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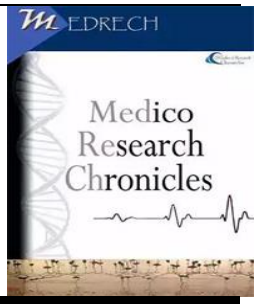

A Case of Paroxysmal Supraventricular Tachycardia Clinical and Electrocardiographic Appraisal

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ABSTRACT

Paroxysmal supraventricular tachycardia refers to a clinical syndrome characterized by rapid regular tachycardia with abrupt onset and termination which originates from or conducts through the atria or atrio-ventricular node. (1) We report 40- year- old male patient who underwent mitral valve replacement for rheumatic heart disease severe mitral stenosis with St Jude's Valve who presented with recurrent palpitations. Systematic clinical and electrocardiographic analysis aids in precise non- invasive diagnosis prior to detailed electrophysiological studies.

CASE REPORT

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INTRODUCTION:

Supraventricular tachycardia includes all tachyarrhythmias (Atrial and /or ventricular rates in excess of 100beats/min at rest) the mechanism of which involves tissues from His Bundle or above (2), which includes inappropriate sinus Tachycardia, atrial Tachycardia, (focal or multifocal and macro reentrant), Atrio ventricular nodal reentrant tachycardia and atrio ventricular (AV) reciprocating tachycardia utilizing an accessory AV connection.

Atrio ventricular nodal reentrant tachycardia (AVNRT) is the most common type, in which the micro reentry circuit is located within the AV node. (3) It can be typical slow fast tachycardia where impulse proceeds down the slow pathway and goes up through the fast pathway or atypical fast slow tachycardia where impulse goes down the fast pathway and proceeds up in the slow pathway. ECG features include no visible P waves in 48% "pseudo-S" in inferior leads or "pseudo r" waves in V1 in 46%," Pseudo q" waves in 2 % and a long RP tachycardia in 4% (fast-slow variety). RP/PR<1 and RP is usually less than 70msecs in typical variety. The P wave axis in frontal plane is caudo-cranial in all the varieties (15).

Atrio ventricular reentrant tachycardia (AVRT) is the next common form which utilizes a bypass tract across the AV ring between atria and the ventricles (1). ECG features favoring AVRT include discrete P

wave following QRS with RP/PR <1 in 90% and >1 in 10% (associated with slowly conducting bypass tract), QRS alternans, ST depression more than 2mm, pre-excitation pattern during sinus rhythm, termination by single ventricular premature beat (14) and slowing in heart rate when bundle branch develops. However, P wave axis depends on the atrial insertion of the bypass tract.

Atrial Tachycardia is the next common variety which originates in the atria and does not require AV node for its maintenance. Atrial Tachycardia can be focal or macro reentry type (also termed as atrial Flutter).

Focal Atrial Tachycardia (FAT) is characterized by regular atrial activity discrete P waves different from sinus P wave with isoelectric segments between the P waves. It can display warm up at onset and have cool down at its termination. Mechanisms can be due to enhanced automaticity, triggered activity or reentry. There can be cycle length variability. P wave precedes the QRS and generally RP interval is prolonged than PR interval. However, RP interval can also be less the PR interval in the presence of pre-existing first degree AV block (4). Atrial rate is usually 160-220 beats /min. The degree of A.V. Block can be variable. Atrial Tachycardia can be unifocal or multifocal conducted normally or with phasic aberrant ventricular conduction. It can vary in duration from few seconds to many hours and may occur repetitively. P wave axis depends on site of origin.

