43. *Spontaneous Intrauterine Maternal-Fetal Blood Transfusion Causing Neonatal Polycythemia*

By Koichi YOSHIMURA,^{*)}^{**)} Sadao YAMANAMI,^{*)}^{**)}
Kenji NAKAJIMA,^{**)} Jun-ichi YANAGIDA,^{*)}
and Hayato HASEKURA^{*)}

(Communicated by Shoei ISEKI, M. J. A., March 12, 1977)

Introduction. Physiological hemoconcentration is observed in almost all normally delivered newborn infants. Their hemoglobin values and hematocrit levels are higher than in adults. In rare cases, however, pathologic neonatal polycythemia with highly concentrated blood is found, which is attributed to several causes including mother-child intrauterine transfusion.¹⁾

Diagnosis of the maternal-fetal transfusion causing neonatal polycythemia is often difficult. This paper presents two infants with maternal-fetal transfusion, judged from the examination of infants' immunoglobulins and blood groups.

Materials and methods. Hematocrit measurements were performed on 160 premature infants by the microhematocrit technic. Among the samples, polycythemic cases showing hematocrit values of 65% or greater were found in 15 premature babies.

Immunoglobulins were estimated by single radial immunodiffusions. Blood groups were examined on erythrocytes. Leucocytes were typed for HLA using the microcytotoxicity test, and red cell enzyme types were tested by starch gel electrophoresis.

Results. Immunoglobulin data from 15 polycythemic babies obtained within the fifth day of life are plotted in Fig. 1. Measurements of immunoglobulins IgM and IgA, which are usually negative in ordinary newborn infants, gave positive results in most infants except three cases.

Blood grouping was performed on seven of the 12 infants with unusually positive IgA or IgM level. Examinations of various blood groups revealed significant abnormalities in two cases (Table I).

Case 1: T.O., male. This infant was born at a gestational age of 32 weeks and weighed 1480 g. The pregnancy was normal. It developed respiratory distress with apnea and cyanosis. Treatment was started with oxygen and transfusion. Hyperbilirubinemia neces-

^{*)} Saitama Medical School, Moroyama, Saitama, Japan.

^{**)} Toshima Metropolitan Hospital, Itabashi, Tokyo.

Fig. 1. Immunoglobulin levels of hypercythemic cases.

N.L.: Normal limit. 1: Case 1, 2: Case 2.

sitated the exchange transfusion and phototherapy on the fourth day of life. At this time the hemoglobin value was 21.6 g/dl; hematocrit, 72%; erythrocyte count, $6.3 \times 10^{12}/l$; platelet count, $226.8 \times 10^9/l$. The serum bilirubin value was 21.6 mg/dl. IgG value was 460 mg/dl; IgA 0 mg/dl; IgM 122 mg/dl without any sign of infections.

The mother's erythrocyte was Rh positive, R_1R_2 (Rh: 1, 2, 3, 4, 5). The infant's erythrocytes had been typed before the exchange transfusion to be Rh positive, R_1R_2 (Rh: 1, 2, 3, 4, 5), the same type as the mother, with weak agglutinations with anti-rh' (Rh3, E) and anti-hr' (Rh4, c). His Rh type changed into R_1R_2 (Rh: 1, 2, -3, -4, 5) as reconfirmed at one month and ten months of age. In addition, Le^b of the Lewis blood groups, which is usually absent during the early neonatal period, was demonstrated suggesting a maternal-fetal transfusion. The absorption test was performed over again using anti- Le^b and the infant's cells with the same positive result.

Other blood groups of the mother were B, M, Le (a-b+), Fy (a+b-), Jk (a+b+), AP (BA), PGM (1).

Blood groups of the infant were B, M, Le (a-b+), Fy (a+b-), Jk (a-b+), AP (BA), PGM (1).

Case 2: M.O., male. This infant was born in full-term weighing 2,220 g. Pregnancy and labor were uncomplicated. He was found plethoric, but his condition was not serious. As serum bilirubin concentration was markedly increased to 21 mg/dl, exchange transfusion was performed on the fourth day of life. At this time the hemoglobin value was 23.3 g/dl; hematocrit 74%; erythrocyte count, $6.0 \times 10^{12}/l$; platelet count, $288.5 \times 10^9/l$, IgG value was 900 mg/dl;

IgA 0 mg/dl; IgM 20 mg/dl.

