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Unmasking the Culprit: Maternal Hyperthyroidism Presenting as Fetal Supraventricular Tachycardia and Hydrops

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Abstract

Management of fetal supraventricular tachycardia at times can be tricky and challenging when they are secondary to underlying metabolic or hormonal problems. It can difficult to unmask the real culprit unless thorough evaluation is performed.

Keywords

- fetal echocardiography
- noncardiac hydrops fetalis
- pediatric cardiology
- supraventricular tachycardia
- thyroid disorder during pregnancy

Introduction

Fetal supraventricular tachycardia (SVT) is a benign condition if diagnosed and treated in time. Occasionally, it can turn malignant and nonresponsive to treatment when underlying contributing factor is not addressed. It is a medical emergency and prompt identification and treatment of the precipitating factor can be rewarding. We wish to discuss a fetal SVT with hydrops, which was secondary to hyperthyroidism in the mother and the management of the same.[1–5] To the best of our knowledge, maternal hyperthyroidism leading to fetal SVT and hydrops has not been reported yet.

Case

A 28-year-old female was referred at 26 weeks of gestation for the evaluation of persistent fetal tachycardia and hydrops fetalis. The first delivery was uneventful normal full-term girl with no complications (5 years back). The second gestation was spontaneous miscarriage at 9 weeks and that was 2 years back. This was her third conception. She was averagely built and nourished. Her resting heart rate was 124/min and blood pressure 100/60 mm Hg in right arm supine. Systemic examination was unremarkable. There was no position significant past medical or surgical history. Routine investigation like complete blood count, urine, and other biochemical parameters were with in normal range.

During the 24-week scan, it was noticed that fetal heart rate was more than 200/min. There were signs of fetal hydrops like ascites and pleural collection. There was flow reversal in ductus venosus. Suspecting SVT, rapid digitalization (1,250 µg loading dose given on first day in 3 divided parts and maintenance dose 500 µg/day in 2 doses) was tried by the primary treating physician but with little benefits and there was no improvement (drug level in maternal serum was 2.1 ng/mL) Fetal weight was appropriate for gestational age.

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Fig. 1 Ascites: Note free fluid collection in peritoneal cavity.

Fetal echocardiography done at 26 weeks, 5 days showed a structurally normal heart (Philips Affinity 70, with C5 curvilinear probe, Philips Medical Systems, the Netherlands). Fetus had ascites (►**Fig. 1**) and flow reversal in the ductus venosus (►**Fig. 2**). Heart rate was persistently in the range of 210 to 240/min (►**Fig. 3**; pulse wave of left ventricular outflow tract) and it never went below 210/min. Sinus rhythm could not be demonstrated throughout the study period. Atrial and ventricular activity was occurring simultaneously that was consistent with short ventriculoatrial (VA) tachycardia (pulse wave Doppler at mitral inflow in ►**Fig. 4** and M mode of right atrium and ventricle; ►**Fig. 5**). Pulse wave Doppler of superior vena cava and ascending aorta or pulmonary artery with vein were not very clear but M mode was clear about nature of heart rhythm differentiating between short versus long VA tachycardia. Ventricular contractility

