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Serial Amniocenteses in the Treatment of Twin to Twin Transfusion Complicated with Acute Polyhydramnios

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Abstract. Twin to twin transfusion, complicated by acute polyhydramnios in a monozygous twin pregnancy, is a difficult clinical problem. A precipitous course usually results in termination of the pregnancy within a few days and often is associated with a high perinatal mortality rate. Two cases are presented that were treated with repeated amniocenteses for the relief of extreme abdominal discomfort and to prevent imminent premature labor. The amount of amniotic fluid removed each time varied from 300 cc to 1200 cc, which was enough to relieve symptoms but not enough to induce uncontrolled uterine activity. A total of 3500 cc and 4750 cc of amniotic fluid were removed from the first and the second patient, respectively. The procedure was found to be safe and resulted in prolonging the pregnancies by 14 and 11 days, respectively. This management, with the addition of tocolysis and close fetal surveillance can offer some hope in an otherwise hopeless situation.

Key words: Serial amniocentesis, Twin to twin transfusion, Polyhydramnios –acute, Monozygous twin pregnancy

INTRODUCTION

Acute polyhydramnios is an uncommon complication of a monozygotic twin pregnancy with a rate ranging from 0.4 to 5% [2,4]. It is characterized by a rapid increase in amniotic fluid volume resulting in increased uterine distention within a short period of

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time [1,12,14]. This leads to severe maternal discomfort with respiratory impairment, premature contractions, premature rupture of the membranes and premature delivery. Because of its association with twin pregnancy and its early occurrence, the outcome is usually very poor [1,14,15].

The etiology of this condition is believed to be a twin-to-twin transfusion which can be demonstrated by an artery-to-vein anastomosis or is the result of intraparenchymal shunting without surface anastomosis [3,14,15]. The recipient twin becomes hypervolemic with hypertrophy of the heart and the entire body. Excessive fetal urination leads to the hydramnios at 20-30 weeks gestation; at the same time, the continuous overload can lead to hydropic changes, namely, scalp edema, ascites, and pleural and pericardial effusion [1,4,15]. The donor twin, on the other hand, becomes anemic with a slower growth rate and oligohydramnios. The size of the shunt is responsible for the severity of this condition, the disparity in growth and volume of the amniotic sac of each twin. It is believed that this is a distinctly separate entity from other forms of polyhydramnios, such as those associated with diabetes, Rh isoimmunization and congenital anomalies [1].

The ultrasonographic criteria of twin-to-twin transfusions are [5]: significant disparity in size with fetuses of the same sex; disparity in the size between the two amniotic sacs; separate umbilical cords with disparity in their size; a single placenta, and evidence of hydrops or congestive heart failure in the recipient.

No uniform management of this condition has been published and, in view of the high fetal mortality when conservative management is attempted, a more aggressive approach seems logical. We would like to present our experience with two such cases.

CASE 1

M.A.D., 23 years old, gravida 4, para 1, abortus 2, was transferred to our hospital at 25 weeks for a twin pregnancy, a rapid increase in abdominal size and abdominal discomfort. Past obstretical history consisted of one full term normal delivery and two spontaneous first trimester abortions.

This pregnancy was uncomplicated until 24 weeks when the patient noticed a rapid increase in abdominal size and abdominal discomfort. On admission, the pertinent finding was an enlarged, tense uterus with a symphysis-fundal height of 48 cm. The cervix was long and closed with bulging of the vaginal fornices, and no contractions.

The ultrasound finding revealed a twin gestation with massive hydramnios in the gestational sac of twin A, a single fundal placenta and a dividing membrane. Twin A had a biparietal-diameter (BPD) of 6.8 cm, femur length (FL) of 5 cm, estimated fetal weight (EFW) of 980 g with no evidence of hydrops. The fetus was very active. Twin B had a BPD of 6.8 cm, FL of 4.6 cm, EFW of 726 and lower than normal amount of amniotic fluid. This fetus was less active than its cotwin. The umbilical veins were identical in diameter. There were no definable abnormalities, by ultrasound. A 50 g glucose challenge was negative. Blood group was 0 positive with negative antibody screen.

The patient was placed on bedrest and given high protein diet. Oral tocolysis was begun due to premature contractions. On the eight hospitalization day, due to extreme

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abdominal discomfort, 500 cc of amniotic fluid were slowly removed. Due to minimal relief of symptoms, on days 11 and 14, repeat amniocenteses were performed, each removing 900 cc of fluid. Ritodrine IV was used to stop ensuing contrastions, but cervical change was noticed on maximal IV doses. Therefore, oral nifedipine was administered (80 mg/day) and uterine activity ceased. Subsequently, four amniocenteses removed 300 cc of amniotic fluid daily (a total of 3500 cc) without causing uterine activity. Steroids were administered twice during the hospital stay. The serum protein on admission was 5.84 g/dl; protein intake was 2.5 g/kg/day and the serum protein level did not show any significant change despite the repeated amniocenteses.

