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Case Report

A long journey to birth: a case of refractory fetal SVT

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ABSTRACT

Fetal tachyarrhythmias occur in approximately 0.4-0.6% of pregnancies, with supraventricular tachycardia (SVT) representing the most common subtype. Although digoxin has traditionally been used as first-line therapy, accumulating evidence indicates that monotherapy is frequently insufficient, particularly in sustained or accessory pathway–mediated SVT. Nevertheless, the optimal timing and escalation strategy for combination therapy remain controversial. We report a case of sustained fetal SVT diagnosed at 29 weeks of gestation in a non-hydropic fetus, managed using a response-guided, stepwise escalation approach. Initial dual transplacental antiarrhythmic regimens failed to achieve sinus rhythm despite therapeutic maternal drug levels and close surveillance. Owing to persistent tachycardia, triple therapy with digoxin, sotalol, and flecainide was initiated under intensive maternal–fetal monitoring, resulting in successful and sustained conversion to sinus rhythm without maternal or fetal adverse effects. This case underscores the importance of individualized, decision-oriented treatment escalation rather than prolonged reliance on monotherapy. Careful maternal electrocardiographic monitoring combined with serial fetal echocardiography enabled the safe administration of triple therapy and was associated with a favorable perinatal outcome.

Keywords: Fetal supraventricular tachycardia, Combination antiarrhythmic therapy, Digoxin, Flecainide, Sotalol

INTRODUCTION

Fetal tachyarrhythmias are observed in approximately 0.4-0.6% of pregnancies and represent a significant cause of fetal morbidity and mortality when sustained.[1-3] Supraventricular tachycardia (SVT) accounts for nearly 70-80% of all fetal tachyarrhythmias and is typically characterized by a regular rhythm with 1:1 atrioventricular conduction and ventricular rates between 220 and 300 beats per minute.[1] The diagnosis of fetal SVT relies primarily on fetal echocardiography using M-mode and doppler techniques, which allow assessment of atrioventricular and ventriculoatrial timing.[1-5] Most fetal SVTs are mediated by atrioventricular reentrant mechanisms involving accessory pathways, which may be transient during fetal life but can persist and lead to clinically significant arrhythmias.[3,4] Sustained fetal SVT may result in heart failure, hydrops fetalis, preterm delivery, or fetal demise if not promptly controlled.[3,6] Although digoxin has historically been used as first-line therapy, increasing evidence suggests that digoxin monotherapy has limited efficacy, particularly in accessory pathway–mediated tachycardias.[9,14–16]

As a result, many centers now favor early combination therapy or rapid escalation when treatment response is inadequate. We present a case of sustained fetal SVT without hydrops, successfully managed using a response-guided, stepwise escalation strategy, emphasizing timely decision-making and individualized combination therapy rather than prolonged reliance on monotherapy.

CASE REPORT

A 24-year-old primigravid woman was referred at 29 weeks of gestation after routine obstetric ultrasonography

revealed persistent fetal tachycardia exceeding 200 beats per minute.

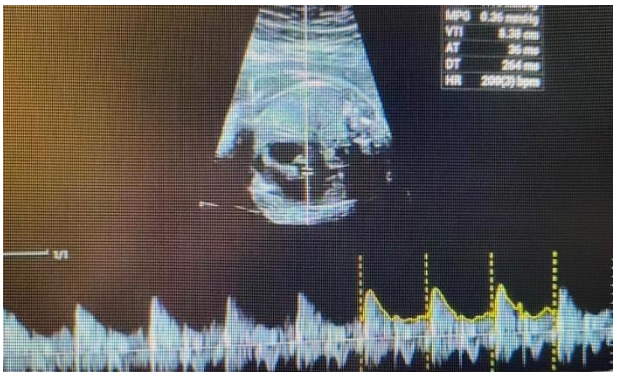

Figure 1: Fetal tachycardia with 200 per minute heart rate and a 1:1 atrioventricular (AV) conduction pattern.

Baseline maternal electrocardiography and renal function tests were normal. Transplacental antiarrhythmic therapy was initiated with oral digoxin at 1.0 mg/day, reduced to 0.5 mg/day after 24 hours. Given the sustained nature of the tachycardia and the limited efficacy of digoxin monotherapy, oral sotalol was added early at a dose of 160 mg/day. Maternal serum digoxin levels were monitored daily and maintained within the therapeutic range (1-2 µg/L), with dose adjustment to 0.75 mg/day due to initially subtherapeutic levels. After five days of combined digoxin–sotalol therapy, fetal tachycardia persisted. Although a gradual reduction in heart rate was observed, sinus rhythm was not achieved.

