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Neonatal Supraventricular Tachycardia – A Diagnostic Challenge

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ABSTRACT

We report a case of a patient admitted to the neonatal intensive care unit at birth for grunting and respiratory distress. A diagnosis was made after the patient presented with signs of serious poor perfusion and cardiorespiratory monitoring showed a rapid cardiac rhythm suggesting supraventricular tachycardia (SVT). Hemodynamic stability was achieved after several adenosine boluses and the SVT was reverted. Episodes of SVT are characterized by abrupt onset and termination and regular narrow QRS complex tachycardia without P waves. This diagnosis should be kept in mind in neonates when a sudden vascular change resembling cutis marmorata happens since it can suggest cardiogenic shock caused by an underlying arrhythmia.

Keywords

Tachycardia, Supraventricular, Shock, Cardiogenic, Infant, Newborn.

Introduction

Supraventricular tachycardia (SVT) is the most common symptomatic arrhythmia in children. It usually appears in the first year of life with a slight male predominance. There is a wide clinical spectrum from hydrops fetalis *in utero* to congestive heart failure in newborns and infants and palpitations in older children [1,2]. Unspecific symptoms like poor feeding, vomiting, lethargy, pallor or poor perfusion may represent a diagnostic challenge in the neonatal period as they can often be caused by sepsis or metabolic disorders. Medical management of SVT consists of vagal maneuvers, adenosine and synchronous cardioversion if hemodynamic instability is present.

We report a case of a full-term male newborn admitted to the neonatal intensive care unit that showed signs of severe poor perfusion and a coincidental abrupt change of rhythm in the cardiorespiratory monitoring that highly suggested supraventricular tachycardia (SVT).

Case Report

The infant was a third-born male in a non-consanguineous marriage. There was no family history of cardiac disease. The antenatal history was uneventful besides isolated ventricular fetal extrasystoles detected at 24 weeks' gestation. He was born at full term from spontaneous vaginal delivery to a 31-year-old mother. Apgar score was 8 and 10 at 1 and 5 minutes, respectively, and birth weight 3,44 kg. On examination, the vital signs were normal but the child presented grunting and nasal flaring. Cardiac auscultation showed rhythmic heart sounds without murmurs and pulmonary auscultation was normal. Chest radiograph showed a normal heart shadow and pulmonary parenchyma without opacity or infiltrating shadows. He was transferred to our neonatal intensive care unit (NICU) for surveillance. Sepsis work-up was normal. 30 minutes after admission the patient presented with respiratory distress, hypotension, sudden pallor and a pinkish-blue mottled skin with a marbled appearance resembling *cutis marmorata* (Figure 1 and 2). Cardiorespiratory monitoring showed a heart rate of 245-260 beats/min and regular narrow QRS complex tachycardia without *P* waves that suggested supraventricular tachycardia. Vagal maneuvers were started by placing an ice pack on the neonate's face followed by administration of intravenous adenosine. After

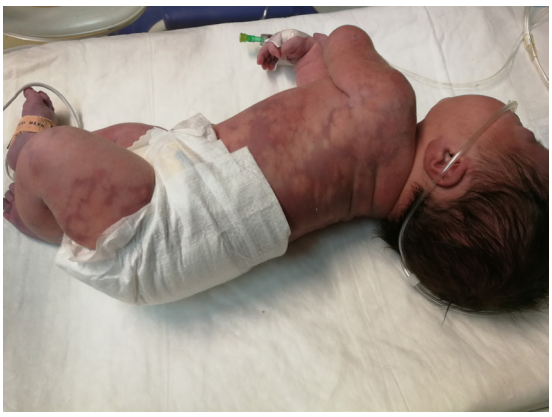
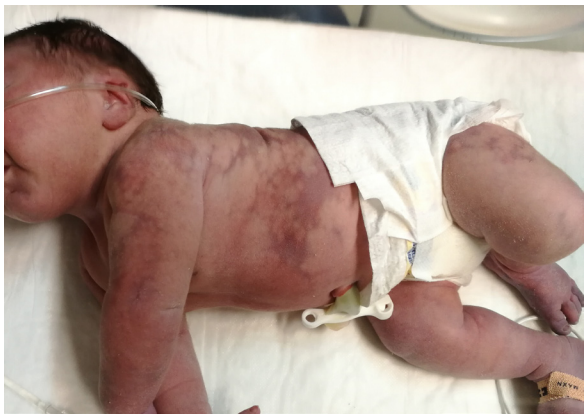

Figure 1 and 2: Newborn showing vascular changes resembling cutis marmorata.

two initial boluses of 0,1 mg/kg and three boluses of 0,2 mg/kg the SVT was reverted in a few minutes. An ECG was performed afterwards showing normal sinus rhythm and the transthoracic echocardiogram displayed a small *foramen ovale* and patent *ductus arteriosus* but no structural lesions and adequate function.

