Placental Transfusion Syndrome in Twins

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The existence of vascular communications between placentas in monochorial twins almost always results in emergency medical treatment.

During a period of a few months we have had three such cases of placental transfusion syndrome, and felt that perhaps the occurrence of this anastomosis in monochorionic twins is more frequent than those clinically diagnosed. Probably only the severe forms are identified, and the less severe may easily be overlooked. In view of the frequency of this syndrome, we decided that all twin placentas should be examined for anastomosis either by the Benirschke technique[1] or with angiograms (which were used in the cases presented). Anastomosis in monochorionic twin placentas plays an important role in perinatal morbidity and mortality, as was clearly indicated by Benirschke,[2 3] in 1961.

Case 1: Twin D

The mother, a 23-year-old, gravida 2, para 1, had given birth to a boy two years previously after an uneventful pregnancy and delivery. This pregnancy was complicated only by mild edema of the feet and a two kg weight loss in three weeks prior to delivery. After forty weeks of gestation, she went into spontaneous labor and in two hours the cervix was completely dilated. When the membranes ruptured on the delivery table it was noticed that the amniotic fluid was deeply stained with meconium.

Twin A: A male infant weighing 2,900 gr; his color was very pale at birth, and his apgar score was two. As soon as oxygen under pressure was given his condition improved and spontaneous respiration started. Inital examination of the baby revealed a second degree of dysmaturation and Hb was found to be 10 gm/100 ml. In spite of an immediate blood transfusion and supportive measures, he made very poor progress and

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developed generalized convulsive activity. Because of the rales in both lung fields he was put on penicillin and streptomycin, but he died 45 hours after birth.

Twin B: A male infant weighing 2,500 gr; his apgar score was one and his color a deep purplish red. He did not respond to resuscitation, and his heart beat stopped soon after delivery.

Examination of the placenta by angiogram revealed vein to vein and artery to vein anastomosis (Figures 1 and 2). Gross and microscopic

