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CASE REPORT

Supraventricular Tachycardia in a Newborn with Camptodactyly: Case Report

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Abstract

Neonatal supraventricular tachycardia (SVT) is defined as an abnormal increase in heart rate originating above the Bundle of His.Camptodactyly is a congenital deformity characterized by progressive and non-traumatic flexion of the proximal interphalangeal joint (PIP). We describe the clinical presentation and management of supraventricular tachycardia in neonates, a condition that is not very common in the daily practice of neonatologists, as well as patients with camptodactyly, and this association has not yet been reported in the literature. We emphasize early diagnosis and adequate treatment, both acutely, during the crisis, and also as prophylaxis for new episodes. New studies may confirm whether this association is just fortuitous (chance).

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INTRODUCTION

Neonatal supraventricular tachycardia (SVT) is defined as an abnormal increase in heart rate, typically above 220 bpm, originating above the His bundle, with an electrocardiographic pattern showing absent or altered P waves and a narrow QRS complex (<80 ms).[1] It is a condition with a variable clinical spectrum that may occur in patients with or without congenital heart disease[1,2].

According to a 2023 systematic review, although uncommon in the neonatal period, SVT represents the second most frequent tachyarrhythmia[3]. The reentry mechanism is the most prominent in the genesis of neonatal SVT[2,4-6].

Its etiology remains uncertain, with an autosomal dominant inheritance pattern reported in many cases, although some are sporadic[7,8].

The classification of camptodactyly is based on age of onset and severity of contractures. The most common form is type I (infantile), which arises in the first years of life, most often affecting the little finger unilaterally or bilaterally and generally being of cosmetic significance only. Type II appears later, usually during the pubertal growth spurt. Type III, syndromic camptodactyly, presents at birth—as in the case reported—affecting multiple fingers bilaterally and potentially being associated with genetic syndromes[8,9].

Management of camptodactyly varies according to the severity of the contracture[7,9]. The literature on the association between SVT and camptodactyly in neonates is limited, and to date, we have not identified any publication establishing a correlation between these two conditions.

CASE REPORT

A full-term male newborn, delivered at 39 weeks of gestation, weighing 4,200 g, classified as large for gestational age. The newborn was delivered via vaginal birth, without complications, with Apgar scores of 9 and 10.

The pregnancy was uneventful. The mother, 21 years old, with no known comorbidities, G2P1A0, attended 11 prenatal consultations and had negative serologies. There was no history of gestational diabetes.

The newborn was admitted to rooming-in, breastfeeding exclusively, and underwent capillary glucose monitoring according to institutional protocol.

On initial examination, he presented palpable and symmetric pulses, a heart rate of 130 beats per minute, respiratory rate of 50 breaths per minute, and no abnormalities on auscultation. The abdomen and genitalia were normal. Primitive neurological reflexes were present and symmetric. Bilateral hand deformities were observed (Figures 1a and 1b), as well as syndactyly of the 4th and 5th toes on both feet, resembling congenital anomalies also present in the mother (Figures 1c and 1d).

With 31 hours of life, during pulse oximetry screening, the newborn presented an isolated episode of tachycardia with a heart rate above 200 bpm, accompanied by cyanosis, and was admitted to the neonatal ICU. The newborn was placed in a heated incubator under continuous monitoring and electrocardiography, breathing room air. The episode resolved spontaneously. After 5 hours of observation in the ICU, he presented a new episode, with a heart rate of 290 bpm, diagnosed as supraventricular tachycardia (Figure 2), accompanied by peripheral cyanosis and decreased activity. A vagal maneuver was performed by applying ice to the forehead, and, as sinus rhythm was not restored, an intravenous loading dose of amiodarone (5 mg/kg) was administered, followed by an oral maintenance dose of 5 mg/kg/day every 12 hours, resulting in reversal of the SVT. Enteral feeding was suspended and intravenous hydration was initiated.

At 46 hours of life, the newborn presented new episodes of SVT. The first was reversed with a vagal maneuver, and the subsequent episodes were reversed with intravenous amiodarone, initially infused at 10 mg/kg/hour and later increased to 20 mg/kg/hour.

