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Monochorionic Twin Pregnancy Complicated by Arrhythmia in One Twin: Longitudinal Hemodynamic Effects of Flecainide Cardioversion Assessed by Fetal Venous Doppler

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Abstract

A parous woman with a monochorionic twin pregnancy presented at gestational week 25 with an uncommon situation. One twin had developed hydrops, but not due to placental transfusion syndrome. The twin had a supraventricular tachyarrhythmia, a rare and severe complication that untreated would threaten the lives of both twins. We present the longitudinal Doppler ultrasound findings when assessing the hemodynamic effects of the arrhythmia and the transplacental treatment with antiarrhythmic medication (flecainide), and how the arrhythmia and the conversion to normal rhythm influenced umbilical and liver circulation in the fetus.

Keywords

- antiarrhythmic drugs
- Doppler ultrasound imaging
- fetal placental circulation
- fetal therapy
- maternal fetal exchange
- monochorionic twins
- tachyarrhythmia

Introduction

Fetal tachyarrhythmia occurs in 0.4 to 1% of singleton pregnancies.[1,2] The incidence in monochorionic (MC) twin pregnancies is not known. Fetal tachyarrhythmias are predominantly supraventricular and can cause cardiac failure, resulting in a reduction in fetal-placental circulation and fetal hydrops with a reported mortality of approximately 17%.[3] In MC twins, little is known about the consequences of cardiac failure due to arrhythmia and its treatment for the unaffected twin.

Transplacental antiarrhythmic treatment has proven successful in singletons with over 60% conversion rate.[4] Flecainide is preferred over digoxin in cases complicated by hydrops due to more effective transplacental transfer, even though similar results are reported with a more aggressive and invasive therapeutic approach.[5,6] We report a case of MC diamniotic (MCDA) twins where one twin developed tachyarrhythmia with hydrops and transplacental antiarrhythmic treatment was successful.

Case Presentation

A 29-year-old parous woman, now pregnant with MCDA twins, had two uneventful pregnancies and births, and there was no

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family history of congenital heart disease. Routine follow-up detected no structural cardiac anomalies, no sign of twin–twin transfusion syndrome, and normal growth patterns in both twins. At 25 weeks, a sustained tachyarrhythmia and fetal hydrops with ascites were diagnosed in twin 1. The woman was transferred to our center for evaluation and treatment.

We found that twin 1 had hydrops with ascites, pericardial effusion, and an echogenic bowel. The placenta appeared more echogenic and thicker than that of the unaffected twin 2 (►**Fig. 1A** and B). The ventricular rate assessed by pulsed wave Doppler across the atrioventricular (AV) valves, in the pulmonary artery and veins, and the umbilical artery was 223 to 244 bpm, and the arrhythmia was classified as supraventricular with 1:1 conduction to the ventricles. Pulsed wave Doppler in the affected twin's ductus venosus showed a severely reversed a-wave, and pulsations in the intraabdominal portion of the umbilical vein (UV; ►**Fig. 1C** and D). The venous liver circulation was compromised with undulating and reversed flow in the left portal vein (LPV; ►**Fig. 2** and ►**Video 1**) and the UV flow (Q_{UV}) was low (►**Fig. 3**).

Video 1

Compromised umbilical venous flow to the fetal liver and rapid arrhythmic pulsations of reversed flow in the left portal vein by pulsed wave Doppler. Horizontal oblique view of the abdomen of the affected twin. Online content including video sequences viewable at: https://www.thieme-connect.com/products/ejournals/html/10.1055/s-0043-1774753.

