—Case Reports—

Placenta with Old, Diffuse Infarction that Was Difficult to Differentiate from a Placental Tumor

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Placental lesions, including placental infarction, are associated with fetal and neonatal mortality and morbidity. We present a case of fetal growth restriction associated with an old, diffuse placental infarction. Because the placenta had only a single viable cotyledon, the others being atrophic, the lesion appeared to be a placental tumor on prenatal ultrasonography. The patient did not have pregnancy-induced hypertension. At 31 weeks of gestation, a cesarean delivery was performed because of fetal growth arrest and breech presentation. A small-for-gestational age infant was delivered with Apgar scores of 8 at both 1 and 5 minutes, and the infant had cleft palate and cleft lips. Pathological examination of the placenta revealed an old, diffuse infarction without neoplastic change. In cases in which a placental tumor causing fetal growth restriction is strongly suspected, diffuse placental infarction should be considered as part of the differential diagnosis, because placental tumors are associated with poor maternal prognosis. (J Nippon Med Sch 2015; 82: 156–158)

Key words: placental disease, fetal growth retardation, prenatal ultrasonography

Introduction

Placental lesions, including placental infarction, are associated with fetal and neonatal mortality, morbidity, and outcomes[1]. Placental infarction is a main cause of maternal vascular underperfusion[1]; however, partial placental infarction is common but of minimal clinical significance[2]. When infarction occurs early, centrally, or extensively, it is strongly associated with pregnancy-induced hypertension, fetal growth restriction, and even fetal death[3]. Some investigators have shown that the incidence of villous infarction is higher in cases of fetal growth restriction than in normal controls[4,5].

We present a case of severe fetal growth restriction with diffusely infarcted placenta in which the prenatal ultrasonography findings and postpartum clinical findings were discrepant.

Case Report

A 36-year-old woman, gravida 2, para 1, was referred to our hospital at 27 weeks 1 day of gestation because of fetal growth restriction. The course of her previous pregnancy and delivery was normal, and her first child had no medical problems. The present pregnancy had occurred through spontaneous conception. The patient had a smoking habit (10 cigarettes per day). The gestational age, established with ultrasonographic examination of the fetal crown-rump length, was 9 weeks 2 days. The results of serological tests (e.g., rubella, syphilis, and toxoplasmosis) showed no abnormalities. At 27 weeks 5 days of gestation, the estimated fetal weight was 604 g (−4.0 standard deviation, fetal growth standard statistical distribution from the Japanese Society of Ultrasonics in Medicine). The ultrasonographic examination of the fetus showed no life-threatening anomalies. In the placental parenchyma, a heterogeneous, hyperechoic lesion with an anechoic center was detected (**Fig. 1**). The lesion showed peripheral blood flow, which was suggestive of a placental tumor.

At 31 weeks of gestation, emergent cesarean delivery was performed because of fetal growth arrest and fetal breech presentation without a nonreassuring fetal status. In her medical course, neither hypertension nor protein-

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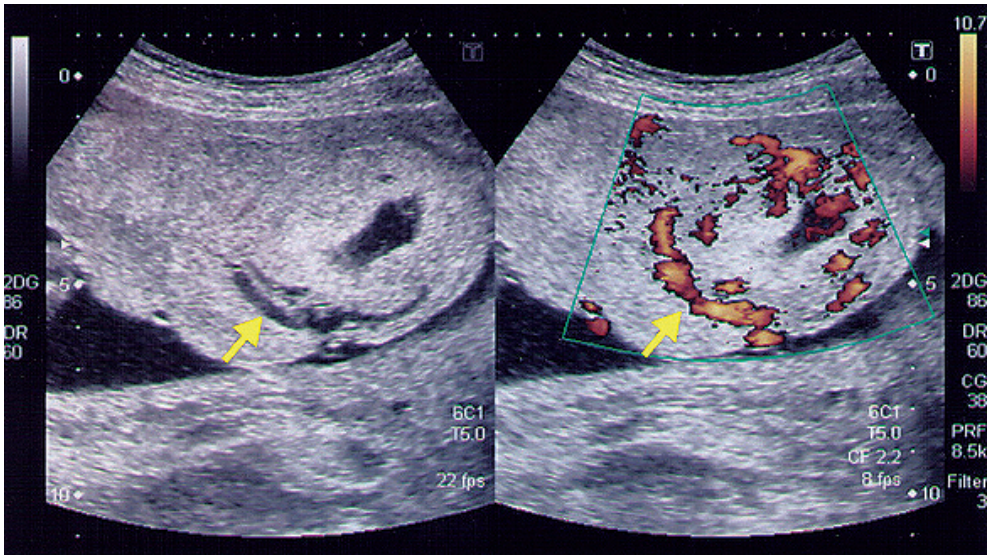

Fig. 1 Prenatal ultrasonography at 27 weeks 5 days of gestation.
The left window is the B-mode image, showing a heterogeneous lesion in the placental mesenchymal tissue (**yellow arrow**). In the right window, power Doppler imaging showed peripheral blood flow to the lesion.

