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Incessant Supraventricular Tachycardia in a Newborn Treated with Combined Antiarrhythmic Drug Therapy

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Abstract

Supraventricular tachycardia is the most common type of tachyarrhythmia seen in childhood. First-line treatments (adenosine, esmolol-propranolol, digoxin, electrical cardioversion) usually suppress tachycardia, but rarely tachycardia is resistant and requiring Class IC and/or Class III agents, such as amiodarone, sotalol, propafenone, and flecainide.

Here, a case of a newborn with an incessant re-entrant supraventricular tachycardia and congestive heart failure, which stable sinus rhythm was finally achieved by amiodarone, esmolol, digoxin and flecainide combination without adverse events, was reported.

Keywords: Heart Failure; Incessant Supraventricular Tachycardia; Newborn

Introduction

Supraventricular tachycardia (SVT) is the most common type of tachyarrhythmia seen in childhood (90%) 1. Atrial flutter, atrial ectopic tachycardia, permanent junctional re-entry tachycardia can be seen, but atrioventricular re-entry tachycardia is the most in newborns and infants. Supraventricular tachycardia is generally well-tolerated but may be accompanied by symptoms of congestive heart failure. Symptoms may vary according to age, heart rate and duration of the tachycardia. Carotid artery massage or applying ice to the face can be tried as a first physical intervention. First-line medical treatments (adenosine, esmolol-propranolol, digoxin, electrical cardioversion) usually suppress the SVT 2. Rarely, SVT may be incessant and refractory to the first-line treatments. Therefore, Class IC and Class III agents (amiodarone, sotalol, propafenone and flecainide) are preferred as second and third-line treatments 3. Cardiac pacing and ECMO (extracorporeal membrane oxygenation) can be considered as the last option when the tachycardia remained and causing heart failure.

Here, a case of a newborn with an incessant re-entry supraventricular tachycardia and congestive heart failure, which was resistant to several anti-arrhythmic drugs in which a stable control of the heart rhythm was finally achieved with the combination of amiodarone, esmolol, digoxin and flecainide, was reported.

Case

A 22-day, 4250 gr male patient was referred to the neonatal intensive care unit due to tachycardia at a rate of 270/min. There were feeding difficulties, weakness and wheezing that started three days ago and increased over time. He was in poor general condition and had tachypnea, hepatomegaly and filiform pulse. Systemic blood pressure was 79/54 mmHg, oxygen saturation was 80%. He had metabolic acidosis (pH 7.16), AST 482 U/l, ALT 158 IU/l, blood urea nitrogen 86 mg/dl, creatinine 0.6 mg/dl, Na: 139 mEq/l (n: 135-145), K: 3.8 mEq/l (n: 3.5-5.1), Cl: 99 mEq/l (n: 96-106), P: 4.2 mg/dl (n: 2.4-4.5) and troponin I 621 pg/ml (n: <15). Infection markers were normal. On electrocardiogram (ECG), there was a regular, narrow QRS tachycardia at a rate of 270/min, suggesting supraventricular tachycardia (Figure 1). His chest X-ray showed an increased cardiothoracic ratio and increased vascularization in the lung (Figure 2).