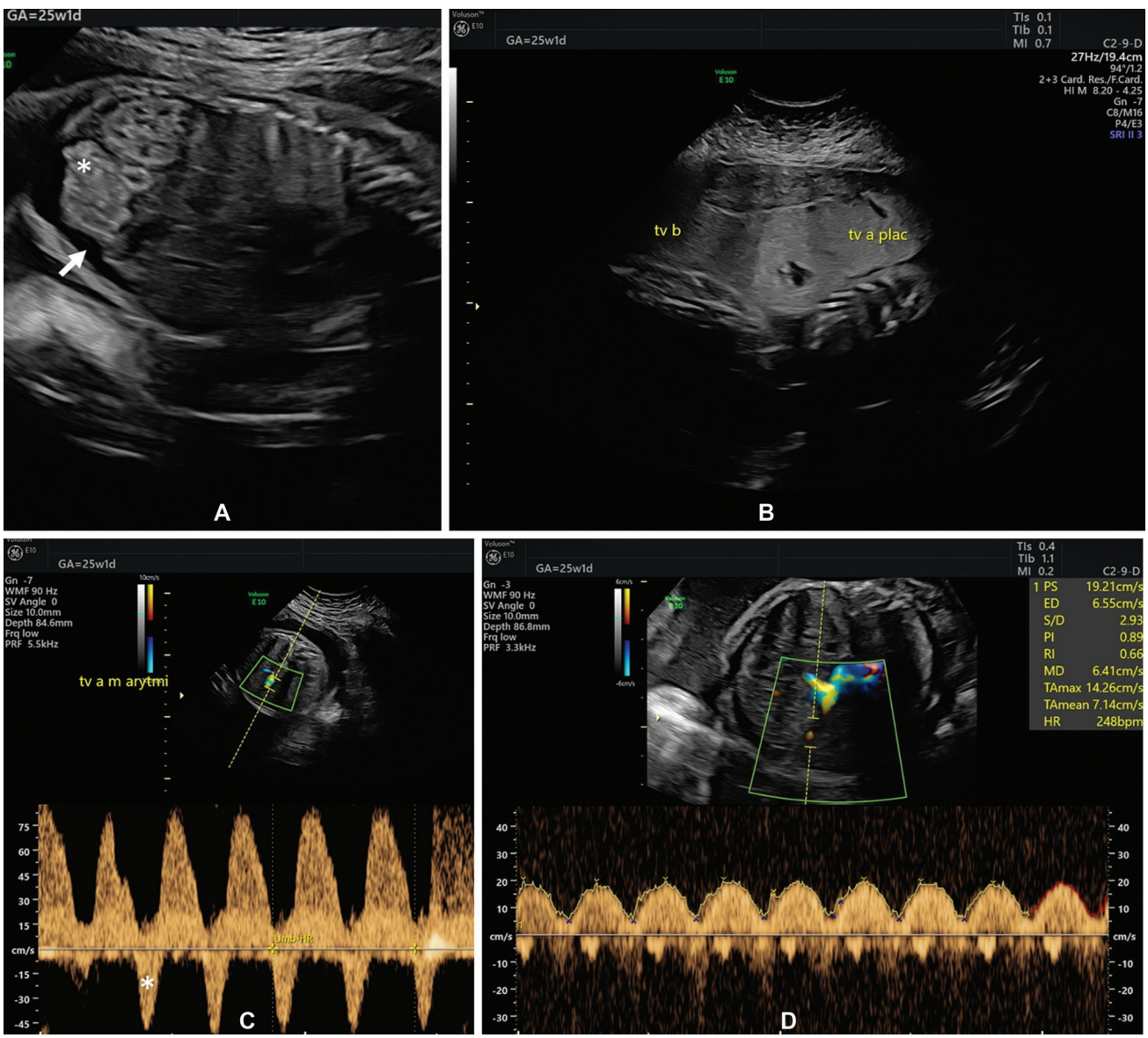

Fig. 1 (**A**) Ascites (arrow) and echogenic bowel (*) in the twin with hydrops. Oblique sagittal view of the fetal abdomen. (**B**) Placentae in monochorionic twins. Placenta of the affected twin with edema to the right. (**C**) Severely reversed a-wave (rate 223 beats per minute) in the ductus venosus by pulsed wave Doppler in the affected twin (*). Horizontal view of the fetal abdomen. (**D**) Pulsations in the intra-abdominal umbilical vein (UV) by pulsed wave Doppler in the affected twin (rate 248 beats per minute). Oblique horizontal view of the fetal abdomen.

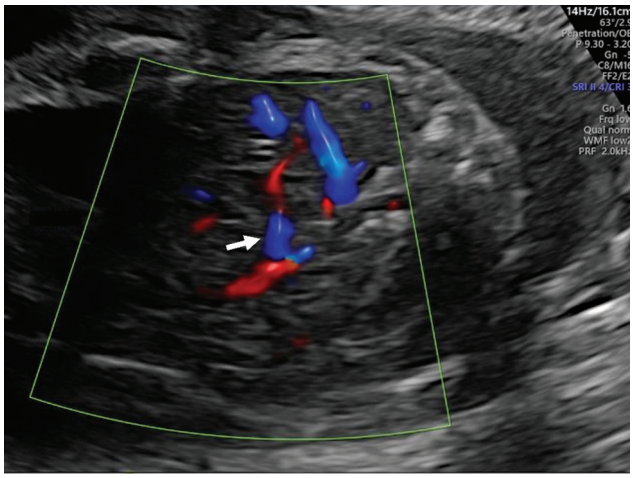

Fig. 2 Reversed blood flow in the left portal vein (LPV) by pulsed wave Doppler in the affected twin (arrow).

The examinations did not identify any fetomaternal explanation for the tachyarrhythmia. Examinations of the mother prior to initiation of treatment were performed according to the institutional guidelines. Maternal echocardiography and echocardiogram (ECG), electrolytes, and thyroid hormone levels were normal. Transplacental medication with per oral 100 + 50 + 100 mg flecainide and administration of betamethasone was initiated due to the risk of preterm delivery. The mother was monitored with continuous ECG during the initiation and adjustment of flecainide dosage and showed a minor increase in QRS duration to 105 ms, QT duration of 370 ms (QTc duration of 420 ms). Serum levels of flecainide were between 1.0 and 1.5 µmol/L (0.5–2.4 µmol/L) during initiation and follow-up of treatment.

