RECURRENT PLACENTAL MICROCALCIFICATIONS IN THE SECOND TRIMESTER

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Placental problems are uncommon during pregnancy. Benign pathologic findings of the placenta include placenta previa or abruption, velamentous cord insertion, succenturiate lobe, circumvallate placenta, battledore placenta, placentomegaly, and chorioangioma. We present the case of a mother who experienced recurrent placental microcalcifications in the early second trimester, with associated intrauterine growth retardation (IUGR).

A 27-year-old woman, gravida 2, para 1, presented at 24 weeks of gestation. She had received prenatal care in our department since early pregnancy. The prenatal ultrasound examination performed before 24 weeks of gestation was uneventful, but progressive microcalcifications of the placenta were noted subsequently. The entire placenta was covered with small calcified spots.

Her past gynecologic and obstetric history was notable for having been a very low birth weight baby (1,450 g, delivered at 29 weeks preterm), and for having had a very low body mass index of 16–18 kg/m^2 since childhood. Her general condition was normal, except for frequent common colds. At the time of her second pregnancy, her body weight was 38 kg and her height was 150 cm. Her body mass index was 17 kg/m^2 (normal, 22–25 kg/m^2). Her first pregnancy had occurred 2 years earlier and severe placental microcalcifications had developed, along with IUGR by 27 weeks of gestation. Her baby was delivered at term by cesarean section because of breech presentation. The baby weighed 2,450 g. The histopathologic findings of the placenta were unremarkable except for grade III chorioamnionitis.

Placental microcalcifications developed earlier during her current pregnancy, at 24 weeks of gestation. The snow-like small calcified spots were scattered around the entire placenta (Figures A–C), but did not occur on the fetal parts of the placenta. Symmetric IUGR was noted at 32 weeks of gestation, earlier than in her last pregnancy. Steroid supplementation (betamethasone 12 mg intramuscularly every 24 hours for 2 doses) was given for lung maturation. The screening results for toxoplasmosis, other congenital syphilis and viruses, rubella, cytomegalovirus, and herpes simplex virus were unremarkable and she had no intrauterine infection, as with her previous pregnancy. She underwent cesarean delivery at 36 weeks of gestation because of severe IUGR, with an estimated fetal weight of 2,200 g. A healthy baby was delivered, with no obvious perinatal problems.

Placental calcium deposition is a normal physiologic process that occurs throughout pregnancy. During the first 6 months, the microcalcification is microscopic; macroscopic plaques appear in the third trimester, most commonly after 33 weeks. The calcium is deposited in the basal plate and septa, but is also found in the perivillous and subchorionic spaces. Calcium plaques are detected as echogenic foci with no obvious acoustic shadows [1–3]. The incidence of placental calcification increases with gestational age, beginning at about 29 weeks [4]. Placental calcification is used to assess maturity. It can be divided into four grades: grade 0, uniform echogenicity (< 18 weeks of gestation); grade 1, occasional parenchymal calcification from 18–29 weeks of gestation; grade 2, occasional basal calcification at about 30 weeks or more of gestation; grade 3, significant basal calcification at more than 39 weeks of gestation. Abnormal maturity of the placenta (grade 3 calcification) is seen in association with maternal smoking, hypertension, and diabetes [5–8]. There is no proof that placental calcification has any pathologic or clinical significance [9,10], except in the case of the early appearance of grade 3 calcification, which is considered to be abnormal and increases the risk of placenta insufficiency.

Some underlying diseases or factors can induce early placental microcalcification. These include intrauterine infection (with *Toxoplasma gondii*), smoking, and hypertension. None of these potential etiologies occurred in the current patient.

