Case Series of Placental Mesenchymal Dysplasia with Live Birth

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Abstract: Objective: 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. The aim is to report the clinical and pathologic characteristics of three cases of placental mesenchymal dysplasia (PMD). Method: Pathologic and clinical data were retrieved from the electronic medical records. The data were reviewed and summarized by the same pathologist. Results: Maternal age was respective 36 years, 31 years and 33 years. Cesarean section was performed at 39 weeks, 37 weeks and 38 weeks, respectively. The newborns were born with birth weight of 1790, 2370 and 3150 grams, respectively, with placenta weight of 590, 579 and 510 grams respectively. Placenta for pathological examination was performed macroscopically thick-walled vessels. Microscopic observation showed that the part of the stem villus was sac-like dilated, the blood vessels were thickened in the villi, the blood vessel wall was thickened, the villus interstitial was loose, and the mucus was changed. Part of the villus edema and degeneration, no trophoblastic hyperplasia, the final villus structure is normal. Conclusion: The size of the lesions in the Placental mesenchymal dysplasia and the location of the lesion is closely related to the birth weight of the newborn. Detailed histology is necessary for diagnosis and differential diagnosis.

Keywords: Placental Developmental Abnormality, Placental Disease, Vascular Disease, Hydatidiform Mole

1. Introduction

Placental mesenchymal dysplasia (PMD) is a relatively rare placental lesion, which is often difficult to identify with vesicular blocks due to the lack of awareness of the disease by clinicians. The present study aims to organize and collect the recent two cases of newborn babies in live births, review the clinical and pathological data, and improve the understanding of the disease by clinical and pathologists through previous literature and clinical-pathological analysis.

2. Clinical Data and Methods

The data of patients with PDM in this hospital were reported. First women were 36 years old, pregnant 2 was born 1, and Down's screening was high risk. Pre-pregnancy check: herpes simplex virus 1+2 antibody IgG>200.000RU/ml, rubella virus antibody IgG 101.000IU/ml; cytomegalovirus antibody IgG 55.400RU/ml; folic acid 54.31nmol/L in the middle and late pregnancy; IgG resistance B potency 128 titer. In the third trimester of pregnancy, the placental parenchyma was uneven, and there were multiple small anechoic areas scattered in the field, which showed a honeycomb change. The patient was not further examined. At 39 weeks of gestation, she was admitted to the hospital due to vaginal bloody secretions. After admission, the ultrasound showed the dark area behind the placenta, and she was given a miscarriage treatment. Subsequently, the fetal membrane spontaneously ruptured, because the pregnant woman has a history of cesarean section, scar uterus, accompanied by cesarean section. A baby girl was born, and the appearance of the newborn was normal. The birth weight was 1790 g, and the Apgar score was 9 at 1 minute and 9 at 5 minutes. Neonatal
examination: blood routine: total white blood cell count
25.35×10^9/L, neutrophil absolute value 17.72×10^9/L, red
blood cell count 4.18×10^12/L; hemoglobin concentration
118g/L, total platelet count 60×10^12/L; biochemical liver
function: total bilirubin 162.5 umol/L, total protein 48.5g/L,
albumin 23.9g/L; C-reactive protein 10.09mg/L; postpartum
placenta routine pathological examination. Clinical diagnosis:
scar uterus, intrauterine infection, premature infants, low birth
weight infants, neonatal anemia, jaundice, hypoproteinemia.
The newborn gave active treatment and the condition
improved and was discharged from the hospital a week later.

Placental pathology, General inspection: Placenta
hypertrophy with size 18cm × 16cm × 4cm, weight 590g. The
fetal membrane was slightly grayish-yellow, umbilical cord
length 14cm, diameter 0.9cm with three umbilical vessels,
local blood clots on the placenta of the placenta, see the gray
saclike expansion area, similar to thick-walled blister-like
structures.

Case 2, pregnant women 31 years old, pregnancy 3
production 1, pre-pregnancy examination: early nausea and
vomiting, and other obvious early pregnancy reaction. Pregnancy
registration, regular checkups, thalassemia negative, normal
sugar screening. Down's screening, prenatal non-invasive
 genetic testing suggest low-risk pregnancy. Pregnancy 24+2
weeks of grade III color ultrasound: intrauterine single live
birth, equivalent to 23 weeks and 1 day, fetal head
circumference, femur, tibia measurement is located under the
M-2SD line, fetal weight is lower than the tenth percentile.
Prenatal diagnosis is recommended for chromosome analysis,
but the woman refused further evaluation. Subsequent ultrasound
showed that the gestational age of the fetus was
less than 1 week of menopause. At the age of 33+2 weeks, the
grade III ultrasound: intrauterine single-lived fetus, placenta
grade II, fetal head circumference, abdominal circumference,
femur length, and tibia length are all located under the M-2SD
line, and the fetal weight is lower than the tenth percentile.
Fetal intrauterine growth restriction was considered. The
placenta is obviously thickened, the internal echo is uneven,
and it is honeycomb-shaped. At 37 weeks of gestation, the
patient was admitted to the hospital; the cesarean section was
performed, and a baby girl was born. The newborn weight was
2370g. Postpartum placental pathology was conducted.
Clinical diagnosis: fetal growth restriction,
small-for-gestation age, premature infants, scar uterus.