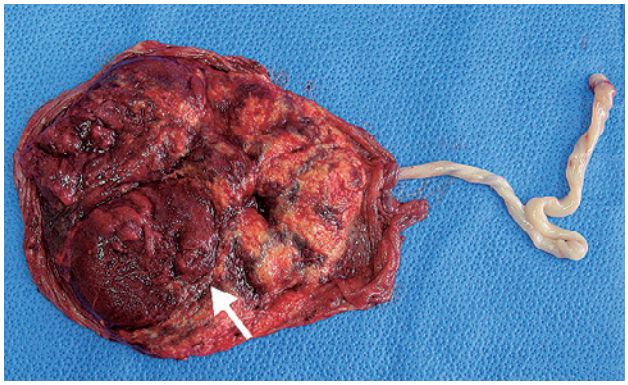

Fig. 2 Macroscopic placental findings.
On the maternal surface of the placenta, only a single cotyledon (**white arrow**) was intact, and the others were atrophic.

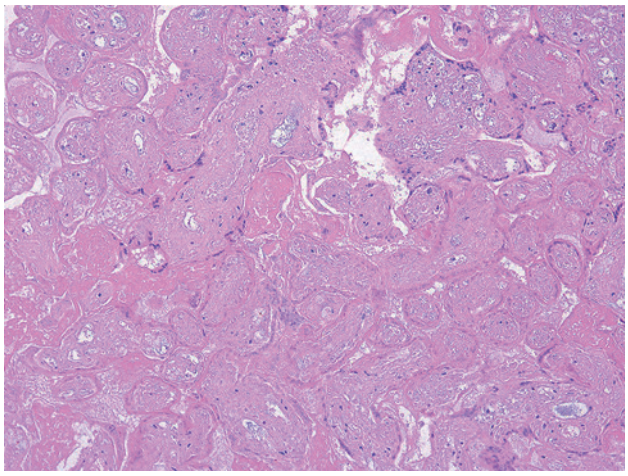

Fig. 3 Microscopic placental findings.
Pathological findings revealed the existence of an old, diffuse infarction. Villous calcifications and intervillous fibrin deposition were widespread, and the changes were more prominent at the rim of the infarcted lesion.

uria was found. The estimated blood loss, including amniotic fluid, during the cesarean section was 600 mL. A 668-g female infant (<5th percentile) was delivered with Apgar scores of 8 at both 1 and 5 minutes. The pH of the umbilical artery blood was 7.293. The newborn had cleft palate and cleft lips; no other anomalies were present. Following cardiopulmonary resuscitation, the infant was transferred to the neonatal care unit.

The placenta weighed 220 g and measured 13.5 (W)× 12.5 (D)×1.8 (H) cm. On the maternal surface of the placenta, only a single cotyledon was intact with the others being atrophic (**Fig. 2**). The umbilical cord was 54.0 cm in length and 0.6 cm in diameter. The amniotic fluid was clear.

Pathological examination (**Fig. 3**) revealed the presence of an old, diffuse infarction without neoplastic change. On the villous surface, the 2-layered structure consisting of the syncytiotrophoblast and cytotrophoblast was obscure, as observed in a typical third-trimester placenta. In addition, villous calcification and intervillous fibrin deposition were widespread but were more prominent in the rim of the infarction lesion. Although no inflammatory change was found in the umbilical cord and the placenta, severe calcification was observed in 1 umbilical artery.

Discussion

Placental infarction can be associated with fetal morbidity and mortality. In the present case, the fetal growth restriction might have been caused by the diffusely infracted placenta, which had only a single functional cotyledon. Vedmedovska et al. have shown that fetal growth restriction with placental infarction is associated with a smoking habit[4]. Maternal smoking during pregnancy is associated with a higher risk of oral clefts in infants[6]. The smoking habit of our patient might be correlated with the perinatal problems, such as fetal growth restriction, placental infarction, and the cleft lips of the infant.

Placental infarction can be detected with ultrasonography, but the sensitivity of this modality is low, with only approximately 10% of all infarctions identified prenatally[7]. The ultrasonographic appearance of the infarcted placenta varies according to the cellularity, fibrin deposition, calcification, and hemorrhage[3]. The low sensitivity of prenatal ultrasonography might be attributed to this diversity. In the present case, a single viable cotyledon was present in the placenta, because the other cotyledons were infarcted. However, the intact area showed placental lakes and peripheral blood flow. Because of its prominence, we had mistaken the intact cotyledon for a placental tumor. Large placental tumors are associated with a number of pregnancy complications[2]. Zanardini et al. reported a series of 19 cases of giant chorioangioma (defined as measuring more than 4 to 5 cm in diameter)[8]. In that study, fetal growth restriction was diagnosed antenatally in 6 cases[8]. On the other hand, Park et al. have reported that multifocal or massive villous infarction was more frequently present in cases with fetal growth restriction (26.7%, 12 of 45 cases) than in cases without (0%, 0 of 24 cases)[5]. Similarly, Vedmedovska et al. have reported that villous infarction is significantly related with fetal growth restriction (controls: 14%, 7 of 50 cases, vs. FGR: 34%, 17 of 50 cases)[4]. In cases in which a placental tumor causing fetal growth restriction is strongly suspected, diffuse placental infarction should be considered as part of the differential diagnosis. Moreover, some types of placental tumor, such as gestational trophoblastic tumors and metastases from cancers, are associated with a poor maternal prognosis[2]. We recommend that the prenatal ultrasonographic examination be performed on the entire placenta, in particular if an abnormal placental lesion is found. Future development of ultrasonography will allow greater reliability for prenatal detection of placental pathological changes.

Conflict of Interest: The authors declare no conflict of interest.

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