A CASE OF INTRAPLACENTAL TWIN TRANSFUSION

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There have been several reports of polycythæmia in one of uniovular twins with anæmia in the other twin. This syndrome has been recently fully recorded and reviewel by Corney and Aherne (1965). We describe here a severe example of this condition, with jaundice in the polycythæmic twin necessitating exchange transfusion; the severe anæmia in the other twin caused death in utero, and the birth of a macerated fœtus.

Case report

This was the mother's first pregnancy, which was complicated by excessive vomiting in the first two months, threatened at three months, and by anæmia (Hb 7.7g%) in the eighth month of pregnancy. The latter was treated with folic acid and intravenous iron dextran complex ("Imferon"). At thirty weeks she developed gross œdema of the legs reaching up to the knee, albuminuria, moderate hypertension and probable hydramnios. Antibodies to Rhesus factor were absent and the Kahn test was negative. Her blood group was 0 Rhesus positive. She was treated by bed rest and Navidrex K. She had no history of serious illness and there was no family history of twinning.

Both twins were delivered normally at 33 weeks' gestation following spontaneous rupture of the membranes. The first twin female was macerated and hydropic, and weighed 4 lbs. 4 ozs. (see *Fig. 1).* The second was born a few minutes later, weighed 3 lbs. 10 ozs.; her length was 16 ins. and head circumference 11 ins. She was linked by a tenuous cord to a deeply-congested portion of the placenta occupying about one quarter of the uniovular placenta; the rest of the placenta, supplying the twin via a thick œdematous cord, was pale and œdematous (see *Fig. 2).*

The second twin was in fairly good condition at birth, and the onset of respiration was spontaneous. However, she was markedly plethoric, limp and irritable. Her cry was high-pitched. She sucked poorly and had a feeble Moro reflex. The heart rate was 120/min., the lungs were clear to auscultation, the liver and spleen were just palpable. A few hours after birth, the Hb (venous sample) was 29 g%, P.C.V. 85%, blood glucose 16 mg%. X-ray of the chest showed normal heart and lungs. In view of the polycythæmia in a monozygotic twin, intraplacental transfusion was diagnosed and at 16 hours 20 ml of blood were withdrawn via a