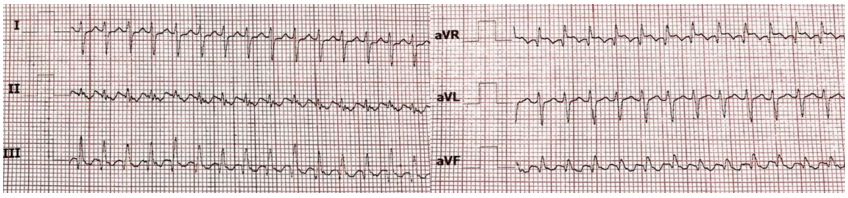

Figure 1: Electrocardiogram showing regular, narrow QRS tachycardia

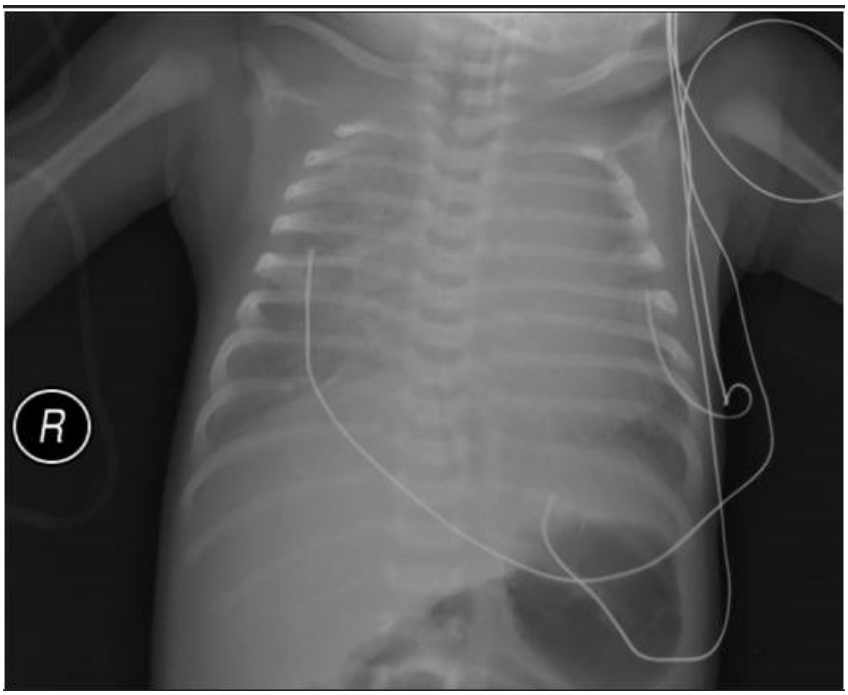

Figure 2: Chest X-ray showing an increased cardiothoracic ratio and increased vascularization in the lung

Transthoracic echocardiography (TTE) demonstrated deterioration of left ventricular systolic function (EF: 35%, FS: 19%), enlarged left ventricle and mild to moderate mitral valve regurgitation. There was no structural cardiac abnormality and there was no thrombus in the cardiac chambers. (Figure 3).

He was intubated and connected to a mechanical ventilator. Although the contraction of the heart was reduced, medical cardioversion was tried first, since the blood pressure values were within normal limits. First, adenosine (200 mcg/kg) was administered to the patient, a cardioversion was not achieved. However, it could be determined that the rhythm was re-entry atrioventricular tachycardia during the short-term blockade. Electrical cardioversion was performed three times because his general condition was poor, but the tachycardia recurred again. Since sinus rhythm could not be achieved, intravenous esmolol (gradually increased to 250 mcg/kg/min) and amiodarone (loading dose of 5 mg/kg, followed by 10 mg/kg/day) were administered. Dopamine and dobutamine (both 5 mcg/kg/min), spiranolactone and furosemide were initiated for heart failure. Giving that the SVT may have developed due to myocarditis, IVIG (2 g/kg) was administered. During the follow-up, amiodarone was increased to 15 mg/kg/min infusion dose because of the prolonged SVT episodes. Repeated administration of adenosine was required due to the recurrence of the SVT episodes. Afterwards, due to persistence of tachycardia, oral digoxin (10 mcg/kg) was administered on the 5th day and oral flecainide (2 mg/kg/day) on the 6th day of the treatment. On the 7th day, while taking amiodarone 15 mg/kg/min, esmolol 150 mcg/kg/min, digoxin and flecainide, the episodes of SVT frequencies decreased progressively. The patient was extubated on the 10th day of his admission. His SVT episodes completely disappeared on the 11th day, then esmolol and digoxin were discontinued gradually. As the left ventricular dysfunction resolved completely, inotropic and decongestive agents were gradually removed from the treatment. He was discharged in sinus rhythm with oral amiodarone (5 mg/kg/day) and oral flecainide (2 mg/kg/day).

Around six months after discharge an ECG showed a sinus rhythm with a heart rate of 130 bpm with good control of the arrhythmia. No further episodes of SVT were detected and no adverse effects were reported.

Consent was obtained from the patient's parents for this case study.

