

Challenges facing paroxysmal supraventricular tachycardia in early infancy

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Abstract

The heart generates its own electrical signal. The sinoatrial node is considered the natural pacemaker of the heart and generates an electrical stimulus regularly, 60 to 100 times per minute under normal conditions. Supraventricular tachycardia is the most common rhythm disturbance in children. The majority of the clinically important Supraventricular tachycardia in healthy children is caused by the presence of an accessory electrical connection between the atrium and the ventricle or within the atrioventricular node. Here is reported the case of a 3-months old infant who was presented with pallor, lethargy, poor feeding and was diagnosed with Supraventricular tachycardia. In infants it presents with nonspecific symptoms. Supraventricular tachycardia is typically not life threatening but can become if left undiagnosed and untreated, so a high index of suspicion should be maintained while evaluating a child even in early infancy.

Keywords: Heart; Rhythm; Supraventricular Tachycardia; Paroxysmal; Infant

1. Introduction

The heart is a "pump" constructed by muscles, which differ from the other types of muscles in human body, smooth and skeletal in shape, appearance, speed of contraction, timing of action and way of control. It is composed approximately by half a billion cells, most of them are located in the ventricular walls [1, 2]. This pump called heart needs electricity to function. The heart generates its own electrical signal. This electrical signal is produced by the sinoatrial (SA) node, which is a small mass of specialized tissue located in the right upper wall of the right atrium. The sinoatrial node generates an electrical stimulus regularly, 60 to 100 times per minute under normal conditions and is under the control of the autonomic nervous system.

The sinoatrial node is considered the natural pacemaker of the heart. After being generated, electricity moves through the heart, causing so the contraction of each part it passes. While all the cardiac muscle can conduct electricity, in specific areas of the heart known as "the electrical conduction system", the electric impulse travels at different speeds. The cardiac conduction system is composed by: the sinoatrial node, the atrioventricular (AV) node, the Bundle of His, Bundle branches, and the Purkinje fibers [3, 4]. The electrical impulse originates in the sinoatrial node, further it spreads across the atriums causing both of them to contract. This process is referred as atrial depolarization which results in flowing the blood into the ventricles. When the electrical cascade reaches the atrioventricular disc, it is stopped except in the atrioventricular node where it travels at a low speed towards the ventricles. This pause allows the atria to contract fully,

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emptying the blood into the ventricles. As the atria are emptied the valves between the atria and the ventricles close, so the atria begin to refill and the electrical stimulus passes through the Atrioventricular node and Bundle of His into the Bundle branches and Purkinje fibers. Consequently all the cells of the ventricles receive an electrical stimulus causing them to contract. Approximately 400 million myocardial cells that build the ventricles contract in less than one third of a second which feels as a single heartbeat. The process of ventricles contraction results in the right ventricle pumping blood to the lungs where carbon dioxide is released and oxygen is absorbed, and the left ventricle pumping blood into the aorta from where it is distributed to the whole body. The sinoatrial and the atrioventricular nodes contain a single stimulus so they need to recharge before releasing a new stimulus. The sinoatrial node recharges while the atria are refilling and the atrioventricular node recharges while the ventricles are refilling, so there is no need for pause in heart function [5, 6, 7].

If there is any disturbance in the heart electrical conduction system, it can cause abnormal heart rhythms and patterns called arrhythmias. Arrhythmias mainly happen in two ways bradycardia and tachycardia. Bradycardia are slow heart rates as a damage to the sinoatrial node, atrioventricular node or the His-Purkinje system. Tachycardia is referred as rapid heartbeats, which often happen as a result of the re-entry phenomenon. This consists of a persistent electrical loop forms, causing parts of the heart beating too quickly or out of order. Tachycardia is named ventricular tachycardia when it initiated in the ventricles, and supraventricular tachycardia when it initiates at the level of atrioventricular node or above [8, 9,10].

This case report aimed to highlight the symptoms and emphasize the importance of Paroxysmal Supraventricular tachycardia in infancy.

2. Case report

A 3-months old male was brought to the emergency by his parents as he had experienced a vomiting episode which was followed by pallor and unresponsive state about 60 minutes before. He was the first born child by an uneventful pregnancy, via vaginalis. He was breastfeeding, fully vaccinated according to age and had been previously healthy.

