Case Series

Evaluation and analysis of influencing factors of placental mesenchymal dysplasia diagnosed using prenatal ultrasonography and pregnancy outcomes: case series

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Background: Placental mesenchymal dysplasia (PMD) is a rare placental vascular malformation of unknown etiology. PMD may coexist with a healthy fetus, and its ultrasound appearance is similar to that of a hydatidiform mole, especially the partial type. Prenatal ultrasonography is vital for accurate diagnosis of these conditions. This study aimed to summarize the characteristics of prenatal ultrasonographic images across different gestational weeks (W) for PMD and evaluate and analyze factors that influence pregnancy outcomes related to PMD. The goal is to improve the diagnosis of PMD, effectively assess fetal prognosis, and provide a reference for prenatal consultations and clinical management.

Case Description: Of the 15 included patients, 4, 8, and 3 had PMD in early pregnancy (<13^{+6} W), mid-pregnancy (approximately 14–27^{+6} W), and late pregnancy (>28 W), respectively. Among the 15 patients, 5 successfully underwent delivery, thereby resulting in fetal survival; 3 experienced intrauterine death, 1 had a miscarriage, and 6 pregnancies were terminated. During early pregnancy, ultrasonographic manifestations of PMD included microscopic anechoic cystic areas in the placental parenchyma. In the second trimester, the placenta exhibited diffuse enlargement and thickening, with the placental parenchyma showing cellular anechoic cystic areas clearly separated from the surrounding normal placental tissue. As the pregnancy progressed, the cystic areas gradually reduced in the third trimester. Additionally, localized umbilical blood vessels showed tumorous lesions, sometimes accompanied by intravascular thrombosis. Some cases exhibited tortuosity and dilation in the umbilical vein.

Conclusions: PMD exhibited varying ultrasonographic characteristics across different gestational stages and demonstrated regular disease evolution corresponding to gestational W. This condition is associated with adverse pregnancy outcomes, with the location, extent, and severity of lesions being crucial factors affecting fetal development in utero.

Keywords: Placental mesenchymal dysplasia (PMD); ultrasound; pregnancy; hydatidiform mole; case series

Submitted Oct 31, 2023. Accepted for publication Jul 22, 2024. Published online Aug 28, 2024.
doi: 10.21037/qims-23-1548
View this article at: https://dx.doi.org/10.21037/qims-23-1548

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Introduction

Placental mesenchymal dysplasia (PMD) is a rare placental vascular malformation with an unknown etiology, which is classified as a non-hydatidiform mole pregnancy. Ultrasonographic findings include multiple cystic hypo anechoic zones within the placental parenchyma, clearly demarcated from normal placental tissues and accompanied by localized placental enlargement and thickening. Placental blood vessel abnormalities exhibit diffuse tortuous dilatation of the villi and may be associated with thrombus formation. PMD can coexist with a normal fetus, and its ultrasonographic appearance shares similarities with hydatidiform mole, particularly partial hydatidiform mole. A hydatidiform mole typically presents with symptoms such as abnormal enlargement of the uterus after menopause, vaginal bleeding, severe nausea and vomiting during pregnancy (hyperemesis gravidarum), hypertension, and luteinized ovarian cysts. The symptoms of a partial hydatidiform mole are similar to those of a complete hydatidiform mole but are generally milder (1). The majority of sonographers have limited knowledge about this disease, thereby leading to potential confusion between the 2 conditions. PMD and partial hydatidiform mole are classified as abnormal placental lesions; however, PMD has a significantly better prognosis than partial hydatidiform mole. Therefore, prenatal ultrasonography plays a crucial role in accurately diagnosing these conditions. Currently, literature on PMD primarily consists of case reports both in China and internationally. This study aimed to summarize the characteristics of prenatal ultrasonographic images at each gestational week (W) for PMD while evaluating and analyzing factors influencing PMD-related pregnancy outcomes. The objective is to enhance PMD diagnosis, effectively evaluate fetal prognosis, and provide reference material for prenatal consultations and clinical management. This study enrolled a total of 15 patients diagnosed with placental interstitial development disorders by prenatal ultrasound at the Gansu Provincial Maternal and Child Health Hospital from March 2020 to August 2023. Comprehensive analysis was conducted on the prenatal echocardiography and clinical data of these 15 patients with placental interstitial development disorders. The average age of all enrolled patients was 29.06±3.01 years, and the average gestational age was 21.73±6.85 W. We present this article in accordance with the AME Case Series reporting checklist (available at https://qims.amegroups.com/article/view/10.21037/qims-23-1548/rc).

Case presentation

Ethical considerations

This study is a retrospective investigation. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013) and was granted an exemption from the Medical Ethics Committee of Gansu Provincial Maternity and Child-care Hospital. The participants provided their written informed consent to publish their cases (including publication of images). A copy of the written consent is available for review by the editorial office of this journal.

Study design and results

This is a retrospective case series study aimed at evaluating the ultrasonographic manifestations of PMD at different stages and its differential diagnosis.

