Case report

A case of successfully treated fetal supraventricular tachycardia in the late first trimester



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Abstract

We present a case of early diagnosed fetal supraventricular tachycardia in the 13th week of gestation. Fetal supraventricular tachycardia was confirmed at the reference centre with a ventriculo-atrial to atrio-ventricular (VA/AV) interval ratio of 1.06, indicating a form of tachycardia with a borderline VA interval. The ventricular inflows were characterized by monophasic filling without any signs of atrio-ventricular insufficiency. The ductus venosus demonstrated an abnormal velocimetry. Given the severity of the condition of the fetus, the medical team faced the challenge of quickly implementing transplacental therapy and the need to rule out trisomy 13. Therefore, a direct karyotyping was immediately performed. Next, the patient was qualified for amiodarone transplacental therapy. Follow-up scans revealed a reduction in nuchal translucency thickness, no signs of fetal oedema, restoration of sinus rhythm, and a normal flow profile in the ductus venosus. At 18 weeks and 4 days the therapy was terminated. The baby was born at 39 weeks of gestation through natural labour. Neonatal mass was 3840 g, and 10 points were scored on the Apgar scale. No abnormalities were found in ECG and neonatal echocardiography. The definitive cause of late first trimester supraventricular tachycardia in the described case has not been determined.

Key words: trisomy 13, first trimester, fetal tachyarrhythmia, supraventricular tachycardia, amiodarone.

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Introduction

Supraventricular tachycardia (SVT) is defined as extreme tachycardia with fetal heart rate between 220 and 250 bpm presenting a 1:1 ratio of the atrial to ventricular rate. It is a form of treatable arrhythmia. The major cause of fetal SVT is explained by an adjunct electrical pathway between the atriums and the ventricles. From a prognostic point of view, in cases of SVT, it is of major importance to measure the time between atrial and ventricular contractions. Fetal tachyarrhythmia can increase fetal morbidity and mortality as a mechanism of progressive cardiac failure based on low cardiac output. Nonimmune oedema of the fetus occurs as a result. The incidence of fetal tachyarrhythmia is 0.4-0.6% of all pregnancies. When left intreated, it leads to cardiac failure in 35-60% of cases [1, 2].

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Case report

We present a case of early fetal supraventricular tachycardia, which was diagnosed in the referring physician's (CP) office in the 13th week of gestation. The mean fetal heart rate (FHR) during the scan measured 223 to 230 beats per minute.





Figure 1. Thickened nuchal translucency (left image) and incipient subcutaneous tissue oedema (right image) in the examined fetus at 13 + 3 weeks of gestation

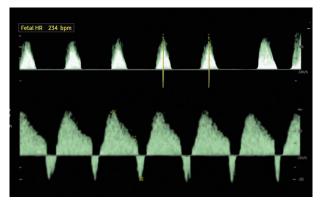


Figure 2. Doppler readings: in the top row, monophasic inflow through the tricuspid valve at a fetal heart rate of 234 bpm; in the bottom row, abnormal flow in the ductus venosus – a deep negative a-wave and significantly reduced D-wave are noticeable

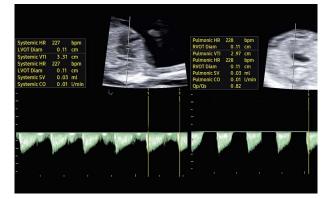


Figure 3. Determinants of total cardiac output: left side – systemic output; right side – pulmonary output

The patient was 25 years old, pregnant for the first time, with insignificant history, without signs of infection, and was not taking any medications. The subject's BMI was 18.7 kg/m². After the fetal tachyarrhythmia was identified, the patient was referred to the reference centre for an expert scan at 13 weeks and 3 days of gestation, dated according to the fetal crownrump length (CRL) measurement. The scan was performed on an HERA W10 ultrasound scanner (Samsung, Seoul, Korea)

using a CA 3-10 transabdominal transducer with a frequency range of 3-10 MHz. It revealed nuchal translucency thickening (NT = 3.2 mm) and marked fetal trunk oedema of the posterior wall. No cardiac or extracardiac structural abnormalities were demonstrated, considering the resolution of the late first trimester scan (Figure 1). In terms of normal variants, only the 2-vessel umbilical cord was noticed.