Table 1 Laboratory examination

WBC	13,000 cell/ml
RBC	4,190,000 cell/ml
Hemoglobin	12.3 g/dl
PLT	536,000 cell/ml
Glucose	92 mg/dL
Urea	17.0 mg/dL
Creatinine	0.37 mg/dL
ALT	55 U/L
AST	54 U/L
LDH	328 U/L
CK	119 U/L
CK-MB	3.9 ng/mL
Na	139 mmol/L
K	5.1 mmol/L
Cl	107 mmol/L

On physical examination he appeared ill, lethargic, poor-feeding and pallor. Breathing rate was mildly elevated 50 breaths in minutes with no rales in auscultation. Heart rate was significantly elevated, peripheral pulses were weak and

skin with poor perfusion. Anterior fontanel was open and soft on palpation, pupils were reactive to light, mucous was wet and no rash was observed on the skin.

Blood examination revealed: White blood cells(WBC) 13,800 cell/ml, Red blood cells(RBC) 4,190,000 cell/ml, Hemoglobin 12.3 g/dl, Platelets(PLT) 536,000 cell/ml. blood glucose level 92mg/dL, Urea 17.0 mg/dL, Creatinine 0.37mg/dL, Alanin amino transferase (ALT) 55U/L, Aspartat amino transferase(AST) 54U/L, Lactate dehydrogenase(LDH) 328U/L, Creatinine kinase(CK) 119U/L, CK-MB 3.9ng/mL, Na 139mmol/L, K 5.1mmol/L, Cl 107mmol/L (Tab.1).

Electrocardiographic examination revealed tachycardia with a frequency of 278 heart beats/minute, QRS complexes were narrow with very short PR interval and P waves at the end of QRS complexes (Fig.1).

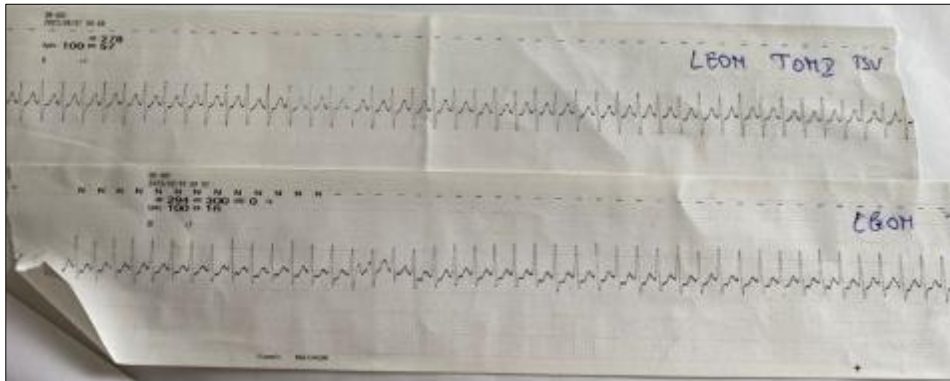


Figure 1 Electrocardiography

Radiologic examination of the thorax and ultrasound of the heart and abdomen resulted normal. Diagnosis of Supraventricular tachycardia was performed. Tachycardia was ceased soon after amiodarone administration and the child was gradually feeling well after a couple of hours.

3. Discussion

Supraventricular tachycardia (SVT) is a common condition that occurs at any age. Supraventricular tachycardia is the most common rhythm disturbance in children. It is mainly defined as a narrow, complex tachycardia that needs atrial tissue or the Atrioventricular node (AVN) as an essential part of the arrhythmia substrate. So depending on the site of origin of the dysrhythmia, Supraventricular tachycardia is classified as an atrial or AV tachyarrhythmia. The vast number of tachycardia are paroxysmal, characterized by sudden onset and termination, and only a relatively small number of them is permanent, named chronic. Paroxysmal tachycardia, in addition, can be either sustained (lasting > 30 seconds) or non-sustained whenever their duration is less. Most SVTs are due to re-entry, and only atrial tachycardia and junctional ectopic tachycardia are caused by enhanced automaticity [8, 9,11]. The majority, almost 90% of the clinically important Supraventricular tachycardia in healthy children is caused by the presence of an accessory electrical connection between the atrium and the ventricle or within the atrioventricular node. The mechanism underlying these forms of supraventricular tachycardia is known as reentry and requires the presence of two electro-physiologically distinct pathways where the electrical impulse cycles and recycles in a repetitive manner similar to a dog chasing its tail. Supraventricular tachycardia caused by concealed or Wolff-Parkinson-White accessory connection pathways predominate in childhood and adolescence, whereas the patients presented with Atrioventricular nodal reentry tachycardia tend to be older [12].