Prenatal ultrasonography assessment of patients with PMD included the following parameters: (I) measurement of placental thickness; (II) evaluation of placental internal echo and blood flow; (III) range and location of placental lesions; (IV) boundary demarcation with normal placental tissue; (V) determination of umbilical cord insertion site on the placenta; (VI) evaluation of fetal growth, development, and presence of structural malformations; (VII) ultrasonic image characteristics at different stages of lesion evolution. Continuous monitoring was performed for fetal growth, amniotic fluid volume, and vascular pulse index. Follow-up visits were scheduled every 2–3 W for pregnancy examination data tracking. Postpartum follow-up for patients with PMD includes monitoring both the postpartum period and the induction of labor, observing the status of postpartum neonates, performing ultrasonography on newborns, and performing follow-up visits at 1 month, 3 months, 6 months, and annually thereafter. Furthermore, this process involves evaluating postpartum neonatal growth and development and keeping detailed records. Maternal recovery within 3 months following labor induction was monitored to observe whether PMD had any effect on maternal recovery, and the results were duly recorded. With the consent of the hospital's ethics committee and the respective parents, a pathological autopsy was performed on a section of the placenta, and comprehensive records were maintained.

In the analysis of clinical data, the lesion area in 6 patients was located at the umbilical cord entrance of the placenta, covering the cord entrance. Among these cases, 3 patients exhibited diffuse placental thickening with

abnormal internal, an increased fetal cardiothoracic ratio, abnormal umbilical artery flow signals, massive effusion of the fetal pericardium, intrauterine growth restriction (IUGR), and intrauterine fetal demise. In 1 case, fetal umbilical vein dilation, an abdominal septate cystic space, oligohydramnios, and IUGR were noted, thereby leading to surgical abortion. In 2 cases, the patients had extensive placental lesions that affected placental function, leading to surgical abortions for both. In the other 5 patients, the diseased area was located at the edge of the placenta, did not cover the umbilical cord entrance of the placenta, and the umbilical cord entrance was situated in the normal region of the placenta. The extent of the placental lesions gradually diminished after 30 W of pregnancy. Among these, 2 cases resulted in preterm births. The main cause of premature birth in fetuses is abnormal placental conditions in the late stage of pregnancy, leading to restricted intrauterine growth and development of the fetus; therefore, medical interventions were necessary in these cases. Meanwhile, 3 cases had full-term deliveries, and the fetuses survived. In 1 patient, abortion occurred at 12 W of pregnancy, and 3 patients underwent early induced abortion following a definitive diagnosis (*Table 1*).

The extent of lesions in patients with PMD within this group varied in our analysis of factors influencing pregnancy outcomes. By employing ultrasound to measure and delineate the extent of the lesion, a comprehensive result was derived after repeated measurements conducted 3 times by experts with extensive experience in ultrasound diagnosis. Ultrasonographic measurements revealed that placental involvement accounted for 15–65% of the placental volume in 10 cases, whereas it exceeded 65% in 5 cases. PMD lesions that are extensive and close to or covering the placental umbilical cord entrance significantly impact placental function. This can result in inadequate blood and oxygen supply to the fetus, thereby leading to

Table 1 Analysis of prenatal ultrasound signs and clinical data of patients with PMD

Order number	MA (years)	Gravidity/ TPAL	Initial gestational age determination (W)	Lesion location	Lesion range (mm)	Abnormal ultrasound signs	Pregnancy outcomes
1	27	G_4P_{1031}	12^{+3}	The right lateral wall of the placenta	65×34	Numerous honeycomb-like anechoic patterns within the placental parenchyma completely enveloped the internal cervical opening	Early induced abortion
2	32	G_4P_{2022}	12^{+4}	In the uterine cavity	38×17	Multiple heterogeneous echoes within the uterine cavity were indicative of PMD	Suspension of embryo
3	30	G_2P_{0020}	21^{+6}	The entrance of the umbilical cord to the placenta	189×32	The placenta appeared as a heterogeneous mass with multiple honeycomb-like anechoic sacs	Induced abortion in the second trimester
4	32	G_3P_{2011}	29^{+2}	Placental margin	44×27	A vesicular cystic lesion was observed at the edge of the placenta, partially covering the internal cervical opening	Natural delivery at term
5	33	G_2P_{2002}	15^{+5}	The entrance of the umbilical cord to the placenta	67×51	The placenta exhibited enlargement and thickening, the inner diameter of the abdominal segment of the umbilical vein was widened, the fetal abdominal cavity was divided and occupied by cysts, and fetal oligohydramnios was detected	Induced abortion in the second trimester
6	29	G_3P_{0121}	13^{+5}	Placental margin	38×29	The placenta exhibited thickening with multiple anechoic honeycomb-like structures	Natural delivery at term

Table 1 (*continued*)

Table 1 (*continued*)

