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A Review on Pharmacological Management of Paroxysmal Supraventricular Tachycardia

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I. Introduction

Paroxysmal supraventricular tachycardia is episodes of rapid heart rate that start in a part of the heart above the ventricles from time to time. There are several risk factors that may provoke PSVT they include: alcohol use, caffeine use, smoking and illicit drug use. Paroxysmal supraventricular tachycardia (PSVT) is a common arrhythmia occurring with an incidence of 2.5 per 1000 adults. PSVT can occur at any age in absence of structural heart disease, but most commonly presents between ages 12 and 30. Most patients with atrioventricular nodal re-entrant tachycardia (AVNT) or atrioventricular re-entrant tachycardia (AVRT) do not have associated structural heart disease, although exceptions (e.g., Epstein's anomaly, familial pre-excitation) do exist. A 12 lead ECG during tachycardia is helpful for defining the mechanism of PSVT. In patients with self terminating episodes, 24 hours holter monitoring is the most effective way. Supraventricular tachycardia's are the most frequent cause of hospital visits with the common ones being; atrial ventricular nodal re-entrant tachycardia, atrial ventricular re-entrant tachycardia and atrial tachycardia, moreover others include atrial fibrillation and atrial flutter. The common symptoms associated with paroxysmal supraventricular tachycardia include; dizziness, chest pain, dyspnea, anxiety, fatigue, diaphoresis and palpitations.

Mechanism of PSVT

There are 3 most common mechanisms of PSVT namely: AV node re-entry accounting for 70%, AP-mediated accounting for 25% and Atrial Tachycardia 5%.[3] The AV node sits in the triangle of Koch (ostium of coronary sinus, anterior-septal leaflet commissure of TV and tendon of todaro) in the floor of the right atrium. Separate pathways, characterized by their conduction velocities as fast or slow, provide input into the AV node. If these pathways have different refractory periods, re-entry using one pathway for ante-grade and one for retro-grade conduction may occur. The p-wave position during AVNRT depends on the types of pathways used. In the most common form, slow pathway ante-grade, fast pathway retro-grade, the P-wave is either not seen or is visible in the terminal portion of the QRS. In AVRT, an extra-nodal accessory pathway connects the atrium and ventricle. Accessory pathways may exhibit both ante-grade and retro-grade conduction, or either only antegrade (rare) or retrograde (concealed pathways) conduction. When the pathway manifests antegrade conduction, a delta wave will be present on the surface ECG, and a diagnosis of Wolff-Parkinson White syndrome is made if the patient has PSVT. [2] Accessory pathways usually exhibit rapid, non-decremental conduction, but a minority of them may manifest slow, decremental conduction. The most common form, orthodromic AVRT, uses the accessory pathway as the retrograde limb, and the AV node –His as the antegrade limb, resulting in a narrow complex QRS. Fixed or functional bundle branch block, a reversal of the circuit (antidromic AVRT), or the presence of 2 accessory pathways can lead to a wide complex QRS complex during PSVT.[2]

Management of PSVT

Adenosine and the non-dihydropyridine calcium antagonists Verapamil and Diltiazem are the intravenous (IV) drugs of choice for termination of PSVT. Adenosine is an endogenous purine nucleoside that slows AV nodal conduction and results in transient AV nodal block. Conduction in rapidly conducting accessory pathways is not affected but the decremental pathways may exhibit block. Adenosine effect is typically seen 15-30 seconds after rapid peripheral infusion as a first pass effect. In adults the dosage is 2.5 to 25mg via peripheral infusion. In children the dose range is 50-250 microgram/kg. Pharmacologically, adenosine hyperpolarizes the cell by stimulating inward potassium current and temporarily inhibiting calcium migration, by doing so the pacemaker activity of the sinoatrial node, spontaneous atrial activity and conduction through the atrioventricular node are dramatically slowed or temporarily stopped. Adenosine has no effect on accessory pathways such as those seen in WPW.[4] mild side effects of Adenosine are common, they include chest pain, difficulty in breathing, facial flushing, sinus arrest or bradycardia may occur but resolve quickly, it might also cause bronchospasms.[2, 4] Adenosine is the drug of choice for PSVT, Wittwer LK et al. Found that 89% of cases

confirmed PSVT were converted by adenosine administration, indicating effectiveness of adenosine in treatment of PSVT.[5] Furthermore, Flavio Tarasouchi concluded that if adenosine fails to restore normal sinus rhythm, diltiazem should be considered.[6]

The AV node action potential is calcium channel-dependent and the non-dihydropyridine calcium channel blockers Verapamil and Diltiazem are very effective for terminating AV-node dependent PSVT. The recommended dose of Verapamil is 5mg IV over 2minutes, followed in 5 minutes to 10minutes followed in 5mg to 7.5mg dose. The recommended dosage for Diltiazem is 20mg followed, by a second dose of 25 to 35mg. PSVT termination should occur within 5 minutes of the end of the infusion, and over 90% of the patients with AV node-dependent PSVT respond. Hypotension may occur with calcium channel blockers, particularly if PSVT does not terminate. Calcium channel blockers are not recommended in infants and neonates with PSVT because of reports of cardiovascular collapse. [2] Verapamil and Diltiazem are effective in terminating PSVT and slowing ventricular response during atrial fibrillation or flutter. Verapamil and Diltiazem slow conduction through the AV-node and increase the AV nodal refractory period.[6]

