Placental Mesenchymal Dysplasia

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- Placental mesenchymal dysplasia is a rare placental lesion characterized by stem villous cystic dilation and vesicle formation, placentomegaly, and vascular abnormalities. It can be associated with growth restriction, stillbirth, Beckwith-Wiedemann syndrome, and some chromosomal abnormalities, and needs to be distinguished from its main differential diagnosis, hydatidiform mole.


Placental mesenchymal dysplasia (PMD) is a rare and fairly new diagnosis first termed in 1991. The term placental mesenchymal dysplasia was chosen in 1991 by Moscoso et al. to characterize a placental lesion previously reported in the literature as "placentomegaly with massive hydrops of placental stem villi and pseudopartial mole." Placental mesenchymal dysplasia is an unusual abnormality of the stem villi of the placenta that may be mistaken for a hydatidiform mole, and in particular, male mole, owing to the mixture of cysts and normal-appearing parenchyma. The stem (anchoring) villi form as outgrowths of the chorionic plate early in placentogenesis and give rise to the branching villous trees (Figure 1). A more detailed description of placental development is available. Placental mesenchymal dysplasia shows stem villous cystic dilation and vesicle formation, placentomegaly, and vascular abnormalities.

It is postulated that PMD occurs when 1 ovum is fertilized by 2 spermatozoa or involves a single ovum and spermatozoa with a maternal nondisjunction error during the first meiotic division. The former produces a mix of diploid and aneuploid cells, and the latter, androgenic meiotic division. The former produces a mix of diploid and triploid cells, while the latter produces a mix of diploid and tetraploid cells.

The most likely entity to be confused with PMD is a partial mole, characterized by stem villous cystic dilation and vesicle formation without trophoblast proliferation. Like PMD, partial moles are characterized by stem villous edema and cystic dilation, but absence of trophoblastic proliferation in the abnormal villi, which shows positive staining of normal villous stroma. Staining for desmin and vimentin is retained in PMD, similarly to normal villous stroma.

The main differential diagnosis includes molar pregnancy. The most likely entity to be confused with PMD is a partial mole; however, a twin pregnancy with 1 normal fetus and 1 complete mole could also mimic PMD. Aside from the clinical distinctions discussed above, histologically, molar pregnancy has hydropic terminal endomesenchyme, with trophoblastic proliferation, which PMD lacks.

PMD shows enlarged edematous stem villi with dilated vessels and absence of trophoblastic proliferation (Figures 3 and 4). Cisterns may be seen within stem villi. The vessels are thick walled, with fibromuscular hyperplasia. Terminal villi are usually normal. The edematous stem villous stroma stains for Alcian blue, but is negative for smooth muscle actin, which shows positive staining of normal villous stroma. Staining for desmin and vimentin is retained in PMD, similarly to normal villous stroma.

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Cases of PMD have shown villous trophoblast staining with p57 but absence of stromal nuclear staining in the abnormal villi, although this finding is inconsistent.

Partial moles, the more common differential diagnosis, are triploid, unlike the usually diploid PMD, and show scalloped villi and trophoblastic inclusions not present in PMD. Partial moles stain for p57 like in a normal pregnancy, with staining of villous trophoblast and nuclear villous stromal staining. Other mimics included in the differential diagnosis with PMD are placental cysts. Placental cysts are hypoechoic and can be located within the placental tissue or under the chorionic plate with proposed origin from the trophoblastic X cell. However, it has been postulated that placental cysts represent massive stem villous hydrops with cisternal formation without trophoblastic proliferation. Like PMD,
these X-cell cysts have been described in association with Beckwith-Wiedemann syndrome. Histologically, placental cysts are cystic spaces containing proteinaceous eosinophilic material. Grossly, PMD may be thought to be chorangiomas, usually more well-circumscribed vascular capillary hemangiomatous placental lesions, which may exhibit a feeder vessel. Multiple chorangiomas in particular may mimic PMD. In addition, subchorionic hemorrhage may mimic PMD, owing to the cystic spaces seen on ultrasonography.10–15

In conclusion, PMD is a relatively rare entity that needs to be distinguished from molar pregnancy to clinically prevent unnecessary termination of the pregnancy.9 If PMD is suspected after antenatal ultrasonography and genetic testing, it is imperative that affected patients be followed up as patients with high-risk pregnancies owing to the association with growth restriction, stillbirth, and other chromosomal abnormalities. The pathologic features in the placenta need to be recognized and distinguished from other placental abnormalities, including molar pregnancy, to aid in clinical decision making with the patient as to future pregnancies and pregnancy outcomes.

References