The pregnancy had been uncomplicated, and the mother had no history of systemic disease, thyroid dysfunction, infection, or medication use. Fetal echocardiography demonstrated a regular tachycardia with ventricular rates ranging from 200 to 239 beats per minute and consistent 1:1 atrioventricular conduction (Figure 1). Simultaneous atrial and ventricular contractions were observed on doppler interrogation. Cardiac anatomy was normal, ventricular systolic function was preserved, and no signs of hydrops fetalis were present.

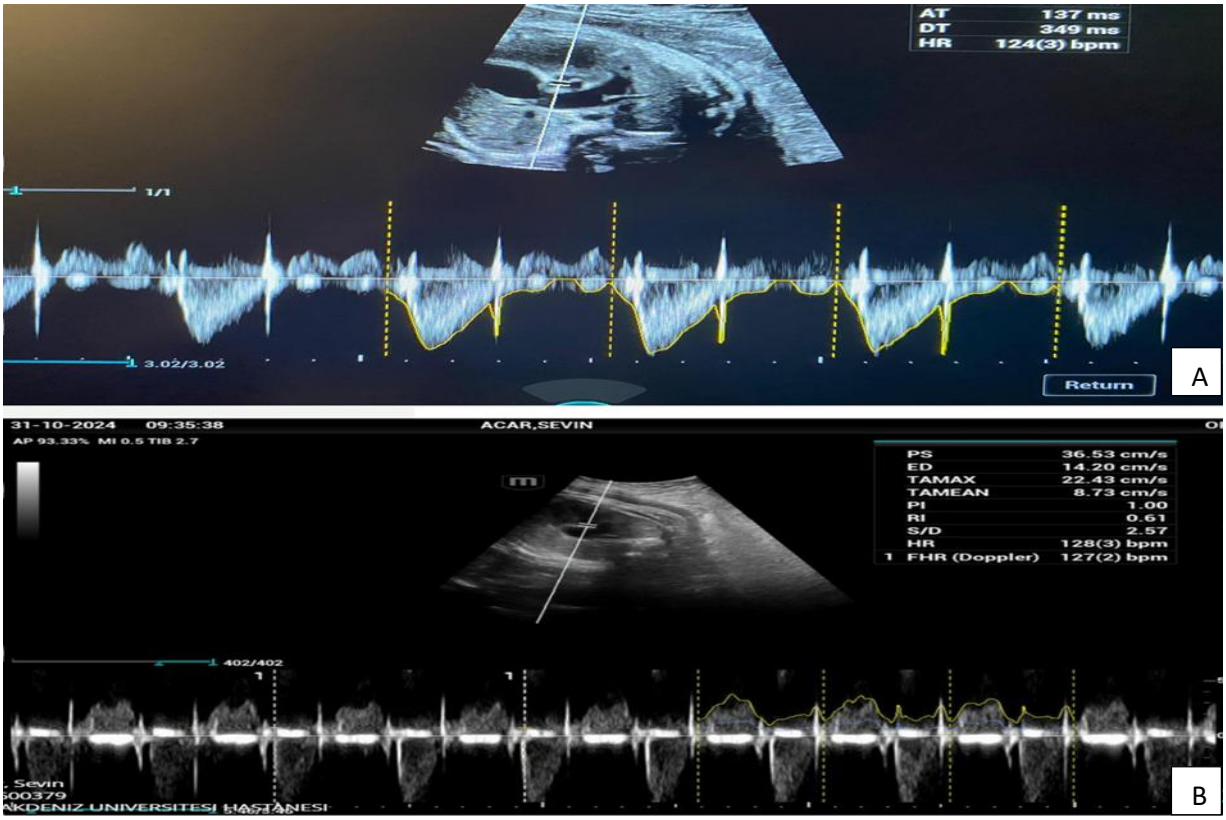

Figure 2 (A and B): Conversion to sinus rhythm at 31 weeks of gestation.

