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Maternal floor infarction: Management of an underrecognized pathology

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Abstract

Maternal floor infarction is a relatively rare condition characterized clinically by severe early onset fetal growth restriction with features of uteroplacental insufficiency. It has a very high recurrence rate and carries a significant risk or fetal demise. Pathological characteristics include massive and diffuse fibrin deposition along the decidua basalis and the perivillous space of the basal plate. We present a case of recurrent maternal floor infarction and propose diagnostic clues as well as potential therapeutic options.

Key words: diagnosis, elevated α-fetoprotein, fetal growth restriction, fibrin deposition, globular placenta, maternal floor infarction, therapy.

Introduction

Maternal floor infarction (MFI) is underrecognized and likely occurring at a higher frequency than previously reported. It is characterized by massive and diffuse fibrin deposition along the decidua basalis and the perivillous space of the basal plate. MFI is associated with an increased risk of fetal demise and severe growth restriction. Unfortunately, the antenatal features of this disorder have not been well defined and unless specific attention is paid to these placental pathological features, the diagnosis is often missed. We present a case of recurrent MFI with associated sonographic features and maternal screening α-fetoprotein (MSAFP) elevations. We suggest that the combination of these antenatal findings should raise the suspicion for the diagnosis of MFI and lead to increased fetal surveillance as well as detailed placental pathology.

Case Report

A 34-year-old woman, G4P2A1L2, was referred with a history of two pregnancies complicated by severe early onset intrauterine growth restriction (IUGR).

In her first pregnancy, an abnormal integrated prenatal screening (IPS) was noted with elevated AFP (MSAFP, 3.43 MoM). Early onset IUGR and oligohydramnios were noted at 20 weeks of gestation. On ultrasound, the placenta was thick, globular and heterogeneous with areas of infarcts along with poor perfusion (Fig. 1). Abnormal umbilical Dopplers (absent end diastolic flow) were evident at 26 weeks with poor growth. At 30 weeks, delivery (by cesarean section) was indicated due to a pulsatile umbilical vein and a non-reassuring fetal status. The infant weighed only 781 g (third centile), and is alive and well. The patient developed pre-eclampsia post-partum. Placental pathology showed massive perivillous fibrin

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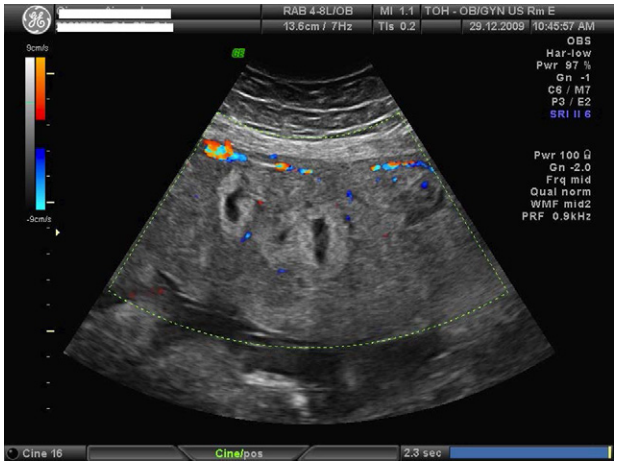

Figure 1 Transabdominal ultrasound image of the placenta at 32 weeks of gestation. The placenta appears heterogenous and bulky with multiple areas of infarction and poor vascular perfusion.

deposition occupying 65% of the placenta (Fig. 2a). The preserved villi showed accelerated villous maturation with increased syncytiotrophoblastic knotting. Investigations for antiphospholipid antibody syndrome and thrombophilia were all negative.

During the second pregnancy, prophylactic aspirin (81 mg/day) and folic acid (5 mg/day) were started. IPS was abnormal (MSAFP, 6.56 MoM). The placental sonogram revealed the same characteristics as during the first pregnancy. Oligohydramnios and abnormal umbilical artery Doppler were seen at 34 weeks. A repeat cesarean section was performed at 35 weeks due to oligohydramnios and a large fetal intra-abdominal umbilical vein varix. Birthweight was 2022 g (10th centile) and the child is alive and well. Postnatally, the patient developed mild preeclampsia. Placental pathology revealed massive perivillous fibrin deposition occupying 50% of the placental volume (Fig. 2b). There was a focal intervillous thrombus measuring 1.0 cm.

