A Case Report of Placental Infarction

DAIJI SATO, YASUYUKI SHINOHARA, AKIHIKO NAKASHIMA, YOSHIAKI HSOKAWA, TERUYUKI NAKASHIMA AND NOBU IDE*

Department of Pathology, Kurume University School of Medicine and *St. Mary's Hospital, Kurume, 830 Japan

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Summary: A 31-year-old Japanese female, gravida 4, para 0, who had previously experienced three spontaneous abortions during 7th or 8th month of gestation was diagnosed with intrauterine fetal death at 26th weeks of gestational age. Investigations for maternal and fetal etiologies, including toxemia, incompatibility of blood groups and types, and infections, proved negative. Anoxia which resulted in the demise of the fetus is believed to have been responsible for the rare combination of extensive placental infarction in an asymptomatic gravid woman.

Key words: placenta — placental infarction — infarction — fetal death — intrauterine fetal death

Introduction

Placental infarction is not a rare lesion. Extensive placental infarction leading to intrauterine fetal death is a not uncommon complication of toxemia (preeclampsia), antepartum hemorrhage, and chronic renal disease. Extensive placental infarction without maternal complication, however, is very rare. Fox (1967) described only one case of extensive placental infarction in 15 cases of unexplained intrauterine fetal death with uncomplicated pregnancies. We report a case of diffuse and large infarctions of placenta without maternal complications that resulted in the demise of the fetus.

Case

A 31-year-old Japanese female, gravida 4, para 0, had previously experienced three spontaneous abortions during the 7th or 8th month of gestation from 1976 to 1979. The third fetus was autopsied, but the cause of fetal death could not be determined.

The patient’s last menstrual period was June 15, 1980. This patient was closely observed as a high risk pregnancy. When examined at 15 weeks of gestation, the patient’s weight gain was less than normal and the height of fundus uteri was observed to be lower than normal for 15th weeks of gestation. She was consequently admitted to St. Mary's hospital for maintenance of graviditas.

Laboratory data included hemoglobin level 15.1 g/dl, hematocrit 42.5 %, leucocyte count 7700, and erythrocyte count $4.5 \times 10^4$, total protein 7.3 g/dl, albumin 3.5 g/dl, blood urea nitrogen 11 mg/dl, creatinine 0.8 mg/dl, and cholesterol 209 mg/dl. Other data for liver function revealed normal levels. Urinary estriol ($E_3$) was 5 mg/day, HPL 1.2 μg/ml, HsALP 2.9 U., and HsLAP 116 U. The antibody titer for Toxoplasma was less than 16 x, Herpes simplex 64 x, Cytomegalovirus 32 x,
Rubella 64x, and Wassermann's reaction was negative. Incompatibility testing of blood groups and types proved negative. At 26th weeks of gestational age, the fetus was spontaneously aborted. The fetus was then autopsied.

Pathologic findings

The fetus was a macerated, extremely premature male infant, and his weight was 450g and crown-heel length 29cm (Fig. 1). All organs appeared expectedly premature. Subarachnoid hemorrhage was recognized at the parietal lobe of cerebrum.

The placenta weighed 140g, 11.5×11.5 cm in diameter (Fig. 2) and 3cm in thickness. Extensive infarction eminating from two large infarcts was seen near the umbilical cord on the cut surface of placenta. The central zones of the large infarcts demonstrated liquefied degeneration. In coloration, one infarct was reddish brown and the other was grayish-white with chocolate-color tinge (Fig. 3 and Fig. 4). In the latter lesion, microscopic examination revealed degenerated, eosinophilic, amorphous materials and massive fibrin deposits in the intervillous spaces (Fig. 5). Leukocytic infiltration was seen among the chorionic villi. In the former lesion severe leukocytic infiltration was observed in the decidua and intervillous spaces. Pooling of erythrocytes and deposi-

Fig. 1. Fetus and placenta
Fetus is a very premature infant, and shows severe post-mortem change with maceration.

Fig. 2. The maternal part of placenta (decidua basalis)
Liquefied degenerations are seen at the central portion.

Fig. 3. Cut surfaces of the placenta
The middle three cuts reveal extensive infarction.
PLACENTAL INFARCTION

Fig. 4. Gross figure of the placental infarction
Two large infarcts are recognized. The right side is a reddish brown colored infarct and the left side is grayish white with chocolate-color tinge, the central zone of which shows liquefaction.

Fig. 5. Microscopic picture of the latter infarction (Fig. 4)
The villi are degenerated and there are fibrin deposits in the intervillous spaces. (×100, H.E.)

Fig. 6. Microscopic picture of the reddish brown colored infarct (Fig. 4)
Leukocytic infiltration and deposition of fibrin are seen in the intervillous spaces. Calcifications are recognized at the periphery of the chorionic villi. (×100, H.E.)

Fig. 7. Microscopic picture of the decidua
Thrombi are formed in the decidual small arteries. (×100, H.E.)

Discussion
This 31-year-old female experienced four spontaneous abortions and in each case, the cause of fetal death was unknown clinically. We have no information concerning the first three fetuses; thus, we are unable to draw any conclusions as to the similarity of our case to the earlier abortions. No fetal abnormalities, such as congenital abnormalities or infections, were notable. Nor were any maternal abnormalities, such as toxemia, incompatibility of blood groups and types, or infections, evident. Only two, large diffuse infarcts were noted primarily in the central zone near the umbilical cord. The necrotic areas

Fig. 6

Fig. 7
extended from the subchorionic space to
the basal plate. Microscopic examination
revealed degenerated villi and fibrin depo-
sition with leukocytic infiltration in the
intervillous spaces. Placental infarction is
classified into four types by Fox (1963):
true infarction, subchorionic fibrin deposi-
tion, intervillous thrombosis, and perivil-
lous fibrin deposition. Subchorionic fibrin
deposition is triangular, its base fused with
the undersurface of the chorionic plate.
Microscopically, this is seen as fibrin with-
out villi. Intervillous thrombosis is round
and recognized in the central or peripheral
areas of the intervillous space. Microscopi-
cally, this shows the same picture of fibrin
thrombus with no villi visible in the cen-
tral area of thrombus. Perivillous fibrin
deposition is found in the peripheral area
of the placenta; microscopically, fibrin
deposits are seen in the intervillous spaces.
Thickened basement membrane of villi and
fibrosis of the villous stroma are charac-
teristic (Fox, 1963).

In this case, fetal anoxia, consequent to
diffuse and large true infarctions of the
placenta, the cause of which are unknown,
is believed to be the cause of death. Other
fetal and maternal pathology were not
evident.

Placental infarction is not rare, and
small infarcts are common in uncomplicated
pregnancies. No relationship to intra-
terine fetal death and intrauterine growth
retardation has been shown (Fox, 1967;
Rolschau, 1978). Placental infarction is
almost never the cause fetal death (Potter
and Craig, 1975). Nevertheless, Little
(1960) and Fox (1967) have stated that
intrauterine fetal death may result from
extensive placental infarction involving
more than 30% of placental tissue. Ex-
tensive placental infarction is more com-
mon in toxemia (preeclampsia), antepart-
tum hemorrhage, and chronic renal dis-
eases than in uncomplicated pregnancies
(Potter and Craig, 1975). Placental infarc-
tion is said to result from the interrupt-
tion of maternal blood flow caused by
thrombosis of a spiral arteriole in the
decidua (Fig. 7) or in the basal plate,
retroplacental hemorrhage, or hemorrhage
into the basal plate (Fox, 1963; Potter
and Craig, 1975).

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