Macro reentrant Atrial tachycardia (MAT) or atrial flutter has been divided into common form with rate of 220-350/min with cranio-caudal activation an uncommon form with rate of 240-300/min with caudo- cranial activation. Type I corresponds to common form. In type II the atrial flutter rate is 340-430/min (13). The classical saw-toothed pattern in ECG is the hallmark for atrial flutter and carotid sinus massage induces AV block without termination (14)

Supra ventricular tachycardia should be clinically and electrocardiographically analyzed for P wave morphology, location, PR/RP ratio, axis in both frontal and horizontal planes, atrial rate and relationship between atrial and ventricular rhythm, any alternation of QRS complex, presence of preexcitation in resting ECG, response to carotid sinus massage, mode of initiation and termination and assessment of jugular venous pulsation during tachycardia (6), (7), (8).

We report a 40-year-old Male patient who has undergone St Jude's mechanical mitral valve replacement a month back with history of recurrent palpitations.

CASE REPORT:

A 40-year-old male patient presented with intermittent palpitations sudden onset and offset for past 2 weeks. There was no history of syncope, breathlessness or features of congestive heart failure. There was no chest discomfort. After surgery for mitral valve, he was regularly taking anticoagulant medications. On examination pulse rate was 100/min regular in rhythm felt in all peripheral arteries. His jugular venous pressure was normal. On auscultation valve clicks well heard second sound was normal and no murmurs. He was serially followed clinically and electrocardiographically.

ECG on 29/10/2024,10:28AM revealed HR 115 /min. Atrial rate equals Ventricular rate, narrow QRS tachycardia PR 0.28secs suggestive of sinus tachycardia with First degree heart block. (Fig 1).

Fig 1 Sinus Rhythm with 1st degree heart Block

ECG on 29/10/2024; 10:58AM one episode of paroxysmal tachycardia revealed narrow QRS, with discrete P waves (Fig 2).