Order number	MA (years)	Gravidity/ TPAL	Initial gestational age determination (W)	Lesion location	Lesion range (mm)	Abnormal ultrasound signs	Pregnancy outcomes
7	26	G_2P_{0020}	23^{+6}	The entrance of the umbilical cord to the placenta	203×96	The placenta was enlarged with multiple abnormal echoes, the umbilical blood flow signal disappeared, and intrauterine growth restriction was observed in the fetus	Intrauterine fetal death
8	24	G_1P_{0010}	13^{+3}	Posterior to the placenta	54×34	Multiple honeycomb-like anechoic areas were noted in the placenta, low-lying placenta, and signs of intrauterine hemorrhage	Early induced abortion
9	28	G_1P_{0010}	22^{+6}	Placental margin	82×30	The placenta exhibited enlargement and thickening, along with the presence of multiple cystic structures, indicative of intrauterine growth restriction	The birth was premature, and the fetus survived
10	23	G_1P_{1001}	25	The entrance of the umbilical cord to the placenta	156×67	The placenta exhibited thickening, with multiple honeycomb-like cystic anechoic masses within the parenchyma, along with localized umbilical hemangioma-like dilatation	Induced abortion in the second trimester
11	29	G_4P_{1122}	15^{+2}	Left lateral wall of the placenta	63×24	The placenta exhibited multiple vesicular anechoic cysts and an abundance of blood flow signals between the uterine muscle walls	Early induced abortion
12	31	G_5P_{2121}	24	Placental margin	113×56	Multiple anechoic areas were observed within the placenta, and an elevated S/D ratio in umbilical blood flow	The birth was premature, and the fetus survived
13	26	G_2P_{2002}	28^{+6}	The entrance of the umbilical cord to the placenta	67×39	The placenta exhibited diffuse thickening, an increased fetal cardiothoracic ratio, tortuous and dilated umbilical veins, massive pericardial effusion and the fetus was experiencing intrauterine distress	Intrauterine fetal death
14	30	G_5P_{3023}	13^{+2}	The entrance of the umbilical cord to the placenta	129×54	The placenta exhibited diffuse thickening and echogenicity, whereas the fetal cardiothoracic ratio was increased. Evidence of umbilical artery diastolic reverse flow with a wave inversion, indicating fetal distress in utero, was noted	Intrauterine fetal death
15	36	G_2P_{1011}	35^{+4}	Placental margin	75×41	The placenta exhibited thickening with multiple cystic masses	Caesarean section was performed at term

PMD, placental mesenchymal dysplasia; MA, maternal age; TPAL, term births, premature births, abortions, living children; W, week; mm, millimeter; S/D, peak systolic velocity/end diastolic.

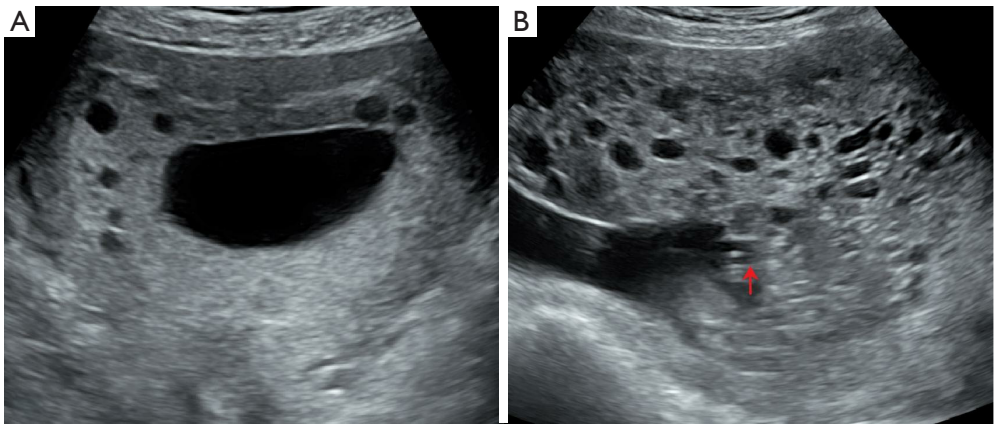

Figure 1 Case 1. (A) Ultrasound image at 13 weeks of pregnancy with PMD. Small cystic hypoechoic areas present within the placental substance. (B) Ultrasound image at 17 weeks of pregnancy with PMD. Gradual increase in cystic hypoechoic areas within the placental substance. The red arrow points to the umbilical cord insertion site, and the affected region is located at the entrance of the placental umbilical cord. PMD, placental mesenchymal dysplasia.

poor blood perfusion and ultimately causing intrauterine fetal hypoxia, which may lead to intrauterine death or stillbirth. Conversely, when the lesion is small and located at the placental periphery away from the umbilical cord entrance, its impact on fetal growth and development is minimal, thereby resulting in a more favorable fetal prognosis.