Fetal supraventricular tachycardia was confirmed (FHR 227-240 bpm), with a ventriculo-atrial to atrio-ventricular (VA/ AV) interval ratio of 1.06, indicating a form of tachycardia with a borderline VA interval (AV interval: 125 ms, VA interval: 132 ms). In addition, the ventricular inflows were characterized by monophasic filling without any signs of atrio-ventricular (AV) insufficiency (Figure 2). There was no evidence of regurgitation of any of the arterial valves. The ductus venosus demonstrated an abnormal velocimetry in the form of a deep negative a-wave, a significantly reduced D-wave, with PVIV and PLI indices of 3.82 and 1.55, respectively (Figure 2).

The evaluation of total cardiac output determinants by Doppler method was feasible thanks to good scanning conditions. Stroke volume (SV) was 0.03 ml; systemic cardiac output was 10 ml/min; pulmonary SV was 0.03 ml; pulmonary cardiac output was 10 ml/min; and Qp/Qs ratio was 0.82 (Figure 3).

First-trimester serum biomarker readings were as follows: free b-HCG: 1.413 MoM; PAPP-A: 0.407 MoM. The result of the risk calculation for the most common trisomies calculated using the FMF algorithm showed the following values: for trisomy 21: 1/13; for trisomy18: 1/1533; for trisomy 13: 1/8.

Given the severity of the fetus' condition, the medical team faced the challenge of quickly implementing transplacental therapy on the one hand, and the need to rule out the most common chromosomal aberration associated with tachycardia – trisomy 13 – on the other. Therefore, a direct karyotyping based on chorionic sampling was immediately performed to rule out chromosomal aberrations within 24 hours. In addition, the chorionic villous sample was sent for aCGH analysis. The nature of the pathology and the associated risk of fetal cardiac failure were explained to the parents. It was raised that there is no literature data on doses of antiarrhythmic medications with proven efficacy at such an early pregnancy stage, and so it was proposed that treatment with the lowest recommended doses of amiodarone would be tried, with the parents bearing the responsibility, to which they agreed. In this situation, an endocrinologist and a cardiologist were consulted on the same day, and both consultations showed no contraindication to using amiodarone in the pregnant woman.

The choice of drug used was primarily dictated by the incipient fetal oedema [2] and the borderline VA interval [3]. The centre's own good experience with using amiodarone in fetuses with oedema at later pregnancy stages was also important. The lowest doses recommended by the American Heart Association were used [4] (saturating dose: 1800 mg/d; maintenance dose 800 mg/d). The pregnant woman reported no side effects during treatment. The first follow-up scan was performed on day 3 and showed reduction in nuchal translucency thickness (NT = 2.4 mm), no signs of subcutaneous tissue oedema, restoration of sinus rhythm with a rate of 138-142 bpm, an increase in the VA/AV interval ratio to 2.1 (AV = 135 ms and VA = 288 ms), and a normal flow profile in the DV (positive a-wave, normalization of D-wave, PVIV = 0.81, PLI = 0.70) (Figure 4).

The determinants of total cardiac output showed: an increase in systemic SV to 0.04 ml with no change in systemic cardiac output; an increase in pulmonary SV to 0.07 ml with no change in pulmonary cardiac output; an improvement in the Qp/Qs ratio to 1.72 ratio; the biphasic nature of flow through the AV valves without regurgitation of these valves or the arterial valves. After 7 days, a normal aCGH result was obtained from the chorionic villus biopsy.

A follow-up scan after next 5 days showed no fetal oedema; sinus rhythm at 147–150 bpm with normal atrioventricular and arterial valve function, and normal DV flow (positive a-wave, normal D-wave, PVIV = 0.84; PLI = 0.71). Systemic SV was 0.1 ml; systemic cardiac output was unchanged; pulmonary SV measured 0.21 ml; pulmonary cardiac output was 30 ml/min; and the Qp/Qs ratio was 2.15.

