—Reports on Experiments and Clinical Cases—

Prenatal diagnosis of acute massive fetomaternial hemorrhage

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Abstract

We present here 2 cases of acute and 2 cases of chronic massive fetomaternial hemorrhage. A sinusoidal fetal heart rate pattern may indicate chronic fetomaternial hemorrhage, but, when increased variability is observed in fetal monitoring, maternal hemoglobin F should be measured to exclude acute fetomaternial hemorrhage. (J Nippon Med Sch 1999; 66: 266—269)

Key words: fetomaternial hemorrhage, sinusoidal fetal heart rate pattern, increased variability

Massive fetomaternial hemorrhage (FMH) has been defined as bleeding of greater than 150 mL of fetal blood in the maternal circulation, which can cause fetal anemia or perinatal death. Some investigations concerning the prenatal diagnosis of FMH have been reported. For example, a sinusoidal fetal heart rate (SHR) pattern has been reported to occur frequently in FMH.

We recently encountered 4 cases of massive FMH that occurred in uncomplicated pregnancies. We performed retrospective examinations to assess the clinical characteristics for prenatal diagnosis of massive FMH.

Case Reports

The medical and genetic family and past histories of the patients and their husbands were unmarked. During the pregnancies, there were no complications such as toxemia of pregnancy, gestational diabetes, maternal anemia, trauma or blood type incompatibility as massived by Coombs’ test. The estimated FMH volumes in these cases were calculated using Kleihauer’s or Mollison’s formulae.

Case 1: A 22-year-old woman, gravida 2, para 0, was admitted for induction of labor at 41 weeks and 3 days’ gestation. On admission, the patient felt no labor pain and noted decreased fetal movement. The cervix was found to be undilated and uneffaced. Fetal heart rate (FHR) monitoring showed a SHR pattern and variable decelerations. A cesarean was performed immediately. A male infant weighing 3,498 g was delivered, with Apgar scores of 8 and 8 at 1 and 5 minutes, respectively. The infant was very pale. The arterial pH, hemoglobin level, hematocrit and reticulocytes of the infant were 7.19, 5.5g/dL, 18% and 28%, respectively. The erythroblast count per 100 white blood cells was 626. At this time, the maternal hemoglobin F (HbF) and α-fetoprotein (α-FP) were 40% and 2,100 ng/mL, respectively. The estimated FMH volume was 180 mL.

Case 2: A 26-year-old woman, gravida 1, para 0, visited the hospital because of a marked decrease in fetal movement at 39 weeks and 3 days’ gestation. FHR monitoring showed increased long-term variability of the fetal heart rate (base line = 120~130 bpm) and variable decelerations. The patient was immediately admitted and a cesarean was performed. A pale female infant weighing 3,352 g was delivered, with Apgar scores of 0 and 1 at 1 and 5 minutes, respectively. The arterial pH, hemoglobin level, hematocrit and reticulocytes of the umbilical cord were 6.63, 3.5 g/dL, 12% and 3.0%, respectively. The infant died 10 hours
after delivery. At this time, the maternal HbF and α-FP were 4.9% and 4,400 ng/mL, respectively. The estimated FMH volume was 220 mL.

**Case 3.** A 30-year-old woman, gravida 1, para 0, was admitted during labor at 41 weeks and 3 days’ gestation. FHR monitoring showed sudden prolonged bradycardia following increased variability of the fetal heart rate (base line = 120 bpm). At this time, the contractions became fairly regular at 3- to 5- minute intervals of 30 seconds’ duration. The cervix was found to be 6–7 cm dilated, 70% effaced and the membranes were intact. A cesarean was performed immediately. A pale female infant weighing 3,638g was delivered, with an Apgar score of 0 at 1 minute. She died soon after delivery. The arterial hemoglobin level and hematocrit of the umbilical cord were 2.2 g/dL and 7.5%, respectively. At this time, the maternal HbF and α-FP were 6.2% and 640 ng/mL, respectively. The esti-
Fetal heart rate monitoring in case 3 (30 mm/min). Sudden prolonged bradycardia following increased variability of fetal heart rate (base line=120 bpm) is observed.

Fig. 3

Case 4: A 28-year-old woman, gravida 1, para 0, was referred to our hospital at 36 weeks and 0 days’ gestation because of the absence of fetal movement and intrauterine fetal death was diagnosed. At 35 weeks’ gestation, the patient felt nauseous, noted decrease fetal movements and visited her family doctor. At this time, FHR monitoring showed a SHR pattern and late decelerations. At 36 weeks and 3 days’ gestation, a pale female infant weighing 2,200g was delivered, and the arterial hematocrit of the umbilical cord was 10%. At this time, the maternal HbF and α-FP were 3.8% and 645 ng/mL, respectively. The estimated FMH volume was 170 mL.

The maternal HbF decreased to normal levels (<0.5%) 2-3 months after delivery in these 4 cases.

Discussion

In this study, none of the pregnant women were considered to be at high risk for FMH according to previously reports. FMH of 30 mL or more has been reported to occur in 3 of 1,000 normal pregnancies. When the FMH is 150 mL or more, severe fetal anemia with the absence of long and short-term variability or fetal death results. In this study, all cases were diagnosed as having massive FMH resulting in severe neonatal anemia or perinatal death. Few studies have investigated the maternal transfusion reaction due to massive FMH. In this study, for example, only 1 patient noted ‘unexplained’ nausea. Thus, selective or
routine screening of the fetal condition for FMH such as continuous FHR monitoring is needed.

Decreased or absent fetal movement and a SHR pattern have been reported to be useful in diagnosing massive FMH. The SHR pattern is well documented to occur with severe fetal anemia. In this study, a SHR pattern was observed in cases 1 and 4, while increased variability with decelerations was observed in cases 2 and 3. Chronic fetal blood loss was suggested in case 1, because increased fetal reticulocytes and erythroblasts were observed. On the other hand, acute blood loss was suggested in case 2 based on the reticulocyte level. Based on our findings, acute fetal blood loss during labor was suspected in case 3, while chronic fetal bleeding was suggested in case 4. In addition, the mother did not note decreased fetal movement in case 3. Increases in variability of response to acute nonacidemic hypoxia have been reported. Acute asphyxia causes stimulation of the autonomic nervous system leading to fetal distress. Thus, a SHR pattern may indicate chronic FMH only. Recently, possible interventions such as intrauterine transfusion for therapy of massive FMH have been reported. However, in cases of acute FMH such as in cases 2 and 3, it may be impossible to perform these interventions. Therefore, when increased variability is observed in FHR monitoring, acute FMH should be excluded as soon as possible.

Zipursky et al. reported that the volume and incidence of fetal red cells in the maternal circulation are greater in primiparous than in multiparous. Our results may support their study. The increased intrauterine pressure associated with rigidity of the cervix may contribute to this increased incidence of FMH in primiparas. However, contradictory conclusions have also been reported. In case 3, the cervix was 6–7 cm dilated and 70% effaced when FMH occurred. Thus, further examinations are needed to clarify the mechanism for FMH.

In conclusion, our observations of 2 cases of acute and 2 cases of chronic massive FMH indicate that when increased variability is observed in FHR monitoring, maternal HbF should be measured to exclude acute fetomaternal hemorrhage.

References


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