Characteristics of prenatal ultrasound images of patients with PMD at different stages of pregnancy: (I) early pregnancy (<13^{+6} W): heterogeneous thickening of the placenta and microcystic anechoic areas may manifest in the placental parenchyma. (II) Mid-pregnancy (approximately 14–27^{+6} W): the placenta exhibited diffuse thickening with uneven internal echoes. The placental parenchyma displayed multiple honeycomb cystic anechoic areas, predominantly oval in shape and distributed parallel to the long axis of the placenta. The cystic regions showed poor tone, whereas both the placental and proximal villous blood vessels were dilated. Furthermore, a clear boundary was observed between the abnormal placental tissue and its surrounding normal counterparts. (III) Late pregnancy (>28 W): as pregnancy progressed, the cystic component gradually decreased; in the third trimester, localized umbilical hemangiomatous lesions with intravascular thrombosis were observed. Some cases exhibited tortuosity and umbilical vein dilation, completely enveloping the internal cervix; color Doppler ultrasonography revealed no abnormal blood flow signals in the lesion area.

Ultrasound images depicting changes in placental and umbilical cord insertion sites at different stages of pregnancy in 2 cases of PMD: case 1 (*Figures 1-3*). A pregnant woman was initially diagnosed with PMD at 13 W and 2 days of gestation. At 35 W of pregnancy, the fetal cardiothoracic ratio increased, and an inversion of the A wave in the fetal ductus venosus was observed, indicating fetal distress in utero. During the preparation for a cesarean section, intrauterine fetal demise occurred; case 2 (*Figures 4-6*). Another pregnant woman was diagnosed with PMD at 15 W and 5 days of gestation. During the follow-up, a cystic mass appeared in the fetus's abdomen, which grew larger with increasing gestational W. The placental lesion also expanded as the pregnancy progressed. With oligohydramnios and fetal growth restriction, the pregnant woman chose to terminate the pregnancy at 27 W.

Monitoring of pregnancy outcomes and placental pathology of 15 patients with PMD: We monitored the pregnancy outcomes and placental pathology of 15 patients with PMD. Of these 15 patients, 5 experienced successful deliveries, whereas 10 opted for termination due to various reasons. Following delivery, the newborns displayed stable vital signs, and no apparent abnormalities in their appearance were observed. Following 1-year follow-up, the mothers and their babies were in good health. There were 3 patients who agreed to undergo placental pathology examinations.

Results of the pathological examination of the placenta in 3 cases: (I) the damage to the fetal membrane was incomplete, with blood clots attached to the maternal surface of the placenta. Additionally, some villi in the near marginal area of the maternal surface appeared

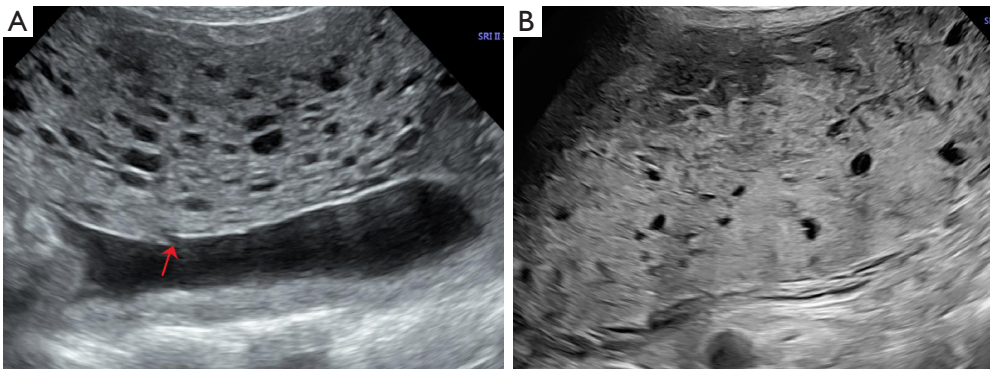

Figure 2 Case 1. (A) Ultrasound image at 24 weeks of pregnancy with PMD. Multiple honeycomb-like anechoic areas present within the placental substance. The red arrow points to the umbilical cord insertion site, and the affected region is located above the entrance of the placental umbilical cord. (B) Ultrasound image at 31 weeks of pregnancy with PMD. The placenta is significantly thickened, and cystic hypoechoic areas gradually decrease. The lesion partially covers the umbilical cord insertion site. PMD, placental mesenchymal dysplasia.

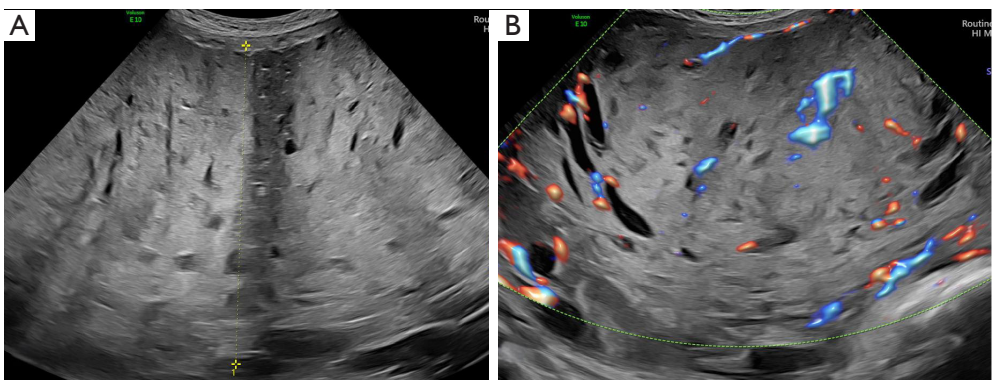

Figure 3 Case 1. (A,B) Ultrasound image at 35 weeks of pregnancy with PMD. The placenta displays diffuse thickening, and there is irregular echogenicity within the placental substance. The affected area is relatively broad, extending over the entrance of the umbilical cord into the placenta. PMD, placental mesenchymal dysplasia.