The treatment strategy was therefore modified to digoxin (0.75 mg/day) and flecainide (300 mg/day in three divided doses). Serial fetal echocardiography showed further reduction in heart rate (239 → 225 → 200 bpm); however, by day 8 of sustained tachycardia, physiological sinus rhythm had not yet been restored. Considering the cumulative duration of tachycardia and incomplete response to sequential dual therapies, sotalol (160 mg/day) was reintroduced, resulting in triple antiarrhythmic therapy. Under intensive inpatient monitoring—including daily maternal ECGs with QTc, PR, and QRS interval assessment, maternal digoxin level and serial fetal echocardiography—conversion to stable sinus rhythm (122-134 bpm) was achieved by day 12 (Figure 2, Table 1, Table 2).Triple therapy was continued until delivery due to concern for recurrence. The mother remained

hospitalized for close surveillance. At 37 weeks of gestation, a healthy male infant was delivered weighing 3090g with Apgar scores of 9 and 10. Postnatal echocardiography was normal; however, Holter monitoring revealed brief episodes of intra-atrial reentrant tachycardia (IART) (150-160 bpm) (Figure 3). Flecainide therapy was continued, and the infant remained asymptomatic at three-month follow-up (Figure 4).

Table 1: Table of serial fetal echocardiographic examination.

1. Day	**11.09.2024 29w Hr: 239/dk**	**Digoxin: a loading dose of 2 tablets twice daily, followed by a maintenance dose of 1 tablet twice daily. Sotalol: 80 mg twice daily.**
5. Day	16.09.2024 29w+5d HR: 225/dk	Due to subtherapeutic serum digoxin levels, the maintenance dose was increased to 1 tablet in the morning and 2 tablets in the evening. Flecainide was subsequently added at a dose of 1 tablet three times daily, and sotalol was discontinued.
6. Day	17.09.2024 29w+6d HR: 199/dk	Digoxin: 1 tablet in the morning and 2 tablets in the evening. Flecainide: 1 tablet three times daily.
8. Day	19.09.2024 31w+1d HR: 225/dk	Digoxin: 1 tablet in the morning and 2 tablets in the evening. flecainide: 1 tablet three times daily sotalol: 1 tablet twice daily was added.
12. Day	23.09.2024 31w+4d HR: 122/dk	Digoxin: 1 tablet in the morning and 2 tablets in the evening. flecainide: 1 tablet three times daily sotalol: 1 tablet twice daily.
13. Day	24.09.2024 31w+5d HR: 120/dk	Digoxin: 1 tablet in the morning and 2 tablets in the evening. flecainide: 1 tablet three times daily sotalol: 1 tablet twice daily.
19. Day	30.09.2024 32w+5d HR: 130/dk	Digoxin: 1 tablet in the morning and 1 tablet in the evening (dose reduced by 1 tablet) flecainide: 1 tablet three times daily sotalol: 1 tablet twice daily.
26. Day	07.10.2024 33w+3d HR: 126/dk	Digoxin: 1 tablet twice daily flecainide: 1 tablet three times daily sotalol: 1 tablet twice daily.
41. Day	22.10.2024 34w+6d Hr: 134/dk	Digoxin: 1 tablet twice daily flecainide: 1 tablet three times daily sotalol: 1 tablet twice daily.
50. Day	31.10.2024 36w HR: 128/dk	Digoxin: 1 tablet twice daily flecainide: 1 tablet three times daily sotalol: 1 tablet twice daily.
58. Day	08.11.2024 37w HR: 122/dk	Digoxin: 1 tablet twice daily flecainide: 1 tablet three times daily sotalol: 1 tablet twice daily.

