Pre-Excitation, Mahaim-Type and brief chronology of pre-excitation - 2008

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Pre-Excitation, Mahaim-Type: It is a rare variant of ventricular pre-excitation form of ventricular pre-excitation characterized by a normal PR interval and a long QRS interval with an initial slow deflection (delta wave). In this syndrome, the atrial impulse travel to the ventricle via the Mahaim fibers which connect ATRIOVENTRICULAR NODE directly to the right ventricle wall (nodoventricular pathway) or to the RIGHT BUNDLE BRANCH OF HIS (nodofascicular pathway).

Mahaim fiber are accessory pathway going directly form the atrioventicular (AV) node to either the ventricular myocardium or one of he conduction fascicles, bypassing the His bundle. There may be a small or absent delta wave and a very short PR interval. This anatomical abnormality predisposes a patient to reentrant arrhythmias.

Mahaim fibre is commonly located between 8 and 10 o'clock at tricuspid annulus at right atrial freewall of tricuspid annulus (1) or right free wall atriofasicular (Mahaim type) accessory pathway.

While usually located along the tricuspid annulus, left-sided Mahaim fibres have been occasionally reported (2).

True nodoventricular (NV) accessory connections, as originally described by Mahaim, are rare entities, with the majority of previously reported cases now recognized as being due to decremental atriofascicular pathways. Radiofrequency ablation at this site eliminate inducibility of both tachycardias and any evidence of antegrade or retrograde accessory pathway conduction. M potential guides to successful RF ablation in most patients. Mahaim junctional acceleration is commonly seen during RF ablation guided by M potential map. Accessory pathways with slow and anterograde decremental conduction (Mahaim fibres) are responsible for a minority of atrioventricular reentrant tachycardias. The atrial origin of accessory

connections with Mahaim-type preexcitation is apparently confined to the anterolateral-to-posterolateral region of the tricuspid annulus. Mechanical conduction block in the atrial input to the accessory fiber induced at the subannular level by catheter manipulation provides an optimal marker to locate the ablation site, even during atrial fibrillation. To expose early recurrence of antegrade accessory pathway conduction, intermittent atrial pacing in the 12 hours after ablation is advisable; in cases of recurrence, a repeat procedure can readily be performed using just the ablation catheter advanced to the target site at the tricuspid annulus.

CHRONOLOGY OF PRE-EXCITATION HISTORY: MAIN LITERATURE CONTRIBUTIONS

Pre-excitation syndrome has a long and interesting history. This ECG abnormality was originally classified as bundle branch block.

1893 **Kent AFS. Researches on the structure and function of the mammalian heart. J Physiol 1893; 14: 233–254.**Kent dramatically advanced the understanding of intracardiac conduction with his study of auriculo-ventricular (A-V) muscular connections. Kent proposed that there were multiple muscular links, which crossed the A-V groove. Specifically, he focused attention on a muscular connection between the right auricle and right ventricle at the lateral right border of the heart

1913: **Cohn AE and Fraser FR. Paroxysmal tachycardia and the effect of stimulation of the vagus nerve by pressure. Heart 1913–14; 5: 93–105.** Cohn and Fraser published the first patient with a short P-R interval, wide QRS complexes, and paroxysmal tachycardia. These authors presented two patients with paroxysmal tachycardia, terminated by vagal stimulation. In the first patient, the resting ECG showed right bundle branch block (RBBB) and the second patient's ECG revealed a slurring of the initial portion of the QRS complex.

1915: **Wilson FN. A case in which the vagus influenced the form of the ventricular complex of the electrocardiogram. Arch Intern Med 1915; 16: 1008–1027.** The first isolated cases reported . Patient with pre-excitation and episodes of supraventricular tachycardia, which were terminated by the Valsalva manoeuver. This patient also had spontaneous and atropine induced AV junctional rhythm.

1921: **Wedd AM. Paroxysmal tachycardia with reference to nomotopic tachycardia and the role of the extrinsic cardiac**

nerves. Arch Intern Med 1921; 27: 571–590. Reported an additional case of pre-excitation which was classified as bundle branch block.

1925 Lewis T. The mechanism and graphic registration of the heart beat 1925; London Shaw and Sons Ltd pp. 13. Sir Thomas Lewis, in 1925 did not find adequate anatomical or physiological evidence to support Kent hypothesis questioned the role of "Kent's bundle" in A-V conduction.

1926 Curtis CW and Hamilton CK. Electrocardiographic changes in rheumatic fever. Lancet 1926; 807–809. Reported an additional cases of pre-excitation syndrome classified as bundle branch block.

1930: Wolff L, Parkinson J, White PD. Bundle-branch block with short P-R interval in healthy young people prone to paroxysmal tachycardia. Am Heart J 1930; 5: 685–704. Wolff and coworkers reported 11 mostly young, healthy subjects with a short P-R interval, wide QRS complexes, and paroxysms of tachycardia. This pattern, which later came to be known as the Wolff–Parkinson–White (WPW) syndrome, was the first characterized pre-excitation syndrome.

1932 Holzmann M and Scherf D. Uber Elektrokardiogramme mit verkurzter Vorhof-Kammerdiztanz und positiven P-Zacken. Z Klin Med 1932; 121: 404–423. These authors suggested bypass tracts as the most likely mechanism of pre-excitation syndrome. in addition to their pioneering theory on the mechanism of pre-excitation, also discussed the possibility of an "excitable centre". A properly timed atrial contraction could initiate a ventricular impulse from an excitable ventricular centre, thereby causing a short P-R interval and a delta wave.