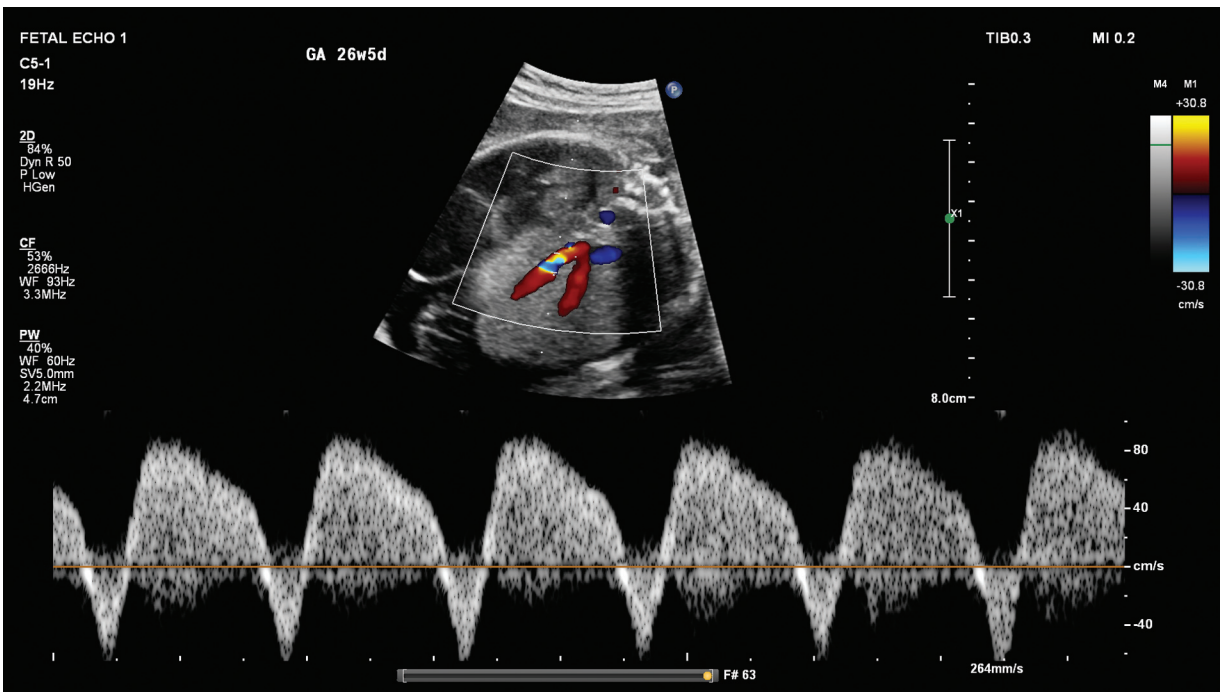

Fig. 2 Flow reversal in ductus venosus.

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Fig. 3 Heart rate: pulse wave of left ventricular outflow tract. Note heart rate of 220/min.

was depressed but it was difficult to comment upon in view of tachyarrhythmia. When patient came to us at 26 weeks, she had already taken 2 weeks of digoxin. In spite of adequate trial of digoxin for 2 weeks[6] (drug level in therapeutic rage as mentioned above), tachycardiac was persistent and fetus had developed hydrops. There was high chances of preterm delivery or fetal demise. As there was no response to digoxin, second-line drug flecainide was started with QT interval monitoring of the mother (50 mg twice a day [BID]).[6] A detailed informed consent was obtained from mother about nature of treatment, possible adverse effect on the mother, and fetus including intrauterine fetal death, preterm labor, and teratogenicity before starting the medication.

As the thyroid profile was not done in the recent past, it was requested. The mother had severe hyperthyroidism (thyroid-stimulating hormone [TSH] < 0.01micU/mL, free