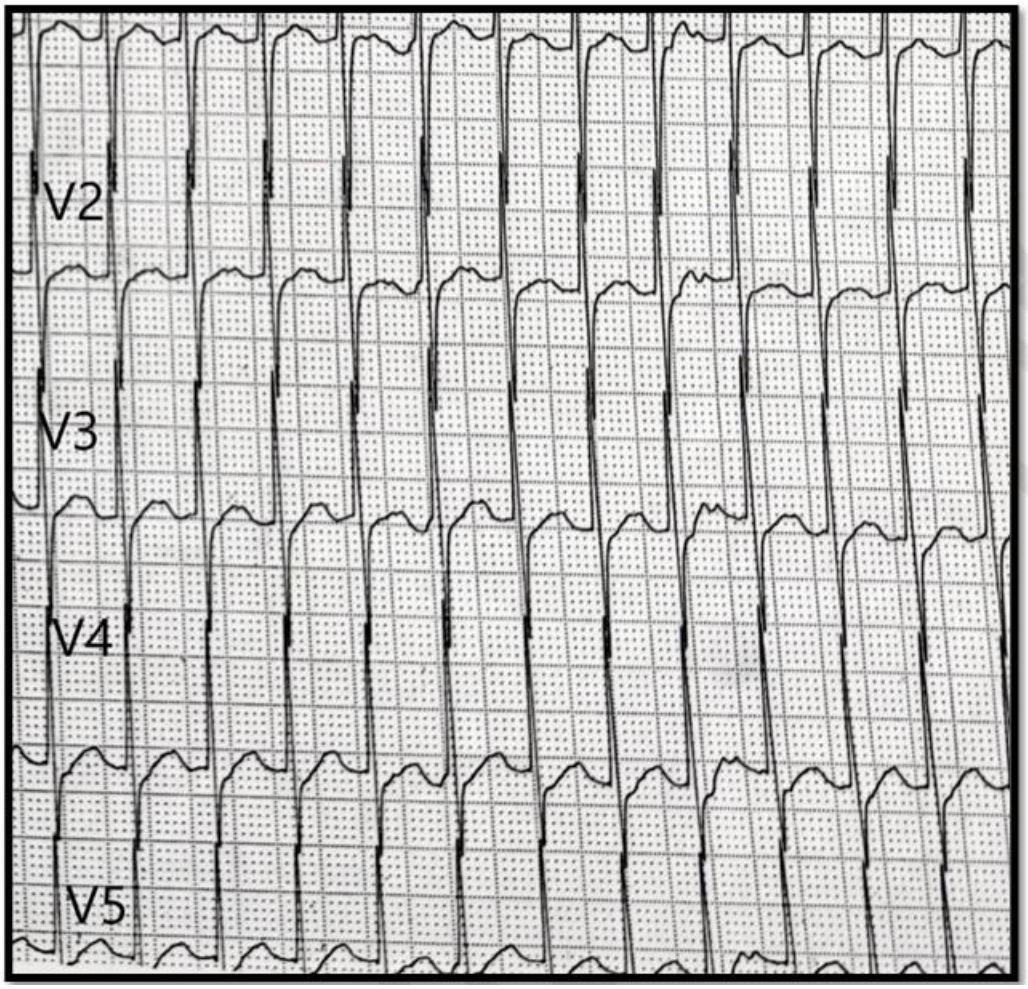

Fig 2: V2 to V5 leads shown Narrow QRS tachycardia with discrete P Waves preceding the QRS as tracing taken on 29/10/2024

Normal sinus rhythm with 1st degree heart block was noted on tracings 29/10/2024 at 09:27AM,10:28AM and 12:47PM, on 30/10/24 at 00:10AM, 1/11/24 at 13:51PM and on 05/11/2024 at 09:57AM. The paroxysmal tachycardia was noted on 29/10/2024 at 10:58AM on 30/10/2024 at 10:57AM and on 01/11/2024 at 12:36PM. On 30/10/2024 at 10:57AM, one paroxysm ended spontaneously with a pause of 0.64 secs which ended in a

ventricular escape beat. During Tachycardia regular canon waves were observed synchronous with pulse and heart rate. Carotid sinus massage immediately terminated the paroxysmal tachycardia on 01/11/2024 and subsequent ECG showed sinus rhythm with 1st degree heart block with PR interval of 0.28 secs.

One episode of paroxysmal tachycardia was recorded lasting for about 8 secs on 6/11/2024 at 9:48AM. The sinus cycle measured 0.68 secs. The Tachycardia began with an atrial premature beat with a coupling interval of 0.32 secs. The Atrial deflection which preceded the QRS during Tachycardia was similar throughout and had a different morphology from the sinus P wave. The atrial cycle started with 0.68 secs gradually accelerated to 0.40 to 0.48 secs with 1:1 atrio Ventricular relationship. The RP interval measured 0.2 secs and PR interval measured 0.24 secs. The atrial deflection was positive in inferior Leads and V4 to V6 leads. It was negative in AVR, AVL, V2, biphasic in V1 and isoelectric in lead 1 suggestive of superior-inferior frontal plane P wave axis with right-left horizontal plane P axis (Fig 3). There was no QRS alternans. QRS duration was 0.80 secs. There was progressive prolongation of PR interval from 0.16 to 0.20 secs noted in the last two beats at the end of paroxysm with dropped beat ended with a pause of 1.04 secs with a sinus escape beat which had shorter PR interval of 0.12 secs (fig4). The second sinus beat after the pause had a PR interval 0.16 secs. From the third beat onwards the PR interval remained at 0.28 secs. The sinus beats after the pause gradually accelerated from 0.76 secs to 0.68 secs. At 9:48.38AM, 9:48.45AM, 9:49.23AM and 9:49.31AM, PR interval during sinus rhythm measured 0.12secs with RR interval of 0.68 secs. At other time intervals first degree heart block was noted with PR interval of 0.28 secs.

