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Paroxysmal Supraventricular Tachycardia In A Child: A Clinical Case Treated With Amiodarone And Management

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Abstract:

This report presents a case of a Vietnamese child with paroxysmal supraventricular tachycardia and discusses diagnosis and management, clinical features, and electrocardiogram to help diagnose arrhythmia in children. This girl child, Acute management has been more successful when using Amiodarone in the absence of some first-line drugs such as Adenosine as well as some other conventional drugs that require caution. However, long-term management continues to be a topic of debate, especially in special situations.

Keywords: Paroxysmal supraventricular tachycardia, pathophysiology, diagnosis, drug treatment, children.

INTRODUCTION

Paroxysmal Supraventricular Tachycardia (PSVT) is a common arrhythmia in all ages, most commonly in children requiring treatment. This arrhythmia is often a manifestation of an accessory pathway in addition to the atrioventricular pathway that allows impulses from the atria to the ventricles to return to the normal pathway, thus completing the circuit and stimulating the atria and ventricles at a rapid rate. Infants and young children often present with poor feeding and tachypnea, while palpitations and chest discomfort are prominent symptoms in older children [1-4]. Electrocardiography (ECG) shows fine QRS complex tachycardia at a rate of > 220 per minute and together with the clinical picture, provides certainty in the diagnosis in the majority of patients [5-9]. In some situations, recognition is difficult due to the nonspecific symptoms and often self-limiting nature of the disorder, and long-term management continues to be a subject of further debate, especially for each specific situation [1-2].

However, in recent years, new insights have emerged about the natural history and mechanisms of supraventricular tachycardia in children [9-11] With advances in arrhythmia prevention and treatment, there are now many treatment options. In this article, we present the recent case of PSVT encountered in a child in Vietnam and discuss the diagnosis as well as a framework that can help select appropriate treatments for children with PSVT attack.

CLINICAL CASE PRESENTATION

A 6-year-old female patient was admitted to the hospital on January 2024, in the Emergency Room of the Pediatrics Department of the International Hospital in Vietnam because of chest pain, difficulty breathing in the first hour, signs of cyanosis, vomiting, and nausea.

On examination, the child is awake, has difficulty breathing, is tired, has no fever, is not vomiting, pale lips, no rales on lung auscultation, fast and small pulse of 228 cycles/minute, hard to detect radial pulse, signs of early peripheral hypoperfusion (Refill < 2s), soft abdomen, no hepatosplenomegaly, negative meningeal syndrome, Sp02 88%, blood pressure is difficult to measure during tachycardia.

The pediatric patient was treated and had an immediate clinical examination performed at the Emergency Department, and emergency treatment was performed. Breathing 1% oxygen, test sympathetic strength with eyeball pressure and carotid sinus massage to bring the sinus rhythm to 202-212 cycles/minute, after a few minutes the tachycardia returns, it's a mild response to vagal testing.

Heart rate electrocardiogram results: Paroxysmal supraventricular tachycardia with frequency of 212 cycles/minute. Dopper echocardiography showed severe tricuspid valve regurgitation (3/4), mild increase in pulmonary artery pressure (32 mmHg), and no atrial septal defect, ventricular septal defect or patent ductus arteriosus detected. Heart function is normal (left ventricular ejection fraction 66.8%), thyroid ultrasound has not detected any abnormalities, and abdominal ultrasound and chest X-ray have not detected pathological abnormalities.

Blood test results: Red blood cells 4.96 T/L (limit index: 3.9-4.6), HGB 133 g/L (limit index 109-163), White blood cells 13.1 G/L (6.0-17.5), Platelets: 335 G/L. Blood biochemical test: Urea 5.59 mmol/L, Creatinine 45.3 mol/L,

GOT/GPT: 29.1/15.1 mmol/L, Na^{+} /K^{+} /Cl^{-} : 140.9/3.9/105 mmol/L and Ca^{++} 1.26 mmol/L, CRP: 0.4 mg/L. Thyroid hormone test: FT3 8.61 pmol/l, FT4 7.9-14.4 pmol/L, TSH 1,195 uIU/mL.