On the morning of the third day of life, he presented SVT with a heart rate reaching 300 bpm, lasting approximately 1 hour, even after vagal maneuvers (application of an ice pack), and was associated with peripheral cyanosis. In the evening, he presented two additional SVT episodes, both controlled after a few minutes with vagal maneuvers.

On the fourth day of life, the newborn had no new episodes of SVT, and enteral feeding was restarted via gastric

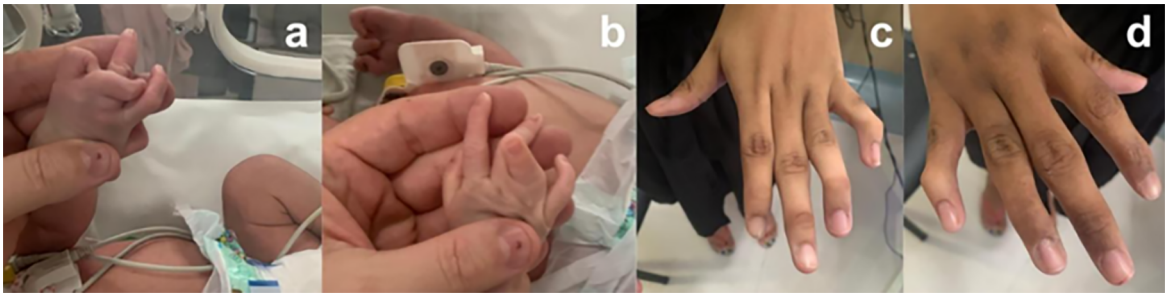
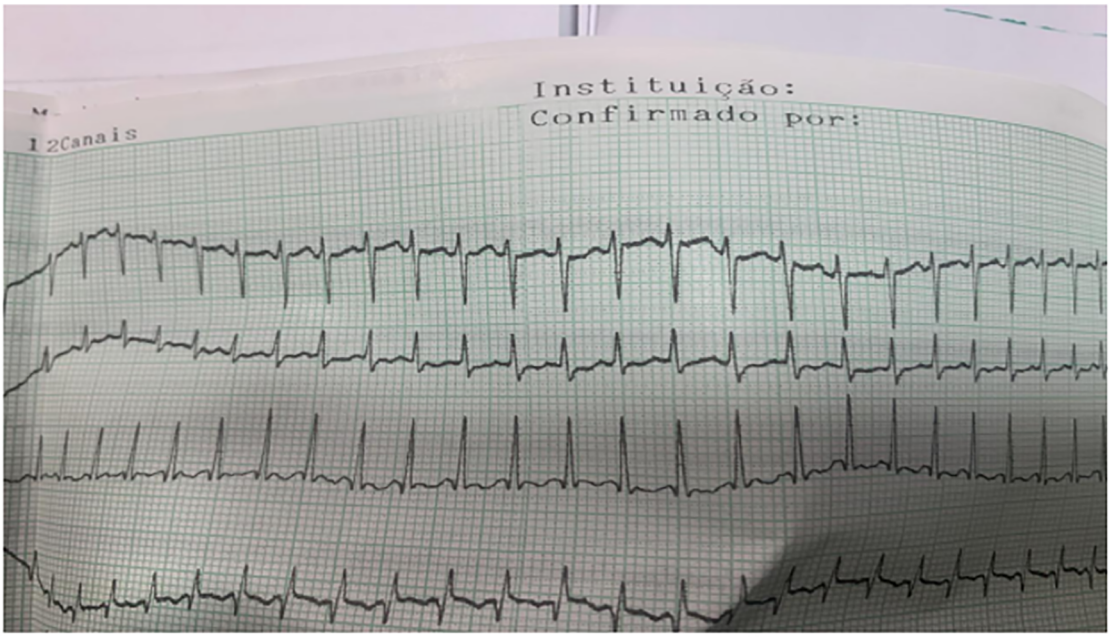


Figure 1. Photographs of hand abnormalities - newborn and mother. Caption: *Bilateral hand deformities in the newborn and mother*. 1a. Clinodactyly of the 4th finger and camptodactyly of the 5th finger of the newborn's right hand. 1b. Clinodactyly and camptodactyly of the 4th and 5th fingers of the newborn's left hand. 1c. Clinodactyly and camptodactyly of the 4th and 5th fingers of the mother's left hand. 1d. Bilateral clinodactyly and camptodactyly of the 4th and 5th fingers of the mother's right hand.



