villous hydrops without trophoblastic proliferation, villous chorangiosis, and chorangimatosis. The fetus usually has a normal karyotype with female predominance and is associated with intrauterine growth retardation (IUGR), stillbirth, Beckwith-Widemann syndrome (BWS), or normal fetal morphology. [1,3]

Case Report

A 22-year-old lady, primigravida with a gestational age of 24 weeks and 5 days, was admitted to this hospital with a chief complaint of decreased fetal movement for the last 3 to 4 days. Her general physical examination and systemic examination did not reveal any abnormality. Per abdominal examination, the uterus was 24 weeks in size, relaxed, no fetal movements felt, and absent fetal heart sounds on auscultation.

Ultrasonographic evaluation showed a single dead fetus, with marked tissue edema, ascites, pleural effusion, and a large hyperechoic mass suggestive of hydrops fetalis. She delivered a single 800-gram dead fetus with hydrops fetalis. Both the mother and the fetus were RH+. Grossly the placenta measured 12 X 10 X 5 cm and weighed 500 grams.

The placenta was slightly enlarged and bulky. The umbilical cord measured 20.5 cm in length and showed marginal insertion. The chorionic plate showed numerous abnormally large, dilated varicose-like vessels. The parenchyma showed few grape-like cysts and a tan-gelatinous appearance. The membranes were unremarkable. (Fig.1)

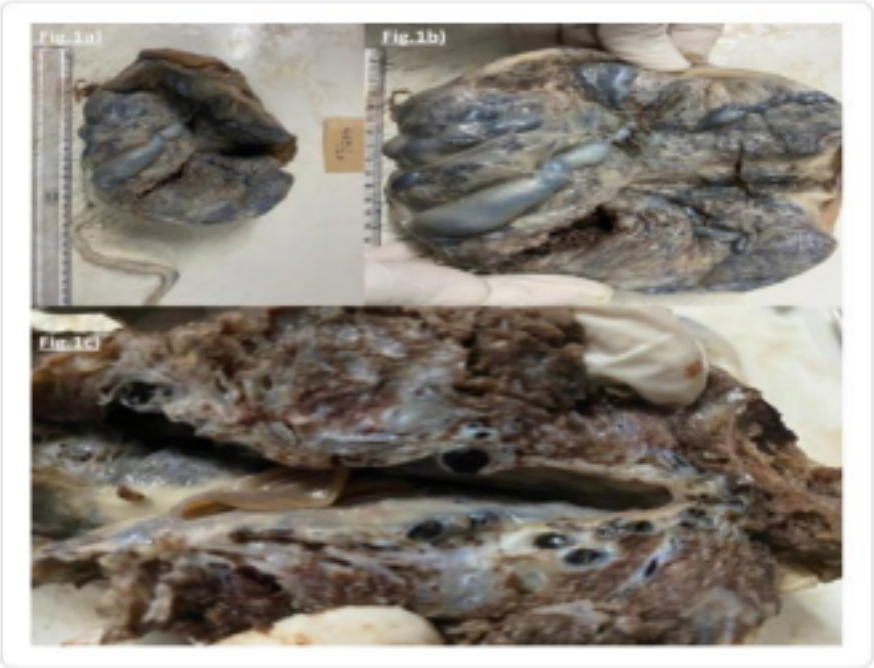
A heterogeneous population of villi, including mature third-trimester villi and larger villi with a myxoid or hydropic look, was visible under a microscope. (Fig.2a). Significant stromal edema, loose connective stroma, and spindle cells were observed in enlarged villi. Because of fluid buildup, the stroma was hypovascular and displayed the development of cisternae (Fig.2b).

There was no trophoblastic hyperplasia or inclusions. Some of the distal villi were large, and dysplastic (Fig.3 a) and showed chorangiosis (Fig. 4 a) with numerous capillary vessels, more than 10 per villus in numerous villi. (Fig. 4 b) Some of the stem villi showed decreased vessels located in the periphery. Many large abnormally thick blood vessels with thick muscular walls showing hemorrhage and thrombosis of the lumen were seen.(Fig3b)

With these gross and microscopic features, we made a histopathological diagnosis of Placental mesenchymal dysplasia.

