Successful Ablation of a Wide Complex Tachycardia with Distinct Intra-Cardiac Electrograms

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Abstract

A 13-year-old boy was hospitalized after a syncopal episode that occurred during exercise. He suddenly felt chest tightness, sweating and palpitations, followed by a transient loss of conciseness. Upon emergency medical team arrival, he was awake and oriented. Baseline ECG showed sinus rhythm at a rate of 98 bpm, with narrow QRS, and no signs of long QT, Brugada, or pre-excitation. Physical examination, blood tests, 24 hours Holter monitoring, transthoracic echocardiography and stress test were all within normal limits. Eight days later he experienced a second episode of palpitations while walking to school. ECG revealed regular wide complex tachycardia (WCT) at a rate of 200 bpm, with LBBB morphology that terminated with Adenosine (Figure 1). The clinical tachycardia was easily induced by programmed electrical stimulation (Figure 2A). Diagnostic electrophysiological maneuver (Figure 2B) was followed by successful ablation, during which a unique phenomenon was noted (Figure 3). What is the diagnosis of the tachycardia and what are the unique findings noted during and after ablation?

Successful Ablation of a Wide Complex Tachycardia with Distinct Intra-Cardiac Electrograms

Short Title: "Wide Complex Tachycardia with unusual electrograms"

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Case presentation

A 13-year-old boy was hospitalized after a syncopal episode that occurred during exercise. He suddenly felt chest tightness, sweating and palpitations, followed by a transient loss of conciseness. Upon emergency medical team arrival, he was awake and oriented. Baseline ECG showed sinus rhythm at a rate of 98 bpm, with narrow QRS, and no signs of long QT, Brugada, or pre-excitation. Physical examination, blood tests, 24 hours Holter monitoring, transthoracic echocardiography and stress test were all within normal limits. Eight days later he experienced a second episode of palpitations while walking to school. ECG revealed regular wide complex tachycardia (WCT) at a rate of 200 bpm, with LBBB morphology that terminated with Adenosine (Figure 1). The clinical tachycardia was easily induced by programmed electrical stimulation (Figure 2A). Diagnostic electrophysiological maneuver (Figure 2B) was followed by successful ablation, during which a unique phenomenon was noted (Figure 3). What is the diagnosis of the tachycardia and what are the unique findings noted during and after ablation?

Discussion

Baseline measurements revealed sinus rhythm with narrow QRS and short HV interval of 25ms. VA conduction was concentric and decremental with VA block at 290ms indicative of retrograde AV nodal conduction.

Incremental atrial pacing reproducibly induced wide complex tachycardia of LBBB morphology following AH prolongation and HV shortening (Figure 2A), suggestive of right sided decremental accessory pathway (AP).[1]

Atrial extra-stimuli administered at the timing of His refractoriness advanced the next V and the next A of the tachycardia (Figure 2B) which is consistent with antidromic tachycardia. The differential diagnosis includes "long" Mahaim atrio-fascicular AP and "short" decremental atrio-ventricular AP. Activation mapping demonstrated earliest ventricular activation at the antero-lateral aspect of the Tricuspid annulus implying of "short" decremental AP rather than the apical portion of the right ventricle as seen in atrio-fascicular accessory pathway.[2] Unexpectedly, a discrete AP potential (Mahaim-like) was noted at the antero-lateral aspect of the Tricuspid annulus, at sites of early ventricular activation during antidromic tachycardia (Figure 3A). The AP was successfully ablated via internal jugular approach at 10 o'clock position in left anterior oblique view.

Following ablation, a unique phenomenon was demonstrated with evidence of infra-AP potential block and AP-ventricular dissociation during atrial pacing without pre-excitation (Figure 3B). During Three-months follow-up the patient was free of any arrhythmias.

