THE JOURNAL OF OBSTETRICS AND GYNECOLOGY OF INDIA

The Journal of Obstetrics and Gynecology of India (July–August 2019) 69(4):383–386
https://doi.org/10.1007/s13224-018-1147-z

SHORT COMMENTARY

Ventricular Tachycardia in a Fetus: Benign Course of a Malignant Arrhythmia

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Received: 10 December 2016 / Accepted: 14 June 2018 / Published online: 29 June 2018

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Abstract

Background Fetal arrhythmias constitute 10 to 20% of the total referrals to fetal cardiology unit. Ventricular tachycardia (VT) is a rare form of fetal tachyarrhythmia.

Materials and methods This report describes the clinical presentation, diagnostic features, and successful management of VT in a fetus at 32 weeks of gestation. Transplacental therapy with sotalol resulted in the termination of tachyarrhythmia in 48 h, followed by good postnatal outcome.

Conclusion Fetal m-mode showing ventricular rate higher than atrial rate with atrio-ventricular dissociation is suggestive of VT. Early diagnosis and institution of transplacental therapy prior to development of hydrops seem to

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carry a good prognosis even in malignant fetal arrhythmias like VT.

Keywords Fetal tachyarrhythmia · Ventricular tachycardia · Prenatal diagnosis · Transplacental therapy

Case Summary

A 24-year-old primigravida was referred at 32 weeks of gestation for management of fetal tachycardia. Fetal echocardiogram revealed structurally normal heart with no evidence of hydrops or ventricular dysfunction. M-mode of the atria and ventricle revealed a regular atrial rate of 131/min and regular ventricular rate of 213/min. There was evidence of atrio-ventricular (AV) dissociation (Fig. 1a). A differential diagnosis of ventricular tachycardia (VT) and junctional ectopic tachycardia (JET) was considered as the ventricular rate was more than atrial rate in the presence of atrio-ventricular dissociation. Doppler interrogation revealed bizarre inflow–outflow relationship with no definite pattern (Fig. 1b). There was no peak-to-peak variability on outflow Doppler.

Maternal serum electrolyte levels were sodium 137 meq/L, potassium 3.2 meq/L, and magnesium 1.9 mg/dl. Intravenous magnesium sulfate and oral potassium supplements were given to maintain adequate levels. She was started on oral sotalol at a dose of 60 mg eighth hourly, which was stepped up to 80 mg eighth hourly after 24 h. Maternal QTc was monitored regularly. Fetal echocardiogram after 48 h of initiation of treatment revealed termination of tachycardia. M-mode revealed a normal sinus rhythm with atrial and ventricular rate of 120/min (Fig. 1c). Inflow–outflow Doppler showed 1:1 AV association with atri-oventricular interval shorter than ventriculo-atrial interval, thus confirming a normal sinus rhythm (Fig. 1d). She was continued on sotalol at a dose of 60 mg eighth hourly. Follow-up assessment at 34 and 36 weeks showed normal heart rate and rhythm. Baby was