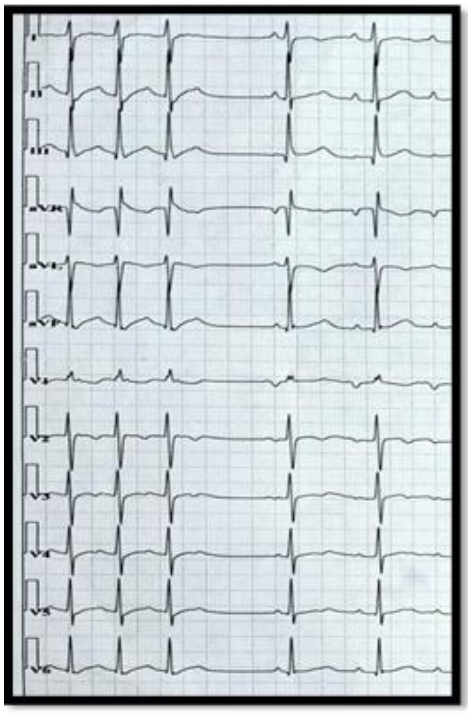

Fig 3 shows superior-inferior frontal plane and right -left horizontal plane P axis

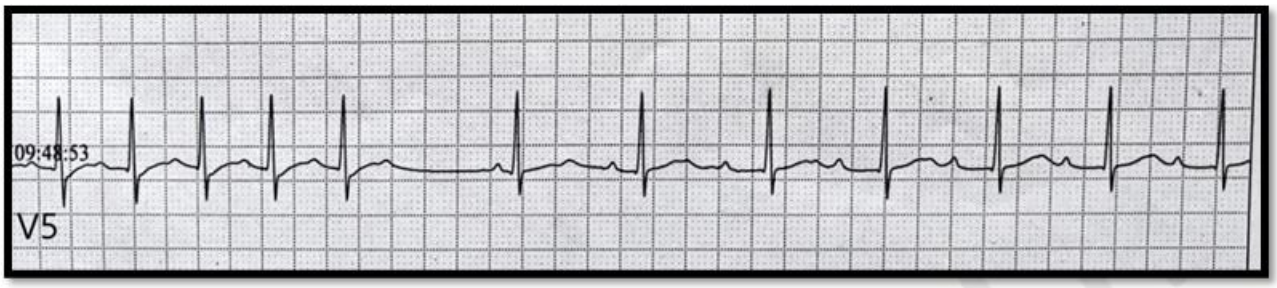

Fig 4. Atrial Tachycardia with Wenckebach periods: prolongation of PR interval in the last two beats at the end of paroxysm from 0.16secs to 0.20 secs with dropped beat and escapes sinus beat with PR interval 0.12secs

Echocardiography revealed normally functioning mechanical mitral valve with Normal LV and RV function Blood test; serum electrolytes, thyroid function test normal and Hb 10.4gms/dl

DISCUSSION:

Paroxysmal supra ventricular Tachycardia can be a sinus Tachycardia, sinus node reentry tachycardia, Atrial Tachycardia, AV nodal reentrant Tachycardia or Atrio Ventricular (AV) reciprocating Tachycardia utilizing an accessory AV connection. The abrupt onset and offset with abrupt termination of tachycardia spontaneously and after carotid sinus massage is less likely in a sinus Tachycardia. Furthermore, in inappropriate sinus Tachycardia the P wave during tachycardia resembles sinus P wave (2). In our case study P waves during tachycardia were different from the sinus P waves.

Discrete P waves separate from the QRS complex was found to be predictive of either AV reciprocating Tachycardia or Atrial Tachycardia (12). The discrete P waves preceding the QRS which are seen positive in inferior leads in general is less likely in reentrant junctional Tachycardias which includes AV nodal and many types of AV bypass reentry. However, the polarity of P wave in AVRT depends on the location of atrial insertion of the bypass tract.

In AVNRT typical or atypical or with block, P waves are usually buried within the QRS. If the P wave emerges as pseudo-S waves in inferior leads, a mid-line caudo-cranial activation with inverted P waves in inferior leads, isoelectric P wave in lead I and equal upright P waves in AVR and AVL is described (15). Similar pattern is observed in AVRT with postero septal bypass conduction (15).