Maintenance doses of amiodarone (800 mg/d) were used, and the fetus was monitored once a week, until reaching 18 weeks + 4 days. The beneficial effect of therapy was reflected in normalization of DV velocimetry (Figure 5).

No abnormalities were shown at that time. In this situation, we decided to terminate therapy and the pregnant woman was referred for further follow-up to referring obstetrician, remaining in contact with the reference centre. By the end of the pregnancy, there were no abnormalities in structural and functional ultrasound assessment of the fetal cardio-vascular system.

The baby was born at 39 weeks of gestation through natural labour. Neonatal mass was 3840 g, and 10 points were scored on the Apgar scale. No abnormalities were found in ECG and neonatal echocardiography (Figure 6). The definitive cause of late first trimester supraventricular tachycardia in the described case has not been determined.

Discussion

The case we present is the earliest report in the literature of the successful use of amiodarone in the late first trimester for supraventricular tachycardia. To date, there has been a single documented case of treating SVT in the 13th week



Figure 4. The primary determinants of fetal improvement after 3 days of amiodarone treatment: reduction in NT thickness (top); improvement in venous flow profile (middle); return of the biphasic nature of inflow to the right ventricle (bottom)

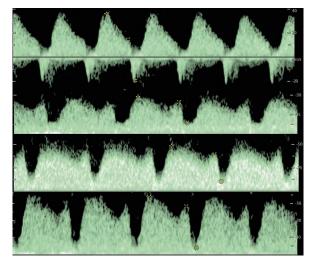


Figure 5. Changes in ductus venosus velocimetry in the first 4 weeks of follow-up

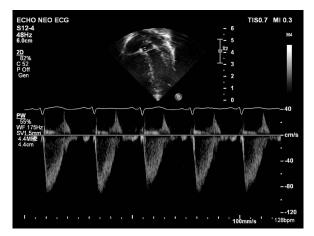


Figure 6. Neonatal echocardiogram presenting the sinus rhythm of the described case

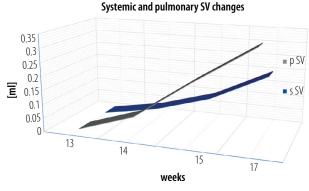


Figure 7. Changes in fetal stroke volume under amiodarone treatment

of pregnancy with digoxin in combination with flecainide [5]. The authors may predict that postponing diagnostic, consultative, and therapeutic management could lead to spontaneous cardiac failure of the presented fetus. It should be noted that with similar clinical images, the first stage of management is often to refer for genetic diagnostics (with regard to trisomy 13), with amniocentesis after 15 weeks of gestation. The waiting time for the test and the results could lead to severe fetal cardiac failure and ultimately result in fetal demise. In the case described, it was assumed that a detailed early anatomy scan and early fetal echocardiography, which showed no anomalies, provided a solid basis for the emergency management implemented. This ultimately led to a normal obstetric outcome [6].

In an earlier paper by the authors, extracardiac abnormalities were demonstrated in 53.6% and cardiac abnormalities in 41.1% of fetuses with trisomy 13 [7]. Tachycardia was raised as a common feature of this condition [8-11]. Typically, in an uncomplicated pregnancy, FHR increases from about 110 bpm at 5 weeks of gestation to 170 bpm at 9 weeks, with subsequent gradual decrease in FHR values observed, reaching about 150 bpm at 13 weeks of gestation⁷. In the previous study we observed mean FHR in fetuses with trisomy 13 at 170 bpm (range: 148-201 bpm) and an FHR above the 95th percentile in 62.5% of them [7]. None of the 56 cases of Patau syndrome analysed previously had FHR values exceeding 210 bpm, which can be considered another argument for the validity of our approach involving early therapy introduction [7].

