Concurrent maternal and fetal tachyarrhythmia in pregnancy

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Abstract

The occurrence of a maternal and fetal tachyarrhythmia together in pregnancy is exceedingly rare. We report a case of a persistent fetal atrial ectopic tachycardia occurring in conjunction with a maternal atrial tachycardia with left ventricular systolic dysfunction. Amiodarone was effective in treating both maternal and fetal arrhythmias.

Keywords

Maternal–fetal medicine, cardiology, cardiac, cardiovascular, high-risk pregnancy

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A 34-year-old gravida 3 para 1 with a bicuspid aortic valve with mild aortic regurgitation was seen at 16 weeks' gestation for the treatment of maternal and fetal arrhythmia. She had a history of an inappropriate sinus tachycardia and an atrial tachycardia (AT) first diagnosed at the age of 15. She had had two radiofrequency ablations at ages 24 and 26 years. She had probable tachycardia-mediated cardiomyopathy, with a left ventricular ejection fraction (LVEF) of 34% at the time of her first ablation. Following the second ablation, her LVEF improved, but she had residual mild left ventricular systolic dysfunction (LVSD).

During her first pregnancy, she had deterioration in left ventricular function from mild to moderate impairment. Despite increasing doses of metoprolol, she remained in AT throughout the pregnancy. Her ventricular function improved postpartum and an echocardiogram performed after six months showed mild LVSD (LVEF: 48%). At the time of this presentation, at 16 weeks' gestation in her third pregnancy, her electrocardiogram (ECG) showed AT at 123 beats per minute (bpm) (Figure 1) and her echocardiogram showed moderate LVSD (LVEF: 35%). She was on metoprolol 100 mg twice daily.

A fetal echocardiography was performed because of the maternal cardiac history and to assess a fetal tachycardia. The study identified a persistent fetal supraventricular tachyarrhythmia (SVA) at 205 bpm with 1:1 atrio-ventricular (AV) conduction and a long ventriculoatrial (VA) pattern (Figure 2). Fetal biventricular systolic function was normal; there was no evidence of heart failure or hydrops fetalis. In view of the fetal risk of tachycardia-induced heart failure and death, the patient was admitted to hospital. An incessant maternal AT was documented, ranging from 104 to 123 bpm. In addition to her regular metoprolol, transplacental therapy (medication administered to the mother with the aim of fetal treatment via placental transfer) was initiated with flecainide (100 mg three times daily). Flecainide failed to convert the SVA and was not continued because of the maternal left ventricular dysfunction. Transplacental therapy with digoxin was started (loading dose: 1.5 mg/day; maintenance dose: 0.5 mg/day, target plasma level: 2.0–2.5 ng/mL) but was ineffective in treating the SVA during the following eight days. Treatment was changed to oral amiodarone (loading dose: 1.2 g/day; maintenance dose: 600 mg/day). After four days of amiodarone, the fetal heart rate decreased to 175 bpm and the patient was discharged from hospital. She remained on amiodarone and metoprolol throughout the pregnancy.

The mother and baby were monitored frequently throughout pregnancy, with fetal echocardiography on a weekly to biweekly basis. The mother converted to sinus rhythm at 19 weeks' gestation. Fetal echocardiograms showed further reduction in heart rate and the dose of amiodarone was decreased. By 27 weeks' gestation, the fetal heart rate

was 160 bpm. The maternal heart rate was 90 bpm in sinus rhythm and there was an improvement in ventricular function (LVEF: 48%). Labour was induced at 38 weeks' gestation in view of maternal and fetal heart disease. The mother was monitored with telemetry and remained stable throughout. She had an uncomplicated vaginal delivery of a female infant (birth weight: 2740 g, <3rd centile; Apgar scores: 9 and 9) with no postpartum haemorrhage. The baby was admitted to the neonatal intensive care for observation. As the infant's ECG revealed normal sinus rhythm, she was discharged home on no medication. Three weeks later, upon re-assessment, the infant was found to be in incessant atrial ectopic tachycardia (AET) 250 bpm with reduced biventricular function requiring re-admission and treatment with amiodarone and propranolol.

