

Intrauterine management of fetal supraventricular tachycardia (SVT) with cardiac failure

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SUMMARY

Fetal arrhythmias are not uncommon in pregnancy. The diagnosis can be established on routine ultrasound scan. Fetal supraventricular tachycardia (SVT) is the most common cause of fetal tachycardia. If left undiagnosed and untreated, these fetuses may develop cardiac failure, hydrops fetalis and eventually death.

We report two fetuses diagnosed antenatally to have fetal SVT. Both fetuses were in cardiac failure and were successfully treated with maternal administration of anti-arrhythmic medications. Digoxin, and in severe instances, a combination with flecainide significantly improved fetal outcomes and prevented fetal mortality. The long term prognosis of such patients are good.

KEY WORDS:

Fetal supraventricular tachycardia, fetal therapy, digoxin, flecainide

INTRODUCTION

Fetal SVT complicates 2% of pregnancies. Although it is most commonly benign and isolated, 10% of fetuses with arrhythmia are associated with significant mortality. Fetal SVT is the most common type of fetal tachycardia. Without intervention, 50% of such fetuses will develop cardiac failure and hydrops fetalis; which eventually leads to death.¹

There has been several case reports of improved fetal outcomes following maternal administration of anti-arrhythmic medications.¹⁻⁵ We report our own experience of two fetuses with fetal SVT in cardiac failure, who were successfully treated with maternal administration of digoxin and a combination with flecainide in severe instances.

The aim of this study is to create awareness on a rare but an essential condition and to highlight available intrauterine management options which significantly prevents neonatal death from fetal SVT.

CASE REPORT

Case 1

A 19 year old healthy primigravida was noted to have fetal tachycardia during a routine ultrasound surveillance at 29

weeks of pregnancy. She was referred to our tertiary fetal medicine unit and a diagnosis of fetal SVT was established. There were no other structural fetal abnormalities.

Following a week of expectant management, the SVT persisted and by this time, the fetus had developed symptoms of cardiac failure as evidenced by the presence of pleural effusion.

Following a multidisciplinary involvement, intrauterine therapy via maternal administration of digoxin successfully reverted the SVT to a normal heart rate within a week. The pleural effusion resolved once the heart rate normalised.

The pregnancy was taken to term and a healthy 2700gms fetus was delivered at 38 weeks of gestation. The fetus was well; in sinus rhythm post-delivery and was discharged home within a week without further interventions.

Case 2

The second patient was a 28 year old healthy multip who was initially noted to have fetal bradycardia on a routine ultrasound scan at the gestation of 23weeks. A diagnosis of a complete fetal heart block was established at our fetal medicine unit. The fetus had no structural cardiac abnormalities. Maternal SLE was ruled out.

Following close surveillance, the complete heart block spontaneously improved to a normal rate three weeks later. In the following weeks, the fetus developed SVT which was apparent at 32 weeks of gestation. The fetus was in cardiac failure leading to hydrops fetalis as evidenced by fetal ascites and scalp odema.

In view of the severity of the symptoms and the possibility of a poor placental transfer of a hydropic placenta, a combination of digoxin and flecainide was administered to the mother.

Within 10 days of treatment, the fetal heart rate was reduced by 50 beats per minute (bpm) from an initial rate of 240 bpm. The fetal hydrops resolved once the heart rate was reduced to 190 bpm. The fetus was delivered electively at 34 weeks of gestation. The fetus required adenosine immediately following delivery and was discharged well on regular digoxin and flecainide.

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DISCUSSION

Fetal SVT can be easily detected on routine ultrasound scans. The mortality following fetal SVT is extremely significant once hydrops fetalis develops; secondary to cardiac failure. Expectant management may not be a viable option while delivery may not be optimal especially if fetus maturity is yet to be established.

Although maternal administration of antiarrhythmic drugs is an established practice, there is little consensus regarding the drug of choice for this purpose. A recent non-randomized multicentre study showed that digoxin and flecainide are significantly better at converting arrhythmias to normal ventricular rhythms compared to other drugs.¹

Maternal digoxin administration has been the first line management of fetal SVT.^{2,3} It has been reported to be associated with a success rate of 51% and is most effective for atrioventricular nodal or atrioventricular re-entrant tachycardia; which are the common causes of fetal SVT.

Although the mother requires a four to eight fold higher doses of digoxin administration, it has an established safety profile and a good response time as rapid conversion to normal rhythms is essential. The mother needs close surveillance and frequent monitoring to ensure therapeutic drug levels, ideally in a coronary care unit.

Recent case reports has suggested that flecainide, which has better placental transfer as compared to digoxin; may be more effective in management of fetal SVT. It has been reported to have a higher success rates of almost 64%.^{1,4,5} This may be more relevant in hydropic fetus which is associated with poor placental transfer. Albeit limited case reports as compared to digoxin, there has been no reported adverse effects with the use of flecainide in pregnancy.

Based on our experience, it took almost a week to effectively reverse fetal SVT following digoxin monotherapy. However,

in fetuses with severe hydrops fetalis, a combination of flecainide and digoxin were administered to ensure appropriate placental transfer of medications and for a rapid response of treatment. It took almost ten days for complete resolution of fetal hydrops despite using combined treatment. Interestingly, the failure symptoms and hydrops significantly improved when the heart rates were reduced by 20%, even at a fetal heart rate of 190bpm.

A multidisciplinary team approach, involving a cardiologist, paediatrician and a fetal medicine specialist are essential in the management of such complex cases.

CONCLUSION

Indirect maternal administration of digoxin is beneficial in treatment of fetal SVT in cardiac failure. Flecainide has a better placental transfer and can be used in combination with digoxin in severe instances, especially in hydropic fetuses. A 20% reduction in the fetal heart significantly improves cardiac failure symptoms.

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