A Patient In Whom Symptoms Of Preeclampsia Improved After Intrauterine Fetal Death Of A Co-Twin

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Citation

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Abstract

Although fundamental treatment of hypertensive disorders of pregnancy is termination of pregnancy, prematurity of infants becomes a problem and careful management is required in the early gestational weeks. We report a 21-year-old woman who had a dichorionic, diamniotic twin pregnancy and conceived naturally, but fetal growth restriction occurred in one fetus from early pregnancy. Symptoms of preeclampsia appeared from 25 weeks' gestation. Maternal symptoms improved at 32 weeks' gestation with intrauterine fetal death of one twin as a trigger. Continuation of pregnancy became possible and a healthy child was delivered at 37 weeks' gestation. Few such cases of complete resolution of preeclampsia following the death of a single fetus have been previously reported. In this report, we discuss our case in the context of these other cases.

CASE PRESENTATION

The patient was a 21-year-old primipara with a dichorionic twin pregnancy who conceived naturally. Her past history was unremarkable. One gestational sac at 4 weeks at the time of the first visit was recognized (twin A) and the second gestational sac appeared 4 weeks later (twin B). We diagnosed her as having dichorionic, diamniotic twins. Twin B was initially small and there was oligohydramnios. A total of 39% discordancy of fetal weight was found at 16 weeks of gestation. The discordant rate was 59% at 20 weeks of pregnancy. Twin B showed blood flow redistribution and the resistance index value of the umbilical artery/middle cerebral artery was 0.83/0.67. The discordant rate became 63% at 22 weeks of pregnancy. Twin A was equivalent to 22 weeks of gestation and showed no obvious abnormality. However, the placenta of twin B was small and amniotic fluid was hardly recognized, and there was disruption of umbilical arterial diastolic blood flow. Twin B was diagnosed with severe fetal growth restriction. Twin A developed without any problems. However, we thought that the consequences of pregnancy for saving the life of twin B would lead to a poor prognosis of twin A. After consultation with the patient and her family, priority was given to growth and maturation of twin A, and we decided to continue the pregnancy. At 24 weeks of pregnancy, the patient had lower extremity edema and showed urine protein 2+, but blood pressure was normal at 125/63 mmHg. At 25 weeks' gestation, the patient had a 24-hour urine protein of 1.12 g and blood pressure was 146/86 mm Hg. She was then diagnosed with preeclampsia and was admitted to hospital. At 29 weeks' gestation, the amount of urine protein increased to 8.02 g/day, blood pressure became 167/85 mmHg, and growth of twin A started to slow from that time.

However, twin B did not increase in size. Twin B was found to be dead at 32 weeks of pregnancy. Because the patient had dichorionic, diamniotic twins, we decided to carefully continue her pregnancy. Blood pressure then naturally improved to the normal range, the amount of urine protein decreased to 4 g/day, and growth of twin A also normalized (Fig. 1). At 37 weeks' gestation, a healthy boy who weighed 2696 g with an Apgar score of 8/9 was born by vaginal delivery. Twin B was 340 g and the fetus showed maceration, but there was no obvious outer surface abnormality. Twin B's placenta was in a thin single umbilical artery with velamentous insertion of the cord (Fig. 2). The patient maintained 3 g/day of proteinuria after parturition, but her blood pressure was normal and she was

discharged. Proteinuria was negative at 2 months postpartum.

Figure 1

Transition of gestational weeks, estimated fetal weight, urine protein, and blood pressure.

