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A Study on Prognosis of Surviving Cotwin

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Abstract. It has been suggested that the surviving cotwin and the mother are seriously affected when intrauterine fetal death of one monozygotic twin occurs. Several authors have reported brain damage in the surviving twin coexisting in utero with dead monozygotic cotwin. Most of such instances are monochorionic and show vascular communication between paired twins in the placenta. In a total of 133 pairs of monochorionic twins, death of one twin during pregnancy occurred in 33 cases (24.8%). Of 33 surviving twins, 8 babies suffered from porencephaly, cerebral palsy and other abnormalities. More unfavorable prognosis was recognized when a cotwin died in the latter half of pregnancy, while when a monozygotic cotwin died in the early stage of pregnancy, the surviving twin uneventfully grew to term in most cases.

Key words: Intrauterine fetal death, Brain damage, Vascular communication, Porencephaly, Twin transfusion syndrome, Cord complications

INTRODUCTION

It has been suggested that the surviving cotwin is seriously affected when intrauterine fetal death of one monozygotic (MZ) twin occurs. Several authors [1,2,3,5,6] have reported brain damage and multiple organ failure in the liveborn cotwin coexisting with a deceased twin. These findings are attributed to thromboplastin-like materials passing through the placenta from the dead twin to the circulation of the surviving twin.

Generally MZ twins having a monochorionic placenta are known to be at increased risk for intrauterine growth retardation, intrauterine fetal death, fetal anomalies and other structural defects. However, the incidence of these fetal abnormalities largely depends on the types of vascular communications between twins [6]. Although the causes

of fetal death in utero of one MZ twin are yet unknown, twin transfusion syndrome is considered to be the most contributory factor [4].

A case is presented and the relationship between outcome of the surviving cotwin and timing of death of one MZ twin is discussed.

MATERIALS AND METHODS

Over a period of 25 years, a total of 218 placentas of twins were examined at the Tokyo Medical College Hospital. These consisted of 133 pairs of monochorionic and 85 pairs of dichorionic twins. Placentas were examined and the outcome of the infants was evaluated according to the types of vascular anastomoses between twins. The simplest and the best way for identifying vascular communication is to inject air from an artery on one side, and thereafter check venous communication in the same way. The infant born first is designated as twin A and the second as twin B.

CASE PRESENTATION

The patient, S.S., was 31-year-old, gravida 1 and para 0. Her menarche was at 12 years, and she had an irregular menstrual cycle, with 45 to 90 days interval and 7 days duration. She was first seen on 20 January 1988 and diagnosed at 10 weeks and 1 day of pregnancy by ultrasound. At 23 weeks of pregnancy, biparietal diameter and fetal femur length for twin A were 59 mm and 35 mm, and those for twin B 63 mm and 38 mm, respectively. The heart movements of both fetuses were recognizable. At 26 weeks of pregnancy, twin A's heart movement disappeared. On 19 May 1988, at 27 weeks of pregnancy, spontaneous labour pains began.

On 20 May 1988, at 00.07, a 724 g female infant was born dead, and at 00:36, a 1066 g (appropriate weight for gestation) female infant was delivered with an Apgar score of 6 at 1 minute and 9 at 5 minutes. Placenta was diamniotic monochorionic, premature, with slight marginal hemorrhage, and weighed 390 g. Vascular anastomoses between twins in the placenta included both artery-to-artery and vein-to-vein.

RESULTS

Although a high incidence of fetal abnormalities in twin pregnancies is generally pointed out, we can recognize that fetal growth retardation, perinatal fetal death and fetal anomalies are very frequent in monochorionic twins, but less so in dichorionic twins. The most unfavorable outcome was in monoamniotic monochorionic twins (Table 1).

As for vascular anastomoses, the commonest vascular communication in monochorionic twins was an artery-to-artery anastomosis. It was present in 51 of the 133 pairs of monochorionic twins (38.3%). In this type of vascular communication, relatively low incidence of fetal abnormalities was found. On the other hand, in those types of anastomoses as coexistence of artery-to-artery and vein-to-vein or artery-to-vein, and as vein-to-vein, and/or artery-to-vein, fetal abnormalities were very frequent (Table 2).

