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CASE REPORT

Massive Perivillous Fibrinoid Degeneration of Placenta/ Maternal Floor Infarct: A Case Report

Veena Mangesh Panat[1]

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Abstract Placental pathology can be a cause of early severe FGR leading to perinatal morbidity and mortality with repercussions in future pregnancies. A diagnosis of a placental lesion on ultrasound should have a detailed clinical and histopathological correlation for better management in next pregnancy. We present a case report of " placental massive perivillous fibrinoid degeneration/maternal floor infarction (MPFD)" with very large avascular placenta. This can lead to fetal growth restriction (FGR) and other complications for the fetus. It is also known to be associated with antiphospholipid syndrome. Management with low dose aspirin and low molecular weight heparin achieves good results.

Keywords Placenta · Massive perivillous fibrinoid degeneration (MPFD) · Maternal floor infarction (MFI) · Fetal growth restriction (FGR) · Anti phospholipid antibody (APLA) · Low dose aspirin · Low molecular weight heparin (LMWH)

Introduction

Massive perivillous fibrnoid degeration/maternal floor infarct (MFI) is a relatively rare condition characterised by severe early fetal growth restriction (FGR) or intrauterine fetal demise (IUFD) with features of uteroplacental insufficiency (1). It was first described by Benirschke in 1961 (2). It is associated with high recurrence rate and major perinatal morbidity. Pathological characteristics include massive and diffuse fibrin deposition along the decidual basalis and perivillous space of the basal plate of the placenta (1). In MPFD, fibrin deposition is so increased (more than 25 % of the placenta) that it obstructs maternal flow to major portions of the placenta with grave fetal consequences.(3). The placenta has also been described as the " black box" of pregnancy (4) and a study of the placenta is important to manage the current pregnancy, and in the future.

Case Report

A young primi from an urban, educated high socio—economic background presented for the first time at 18 weeks for an anomalies scan; no earlier scans had been done.

Fetal biometry showed gestational age as 21 weeks from BPD and HC but the AC and FL were less than the 3rd percentile and so were all long bones. There was no gross structural anomaly in the fetus. The umbilical cord showed a 2 vessel cord. The finding apart from the early severe FGR was a markedly large placenta (Fig. 1), with a homogenous ground glass appearance as shown in the Fig. 2. On color flow, the placenta appeared devoid of any vascularity (as in Fig. 3). The patient was counseled about early severe FGR and "short" long bones and she was offered amniocentesis.

The amniocentesis was uneventful.

The patient came again after 15 h with an inevitable abortion with fetal parts in the vagina. The patient was informed and admitted. The miscarriage was uneventful. The placenta was examined grossly and showed fibrous changes over a large part. Placental tissue was sent for histopathological examination which showed changes of perivillous fibrinoid degeneration

