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Supraventricular tachycardia in children – a case series with short-term follow-up

Częstoskurcz nadkomorowy u dzieci – seria przypadków z obserwacją krótkoterminową

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Abstract

Aim: Tachyarrhythmias are the most common symptomatic cardiac rhythm disorders in children, with an incidence of approximately 1 in 250–1,000 and possible presentation at any age. This study aimed to present illustrative paediatric cases of supraventricular tachycardia and their short-term follow-up. **Materials and methods:** Five paediatric patients with supraventricular tachycardia admitted to the Department of Paediatrics at the Multispecialty Hospital in Rybnik between 2021 and 2023 were analysed. Follow-up data were obtained from outpatient evaluations and cardiology records through 2025. **Results:** Non-pharmacological measures were ineffective in all patients. Intravenous adenosine successfully terminated supraventricular tachycardia in every case: three patients converted after a single dose, and two after a second dose. Long-term management included beta-blockers; one patient required additional flecainide therapy, and one is awaiting catheter ablation. **Conclusion:** Intravenous adenosine is highly effective for acute termination of supraventricular tachycardia in children, while individualised long-term pharmacological therapy remains essential for rhythm control.

Keywords: children, supraventricular tachycardia, tachyarrhythmias

Streszczenie

Cel: Tachyarytmie są najczęstszymi objawowymi zaburzeniami rytmu serca u dzieci, z częstością występowania około 1 na 250–1000 i możliwością ujawnienia się w każdym wieku. Celem badania było przedstawienie ilustracyjnych przypadków częstoskurczu nadkomorowego u dzieci i ich obserwacji. **Materiał i metody:** Przeanalizowano pięcioro dzieci z częstoskurczem nadkomorowym hospitalizowanych na Oddziale Pediatrii Szpitala Specjalistycznego w Rybniku w latach 2021–2023. Dane z obserwacji uzyskano na podstawie dokumentacji kardiologicznej do 2025 roku. **Wyniki:** Metody niefarmakologiczne przerywania częstoskurczu okazały się nieskuteczne u wszystkich pacjentów. Dożylne podanie adenozyny skutecznie przerwało częstoskurcz nadkomorowy we wszystkich przypadkach: u trzech pacjentów konwersja nastąpiła po jednej dawce, a u dwóch – po drugiej. Leczenie długoterminowe obejmowało stosowanie beta-adrenolityków, u jednego pacjenta konieczne było dodatkowe włączenie flekainidu, a jeden pacjent oczekuje na zabieg ablacji. **Wnioski:** Dożylne stosowanie adenozyny jest wysoce skuteczne w ostrym przerywaniu częstoskurczu nadkomorowego u dzieci, a indywidualnie dobrana długoterminowa terapia farmakologiczna ma zasadnicze znaczenie dla kontroli rytmu.

Słowa kluczowe: dzieci, częstoskurcz nadkomorowy, tachyarytmie

INTRODUCTION

Tachyarrhythmias are the most common symptomatic cardiac arrhythmias in children, with an incidence of roughly 1 in 250–1,000 and potential onset at any age[1]. Prolonged tachycardia may lead to heart failure, particularly in younger children; therefore, accurate identification of the tachycardia type and prompt initiation of appropriate treatment are crucial[2].

INCLUSION CRITERIA

Among 11 paediatric patients with supraventricular tachycardia (SVT) hospitalised in the Department of Paediatrics at the Multispecialty Hospital in Rybnik between 2021 and 2023, five clinically illustrative cases representing different age groups were selected for detailed analysis. Follow-up data were obtained through 2025 from specialist outpatient evaluations and cardiology records.

CASE 1

A 7-day-old neonate (second pregnancy, second delivery, spontaneous vaginal birth at 38 weeks of gestation; birth weight: 2,895 g; Apgar score: 10; unremarkable antenatal and perinatal history) was admitted to the Department of Paediatrics due to poor weight gain and jaundice.
Physical examination revealed jaundice (total bilirubin: 255.1 μmol/L), SpO_2: 99%, and a heart rate of 140 bpm. Biochemical and microbiological tests confirmed a urinary tract infection, and treatment with ampicillin and gentamicin was initiated.
On the third day of hospitalisation, the infant developed hypotonia, apathy, and peripheral cyanosis. Examination showed rapid jugular venous pulsations, weak peripheral pulses, and a heart rate of approximately 270 bpm. SVT could not be confirmed due to the unavailability of appropriately sized ECG leads, and the patient was transferred to the Neonatal Intensive Care Unit.

