

Terbutaline-induced neonatal ventricular tachycardia: A case report and review of literature

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ABSTRACT

Neonatal ventricular tachycardia (VT) is an extremely rare condition. We present a 35-week-old gestation neonate who developed tachycardia following maternal exposure to terbutaline. Upon transfer to our neonatal intensive care unit, an electrocardiogram (ECG) was obtained which was consistent with VT. The arrhythmia did not respond to vagal maneuvers or adenosine but resolved following cardioversion demonstrated on postconversion ECG. At outpatient follow-up, the infant had no further episodes of arrhythmia. To the best of our knowledge, this represents the first case describing terbutaline-induced fetal or neonatal VT.

Keywords: Atrioventricular dissociation, fetus, neonate, terbutaline, ventricular tachycardia

INTRODUCTION

Ventricular tachycardia (VT) in neonates is exceedingly rare,^[1] with the majority of VT occurring in the setting of myocarditis, long QT syndrome, or structural heart disease.^[2] Perinatal exposure to medications is another cause of VT in neonates. Beta-2 agonists are sometimes used for short-term tocolysis,^[3] with fetal sinus tachycardia as the most common side effect.^[4] Rarely, arrhythmias including VT may occur. Previously, ritodrine exposure was reported as an etiology for short-duration VT in a neonate.^[5] We report a case of VT in a neonate, most likely resulting from *in utero* exposure to terbutaline.

CASE REPORT

A female neonate born at 35 weeks of gestational age was transferred to our neonatal intensive care unit (NICU) for the evaluation and management of persistent tachycardia. She was born at an outside facility via cesarean delivery due to a concern for

partial placental abruption. Before the delivery, the mother was given two subcutaneous doses of 0.25 mg of terbutaline to achieve tocolysis so as to give time for the antenatal corticosteroid to act. However, approximately 20 min after the administration of the second dose of terbutaline, the fetal heart rate was noted to rise from 150–160 beats per minute (bpm) to 180–190 bpm, necessitating an expedited delivery for suspected nonreassuring fetal status.

At the delivery, no placental abruption was noted. The infant was vigorous with 1 and 5 min Apgar scores of 8 and 9, respectively. Once placed on cardiac monitoring, the infant was noted to have tachycardia with a heart rate of 205 bpm. Laboratory studies were obtained for suspected sepsis, and infant received a dose of both ampicillin and gentamicin. Both umbilical cord blood gas and postnatal arterial blood gases were reassuring, thus ruling out significant perinatal asphyxia. Further laboratory evaluation for electrolyte abnormalities, including serum calcium levels, yielded normal results.

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Twelve-lead electrocardiogram (ECG) was concerning for a wide QRS complex tachyarrhythmia, so a decision was made to transfer the infant to our NICU for further evaluation and treatment.

Upon arrival to the NICU, the infant was noted to be persistently tachycardic with heart rates ranging from 190 to 210 bpm. Pediatric cardiology was consulted, and an ECG was obtained. Supraventricular tachycardia with aberrancy versus VT was the initial impression because of the width of the QRS complexes, left-axis deviation, and the random occurrence of P waves in relation to QRS complexes, suggesting atrioventricular (AV) node dissociation [Figure 1, arrows]. Vagal maneuvers were unsuccessful, so the patient was given two doses of adenosine, which were also unsuccessful. Synchronized cardioversion was then performed, and the arrhythmia resolved after the second shock. Postconversion heart rate ranged from 120 to 140 bpm, and the postconversion ECG showed the typical rightward QRS axis for a neonate with a normal and shorter QRS duration compared to the preconversion ECG [Figure 2]. Echocardiogram showed a structurally normal heart with normal cardiac function. The infant remained asymptomatic with no breakthrough episodes during the 1st year of follow-up at the pediatric cardiology clinic.

DISCUSSION

VT is extraordinarily rare in neonates.^[1,2] In the current case, the diagnosis of VT was based on an elevated heart rate with wide and bizarre QRS complexes with irregular appearance of P waves with AV dissociation. A major differential diagnosis includes supraventricular tachycardia with aberrant conduction pathway or antidromic conduction through the AV node. However, in the index case, AV dissociation with left-axis deviation on the ECG strongly suggested the presence of VT.