Discussion

The cardiovascular adaptations to pregnancy including the increase in maternal heart rate and cardiac output, elevation in plasma catecholamine concentrations and adrenergic receptor sensitivity can lead to an increased susceptibility to arrhythmias. Arrhythmias can occur for the first time in pregnancy.

AT is defined as regular atrial activation independent of the sinus node, with atrial rates typically of 100 to 250 bpm. It can be incessant and, rarely, associated with left ventricular systolic dysfunction. Incessant maternal AT is rare, but has been described in pregnancy. AT can be treated with beta blockers or calcium channel blockers; however, pharmacologic management is often ineffective in the treatment of incessant AT. Higher doses of metoprolol are often required in pregnancy due to its altered metabolism.¹ For refractory cases, antiarrhythmics can be used. The safety profile of many antiarrhythmic

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Figure 1. Maternal electrocardiogram at presentation (16 weeks' gestation) showing an atrial tachycardia at 123 bpm. The patient was taking metoprolol 100mg twice daily.

Figure 2. A simultaneous SVC/aorta Doppler tracing of the fetal arrhythmia at 16 weeks' gestation.

A: a-wave flow reversal in the SVC during atrial contraction; V: systolic forward flow in the ascending aorta, V–A duration is longer than A–V duration making this a long-VA tachycardia. Possible mechanisms include AET and PJRT.

AET: atrial ectopic tachycardia; PJRT: persistent junctional reciprocating tachycardia.

drugs is not well established in pregnancy making the management of maternal arrhythmia additionally challenging, particularly in the setting of LVSD. Amiodarone is generally avoided in pregnancy due to the risks of adverse fetal effects including thyroid dysfunction, low birth weight and neurological abnormalities, but can be used in women with underlying structural heart disease.² Catheter ablation is reserved for patients with symptomatic and drug-refractory arrhythmia. Ablation without fluoroscopy (to reduce radiation exposure to the fetus) has been used in a pregnant woman with incessant AT.³

Fetal SVA is rare, occurring in 1/5000 pregnancies. Reentrant or short VA tachycardia accounts for 60% of fetal SVAs and atrial flutter 30%.⁴ Long VA tachycardia, as seen in this fetus, is rarely observed (10%); these tachycardias are typically slower, occur earlier and are

more difficult to manage. Long VA tachycardia includes AET and persistent junctional reciprocating tachycardia. These cannot be distinguished by fetal echocardiography. In this infant, AET was confirmed after birth. With fetal SVAs, higher ventricular rates and earlier gestational age at presentation are associated with an increased risk of hydrops. Transplacental drug treatment is recommended to most mothers of a fetus with persistent SVA. The most commonly used antiarrhythmics are digoxin, flecainide and sotalol.⁴ Digoxin and flecainide have been associated with higher rates of termination of SVAs and greater slowing of ventricular rates compared to sotalol.⁴ Increased digoxin doses are often required in pregnancy due to increased renal clearance. In the case of fetal hydrops, flecainide and sotalol are the preferred agents as they continue to cross the placenta

readily, while placental transfer of digoxin is impaired in this setting.⁴ While there are hypothesized concerns of the pro-arrhythmic effects of flecainide and sotalol, there is no reported objective evidence to support this.⁴

The occurrence of co-existing maternal and fetal arrhythmia is exceedingly rare. There is one reported case of familial AT in the literature.⁵ It is not clear, in our case, that the two arrhythmias were linked. The case was also more complex because the underlying maternal cardiomyopathy also limited the options available for antiarrhythmic therapy. Despite its relatively poor placental transfer, amiodarone has been shown as an effective choice for the treatment of fetal tachycardia, even in the presence of hydrops.⁶ It is generally reserved for refractory cases and only after careful counseling given the potential for adverse fetal outcomes.^{2,6} In this case, amiodarone was an effective treatment for both maternal and fetal arrhythmias. The infant's low birth weight is likely multifactorial in etiology, potentially linked with amiodarone therapy, beta blocker use and maternal heart disease, all of which are associated with low birth weight.^{2,7}

This rare presentation highlights the delicate balance in maternal and fetal well-being when treating arrhythmias in pregnancy, the need for multidisciplinary teams and the additional complexity of treating fetal tachycardia in the context of maternal arrhythmia and ventricular dysfunction.

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Guarantor

CS.

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