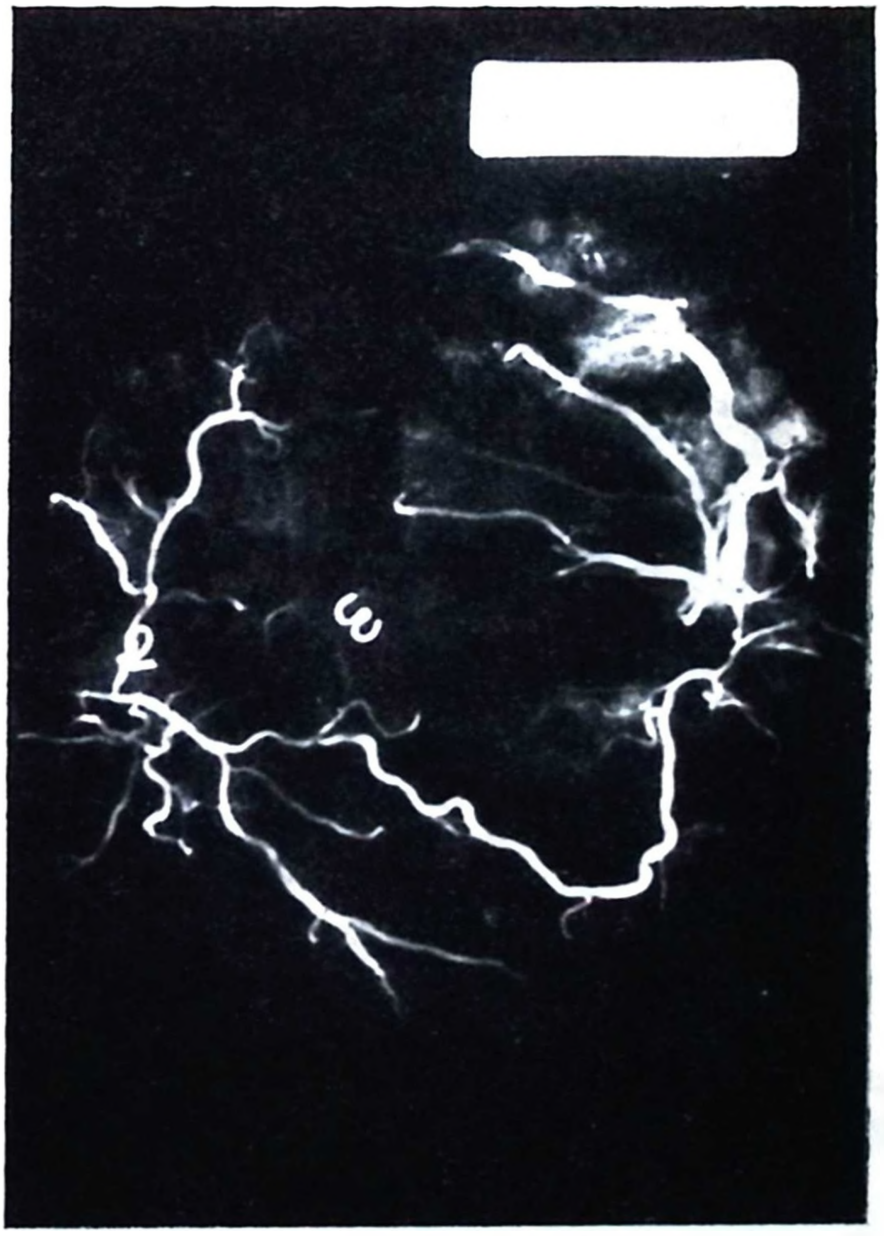

Figure 1. Angiogram of Placenta in Case 1.
The stump of the cord of Twin A is labelled No. 2
The stump of the cord of Twin B is labelled No. 1
Divided membrane between placentas is labelled No. 3
Urografin was given through Twin B's cord stump via the umbilical vein.
The figure show artery to vein anastomosis between two parts of the placentas.

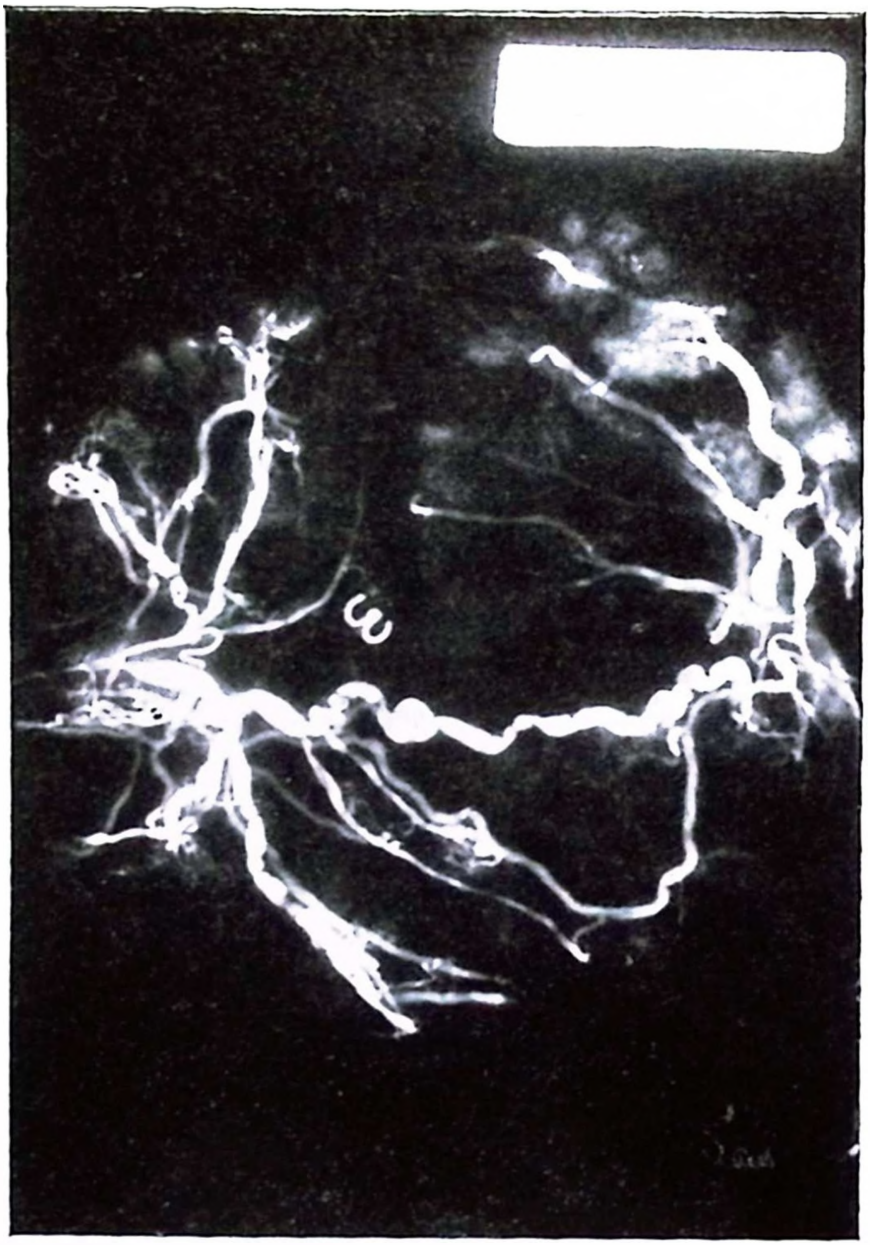

Figure 2. In the same placenta, urografin given through Twin A's cord stump via umbilical vein. The figure shows vein to vein as well as artery to vein anastomosis from previous angiogram.