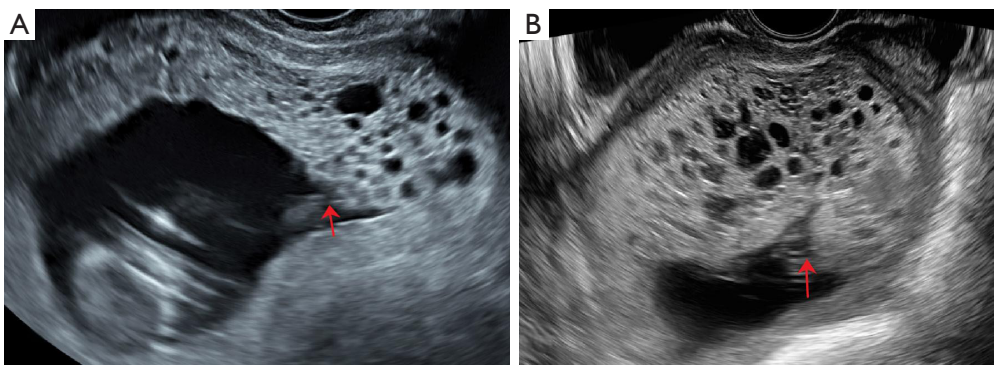

Figure 4 Case 2. (A) Ultrasound image at 15 weeks of pregnancy with PMD. Multiple cystic, anechoic structures are observed within the placental substance. The red arrow indicates the entrance of the umbilical cord into the placenta, and the affected area is situated on the right side of the placental wall, in close proximity to the entrance of the umbilical cord. (B) Ultrasound image at 18 weeks of pregnancy with PMD. The number of cystic, echo-free structures within the placental substance gradually increases. The red arrow points to the entrance of the umbilical cord into the placenta, and the affected area is located above the entrance of the umbilical cord into the placenta. PMD, placental mesenchymal dysplasia.

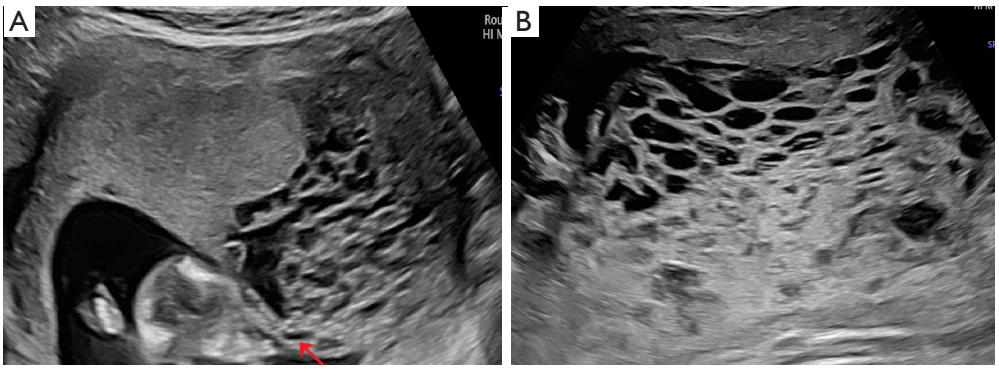

Figure 5 Case 2. (A) Ultrasound image at 20 weeks of pregnancy with PMD. The red arrow indicates the entrance of the umbilical cord into the placenta. The demarcation between the affected area and normal placental tissue is distinct, with the affected region situated at the entrance of the umbilical cord into the placenta. (B) Ultrasound image at 22 weeks of pregnancy with PMD. The placenta exhibits thickening, and the extent of cystic, echo-free structures gradually decreases. The affected area partially encompasses the entrance of the umbilical cord into the placenta. PMD, placental mesenchymal dysplasia.

