—Report on Experiments and Clinical Cases—

Massive Subchorionic Hematoma (Breus' Mole) Complicated by Intrauterine Growth Retardation

Naoko Nishida[1], Shunji Suzuki[1], Yukie Hamamura[1], Kenji Igarashi[1], Zuisei Hayashi[1], Rintaro Sawa[1], Yoshio Yoneyama[1], Hirobumi Asakura[1], Ken Kawabata[2], Yoshio Shima[2], Sumio Shin[3] and Tsutomu Araki[1]

[1]Department of Obstetrics and Gynecology, Nippon Medical School
[2]Department of Pediatrics, Katsushika Red Cross Maternity Hospital
[3]Department of Obstetrics and Gynecology, Katsushika Red Cross Maternity Hospital

Abstract

We present here a case of massive subchorionic hematoma complicated by intrauterine growth retardation and oligohydramnios diagnosed at 22 weeks' gestation. The patient was managed with the following medications: (1) tocolysis with ritodrine infusion, (2) 10% maltose infusion therapy (1500 mL/day), (3) antibiotic infusion (cefotaxim sodium, 2 g/day×7) and (4) kampo therapy with Sairei-to until delivery. At 33 weeks and 0 days' gestation, a female baby weighing 1,342 g was delivered without complication by caesarean section. During surgery, an escape of about 500～600 g of dark brown blood with no clots was noted from the subchorionic space of the placenta. Examination of the placenta showed a large fibrosis with well-defined margins on the fetal surface. (J Nippon Med Sch 2001; 68: 54—57)

Key words: massive subchorionic hematoma, intrauterine growth retardation

Introduction

Massive subchorionic hematoma (MSH), which was first described by Breus[1] in 1892 (Breus' mole), is a serious condition in pregnancy that is frequently complicated by perinatal abnormalities such as intrauterine growth retardation (IUGR) and intrauterine fetal death (IUFD). In 29 cases of MSH at 18～39 weeks' gestation reported by Shimizu et al.[2], 11 cases (38%) resulted in abortion or IUFD, and 12 cases (41%) resulted in light-for-detes. We encounted a case of MSH complicated by IUGR and oligohydramnios diagnosed at 22 weeks' gestation and present here the details of the case.

Case Report

A 28 year-old primigravida was found to have placental thickening and oligohydramnios on ultrasound scan at 22 weeks' gestation. The medical and genetic family and past histories of the patient and her husband were unremarkable. The patient does not smoke. During the first trimester of the pregnancy, there were no complications such as infection, anemia or trauma in the patient. At 23 weeks' gestation, ultrasonographic examination revealed symmetric growth retardation: both the biparietal diameter and femoral length were − 1.5 SD. The fetal head circumference (HC) was also − 1.2 SD, as shown in **Fig. 1**. The amniotic fluid index (AFI) was 4.5 cm. A large homogenous

Correspondence to Shunji Suzuki, MD, Department of Obstetrics and Gynecology, Nippon Medical School, 1−1−5 Sendagi, Bunkyo-ku, Tokyo 113−8603, Japan.
Journal Website (http://www.nms.ac.jp/jnms/)

and hyperechoic zone (13×10 cm) was detected at the lower pole of the placenta (**Fig. 2**), while the insertion of the umbilical cord was recognized at the upper pole of the placenta. Pulsed Doppler examinations of the umbilical artery and of the fetal middle cerebral artery were normal. No blood flow was present in the hyperechoic zone of the placenta. The patient felt irregular uterine contractions. Her hematocrit was 31%, platelet count was 204,000/mm^3, antithrombin (AT)-III was 92.3%, hemoglobin F (HbF) was 1.1% (normal: 0.0～0.7%), C-reactive protein was <0.4 mg/dL, cervical granulocyte elastase was 4.59 μg/mL (normal: < 1.60), anticardiolipin antibody was negative, and antinuclear antibody was positive (×320).