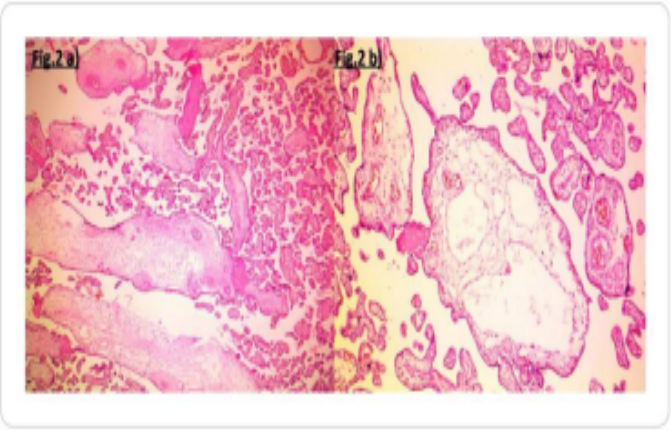
A single deceased fetus, significant tissue edema, ascites, and pleural effusion, as well as a sizable hyperechoic mass suggestive of hydrops fetalis with placental SOL and several loculated cysts, were all visible on the ultrasound evaluation. An MRI showed a single intrauterine pregnancy with hydrops fetalis and placental mesenchymal dysplasia, with an average gestational age of 21 weeks and 4 days.

Figure 1:



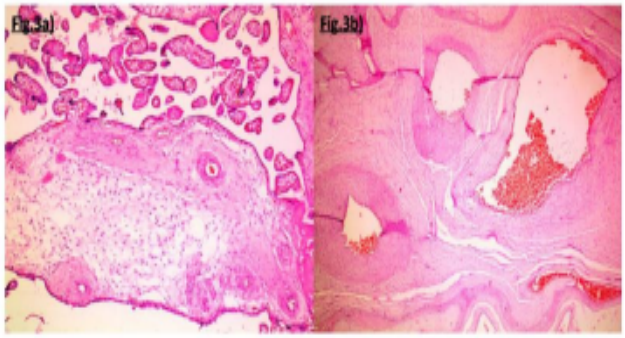
Gross images of the case showing a): Enlarged placenta with marginal umbilical cord insertion and dilated blood vessels; b): Placenta showing numerous abnormally large, dilated varicose like blood vessels; c): Placenta showing dilated blood vessels and few cystic myxoid spaces

Figure 1(a,b):



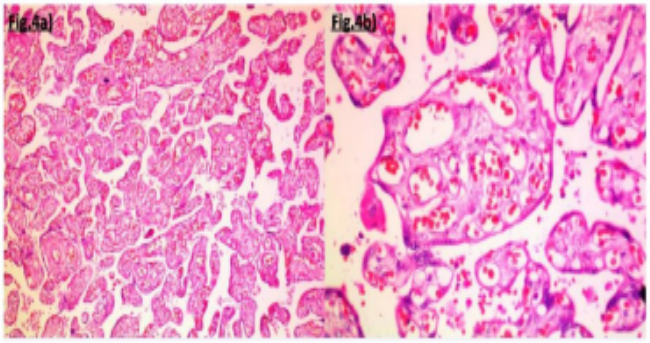
H&E stained sections from the placenta showing; a): mixed population of enlarged dysplastic villi among mature third trimester villi (40X); b): Stroma of dysplastic villi composed of loose connective tissue and formation of cisternae as a result of fluid accumulation(100X)

Figure 2(a,b):



H&E stained sections from the placenta showing; a): Large dysplastic villi with loose oedematous stroma with spindle cells and peripherally located thick walled blood vessels. There is no trophoblastic hyperplasia (100X); b): Abnormally large thick walled blood vessels(40X)

Figure 3 (a,b):



H&E stained sections from the placenta showing; a): Immature 3rd trimester villi with chorangiosis (100X); b): Immature 3rd trimester villi showing chorangiosis with numerous capillary vessels, more than 10/villous in numerous villi(400X)

Figure 4 (a,b):

Discussion

Placental mesenchymal dysplasia (PDM) is a rare disease, the cause is unknown, some are called mesenchymal stem hyperplasia, and some are called pseudo-vesicular block or vascular malformation. [4,2] PMD was initially described by Moscoso et al in 1991 as placentomegaly which gives the image of partial hydatidiform mole with an elevated level of alpha-fetoprotein. [5] Due to a lack of clinical knowledge, it is challenging to estimate the incidence of PDM, which could lead to underreporting overall.