Adenosine and Verapamil have shown to have equivalent efficacy in several randomized clinical trials. Most patients with PSVT can be acutely managed with either agent. To minimize the potential for adverse effects, adenosine should be selected in patients with severe hypotension or heart failure, in infants and neonates, and in those at risk for severe bradycardia. Verapamil and Diltiazem should be considered for patients with bronchospasms, and those taking Theophylline and other methylxanthines block A_1 receptor, in which Adenosine mediates its effects.[2]

Sodium current blockers are also used to convert to sinus rhythm, they decrease the sodium influx during phase 0 of rapid depolarization of fast response cardiac action potentials. This type of action potential is found in non pacemaker cells, the Cardiomyocyte's (the atrial and ventricular myocytes, purkinje tissues). Because the slope of phase 0 depends on the activation of fast sodium channels and the rapid entry of sodium ions into the cell, blocking these channels decreases the slope 0, which also leads to a decrease in the amplitude of the action potential, in so doing reducing the conduction velocity in a non-nodal, Cardiomyocyte's. Furthermore sodium channel blockers increase the Effective Refractory Period (Class IA), while others decrease the Effective Refractory Period (Class IB), or have no effect on Effective Refractory Period (Class IC), class IA moderate sodium channel blocker and increases Effective refractory period for example: Quinidine. Class IB weak sodium channel blocker and deceases effective refractory period for example: Lidocaine, Class IC strong sodium channel blocker with no effect on Effective Refractory Period for example flecainide, Flecainide is used in treatment of supraventricular tachycardia such as atrial fibrillation, maintaining sinus rhythm in patients whom structural heart disease is absent.

Beta blockers such as electrophysiological effects of Sotalol are manifested by increased sinus cycle length (slowed heart rate), decreased AV nodal conduction and increased AV nodal refractoriness. The Class III electrophysiological effects in man include prolongation of the atrial and ventricular monophasic action potentials, and effective refractory period prolongation of atrial muscle, ventricular muscle, and atrioventricular accessory pathways (where present) in both the anterograde and retrograde directions. With oral doses of 160 to 640 mg/day, the surface ECG shows dose-related mean increases of 40–100 msec in QT and 10–40 msec in QTc.[2] Intravenous beta blockers such as Metoprolol and Esmolol have been used in treatment of acute SVT but data regarding this practice is limited, a small randomized study comparing diltiazem to Esmolol for acute SVT was terminated early because of the marked superiority of Diltiazem.[7] furthermore in another study Effects of intravenous injection of 0.6 mg/kg sotalol, a beta-blocking agent with additional class III properties, were studied by means of electrophysiological techniques in 14 patients, seven with the Wolff-Parkinson-White syndrome and seven with concealed atrioventricular (AV) accessory pathways. Sotalol brought about a significant increase in the retrograde effective refractory period of the anomalous pathway, whereas changes in the antegrade effective refractory period were more variable. In five of nine patients with electrically induced reciprocating tachycardia sotalol prevented the initiation of sustained re-entry. In most cases the suppression of the circus movement was the result of the development of AV nodal block. Thus our data support the use of sotalol for the treatment of tachycardia's incorporating anomalous AV conduction pathways.

Moreover numerous trials performed over the last 20 years, although uncontrolled, have shown the racemic d, l-sotalol is effective for the acute conversion and for long-term prevention of recurrences of supraventricular tachyarrhythmias. Sotalol appeared to be moderately effective in atrial fibrillation or atrial flutter, having somewhat greater efficacy in the case of atrioventricular (AV) nodal re-entrant tachycardia due to Wolff-Parkinson-White syndrome or concealed accessory pathway. These effects may stem from the combined class II and class III electrophysiological properties of this drug. However, studies comparing d,l-sotalol to pure beta blockers in different 'models', especially postsurgical arrhythmias and Wolff-Parkinson-White syndrome, have suggested that the observed clinical benefit may be related to d, l-sotalol's class III properties. Thus, d-sotalol may be efficacious in this indications.[9]

Amiodarone is a class III, antiarrhythmic agent, and prolongs phase 3 of the cardiac action potential, the repolarization phase where there is normally decreased calcium permeability and increased potassium permeability. Amiodarone slows conduction rate and prolongs the refractory period of the SA and AV nodes. Amiodarone given intravenously for acute termination of supraventricular tachyarrhythmias is completely safe and seems effective. Amiodarone is a potent antiarrhythmic drug which can be considered for antiarrhythmic intervention in patients with PAF or PSVT. Oral treatment with Amiodarone has been shown in many studies to be useful in the termination of supraventricular tachyarrhythmias and in the maintenance of sinus rhythm, although effects are only achieved after several hours or even days. Furthermore, the other mechanism to correct the PSVT includes Catheter ablations which are performed in cardiac electrophysiology laboratories specially equipped with recorders, programmed stimulators and ablators. The procedure is typically performed using conscious sedation. Two to five multipolar electrode catheters are inserted percutaneous under local anaesthesia into femoral, brachial, subclavian, or internal jugular veins and positioned in the heart under fluoroscopic guidance. Each electrode catheter has four or more electrodes. The most distal electrode pair is usually used for pacing and the delivery of critically timed extra stimuli, while all of the electrodes are used to record electrograms from localised regions within the heart. [11]

II. Conclusion

Management of Paroxysmal supraventricular tachycardia involves pharmacological and radial frequency ablation, I explained pharmacological management with options and their mechanism of actions, as PSVT is broad it provides challenges for choice of medication for the physicians but with current evolution of cardiac electrophysiology for those with recurrent PSVT they are opted for radial frequency catheter ablation to cardiovert, this does not mean we don't use the pharmacological management, because it has its role too in management of PSVT, therefore I conclude that the understanding of the mechanism of PSVT will facilitate and ease the choice of pharmacological agent of choice to treat PSVT.

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