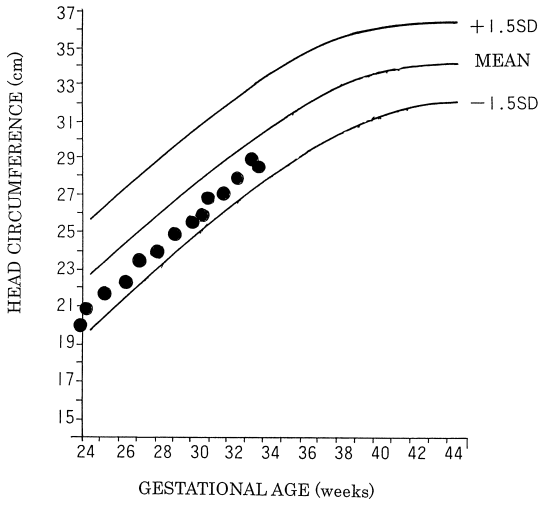

Fig. 1 The change in fetal head circumference plotted on an appropriate reference range (±1.5 SD)

The patient was admitted the following day and gave informed consent to be placed on the following medications: (1) tocolysis with ritodrine infusion until delivery (50 μg/min), (2) 10% maltose infusion therapy[3] until delivery (1,500 mL/day), (3) antibiotic infusion (cefotaxim sodium, 2 g/day×7) and (4) kampo therapy with Sairei-to until delivery (8.1 g/day, *per os*). Magnetic resonance imaging was performed at 24 weeks 3 days' gestation, and the lower portion of the placenta showed heterogenous low-intensity on T_2-weighted images, suggesting a massive hematoma of about 370 cm^3 (=0.52×10×9×8 cm, **Fig. 3**). Weekly ultrasound examinations, including Doppler studies of umbilical arteries and fetal middle cerebral arteries, were performed, and the fetus was monitored daily with cardiotocograms.

The patient was managed conservatively for 9 weeks, and the fetal HC grew compatible with about −1.2 SD of the dates (**Fig. 1**), although fetal growth retardation worsened. The ultrasonographic findings of the placenta, the AFI and the Doppler examinations did not change significantly. The fetal cardiotocograms showed a reactive pattern. At 32 weeks' geatation, the patient's hematocrit was 28%, platelet count was 185,000/mm^3, AT-III was 81.9%, HbF was 0.7%, C-reactive protein was <0.4 mg/dL, and antinuclear antibody was×80.

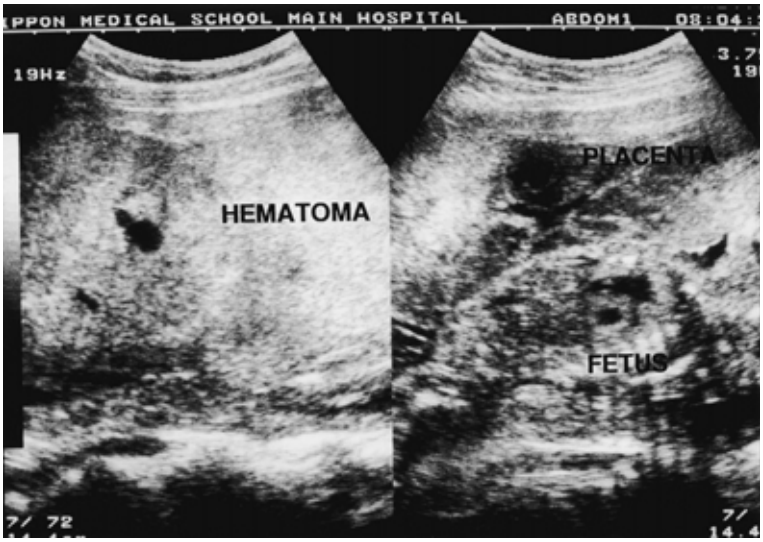

Fig. 2 A large homogenous and hyperechoic zone detected at the lower pole of the placenta at 23 weeks' gestation by ultrasonography

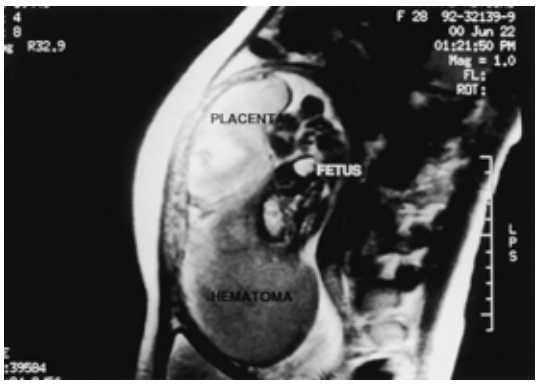

Fig. 3 Magnetic resonance imaging of the palcenta at 24 weeks' gestation. The lower portion of the placenta showed heterogenous low-intensity on T_2-weighted images.