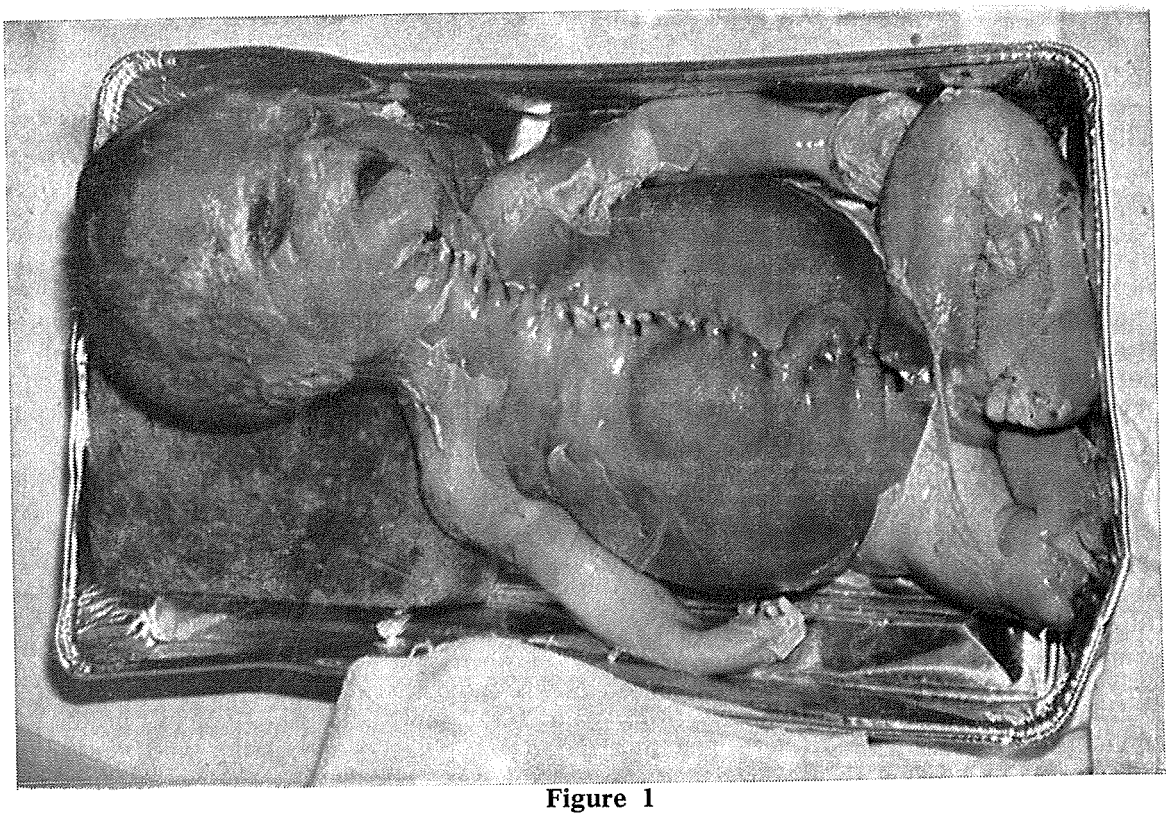

Figure 1

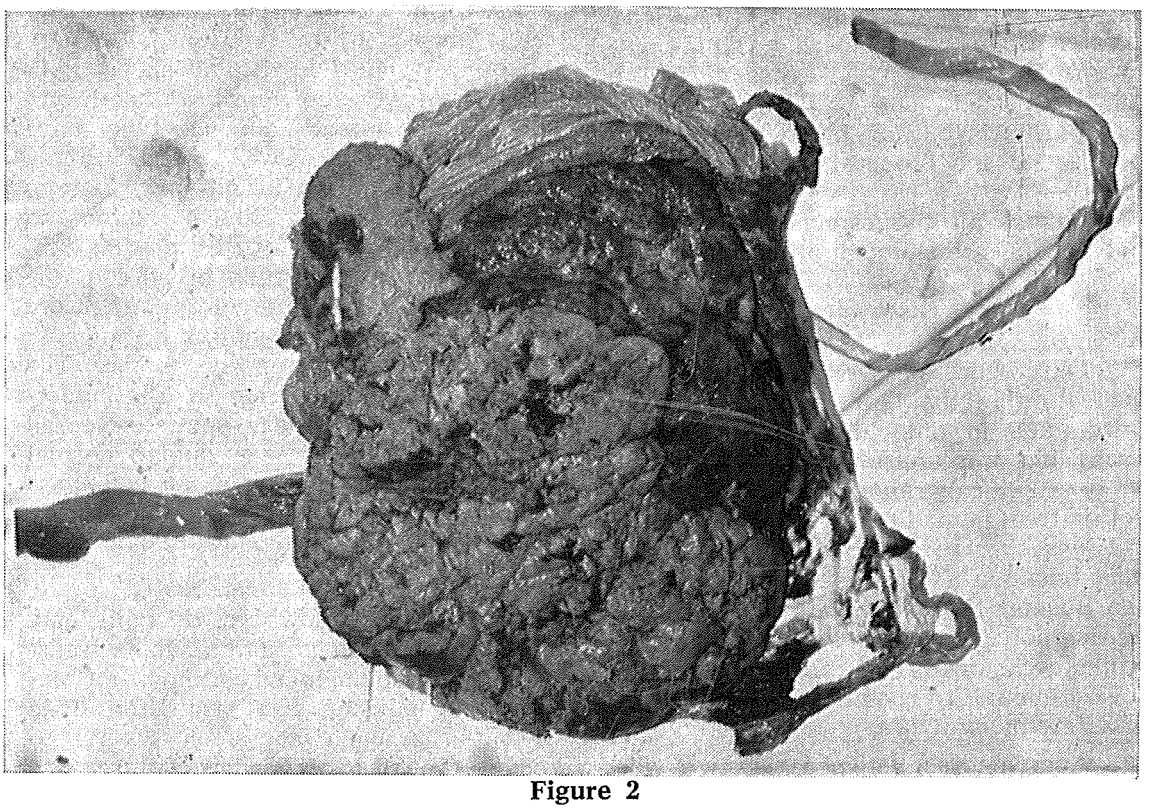

Figure 2

catheter inserted into an umbilical vein. Six hours later a further 40 ml were withdrawn. At 30 hours the hæmoglobin had fallen to 22 g% and P.C.V. to 74%. The baby's condition had improved, and a normal cry and a sucking reflex were present. At 36 hours the indirect serum bilirubin level had risen to 21.8 mg%, though clinically severe jaundice was not present. There were no antibodies, the baby's blood group was A Rhesus negative, and the Coombs' test was negative. 90 ml per pound body weight were exchanged via the umbilical vein.