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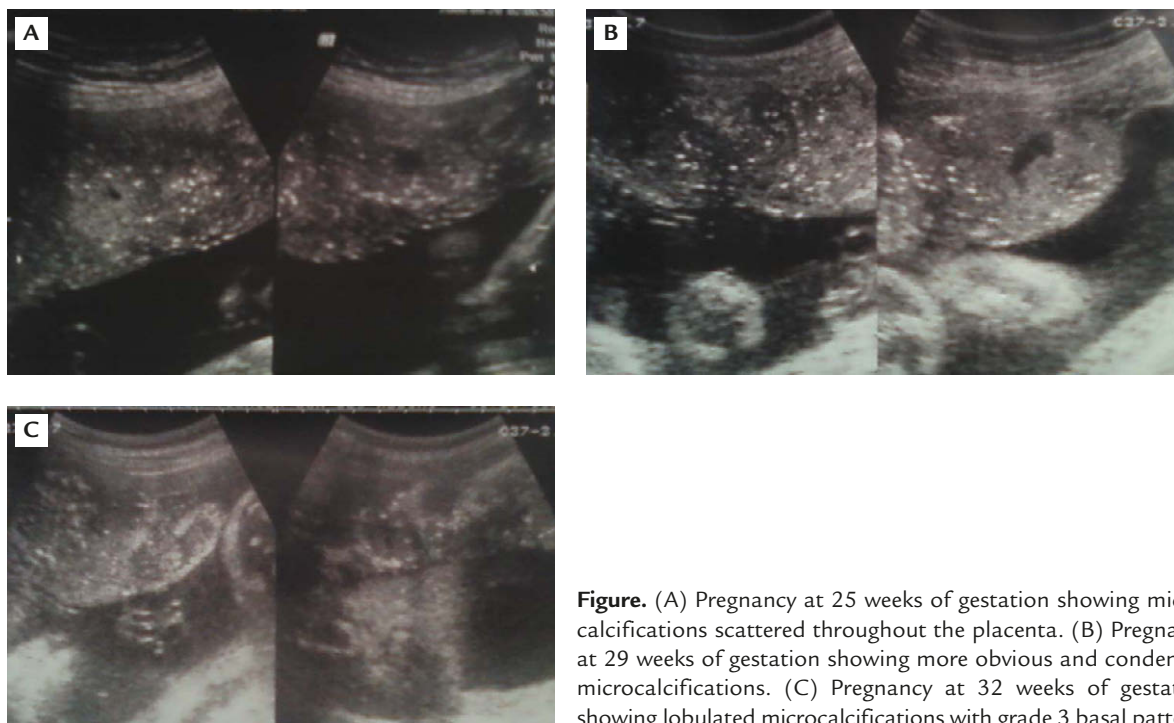E-mail: chia007@iris.seed.net.tw

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Figure. (A) Pregnancy at 25 weeks of gestation showing micro-calcifications scattered throughout the placenta. (B) Pregnancy at 29 weeks of gestation showing more obvious and condensed microcalcifications. (C) Pregnancy at 32 weeks of gestation showing lobulated microcalcifications with grade 3 basal pattern.

A sonographically estimated fetal weight during the prenatal period of less than the tenth percentile is an indicator of IUGR. Because this approach is based purely on a weight threshold, it only identifies small fetuses at risk of adverse outcomes. Approximately 70% of infants with birth weights below the tenth percentile develop normally, but are constitutionally small and are not at risk of adverse outcomes; they present one end of the normal spectrum for neonatal size.

The anatomic survey should focus on the detection of markers of aneuploidy, non-aneuploid syndromes, fetal infection, and anatomic defects. There is a relationship between aneuploidy and fetal anomalies such as gastroschisis, omphalocele, diaphragmatic hernia, congenital heart defects, and sonographic markers such as echogenic bowel and nuchal thickening. Markers for viral infection may be nonspecific, but include echogenicity and calcification in organs such as the brain and liver. Identification of any of these abnormalities on ultrasound significantly affects the differential diagnosis and frequently has a decisive impact on outcome.

In conclusion, after evaluating several possible causes of early placental microcalcifiation, the cause of early placental microcalcification with IUGR remains obscure, and babies delivered from these pregnancies are constitutionally small.

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