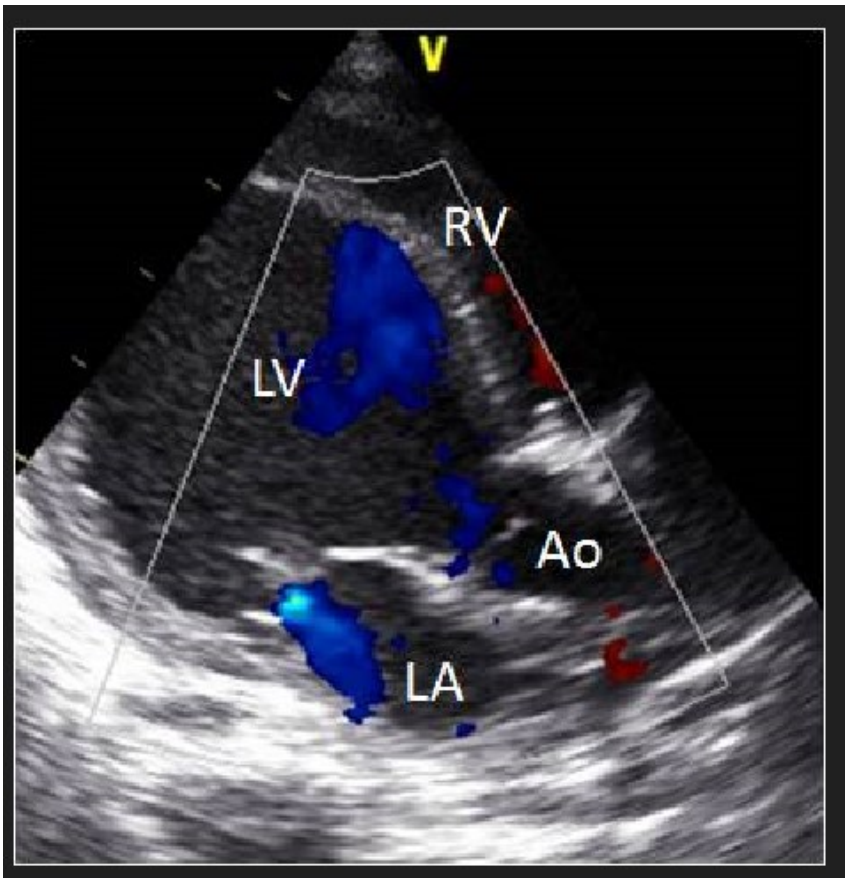

Figure 3: Transthoracic echocardiogram in parastrenal long axis view demonstrating enlarged left ventricle and mild to moderate mitral valve regurgitation, which indicates tachycardiomyopathy.

Discussion

The immaturity of the cardiac structures, including conduction tissue, facilitates the occurrence of tachycardia in newborns and infants. As a result of the maturation of the cardiac structures, SVT may disappear around the first year of age [4]. Atrioventricular re-entry is the most common type of SVT in both newborns and infants. Since other tachycardia types may also be incessant, it should be distinguished from atrial flutter, atrial ectopic tachycardia, permanent junctional re-entry tachycardia and ventricular tachycardia 5. The response of arrhythmia to the administration of an intravenous bolus of adenosine is useful to distinguish between re-entry and automaticity. Therefore, to terminate or differentiate tachycardia, we first applied adenosine. Unfortunately, sinus rhythm could not be remained and re-entry SVT recurred. Although amiodarone alone or in combination with digoxin/propranolol [6-8] and flecainide alone [9] have been reported as successful first-line treatments for SVT, adenosine, propranolol, esmolol and digoxin, alone or in combination, are recommended as first-line treatements [1]. Firstly, esmolol was administered in our case, but amiodarone and digoxin had to be added because of the tachycardia persisted. Amiodarone is a Class III antiarrhythmic agent, effective for supraventricular and ventricular tachyarrhythmias, that is often used as a second-line treatment. It causes prolonged effective refractory period of myocytes by blocking the potassium channels responsible for repolarization of the heart during the third phase of the cardiac action potential [10]. Amiodarone can cause mild or serious adverse effects, involving several organs, such as arrhythmia, hypersensitivity pneumonitis, hypothyroidism and hyperthyroidism, optic neuritis, abnormalities in liver function tests and allergic rash [11]. In our case, amiodarone was able to increase to the maximum dose without any adverse effects. Finally, flecainide was added to the treatment as long-term sinus rhythm could not be achieved with esmolol, digoxin and amiodaron combination. Flecainide is a Class IC antiarrhythmic agent that blocks fast sodium channels, thus slowing conduction throughout myocardium [12]. The use of flecainide for the treatment of the SVT in newborns and infants is still controversial due to the potentially severe proarrhythmic effects found in the results of the Cardiac Arrhythmia Suppression Trial (CAST) study

[13]. However, it reported that flecainide is well-tolerated and it provided an antiarrhythmic effect for the prevention of the SVT in newborns without structural heart disease [9]. Because of their potential and often dangerous adverse effects, the patient receiving a Class IC and/or Class III agents must be monitored very closely. If the SVT persists despite conventional pharmacologic therapy, catheter ablation can be used for definitive treatment [14]. However, [3] found that oral flecainide combined with amiodarone and esmolol-propranolol in preventing arrhythmic activity and reducing the need for ablation in critical neonates and infants. In addition, [15] reported that an incessant automatic atrial tachycardia in a newborn successfully treated with nadolol and flecainid. In our case, the incessant re-enty SVT could only be terminated with a quadruple treatment regimen consisting of esmolol, digoxin, amiodarone and flecainide, without any adverse effects.

Conclusion

Although the proarrhythmic effects of flecainide and amiodaron are known, they can be used in the incessant SVT with careful monitoring. Thrombus may occur in incessant SVT [16]. Therefore, echocardiography should be performed before the treatment to assess structural heart disease, ventricular size and function, as well as thrombus examination. Another issue that should not be forgotten is that, since incessant tachyarrhythmias may develop reversible heart failure, inotropes may be required in addition to antiarrhythmic treatment until the symptoms resolve.

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Conflicts of interest

None

Authors' Contributions

Eyup Aslan conceptualized the idea, reviewed literature, and wrote the draft; Eyup Aslan, Kazim Kucuktasci and Cem Karadeniz managed the cases, reviewed, and finalized the manuscript.

Declaration of Conflicting Interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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