Repeat ultrasound revealed normal growth of twin A, arrested growth of twin B, enlarged umbilical vein of twin A with no sign of hydrops. The enlarged umbilical vein of twin A, the discrepancy in their growth and the size of their amniotic sacs led us to the presumptive diagnosis of twin-to-twin transfusion. Nifedipine was discontinued at 28 weeks. At this time, the patient refused further amniocenteses. Three days following the last amniocentesis, contractions recurred with further dilatation of the cervix. At this time the biophysical profile was 8 out of 10 for twin A and 4 out of 10 for twin B. Prior to this, daily fetal assessments had been normal. Because of fetal distress in twin B, a cesarean section was performed. Identical twin girls were delivered. Twin A (the recipient) had Apgar scores of 4 and 7, weighed 1265 g, and had an hematocrit (Hct) of 77%. She required intubation and mechanical ventilation for a week and had a reduction exchange transfusion on the first neonatal day. The postexchange Hct was 64%.

The neonatal outcome of twin A was significant for bilateral periventricular leukomalacia without hyperactivity confirmed by ultrasound and CT scan, a transient left upper extremity monoparesis and normal neurological development at three months of age. The infant weighed 2600 g at discharge on day 66 of life.

Twin B weighed 790 g at birth with Apgar scores of 1 and 5. Her postnatal course was complicated by mild hyaline membrane disease requiring intubation, anemia to a Hct of 37% for which she was transfused on day 1, and neonatal hyperbilirubinemia for which she underwent a double volume exchange transfusion on day 3. On day 6 her condition suddenly deteriorated and she expired from what postmortem examination revealed to be a massive pulmonary embolus. The placenta was found to be diamniotic monochorionic with several large anastomoses. The cord of twin A measured 2.0 cm in diameter and that of twin B measured 0.8 cm.

CASE 2

S.M., 33 years old gravida 2, para 1, with one previous term vaginal delivery, was transferred to our hospital at 26.5 weeks gestation with a twin gestation, acute polyhydramnios and premature contractions. She was being treated with IV ampicillin for a presumed urinary tract infection as noted by right flank pain and an abnormal urinalysis. Her medical history was remarkable for retinitis pigmentosa and congenital deafness. Her admitting physical examination was remarkable for a uterine fundal height of 52 cm, right costovertebral angle tenderness, abnormal urinalysis, a cervix dilated to 3 cm, 75% effaced, bulging of the fornices and occasional contractions.

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Ultrasound revealed a twin gestation with massive polyhydramnios in the gestational sac of twin A, a single posterior placenta and a dividing membrane. Twin A was vertex, had a BPD of 7.4 cm, FL of 5 cm, EFW 1259 g, no signs of hydrops and good fetal activity. Twin B was in transverse lie, with a BPD of 5.8 cm, FL of 4.5 cm, EFW 640 g, reduced amount of fluid and it was less active than its cotwin. Biophysical profiles on both infants were 8/8. A 50 g glucose challenge was negative; blood type was A positive and antibody screen was negative; there were no definable abnormalities by ultrasound . Treatment on admission included IV ritodrine, steroids and ampicillin. Eighteen hours after her admission the patient developed chest pain without ECG changes. Ritodrine was discontinued and IV magnesium sulfate was administered for tocolysis. Ten hours later cervical change (4 cm) was noted, $MgSO_4$ was stopped and nifedipine (20 mg PO every 6 hours) was started. This continued until delivery.

Serial amniocenteses for extreme abdominal discomfort and premature contractions were performed on days 1, 2, 3, 5, and 11. The amounts of fluid removed ranged from 73 cc to 1200 cc. A total of 4750 cc were removed. Her hospitalization was further complicated beginning on day six by stable preeclampsia as manifested by elevated blood pressure of 130/90 mmHg and mildly elevated transaminases. Her serum albumin fell from 3.12 g/dl to 2.9 g/dl.

Two hours following the last amniocentesis the patient began contracting. Her cervix was found to be 9 cm dilated with no presenting part palpated. An immediate cesarean section was performed and two viable female infants were delivered both requiring mechanical ventilation. Twin A had Apgar scores of 3 and 6, and weighed 1140 g with a cord that measured 2 cm in diameter. After transient anuria the baby did well. Twin B had Apgar scores of 4 and 8, and weighed 665 g with a cord that measured 0.5 cm in diameter. She developed renal cortical necrosis that led to neonatal death. The placenta was monochorionic with 80% of the weight belonging to twin A, with several large anastomoses. There was no significant difference in the hematocrits between the twins.

Post operatively, the patient's preeclampsia worsened and she was treated with magnesium sulfate for 36 hours. Otherwise, her course was unremarkable.

