症例報告

AN INFANT WITH HEART FAILURE AND RESPIRATORY DISTRESS SYNDROME DELIVERED BY EMERGENT CESAREAN SECTION WITH PLACENTA PREVIA TOTALIS

TOSHIYA NISHIKUBO, TETSUYA SAKAUE, YUMIKO UCHIDA, KEIJI NOGAMI, ISAO KUWAHARA, *HIDEKI MINOWA and HIDEKAZU KAMITSUJI

Neonatal Intensive Care Unit, Perinatal Medical Center, Nara Prefectural Hospital
*Neonatal Intensive Care Unit, Nara Medical University

Received May 8, 1998

Abstract: We report an infant with heart failure and respiratory distress syndrome (RDS) born by emergent cesarean section due to placenta previa totalis after massive vaginal bleeding. The patient was at 31 gestational weeks, and birth weight was 1785 g. He was admitted to our neonatal intensive care unit due to respiratory failure and low birth weight. However, he also demonstrated severe heart failure. Blood pressure was 44/25 mmHg on admission and the low blood pressure continued following dopamine administration. He was administered artificial pulmonary surfactant (S-TA) soon after admission, but respiratory failure did not sufficiently improve. In addition to dobutamine, S-TA was administered two more times, 6 and 16 hours after birth. Furthemore, in order to alleviate respiratory failure, high frequency oscillation was started at 16 hours. Then his general condition improved gradually and he was extubated 27 days after birth. In conclusion, infants delivered by emergent cesarean section due to placenta previa totalis with massive vaginal bleeding may demonstrate severe heart failure and require more intensive respiratory and circulatory support than standard RDS infants.

(奈医誌. J. Nara Med. Ass. 49, 261~265, 1998)

Key words: placenta previa totalis, infant, neonatal asphyxia, heart failure, respiratory distress syndrome

Placenta previa totalis is a common obstetric disease which necessitates cesarean section for delivery. If vaginal bleeding occurs, emergent cesarean section should be performed to prevent massive bleeding from the disrupted placenta. Cesarean section is one of the predisposing factors of respiratory distress syndrome (RDS)¹⁾. In addition, placenta previa totalis itself is also one of the risk factors for RDS²⁾. However, few studies have reported infants complicated with severe heart failure, in addition to RDS, born by emergent cesarean section due to placenta previa totalis after massive vaginal bleeding.

We examined here an infant with severe heart failure and RDS born by emergent cesarean section after massive vagilal bleeding due to placenta previa totalis.

CASE REPORT

The pregnant woman was diagnosed with placenta previa totalis at 28 weeks of gestation by fetal echography. She was transferred to our hospital due to massive vaginal bleeding associated with uterine constriction at 31 weeks of gestation. Massive vaginal bleeding of more than 170 ml on admission due to placenta previa totalis continued after admission. Non-stress test of the fetus did not reveal fetal distress on admission. Emergent cesarean section was performed, and a male infant, body weight 1785 g, Apgar score at 1 and 5 minute 6 and 8, respectively, was delivered. He was intubated soon after birth due to severe cyanosis and respiratory retraction. Chest X-ray film revealed a reticulogranular pattern with air bronchogram (Fig. 1-1), microbubble stable test using gastric residue showed a weak pattern, and he was diagnosed with RDS³).

Blood pressure and heart rate on admission were 44/25 mmHg and 180 beats/min, respectively. Though echocardiography could not detect any congenital heart disease, ejection fraction (EF) decreased to 45 % on admission. Blood gas analysis revealed moderate metabolic acidosis on admission (Table 1). Lactate dehydrogenase (LDH) and creatinine kinase (CK) were not markedly elevated. There was no sign of infection on peripheral blood analysis or blood culture.

Artificial pulmonary surfactant (S-TA, Surfacten, 120 mg/kg) was administered soon after the diagnosis of RDS. Dopamine (5 micrograms/kg/min) was started (Fig. 2), and total water intake was restricted to 60 ml/kg/day. The respiratory condition improved on blood gas analysis 6 hours after birth; however, chest X-ray findings did not improve (Fig. 1-2). Though a second administration of S-TA was performed in addition to dobutamine (3 micrograms/kg/min) and hydroxyl carbonate, blood gas analysis gradually worsened. A pulmonary air leak

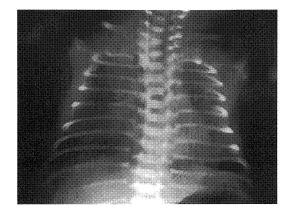
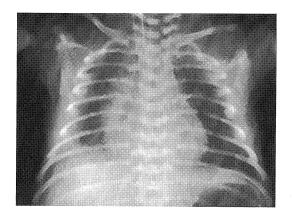


Fig. 1-1. Chest X-ray on admission.