Thyroid test results were normal. In a situation where Adenosine was not available at this time, it was decided to treat the tachycardia by intravenous infusion with an electric syringe with Amiodarone bolus starting dose's 150 mg/tube mixed with 50ml Glucose 5% intravenous bolus infusion for 10 minutes. After 10 minutes of recording the electrocardiogram, the heart rate returned to sinus rhythm with a frequency of 136 cycles/minute, the patient was awake, had much less difficulty breathing, and continued to maintain the dose of Amiodarone 1mg/minute intravenously for 6 hours, only After 3 hours, continue to maintain the dose, measure the Heart Rate again to Sinus Rhythm frequency of 109 Cycles/minute, SpO2 98%. Blood pressure is stable, the patient is awake, and his heart is no longer short of breathing, We stopped the Amiodarone infusion and continued monitoring on the Monitor, no more episodes of paroxysmal supraventricular tachycardia appear during the day. The results of the Holter electrocardiogram were normal followed by paroxysmal supraventricular tachycardia as well as other arrhythmias on the Holter electrocardiogram after the day of ablation of paroxysmal supraventricular tachycardia.

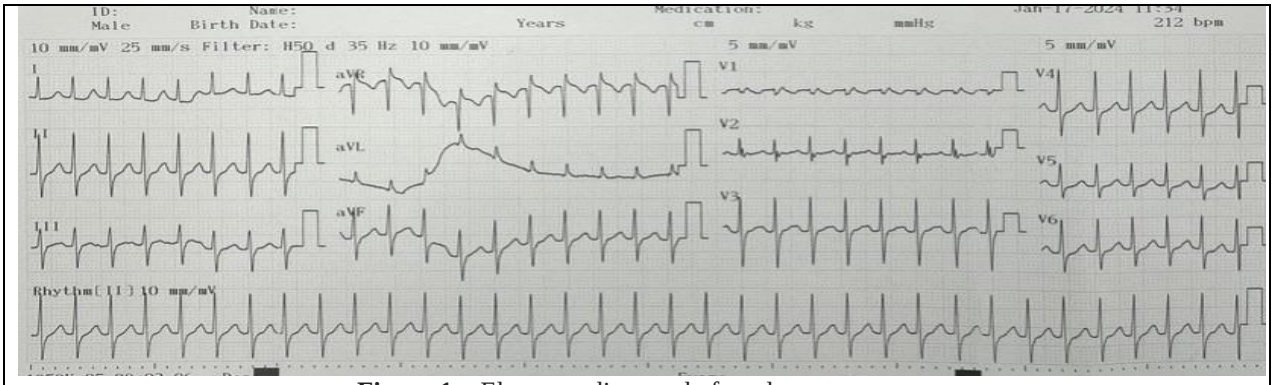

Figure 1a: Electrocardiogram before drug treatment

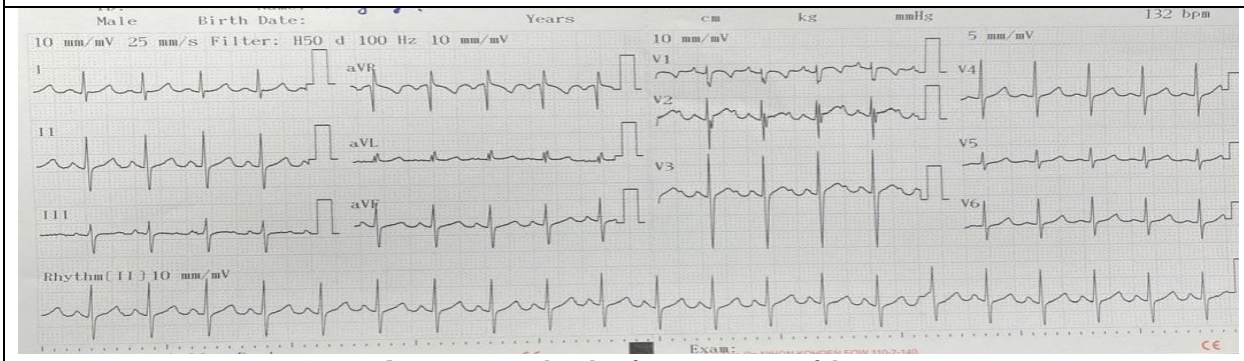

Figure 1b: ECG immediately after 15 minutes of drug treatment

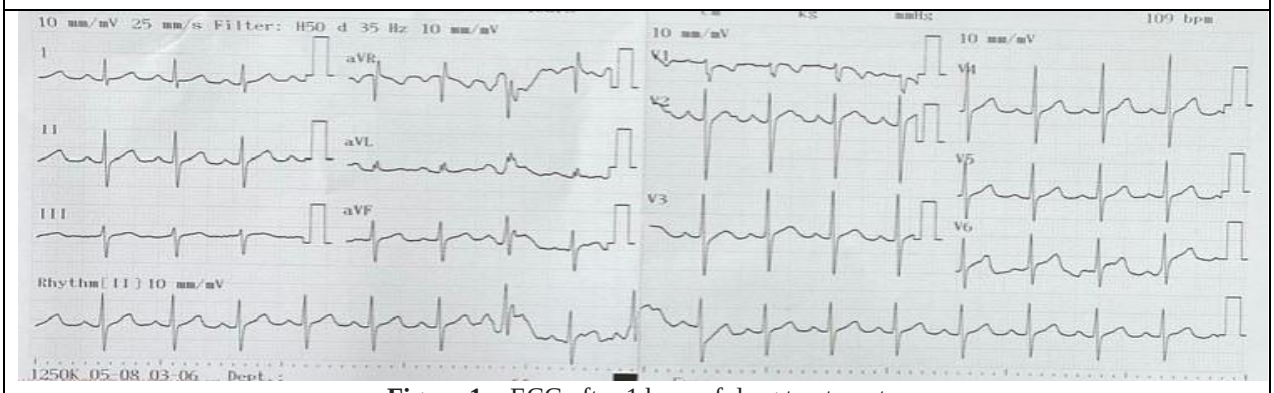

Figure 1c: ECG after 1 hour of drug treatment

Figure 2: Doppler echocardiography image

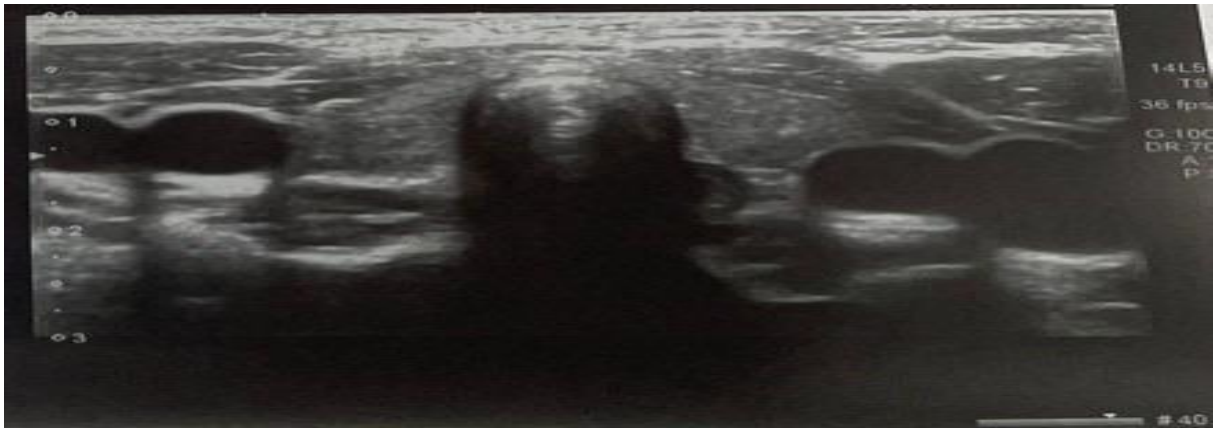

Figure 3: Ultrasound image of the thyroid gland

DISCUSSION

In the general population, the rate of occurrence of acute respiratory syndrome (PSVT) is about 0.225%, the females more than the male group, and the elderly is higher than in young people [12], [13], [14].

