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Ductus Venosus Flow Velocity in Acute Fetal Congestive Heart Failure During Fetal Transfusion

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Objective: To evaluate ductal flow pattern in a fetus in acute congestive heart failure.
Methods: We used real-time, M-mode, pulsed wave, and color Doppler imaging to study a fetus with severe Kell erythroblastosis fetalis and acute right ventricular congestive failure after intravascular transfusion.
Results: We documented reverse flow velocity waveforms in the ductus venosus.
Conclusion: Fetal ductus venosus may play a physiologic role in the alleviation of congestive heart failure.

Key words: Kell sensitization—Ductus venosus Doppler—Congestive heart failure—Fetus—Intrauterine transfusion.

Introduction

Ductus venosus is a venous connection between the intrahepatic portion of the umbilical vein and the inferior vena cava, which is functional only during intrauterine life. The existence of an anatomical or functional sphincter has been suggested.[1] This sphincter regulates the blood flow from the placenta to the right atrium. Ductus venous flow velocity waveforms have been obtained by means of pulsed wave Doppler methodology in normal pregnancies.[2] In this report we describe the changes in the flow pattern of the ductus venosus immediately after intravascular fetal transfusion in a hydropic immature fetus at 21 weeks.

Case Report

HMc is a 27-year-old caucasian female, G_2P_{1001} with Kell isoimmunization. Percutaneous fetal blood sampling at 21⅗ weeks gestation revealed a Kell positive fetus with Hb of 2.5 g/dl, Hct of 7.6%, and direct Coombs of 2+. We transfused the fetus with 15 ml of appropriately prepared packed red blood cells (Hct of 76%) at a rate of 1–1.5 ml/min. Post-transfusion Hct increased to 24.7% and Hb to 8.4 g/dl. M-mode measurement of the right ventricular inner diameter was greater than the 95th percentile for gestation while the left ventricular inner diameter remained in the upper normal range for gestation.[3] Real-time M-mode evaluation revealed minimal right ventricular contractility (systolic fractional shortening less than 15%). Fetal heart rate increased to 188 bpm. Color Doppler imaging during diastole revealed a scant amount of right ventricular inflow, and pulsed wave Doppler imaging revealed the tricuspid peak flow velocity to be at less than 10 cm/s. Umbilical vein flow pattern became pulsatile, and inferior vena cava flow velocity waveforms (FVWs) demonstrated significant reverse flow component (>30%). Concurrently, the ductus venosus flow pattern became biphasic with a significant reverse component (Fig. 1). These findings represent substantial changes from the previously normal waveforms. Middle cerebral artery FVWs revealed significantly decreased pulsatility index of 0.59 (5th percentile = 1.5). Ten min later right ventricular contractility started to improve along with the central and peripheral Doppler findings. The flow velocity of the ductus venosus demonstrated continuous improvement and returned to normal with biphasic forward flow pattern over the next several hours post-transfusion. Prior to discharge, right ventricular systolic fractional shortening ranged between 30 and 40%. The patient was discharged, and follow-up fetal evaluation the next day revealed normal cardiovascular function. After a series of five uncomplicated transfusions, the patient was delivered by elective primary cesarean section at 34 weeks. At 18 months of age, the infant is in good health with normal physical and neurologic development.

Comment

Ductus venosus (DV) is an important fetal vascular structure, which obliterates afterbirth. It has been suggested that DV regulates fetal flow of oxygenated blood from the placenta to the heart by constriction and dilatation in response to variable fetal oxygen content or PO_2. There is limited published experience with regard to DV flow velocity

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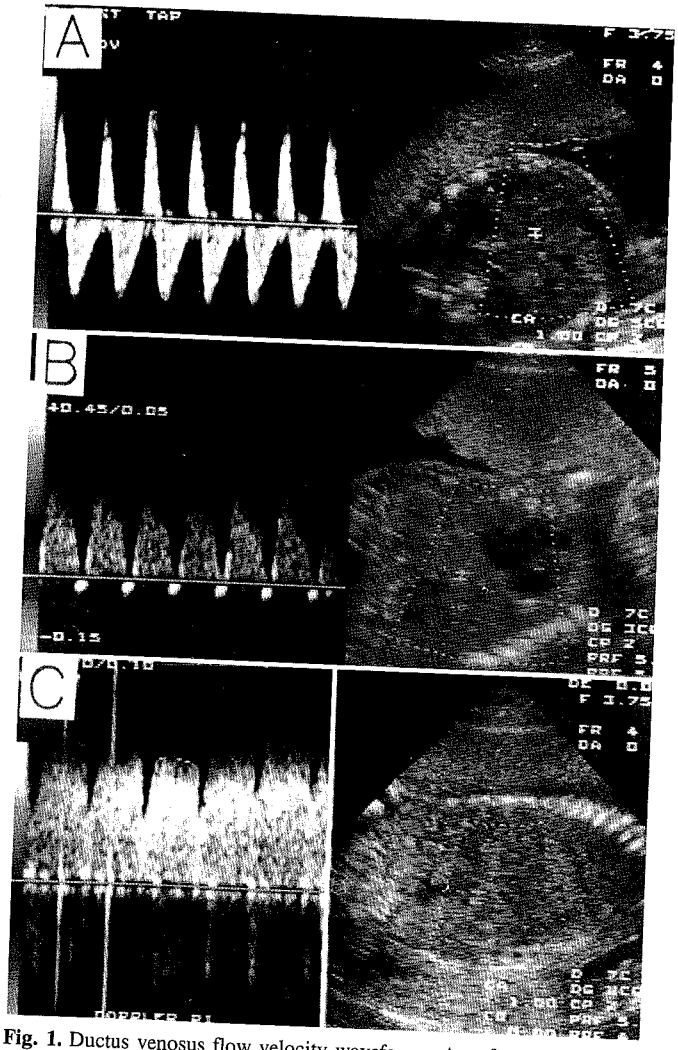

Fig. 1. Ductus venosus flow velocity waveforms: A, a few minutes after completion of the transfusion; B, approximately 30 min after transfusion; and C, several hours after transfusion.

waveforms. Recent evidence with Doppler documented the role of DV in the normal fetus and the absence of reverse flow patterns.[2] The authors of that report speculated that the interstitial placental pressure exceeds the venous pressure. This may be true under normal conditions, but it apparently changes under conditions of acute cardiac congestion as in this case.

Several factors may have played a role in the right ventricular failure in this fetus. Although the transfused blood volume was not overestimated (post-transfusion fetal hemoglobin was only 8.4 g/dl), volume overload may still have been one of the reasons for the fetus' congestive heart failure. Preexisting myocardial depression from severe anemia may have also contributed significantly to the cause of the cardiac failure. A less important but not negligible reason could have been the immaturity of the fetus whose heart was unable to cope with the additional volume (decreased compliance). The increased pressure in the central venous system may have caused secondary volume shifting into the placenta and possibly the hepatic sinusoids. The small amount of blood entering the right ventricle was returned to the right atrium, and the circulation was sustained by the left ventricle alone. During atrial systole, a substantial amount of blood moved in reverse direction through the DV. We have no evidence of the possible mechanism of this phenomenon, although it appears that the changes that took place were a passive response to increased central venous pressure and not the result of some physiologic protective mechanism. Regardless of the physiology involved, we believe that this mechanism may have allowed the fetus to overcome the state of acute right ventricular failure and gradually return to a normal cardiovascular state with competent right ventricular function.

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