Table 1. Laboratory findings on admission

Peripheral Blood			Immunological test		
RBC 3.	87×10^{6}	$/\mu$ 1	CRP	0.2	mg/dl
Hb	Hb 14.2 g/dl		Blood chemistry		
Ht	44.8	%	T. Bil	3.8	mg/dl
Plt 389×10^3 / μ l		GOT	65	IU/l	
WBC	13600	$/\mu$ 1	GPT	7	IU/l
Eos	0		LDH	952	IU/l
Bas	1		CK	275	IU/l
St	12		ALP	533	IU/l
Seg	50		T. P.	4.4	mg/dl
Lym	30		Alb	2.7	mg/dl
Mon	7		BUN	19	mg/dl
Blood gas analysis			CRN	0.9	mg/dl
pН	7.238		FBS	77	mg/dl
PCO_2	48.5	mmHg	Na	131	mEq/l
PO_2	75.4	mmHg	K	5.3	mEq/l
BE	-7.6	mmol/l	Ca	6.7	mg/dl
HCO_3	20.1	mmol/l			
heel prick blood			Blood culture: negative		
PIP/PEEP=25/4 cm H ₂ O			Ulynalysis : n. p.		
IMV = 40, $Ti = 0.6$,			Microbubble test: weak		
$FiO_2=1.0$					

was detected on chest X-ray 16 hours after admission (Fig. 1-3). Low blood pressure continued as 44/26 mmHg and 45/26 mmHg 6 hours and 16 hours after birth, respectively. EF did not normalize 16 hours after birth in spite of the administration of catecholamines. Mechanical



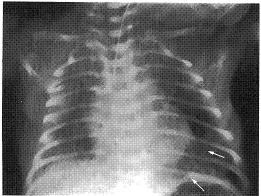


Fig. 1-2. Chest X-ray 6 hours after birth.

Fig. 1-3. Chest X-ray 16 hours after birth. Bulla was detected in the left lung (arrow).

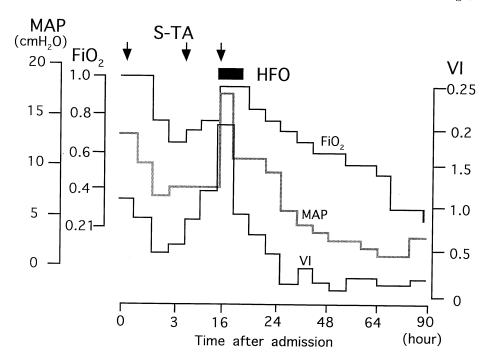


Fig. 2. Respiratory time course after admission.

MAP: mean airway pressure FiO_2 : fraction of inspired oxygen

VI: ventilatory index

S-TA: artificial pulmonary surfactant (Surfacten)

HFO: high frequency oscillation

ventilation changed from intermittent mandatory ventilation to high frequency oscillation (HFO) 16 hours after birth with a third replacement therapy of surfactant to improve respiratory condition and to prevent distended pneumothorax. After these treatments, respiratory and circulatory conditions gradually improved without the complication of distended pneumothorax. He was extubated 27 days after birth.

DISCUSSION

Abruptio placenta and placenta previa totalis are common and serious placental abnormalities causing massive vaginal bleeding of pregnant women in the third trimester⁴). It is well known that abruptio placenta causes severe problems for the mother and fetus such as massive maternal blood loss, maternal coagulation failure, neonatal asphyxia and stillbirth^{4,5}). Despite placenta previa totalis also causing perinatal disorders⁶), the problems for the fetus do not seem as severe as with abruptio placenta.

Infants born from placenta previa totalis are reported to be high risk infants for RDS^{2,7)}, preterm birth⁸⁾, and cerebral palsy⁹⁾. However, few studies have reported infants complicated with heart failure due to placenta previa totalis.

In our case, the infant demonstrated both RDS and heart failure. The therapy for RDS is established to be the administration of artificial pulmonary surfactant^{10,11}. In standard cases of RDS infants after surfactant-replacement therapy, respiratory failure improves within 12 hours on blood gas analysis and chest X-ray findings. In our case, blood gas analysis improved transiently 2 hours following the worsening of both blood gas analysis and chest X-ray findings. If the efficacy is not sufficient, multiple-dose surfactant replacement therapy is reported to be useful¹². Despite a second replacement therapy, respiratory distress did not improve on blood gas analysis or chest X-ray analysis, and the problem in our case was thought not to be just the deletion of pulmonary surfactant.