Pathological examination of placenta Gross examination:
broken, 24×18×9cm, thickness 4-5.5cm, weight 579g,
umbilical cord length 1cm, diameter 1.5cm, three umbilical
vessels. The membrane of the membrane is gray, and there are
a lot of vesicular structures in the fetal surface and the middle
part of the placenta. The other placental villi have no special
changes in the naked eye. The maternal surface is rough and
the texture is slightly brittle.

Case 3, pregnant women 33 years old, pregnancy 4
production 1, pre-pregnancy examination: early nausea and
vomiting and other early pregnancy reactions, lasting for 1
month, no history of exposure to toxic radiation. Pregnancy 4+
month consciously fetal movement, activity so far, pregnancy
during the construction of the book, regular checkup,
thalassemia, Down's screening, OGTT low risk, smooth
pregnancy, no complaints discomfort, patients without
dizziness, double lower limb edema. At 38 weeks of this
pregnancy, vaginal fluid discharge 7 hours before, because the
scarred uterus, the infant was delivered through cesarean
section, a baby girl was born, and the newborn weight was
3150g. Clinical diagnosis: scar uterus; premature rupture of
membranes.

Placental pathological examination: the placenta size is
17×17×3.5cm, the weight is 510g, the membrane was slightly
grayish-yellow, the damage was not complete, the umbilical
cord was 14cm long, the diameter was 1.4cm, the cut surface
was separate in three, and the part of the maternal surface near
the marginal area was expanded. It has a blister-like structure
with a range of 12×7 cm.

![Representative pictures of placental mesenchymal dysplasia (PDM). A, stem villi are saclike, loose connective tissue, normal villi around, no proliferation of trophoblastic cells; B, thick-walled vessels with cystic dilatation; C, interstitial edema of villi. 100×.](image)

3. Results

Case 1 under the microscope: visible lesions of the stem
villus saclike expansion, stem villus blood vessels thickened,
thickened blood vessel wall, see thick wall small blood vessels,
villus interstitial loose, mucinous changes, partial villous
edema degeneration, no nourishment leaves The cells
proliferated and the terminal villus was normal. Lymphocyte
aggregation infiltration was observed in the fetal membrane.

Pathological diagnosis: mesenchymal dysplasia (PMD); stage
II of fetal membrane inflammation. Case 2: Microscopic
edema of placental villi with mucous-like interstitial, dilated
thick-walled blood vessels, partial villi of different sizes,
partial villous edema, partial villous interstitial cell
proliferation, partial placental villus structure, fetal membrane,
and the umbilical cord was slightly edematous. The
pathological diagnosis was: dysplasia of the placenta. In the
third case, the part of the placenta under the microscope had
large edema of the villus, accompanied by mucus-like interstitial, and the expanded thick-walled blood vessels were seen individually, and most of the villi were normal. Immune cells infiltrated in the fetal membrane, but not in the umbilical cord. Pathological diagnosis: dysplasia of the placenta.

4. 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. According to the literature, the incidence of PDM is reported at about 0.002% to 0.02%, or even lower [1, 2]. The incidence of PDM is difficult to determine because clinical and pathological understanding is inadequate, which may result in overall underestimation and underreporting. On the other hand, it is currently only detected when the placenta is infected or complicated [3, 4]. The incidence of PDM may increase as the pathologist's clinical awareness increases [5, 6]. At present, the pathogenesis of PDM is still unclear. Chen CE [7] and others believe that the proliferation of stem choriocarcinoma mesenchymal cells is accompanied by hyperplasia of mesenchymal cells, and there is a normal villus mesoderm connective tissue in the expanded stem villi. The presence of eosinophilic mucopolysaccharide, presumably congenital mesoderm malformation. Some patients with Beckwith-Wiedemann syndrome are overgrowth presumably congenital mesoderm malformation. Some patients with Beckwith-Wiedemann syndrome are overgrowth syndrome, also known as an umbilical bulge-megaglobe-macro-complex syndrome. There are no consistent clinical diagnostic criteria for this disease, which is considered to be the maternal source or parent of the chromosome 11p15.5 region, which may affect the expression of IGF-2 and Xp22.31, which is expressed as vascular endothelial growth factor D (VEGF-D) [8]. Deletion of expression of the source imprinted gene [9]. The karyotype of fetal chromosomal examination in PDM patients is normal, and a small number of patients are accompanied by chromosomal abnormalities, including 13-trisomy, triploid and 13q12.11 involving the GJB6 gene [10, 11].