Accessory pathways with decremental properties connect the right atrium or the AV node with the right ventricle or the right bundle branch. Although the historical report by Mahaim refers to a nodo-ventricular accessory pathway, the term Mahaim has been adapted to describe other decremental AP's with different anatomical features, including atrio-fascicular and atrio-ventricular pathways.[1]Further classification of decremental AV pathways into "long" pathways that insert into or near the right bundle branch and "short" pathways that insert into the base of the right ventricle was later described by Haıssaguerre in 1995.[3] The latter pathways are characterized by atrial and ventricular insertion immediately contiguous to the tricuspid annulus as seen in the present case in which the distal insertion is adjacent to the Tricuspid annulus.[3]

Thus far, Mahaim potential, described as an AV nodal His like structure, has been reported in "long" atrio-fascicular and atrio-ventricular pathways [1,2], where it also facilitates successful ablation. Typically, catheter ablation of these pathways is accomplished by identifying the proximal and distal insertions and the recording of a Mahaim potential at the tricuspid annulus or on the right ventricular free wall.

In the present case, we have found that Mahaim-like potential can be also identified in short decremental accessory pathway in which the earliest ventricular activation is adjacent to the Tricuspid annulus rather than the right bundle branch or the right ventricular free wall. The location of the Mahaim-like potential was correlated with the earliest site of ventricular activation during maximal pre-excitation and assisted in localization of the AP and ultimately with successful ablation. Post ablation, a unique phenomenon of an infra-AP potential block was documented with evidence of AP-ventricular dissociation, which proves our concept that the Mahiam-like potential was indeed associated with the localization and ventricular insertion of the accessory pathway.

Figure 1

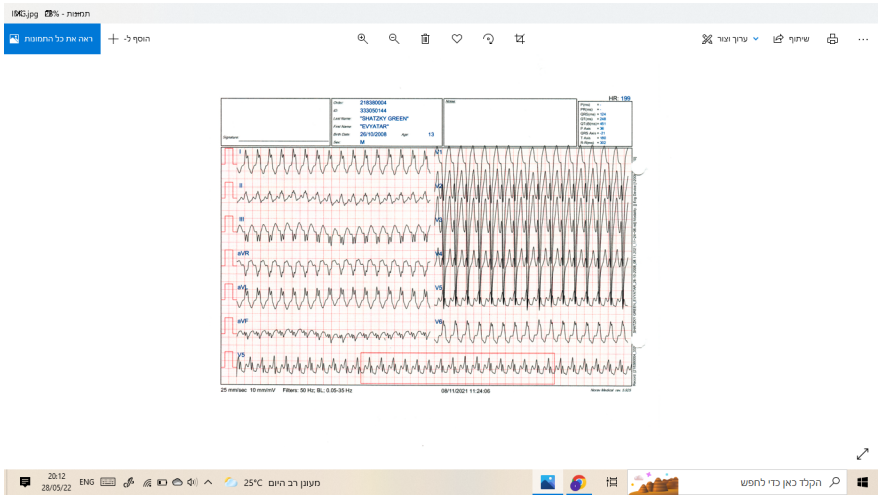
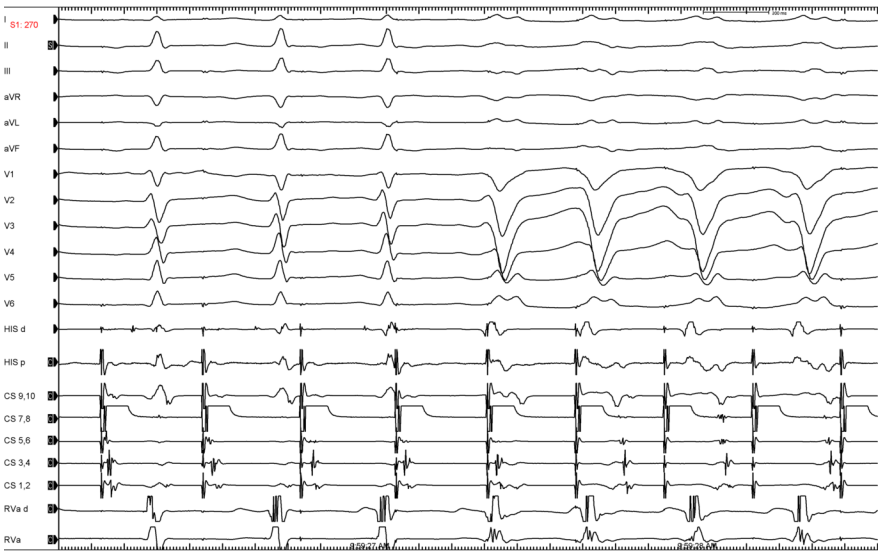