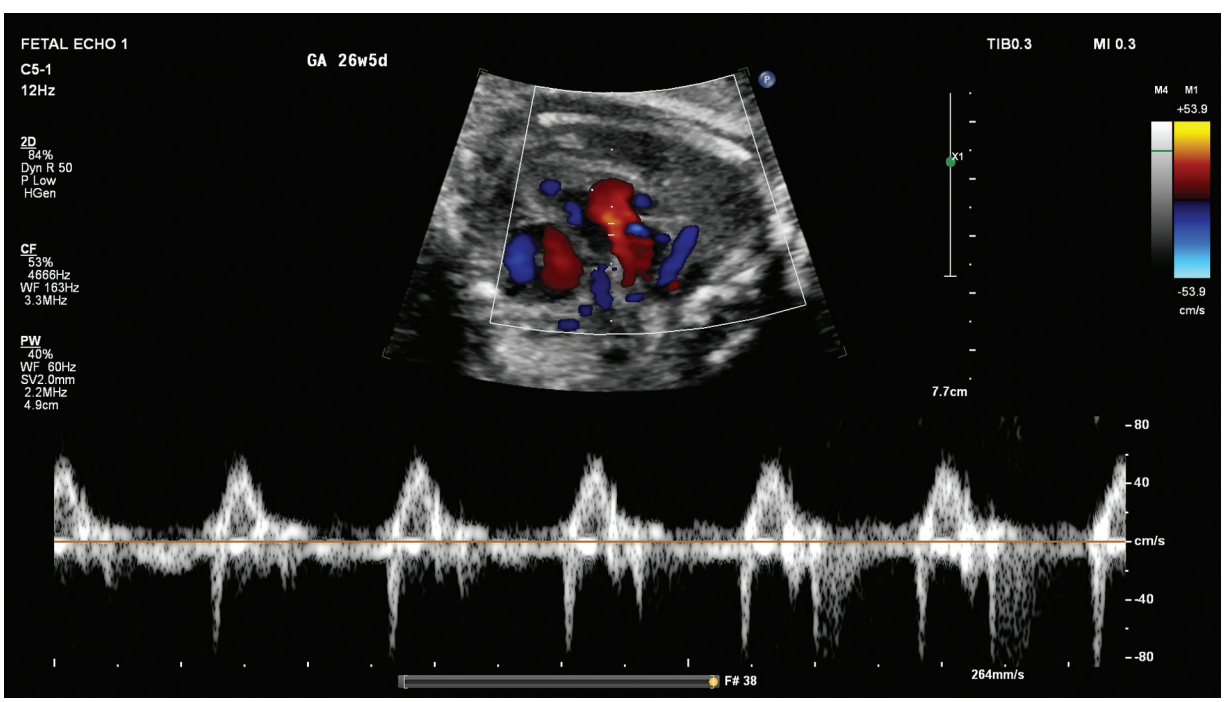

Fig. 4 Pulse wave Doppler: Single wave seen at mitral inflow. Separate "E" and "A" waves are not seen.

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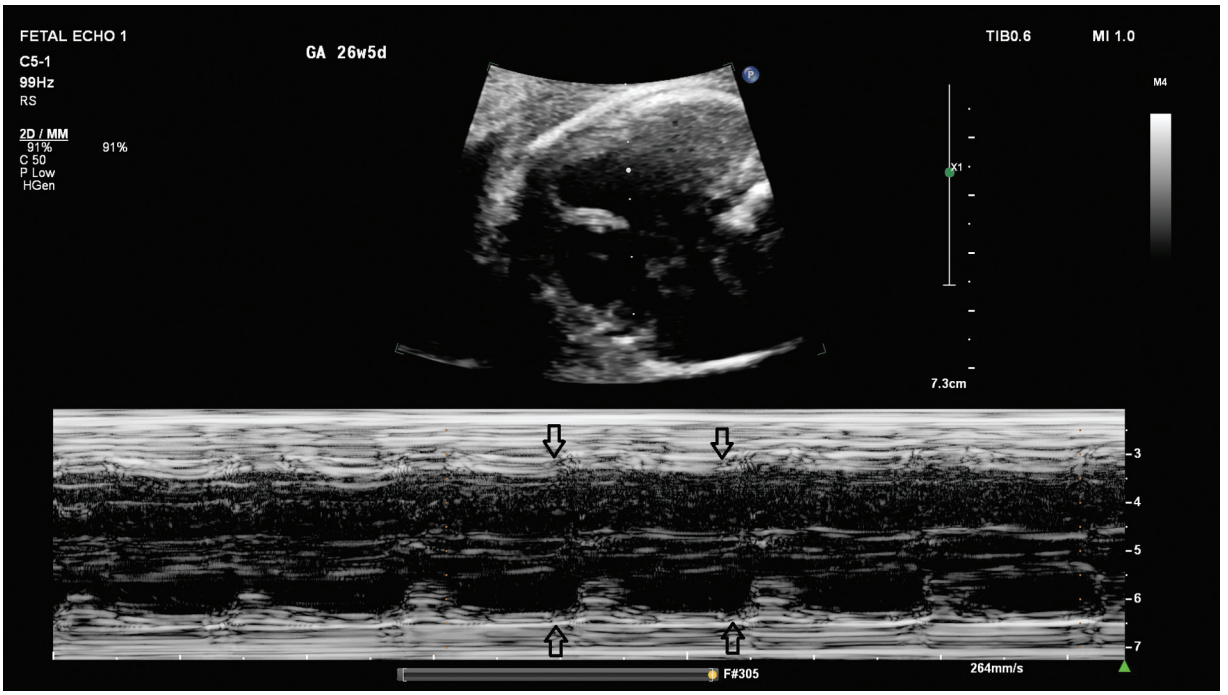

Fig. 5 M mode across atria and ventricle: Short ventriculoatrial tachycardia. Downward arrow: atrial contraction. Upward arrow: ventricular contraction.

T3: 16.5 pg/mL, free T4 27.8 ng/mL) and anti-TSH receptor antibody was positive. Immediate antithyroid medication was instituted after informed consent as mentioned above (methimazole 10 mg BID orally). Within 5 days of starting treatment, there was marked improvement in fetal tachycardia. Heart rate improved to 160/min and there was a sinus rhythm. Flecainide was stopped after 1 week and there was no recurrence of arrhythmia.

The mother had normal vaginal delivery at 36 weeks, 5 days. Birth weight was 2.32 kg. The neonate was monitored for possible development of thyroid problem in early neonatal period and had uneventful course. Electrocardiogram as well as two-dimensional echocardiogram at birth was normal.