PDM has no clinical special symptoms in early pregnancy. It can be diagnosed by routine prenatal examination or ultrasound. Some of the serum βHCG is normal or slightly elevated, and serum α-blood protein is elevated because of PDM surface transformation. The increase in area, as a result of an increased volume of stem villus and increased number of the placenta, is thought to be an increase in alpha-fetoprotein transferred to the maternal circulation [12]. In the third trimester, fetal growth restriction or fetal death may occur, and some amniotic fluid may occur. It may be caused by difficulty in swallowing the fetus with Beckwith-Wiedemann syndrome. In patients with PDM, intrauterine growth restriction during pregnancy, intrauterine stillbirth, premature delivery, severe fetal growth restriction may be due to poor perfusion of the placental stem villus vascular thrombosis and hypoxia or fetal angiogenesis. The complications of the fetus are mainly anemia, thrombocytopenia or microangiopathic hemolysis, which may be related to the abnormal distribution of blood vessels. Its etiology and pathogenesis are unclear and may be related to abnormal development of X chromosome and trophoblastic cells [13]. The current prevailing view is that the adverse consequences of PDM are mainly concentrated on the fetus, while the influence of PDM on the mother is mainly manifested as eclampsia and pregnancy-induced hypertension. This study found that one of the pregnant women, the lesion in the marginal zone, the fetus was 3150g, the fetal weight was normal. It suggests that the lesions are small in scope and concentrated in the marginal area of the placenta, with little or no effect on the fetus. Another pregnant woman's lesions were concentrated in the middle of the placenta and the placenta fetal area. The neonatal weight was 2370g, suggesting that the placental dysplasia may be related to the site of placental lesions. The lesions are concentrated in the center of the placenta, affecting the degree of fetal growth and development.

PMD ultrasonography mainly showed cystic anechoic zone; before 13 weeks of gestation, patients with early ultrasound examination were not obvious, and vesicular lesions could not be detected. As the pregnancy progresses, the congested blood vessels resemble a black-red or grayish-white scorpion-like structure that can be identified in the placental parenchyma, mainly in the place where the chorionic plate is close to the fetal surface, consistent with the placental gross examination. Subsequent ultrasound showed similar similarity to some of the blister-like blocks. The imaging findings of PDM 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 and the edema of the missed abortion. In this paper, the aneurysm-like dilatation of the placental villus is localized, the local villus is cystic, or accompanied by the formation of mucin-like interstitial and mucus pools, and the terminal villous structure around the other parts is normal, and the nourishing leaves lacking partial vesicular blocks Cell proliferation characteristics [14].

There are few reports on the effect of the virus on the placenta. Rubella virus, cytomegalovirus, and herpes simplex virus are a group of pathogens that cause intrauterine infection in pregnant women. Studies have shown that TORCH infection can inhibit the proliferation of human placental trophoblast cells and arrest human placental trophoblast cells in the G1 phase to induce apoptosis of human placental trophoblast cells, which may be related to the inhibition of Cdc-6 protein expression [15]. The study of folic acid is mainly about the lack of effects on the fetus, as well as the side effects of excessive folic acid intake, which can mask the early symptoms of vitamin 12 deficiency. Vitamin 12 deficiency can cause damage to the nervous system, interfere with antiepileptic effects and induce convulsions, and also affect zinc absorption. There is no relevant research on the influence of the placenta, and further understanding is needed.

In this case, one of the pregnant women was a senior pregnant woman. The literature review reported that 23-42 years old were reported, and there were more reports in 20-30 years old. It is currently considered that there is no significant
age-related relationship between PDM and age. There was no report on the number of births and the number of maternal births. There was no correlation between the study and the number of maternal births. There may be fewer cases and further research is needed.

Placental mesenchymal placental size of the placental lesions may be an important factor affecting the fetus or infant. However, its pathogenesis and biological basis need further study. Detailed histology is essential for diagnosis and differential diagnosis. Although PMD often exhibits perinatal adverse outcomes, it may be related to the extent of placental dysplasia. With the advancement of ultrasound technology, prenatal identification of these cases is very important to reduce fetal morbidity and mortality. The prognosis is better than a partial hydatidiform mole. Once the PMD is suspected, the fetal karyotype is normal. Through prenatal screening and patient counseling, strict B-ultrasound detection of fetal growth and development, continued pregnancy is acceptable, thus avoiding unnecessary abortion.

5. Conclusion

The size of the lesions in the Placental mesenchymal dysplasia and the location of the lesion is closely related to the birth weight of the newborn. Detailed histology is necessary for diagnosis and differential diagnosis.

References