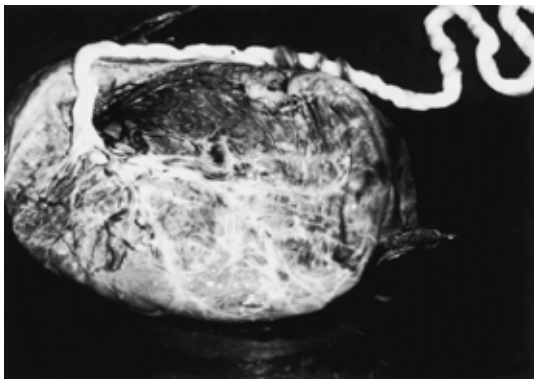

Fig. 4 The fetal surface of the placenta showing a large fibrosis of 16×5 cm with well-defined margins. The fibrosis did not extend beneath the insertion of the umbilical cord.

At 33 weeks and 0 days' gestation, a female baby weighing 1,342 g (−3.0 SD) was delivered without complication by caesarean section because of regular uterine contractions, IUGR and oligohydramnios. The amniotic water was clear. During manual removal of the placenta, an escape of about 500～600 g of dark brown blood with no clots was noted from the subchorionic space of the placenta. The placenta measured 20×19×4 cm and weighed 790 g. Examination of the placenta showed a large fibrosis of 16×5 cm, which did not extend beneath the insertion of the umbilical cord, with well-defined margins on the fetal surface (**Fig. 4**). Several branches of the umbilical vessels ran along the surface of the fibrosis. At the base of the fibrosis, there was a subchorionic space in which we speculate a large amount of blood was stored (**Fig. 5**). Microscopically, the subchorionic infarction and hematoma of the placenta were confirmed.

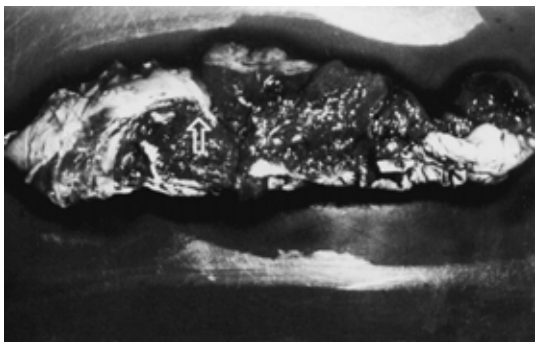

Fig. 5 The cut surface of placenta showing a subchorionic space at the base of the fibrosis.

Discussion

In this study, the sonographic features of the involved placenta were perfect for a correct diagnosis of MSH[4-6]. Although the reported frequency of MSH is very low, some cases of IUGR associated with MSH have been investigated[4-6]. The mechanism of IUGR has been suggested, at least in part, to be due to uteroplacental insufficiency from the MSH. Thomas et al.[4] observed the presence of redistribution in the fetoplacental circulation in their two cases of MSH by using a Doppler study. In the current case, on the other hand, the Doppler examinations were normal except for the presence of MSH, which might be because the MSH did not extend beneath the umbilical cord. The location of the MSH has been suggested to impede the cord insertion[5,7]. The MSH of maternal blood in the intervillous space can easily compress the umbilical vessels. The findings in this case may support the porsibility of these mechanisms. If MSH is found on ultrasound examination, we believe that the insertion portion of the umbilical cord should be examined immediately.

