

Video 5. Complete disappearance of mitral regurgitation following Amplatzer occluder implantation

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Intrauterine idiopathic severe ductal constriction diagnosed by fetal echocardiography: a cause of hydrops fetalis

Intrauterin dönemde fetal ekokardiyografi ile tanı alan ve hidrops fetalise yol açan idiyopatik ciddi duktus darlığı

Introduction

The premature closure or constriction of the ductus arteriosus (DA) is rare and can result in right heart failure, fetal hydrops, persistent pulmonary hypertension and death (1-3). Thus, the recognition of this condition is of vital importance.

We diagnosed idiopathic severe ductus constriction in a fetus whose mother had no risk factors for DA restriction. The case is described with the features of prenatal and postnatal fetal echocardiography.

Case Report

A 29-year-old gravida 2, para 1 woman was referred to our department at 33 weeks of gestation for fetal echocardiography. Obstetric ultrasound examination showed normal fetal growth with a small pleural effusion, and a moderate amount of ascites and polyhydramnios. The patient had no history of medication use during pregnancy, including anti-inflammatory drugs.

Fetal echocardiography ruled out structural cardiac defects; however, it demonstrated a prominent, dilated and hypertrophic right ventricle and an enlarged right atrium and moderate tricuspid regurgitation (Fig. 1 and Video 1. See corresponding video/movie images at www.anakarder.com).

Estimate of right ventricular pressure was 80 mmHg. A small pleural effusion and moderate ascites was also observed. The DA was difficult to visualize, and the flow pattern at the ductus insertion site was consistent with constriction of DA (Video 2. See corresponding video/movie images at www.anakarder.com). Ductal velocity was at least 2.68 m/sec during the systolic phase and 1.57 m/sec during the end-diastolic phase (Fig. 1). The pulsatility index was 0.68. We decide to deliver the baby due to hydrops fetalis, and a cesarean section was performed one day after the mother's examination. A male infant weighing 3.4 kg was delivered at 33+1 weeks of gestation with Apgar scores of 8 and 9 after 1 and 5 min, respectively. The baby was noted to be tachypneic soon after birth and required supplemental oxygen with an FiO₂ of up to 30%; however, mechanical ventilation was not required. The infant was noted to have hydrops with common ascites and subcutaneous edema. An echocardiogram was performed within one hour of birth, which revealed spontaneous closure of the DA. Thus, the prenatal diagnosis was confirmed. A repeat echocardiogram showed a decrease in right ventricular hypertrophy with functional improvement and a marked decrease in the severity of tricuspid regurgitation. There was no risk factor for hydrops fetalis in this infant with the exception of prenatal DA constriction. The neonate was without clinical complications after delivery. The infant's ascites and subcutaneous edema resolved gradually. He was discharged on day 8 weighing 2.9 kg. An echocardiogram performed at this time showed no abnormalities.

Discussion

In utero closure of DA is rare. The patency of DA in utero is largely maintained by high levels of circulating PGE₂ and locally produced PGE₁. Thus, it has been noted that maternal administration of PG synthase inhibitors, such as NSAIDs and corticosteroids, is associated with an increased risk of premature closure of the PDA (4, 5). Another cause of premature PDA closure is the ingestion of polyphenols (i.e., herbal teas, grape and orange derivatives, dark chocolate, berries) (6).