After evaluation by a pediatric cardiologist, he started oral propranolol 1 mg/kg/dose three times a day, which was suspended at 6 months of age. The infant is currently 2 years old and there was no recurrence of SVT.

Discussion

SVT is the most common symptomatic arrhythmia in childhood that can be a recurrent and persistent condition. The estimated incidence of SVT in neonates is 1 in 200 to 250 for neonates. SVT is a tachyarrhythmia with an origin proximal to the bundle of His. The typical infant who has SVT has a regular R-R interval, with rates often greater than 230 beats/min. The normal cardiac impulse originates in the sinus node and travels antegrade through the atria to the atrioventricular (AV) node. Under normal physiologic conditions, the ventricles are protected from a rapid atrial rate because of the innate delay in the AV node. Abnormal reentrant circuits in the myocardium, resulting in SVT, bypass this delay. The most common type of SVT in infancy and childhood, representing approximately 70% of cases, is atrioventricular reentrant tachycardia. Most patients with SVT have structurally normal hearts but it may be associated with congenital heart disease [1-3].

There is a wide clinical spectrum for SVT and infants tend to present more nonspecific symptoms than older children do. In our case, the neonate presented respiratory distress and sudden signs of severe poor perfusion (Figure 1 and 2) coincidental with abrupt regular narrow complex QRS tachycardia without *P* waves shown in the cardiorespiratory monitor. To our knowledge, this is the first case report of neonatal SVT manifested by skin changes resembling *cutis marmorata.* This vascular change represents an accentuated physiologic vasomotor response in the newborn that is sometimes discernible in cardiogenic shock [4,5].

A 12-lead ECG should ideally be performed during the SVT episode but this should not delay treatment in order to prevent cardiac failure and cardiogenic shock. In this case, the ECG was performed only after SVT was resolved and showed no evidence of ventricular pre-excitation suggestive of Wolf-Parkinson-White syndrome.

When considering treatment for acute SVT, the most important diagnostic decision is if there is hemodynamic compromise since unstable SVT is a medical emergency warranting immediate action. Hemodynamically unstable patients with a tachydysrhythmia and signs of shock should receive immediate synchronized cardioversion. In stable patients with SVT, vagal maneuvers, such as Valsalva, and application of ice to the patient's face have been shown to successfully terminate SVT in 20%–63% of cases. Other maneuvers such as inducing a gag reflex, applying ocular pressure and carotid massage are no longer recommended. Adenosine is an AV nodal blocking agent and is the initial drug of choice for conversion of SVT. The most common starting dose is 0.1 mg/kg, followed by 0.2 mg/kg (to a maximum of 12 mg) given in the most central location possible via a rapid IV push with immediate saline flush. If medical therapy with adenosine is unsuccessful, or stable SVT deteriorates to unstable SVT, synchronized cardioversion is needed. The initial charge is 0.2-0.5 J/kg and may be increased up to 1 J/kg on the second attempt. Diagnosis and termination can also be achieved with transesophageal electrophysiology testing [6,7]. In our case despite hemodynamic compromise, the first-line approach was adenosine bearing in mind its rapid administration and the vast experience with this drug in our NICU.

Beta-blockers, particularly propranolol, have become established as the most common first-line agent for maintenance therapy in infants being the standard practice to continue treatment for 6 months to a year followed by a period of weaning. A randomized, blinded clinical trial comparing digoxin *versus* propranolol demonstrated no first recurrences in either group between 6 and 12 months suggesting that our follow-up treatment practice was adequate [8,9].

60 to 90% of patients have spontaneous SVT resolution during the first year of life and the prognosis seems to be better if the first episode occurred in the first month of life [10].

In conclusion, this case report highlights the atypical presentation of neonatal SVT resembling *cutis marmorata* promptly diagnosed due to cardiorespiratory monitoring. This diagnosis should be kept in mind in infants with sudden signs of poor perfusion since SVT in early infancy is dangerous and potentially fatal if not recognized and treated appropriately.

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