However, in AVRT utilizing left sided accessory pathway during tachycardia, P wave in lead I is frequently negative. P waves are inverted in inferior leads if left accessory pathways are septal or posterior. If the accessory pathway is left lateral P wave although negative in lead I, it can be positive in Lead II and III. P wave in V1 is frequently positive when left by pass is utilized (13).

In AVRT using right sided bypass tract P wave is positive in Lead I and II and morphology in Lead III and AVF is variable. P wave in AVL and V6 are positive, isoelectric in AVR and Negative or biphasic in V1 (15). However, in our patient during paroxysm, there is a superior-inferior frontal P wave axis and AVR and AVL were negative which is different from the typical patterns described for AVRT and AVNRT. A caudo-cranial activation of frontal plane P axis could however occur in an atrial tachycardia or in an uncommon form of atrial flutter.

Atrial flutter can closely resemble FAT. The ECG of MAT may resemble FAT when there are areas of very slow conduction in the re-entry circuit wherein, they generate very low amplitude potentials not detected on surface ECG. Furthermore, FAT can produce continuous oscillation pattern if the cycle

length is sufficiently short to approach duration of atrial depolarization (25). Evans has mentioned the difficulty on clinical grounds to separate atrial tachycardia from atrial flutter and stipulated that atrial flutter is favored if the attack is prolonged (10). Atrial Flutter which are macro reentrant Tachycardias generally do not occur in spontaneous bursts (2) and carotid sinus massage increases the AV block without terminating the Tachycardia (13). After exclusion of atrial flutter superior to inferior P wave axis and a right to left P wave axis in horizontal plane as noted in our patient is mentioned very suggestive of atrial tachycardia (6).

Furthermore, Wenckebach block noted in our study is less likely to occur in AVRT (22). The differential diagnosis of SVT with Wenckebach block includes AVNRT with lower common pathway Wenckebach, atrial tachycardia or junctional tachycardia (22). Wenckebach phenomenon in atrial tachycardia has also been described by Vincenzo Carbone et al, wherein, they have described the beat following the pause could be pseudo escape beats due to slow pathway conduction of a supra-ventricular impulse (23). John Parkinson et al in their case series of paroxysmal atrial tachycardia have described progressive prolongation of PR with dropped beat (Wenckebach periods). They also noted slight prolongation of PR interval at the end of paroxysm (9) as noted in our case study. Harry G Mond et al have also reported Wenckebach AV sequences in atrial tachycardia. They have also described non-conducted focal atrial tachycardia which demonstrated a Wenckebach block at the ectopic -myocardial junction in the atrium (24).

Regular canon waves during SVT are noted to occur during AV nodal reentrant tachycardias, orthodromic tachycardia ectopic junctional tachycardia, sinus tachycardia and ectopic atrial tachycardia with first degree AV block (18). First degree heart block noted in the sinus rhythm could account for short RP compared to PR interval during Tachycardia.

Carotid sinus massage on paroxysmal Atrial Tachycardia can either cause cessation of Tachycardia or have no effect (7). Henkens et al have mentioned abnormal automaticity or micro-reentry related focal atrial tachycardia is generally unresponsive to vagal maneuvers. However, triggered activity related atrial tachycardia may be terminated by carotid sinus massage (20). The variation in the PR interval during sinus rhythm at 9:48 and 9:49 AM could be due to variable autonomic influence on AV conduction.

In conclusion, the electrocardiographic features of our patient presenting as paroxysmal narrow QRS tachycardia with a discrete similar P wave preceding the QRS during tachycardia which has different morphology than the sinus P wave with superior to inferior frontal P axis and right to left horizontal P wave axis with intermittent Wenckebach is suggestive of atrial tachycardia. Even though it has been mentioned electrocardiography can identify 80% of AVNRT and AVRT but incorrectly categorize 20% of paroxysmal supraventricular tachycardias and unable to differentiate atrial tachycardias from the other two types (8), a systematic analysis of clinical and ECG features is definitely helpful when planning an electrophysiological test which is conclusive to precisely diagnose the exact tachycardia mechanism.

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