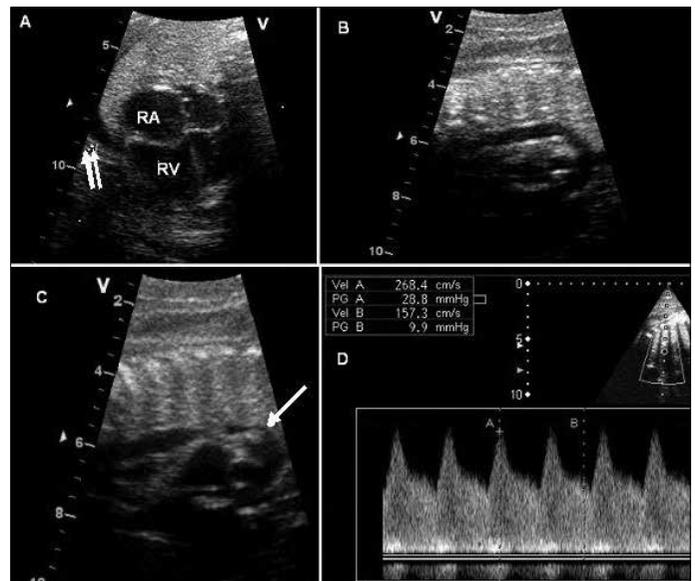


Figure 1. A) The four-chamber view shows marked right ventricular enlargement and hypertrophy as well as right atrial enlargement and a pleural effusion (white double arrow). B) Normal aortic arch in the sagittal view. C) Constriction of the ductus arteriosus in the sagittal view (single arrow). D) Pulsed wave Doppler flow showing a peak velocity of 2.60 m/sec in the systolic phase and 1.5 m/sec in the end-diastolic phase

There are a few additional case reports that have described idiopathic fetal ductus constriction/closure without evidence of maternal use of a PDA constricting agent or structural cardiac defect (7, 8).

In this case, we excluded all known secondary causes of premature ductus constriction by history, including NSAIDs, teas, and herbal remedies as well as structural cardiac and ductus abnormalities. We believe that our case represents idiopathic premature restriction of the DA. We have no clear explanation or theory as to why this ductus was restricted.

In the healthy fetus, ductal flow velocity increases gradually with advancing gestational age. In previous reports, ductal restriction has been defined as a peak systolic velocity of greater than 1.4 m/s, a persistent diastolic peak flow velocity greater than 0.35 m/s and a pulsatility index less than 1.9 (9, 10). Each of these criteria was found in our patient. The four-chamber view is a key to the detection of ductal constriction or closure. Although a dilated right ventricle and significant tricuspid regurgitation may be the first indication of DA restriction on this view, when the constriction is initially occurring, the four-chamber view may appear to be normal. Thus, the diagnosis should be made based on the velocity of the DA upon Doppler examination in the sagittal view. Thus, the determination of DA velocity should be a routine part of fetal echocardiographic examination.

Conclusion

The outcomes associated with premature ductus constriction include right heart failure, fetal hydrops, persistent pulmonary hypertension and death. The wide spectrum of clinical presentations is dependent on the duration and severity of flow obstruction. If ductal arteriosus flow is not checked using color and pulsed Doppler, the diagnosis of this serious condition could be missed.

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Video 1. A four-chamber view showing marked right ventricular enlargement and hypertrophy with tricuspid regurgitation

Video 2. A Sagittal views showing that the ductus arteriosus is severely constricted. Color flow imaging of the ductus arteriosus shows a narrowed jet with increased velocity, indicating ductus arteriosus constriction

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Subclavian artery stenosis in a patient undergoing coronary bypass using composite t-grafting technique: is it subclavian artery stenosis or more?

Kompozit T-graft tekniği kullanılan koroner baypass bir hastada subklavian arter stenozu: Subklavian arter stenozu ya da fazlası?

Introduction

Occlusive arterial disease of the upper extremity occurs at a much lower frequency than disease of the lower extremity. They are mostly seen in the subclavian and the innominate artery. The common cause is atherosclerosis (1). With an increase in the use of internal mammary artery in heart surgery, detection of subclavian artery stenosis (SAS) has gained importance. In a patient with history of coronary bypass surgery (CABG) 6 months ago, we determined SAS. What makes this case interesting is that left internal mammary artery (LIMA) was anastomosed to the left anterior descending artery (LAD); furthermore, the proximal anastomoses of the arterial grafts belonging to the circumflex (CX) and the right coronary arteries (RCA) were anastomosed to LIMA using the composite T-grafting technique. Hence, perfusion of the heart was rendered totally dependent on LIMA and consequently on the left subclavian artery.

Case Report

A 65-year-old woman was admitted to our clinic with pulmonary edema. Her medical history revealed that she had undergone coronary artery bypass grafting with a LIMA conduit to the LAD and with a composite T-grafting technique applied to the RCA and CX six months ago. On