Discussion

Fetal SVT are generally secondary to a reentrant mechanism (SVT and atrial flutter) or triggered activity (atrial tachycardia). Very occasionally they are secondary to metabolic problems. In nonresponders of standard medical management, it is worth, considering alternative causes for the same. In some series, close to 20% of SVT were secondary to thyroid problems.[5]

Anti-TSH receptor antibody in mother crosses into fetal circulation and stimulates the thyroid gland and leads to fetal hyperthyroidism. It presents as fetal tachycardia, low birth weight, neonatal hyperthyroidism, and occasionally fetal death. Antibodies remain in circulation in spite of thyroidectomy or ablation of thyroid with radioactive iodine done in past and the mother being euthyroid at the time of pregnancy. Thus, it is important to note past history of treatment for thyroid disorder is important even if the mother is euthyroid at present.[1–4]

Certain aspects of this case are unique. Though fetal tachycardia is commonest manifestation of fetal hyperthyroidism, SVT is not so common. It was further complicated by development of hydrops fetalis, imminent fetal jeopardy, and not responding to therapy. Other markers of fetal hemodynamic compromise like flow reversal in venous duct and cardiomegaly were also present. The clinical dilemma was of immediate delivery with extreme preterm baby versus expectant medical management and variable outcome.

Treatment options for fetal tachyarrhythmias are determined by mechanism of arrhythmia and associated complicating factors. M mode of atria and ventricle or pulse wave of aorta/superior vena cava are diagnostic tools used to ascertain the mechanism. First step in the diagnosis of tachyarrhythmia is to identify atrioventricular association or dissociation. Mechanisms for dissociation include ventricular tachycardia and paroxysmal junctional reentrant tachycardia. If atrioventricular association is present, the next step is to differentiate between short versus long VA tachycardia. It is important to distinguish between short VA and long VA tachycardia. Reentrant tachycardia is generally short VA, while others are long VA (ectopic atrial tachycardia). There are chances of variable atrioventricular block in case of atrial flutter.[6] Digoxin, flecainide, sotalol, and amiodarone are preferred drugs for treatment of fetal tachyarrhythmia for detailed discussions, please refer to references.[6]

Antithyroid drugs are preferred therapy for maternal and fetal hyperthyroidism. Though there are some reports to suggest propyl thiouracil may be less teratogenic than methimazole, there is no conclusive evidence for the same. We offered treatment as per institutional protocols. It is recommended to keep mother at higher limit of normal

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thyroid hormone range to prevent fetal hypothyroidism. Beta-blockers like propranolol cross placental barrier and can control tachycardia but carry risk of growth retardation.[1–4]

Whether routine thyroid screening in first trimester would have avoided this complication remains a debatable issue. In retrospect, one can strongly argue for the same.

Take-Home Message

Fetal SVT is occasionally secondary to noncardiac causes and it does not respond to standard medical management unless underlying anomaly is corrected. Maternal screening for thyroid dysfunction or related past history can be worthwhile.

Funding
None.

Conflict of Interest
None declared

References

1 Polak M, Van Vliet G. Therapeutic approach of fetal thyroid disorders. Horm Res Paediatr 2010;74(01):1–5
2 Polak M, Legac I, Vuillard E, Guibourdenche J, Castanet M, Luton D. Congenital hyperthyroidism: the fetus as a patient. Horm Res 2006;65(05):235–242
3 Moleti M, Di Mauro M, Sturniolo G, Russo M, Vermiglio F. Hyperthyroidism in the pregnant woman: maternal and fetal aspects. J Clin Transl Endocrinol 2019;16:100190. Doi: 10.1016/j.jcte.2019.100190
4 Léger J. Management of fetal and neonatal Graves' disease. Horm Res Paediatr 2017;87(01):1–6
5 Johnson JA, Williams P, Lu Z, Kavanaugh-McHugh A, Fish FA, Killen SA. Fetuses of mothers with thyroid disease may be at higher risk of developing supraventricular tachycardia. Am J Perinatol 2015;32(13):1240–1246
6 Donofrio MT, Moon-Grady AJ, Hornberger LK, et al; American Heart Association Adults With Congenital Heart Disease Joint Committee of the Council on Cardiovascular Disease in the Young and Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and Council on Cardiovascular and Stroke Nursing. Diagnosis and treatment of fetal cardiac disease: a scientific statement from the American Heart Association. Circulation 2014;129(21):2183–2242

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