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## Neonatal Polycythemia (Case Report)

*by*

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### Introduction

A venous hematocrit of greater than 65% or a venous hemoglobin concentration in excess of 22.0 gm/100 ml at any time during the first week of life should be considered as evidence of polycythemia (Oski and Naiman, 1972).

Many cases of polycythemia have been described in the literature. This condition has a variety of recognized etiology, although in many instances its etiology is obscure. Some of the recognized causes are twin to twin transfusion, maternal-fetal transfusion and delayed cord clamping i.e. intentional, unassisted home delivery.

The symptoms observed in the polycythemic infant appear to be primarily a consequence of the associated increase in blood viscosity. Symptoms and signs of neonatal polycythemia which have been described in the literature are as follows:

Lethargy, plethora, respiratory

distress, cyanosis, congestive heart failure, convulsion, priapism, jaundice, renal vein thrombosis, tetany, hyperbilirubinemia, hypoglycemia, hypocalcemia and thrombocytopenia. Most infants with polycythemia manifest no symptoms of any type (Gatti et al., 1966). However, once symptoms exist and hyperviscosity is demonstrated, a partial exchange transfusion with plasma to lower the viscosity should be considered (Kontrass, 1972). Whether asymptomatic children with polycythemia should also undergo exchange transfusion remains open to question (Oski and Naiman, 1972).

The purpose of this paper is to report a case of neonatal polycythemia thought to be caused by delayed cord clamping. Maternal-fetal transfusion is also a probable cause.

### Case Report

A 9-hour-old, first male twin, weight 2720 gm, length 48 cm, was ad-

mitted to the newborn nursery on June 11, 1973, with the following history:

This infant was born on June 11, 1973, at home; the delivery was assisted by an indigenous midwife. The mother was a healthy 23-year-old Indonesian woman gravida III, PII, T0. The first day of the mother's last menstrual period was undetermined. The cord was clamped 5 hours after birth.

The second twin, a male, weight 2590 gm, in transverse lie was born dead 9 hours after the delivery of the first child. The placenta weighed 1000 gm with monochorionic and monoamniotic membrane.

On admission the infant was plethoric but not cyanotic. Moro, grasp and suction reflexes were normal. No abnormalities were found on examination of the heart, lungs, abdomen or genitalia.

Capillary hemoglobin value on the first day of life was 22.3 gm%. Leukocyte count and differential count were normal. Early bottle feeding was started and was well tolerated.

On the second day the infant was noted to be severely ill, lethargic, plethoric and showed signs of respiratory failure. The extremities and face were edematous. Respiratory rate was 82/m, and of abdominocostal type. Pulse 150/m, regular, equal. No abnormalities were found on examination of the heart and lungs except a loud P2. The liver was palpable 3 cm under the costal margin with smooth surface, rounded edge and elastic in consistency.

Grasp, suction and Moro reflexes were weak. Venous hemoglobine and hematocrit value were respectively 22.8 gm% and 69%. The platelet count was 320.000/mm<sup>3</sup>. A rontgenogram of the chest revealed increased lung vascularity, slight enlargement of the heart to the left with heart thoracis ratio of 56%. An E. C.G. showed mild hypocalcemia with ventricular strain signs.

Polycythemia with cardiac failure was considered, a partial exchange transfusion was performed and the hematocrit was reduced from 69% to 60%. The formula of Oski and Naiman (1972) was used.

$$\text{Volume of exchange (ml)} = \frac{\text{Blood volume} \times (\text{observed Hct} - \text{desired Hct})}{\text{Observed Hct}}$$

$$\text{Blood volume} = 85 \text{ cc/Kg. B.W.}$$

$$\text{Volume of exchange} = \frac{85 \times 2.72 \times (0,69 - 0,60)}{0,69} = 30 \text{ ml.}$$

A gastric tube feeding was inserted into the umbilical vein with the location of the catheter tip in the inferior vena cava. Thirty millilitres of

blood were withdrawn and replaced with 30 ml of one fifth normal saline in 5% glucose in 5 ml. increments over a priod of 30 minutes. One fifth

of normal saline in 5% glucose was used because plasma was not available.

Besides partial exchange transfusion being done, the infant was digitalized with digoxin. The infant was also treated with penicillin procain, kantrex, calcium and luminal. Because of severe weakness, intravenous fluid drip of one fifth normal saline in 5% glucose and NaHCO<sub>3</sub> was also given.