Tocolysis and antibiotic therapy are common for the management of MSH, because subchorionic hematoma has been reported likely to result in abortion or premature delivery associated with chorioamnionitis[8]. In the current case, kampo therapy with Sairei-to and 10% maltose infusion therapy were also performed. In 1991, Baxi and Pearlstone[9] suggested that the presence of antibodies is an etiologic factor for subchorionic hematoma of the placenta. The antibodies have

been suggested to increase the tendency of platelets to aggregate, which leads to thrombosis and/or vasculitis and thereby to an increased possibility of subchorionic hematoma. In Japan, Sairei-to has been suggested to have an effect like corticosteroids, and is therefove used for the treatment of patients with a poor obstetric history such as habitual abortion[10]. In this case, the antinuclear antibody decreased from (×320) to (×80) with kampo therapy using Sairei-to, while intravenous infusion of 10% maltose is a technique for the treatment of high risk pregnancies that has been used for the past 18 years in Japan[11]. Maltose infusion not only increases the supply of glucose to the fetus in the absence of fetal insulinogenesis, but also improves the metabolism of amino acids and the circulation in growth-retarded fetuses[3,11]. In the current case, the findings of fetal HC and Doppler studies may support these previous studies.

In the current case, an increased maternal HbF was also observed on admission, indicating the presence of fetomaternal hemorrhage (FMH)[12,13]. There were no typical fetal symptoms of FMH such as decreased or absent fetal movement or a sinusoidal heart rate pattern in cardiotocograms because the onset of MSH might have been during the first trimester of pregnancy. No maternal transfusion reaction due to FMH was observed. MSH is a condition in which a large amount of blood mainly of maternal origin collects and separates the chorionic plate from the villous chorion[6]. Thus, further examination is needed of this mechanism.

In Summary, we present a case of MSH complicated by IUGR and oligohydramnios diagnosed at 22 weeks' gestation. Sairei-to and 10% maltose infusion with tocolysis and antibiotic therapy may be useful in the management of MSH.

References

1. Breus C: Das Tuberose Subchoriale Haematom der Decidua. Leipzig, Deuticke, 1892.
2. Shimizu I, Shikado K, Mituda N, Iwata M, Nagata M, Fuke S, Bekku S, Wasada K, Kawamoto A, Nakayama M, Suehara N: Ultrasonographic findings in massive subchorionic hematoma (Breus' mole). Ultrasound Obstet Gynecol 1995; 6(suppl): 163.
3. Suzuki S, Mine K, Sawa R, Yoneyama Y, Araki T: 10% maltose infusion therapy for oligohydramnios. Aust NZ J Obstet Gynecol 1999; 39: 373–375.
4. Thomas D, Makhoul J, M, ller C: Fetal growth retardation due to massive subchorionic thrombohematoma: report of two cases. J Ultrasound Med 1992; 11: 245–247.
5. Tam W, Fung HY, Fung T, Lau T, To K: Intra-uterine growth retardation and transverse lie due to massive subchorionic thrombohematoma and overlying large subchorionic cyst. Acta Obstet Gynecol Scand 1997; 76: 381–383.
6. Richards DS, Bennett B: Prenatal ultrasound diagnosis of massive subchorionic thrombohematoma. Ultrasound Obstet Gynecol 1998; 11: 364–366.
7. Kirkinen P, Jouppila P: Intrauterine membranous cyst: A report of antenatal diagnosis and obstetric aspects in two cases. Obstet Gynecol 1986; 67: 26 S–30 S.
8. Seki H, Kuromaki K, Takeda S, Kinoshita K: Persistent subchorionic hematoma with clinical symptoms until delivery. Int J Gynaecol Obstet 1998; 63: 123–128.
9. Baxi LV, Pearlstone MM: Subchorionic hematomas and the presence of autoantibodies. Am J Obstet Gynecol 1991; 165: 1423–1424.
10. Kano T: Kampo therapy for habitual abortion (in Japanese). Obstetrical and Gynecological Therapy 1997; 75: 538–540.
11. Chimura T, Funayama T, Mituu T, Kaneko N: Effect of infusion therapy on intrauterine fetal growth retardation. Acta Obst Gynec Jpn 1982; 34: 551–558.
12. Giacoia GP: Severe fetomaternal hemorrhage: A review. Obstet Gynecol Surv 1997; 52: 372–380.
13. Ohshita T, Suzuki S, Sawa R, Yoneyama Y, Asakura H, Araki T: Prenatal diagnosis of acute massive fetomaternal hemorrhage. J Nippon Med Sch 1999; 66: 266–269.

(Received, September 8, 2000)
(Accepted, September 26, 2000)