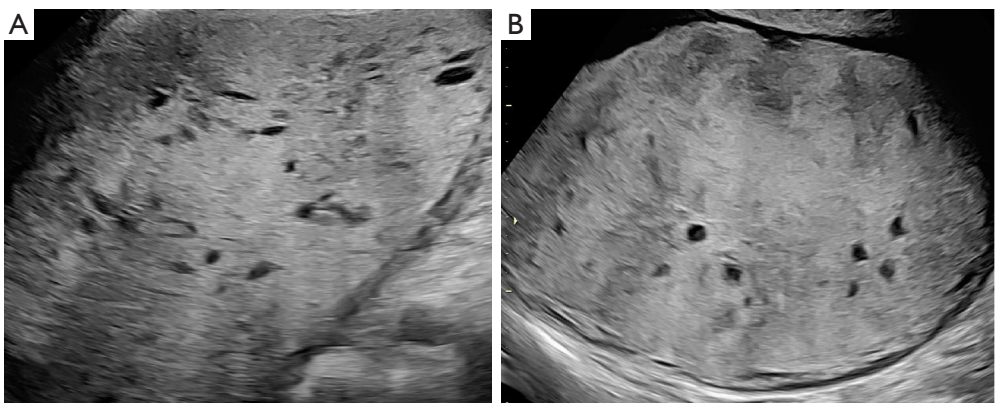

Figure 6 Case 2. Ultrasound image at 26 weeks of pregnancy with PMD. (A,B) The placenta exhibits diffuse thickening, with abnormal echoes present within. The affected area encompasses the entrance of the umbilical cord into the placenta. PMD, placental mesenchymal dysplasia.

enlarged and exhibited vesicular-like structures. Upon gross examination, no significant changes were observed in other placental villi (*Figure 7A*,*7B*). (II) The placenta demonstrated hypertrophy with incomplete damage, and an expansion of villi near the fetal surface margin was noted, resembling a thick-walled bubble structure (*Figure 7C*). (III) The maternal surface appeared rough and slightly brittle. An expanding area containing white sacs was observed, along with numerous vesicular structures near both the fetal surface and the middle region of the placenta. Furthermore, this section visibly displayed 3 umbilical blood vessels. The umbilical cord measured 18 cm in length with a 1.2-mm diameter (*Figure 7D*). (IV) Placental tissue slice microscopic examination results [hematoxylin and eosin (HE), 100× magnification] (*Figure 8A*,*8B*).

Discussion

The incidence of PMD, a rare placental disease that is distinct from hydatidiform pregnancy or pseudopartial hydatidiform mole, is approximately 0.02% (2). Placental enlargement or thickening, vesicular cystic changes in the affected area, enlarged cystic villi, and localized dilated and tortuous placental chorionic vessels and proximal villi, which are sometimes accompanied by thrombosis, are typical ultrasonographic manifestations. PMD is frequently

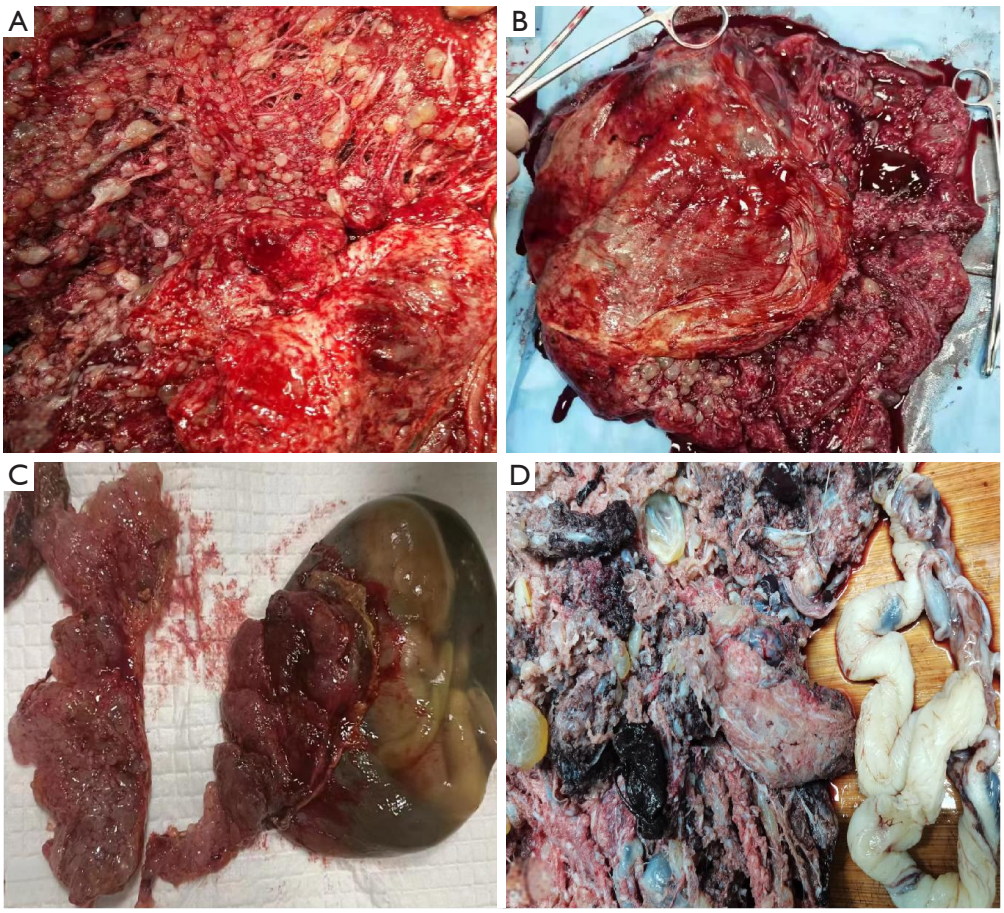

Figure 7 Case 2. PMD patients' gross specimen images of the placenta after induced abortion. (A,B) The placental surface is uneven with diffuse vesicular changes within the placental parenchyma. (C,D) Some local blood vessels of the placenta exhibit features of vascular tumor-like lesions, accompanied by intravascular thrombosis within some areas of the placental parenchyma.

