Emergent intrauterine resuscitation in a fetus with transient congenital anemia - case report

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Summary

A 24-week gestation complicated by fetal hydrops was prepared for intrauterine transfusion to treat possible fetal anemia. During the therapeutic transfusion procedure, blood samples could be not taken from the umbilical vein, and severe fetal bradycardia developed. Given the severity of the condition and the fetal position, transfusion was performed through the most accessible part of the fetal heart, which was the right atrium. Just after the transfusion procedure, fetal cardiac tamponade developed, and the fetal heart collapsed. Subsequently, urgent fetal pericardiac tamponade decompression was performed.

Key words: Cordocentesis; Intrauterine transfusion; Cardiac tamponade; Pericardiocentesis.

Introduction

Intrauterine intravenous transfusion can be performed in the treatment of fetal hydrops complicated with fetal anemia. The procedure has complications like fetal loss, vascular damage, fetal bradycardia, and preterm labor [1]. When it is not possible to perform the transfusion through the umbilical vein, intracardiac transfusion can be chosen as an alternative route which is more dangerous. The intracardiac transfusion procedure has severe complications like fetal loss, bradycardia, asystole and pericardiac tamponade [2].

We report a case of intrauterine transfusion complicated with fetal bradycardia, which was overcome with intracardiac transfusion, but complicated with pericardiac tamponade and treated with urgent pericardiac tamponade decompression incidentally.

Case Report

A 34-year-old woman was referred to our clinic in the 24th week of her gestation due to a diagnosis of fetal hydrops. In the detailed scan, fetal abdominal ascites, pleural effusion, a MCA-PSV level of 88.7 cm/s (> 2 MoM), a dicrotic pattern in the umbilical artery blood flow and a pulsatile flow in the umbilical vein were detected. The indirect Coombs test was negative, and the patient was found to be immunized for toxoplasmosis, rubella and cytomegalovirus. Cordocentesis was performed. During the procedure, a small quantity of dark red fetal blood was removed from the umbilical vein. Hematological and serological parameters were: hemoglobin (Hb) 1.7 g/dl, hematocrit (Hct) 5.14%, a negative result for Parvovirus B19 IgM and a positive result for IgG. Due to severe fetal anemia, we decided to perform intrauterine intravascular transfusion the following day. A Type O, Rh (-), freshly packed, cytomegalovirus antibody-negative, irradiated erythrocyte suspension with Hb of 22.6 g/dl and 62.8% Hct was prepared. Pancuronium bromide was used to paralyze the fetus. Using a 22-gauge spinal needle, we twice attempted to aspirate blood in order to measure the initial Hb level. Despite the correct positioning of the tip of the needle in the umbilical vein of the transplacental cord, no sample could be taken. Meanwhile, acute fetal bradycardia lasting more than 60 seconds and vasospasm were detected, so we decided to execute an emergent intrauterine intracardiac transfusion instead of accessing the hepatic portion of the umbilical vein. We immediately entered the amniotic cavity with a new 22-gauge spinal needle and directed it to the fetal heart. Given the position of the fetus, it was impossible to puncture either of the ventricles, so we penetrated the right atrium as the most accessible heart region and took a blood sample (fetal Hb level was not measured and Hct level was 2.9%), and we then transfused 150 ml of erythrocyte suspension into the right atrium. After the transfusion, the fetal heart rate recovered to normal. After removing the needle, an echo lucent area developed around the fetal heart. The heart started flickering, and heart dimensions decreased. Pericardiac tamponade was therefore strongly suspected, and in response, the pericardial area was again penetrated with the spinal needle, and 6 ml of blood was promptly taken. Thereafter, we observed that the heart became dilated, the dimensions returned to normal and fetal cardiac activity improved within a few seconds. After the transfusion, fetal Hb and Hct levels and MCA-PSV were all normal. Routine follow-up was performed. Two weeks after the transfusion oligohydramnios was detected. Thereafter, the amniotic fluid index (AFI) was increased to 18 cm on the third week of transfusion and increased echogenity was detected, possibly due to intraamniotic bleeding. Six weeks after the transfusion AFI was 12 cm.

At the 32nd gestational week, two months after intrauterine transfusion, pleural effusion and increased MCA-PSV (66.4 cm/s) were diagnosed and a second transfusion through the intravascular route was successfully performed. At the 35th week of pregnancy, due to premature rupture of membranes and a previous cesarean scar, a cesarean section was performed. At the end of the first postpartum month, the baby developed anemia, and a third transfusion was performed postnatally. The pediatric hematology unit made a diagnosis of transient intrauterine hematopoiesis repression, possibly due to Parvovirus B19 infection. After two years of follow-up, the child was healthy and had normal developmental scores.

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Discussion

We have reported an unusual case in which an intrauterine intravascular transfusion performed to treat severe anemia was adjusted to an intracardiac transfusion procedure upon complication by fetal cardiac tamponade, and which we managed with urgent pericardiocentesis. Several points warrant discussion. First, regarding the repeated cordocentesis on the running day, Mari *et al*. [3] reported a false-positive rate of 12% for MCA-PSV. Therefore, we suspected a possible false-positive MCA-PSV and decided to perform a two-step approach for diagnosis and transfusion. In our opinion with this approach, we obviate preparing blood due to a false indication.

Another point related to repeated cordocentesis is the risk of fetal loss in a hydropic fetal condition. However, this risk is actually not that high when the procedure is performed correctly. Previously, van Kamp *et al*. [1] reported perinatal loss rates per procedure in the absence and presence of fetal hydrops as 1.4% and 2.5%, respectively. Nonetheless, the failure of the second cordocentesis procedure could be attributed to severe fetal anemia and low intravascular pressure as well as local vasospasm that might have developed due to the procedure.

Another event of this case was that the intrauterine intracardiac transfusion was performed via the right atrium. The safety of ventricular punctures, especially the apical part of the left ventricle, has previously been reported [4, 5]. This should be the site of choice both anatomically and physiologically because it is safe and distant from the great arteries and the transfused blood will easily traverse to the fetal brain and fetal coronaries, as well as general circulation and the placenta. Although when transfusion via the umbilical vein fails, the intrahepatic route should be considered as a secondary option for access. However in our case, due to the speed of events, gravity and urgency of the life-threatening condition, we entered into the closest and most accessible part of the fetal heart that the fetal position permitted. Westgren *et al*. [2] reported two cases of right atrium puncture during intracardiac transfusion. One of their cases was complicated with hemopericardium, while the other case was complicated with severe fetal bradycardia. In our opinion, this case and Westgren *et al's* experience suggest that great caution should be taken with puncture of the right atrium, which might end with hemopericardium collection. The most common complications of intracardiac transfusion are severe bradycardia and asystole, which might occur even when the transfusion is performed through the left ventricle. Another serious complication following intracardiac transfusion is fetal death [6, 7].

The fluctuation in the AFI after the transfusion could be related with the olyguric and polyuric resolution phases of acute renal failure of the fetus due to severe anemia. An important clinical observation is that fetal physiologic reactions may mimic the same physiological renal response as in adults in the case of severe fetal hypotension. This observation can be re-evaluated in experimental animal models to make a strict conclusion.

In summary, this case shows that in cases of fetal hydrops and severe fetal anemia, when intravascular transfusion is complicated, fetal intracardiac transfusion could be life-saving. As a caution, when intracardiac transfusion is selected, the ventricles should be the main or ideal targets of the needle. If the right atrium is chosen instead, the risk of cardiac tamponade development should be taken into account, and the operator must be prepared for urgent pericardiocentesis.

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