1933 Wolferth CC and Wood FC. The mechanism of production of short P-R interval and prolonged QRS complexes in patients with presumably undamaged hearts; hypothesis of an accessory pathway of auriculo-ventricular conduction (bundle of Kent). Am Heart J 1933; 8: 297–311. These authors introduce the notion that these Kent bundles might play a role in A-V conduction

1941: Levine SA and Beeson PB. The Wolff–Parkinson–White syndrome with paroxysms of ventricular tachycardia. Am Heart J 1941; 22: 401–409. These authors reported three patients with paroxysms of tachycardia classified by the authors as "ventricular

tachycardia". All of their patients had pre-excitation and irregular wide QRS complex tachycardia, most likely due to atrial fibrillation. The most notable patient was a 36-year-old steamfitter who "while lifting a heavy box, suddenly became conscious of a "knock" in the centre of his chest". The initial ECG showed "ventricular tachycardia," which was treated with 0.8 mg of oral digitalis. After restoration of sinus rhythm the ECG showed a short P-R interval and bundle branch block, with an infarct pattern. The patient was hospitalized for six weeks with the diagnosis of acute myocardial infarction. However, according to Levine, who saw the patient two months later, the diagnosis of myocardial infarction was erroneous. He still believed however, that the clinical syndrome could be attributed to "an attack of ventricular tachycardia". The case was recognized for its similarity to those described by Wolff, Parkinson, and White, in that the young steamfitter was free of organic heart disease, yet a ventricular origin for the arrhythmias was assumed.

1943: Wood FC, Wolferth CC, Geckeler GD. Histologic demonstration of accessory muscular connections between auricle and ventricle in a case of short P-R interval and prolonged QRS complex. Am Heart J 1943; 25: 454–462. Theses authors documented the first accessory connection at autopsy. They reported the first histological proof of muscular connections between the right auricle and the right ventricle in a human autopsy specimen. The patient was a 16-year-old boy with episodes of paroxysmal tachycardia. Three years after the initial presentation, he presented with palpitations, severe substernal distress, and a heart rate over 150 beats per minute (bpm). The patient died 2 h later after drinking cold water. Autopsy revealed a normal heart with three muscular A-V connections between the right atrium and ventricle.

1944: Ohnell RF. Pre-excitation, a cardiac abnormality. Acta Med Scand 1944; 52:Suppl. 1–167. Ohnell introduced the term pre-excitation syndrome for an electrocardiogram (ECG) manifested by a short P-R interval and a wide QRS complex, regardless of the presence or absence of tachycardia.

1944: Segers M, Lequime J, Denolin H. L'activation ventriculaire precoce de certain coeurs hyperexcitables. Etude de l'onde de

l'electrocardiogramme. Cardiologia 1944; 8: 113–167. Segers and coworkers proposed the term delta wave for the initial slurred component of the QRS complex.

1947: Mahaim I. Kent's fibers and the A-V paraspecific conduction through the upper connections of the bundle of His-Tawara. Am Heart J 1947; 33: 651–653. Mahaim postulated that if conduction by Kent's fibres is accepted (and it has still not been proved that these fibres regularly exist, and one can even doubt it), it should be regarded as an accessory form of conduction: para-specific conduction"

1961: James TN. Morphology of the human atrioventricular node with remarks pertinent to its electrophysiology. Am Heart J 1961; 62: 756–771. Documented a series of patients with pre-excitation syndromes

1966: Lev M. Anatomic consideration of anomalous AV pathways. In Dreifus LS and Likoff W (Eds.). Mechanisms and therapy of cardiac arrhythmias 1966; New York Grune & Stratton pp. 665–670. Documented a series of patients with pre-excitation syndromes.

1970: Durrer D, Schuilenburg RM, Wellens HJJ. Pre-excitation revisited. Am J Cardiol 1970; 25: 690–697. A-V bypass tracts as the mechanism of pre-excitation was not generally accepted until the 1970s. A major advance was achieved when Durrer and Roos performed epicardial excitation mapping on a patient with the WPW syndrome Mapping was performed on a 21-year-old woman during surgery for a large atrial septal defect. The right ventricle was activated earliest as a result of conduction of the atrial impulse through a connection near the right lateral atrioventricular sulcus. This finding laid the foundation for later invasive electrophysiological studies

1988: Tchou P, Lehmann MH, Jazayeri M, et al. Atriofascicular connection or a nodoventricular Mahaim fiber? Electrophysiologic elucidation of the pathway and associated reentrant circuit. Circulation 1988; 77: 837–848.) (Klein GJ, Guiraudon GM, Kerr CR, et al. Nodoventricular accessory

pathway: evidence for a distinct accessory atrioventricular pathway with atrioventricular node-like properties. J Am Coll Cardiol 1988; 11: 1035–1040.) The role of the Mahaim fibres was considered in patients with accessory pathways and decremental conduction properties. However, electrophysiological studies and surgical results in the 1980s and 1990s challenged the participation of Mahaim fibres in the pre-excitation syndrome.

References

1) Bohora S, Dora SK, Namboodiri N, Valaparambil A, Tharakan J.Electrophysiology study and radiofrequency catheter ablation of atriofascicular tracts with decremental properties (Mahaim fibre) at the tricuspid annulus. Europace. 2008;10:1428-1433.

2) Francia P, Pittalis MC, Ali H, Cappato R.Electrophysiological study and catheter ablation of a Mahaim fibre located at the mitral annulus-aorta junction.. J Interv Card Electrophysiol. 2008;23:153-157.