Pediatric PSVT Presenting with Blindness and Paresthesia

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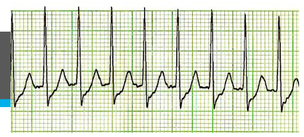
Background



Narrow complex tachycardia, particularly supraventricular tachycardia (SVT), is a prevalent pediatric cardiac rhythm pathology estimated to occur in 0.1-0.4% of children. A heart rate of 220-320 beats/minute has been recorded in infants with paroxysmal SVT (PSVT), and 160-280 beats/minute in older children. Symptoms of poor appetite, fatigue, pallor, cyanosis, irritability, dizziness, emesis, and chest pains, as well as chest fluttering/palpitations have been exhibited by children with SVT, with eventual self-resolution in seconds or days. SVTs originate from heart tissue at or above the Bundle of His.

Case Report

A ten-year-old male with past medical history of congenital umbilical hernia, abdominal migraines, cyclical vomiting syndrome, and recurrent otitis media, was accompanied by his mother to the emergency department (ED). Per mother, patient had a two-day history of four 15-20 second episodes of heart palpitations associated with headaches, numbness, tingling, and loss of sensation of lower extremities, and vision blacking out bilaterally, which self-resolved as the heart symptoms self-obliterated. The patient also complained of loss of appetite and fatigue in the past two days, which deviate immensely from his baseline norm. In the ED, a CT scan of brain without contrast did not show any hemorrhage or mass effects. Workup included a urine drug screen, urinalysis, CBC with differential, CMP, Protime INR, CK, and Troponins, which all demonstrated unremarkable results. While in the ED, patient had one episode of SVT that was captured on his rhythm strip showing heart rate in the 220s, partially reproducing his symptoms, which abruptly resolved after 20 seconds. Patient was discharged on a prescription of atenolol and was referred to a pediatric cardiologist, who proceeded to order a pediatric echocardiogram which showed a structurally intact heart with good ventricular function, albeit a small patent foramen ovale (PFO) with left to right shunt and atria of normal sizes. Since discharge, recurrence of symptoms has transpired. The pediatric cardiologist suggested the most likely cause of the SVT to be a concealed accessory pathway, possibly within the PFO, and recommended management with vagal maneuver, antiarrhythmic therapy, and consideration of catheter ablation to achieve freedom from pharmacologic dependence for symptomatic control.

Pathophysiology

Supraventricular tachycardia (SVT), also called paroxysmal supraventricular tachycardia (PSVT), is defined as an abnormally fast heartbeat that begins and ends spontaneously and originates in heart tissue other than the ventricles. It is a broad term that includes many forms of heart arrhythmias that originate above the ventricles. Supraventricular tachycardias are divided into re-entrant tachycardias and automatic tachycardias[1]. Re-entrant tachycardias occur when the electrical signal is conducted more than once in a closed circuit, often having two pathways. Re-entrant tachycardias can be further divided into atrioventricular reciprocating tachycardia (AVRT) where the re-entry is caused by an accessory pathway, or atrioventricular nodal re-entrant tachyardia (AVNRT), where the re-entry is occurring within the AV Node itself[2]. In the pediatric age range, the accessory AV pathways are the most common cause of SVT, making up 80% of cases[1].

Automatic tachycardia arise from abnormal automaticity. Normally, automaticity is a property of the sinus node and other conduction tissues. It allows for the slow spontaneous depolarization of cells until a threshold is reached allowing for an action potential to occur. In certain conditions, often when there is damage to the myocardium, some heart cells may acquire automaticity[1]. If their intrinsic rate is higher than that of the sinus node, they begin to set pace for the rest of the heart. One such tachycardia that falls into this category is the atrial ectopic tachycardia. In this tachycardia, an ectopic site in the atria gains automaticity and acts as the pacemaker. In this condition, heart rates of 250-300 can be achieved in pediatric patients, though it is relatively rare in children, making up only 5-10% of all SVTs[1].

References

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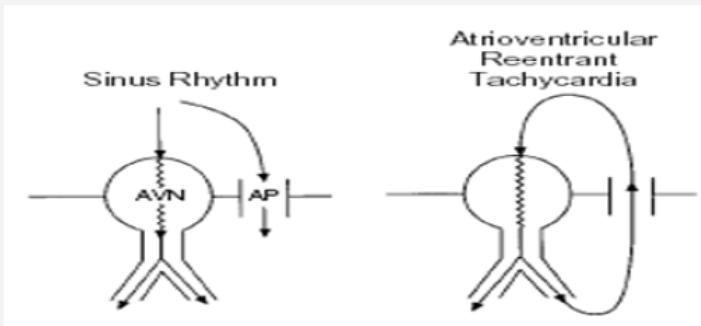

Figure 1: Mechanism of Atrioventricular Reentrant Tachyarrhythmia.

This figure shows the mechanism of a re-entrant tachyarrhythmia through an accessory pathway (AP), the most common form of pediatric SVT.[3]

Discussion

The concomitant complete vision loss and onset of neurological symptoms that superimposed on the cardiac rhythm disturbance have been unprecedented. SVT originating from foci in the inter-atrial septum and anterior atrial septum have been common, whereas foci in the PFO have been a rarity in medical literature. Most supraventricular tachycardias affect structurally healthy hearts[1]. Often, most tachycardias in this age group are revealed by heart failure signs, such as poor feeding, sweating and shortness of breath[1]. The main symptom reported by school-age children is palpitations[1]. SVT, although generally a benign condition, can cause complications such as hemodynamic instability, left ventricular dysfunction, tachycardia-induced cardiomyopathy, and heart failure if not recognized and managed promptly[1].

Vagal manoeuvres are effective in patients with AV re-entrant tachycardia[2]. Adenosine is the drug of choice at all ages for tachycardias involving the atrioventricular node[2]. However, given that this patient's SVT was most likely occurring from a pathway within the PFO, guidelines become less clear. Patients with AV re-entrant PSVT can be treated effectively by class Ic drugs, such as propaphenone and flecainide[1]. Amiodarone has the greatest anti-arrhythmic effect, but should be used with caution owing to the high incidence of side effects[1]. Destroying the accessory pathways by Catheter Ablation can be a permanent and long-term solution and allow for freedom from pharmacological management[2].