Regarding clinical characteristics: Paroxysmal supraventricular tachycardia (PSVT) attacks often occur in patients without structural heart disease. However, some cases can also occur in patients with organic heart disease. Taking the history, it can be seen that the patient has palpitations, and rapid heartbeat, and the appearance and end of tachycardia are quite sudden. During the attack, the patient feels palpitations, and anxiety, and the heart beats very fast. The PSVT attacks usually have little effect on hemodynamics and usually do not last long, But in a few cases, the attack can last for days and can cause low blood pressure or heart failure [9], [13], [14], [15]. Listen to the heart and the heart rate is usually very regular, with an average frequency of 180-200 cycles/minute. The PSVT attacks can end suddenly or when the patient inhales deeply and then exhales but closes the glottis tightly or when the doctor massages the carotid sinus or presses the eyeball... The electrocardiogram usually shows the image of the QRS complex usually slender, evenly, frequency of 180 - 200 cycles/minute. The P wave is not visible due to mixing with the QRS complex or can sometimes be seen as a small r wave in V1. At the end of the attack, a short period of sinus arrest or bradycardia may be seen before sinus rhythm is restored [14-18].

In terms of pathophysiology: Paroxysmal supraventricular tachycardia is a paroxysmal tachyarrhythmia. In young patients with paroxysmal tachycardia with a thin, regular QRS complex, the differential diagnosis often includes atrioventricular nodal reentry tachycardia (AVNRT) and atrioventricular reentry tachycardia (AVRT). The mechanism of both types of tachycardia mentioned above is due to re-entry. The anatomical basis for the formation of an AVNRT attack is the presence of a dual atrioventricular nodal pathway and an AVRT attack is the presence of an accessory pathway. Sometimes it is necessary to differentiate from atrial tachycardia which can also have similar manifestations on the electrocardiogram. Identifying arrhythmia with pathophysiological mechanism is important in the management and treatment of this arrhythmia, drugs that act on the atrioventricular (AV) node (the AV node is part or all of the reentry circuit) will have the ability to relieve acute respiratory syndrome [14], [16-18].

Regarding electrocardiographic characteristics in the diagnosis of atrioventricular nodal reentry tachycardia (AVNRT): Regular QRS complexes (both ST-T are the same), QRS frequency is about 150-250 cycles/minute, P waves are normal P waves may not be seen or may appear immediately after the QRS complex (P'), creating a slightly wider QRS complex. If there is the presence of a pseudo r' wave in V1 or a pseudo S wave in leads II, and III, AVF can be identified as AVNRT with very high accuracy 14], [16], [18].

The ECG characteristics in the diagnosis of Atrioventricular reentry tachycardia (AVRT): Frequency is usually fast > 180 cycles/minute, P waves tend to appear later in the cardiac cycle, RP interval is short, P wave falls in the ST segment, time and shape The state of the P wave depends on the location and conduction velocity of the accessory pathway. If the P wave is reversed in lead II and there is a relatively long distance from the preceding R wave, these are characteristics that help us diagnose AVRT 14], [16], [18].

Atrial tachycardia (AT): P wave is different from sinus P, frequency is about 150-250 cycles/minute, RP and PR intervals change because atrioventricular conduction depends on the properties of the atrioventricular node and heart rate. In atrial tachycardia, the morphology and axis of the P wave depend on the location of the pulse source and the tachycardia mechanism [17].

Results of biochemical blood tests were within normal limits, blood formula tests and infection indicators showed no signs of infection were detected. Thyroid hormones were within normal limits. Thyroid test results are normal, and safety in using the Amiodarone drugs group, in situations where at this time there is no access to Adenosine, it is decided to treat tachycardia by intravenous infusion with a syringe. The starting dose of Amiodarone bolus is completely reasonable [16], [19-21].

Treatment: Tachycardia can be stopped by vagal force testing (Tell the patient to breathe in deeply and then exhale but close the glottis, press the eyeballs, and carotid sinus massage) [22]. Often, medication must be used to stop the attacks of PSVT using intravenous Adenosine [23], [24], [25]. Can be stopped with 3 groups (Amiodarone and sotalol) or an IC group (flecainide, propafenone). In the long term, persistent attacks can be treated with beta-blockers/calcium channel blockers (Verapamil, diltiazem) [15], [18].