The next day the infant showed much improvement, the respiratory rate fell within normal limits, plethora and cyanosis disappeared, only a slight icterus and praetibial edema were noted, which disappeared on the fifth day. A physical examination showed a normal infant with normal reflexes, no abnormalities of the heart and lungs were found. The liver was palpable 1 cm under the costal

margin with smooth surface, sharp edged and elastic in consistency. An E.C.G showed signs of digitalis effect but no signs of hypocalcemia. Tube feeding was begun on the third day of life and was gradually changed to bottle feeding.

#### Laboratory Findings.

Venous blood on the second day examination:

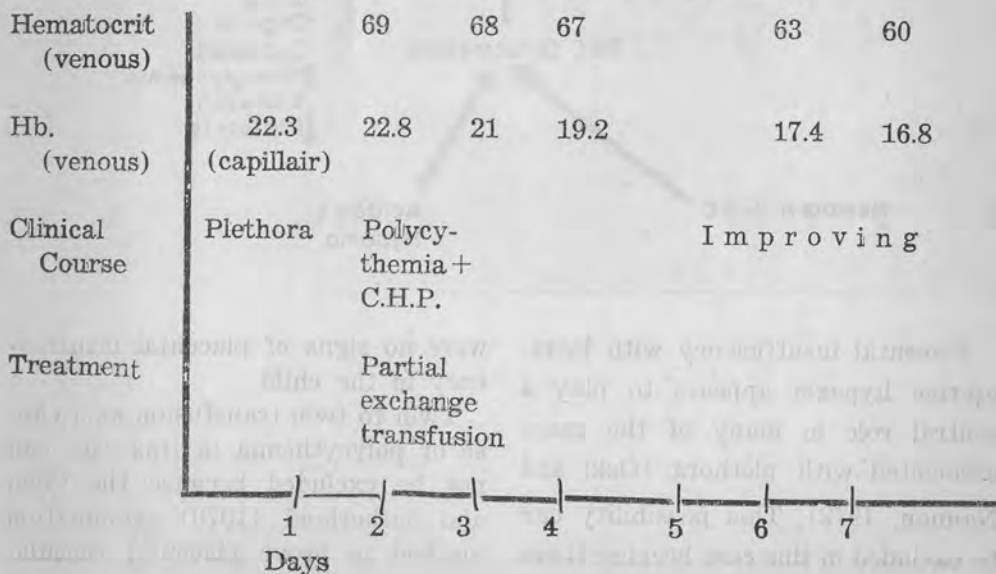
Bilirubin total 3.095 mg%, bilirubin direct 0.4 mg%, Ca. 7.6 mg%, glucose 50 mg%.

Blood culture was sterile.

On the third day: bilirubin total 5.93 mg%, bilirubin direct 0.593 mg%. Leukocyte and differential count were within normal limits.

Fetal Hb. : 33.1%.

The venous hematocrit was followed up until the seventh day of life as shown in Figure I.



Although the Hb. and hematocrit were still high on the following days after treatment, a partial exchange transfusion was not repeated.

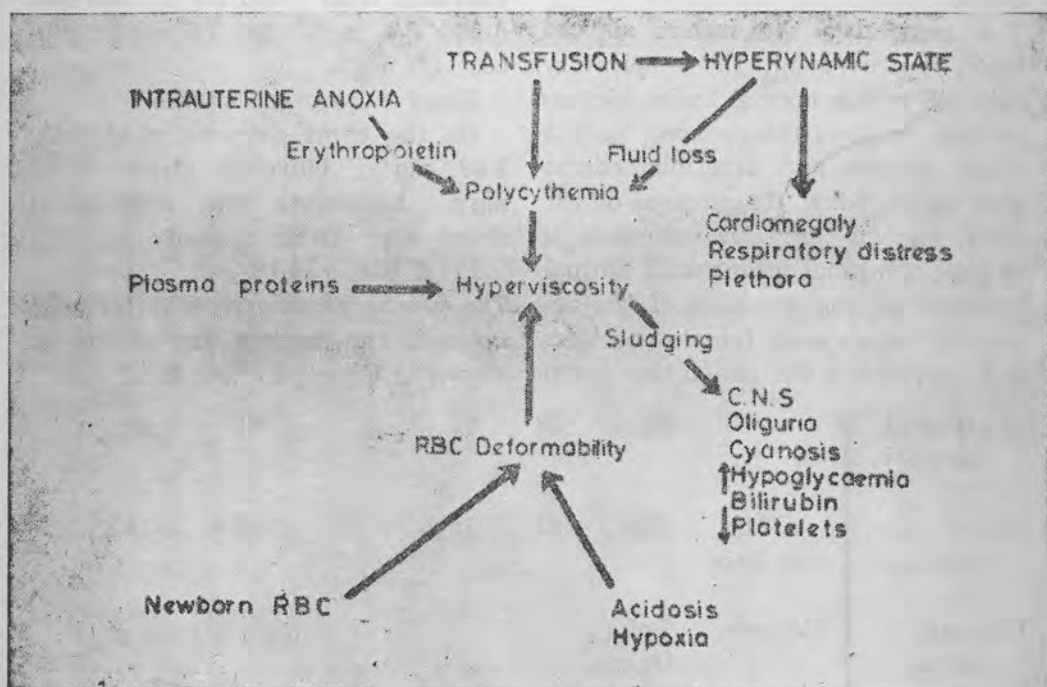
Clinically the infant showed no abnormal signs that would indicate a retreatment. Both venous Hb. and hematocrit decreased gradually. The results in this case are the same as reported by Wood (1959).