2A - taquicardia sinusal



2B - TSV - DII longo - velocidade 50m\s (dobrada)

Figure 2. ECGs of the clinical case. Caption: (2a) Electrocardiograms performed on the newborn during hospitalization. ECG showing sinus tachycardia, with a narrow QRS complex, regular rhythm, and heart rate around 175 bpm. (2b) ECG showing paroxysmal supraventricular tachycardia, characterized by a fast and regular heart rate (280 bpm), with narrow QRS complexes and difficulty identifying P waves.

tube using infant formula. The following day, propranolol was added at 2 mg/kg/day, orally, and feeding was advanced.

On the sixth day of life, the newborn had episodes of vomiting, and amiodarone was discontinued, maintaining only propranolol. A transthoracic echocardiogram revealed a patent foramen ovale and indirect signs of mild pulmonary arterial hypertension, with preserved biventricular systolic function.

After 12 days of hospitalization, the newborn was discharged with a prescription for propranolol and vitamin D, feeding exclusively on breast milk, and instructed to follow outpatient care. The echocardiogram performed on the day of discharge showed persistence of the patent foramen ovale, normal biventricular systolic function, and no signs of pulmonary hypertension.

The newborn completed neonatal screening tests, which revealed no abnormalities. During hospitalization, blood gas analysis, complete blood count, electrolyte panel, renal and liver function tests were performed, as well as cranial ultrasound via the fontanelle and abdominal ultrasound, with no abnormalities detected.

The newborn continues to be followed in outpatient pediatric cardiology and orthopedics, with continued use of propranolol and the need for bilateral orthoses on the fingers.

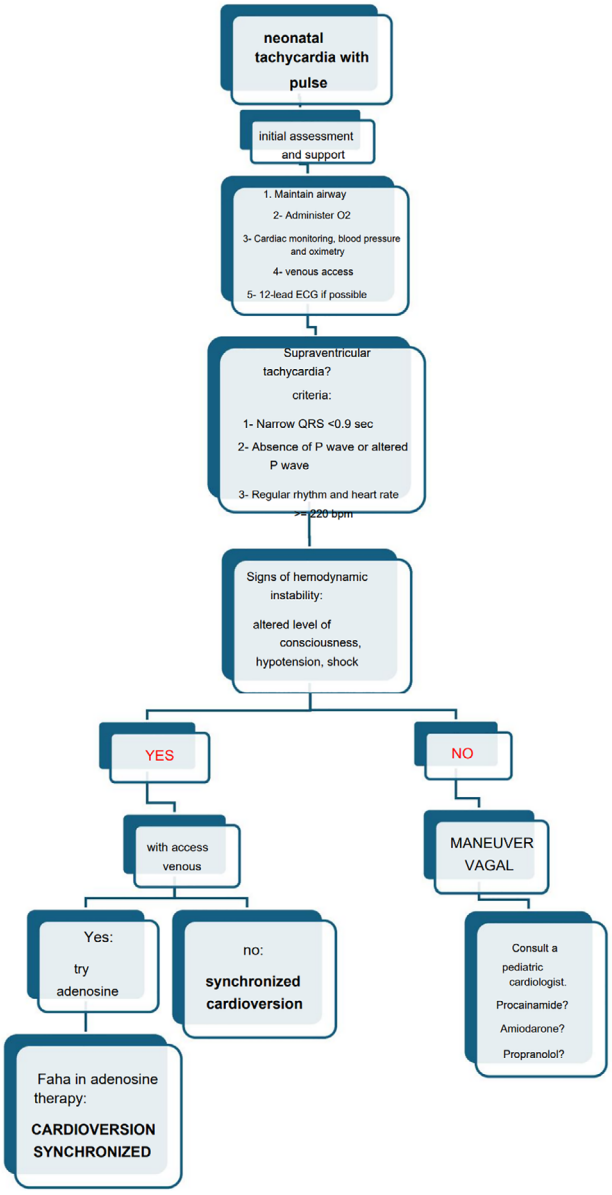


Figure 3. Flowchart of neonatal supraventricular tachycardia treatment - based on: Oeffl et al. (2023)[3] Arrhythmias Requiring Electric Countershock during the Neonatal Period - A Systematic Review (reference 3 of the article).