examination of the placenta indicated a diamniotic, monochorionic fused twin placenta. Postmortem examination of Twin A revealed bronchopneumonia, and that of Twin B congestion and hemorrhages in all organs.

Case 2: Twin C

The mother was a 25-year-old, gravida 2, para 1, whose pregnancy was complicated by hypertension (160/100 mm Hg) three weeks prior to delivery. On the day of delivery she was admitted to the hospital with full dilatation of the cervix, and prolapse of the cord was noticed. She was delivered immediately.

Twin A: A male infant delivered by breech extraction, whose birth weight was 2,850 gm, length 49 cm and apgar score six. Within three minutes his condition improved and he started good spontaneous respiration. The baby did well during a hospital stay of five days. His initial Hb was found to be 11.6 gm/100 ml, and because of his good clinical condition he was not transfused, but given elemental iron 2 mg/kg three times daily for three months.

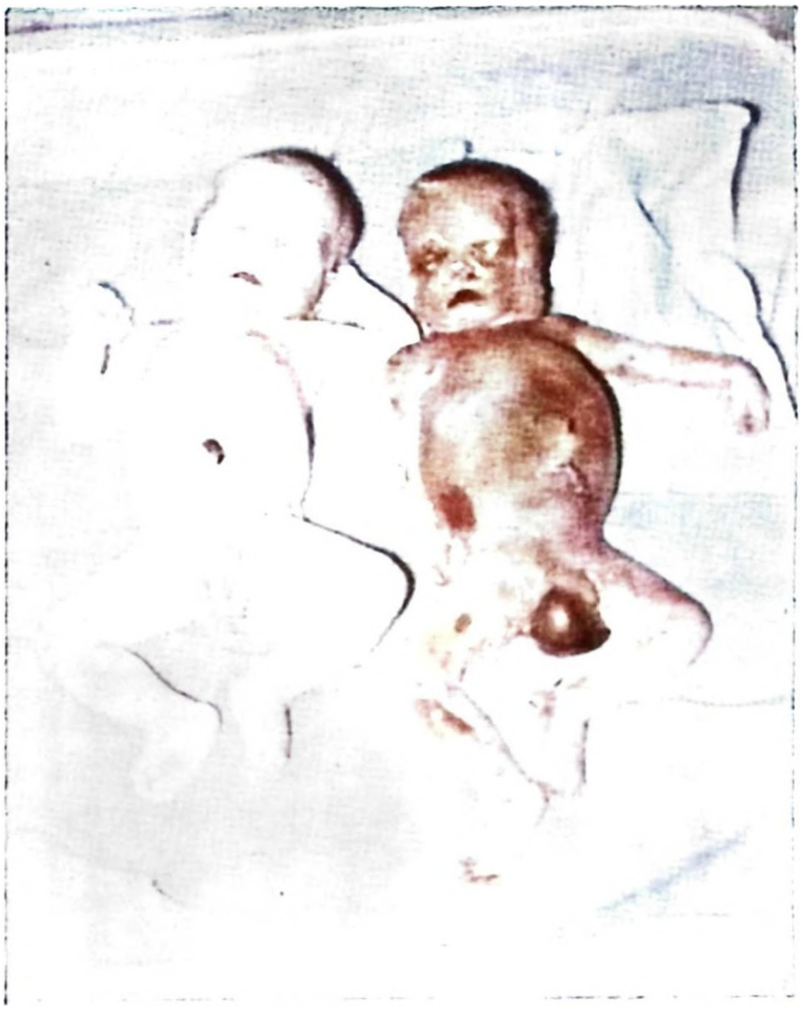

Figure 3. Twins of Case 1: Twin A appears anemic and Twin B plethoric.

Twin B: A macerated stillborn male infant weighing 1,850 gm was delivered in transverse presentation by version and extraction. He was deep purple in color. Unfortunately no postmortem examination could be obtained.