Follow-up

Upon admission, an ECG demonstrated a narrow-QRS tachycardia with a short RP interval and a heart rate of 270 bpm. Following intravenous administration of 0.3 mg (equivalent to 1 mg/kg) of adenosine, the tachycardia resolved and the infant's clinical condition improved.
Echocardiography excluded any haemodynamically significant congenital heart defect, and myocardial contractility was within normal limits. Propranolol therapy was initiated; however, due to recurrence of the arrhythmia, treatment was modified to oral propafenone in a dual-drug regimen, resulting in cessation of tachycardia episodes.
Follow-up ECG recordings demonstrated widening of the QRS complexes to 100 ms. Therapy was adjusted accordingly, and normalisation of cardiac rhythm was ultimately achieved with combination treatment consisting of amiodarone and propranolol, as confirmed by 24-hour Holter monitoring.
During the following days of hospitalisation, the patient's condition remained stable. Oral antiarrhythmic therapy was continued without recurrence of paroxysmal arrhythmia. The patient remains under follow-up at the Paediatric Cardiology Outpatient Clinic.

CASE 2

A 2.5-year-old boy was admitted to the Department of Paediatrics due to the sudden onset of weakness, pallor, and excessive sweating. Similar episodes had occurred several times since the age of one year, each resolving spontaneously. Family history was notable for paroxysmal tachycardia in the mother.
Physical examination revealed tachycardia exceeding 200 bpm, with no other abnormalities. ECG demonstrated SVT at a rate of 250 bpm. Laboratory investigations showed no significant abnormalities.
Intravenous adenosine was administered twice at a dose of 0.1 mg/kg, without restoration of sinus rhythm. After increasing the dose to 0.2 mg/kg, conversion to normal sinus rhythm was achieved. The patient was transferred to the Department of Paediatric Cardiology.

Follow-up

Upon admission, echocardiography revealed a structurally normal heart with normal contractility. The ECG showed sinus rhythm with no features of pre-excitation (no shortened PR interval, no prolonged QRS duration, no delta waves, and no secondary ST-T abnormalities). Treatment with propafenone at a dose of 30 mg three times daily (6 mg/kg/day) was initiated.
Subsequent 24-hour Holter monitoring recorded two episodes of tachycardia at 250 bpm. Pharmacotherapy was modified by adding propranolol at a dose of 7 mg three times daily (1.4 mg/kg/day), and the patient was discharged home with a recommendation for follow-up at the Paediatric Cardiology Outpatient Clinic in two months.
During the first outpatient visit, according to the mother's report, three episodes possibly corresponding to tachycardia had occurred since discharge (manifesting as restlessness and rapid heartbeat, with normalisation after sleep). The current treatment regimen was continued.
One week after the clinic visit, the patient presented to the Department of Paediatric Emergency with SVT at approximately 200 bpm lasting about one hour, accompanied by malaise. The episode was terminated following administration of adenosine at a dose of 0.2 mg/kg. The patient remained under observation in the Department of Paediatrics, and following consultation with the Department of Paediatric Cardiology, the propafenone dose was increased to 40 mg orally three times daily (200 mg/m^2).

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During subsequent follow-up visits at one, two, four, six, nine, and fifteen months, no further tachycardia episodes were observed, and the current treatment was maintained. The next follow-up visit is scheduled in one year.

CASE 3

A 3-year-old girl was admitted to the Department of Paediatrics with pneumonia. On initial assessment, her heart rate was within normal limits. During an evening examination, marked tachycardia at a rate of 250 bpm was noted and confirmed on ECG, establishing the diagnosis of SVT. The child's general condition was good; she reported no complaints, and vital signs remained within normal limits.