She presented for care in her third pregnancy at 13 weeks, already on folic acid as well as aspirin. An earlier ultrasound confirmed dates. A repeat study at 13 weeks revealed a 1-week discrepancy. IPS was abnormal (MSAFP, 3.84 MoM). Ultrasounds showed oligohydramnios starting at 17 weeks and decreased growth velocity. The placenta was globular and heterogeneous. Uterine artery Dopplers were normal. At 21 weeks, increased resistance in umbilical artery and severe oligohydramnios were noted. At 23 weeks, biometry was consistent with IUGR (<3rd %) and anhydramnios was found. She was counseled

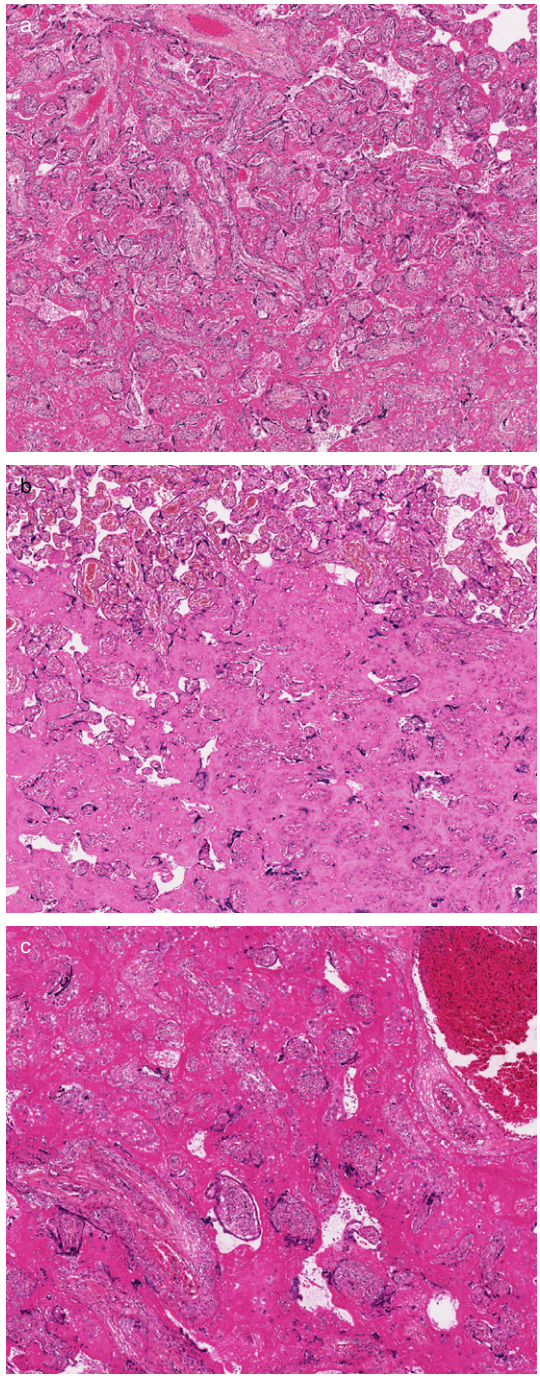

Figure 2 Representative histology of placenta from each pregnancy (hematoxylin phloxine saffron stain, original magnification ×40). Selected fields of routinely stained slides from the placentas of each pregnancy, consecutively. In these fields, villi can be seen surrounded by abundant fibrin which appears as bright pink on routine histological stains. By correlating the gross and histological appearance an estimate of the total volume of fibrin is arrived at.

regarding the poor prognosis and the high possibility of fetal demise. At 25 weeks, absent end diastolic flow in umbilical arteries and lack of growth were noted. The patient was started on sildenafil citrate to try to optimize placental perfusion and promote growth. Within the next 2 weeks, a 45-g increment in weight was noted; however, middle cerebral artery redistribution, reversed end diastolic umbilical flow and a reversed 'A' wave in ductus venosus were present at 29 weeks. Intrauterine fetal demise was diagnosed at 30 weeks with estimated fetal weight still at 387 g. Placental pathology revealed massive perivillous fibrin deposition occupying 75% of the placental volume (Fig. 2c).