He was diagnosed with heart failure because of the refractory low blood pressure and low ejection fraction. For treatment of heart failure, dopamine was administered on admission and dobutamine was supplemented¹³. The heart failure was suspected to have occurred just before delivery because there were no marked increases of LDH or CK on admission. Therefore, the heart failure was likely due to hypoxia induced by desperation of the placenta as a result of placenta previa totalis. The ineffectiveness of S-TA was suspected to be related to heart failure. Low heart output may induce the increase of intra-pulmonary arterio-venous shunt, resulting in insufficient pulmonary oxygenation.

HFO for infants with severe respiratory failure has been assessed^{14–16)}. HFO may improve respiratory failure in infants who do not respond to conventional mechanical ventilation¹⁶⁾. However, pulmonary air leak and respiratory failure indicating early improvement in respiratory status were not effective when HFO was used as rescue treatment¹⁴⁾. In our case, HFO with surfactant replacement as rescue therapy improved the blood gas analysis. However, it is difficult to conclude the efficacy of HFO for infants complicated with RDS and heart failure.

In conclusion, infants delivered by emergent cesarean section due to placenta previa totalis with massive vaginal bleeding may demonstrate severe heart failure and need more intensive respiratory and circulatory support than standard RDS infants.

REFERENCES

- Martin, R. J., Fanaroff, A. A. and Skalina, M. E.: The respiratory distress syndrome and its management. In Behrman's Neonatal Perinatal Medicine. Mosby, Missouri, pp427-443, 1983.
- 2) **Niswander, K. C.**: Obstetric factors related to prematurity. In the Epidermilogy of Prematurity. Stanley, Baltimore, pp249-264, 1977.
- 3) Chida, S., Fujiwara, T., Konishi, M., Shimada, S. and Takahashi, A.: Surfactant proteins and stable microbubbles in tracheal aspirates of infants with respiratory distress syndrome: relation to the degree of respiratory failure and response to exogenous surfactant. Eur. J. Pediatr. 156: 131-138, 1997.
- 4) Cunningham, F. G., MacDonald, P. C., et al. eds: Williams obstetrics. 20th ed., Appleton & Lange, Connecticut, pp745-782, 1997.
- 5) Letsky, E.: Medical disorders during pregnancy. (Barron. W. M. and Lindheimer. M. D., eds,). Mosby Year Book, St. Louis, pp272-322, 1990.
- 6) **Agdestein. S.**: Perinatal and infant mortality: trends and risk factors in Norway 1967-90. Acta Obstet. Gynecol. Scand. Suppl. **160**: 1-30, 1994.
- 7) Matsuda, Y., Ikenouue. T., Hokanishi, H.: The perinatal factors associated with the development of neonatal RDS in cases of placenta previa. Nippon Sanka Fujinka Gakkai Zasshi 38: 903-908, 1986.
- 8) Chen, C. P., Wang. K. G., Yang, Y. C. and See, L. C.: Risk factors for preterm birth in an upper middle class Chinese population. Eur. J. Obstet. Gynecol. Reprod. Biol. 70: 53-59, 1996.
- 9) Petridou, E., Koussouri. M., Toupadaki, N., Papavassiliou, A., Youroukos, S., Katsarou, E. and Trichopoulos, D.: Risk factors for cerebral palsy: a case-control study in Greece. Scand. J. Soc. Med. 24:14-26, 1996.
- 10) **Fujiwara, T., Maeta, H., et al.**: Artificial surfactant therapy in hyaline membrane disease. Lancet 1: 55–59, 1980.
- 11) Horbar, J. D., Soll, R. F., Sutheland, J. M., et al.: A multicenter randomized, placebo-controlled trial of surfactant threrapy for respiratory distress syndrome. New Eng. J. Med. 320: 959-965, 1989.
- 12) **Dunn, M. S., Shennan, A. T.** and **Possmayer, F.**: Single-versus multiple-dose surfactant replacement therapy in neonates of 30 to 36 weeks' gestation with respiratory distress syndrome. Pediatr. 86: 564-571, 1990.
- 13) **Klarr, J. M., Faix, R. G., Pryce, C. J.** and **Bhatt Mehta, V.**: Randomized, blind trial of dopamine versus dobutamine for treatment of hypotension in preterm infants with respiratory distress syndrome. J. Pediatr. **125**: 117-22, 1994.
- 14) Chan, V., Greenough, A., Gamsu, H. R.: High frequency oscillation for preterm infants with severe respiratory failure. Arch. Dis. Child. Fetal Neonatal. Ed. 70: 44-46, 1994.
- 15) Sakai, T., Igarashi, Y., Aiba, S., Kuba, Y., Asanuma, A.: New interpretation and management of dry lung syndrome: a case report. Acta Paediatr. Jpn. 36: 510-4, 1994.
- 16) Ito, Y., Kawano, T., Miyasaka, K., Katayama, M., Sakai, H.: Alternative treatment may lower the need for use of extracorporeal membrane oxygenation. Acta Peadiatr. Jpn. 36: 673-7, 1994.