In the case of this clinical case report, there were no drugs available in the Adenosine group. Calcium channel blockers such as Verapamil or beta-blockers such as Propranolol or Esmolol are considered cautiously in this case because the patient is showing signs of hypotension. The vagal test was ineffective in this case, so it was decided to use Amiodarone to relieve the seizure by intravenous infusion. As a result, after the first bolus infusion dose of Amiodarone, the electrocardiogram returned to sinus rhythm with a frequency of 136 cycles/minute, which is the effective threshold in the first hour. Continue maintaining Amiodarone, an electrocardiogram of sinus rhythm at 109 cycles/minute is the safe threshold for children. Holter electrocardiogram results after 1 day did not detect a recurrence of ventricular tachycardia (VT) as well as other arrhythmias. The patient was stable and was discharged from the hospital; For these children, after being discharged from the hospital, they may not need to take medication and continue to be managed and monitored. If they relapse, they can be considered for electrophysiological testing to consider treatment to eliminate the accessory pathway using radio frequency (RF) energy. Thus, the treatment of paroxysmal supraventricular tachycardia in children with normal thyroid function is effective in the treatment of paroxysmal supraventricular tachycardia with Amiodarone and is effective and safe.

Until now, to stop paroxysmal supraventricular tachycardia, the first drug product is still Adenosine: 6mg syringe. The injection site should be in the basilic vein and when injecting, the injection must be done quickly because the half-life of the drug is extremely fast. The first time use 6 mg, if there is no result, repeat the injection with 6 mg And if there are still no results, continue using 12 mg (2 tubes) [23], [24], [25]. Some other drugs are also used to stop paroxysmal supraventricular tachycardia such as calcium channel blockers and beta blockers. can be used when Adenosine fails. People often use Verapamil intravenously, the Dosage of Verapamil is 5-10 mg intravenously over 2-3 minutes. Contraindicated in patients with impaired function. left ventricular dysfunction, hypotension, be careful in the elderly [16], [26]. Commonly used beta blockers: Propranolol or Esmolol IV injection. The dose of Propranolol is 0.15 mg/kg intravenously at a rate of 1 mg/min. Pay attention to the side effects and contraindications of these drugs. Digitalis: caution should be exercised when patients have Wolff-Parkinson-White (WPW) syndrome or intend to continue rubbing the carotid sinuses afterward, because Digitalis may increase the sensitivity of the carotid sinuses. Amiodarone is a drug that can be considered when the above measures fail [16], [26-29].

In cases where medical treatment of paroxysmal supraventricular tachycardia is ineffective, electroshock can be performed to stop the attack; Electroshock to relieve seizures: is indicated when the seizure is persistent, affects hemodynamics (causing heart failure, hypotension) or drugs cannot relieve the seizure. Usually only requires a small amount of energy (50J) and synchronization to be effective. can stop the attack [30].

In the long term, consider radical treatment with cardiac electrophysiological investigation and radiofrequency energy therapy that can be applied at facilities with Intensive Care Unit (ICU) - Interventional Cardiology. Thanks to the electrophysiological exploration method to detect secondary transmission pathways and thereby using high-frequency radio waves to destroy (burn) the secondary transmission pathways, the disease can be completely cured. This is the first choice method for patients with recurrent PSVT attacks that do not respond to conventional medical treatments. For all

patients diagnosed with PSVT attacks, they should be sent to centers with electrophysiological testing, to consider thoroughly treating the patients with PSVT attacks [16], [31-33].
Drugs used to prevent reentry at the AV node are beta blockers, Digitalis, or Verapamil... Newborns or young children need long-term treatment with drugs. However, the use of these drugs is In the long term, attention must be paid to their side effects, while older children who have had controlled PSVT without WPW syndrome appear to have less recurrence and can be monitored and managed with the option of not need medication [11], [16], [29], [34].

CONCLUSION

We have reported a clinical case of a child with paroxysmal supraventricular tachycardia Who was successfully treated with intravenous bolus injection, which is the treatment of choice to abort paroxysmal tachycardia. Supraventricular does not respond to vagal testing as well as when first-line drugs such as Adenosine are not available... In cases of drug resistance, ablation of the accessory pathway through radiofrequency catheters is still a method that needs to be considered for treatment, even for children.

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