A Valsalva manoeuvre was attempted to restore sinus rhythm, but without effect. Follow-up laboratory tests showed normal electrolyte levels and compensated metabolic acidosis on blood gas analysis.

After consultation with the on-call physician from the Department of Paediatric Cardiology, a rapid intravenous bolus of adenosine was administered. Following a dose of 1.5 mg (0.09 mg/kg), a regular rhythm of 150 bpm was restored. After consultation with the Department of Paediatric Cardiology, metoprolol was subsequently initiated at a dose of 6 mg twice daily.

Follow-up

The patient was hospitalised twice due to recurrent episodes of SVT. As metoprolol therapy proved only partially effective, propafenone was added at a dose of 40 mg three times daily (8 mg/kg/day). However, the medication was poorly tolerated because of its taste. After two months, the patient was admitted to the Paediatric Cardiology Unit, where the metoprolol dose was increased to 1.35 mg/kg/day and flecainide was initiated under continuous ECG monitoring, with gradual dose escalation to 4 mg/kg/day.

Over the following seven months, the patient experienced six episodes of SVT lasting from approximately 30 minutes to two hours. During longer arrhythmic events, she was initially restless and later became apathetic. During another hospitalisation in the Paediatric Cardiology Unit, her previous therapeutic regimen was maintained.

During the subsequent two years, outpatient follow-up visits occurred every three months. During this period, one SVT episode occurred in the context of fever, and two additional episodes were documented on 24-hour Holter ECG monitoring. Most recently, the patient was hospitalised for a respiratory tract infection, during which three further SVT episodes were recorded.

The patient is currently receiving oral metoprolol and flecainide. Dosages have been adjusted according to increasing body weight to 1.4 mg/kg/day and 3.3 mg/kg/day, respectively.

CASE 4

A 5-year-old boy was admitted with his first episode of SVT. His history included surgical repair of a ventricular septal defect at five months of age, with ongoing cardiology follow-up. On the day of admission, he developed left ear pain with purulent otorrhoea and palpitations. Emergency medical services documented SVT on ECG (Fig. 1), and administration of 3 mg of adenosine (0.17 mg/kg/day) restored sinus rhythm.

On arrival at the Department of Hospital Emergency, he was haemodynamically and respiratory stable, febrile to 38.6°C, and ECG showed sinus rhythm at 125 bpm with right bundle branch block.

Physical examination revealed generalised warmth, a postoperative chest scar, a soft systolic murmur at Erb's point, pharyngeal inflammation, and bilateral acute otitis media with left tympanic membrane perforation. Laboratory tests revealed mildly elevated inflammatory markers, leucocytosis with segmented predominance, hypomagnesaemia, and low-normal potassium.

Treatment included antibiotic therapy and electrolyte supplementation. Following cardiology consultation, a beta-blocker was initiated. The patient was discharged home with instructions to continue metoprolol 12.5 mg twice daily (1.43 mg/kg/day) and electrolyte supplementation until the next cardiology follow-up.

Follow-up

During subsequent follow-up visits at the Paediatric Cardiology Outpatient Clinic, conducted every six months, the metoprolol dose was gradually reduced and ultimately discontinued over the course of one year. The patient is currently off beta-blocker therapy, has had no further episodes of SVT, and has been advised to continue annual follow-up at the outpatient clinic.

CASE 5

A 17.5-year-old girl was admitted to the Department of Paediatrics for a week-long history of chest pain and palpitations accompanied by weakness and reduced exercise tolerance. She had experienced three similar exertion-related episodes in the past, each resolving spontaneously. Family history was notable for maternal arrhythmia treated with bisoprolol, although the specific type was not documented.

On physical examination, her heart rate was 180 bpm. ECG demonstrated SVT. Laboratory tests revealed hypomagnesaemia and hypochloraemia. A Valsalva manoeuvre was attempted without effect. Ultimately, administration of 6 mg adenosine restored normal heart rate; however, the ECG showed rhythm disturbances with additional ventricular ectopic beats.