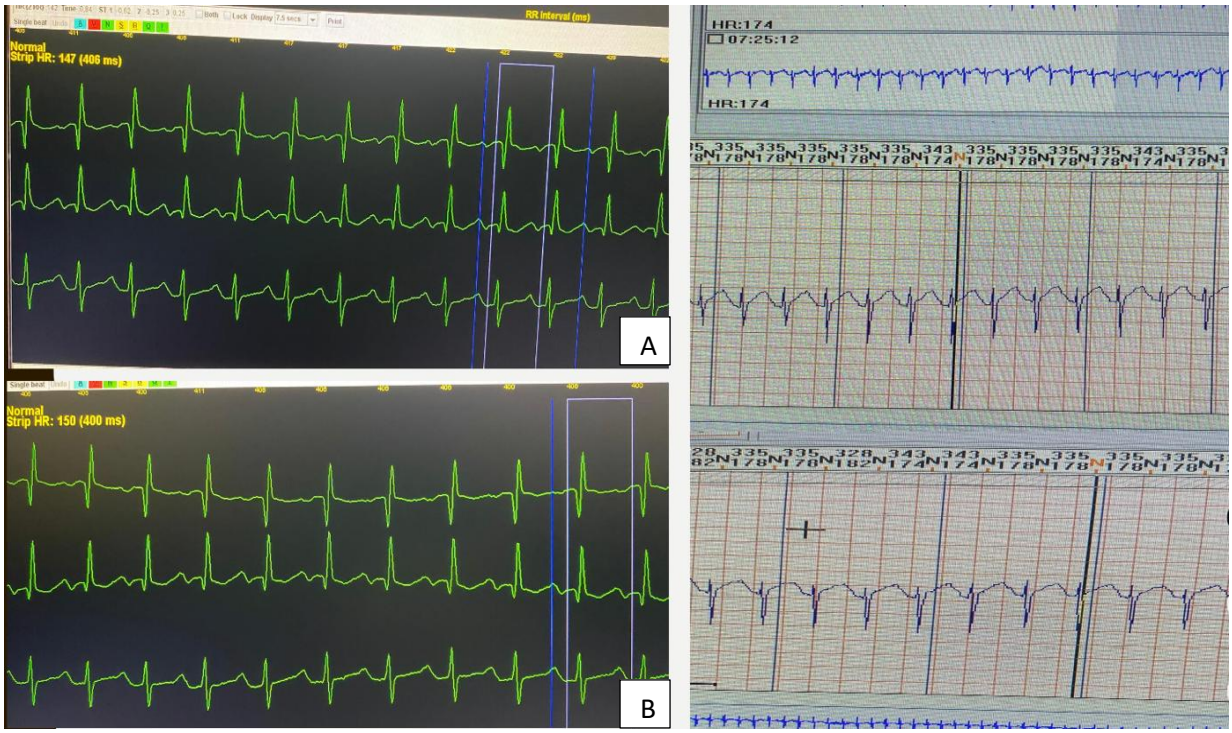

Figure 3 (A and B): IART on the baby's holter ECG recording.

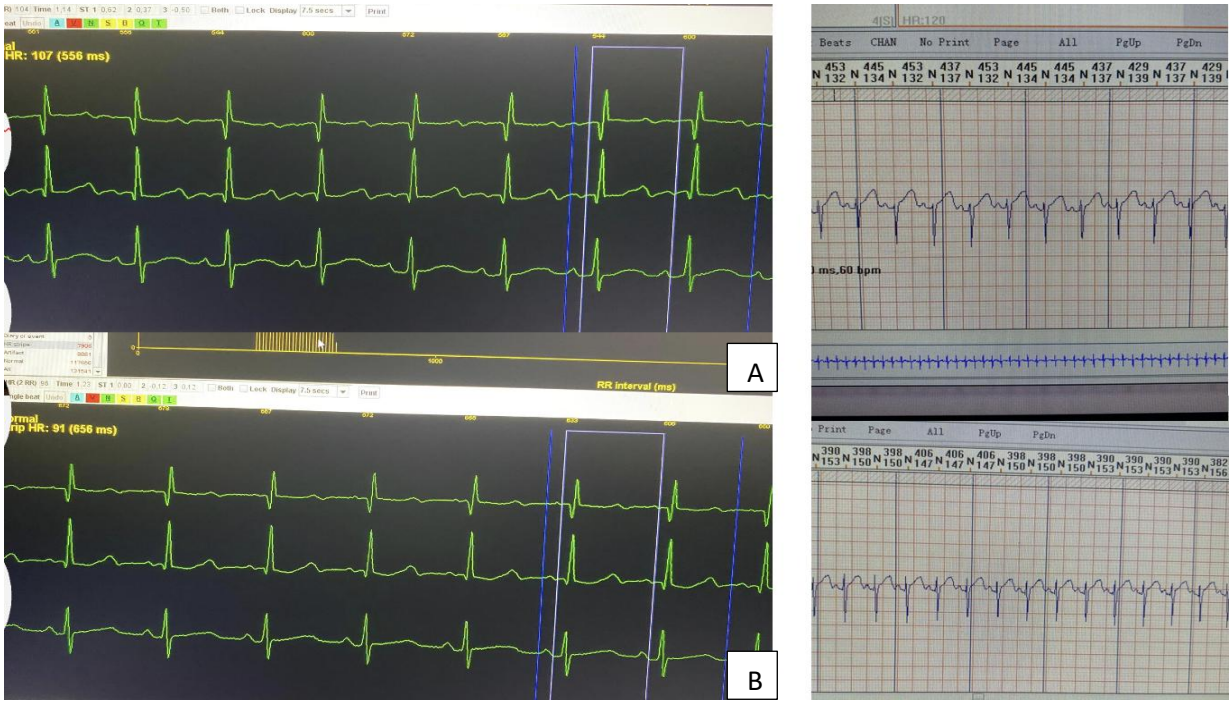

Figure 4 (A and B): Normal sinus rhythm on baby's holter ECG recording.

