Original Research Paper

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MASSIVE PERIVILLIOUS FIBRIN DEPOSITS ON PLACENTA PRESENTING AS INTRAUTERINE GROWTH RESTRICTION IN A WOMAN WITH BAD OBSTETRIC HISTORY.

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ABSTRACT **Introduction:** Massive perivillous fibrinoid, otherwise known as massive perivillous fibrin deposition or maternal floor infarction, is a condition characterised by enmeshment of chorionic villi in a fibrinoid material and in placental insufficiency of varying degrees. The consequences include a significant risk of intrauterine growth restriction, intrauterine death and preterm delivery. **Case Report:** 25-year-old, G2P0100, came to at 8 weeks of gestation, with history of previous pregnancy complicated by MPVFD of the placenta, resulting in third-trimester intrauterine fetal death. She was started on low molecular weight heparin 40IU and aspirin 150 mg daily. Ultrasounds were done timely which realveled MPVFD and early onset growth restriction, which was managed with timely doppler studies. The patient received two doses of betamethasone at 34 weeks, and she delivered a live male baby weighing 2100 grams via cesarean section for non reassuring fetal heart rate. Placenta was grossly bulky and was send for histopathology examination. Placental histopathology showed increased intervillous fibrin deposits, calcifications and multiple thrombii. These findings are consistent with diagnosis of recurrent MPVFD. **Discussion:** The amount of fibrin in placenta determines pregnancy outcome. It is associated with higher incidence of prematurity, small for gestational age and intrauterine fetal death compared to moderate or low MPVFD. **Conclusion:** It is very important to keep suspicion of placental causes in pregnancies presenting with intra uterine growth restriction and antepartum haemorrhage. Timely intervention and simple management may improve the maternal and fetal outcome in cases of massive perivillous fibrin deposits on placenta.

KEYWORDS : MPVFD (massive perivillous fibrin deposition), intrauterine growth restriction, placental infarction.

INTRODUCTION:

Massive perivillous fibrinoid, otherwise known as massive perivillous fibrin deposition or maternal floor infarction, is a condition characterised by enmeshment of chorionic villi in a fibrinoid material and in placental insufficiency of varying degrees. The consequences include a significant risk of intrauterine growth restriction, intrauterine death and preterm delivery. There is a high risk of recurrence. Massive perivillous fibrin deposition (MPFD) and maternal floor infarction are closely related placental lesions characterized by the extensive deposition of fibrin or fibrinoid material in the intervillous space, extending from the basal chorionic plate to subchorionic placental area [[1], [2], [3]]. The condition is thought to be rare, occurring in approximately 0.1% of deliveries. In a recent prospective study evaluating the incidence of MPVFD in pregnancies with intrauterine fetal growth restriction (IUGR), fibrin deposition exceeding 25% was reported in 11.8% of the placentas, which is higher than previously reported [[4], [5], [6]].

Case Report: A 25-year-old woman, G2P0100, came to us at 8 weeks of gestation, with history of previous pregnancy complicated by MPVFD of the placenta, resulting in third-trimester intrauterine fetal death.

In the patient's first pregnancy, her prenatal course was largely unremarkable. She was booked at Primary health center. Past medical and surgical history was insignificant. Her initial prenatal body mass index (BMI) of 24. She had her level 1 and level 2 scans done which were told to be normal. The patient has sudden onset of labor pain at 34 weeks of gestation, she self transferred to a tertiary care unit. On admission she was diagnosed with an IUFD and had an uncomplicated spontaneous vaginal delivery. On autopsy, the fetal exam was notable for maceration and organs small for gestational age. There were no signs of intrauterine infection. However, placental pathology was notable for MPVFD covering 75% of the placenta. APLS work-up including anticardiolipin antibodies, anti-beta-2-glycoprotein antibodies, and lupus anticoagulant testing was negative. Infectious testing for syphilis, hepatitis B, HIV and hepatitis C were negative, as were the patient's, hemoglobin A1c (HbA1c), thyroid function testing (TFT), and tests evaluating for preeclampsia including a complete blood count, complete metabolic panel, and a protein to creatinine ratio. TORCH profile was positive for herpes simplex virus IgG, hinting a previous infection. She was counselled for early booking and proper treatment at tertiary care center in her subsequent pregnancy

One year later when she concieved again, the patient had patient was started on low molecular weight heparin 40IU and aspirin 150 mg daily. Her level 1 scan was normal, level 2 scan showed heterogeneous iso to hyperechoic striated pattern fusiform shaped avascular structures along the fetal surface of placenta giving appearance of jungle vines as shown in the figure no 1 Early onset Intrauterine growth restriction was evident, and patient was managed accordingly with timely doppler studies. The patient received two doses of betamethasone at 34 weeks, and she was admitted for expectant management.

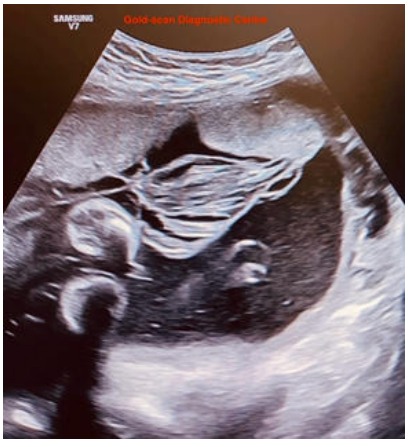

Figure No. 1 USG showing MPVFD "Jungle vines sign".

She spontaneously went into labour. Intrapartum her cardiac tocogram was non reassuring. Patient delivered a live male baby weighing 2100 grams via cesarean section for recurrent decelerations. Apgar scores were 8 and 9 at 1 and 5 min respectively, baby was admitted in neonatal intensive care unit. Placenta was grossly bulky and was send for histopathology examination. Placental histopathology showed increased intervillous fibrin deposits, calcifications and multiple thrombii. These findings are consistent with diagnosis of recurrent MPVFD.

Postpartum period was uneventful. Baby was discharged on day 4 of lufe and mother was discharged on 5th postoperative day. She was educated regarding contraception.

DISCUSSION:

The term massive perivillous fibrinoid or massive perivillous fibrin deposition on placenta is self explanatory, the pathological condition which is characterised by heavy deposition of fibrin in the placenta and distorting the chorionic villii. Synonymously also referred to as maternal floor infarction, which is misleading as there is no infarction nor ischemia. The amount of fibrin in placenta determines pregnancy outcome. It is associated with higher incidence of prematurity, small for gestational age and intrauterine fetal death compared to moderate or low MPVFD.

CONCLUSION:

It is very important to keep suspicion of placental causes in pregnancies presenting with intra uterine growth restriction and antepartum haemorrhage. Timely intervention and simple management may improve the maternal and fetal outcome in cases of massive perivillous fibrin deposits on placenta.

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