At the end of the exchange transfusion the hæmoglobin was 16.4 g%, P.C.V. 45%, and the serum bilirubin 12 mg%. Umbilical swab and blood cultures were sterile. Subsequent progress was uneventful, the baby being discharged at 5 weeks weighing 5 lbs. 3 ozs. At follow-up, she thrived, weighing 11 lbs. 7 ozs. at 5 months and appeared a normal baby. She smiled at 4 months, and there was no impairment of hearing or any evidence of cerebral palsy.

Postmortem examination on the macerated first twin revealed no anatomical abnormality apart from gross œdema of all the tissues. The blood vessels were singularly devoid of blood. There were pericardial and bilateral pleural effusions, together with gross ascites. Microscopic sections of all tissues examined showed only changes due to autolysis.

Discussion

Differences in the hæmoglobin concentration in the first few days are not uncommon in uniovular twins (M. Seip, 1956), but it is unusual for this difference to be significant enough to warrant any active treatment. Lethargy, cyanosis, convulsions, heart failure and early hyperbilirubinæmia (with risk of kernicterus, see Bosma, 1954) have all been recorded in the polycythæmic twin (Wood, 1959), while occasionally the anæmia has been severe enough to require correction in the first few days of life.

Anastomoses between the two sides of the uniovular placenta have been amply demonstrated anatomically by Schatz (1882), and more recently by Klingberg *et al.* (1955) and Benirschke (1961). These may be superficial (artery to artery, vein to vein, or both types) or deep (always arteriovenous). It is postulated that at least some of the superficial anastomoses develop as a compensatory mechnism to balance previously-existing deep anastomoses. When the compensatory mechanism fails to develop, all gradations of anæmia and polycythæmia may occur (Becker and Glass, 1963), and in extreme instances the donor twin may exsanguinate in utero, as in the case reported here (see also Benirschke, 1961). No superficial placental anastomoses were demonstrable in our case, and it has already been noted that the anæmic twin's blood vessels were practically devoid of blood.

The degree of polycythæmia may be intense, and the increased viscosity of the blood and increased blood volume predispose to a sluggish circulation and venous thromboses. Cerebral (lethargy, poor sucking, feeble Moro response, convulsions), cardiovascular (peripheral and central cyanosis, heart failure), pulmonary and renal (renal vein thrombosis) complications may develop. Purpura and thrombocytopœnia in the anæmic twin have been recorded by Corney and Aherne (1965). The serum bilirubin may rise to dangerous levels in the first 48 hours, and as in the present patient this may be difficult to recognise clinically because of the polycythæmia. It is important therefore to estimate the serum bilirubin, however mild the jaundice appears to be. The mechanism of production of the hyperbilirubinæmia is not clear, but an increased bilirubin load to the liver due to normal breakdown of an increased red cell mass is likely to be an important contributory factor (Valaes and Doxiades, 1960; Bergstedt, 1957).

The anæmic newborn or twin may need urgent treatment and a simple transfusion is recommended, though an exchange transfusion has also been done when the anæmia has been severe, to obviate the risk of cardiac failure (see Corney and Aherne, 1965). Blood from the

infant with polycythæmia has been used by Valaes and Doxiades (1960) to transfuse the anæmic baby. This may be dangerous if the serum bilirubin of the "donor" twin is high.

The polycythæmic twin has been variously treated by venesection alone or by venesection and replacement with normal saline, albumin, plasma or 5% dextrose. Removal of blood not only corrects the polycythæmia but also decreases the risk of hyperbilirubinæmia. The hyperbilirubinæmia *per se* may require exchange transfusion.

Summary

1. Twins showing intraplacental transfusion are described.

2. Successful treatment of the polycythæmia and hyperbilirubinæmia is described. Bilirubin estimation should be performed routinely in such cases because polycythæmia may mask severe jaundice.

3. Previous reports of the condition and its management are briefly discussed.

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