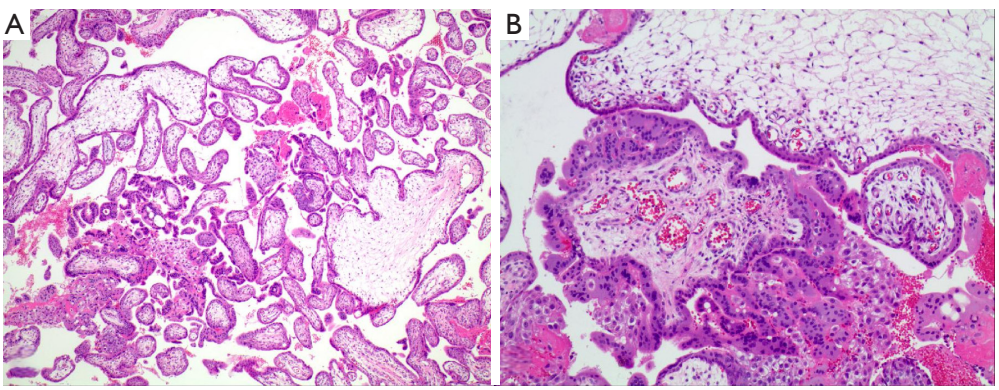

Figure 8 Case 2. Under the microscope, the results of the placental tissue slice (HE, ×100) are as follows: (A,B) the chorionic villi are notably expanded, forming cystic structures. Loose connective tissue and lacunar structures are observed, with normal villi seen in the surrounding periphery. HE, hematoxylin and eosin staining.

mistaken for hydatidiform mole, particularly partial hydatidiform mole (3). However, unlike hydatidiform mole pregnancies characterized by trophoblastic cell hyperplasia, PMD does not exhibit this feature. Although the ultrasonographic appearances of PMD and partial hydatidiform mole may be similar, their prognoses significantly differ. Most fetuses affected by PMD have normal structures and frequently reach full term, whereas those affected by partial hydatidiform mole frequently experience fetal demise or multiple fetal malformations, with very few reported cases of full-term normal pregnancies. Therefore, prenatal differential diagnosis of these 2 diseases is crucial. Prenatal ultrasonography is increasingly utilized for early pregnancy monitoring and early detection of placental vesicular lesions, serving as the primary means of diagnosing PMD. This study aimed to summarize the ultrasonic image characteristics of PMD at various stages of pregnancy, evaluate and analyze factors influencing PMD's pregnancy outcomes, and provide clinicians with a diagnostic foundation while assisting in appropriate treatment plan selection.

The ultrasonic manifestations of PMD are characterized by distinct features and follow a regular lesion progression, which are of significance for prenatal ultrasound diagnosis (4). In this study, we conducted a retrospective analysis of ultrasound images and clinical data from 15 patients with PMD. Prenatal ultrasonographic examination revealed multiple honeycomb-like cystic areas within the placental parenchyma, absence of tension in these cystic regions, an oblong distribution parallel to the long axis of the placenta, complete coverage extending to the inner cervix, and clear demarcation from the surrounding normal placental tissue. These findings align with the typical ultrasonic characteristics observed in cases of PMD (5). The ultrasound examination of PMD during early pregnancy frequently yields inconclusive results, revealing the presence of small cystic echoes within the placental parenchyma. In the second trimester, diffuse placental enlargement and thickening are observed, accompanied by dilation in both the villous and proximal villous vessels. The cystic portion of the placental parenchyma reaches its maximum dilation while maintaining a well-defined boundary with the surrounding normal tissues. As pregnancy progresses, the cystic components gradually diminish, frequently presenting localized umbilical hemangiomatous lesions accompanied by intravascular thrombosis during the third trimester. In some cases, umbilical vein tortuosity and dilation may also be observed (6).