DISCUSSION

Neonates with a heart rate above 180 BPM may exhibit hemodynamic compromise, constituting a medical emergency[3].SVT comprises a group of tachyarrhythmias originating above the His bundle[1,6]. The primary goals are to restore normal cardiac rhythm and stabilize hemodynamics. After clinical stabilization, it becomes essential to confirm the specific type of supraventricular tachyarrhythmia based on surface electrocardiogram analysis[6] recorded during and after arrhythmia resolution. In this case, the analysis identified supraventricular tachycardia.

Supraventricular tachycardia (SVT) can be defined as an abnormal heart rate originating above the ventricles, usually (but not always) with a narrow QRS complex, excluding atrial flutter and atrial fibrillation from this definition.

The literature describes congenital heart disease, electrolyte disturbances, sepsis, and placement of central venous catheters as risk factors[3].

Clinical management of newborns with SVT can be challenging because the exact arrhythmia mechanism is often unknown. SVT may present in utero or in the neonatal period[4]. With advances in modern fetal echocardiography, the diagnosis of sustained fetal arrhythmias has become increasingly common[3]. In the case presented, no abnormalities were identified in the prenatal fetal echocardiogram.

The clinical presentation, although nonspecific, may include pallor, cyanosis, irritability, diaphoresis, tachypnea, feeding difficulties, or palpitations[1,4].

The treatment of neonatal SVT considers the pathophysiological involvement of the atrioventricular node and is structured differently for the acute and maintenance phases[1,3,5].In the acute approach, the goal is to restore sinus rhythm[5]. Among therapeutic options, vagal maneuvers are preferred in hemodynamically stable cases, while cardioversion is reserved for patients with hemodynamic instability or signs of heart failure[1-4,6]. A commonly used vagal maneuver in neonatal ICUs involves applying a bag of ice and water to the infant's face, simulating the "diving reflex." Ocular compression and carotid sinus massage are avoided as they may cause harm to the newborn.

In the present case, vagal maneuvers and amiodarone were used. The literature recommends adenosine when vagal maneuvers fail. It should be administered as a rapid infusion through an adequate intravenous line placed as close to the heart as possible. Adenosine administration also aids in diagnosing SVT when an ECG is recorded simultaneously[6]. For maintenance therapy, first-line antiarrhythmic options include non-selective beta-blockers such as propranolol or digoxin[1-3,5]. Digoxin should be avoided when pre-excitation syndromes, such as Wolff-Parkinson-White, are suspected[2,3]. Studies highlight that in cases of refractory SVT, cardioversion offers no benefit, making antiarrhythmic therapy or radiofrequency ablation necessary for definitive treatment[4,6].

To detect congenital structural heart anomalies and ventricular function abnormalities, routine transthoracic echocardiography is essential in the follow-up of neonates with SVT[1,3]. In the case described, the initial imaging showed only indirect signs of mild pulmonary arterial hypertension unrelated to the arrhythmia. Subsequent examinations did not reveal such alteration.

The literature suggests that appropriate management of neonatal SVT reduces complications and fatal outcomes. In most cases, spontaneous resolution occurs during the first year of life[1,2,4,6].

In the present case, the newborn exhibited bilateral camptodactyly with a family history of the same condition, suggesting a possible autosomal dominant inheritance.[7,8] The use of orthoses was recommended, with surgical treatment reserved for more severe cases[9].

The combined manifestation of SVT and camptodactyly has not been previously reported in the literature according to the authors' review and is most likely a coincidental finding.

CONCLUSION

Supraventricular tachycardia remains a clinical challenge for neonatologists and pediatric cardiologists, both in its proper management during the acute phase—also referred to as the conversion phase aimed at restoring normal sinus rhythm—and in the maintenance phase to prevent new episodes. Hemodynamic stability is the most important factor guiding treatment decisions. The association with non-cardiac malformations appears to have been merely incidental and lacks support from comparable studies in the literature. Neonatology services must be adequately equipped and have access to pediatric cardiologists for guidance in such cases. Prognosis depends on the presence of concomitant structural cardiac malformations and the initial response to treatment. Cardiologic follow-up is essential to elucidate the underlying pathophysiological mechanism and to guide definitive therapy, such as radiofrequency ablation.

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