Table 1 - Incidence of SFD babies, perinatal death, and fetal anomalies in twins according to placentation

| Type of placentation | No. of cases | No. of fetuses | SFD babies (%) | Perinatal death (%) | Fetal anomalies (%) |
|---------------------------------|--------------|----------------|-------------------|---------------------|---------------------|
| Monoamniotic monochorionic | 8 | 16 | 12 (75.0) | 7 (43.8) | 4 (25.0) |
| Diamniotic monochorionic | 125 | 250 | 105 (42.0) | 76 (30.4) | 41 (16.4) |
| Monochorionic twins | 133 | 266 | 117 (44.0) | 83 (31.2) | 45 (16.9) |
| Diamniotic dichorionic fused | 49 | 98 | 33 (33.7) | 9 (9.2) | 7 (7.1) |
| Diamniotic dichorionic separate | 36 | 72 | 21 (29.2) | 6 (8.3) | 2 (2.8) |
| Dichorionic twins | 85 | 170 | 54 (31.8) | 15 (8.8) | 9 (5.3) |

SFD: Small for date.

Table 2 - Incidence of fetal abnormalities according to vascular anastomoses

| Type of anastomoses | No. of fetuses (No. of cases) | SFD babies (%) | Perinatal death (%) | Fetal anomalies (%) |
|--|-------------------------------|----------------|---------------------|---------------------|
| Artery-artery | 102 (51) | 36 (35.3) | 12 (11.8) | 3 (2.9) |
| Artery-artery & vein-vein or artery-vein | 68 (34) | 37 (54.4) | 27 (39.7) | 13 (19.1) |
| Vein-vein with/without artery-vein | 10 (5) | 6 (60.0) | 7 (70.0) | 1 (10.0) |
| Artery-vein | 18 (9) | 4 (22.2) | 7 (39.8) | 2 (11.1) |

SFD: Small for date.

In the reference case, intrauterine death of one of the twins was estimated to have occurred a week before delivery at 27 weeks of pregnancy. The live cotwin suffered from respiratory distress immediately after birth and her growth has been disturbed physically and neurologically. CT-scan of brain at 7 weeks demonstrated multicystic encephalomalacia (Figure). Placental examination revealed a diamniotic monochorionic placenta with coexistence of artery-to-artery and vein-to-vein anastomoses.

In 33 out of the 133 pairs of monochorionic twins, one of the twins died during pregnancy. Of the 33 liveborn cotwins, 25 are known to be neurodevelopmentally normal,