✉ Veena Mangesh Panat
veenamoghepanat@gmail.com

[1] Panat Hospital, Aurangabad, Maharashtra, India

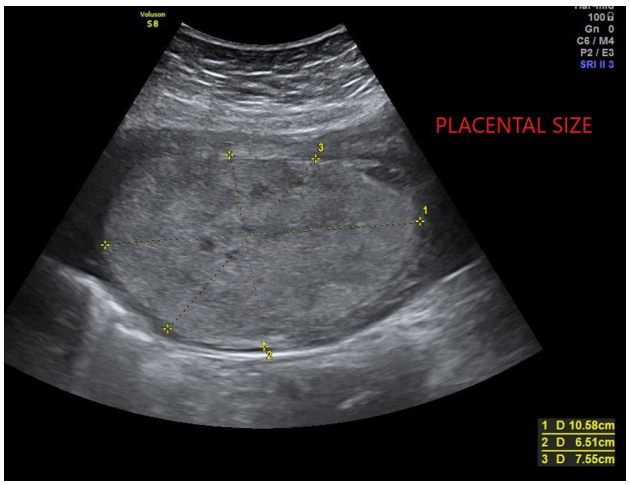

Fig. 1 Huge size of placenta

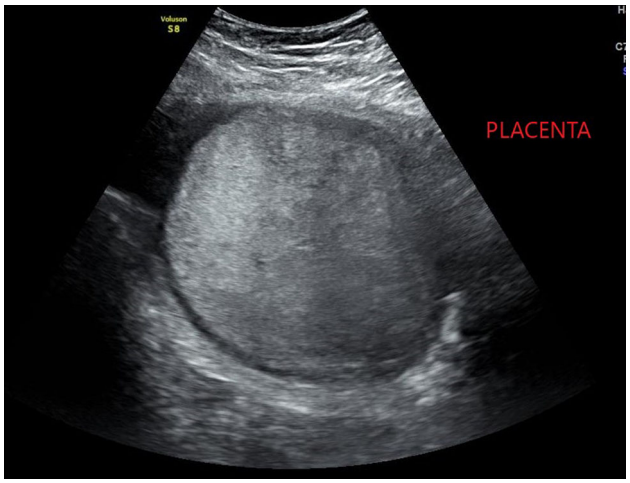

Fig. 2 Groundglass appearance of placenta

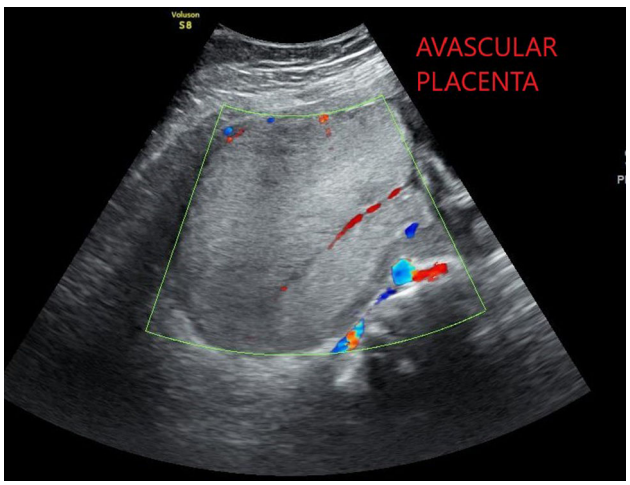

Fig. 3 Avascular placenta

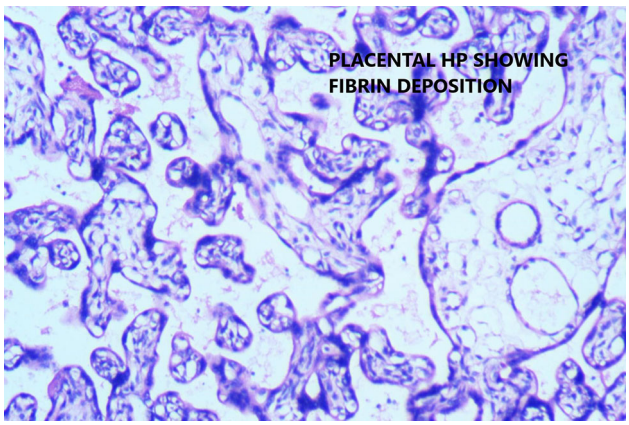

Fig. 4 Placental histopathological evidence of excess fibrin deposition

(Fig. 4). FISH and conventional karyotype reports were normal.

The patient was advised for a laboratory evaluation for antiphospholipid syndrome and counseled about positive benefits of low dose aspirin and LMWH even if the reports came back negative. She had a missed miscarriage after three months inspite of low dose aspirin and LMWH. Her antiphospholipid evaluation was negative.

She conceived again in 6 months, low dose aspirin and LMWH were started in a dose of 75 mg and LMWH 5000 IU respectively daily. Folic acid was also started. The NT and fetal abnormality scan were reported as normal. Her uterine artery PI was in the low risk range. After 28 weeks, tests for fetal surveillance were done and were normal. She underwent a cesarean section at 37 weeks for breech presentation. Her intra and post operative period was normal. The weight of the neonate was normorange.