The infant was discharged on the 14th day of life in good condition.

### Discussion

The clinical observations and the laboratory findings made the diagnosis of symptomatic polycythemia obvious in this case.

Several etiologic factors may have played a role as shown by Gross et al., (1973) in the following scheme:



Placental insufficiency with intra-uterine hypoxia appears to play a central role in many of the cases associated with plethora (Oski and Naiman, 1972). This possibility can be excluded in this case because there

were no signs of placental insufficiency in the child.

Twin to twin transfusion as a cause of polycythemia in this case can not be excluded because the Coen and Sutherland (1970) examination method to prove placental vascula-

ture communication was not done. However, the possibility of twin to twin transfusion in this case does not seem to be the only causative factor.

Another kind of transfusion can be maternal-fetal transfusion. According to Michael and Mauer (1961), a maternal-fetal transfusion can be diagnosed by demonstrating a low fetal hemoglobin concentration in the infant. Oski and Naiman (1972) stated that although the range of fetal hemoglobin concentration is rather wide at any given gestational age, the presence of fetal hemoglobin of less than 60% in a plethoric infant also suggests the presence of a maternal transfusion. In this case the fetal hemoglobin concentration was 33.1%.

Maternal-fetal transfusion is also diagnosed by the detection of red cells of maternal blood type in infant blood. The mixed agglutination technique demonstrating the increased quantities of Ig. A and Ig. M in the infant's serum is used. Unfortunately this technique can not be done in our laboratories.

Besides the two factors causing transfusion syndromes mentioned above, delayed cord clamping can also produce polycythemia in the newborn. The hematocrit of the newborn is greatly influenced by the elapsed time before the umbilical cord is clamped. Late clamping results in large placental transfusion and a

smaller residual placental blood volume (Mackintosh and Walker, 1973). It is also estimated that the proportion of total infant/placental blood volume in the infant at birth is 67% and it increases to 80% after one minute and to 87% at the termination of placental transfusion (Yao, Monian and Lind, 1969). Electron microscopical studies have revealed large numbers of fenestrated small blood vessels in infants with late cord clamping. This explains an increased transudation (Pietra et al., 1968) up to one half of plasma volume (Buckels and Usher, 1965) into the extravascular space. After subsequent fluid shifts polycythemia results.

The symptoms appear to be a consequence of the associated increased blood viscosity. Besides, transfusion may also result in a hyperdynamic state of the circulation with symptoms suggestive of circulatory overload. In this case the infant showed lethargy, plethora, cyanosis, jaundice, congestive heart failure and hypocalcemia.

It has been agreed that once symptoms exist in neonatal polycythemia, a partial exchange transfusion to lower the viscosity of blood should be considered. In this case the formula of Oski and Naiman (1972) was used. Mackintosh and Walker (1973) proposed plasma or albumin exchange transfusion using 30 ml/kg. bodyweight. This method has been reported with good results. They also recommended that as polycythemia is

so easily diagnosed and treated, hematocrit estimation is advised for any infant with cerebral, cardiovascular, or respiratory signs.

Until now a partial exchange transfusion is recommended only for symptomatic polycythemia. However, asymptomatic polycythemia should be carefully observed for evidence of hypoglycemia, hypocalcemia and hyperbilirubinemia (Oski and Naiman, 1972).

#### Summary

A case of symptomatic polycythemia has been reported.

Delayed cord clamping may be the cause of polycythemia, but because of low fetal hemoglobin concentration the possibility of maternal-fetal transfusion could not be excluded.

The signs and symptoms were plethora, cyanosis, lethargy, jaundice, congestive heart failure and hypocalcemia. A partial exchange transfusion using one fifth normal saline in 5% glucose was done.

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