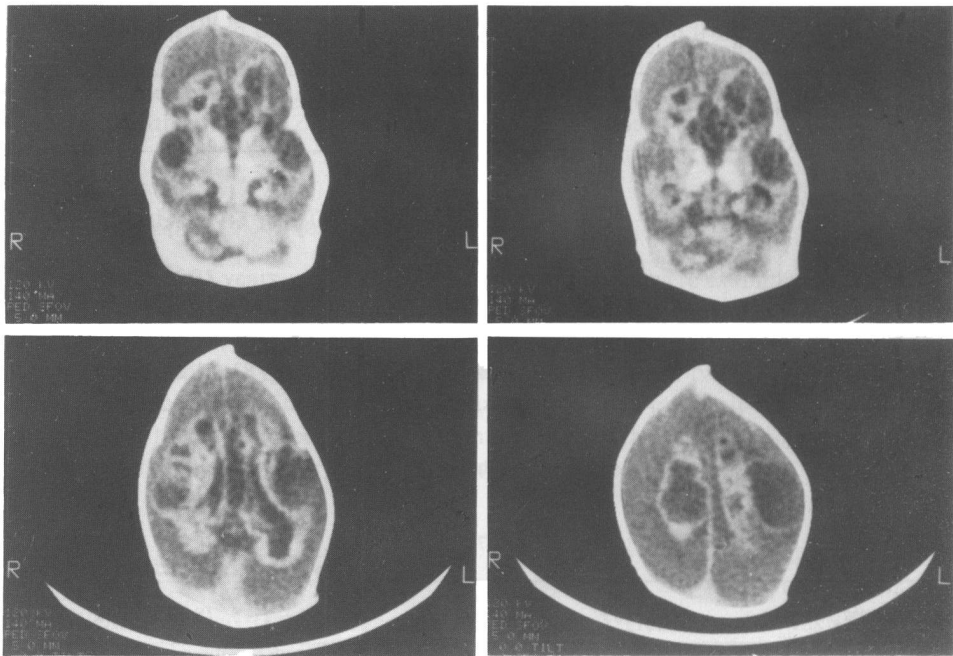


Figure. CT-scan of brain at 7 weeks old. Porencephaly due to multiple infarctions is noticeable.

Table 3 - Unfavorable outcome of the survived twins

| Term when one twin died | Case ref. no. | Body weight and gest weeks at birth | Remarks on liveborn twins |
|-------------------------|---------------|-------------------------------------|------------------------------|
| 1st trimester | 18 | M : 2934g (39w) | Fallot's tetralogy |
| 2nd trimester | 3 | M : 3322g (40w) | Spina bifida |
| | 16 | M : 3280g (41w) | Congenital skin defects |
| | 32 | M : 1148g (29w) | Died of RDS soon after birth |
| 3rd trimester | 14 | F : 2210g (35w) | Cerebral palsy |
| | 29 | M : 2104g (35w) | Porencephaly |
| | 30 | F : 1800g (33w) | Died of RDS at 3 days |
| | 33 | F : 1066g (27w) | Porencephaly |

but 8 had an unfavorable outcome (Table 3). Abnormalities such as skin defect (case 16), cerebral palsy (case 14), and porencephaly (cases 29 and 33) may result from embolism and infarction affected by intrauterine fetal death of the cotwin in rather late stage of pregnancy. However, the other fetal abnormalities shown in Table 3 (cases 18, 3, 32 and 30) may not be directly related to the cotwin's fetal death.

DISCUSSION

Although the etiology of intrauterine death of one MZ twin is yet to be known, the most contributory cause to fetal death in the 3rd trimester is likely to be the twin transfusion syndrome, which can occur even at an early stage of pregnancy [2]. However, in earlier stages, such as 1st or 2nd trimester, causes for fetal death other than twin transfusion syndrome should also be considered. When intrauterine fetal death of one twin occurred in the 2nd trimester, the dead fetus was delivered as fetus papyraceus with the survived cotwin and the placenta near term, and all dead fetuses we have observed had velamentous or marginal insertion of the umbilical cord [6]. Fetal death of one twin in the 1st trimester might occur by when twin transfusion syndrome and cord complications affect it earlier and more severely.

Twin transfusion syndrome and abnormal insertion of the umbilical cord, which are found with a high incidence in monochorionic twins, frequently result in fetal morbidity and mortality on account of an imbalance and disruption of fetal circulation between twins. When one twin dies, embolisation and infarction may occur by passing of thromboplastin-like substances through the placental anastomoses from the dead to the live twin [1,2,5,].

The later the stage of pregnancy one twin dies, the worse the outcome for the surviving cotwin [6]. This suggests the possibility that the immature fetus has not yet produced enough coagulative factors to affect the live cotwin. Therefore, the surviving cotwin can grow without adverse effects and with a wider space in utero when early fetal death of a twin occurs.

CONCLUSION

1. Out of 133 pairs of monochorionic twins, intrauterine fetal death of one twin occurred in 33 cases (24.8%).
2. The MZ pairs with a placenta with both artery-to-artery and vein-to-vein anastomoses were at high risk for intrauterine fetal death of one twin, and the surviving cotwin also had unfavorable outcome.
3. The later the stage of pregnancy one twin died, the worse the outcome of the surviving cotwin.
4. Of 33 cases where one twin died during pregnancy, the surviving cotwin suffered from porencephaly, cerebral palsy and/or other structural defects in 8 cases.
5. The main cause of such lesions in the surviving twin is likely to be arterial embolism and infarction.

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