Clinical manifestations of Supraventricular tachycardia are age and duration dependent. In older children and adolescents with paroxysmal Supraventricular tachycardia heart rate is between 160-280 beats/minute, whereas in infants the heart rate is usually between 220-320 beats/minute. In older children and adolescents the most common clinical presentations are: palpitations, dizziness, shortness of breath, syncope, chest pain, fatigue, diaphoresis, nausea. In infants, symptoms are mostly nonspecific and include poor feeding, irritability, vomiting, cyanosis, and pallid spells. If the symptoms go unrecognized for hours to days, the infant can present with significant hemodynamic compromise or heart failure symptoms [13,14]. When the presenting 3-months old boy presented in emergency with compromised health conditions and changes in vital signs (tachycardia, mild tachypnea, color change), the main concern was that of a serious health condition more specific such as an imminent sepsis. A serious bacterial infection and sepsis in early

infancy is manifested with nonspecific signs such as a racing heart, rapid breathing, cool extremities, color change and compromised activity level, fever is an important sign but not consistent one. The hypermetabolic state which characterizes sepsis results in circulatory compromise, increased cardiac output, peripheral vasodilation and increased tissue oxygen consumption. However after a careful physical examination, the results of normal blood count and no alteration in biochemical panel, despite the circulatory compromise the diagnosis of sepsis was no longer the main suspicion. Findings on electrocardiographic examination permitted the accurate diagnosis of Supraventricular tachycardia. The rhythm resulted in 278 heart beats/minute, QRS complexes were narrow with very short PR interval and P waves at the end of QRS complexes.

Infants who have Supraventricular tachycardia less than 24 hours rarely develop signs of congestive heart failure, however this risk is proportionally linked to the duration of the disorder so congestive heart failure is present in 19% of infants who have Supraventricular tachycardia for 24-36 hours and in 50% of infants who have it for more than 48 hours. Its onset is sudden, as a light switch turned on, but the offset is less dramatic because of the elevated level of catecholamine which results in sinus tachycardia at the termination of Supraventricular tachycardia, bringing about a gradual slowing of the heart rhythm.

Supraventricular tachycardia is not a rare condition in children, although the exact incidence is not known it is estimated to be 1 in 250 to 1 in 1000 children. About half of children suffering Supraventricular tachycardia develop the first episode in the first year of life. After infancy, the incidence peaks in early childhood (6-9 years) and in adolescence. Spontaneous resolution occurs in more than 90% of infants when they complete the first year of life. Although about one-third may have recurrence at a mean age of 8 years. In contrast to infants only a small proportion of children who develop the disorder after the first year of life may have spontaneous resolution. Most children with Supraventricular tachycardia have a structurally normal heart [15,16,17].

Acute management of paroxysmal Supraventricular tachycardia includes controlling the rate and preventing hemodynamic collapse. Antiarrhythmic medications intend to slow conduction, especially within one limb of the reentrant circuit, as a result terminating tachycardia when the circulating wave front encounters refractory tissue. Nearly all classes of antiarrhythmic agents are used to treat Supraventricular tachycardia successfully [18,19].

4. Conclusion

Paroxysmal Supraventricular tachycardia is the most common dysrhythmia in children, including early infancy. In infants it presents with nonspecific symptoms including poor feeding, irritability, vomiting, cyanosis or pallor which characterize other serious childhood conditions too. Supraventricular tachycardia is typically not life threatening but can become if left undiagnosed and untreated, so a high index of suspicion should be maintained while evaluating a child even in early infancy.

Compliance with ethical standards

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Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed Consent was taken from the parents of the hospitalized child, reported in the study, for using the data of the medical records, providing anonymity.

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