The etiology for the occurrence of VT was attributed to terbutaline exposure based on the temporality of the tachycardia, absence of structural heart disease, normal electrolytes, and a normal postconversion ECG. The absence of breakthrough episodes of VT during the 1st year of follow-up further substantiated the association of terbutaline with VT. Our case highlights the fact that in the absence of common etiologies for VT, iatrogenic causes of VT need to be strongly considered.

Terbutaline is a beta-2 sympathomimetic agent occasionally used in the perinatal period for tocolysis. Terbutaline can readily cross the placenta, and the fetal blood levels of terbutaline can rapidly rise up to 55% of maternal blood levels, following subcutaneous administration^[6] Subcutaneous terbutaline has only been

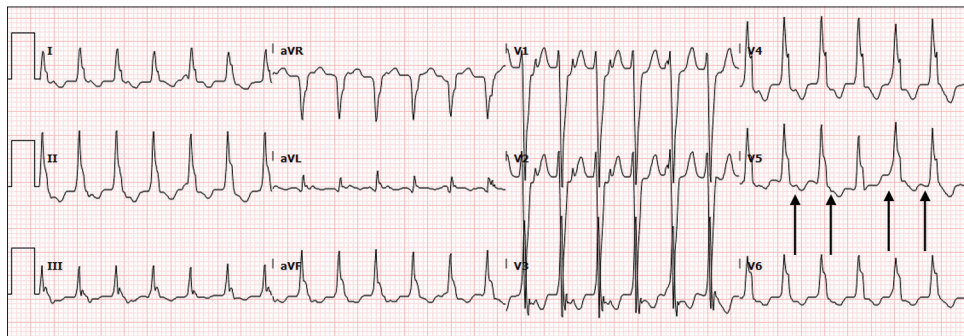


Figure 1: Electrocardiogram before cardioversion showing wide complex tachycardia with left-axis deviation and atrioventricular node dissociation (note annotated P waves) consistent with ventricular tachycardia

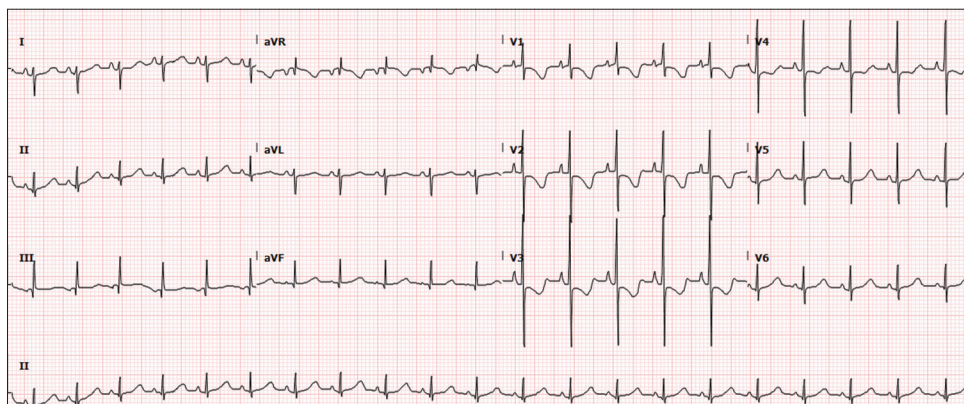


Figure 2: Electrocardiogram postcardioversion showing sinus rhythm

used off-label for short-term tocolysis, and caution against the repeated or prolonged subcutaneous administration of terbutaline for tocolysis beyond 48–72 h among pregnant women exists on the label.^[7] One of the well-known side effects of beta-2 agonists is fetal^[8] and neonatal sinus tachycardia, because they exert a chronotropic effect by decreasing the refractory period and sinus cycle length of the sinoatrial node.^[4] By also shortening the refractory periods of cardiac myocytes, they facilitate enhanced automaticity of both atria and ventricles.^[8] Other beta-2 sympathomimetic agents such as ritodrine have also been implicated as the cause of neonatal VT.^[5]

CONCLUSION

We report a rare case of VT in a neonate due to perinatal exposure to terbutaline, a beta-2 sympathomimetic medication used as a tocolytic agent. To the best of our knowledge, this is the first case describing terbutaline-induced fetal or neonatal VT. Our case represents a rare yet important side effect of antenatal administration of terbutaline of which providers should be aware.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the legal guardian has given consent for images and other clinical information to be reported in the journal. The guardian understands that the names and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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