Examination of the placenta by angiogram revealed vein to vein anastomosis. Macroscopic and microscopic examination of the placenta disclosed a diamniotic, monochorionic fused twin placenta.

Case 3: Twin K

The mother was a 33-year-old, gravida 1, para 0, who had an uneventful pregnancy until 29 weeks of gestation. During an examination because of right upper quadrant pain it was noted that there was no fetal heart sound in one of the twins. Due to this diagnosis of intrauterine death, a cesarean section was performed at 30 weeks of gestation at another hospital. Twin A was a slightly macerated, deep purple colored female infant, and Twin B was a hydropic female with an apgar score of three. Her condition improved after five minutes of resuscitation.

A postmortem examination of Twin A revealed a macerated stillborn plethoric infant with congestion in all the organs. Since she was delivered in another hospital the placenta could not be obtained for examination.

Twin B was transferred to Hacettepe premature unit. On admission she was very pale, mimicking hydrops fetalis. Her birth weight was 1,890 gm. The direct Coombs test was negative, her blood group was O Rh positive, the reticulocyte count was 11.2 per cent and she had many normoblasts on her peripheral blood smear; Hb was low (11.16 gm/100 ml). There were rales at the base of both lungs due to congestive heart failure. Since Twin B had anemia and Twin A had a plethoric appearance, the former was immediately diagnosed as having placental transfusion syndrome. The anemia was treated by packed cell transfusions which the infant tolerated and responded to well. Within 24 hours the rales in both lung fields disappeared and the child put out a large amount of urine. After this initial therapy she was put on elemental iron, as in case two. She made good progress and was discharged at the age of 47 days in excellent condition with Hb 12.5 gm/100 ml and a 1.8 per cent reticulocyte count.

Discussion

This syndrome has been mentioned in the literature as «Twin transfusion syndrome.»[3,5] «Parabiotic syndrome,»[4] or «Fetal transfusion syndrome».[6] We prefer to use the term «Placental transfusion syndrome» because it originates from the vascular anastomosis of monochorionic twin placentas.

Naeye (1963)[4] reported nine cases with a thorough study of their anatomical findings. Rausen et al [5] gave detailed reviews of 36 cases (including Naeye's) of placental transfusion syndrome in the literature in 1965. They added their own series of 19 cases, and pointed out that the incidence of this syndrome in monochorial twin placentas was 15 per cent.

The treatment of these twins can be accomplished only by early recognition of the condition. It is important for both pediatricians and obstetricians to make a quick diagnosis in the delivery room, or immediately

after in the nursery. The color differences of the twins are the most striking clinical features. Immediate examination of the placentas in suspected cases is important; if they are separate they are usually dichorionic. Vascular anastomosis of twin placentas usually occurs in monochorionic, diamnotic twin placentas.

The donor twin is anemic and may be in shock due to hypovolemia; it is usually small and malnourished, and oligohydramnios may be present. Microcardia, small or normal glomeruli in the kidney and thin walled arterioles may be found in histological sections.

The recipient twin is plethoric and usually larger than the donor. Hyperbilirubinemia may be found as a result of polycythemia. Hypervolemia, cardiomegaly and an increase of muscle around the pulmonary and systemic arteries suggesting antenatal hypertension is well documented by Naeye.[4] Polyhydramnios in the recipient twin usually results in premature delivery and increased risk to the babies.

In our three sets of twins, the recipient died *in utero* in two cases and one died shortly after birth. Better tolerance of slow developing anemia in the donor twin could be explained by increasing hematopoietic activity.

Treatment of these twins should begin immediately. The donor may be severely anemic and in shock, for which immediate blood transfusion is indicated. If placental transfusion has been chronic, the anemia may be well tolerated and such babies do well on iron therapy alone.[7]

The recipient twin's life may be saved by partial exchange transfusion with plasma to reduce the hematocrit to approximately 60 per cent, or by phlebotomy to decrease the blood volume. Sometimes digitalization is indicated to overcome the cardiac loading. In addition to this therapy, intensive premature care should be given to these high risk babies.

Summary

Three sets of twins with placental transfusion syndrome are presented. The importance of this syndrome in perinatal morbidity and mortality is once again emphasized, and the findings in these babies, immediate care after delivery, and terminology are discussed.

Acknowledgemets

We wish to thank Hüsnü Kişnişçi, M. D., Chief of the Department of Obstetrics and Gynecology and Tekin Durukan, M. D., Chief Resident in Obstetrics and Gynecology for their kind co-operation.

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