Discussion

While obstetricians are always aware of the relevance of placental position, one distinct process that must also be borne in mind is Maternal floor infarct/ Hyaline placenta/massive perivillous fibrinoid degeneration (5). It is associated with significant fetal risk and a high chance of recurrence in the next pregnancy. It is characterised by massive deposition of fibrin in the basal plate of the placenta and perivillous space of basal plate, encasing the villi, which then become avascular and necrotic leading to impaired perfusion of the intervillous space (1). There is hypoplasia and sclerosis of the engulfed villi as well (6).

It is also characterised by early onset FGR with features of utero—placental insufficiency. It has a relatively high

recurrence rate and carries significant risk of fetal demise.(1). A subset of patients has a signature of maternal antifetal rejection as a mechanism and 80% patients with MFI have pregnancy loss (40% IUD and 40% second trimester spontaneous abortion(6). Some other studies have shown an incidence of 40% fetal death, preterm birth 58% and FGR 54% (7). Recurrence in the same study was around 12.2% . Some studies have quoted an incidence of around 0.028–0.5% and morbidity such as prematurity (35.4%), FGR (31%0 and mortality—stillbirth and IUD around 24%. (8). The etiology of MFI is uncertain, but thrombophilia maybe associated with it in upto 40 % cases (9).

Some others show an incidence of 0.028%, with recurrence rate of 18% and all infants in this study showed FGR. The incidence of fetal death was 31% and incidence of preterm birth was almost 33 % (10).

MFI was originally described by Benirschke in 1961 as a passing comment in an article focussed on examination of the placenta. In 1967, it was Benirschke and Driscoll who described it as a diffuse thickening of the maternal floor of the placenta.(11). They suggested that the " lack of fissuring of decidual basalis " in a term placenta was abnormal and called it MFI to explain the deposition of fibrin (11). Although the term infarction is used repeatedly, it is a misnomer because the lesion is not an "infarct", but the term has stuck. The term MFI is used more by obstetricians whereas MPFD is preferred by pathologists.

One hallmark of this condition on ultrasound is the typical basal location near the decidua (maternal surface) which is unique to maternal floor infarct (5). The increased density of the placenta is seen on ultrasound (1). MPFD/MFI are considered to represent the same process with different severity.

Studies have suggested a maternal fetal immunological rejection, with the fibrinoid degeneration leading to obstruction of blood flow towards the chorionic villi by microthrombi (12). If that may be a possible etiology, then use of low dose aspirin and LMWH may have a positive effect on the pregnancy outcome in the patient in future pregnancy. Heparin has also been reported in cases with with or without APS (1).

Studies of dizygotic twins has shown that there may be a genetic—immunological cause for MFI, as it affected one of a pair of dizygotic twins (13).

PAPS, primary antiphospholipid syndrome, may be a predisposing factor in the development of this disease. Some studies have shown that when MPFD/MFI occurred with PAPS at a RPL clinic, use of low dose aspirin and LMWH showed promising results. The increased prevalence of late pregnancy complications in PAPS with history of early loss suggests that low dose aspirin and LMWH treatment does not eradicate the underlying pathology, but reduces its severity. Therefore early losses may be converted to treated pregnancy with late antenatal complications (12).

In the presence of APS, treatment did not eradicate the problem, but the severity was less and in its absence, Heparin improved outcome and maintained growth (14).

Others have shown that recurrent MPFD can be treated not only with Low dose aspirin and LMWH but by adding IgG also (15).

The therapeutic benefit of heparin and aspirin in improving outcomes in women with obstetric APS has been presumed to relate to their anticoagulant properties. Heparin may inhibit aPL binding to trophoblastic cell membranes, modulating trophoblastic apoptosis, promoting trophoblastic cell invasiveness and reducing complement activation with the ensuing inflammatory response at the decidual—placental interface (16).

Some studies have shown therapeutic benefit of using statins like Pravastatin in future pregnancies in patients with earlier MPFD. If routine treatment with Aspirin, LMWH has not helped patients with MPFD, then this novel treatment can be offered after thorough counselling. That study used the drug after 5–6 pregnancy failures for a patient who was thoroughly counselled about that drug being an " innovative" therapy(17). It is a cholesterol lowering drug that reverses the angiogenic/anti—angiogenic imbalance. Although statins are category 'X" drugs and anomalies like VACTERL or isolated limb anomalies may be seen, abnormal pregnancy has not been reported in studies with exposure to Pravastatin till now(18). But this treatment is presently in the experimental realm.