Table 2: Table of maternal digoxin levels.

	Digoxin level: mcg/L
12.09.24	0.46
13.09.24	1.64
13.09.24-30.09.24	1-2
30.09.24	1.06
01.10.24	0.92
03.10.24	0.93
09.10.24	0.97
16.10.24	0.66
23.10.24	1.18
30.10.24	1.0
06.11.24	0.97
07.11.24	0.85

DISCUSSION

Fetal SVT remains the most common fetal tachyarrhythmia and continues to pose significant management challenges due to variability in arrhythmia mechanisms, treatment response, and lack of universally accepted therapeutic algorithms.[1-3] While advances in fetal echocardiography have improved diagnostic accuracy, optimal treatment strategies—particularly regarding the timing and extent of combination therapy—remain a subject of ongoing debate. Accurate mechanism-oriented diagnosis is fundamental to effective management. M-mode and doppler echocardiography allow assessment of atrioventricular and ventriculoatrial relationships, which guide differentiation between reentrant SVT and other tachyarrhythmias such as atrial flutter.[1-5] Most fetal SVTs are mediated by accessory pathways, a mechanism associated with reduced responsiveness to digoxin monotherapy.[3,4] In the present case, persistent 1:1 atrioventricular conduction and sustained tachycardia supported the diagnosis of SVT, justifying an aggressive rhythm-control strategy. Historically, digoxin has been recommended as first-line therapy, particularly in non-hydropic fetuses, due to its long-standing clinical experience and maternal safety profile.[7,8] However, accumulating evidence indicates that digoxin monotherapy frequently fails to achieve rhythm conversion, especially in reentrant SVT mediated by accessory pathways.[9,14-16] Partial heart rate reduction without restoration of sinus rhythm should not be interpreted as therapeutic success, as ongoing tachycardia continues to expose the fetal myocardium to adverse hemodynamic effects.

In recent years, a paradigm shift toward protocol-based and early combination therapy has emerged. The multicenter Japanese study by Miyoshi et al represents a landmark contribution in this regard. In their prospective, protocol-defined cohort of fetuses with SVT and atrial flutter, transplacental treatment strategies incorporating early combination therapy were associated with high conversion rates and favorable perinatal outcomes, even in complex or refractory cases.[16] Importantly, this study emphasized structured escalation based on treatment response rather than prolonged reliance on monotherapy. Comparative studies further support this approach. Jaeggi et al demonstrated superior efficacy of flecainide and sotalol compared with digoxin, particularly in non-hydropic fetuses with reentrant SVT.[15] Similarly, meta-

analyses have shown higher rhythm conversion rates with flecainide-based regimens and combination therapies, without a corresponding increase in serious maternal adverse events when careful monitoring is employed.[9-16]

The Utrecht treatment protocol advocates stepwise escalation when sinus rhythm is not achieved within a predefined timeframe, highlighting that delayed escalation may unnecessarily prolong fetal exposure to tachycardia.[10] Our management strategy aligns closely with this concept. In the present case, sequential dual therapies achieved only partial heart rate reduction, prompting escalation to triple therapy after careful consideration of maternal safety and fetal risk. This decision was driven not by refractoriness alone, but by a decision-making framework prioritizing rhythm normalization over rate control. Triple antiarrhythmic therapy should not be considered routine; however, evidence suggests that it can be safely administered in selected refractory cases under intensive inpatient surveillance.[11,12] In our case, strict maternal ECG monitoring and serial fetal echocardiography enabled safe administration and successful rhythm conversion without maternal or fetal complications.

CONCLUSION

In conclusion, this case reinforces emerging evidence that early recognition of treatment failure and timely escalation to combination therapy are critical in the management of fetal SVT. Rather than prolonged monotherapy, a response-guided, protocol-based approach may optimize fetal outcomes while maintaining maternal safety.

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