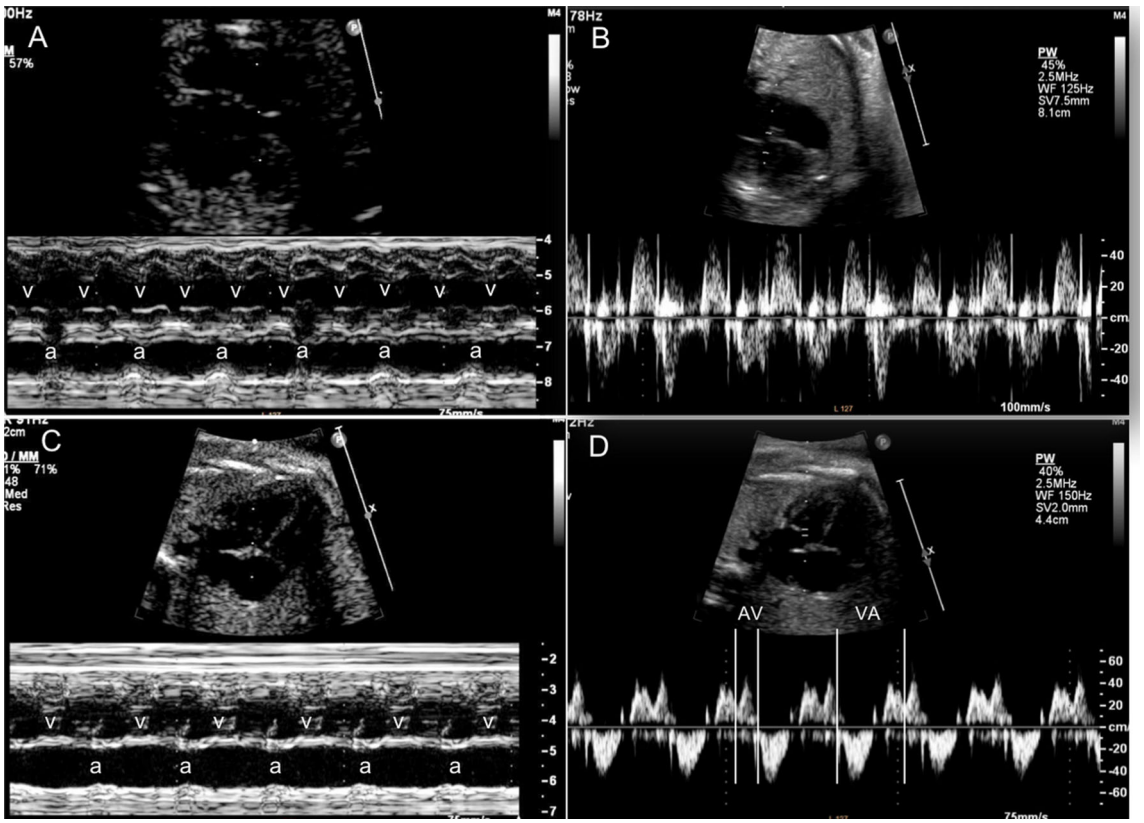

Fig. 1 Fetal echocardiographic images before and after arrhythmia management. **a** M-mode of ventricle and atria showing regular ventricular contractions (v) at the rate of 213/min in the upper panel and regular atrial contractions (a) at the rate of 131/min in the lower panel. The ventricular rate is higher than the atrial rate with atrio-ventricular dissociation. **b** Doppler interrogation during tachyarrhythmia showing bizarre inflow–outflow relationship with no definite pattern. **c** M-mode following arrhythmia management showing regular ventricular (v) and atrial (a) contractions at the rate of 120/min. **d** Doppler interrogation following arrhythmia management showing 1:1 atrio-ventricular association. Note that the atrio-ventricular (AV) interval is less than the ventriculo-atrial (VA) interval

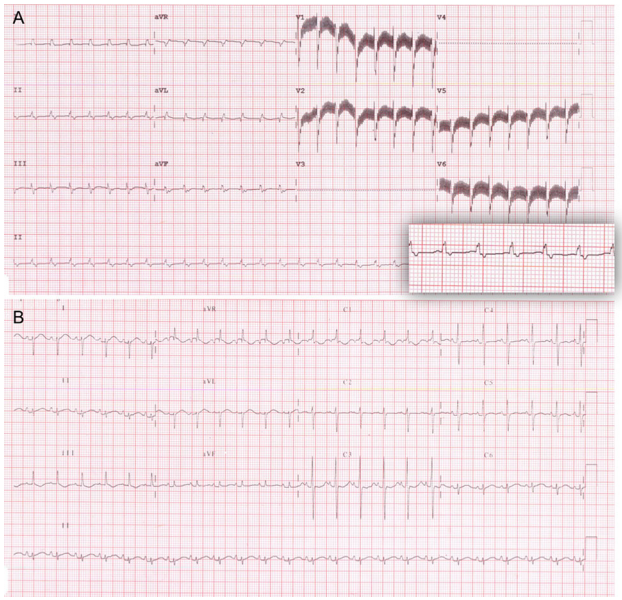

Fig. 2 **a** Electrocardiogram of the neonate immediately after birth showing a broad complex tachycardia at the rate of 185/min. Rhythm strip shown in the inset reveals 1:1 ventriculo-atrial conduction. **b** Electrocardiogram showing sinus rhythm at the rate of 110/min. Note that there is a shift in QRS axis along with morphology after termination of tachyarrhythmia