Conclusion

Placenta, the interface between the maternal and fetal compartment has to be studied in detail not just during antenatal ultrasound, but a gross examination is warranted after delivery and in many cases histopathological study should also be included as part of complete study. The detailed examination helps the treating clinician in managing the index pregnancy and future pregnancies to achieve good results. Perivillous fibrinoid degeneration can have good results if proper management as per above discussion is done.

References

1. Al Sahan N, Gynspan D, Von Dadelszen P, Guslin A. Maternal floor infarction – Management of an unrecognised pathology. J Obst Gynecol Res. 2014;40:293–6.
2. Benirschke K. Examination of placenta. Obst Gynecol. 1961;18:309–33.
3. Chen A, Roberts DJ. Placental pathologic lesions with a significant recurrence risk, What not to miss. J Pathol Microbiol Immunol. 2017;126:589–601.
4. Kulkarni AD, Palaniappan N, Evans MJ. Placental pathology and stillbirth, A review of the literature and guidelines for the less experienced. J Fetal Med. 2017;4:177–85.
5. Dashe JS, Hoffman BL USG evaluation of the placenta, membranes and umbilical cord. Callen's USG in OBGY, 6th edition 2017. P. 674–683.
6. Romero R, Whitten A, Korzeniewski SJ, et al. Maternal floor infarction/massive perivillous fibrin deposition: a manifestation of maternal antifetal rejection? Am J Reprod Immunol. 2013;70:285–98.
7. Andres RL, Kuyper W, Reshik R, Piacquadio KM, Benirschke K. The association of maternal floor infarction of the placenta with adverse perinatal outcome. Am J Obstet Gynecol. 1990;163:935–8.
8. Katzman PJ, Genest DR. maternal floor infarction and massive perivillous fibrinoid degeneration—histological definitions, association with IUGR and risk of recurrence. Paed Dev Pathol. 2002;5:159–64.
9. Gogia N, Machin GA. Maternal thrombophilias are associated with specific placental lesions. Pediatric Dev Pathol. 2008;11:424–9.
10. Bane AL, Gillan JE. Massive perivillous fibrin deposition causing recurrent placental failure. BJOG. 2003;110:292–5.
11. Benirschke K, Driscoll SG. maternal floor infarction—the Pathology of human placenta. New York: Springer Verlag; 1967. p. 328–30.
12. Sebire NJ, Backos M, Goldin RD, et al. Placental massive perivillous fibrinoid degeneration associated with antiphospholipid syndrome. BJOG. 2002;109:570–3.
13. Redine RW, Jiang JG, Shah D. Discordancy for maternal floor infarction in dizygotic twin placentas. Hum Pathol. 2003;34:822–4.
14. Makino A, Suzuki Y, Yamamoto T, Sato T, Kojima K. Use of aspirin and LMWH to prevent recurrence of maternal floor infarction in women without evidence of antiphospholipid anibody syndrome. Fetal Diagn Ther. 2004;19:261–5.
15. Morreti SAF, Gruslin A, Grynspan D. Recurrent massive perivillous fibrinoid degeneration and chronic intervillositis treated with heparin and Intravenous immunoglobulin- a case report. J OBGY Can. 2017;39(8):676–81.
16. Kutteh WH, Hinote C. Antiphospholipid antibody syndrome. Obstet Gynecol Clin N Am. 2014;41:113–32.
17. Chaiworapongsa T, Romero R, Korzeniewski S, Chaemsaithong P, Andrade EH. Provastatin to prevent recurrent fetal death in MPFD. J Matern Fetal Neonatal Med. 2016;29(6):855–62.
18. Edison RJ, Muenke M. Mechanistic and epidemiologic consideration in the evaluation of adverse birth outcome following gestational exposure to statins. Am J Med Genet. 2004;131:1287–98.