Following cardiology consultation, intravenous potassium, metoprolol, and magnesium supplementation were

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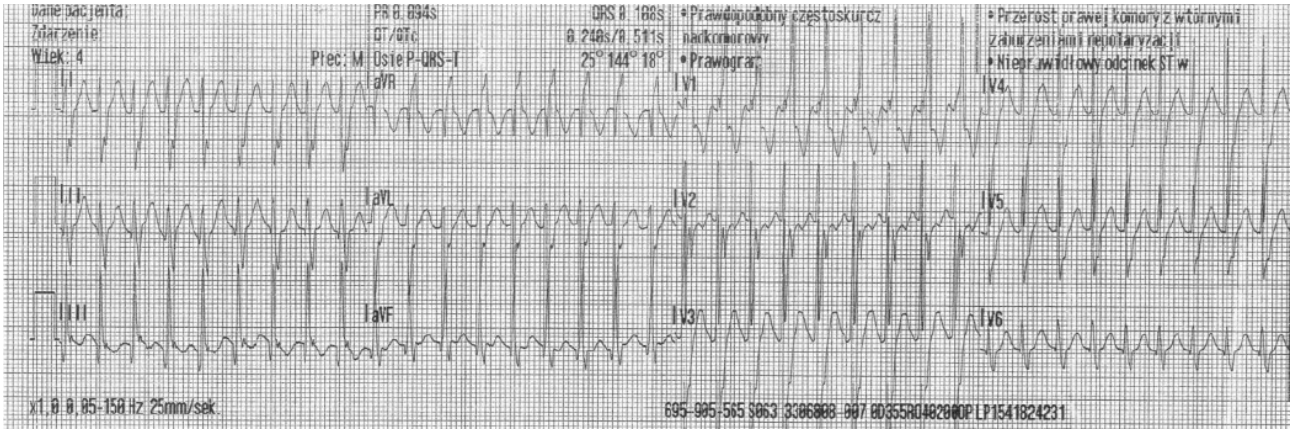

Fig. 1. Narrow-QRS tachycardia with a regular ventricular rate of 220 bpm and no clearly visible P waves. The ECG pattern is consistent with orthodromic atrioventricular re-entrant tachycardia or atrioventricular nodal re-entrant tachycardia

initiated, resulting in gradual normalisation of the ECG. The patient was discharged with recommendations for ongoing pharmacotherapy, including metoprolol 50 mg orally twice daily and oral magnesium supplementation, as well as cardiology follow-up.

Follow-up

The patient was admitted to the Department of Paediatric Cardiology four months later, having had no further SVT episodes. Echocardiography showed a structurally normal heart with preserved systolic function. 24-hour Holter monitoring recorded approximately 4,000 aberrantly conducted premature beats. She was discharged on the same medications and is awaiting radiofrequency catheter ablation while remaining under follow-up in the Adult Cardiology Clinic.

RESULTS

Non-pharmacological measures failed to restore sinus rhythm in all cases; conversion was achieved only after adenosine. Three patients responded to a single dose, while two required a second. For long-term prophylaxis, most were started on a beta-blocker, one received combined beta-blocker and flecainide therapy, and one is awaiting catheter ablation.

DISCUSSION OF CHARACTERISTIC SYMPTOMS

In paediatric practice, the most common rhythm abnormality is sinus tachycardia, typically triggered by exertion, fever, pain, anxiety, anaemia, dehydration, hyperthyroidism, heart failure, or myocarditis.

It is usually well tolerated, and treating the underlying cause generally restores a normal heart rate[2].

In contrast, SVT often requires non-pharmacological or pharmacological termination, making accurate differentiation from narrow-complex sinus tachycardia essential (Tab. 1)[3].

Symptoms of tachycardia in children vary by age (Tab. 2). Even marked arrhythmias may go unnoticed, and in young children – especially infants – SVT often presents with non-specific symptoms[4].

TREATMENT

Non-pharmacological methods for terminating supraventricular tachycardia

Management of SVT includes attempts to stimulate the vagus nerve. In infants, this may involve the application of a cold compress to the head. In older children, vagal manoeuvres include performing a Valsalva manoeuvre (e.g. blowing through a straw or pushing the plunger of a syringe)[1].