Given the one case described so far in the literature, the authors believe that individualization of treatment is essential. Therefore, we used direct karyotyping, also known as overnight karyotyping [12] and implemented detailed monitoring of treatment effects. Because of the low cardiac output that is known to result from supraventricular tachycardia, we suggest that in similar cases, in addition to the traditional parameters of atrioventricular valve and ductus venosus velocimetry, evaluation of total cardiac output parameters should also be used. In the case described herein, their improvement went hand in hand with the normalization of the ductus venosus flow profile and the return of flow phasicity of the ventricular inflows, and, most importantly, the resolution of the incipient subcutaneous tissue oedema. The most pronounced changes over the course of treatment were observed in terms of an increase in both stroke volumes, especially that of the right ventricle (Figure 7),

which is considered one of the more significant prognostically favourable parameters.

It should be emphasized that cardiac output parameter monitoring in such an early fetus, considering the determinants of optimal translucency of the patient's abdominal wall and the associated prime BMI for the scan, as well as the availability of transabdominal high-frequency transducer, will not be possible in every case.

Conflict of interest

The authors declare no conflict of interest.

REFERENCES

- 1. Yuan SM, Xu ZY. Fetal arrhythmias: prenatal evaluation and intrauterine therapeutics. Ital J Pediatr 2020; 46: 21.
- Macalintal JCM. Transplacental therapy of supraventricular tachycardia and hydrops fetalis in a twin pregnancy – case report and literature review. Prenat Cardio 2021; 11: 59-69.
- Jaeggi E, Fouron JC, Fournier A, Van Doesburg N, Drblik SP, Proulx F. Ventriculo-atrial time interval measured on M mode echocardiography: a determining element in diagnosis, treatment, and prognosis of fetal supraventricular tachycardia. Heart 1998; 79: 582-587.
- Donofrio MT, Moon-Grady AJ, Hornberger LK, Copel JA, Sklansky MS, Abuhamad A, et al. Diagnosis and treatment of fetal cardiac disease: a scientific statement from the american heart association. Circulation 2014; 129: 2183-2242.
- Porat S, Anteby EY, Hamani Y, Yagel S. Fetal supraventricular tachycardia diagnosed and treated at 13 weeks of gestation: a case report. Ultrasound Obstet Gynecol 2003; 21: 302-305.
- Bravo-Valenzuela NJ, Araujo Júnior E. Inappropriate fetal sinus tachycardia at 9 weeks of gestation. Ann Pediatr Cardiol 2022; 15: 399-403.
- Rajs B, Nocuń A, Matyszkiewicz A, Pasternok M, Kołodziejski M, Wiercińska E, et al. First-trimester presentation of ultrasound findings in trisomy 13 and validation of multiparameter ultrasound-based risk calculation models to detect trisomy 13 in the late first trimester. J Perinat Med 2020; 49: 341-352.
- Hyett JA, Noble PL, Snijders RJM, Montenegro N, Nicolaides KH. Fetal heart rate in trisomy 21 and other chromosomal abnormalities at 10-14 weeks of gestation. Ultrasound Obstet Gynecol 1996; 7: 239-244.
- Liao AW, Snijders R, Geerts L, Spencer K, Nicolaides KH. Fetal heart rate in chromosomally abnormal fetuses. Ultrasound Obstet Gynecol 2000; 16: 610-613.
- Kagan KO, Wright D, Valencia C, Maiz N, Nicolaides KH. Screening for trisomies 21, 18 and 13 by maternal age, fetal nuchal translucency, fetal heart rate, free beta-hCG and pregnancy-associated plasma protein-A. Hum Reprod 2008; 23: 1968-1975.
- Rajs B, Pasternok M, Nocuń A, Matyszkiewicz A, Ziętek D, Rozmus-Warcholińska W, et al. Clinical article: screening for trisomy 13 using traditional combined screening versus an ultrasound-based protocol. J Matern Neonatal Med 2019; 34: 1048-1054.
- 12. Brambati B, Simoni G. Diagnosis of fetal trisomy 21 in first trimester. Lancet (London, England) 1983; 8324: 586.

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