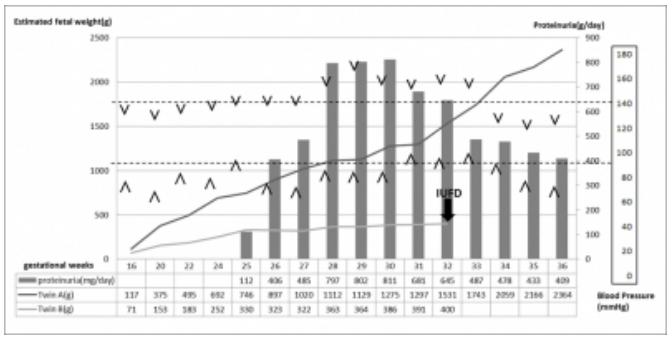
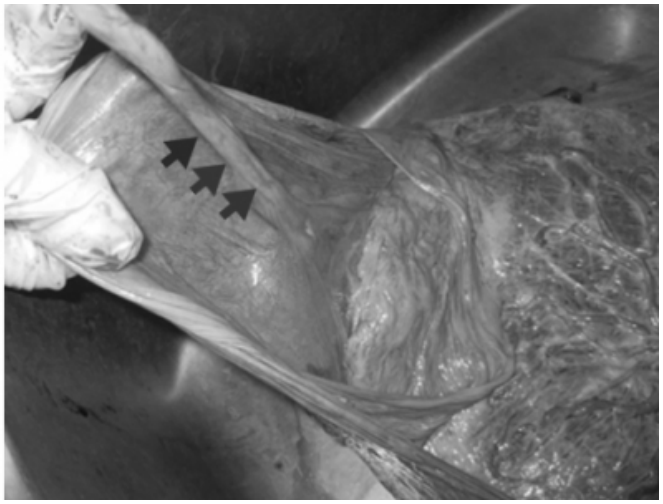


Figure 2

Twin B's placenta was in a thin single umbilical artery with velamentous insertion of the cord



DISCUSSION

Preeclampsia is likely caused by placental dysplasia, and the only fundamental treatment is termination of pregnancy. Preeclampsia affects 3%–5% of pregnancies and is a major cause of maternal and perinatal mortality [1]. Twin gestations occur in 3.2% of pregnancies and are associated with an increased risk of gestational diabetes, preterm delivery, fetal growth restriction, hypertension, and hemorrhage. The risk of preeclampsia in twin pregnancies is more than twice that of singleton pregnancies [2]. Our case was a patient with dichorionic, diamniotic twins. Twin B was small from early pregnancy and the patient's diagnosis was preeclampsia at 25 weeks' gestation. Symptoms of preeclampsia dramatically improved after intrauterine fetal death of twin B at 32 weeks' gestation and pregnancy was able to be continued. Twin A was delivered vaginally at 37 weeks' gestation. Few cases of resolution of preeclampsia after spontaneous intrauterine death of one twin have been reported [3-6].

Hagay et al. [3] hypothesized that genetic susceptibility to preeclampsia could be conferred by homozygosity for the same single recessive gene that is expressed in both the mother and fetus. They predicted that one of the dizygotic twins had a recessive gene susceptible to preeclampsia and preeclampsia improved as the fetus died.

Narashimhulu et al. [4] reported resolution of preeclampsia in a surviving fetus after intrauterine death of its co-twin. These authors suggested that placental involution after fetal demise is important for resolution of preeclampsia and it takes 1 to 3 weeks to improve. In our case, blood pressure became less than 140 mm Hg 2 weeks after the death of twin B.

However, Balci et al. [5] reported two cases in which preeclampsia did not improve after the co-twin died. They stressed the importance of severity of preeclampsia. In the case of severe preeclampsia, it might not improve even if one fetus dies. Balci et al. [5] found that preeclampsia was severe because proteinuria was 5 g/day, but in our case, it rose to 8 g/day. However, in our case, in contrast to proteinuria, blood pressure was relatively mild with no medication required. In the case of severe preeclampsia, symptoms may not be improved after death of a fetus, but evaluating severe preeclampsia only with proteinuria may not be accurate.

In our case, weight discordancy was observed from the beginning of pregnancy. At 22 weeks of pregnancy, twin B was determined to be in a state of severe fetal growth restriction and a high risk of intrauterine fetal death. When considering saving the life of twin B at that time, termination of pregnancy was necessary, but there was a high risk that twin A would have a poor prognosis. We decided to continue pregnancy as a result of discussion with the patient. Twin B then died and twin A was delivered in a healthy condition at 37 weeks. Audibert et al. [6] reported resolution of preeclampsia after selective termination of a dichorionic pregnancy in the third trimester. They considered that selective termination appears to be a reasonable option when one fetus is found to be abnormal in a multifetal pregnancy with dichorionic placentation.

If preeclampsia develops in a twin pregnancy and there is

abnormality in one twin, placental abnormality of that twin may be the cause of preeclampsia. If a co-twin with fetal growth restriction is thought to have a severe prognosis, one option is that priority can be given to continuing the pregnancy with the normal twin. This presents an ethical problem and each case needs to be carefully considered.

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