Discussion

Maternal floor infarction is a rare condition first described in 1961 by Benirschke and Driscoll. Its incidence has been reported to be as high as 0.5%.[1]

It is characterized by marked, diffuse increase in fibrin deposition along the decidua basalis and the perivillous space of the basal plate.[2] Histologically, the basal villi are enmeshed in eosinophilic fibrinoid material.

The etiology of MFI is uncertain but thought to be associated with thrombophilia in up to 40% of cases.[3] Uxa *et al*. failed to demonstrate an association between genetic polymorphisms in plasminogen activator inhibitor-1 (*PAI-1*), thrombin activated fibrinolysis inhibitor (*TAFI*), plasminogen activator urokinase (*u-PA*) and plasminogen activator tissue (*t-PA*), which may mediate defective fibrinolysis in MFI.[4]

Maternal floor infarction causes IUGR and intrauterine fetal death with a reported risk of 69% and 40%, respectively. The recurrence rate varies from 14% to 39%.[1,5]

Maternal floor infarction has important implications for the fetus that extend beyond the perinatal period. Up to 70% of fetuses born from a pregnancy complicated by MFI have neurological and/or developmental impairment, especially white matter infarction.[6]

In our case, the patient's three pregnancies were complicated by early onset IUGR with oligohydramnios in the context of a high MSAFP. Placental morphology by ultrasound showed a thickened, globular, inhomogeneous placenta with areas of infarction. No evidence of thrombophilia or antiphospholipid antibody syndrome was found. Although there appeared to be some improvement in outcome in the second pregnancy that may be attributable to aspirin and folic acid, the third pregnancy, managed similarly, carried the worst outcome. Given this apparent failure of therapy and in the hope of achieving an improvement in growth, sildenafil citrate was administrated as reported by von Dadelszen.[7] An increase in abdominal circumference was noted after 2 weeks of sildenafil and this, after a period of absent fetal growth. Whether this was related to sildenafil requires further investigation.

Other therapies have been explored in the context of MFI. Heparin was reported in cases with MFI with or without antiphospholipid syndrome (APAS). In cases with APAS, treatment did not eradicate the underlying pathology but reduced its severity,[8] whereas in absence of APAS, heparin improved outcome and maintained normal fetal growth.[9] In a case of APAS and recurrent MFI where aspirin and heparin had failed, i.v. immunoglobulin was started preconceptually and improved the outcome of two consecutive pregnancies with evidence of MFI on placental pathology.[10]

Our report illustrates the value of MSAFP determination and placental sonographic evaluation in cases at risk of MFI. The raised AFP in the maternal circulation likely occurs as a result of the excess fibrin deposits found at discontinuities in the syncytial layer which allows AFP transfer from the fetal to the maternal circulation. Furthermore, the early discrepancy in growth also raises suspicion of severely abnormal placentation.

In conclusion, a suspicion of MFI arises in the context of early onset or recurrent IUGR. Additional diagnostic clues include elevated MSAFP and a thick, globular, inhomogeneous placenta on ultrasound. To confirm the diagnosis, placenta pathology is required. Given the described association with APAS and thrombophilia, screening may be beneficial. Treatment with aspirin and heparin has suggested improvement in outcome in some cases although further research is needed. To our knowledge, sildenafil use has not been reported in the context of MFI. Although its vasodilatory activity is unlikely to prevent such an insult, it may improve uteroplacental circulation and fetal growth once the disease is established.

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Disclosure

None.

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