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PLACENTAL MESENCHYMAL DYSPLASIA- A CASE REPORT

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ABSTRACT
Placental mesenchymal Dysplasia is a rare benign placental vascular anomaly, characterized by placentomegaly and grape like vesicles resembling molar pregnancy on Ultrasonography with heterogenous solid and cystic areas. Colour Doppler shows increased blood flow due to aneurysmal dilatation od chorionic arteries and veins. Incidence of PMD is approximately 0.02%. In PMD, there is slight increase in B-HCG and Alpha Feto protein is elevated. In present case, a 36 year old female underwent USG at 16 weeks gestation showing large cystic and solid areas in placenta, with single live fetus. USG diagnosis of PMD was made. Termination of pregnancy was done and placenta was sent for histopathological examination which showed admixture of normal and abnormal appearing chorionic villi, absence of trophoblastic proliferation, favouring diagnosis of PMD.

KEYWORDS: PMD, placentomegaly, grape like vesicles.

INTRODUCTION
Placental Mesenchymal Dysplasia is a rare benign placental vascular anomaly. It is characterized by placentomegaly and grape like vesicles resembling molar pregnancy on Ultra sonography. Incidence of PMD is 0.02%[1] with female preponderance.[2] It is important to distinguish PMD from molar pregnancy in order to prevent unnecessary termination of pregnancy. In PMD fetus is normal but has high incidence of fetal growth retardation (FGR) and intrauterine fetal death (IUFD) and is associated with Beckwith-Wiedemann syndrome (BWS).[2] PMD placentas are large showing edema of stem villi with intact terminal villi and vascular anomalies such as cirsoid chorionic vessels, thrombosis, increased thickness of vessel wall, vascular stenosis, villous chorangiosis, chorioangioma, fetal thrombotic vasculopathy. Abnormal umbilical cords, tortuous, twisted long cords lead to FGR and IUFD.[2,3] In PMD, there is absence of trophoblastic proliferation differentiating it from partial moles.[4]

In the present case reported, a 36 year old female with intrauterine live fetus without any obvious anomaly at 16 weeks gestation was diagnosed on Ultrasonography as PMD because of presence of solid and cystic areas and increased vascular flow in cystic areas. As there is increased incidence of FGR and IUFD, termination of pregnancy was done and placenta subjected for histopathological examination. Histopathological diagnosis of PMD was given.

CASE REPORT
A 36 year old female underwent USG at our centre at 16 weeks gestation. At USG, a single live fetus with no obvious anomalies and 16 weeks gestational age was reported. Placenta was in anterior, upper segment, mild bulky with multiple small cystic like spaces. Provisional diagnosis of Placental Mesenchymal Dysplasia was made on USG. Serum B-HCG level was slightly raised and Alpha Feto Protein level was raised. After counselling the patient, pregnancy was terminated and placenta sent for histopathological examination. Grossly, placenta showed cystic and solid areas. On Cut section, cysts were filled with blood. Histological examination of placental tissue showed admixture of normal and abnormal appearing chorionic villi at low power (Fig 1). Few villi were enlarged and showed edematous stroma with cistern formation and thick walled and tortuous blood vessels within the villi. There were increased capillaries in villi, absence of trophoblastic proliferation (Fig 2), favouring a diagnosis of Placental Mesenchymal Dysplasia.

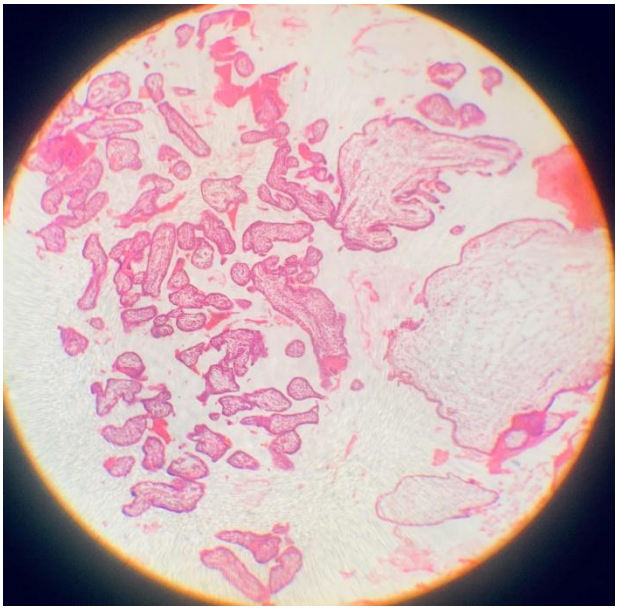

Fig 1. H&E stain, Section shows normal and abnormal appearing chorionic villi at low power.