The placenta, an essential organ that connects the fetus and the mother, plays a crucial role in fetal growth and development, as well as in the prognosis of newborns (7). PMD lesions that are extensive and close to or covering the placental umbilical cord entrance significantly impact placental function. This can result in inadequate blood and oxygen supply to the fetus, thereby leading to poor blood perfusion and ultimately causing intrauterine fetal hypoxia, which may lead to intrauterine death or stillbirth. Conversely, when the lesion is small and is located at the placental periphery away from the umbilical cord entrance, its impact on fetal growth and development is minimal, thereby resulting in a more favorable fetal prognosis. Previous studies have indicated a potential correlation between the location and extent of PMD lesions and fetal outcomes (8,9). In this study, 6 patients exhibited umbilical cord placental insertion within the affected area, thereby resulting in significant placental function impairment. Consequently, intrauterine hypoxia occurred, thereby leading to restricted fetal growth and development. Among these cases, 3 resulted in intrauterine demise, whereas 3 required surgical abortion. The affected area in the additional 5 patients was located at the placental edge, with normal placement of the umbilical cord insertion within the placenta. During the follow-up period, no significant abnormalities were observed in fetal growth and development. The diseased area gradually decreased after 30 W of pregnancy, thereby leading to full-term delivery of the fetus. The vital signs of all 5 newborns remained stable during the follow-up period. These findings suggest a correlation between PMD lesion location and adverse pregnancy outcomes, supporting conclusions drawn in previous studies.

Moreover, the extent of PMD lesions is a crucial factor influencing intrauterine fetal development and neonatal prognosis (10). Considerable variation was noted in the range of lesions observed in patients with PMD. Ultrasonographic measurements revealed that placental involvement accounted for 15–65% of the placental volume in 10 cases, whereas it exceeded 65% in 5 cases. Studies have indicated that larger lesion areas within the placenta have a greater impact on its function, potentially leading to IUGR or fetal death (11,12). Even cases with smaller lesion areas can result in adverse pregnancy outcomes, which are closely associated with lesion location. When the lesion predominantly affects the root of the umbilical blood duct (13), it significantly impairs fetal blood and oxygen supply, thereby causing chorionic blood vessel rupture and

intravascular thrombosis, ultimately resulting in inadequate perfusion and subsequent fetal demise or premature birth.

In this study, the lesions in 1 patient with PMD occupied 60% of the placental area. However, these lesions were mainly concentrated at the placental edge and did not significantly impact placental function or fetal blood and oxygen supply. Remarkably, a healthy fetus was delivered in a post-term pregnancy. Literature reports have indicated that approximately 20% of patients with PMD can be complicated with the Beckwith-Wiedemann syndrome (BWS) (14). Ultrasonographic manifestations of BWS include macrosomia, omphalocele, enlarged liver, large kidneys, excessive amniotic fluid, and other symptoms. BWS may be linked to chromosomal abnormalities. Therefore, during prenatal ultrasound examinations of patients with PMD, careful attention should be paid to whether the fetus is affected by BWS, as indicated by relevant abnormalities detected through ultrasonography. Fetal chromosomal examination is strongly recommended (15). If the fetal chromosomal karyotype is normal and no structural deformities or growth restrictions are noted, the fetus is less affected at this stage; pregnant women can consider continuing the pregnancy under these circumstances.

Ultrasound diagnosis of PMD remains an evolving area of research, and the choice of pregnancy outcomes depends on patients' awareness of the disease and their tolerance for the risks of continuing the pregnancy. The adverse consequences of PMD are mainly concentrated in the fetus (16), which should be considered when ultrasonography reveals multiple abnormal cystic lesions in the placental parenchyma and sub-chorionic angiomatous dilatation. Comprehending the ultrasonic image characteristics of PMD at every stage of pregnancy is crucial for precise disease diagnosis. Prenatal diagnosis in patients with PMD can confirm fetal chromosomal karyotype, closely monitor fetal growth and development through ultrasonography, and track the extent, location, and severity of PMD lesions. The integration of prenatal ultrasonography with clinical symptom assessment, fetal intrauterine development evaluation, and chromosomal karyotype analysis can facilitate prenatal consultations for patients and provide valuable insights for clinical treatment decisions, ultimately facilitating the prevention of unnecessary pregnancy terminations.

Conclusions

Prenatal ultrasound plays a crucial role in the early diagnosis and differential diagnosis of PMD. When ultrasound reveals cystic lesions in the placenta, it is important to consider that PMD has characteristic ultrasonographic features and a regular pattern of disease progression.

Acknowledgments

The authors would like to express their gratitude to the patients and their families who participated in this study. The authors would also like to thank the medical and research staff at Gansu Maternal and Child Health Hospital for their support and assistance in conducting this research.

Funding: This work was supported by the Science and Technology Program of Gansu Province (No. 23JRRA1383).

Footnote

Reporting Checklist: The authors have completed the AME Case Series reporting checklist. Available at https://qims.amegroups.com/article/view/10.21037/qims-23-1548/rc

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://qims.amegroups.com/article/view/10.21037/qims-23-1548/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. This study is a retrospective investigation. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013) and was granted an exemption from the Medical Ethics Committee of Gansu Provincial Maternity and Child-care Hospital. The participants provided their written informed consent to publish their cases (including publication of images). A copy of the written consent is available for review by the editorial office of this journal.

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Cite this article as: Wei CM, Li TG, Ma B, Xu XY. Evaluation and analysis of influencing factors of placental mesenchymal dysplasia diagnosed using prenatal ultrasonography and pregnancy outcomes: case series. Quant Imaging Med Surg 2024;14(9):6934-6944. doi: 10.21037/qims-23-1548

