

—Reports on Experiments and Clinical Cases—

A Case of Maternal Reaction Due to Fetomaternal Transfusion

Shunji Suzuki^{1,2}, Toyohiko Kuwajima^{1,2}, Tomoaki Murata² and Tsutomu Araki²¹Department of Obstetrics and Gynecology, Tokyo Rinkai Hospital²Department of Obstetrics and Gynecology, Nippon Medical School**Abstract**

We present here a case of maternal reaction to fetomaternal transfusion complicated by subchorionic hemorrhage. A 40-year-old woman, gravida 2, para 0, was admitted to our hospital at 25 weeks' gestation because of high blood pressure. On the morning of 32 weeks and 2 days' gestation, she developed sudden onset nausea, dyspnea and regular uterine contractions. Blood pressure was 80/50 mmHg and pulse was 110 beats/minute. At this time, her WBC, hemoglobin and platelets were decreased significantly. Two hours after onset, the patient's condition improved spontaneously. Increased serum alpha-fetoprotein level (3.0 multiple of median) was observed. She was suggested to be a case of acute fetomaternal transfusion.

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Key words: ABO incompatibility, Alfa-fetoprotein, Fetomaternal transfusion**Introduction**

Few studies have investigated the maternal transfusion reaction due to fetomaternal transfusion¹. In a 1978 literature by Bergin et al.,² the signs and symptoms of an incompatible transfusion, such as fever, chills and symptoms of hemolysis with intravascular coagulation and defibrination, were reported as maternal reactions in a case of ABO-incompatible fetomaternal transfusion. We present here a case of maternal reaction due to fetomaternal transfusion complicated by subchorionic hemorrhage.

Case Report

A 40-year-old woman, gravida 2, para 0, was admitted to our hospital at 25 weeks' gestation because of high blood pressure, 150~170/90~100 mmHg. Her blood type was O-type, Rh positive. Her

first pregnancy at 36 years old was terminated at 16 weeks' gestation because of severe hypertension and liver dysfunction. She was diagnosed with chronic hypertension based on the findings of eye-grounds (Scheie: H₂S₁), and gave informed consent to receive medication with methyldopa (1,500 mg/day, *per os*) at hospital until delivery. Weekly ultrasound examinations were performed, and the fetus was monitored daily with cardiotocograms. She was managed conservatively, and the fetus grew compatible with about -0.4 SD of the dates. The ultrasonographic findings of the placenta and the amniotic fluid index (AFI) did not change significantly. The fetal cardiotocograms showed a reactive pattern.

At 31 weeks' gestation, her blood pressure was 130~140/80~90 mmHg. Laboratory tests revealed a white blood count (WBC) of 7,000/mm³ (normal range 4,000~8,000/mm³), hemoglobin of 9.5 g/dl (normal range 12~16 g/dl) and platelets of

Correspondence to Shunji Suzuki, MD, Department of Obstetrics and Gynecology, Tokyo Rinkai Hospital, 1-4-2 Rinkai-cho, Edogawa-ku, Tokyo 134-0086, Japan

E-mail: czg83542@mopera.ne.jp

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225,000/mm³ (normal range 200,000~400,000/mm³). Thrombin-antithrombin-III complex (TAT) was 8.3 µg/l (normal range <3.0 µg/l).

On the morning of 32 weeks and 2 days' gestation, she developed sudden onset nausea, dyspnea and regular uterine contractions. Blood pressure was 80/50 mmHg and pulse was 110 beats/minutes, and her body temperature was 39.0°C. At this time, her WBC, hemoglobin and platelets had decreased to 2,900/mm³, 8.3 g/dL and 130,000/mm³, respectively. Her blood gas levels were normal. Chest auscultation revealed no rale, and chest radiography and echocardiography were normal. Two hours after onset, the patient's condition improved spontaneously, and her blood pressure, pulse and body temperature were 108/56 mmHg, 90 beats/minute and 36.4°C, respectively. On the next day, her WBC, hemoglobin and platelets recovered to 10,900/mm³, 9.2 g/dL and 167,000/mm³, respectively. Her TAT increased to 28.2 µg/l. Increased serum alpha-fetoprotein level (840 ng/ml; 3.0 multiple of median) was observed. The concentration of serum sialyl Tn (STN) was normal (22.0 U/ml). There were no serologic findings for viral infections, and no abnormal findings were observed in her peripheral vessels. Based on these findings, she was suggested to be a case of acute fetomaternal transfusion. Fetal cardiocograms showed a reactive pattern and the ultrasonographic findings showed normal AFI. However, a relatively hypoechoic layer between the chorionic plate and remainder of the placenta suggesting subchorionic hemorrhage was noted.

At 32 weeks and 6 days' gestation, a female infant weighing 2,026 g was delivered by cesarean section because of increased maternal blood pressure (190/110 mmHg). The placenta weighed 530 g. Microscopically, subchorionic infarction and hemorrhage of the placenta were confirmed. The baby's blood type was B, Rh positive, and the hemoglobin level of the umbilical vein was 13.6 g/dl. Thirty days after the surgery, the patient's serum alpha-fetoprotein levels decreased to normal.

Discussion

Placental abnormalities such as intravillous throm-

bosis have been reported to be associated with elevated maternal serum alpha-fetoprotein level, which indicates the presence of fetomaternal transfusion due to a breakdown of the placental barrier^{3,4}. Fetomaternal transfusion with the presence of maternal-fetal ABO incompatibility such as the current case resulted in the destruction of fetal blood cells in the maternal circulation⁵. In most pregnancies, a few red blood cells enter the maternal circulation, but the occurrence of maternal transfusion reaction due to fetomaternal transfusion such as that in the current case is rare. In our previous 4 case reports on massive fetomaternal transfusion⁶, only one patient, whose blood type was different from that of her baby (unpublished data), noted 'unexplained' nausea. In this case, in addition, the normal level of STN in the serum might indicate the absence of amniotic fluid embolism. Fortunately, this case was not severe because of the normal hemoglobin level of the umbilical vein. In patients with unexplained nausea, however, examinations for fetomaternal transfusion and placental abnormalities should be performed.

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