Pharmacological methods for terminating supraventricular tachycardia

If first-line non-pharmacological interventions are ineffective, adenosine should be administered as a rapid intravenous bolus at a dose of 0.1 mg/kg body weight, and in children under one year of age at a dose of 0.15 mg. If the initial dose is ineffective, adenosine may be repeated after three minutes at twice the previous dose, i.e. 0.2 mg/kg body weight. Adenosine slows conduction through the atrioventricular node and terminates nodal re-entrant tachycardia[5]. Adenosine is rapidly metabolised, with a half-life of only 5–10 seconds. Therefore, it may be safely re-administered within one minute of the preceding dose[6]. Adenosine should be administered via central access or a peripheral vein in the upper limb as a very rapid bolus, immediately followed by a rapid saline flush[7]. The expected antiarrhythmic efficacy exceeds 90%[6].

Adenosine is contraindicated for tachyarrhythmia termination in several situations. It should not be used in atrial fibrillation or flutter with pre-excitation (e.g. Wolff–Parkinson–White syndrome), as it may enhance conduction

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Type of tachycardia	Sinus tachycardia	Supraventricular tachycardia with narrow QRS complexes
History	Fever, fluid loss, pain, stress	Non-specific
Heart rate	<220/min – neonates <180/min – children	>220/min – neonates >180/min – children
P wave	Present/normal >200/min difficult to assess	Absent/abnormal
R-R variability	Variable depending on situation, respiratory sinus arrhythmia	Constant
Onset/termination	Gradual	Sudden

Tab. 1. Differentiation of sinus tachycardia from supraventricular tachycardia with narrow QRS complexes

Neonates and infants	Older children
• Child distress • Loss of appetite • Tachypnoea • Excessive sweating • Pallor • Irritability • Cardiogenic shock	• Palpitations – predominant symptom • Fatigue • Chest discomfort • Stabbing chest pain • Neck pulsations (characteristic) • Shortness of breath • Visual disturbances • Paroxysmal cough and dyspnoea • Abdominal pain • Syncope/near-syncope

Tab. 2. Clinical symptoms of supraventricular tachycardia according to the child's age

through the accessory pathway and trigger ventricular fibrillation. It is also contraindicated in second- or third-degree AV block and in sick sinus syndrome due to the risk of worsening conduction abnormalities. Caution is required in patients with asthma or reactive airway disease because of the potential for bronchospasm[6].

Adenosine is associated with a relatively high incidence of adverse effects; however, these are generally transient and clinically benign, reflecting the drug's ultra-short plasma half-life (<10 seconds). The most frequently reported immediate adverse effects include facial flushing, dyspnoea, and chest pain[8].

Adenosine may induce transient electrophysiologic effects, including sinus pauses, sinus bradycardia, transient complete atrioventricular block, and brief episodes of asystole. Following rapid bolus administration, premature atrial and ventricular depolarisations may occur; these ectopic beats can occasionally trigger reinitiation of re-entrant tachyarrhythmias[8,9].

Although uncommon, adenosine may precipitate atrial fibrillation, likely secondary to shortening of the atrial refractory period. In most cases, adenosine-induced atrial fibrillation is self-terminating and clinically insignificant. However, in patients with an accessory atrioventricular pathway, atrial fibrillation with rapid conduction over the accessory pathway may occur and can be potentially life-threatening[8].

Clinically significant bronchoconstriction is rare but has been reported following intravenous adenosine administration for the treatment of SVT[6].

Alternative methods for SVT termination are generally used in specialised care. Pharmacological options include flecainide, propafenone, and procainamide; in infants, amiodarone may be used when other treatments fail, although conversion may take hours. Transoesophageal atrial overdrive pacing is another option. Synchronised electrical cardioversion should be reserved for haemodynamically unstable patients[10].

CONCLUSIONS

Intravenous adenosine is highly effective for acute SVT termination in children, and individualised pharmacological therapy is essential for long-term rhythm control.

Conflict of interests

The authors do not report any financial or personal connections with other persons or organisations which might negatively affect the content of this publication and/or claim authorship rights to this publication.

Author contribution

Original concept of study; collection, recording and/or compilation of data: WGK, TB, AZ. Analysis and interpretation of data; writing of manuscript: AP. Critical review of manuscript; final approval of manuscript: KM.

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