However, it is only identified in cases where the placenta is complex or diseased. [6,7] The incidence of PMD may rise as the pathologist's awareness increases. [8,9] The pathogenesis of

PMD is unclear. [10] According to Chen CE and others, stem chorionic mesenchymal cells' growth is followed by mesenchymal cell hyperplasia, and the increased stem villi contain normal villus mesoderm connective tissue. Eosinophilic mucopolysaccharide is seen, which is likely a congenital mesoderm abnormality. A small percentage of individuals have chromosomal abnormalities, such as 13-trisomy, triploidy, and 13q12.11 affecting the GJB6 gene, but the majority of PMD patients have a normal karyotype of prenatal chromosomal analysis. [11,12] Androgenetic/biparental mosaicism has been proposed as the fundamental etiology of PMD, even though the pathophysiology of the condition is yet unknown. [13] Typical clinical signs of PDM in the early stages of pregnancy are absent. Ultrasound or a routine prenatal examination can be used to diagnose it. Due to the placenta's enlarged surface area in PDM, blood alfetoprotein is higher while serum ßHCG is either normal or slightly elevated. [14] Poor placental perfusion may be the cause of intrauterine growth limitation, intrauterine mortality, and early birth in people with PMD. Anemia, thrombocytopenia, or microangiopathic hemolysis are the most common fetal consequences, and they may be connected to the aberrant blood vessel distribution. [15] To avoid needless pregnancy termination, it's critical to distinguish PMD from its mimics, particularly molar pregnancy. In contrast to molar pregnancies, PMD typically results in a healthy fetus. But PMD is unique in that it is linked to Beckwith-Wiedemann syndrome (BWS), which is characterized by omphalocele, macrosomia, visceromegaly, and macroglossia, and it has a high incidence of fetal growth restriction (FGR) and intrauterine fetal death (IUFD). [3] The imaging findings of PMD are similar to those of hydatidiform moles, which are easily misdiagnosed before delivery. Placental dysplasia is due to the cystic expansion of some of the villi, which should be differentiated from the vesicular vesicles. [5]

In terms of pathology, PMD placentas are typically large and exhibit edema of stem villi with intact terminal villi as well as a variety of vascular abnormalities, including cirsoid chorionic vessels, thrombosis, vessel wall thickness, vascular stenosis, villous cholangitis, chorioangioma, and fetal thrombotic vasculopathy. Additionally, aberrant umbilical cords, such as twisted, tortuous, or too lengthy cords. [3,14] Absence of trophoblastic proliferation in PMD placentas is a histological difference from partial moles. [5]

Imaging tests in this case report suggested a molar pregnancy. The placenta appeared much larger and heavier upon physical inspection. There was minimal implantation in the umbilical cord. Numerous unusually big, dilated varicose-like veins were visible on the chorionic plate. The parenchyma had a tan-gelatinous appearance with a few grape-like cysts. The membranes weren't particularly noteworthy. Mature distal villi and dysplastic stem villi were both present in the mixed villi population observed under a microscope. Cistern formation and stromal edema were visible in the larger villi. The immature villi had cholangitis symptoms. Neither inclusions nor trophoblastic hyperplasia were present. These microscopic and gross findings verified PMD and ruled out hydatiform mole.

Conclusion

For diagnosis and differential diagnosis, a thorough histological examination is necessary. In the event that a fetus with a phenotypically normal appearance may be recognized, PMD should be included in the differential diagnosis of placental cystic lesions by sonography. Since sudden IUFD, including rupture of the cirsoid chorionic vessels, can occur at any time during gestation, termination of the pregnancy at an optimal time could be considered when prenatal characteristics of PMD are detected, such as an elevated AFP and normal hCG level in the mother, a normal female karyotype revealed by amniocentesis, and dilated subchorionic vessels revealed by ultrasonography during the third trimester. In this instance, the placenta's significant vascular alterations may have caused cardiac decompensation, hydrops fetalis, and intrauterine death.

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