The mother's blood groups were O, Rh positive, R₁r (Rh: 1, 2, -3, 4, 5), MN, Le (a-b+), Fy (a+b-), Jk (a-b+), Di (a-b+), AP (B), PGM (1), HLA (A2A9, B5BW35).

The infant was O, Rh positive, R₁r (Rh: 1, 2, -3, 4, 5), MN, Le (a-b+), Fy (a+b-), Jk (a-b+), AP (B), PGM (2-1), HLA (A9, B5).

The presence of Le^b on the infant's red cells suggested maternal-fetal transfusion.

Discussion. Polycythemic syndromes have been reported in the field of pediatrics since 1960. To define neonatal polycythemia Kontras proposed the value above 20 g/dl for hemoglobin and 65% for hematocrit.¹⁾

Polycythemic infants show several pathologic symptoms as convulsion, cyanosis, jaundice, and respiratory distress.^{2),3)}

Syndromes of hyperviscosity, i.e., plethora, hypoglycemia, cyanosis, and central nervous manifestation, occur in relation to polycythemia. Thus, pathologic polycythemia can signify hyperviscosity syndrome. Causes of neonatal hyperviscosity have been summarized by Gross⁴⁾ and reported by others to be intrauterine blood transfusion from the mother,⁵⁾ from one of twins,⁶⁾ or from the placenta due to late clamping of the umbilical cord.⁷⁾

In order to confirm the diagnosis of maternal-fetal transfusion causing neonatal polycythemia, various methods have been used. Michael and Mauer⁵⁾ first demonstrated 3 cases plethoric with maternal transfusions through the Ashby agglutination technic. Presence of IgM or IgA in the serum, which are usually absent in the neonatal period, and a low fetal hemoglobin concentration for gestational age are the other ways to confirm the phenomenon.¹⁾ Erythrocytes labelled with radioisotope are used by Mengert *et al.*⁸⁾ to demonstrate the maternal cells in the newborn infant's blood.

Our aim was to use blood groups and other hematonomical markers for this purpose. Abnormalities of blood groups were recognized in only two of the 15 plethoric infants. In case 1, the infant revealed extra blood group antigens, Rh3 (rh'', E) and Rh4 (hr', c) of the Rh blood groups and Le^b of the Lewis blood groups, which were considered to derive from the mother. In case 2, the unusual presence of the Le^b antigen of the Lewis blood groups gave the evidence for the maternal transfusion. Although the Le^b antigen was not detected in the other hyperemic infants, possibility of a maternal-fetal transfusion cannot be denied, as the anti-Le^b serum used was not sufficiently strong. The enzyme groups and HLA groups

did not give any significant informations in the present study.

No treatments were needed in the other plethoric infants tested. In severe cases, however, partial or total exchange transfusions would be indicated in order to reduce the erythrocyte mass.

Summary. Spontaneous maternal-fetal transfusion was detected in two cases among 15 plethoric newborn infants through blood groups and immunoglobulin tests.

References

- 1) S. B. Kontras: Polycythemia and hyperviscosity syndromes in infants and children. *Pediat. clin. N. Amer.*, **19**, 919-933 (1972).
- 2) J. L. Wood: Plethora in the newborn infant associated with cyanosis and convulsion. *J. Pediat.*, **54**, 143-151 (1959).
- 3) R. A. Gatti, A. J. Muster, R. B. Cole, and M. H. Pole: Neonatal polycythemia with transient cyanosis and cardiorespiratory abnormalities. *J. Pediat.*, **69**, 1063-1072 (1966).
- 4) G. P. Gross, W. E. Hathaway, and H. R. McGaughey: Hyperviscosity in the neonate. *J. Pediat.*, **82**, 1004-1012 (1973).
- 5) A. F. Michael, and A. M. Mauer: Maternal-fetal transfusion as a cause of plethora in the neonatal period. *Pediat.*, **28**, 458-461 (1961).
- 6) A. R. Rausen, M. Seki, and L. Strauss: Twin transfusion syndrome. *J. Pediat.*, **66**, 613-628 (1965).
- 7) R. Usher, M. Shephard, and J. Lind: The blood volume of the newborn infant and placental transfusion. *Acta Pediat.*, **52**, 497-512 (1963).
- 8) W. F. Mengert, *et al.*: Placental transmission of erythrocytes. *Am. J. Obst. Gynec.*, **96**, 678-685 (1955).