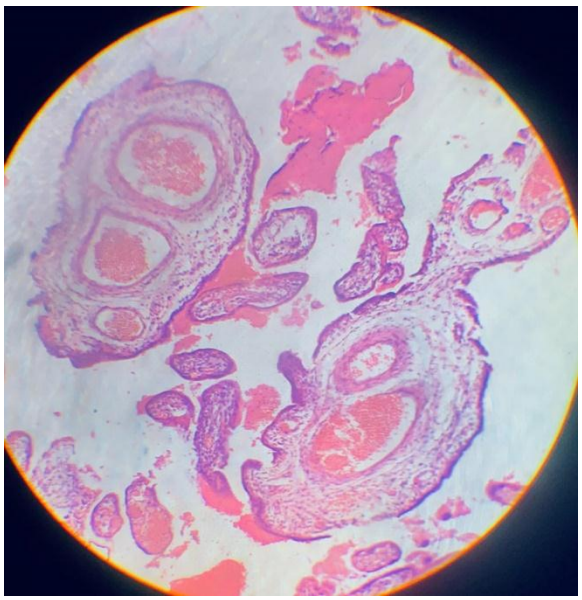

Fig 2. H &E stain, Section shows enlarged and edematous stroma with cistern formation and thick walled and tortuous blood vessels within the villi, absence of trophoblastic proliferation.

DISCUSSION

Placental Mesenchymal Dysplasia is a rare disease entity, initially described by Moscoso et al. in 1991 as placentomegaly resembling image of partial hydatidiform mole with elevated level of AFP.[4] Incidence of PMD is approximately 0.02%.[1] 50% cases show placentomegaly while 80% show cystic placenta.[5] The main differential diagnosis of PMD are partial hydatidiform mole, dichorionic twins of a normal fetus and complete mole, confined placental mosaicism. Human Chorioinic Gonadotropin is useful in differential diagnosis. Abnormally high level of B-HCG indicates molar disease. AFP is elevated in PMD with slight increase in B-HCG. There is possible genetic basis for the placental abnormality in PMD. 23% PMD cases are associated with paternal isodisomy of the 11p15.5 region. BWS is characterized by macrosomia, exomphalos, macroglossia, omphalocele, internal visceromegaly, placentomegaly and increased childhood tumors. There are reports describing coincidence of PMD with fetal hepatic mesenchymal tumors, probably sharing common pathogenetic origin.[6,7]

On USG, Placenta of complete molar pregnancy with co twin and partial molar pregnancy look heterogenous with partially solid and cystic areas.[6,8] Histopathologically, trophoblast proliferation, is seen in molar pregnancy but not in PMD. Partial molar pregnancy is accompanied by an abnormal triploid fetus.

On colour Doppler, in first trimester in PMD, placenta does not show blood flow in cystic spaces whereas in

third trimester, large vascular areas with turbulent blood flow are seen (either arterial or venous), located mainly under and at the level of chorionic plate, and due to progressive dilatation of chorionic arteries and vein become aneurysmal.[9] Diagnosis of PMD is confirmed after evaluation of placental pathology.

Grossly it is characterized by placentomegaly, dilated or aneurysmal chorionic vessels and fibromuscular hyperplasia or cystic villi.[8] As pregnancy advances, tangled congested vessels grossly resemble grey-white or dark-red worm like structures within the parenchyma and are prominent in subchorioinc plate near fetal surface.

Microscopically mesenchymal hyperplasia and edema of stem cell villi containing thick walled vessels are seen. There is characteristic absence of trophoblastic hyperplasia differentiating Placental Mesenchymal Dysplasia from Gestational Trophoblastic Disease.

IHC tests using antibodies against P57 K1P2 protein (expressed in maternal genome) is helpful in distinguishing PMD from molar pregnancy.[10]

The cause of intrauterine fetal death (IUFD) may be due to thrombosis of chorionic vessels and umbilical cord anomalies. Chronic hypoxia due to obstruction of fetal vascular thrombosis and decrease in maternal-fetal gas exchange due to insufficient amount of chorionic villi.[2]

CONCLUSION

PMD should be considered in differential diagnosis of sonographic diagnosis of cystic lesions of the palcenta esp. with phenotypically normal appearing fetus. As there is increased incidence of IUFD, accurate monitoring with serial growth scans, S. B-HCG, S. AFP levels and third trimester assessment of well being is required. USG appearance suggestive of molar pregnancy alongwith increase maternal serum AFP and normal or slightly increased B-HCG level can suggest the diagnosis of PMD.

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