DISCUSSION

The first reported case of amniocentesis as a treatment for polyhydramnios in twin pregnancy was in 1944 by Erskin [9]. This was done to prolong the pregnancy until spontaneous resolution of the condition occurred following the death of the donor twin.

More recently, Brown reported of a case in which both fetuses survived [6]. In 1980 he reported another case [7] in which 6700 cc and 4000 cc were removed by amniocentesis at 28 and 32 weeks, respectively, with a successful delivery of both twins. The indication for the amniocentesis was not acute polyhydramnios but rather severe vulvar edema. The largest study is that of Weir et al [14]. His conservative approach resulted in 100% perinatal-neonatal mortality. Mills [10] reports a better success rate and believes that amniocentesis should start immediately and as much fluid as possible should be removed each time. He reported a case requiring 13 amnio-

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centeses before delivering the patient at 33 weeks gestation.

Avris [1] attempted removing of large quantities of fluid (1.2-1.9 l) in treating 5 of his patients but only 3 babies survived.

The major consideration in the therapy is the amount of amniotic fluid to be removed with each amniocentesis. The amount of fluid to be removed at each amniocentesis should ideally be enough to relieve symptoms, but not enough to induce uterine activity. This amount may vary in each individual case. Our first patient tolerated the removal of 500 cc without initation of contractions, but with little symptomatic relief. The removal of 900 cc relieved her discomfort but initiated contractions that failed to stop with maximum doses of IV ritodrine and stopped only with PO nifedipine. An additional 4 amniocenteses, removing 300 cc each, under nifedipine coverage, did not provoke contractions. Our second patient was put on ritodrine and was later changed to magnesium sulfate. The repeated amniocenteses resulted in cervical dilatation that stopped when nifedipine was used, but 2 hours following the fifth amniocentesis she started contracting again and dilated her cervix to 9 cm.

Therefore, it is our feeling that one has to tailor the amount of fluid that is removed each time and to use prophylactic tocolysis. In addition to the initiation of uterine contractions, there are other complications associated with serial amniocentesis; it is believed that the removal of large quantities of amniotic fluid might cause premature separation of the placenta [11,12]. This was not our experience or the experience of those who removed even larger quantities of fluid than we did. We believe that the slow removal of fluid is the key in reducing the likelihood of this complication.

The psychological impact of repeated amniocenteses on the mother should not be underestimated. Despite the best support we could supply to our first patient, she refused further amniocenteses after 28 weeks and soon after went into labor. We did not experience a decrease in serum proteins as a result of our serial amniocentesis. This could be explained by the relatively small amount of fluid that was removed in our two patients. Queenan [12] reports a marked decrease in serum proteins (from 7.2 to 5.3 g/dl) after the removal of 7820 ml of amniotic fluid over a 3 week period. This required the adminstration of 25 g of albumin intravenously. Therefore, we believe that one should increase the protein intake in the patient's diet, follow her serum protein level and be prepared for IV supplementation when it drops.

An extremely rare complication of amniocentesis is an amniotic fluid embolism [13]. In face of the high mortality rate associated with this event, it should be explained to the patient and a trained anesthesiologist should be available to initiate an immediate and aggressive treatment.

Our experience with nifedipine in premature labor is the subject of another report [8]. Because the survival rate at our institution for a twin pregnancy at 28 weeks is 85% and experience with nifedipine is limited, we consider it prudent to discontinue its use at 28 weeks.

The decision to intervene in a twin pregnancy when one fetus is compromised is always difficult. Because of good survival statistics at 27 weeks, in our hospital, we would intervene at this point if either of the twins were showing signs of distress. The first patient reached 28 weeks and 3 days, and because there was evidence of fetal distress in one twin, a cesarean section was performed. If we had not intervened with repeated amniocenteses, it is almost certain that delivery would have occurred at 26

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weeks with a slim chance for survival [14]. It is unfortunate that despite prolongation of the gestation by 14 days the outcome was less than what we hoped for. However, the death of the donor twin B in Case 1 was due to an unexpected pulmonary embolus, possibly related to a complication of the transfusion. The fact that the recipient twin A has periventricular leukomalacia may be related to the hyperviscosity syndrome. Although the prognosis is guarded, ongoing follow-up of this baby indicates that the outcome may be reasonable.

In our second patient, we managed to prolong the pregnancy by 11 days, carrying her from 26.5 weeks gestation (with slim chances of survival of any of the twin babies) to 28 weeks with much higher chances of survival. The rare renal complication that resolved in one twin and persisted in the other is again unfortunate, but at least one baby survived.

In summary, there are few options for the management of twin-to-twin transfusion in the late second trimester. Our management of repeat amniocenteses is a safe procedure which may allow symptomatic relief, prevent premature labor and enable continuation of the pregnancy to gestational age compatible with survival. The combination of serial amniocenteses, tocolysis and close fetal surveillance may offer some hope in an otherwise hopeless situation.

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