delivered at 39 weeks gestation by elective cesarean section at a perinatology center. APGAR was 10 at 1 min. Electrocardiogram of the neonate immediately after birth revealed a broad complex tachycardia (heart rate 185/min) with 1:1 ventriculo-atrial conduction (Fig. 2a). However, the arrhythmia got terminated during transfer to cardiac intensive care unit. Subsequent ECG revealed a normal sinus rhythm with heart rate of 110/min. QTc interval was normal (440 ms). On comparing the ECGs during tachycardia and sinus rhythm, it was noted that there was change in QRS axis and morphology during tachycardia (Fig. 2b). This confirmed the diagnosis of VT. Transthoracic echocardiogram showed normal cardiac function, no evidence of any tumors, and absence of structural heart defects. Baby was started on oral β-blocker (propanolol 1 mg/kg/dose 12th hourly) and discharged after 2 days. Periodic assessment during the follow-up showed sinus rhythm and normal QTc. Propranolol was discontinued at one year.

Discussion

Fetal tachyarrhythmias constitute 10–20% of the total referrals to fetal cardiology [1]. Most of these are benign atrial ectopics; only a few are clinically significant. The most common tachyarrhythmias are atrio-ventricular re-entry tachycardia and atrial flutter. VT is rare, constituting only 1–2% of fetal arrhythmias.

A systematic assessment of atrial rate, ventricular rate and relationship of atrial and ventricular events on m-mode and Doppler would help in the differential diagnosis of fetal tachycardias. In our case, a ventricular rate higher than atrial rate in the presence of atrio-ventricular dissociation led us to a differential diagnosis of VT and JET. A definite differentiation between the two is difficult by fetal echocardiography. The diagnosis was confirmed only after birth in our case.

Prenatal VT can be secondary to fetal myocarditis, cardiac tumors and long QT syndrome [2]. Fetal myocarditis due to viral or isoimmune etiology carries a poor prognosis. Solitary or multiple rhabdomyomas can present as VT and long-term prognosis generally depends on association with Tuberous sclerosis complex. VT secondary to long QT syndrome (LQTS) has to be ruled out as the management and prognosis would differ from other etiologies. Family history of sudden deaths, long QTc on ECG of parents, alternating bradycardia and tachycardia, intermittent 2:1 atrio-ventricular block, and evidence of polymorphic VT (peak-to-peak variability on outflow Doppler) are suggestive of congenital LQTS [3, 4]. None of these features were present in our case, thus making the diagnosis of LQTS unlikely. The etiology of VT appears to be idiopathic in our case.

Though some authors have suggested conservative management of fetal VT [3] in the absence of hemodynamic compromise, we believe in instituting transplacental therapy on diagnosis as persistent tachycardia can result in ventricular dysfunction. We started on sotalol as a possibility of LQTS was unlikely in our case. Beta-blockers may be started if LQTS is suspected. Intravenous magnesium sulfate to the mother is the drug of choice in polymorphic fetal VT [3]. Amiodarone and flecainide are the other drugs which are shown to be effective [2]. Dexamethasone and intravenous immunoglobulin may be useful if it is secondary to myocarditis. As most of the data are based on individual case reports, the treatment has to be individualized by the clinician. The prognosis is good in the absence of structural heart defects, myocardial disease, and LQTS, as in our case.

Compliance with Ethical Standards

Conflict of interest The authors declare that they have no conflict of interest.

Informed Consent Informed consent was obtained from the patient for inclusion in this publication.

Research Involving Human Participants or Animals This article does not contain any study with human or animal subject.

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