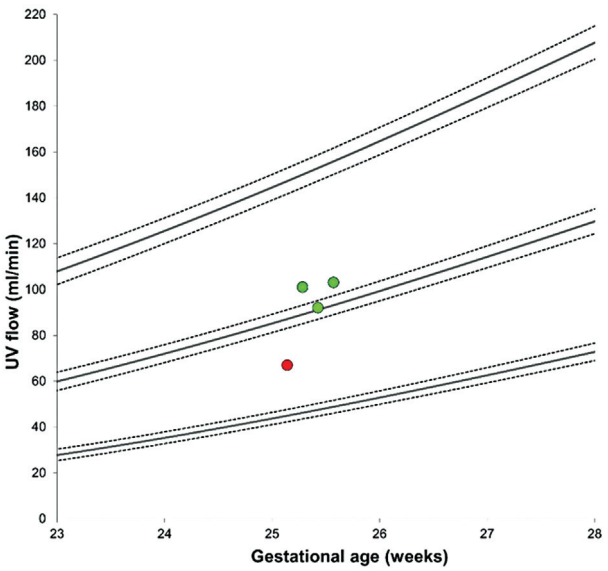

Fig. 3 Serial measurements of the umbilical vein (UV) flow in the affected twin before (red dot) and after cardioversion (green dots; solid lines indicate the 5th, 50th, and 95th centiles and dashed lines indicate the 95% confidence intervals). (Adapted from Ebbing C. Fetal splanchnic arteries. A longitudinal study and hemodynamic relations to common Doppler parameters. 2008. Accessed August 14, 2023 at: https://bora.uib.no/bora-xmlui/handle/1956/3158.)

Conversion to sinus rhythm in the fetus was successful 24 hours after initiation of treatment, with a short relapse of supraventricular tachycardia (SVT), following an increase in flecainide dosage to a total of 300 mg/24 h, after which no further arrhythmias were recorded. The mother tolerated this dosage well. The ascites in twin 1 resolved after 5 days, and fetal Doppler velocities (middle cerebral, umbilical artery, UV, and ductus venosus) and Q_{UV} normalized (►**Fig. 3**). The unaffected twin showed no signs of hemodynamic compromise before or after flecainide treatment.

After 5 days, the mother was transferred to her local hospital for further follow-up. At 28 weeks of gestation, the placentas appeared with equal thickness and echogenicity, indicating resolved placental edema. At gestational weeks $35^{3/7}$, she had a spontaneous vaginal delivery of two healthy girls. Twin 1 and 2 had birthweights and hemoglobin levels in the umbilical cord of 2,506 and 2,052 g and 23.5 and 17.4 g/dL, respectively. Both had normal Apgar scores, and pediatric evaluation was normal in both twins.

Discussion

Reported cases of twin arrhythmia are scarce, especially in MC pregnancies (►**Supplementary Table S1**, available in the online version).[7] These cases may represent a challenge in a situation where one twin develops arrhythmia with subsequent hydrops and cardiac failure. It is unforeseeable how the hemodynamic consequences of the arrhythmia may influence the unaffected twin through placental communications.

Insufficient cardiac output in tachyarrhythmia may cause a fall in blood pressure and increased central venous pressure transmitted to the placental bed in the affected twin. The effect of this redistribution on placental flow and the nonaffected twin is likely dependent on the number and nature of the placental vascular communications. There were no signs of twin–twin transfusion syndrome, unequal placental resources, or abnormal cord insertions before the arrhythmia, which was reassuring. Regretfully, no evaluation of the placenta was performed to characterize communicating vessels. The affected twin developed hydrops, a low Q_{UV}, and a reversal of flow in the precordial veins, suggesting that the cardiac output to the placenta and the venous return to the liver was compromised due to reduced diastolic relaxation and filling time. The repeated observations of a gradual increase in Q_{UV} and normalization of LPV flow velocities after conversion indicated normalization of the cardiac output to the placenta and venous liver flow.

Flecainide passes the placenta with rapid fetal response, also in cases of hydrops, but may have a proarrhythmic and negative inotropic effect in the mother and possibly a nonaffected twin.[8] There is limited knowledge about the effect of flecainide in a nonarrhythmic fetal heart. Future studies should aim to include placental sharing, quantification of vascular communications, maternal and umbilical blood examinations, and neonatal cardiac assessment.

Conclusion

We demonstrate a case of successful cardioversion of SVT with flecainide in an MC twin pregnancy with no adverse effects for the mother and the nonaffected twin. Fetal venous Doppler ultrasound gave useful insights into circulatory pathophysiology.

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Conflict of Interest
None declared.

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