Figure 2



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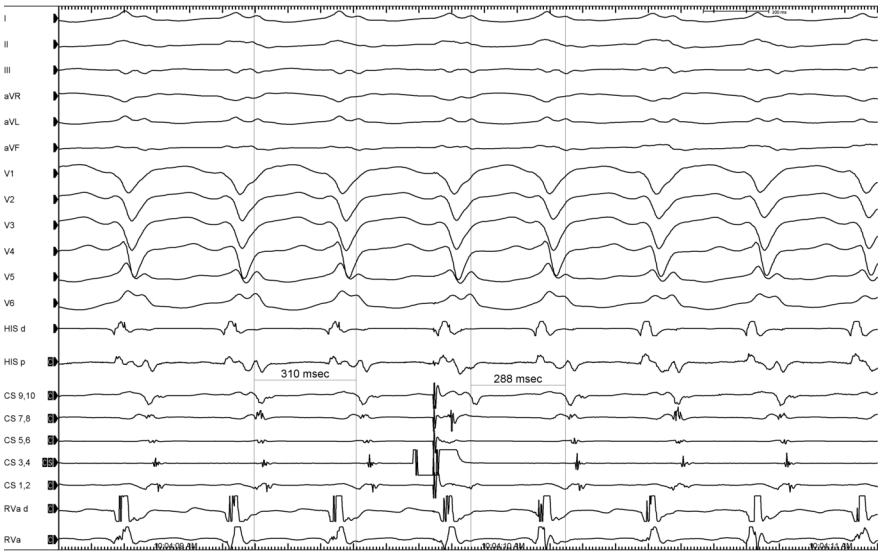


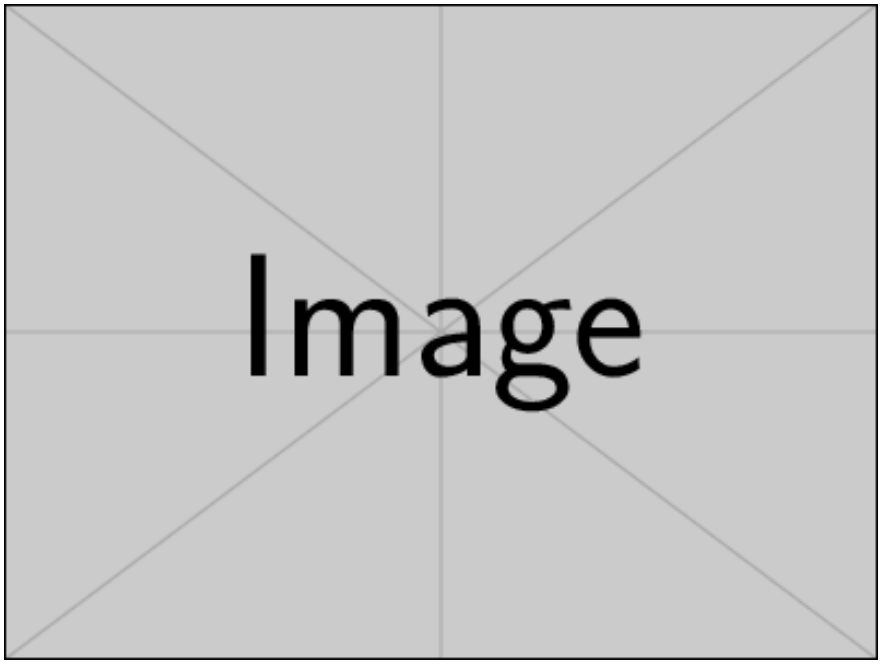


Figure 3

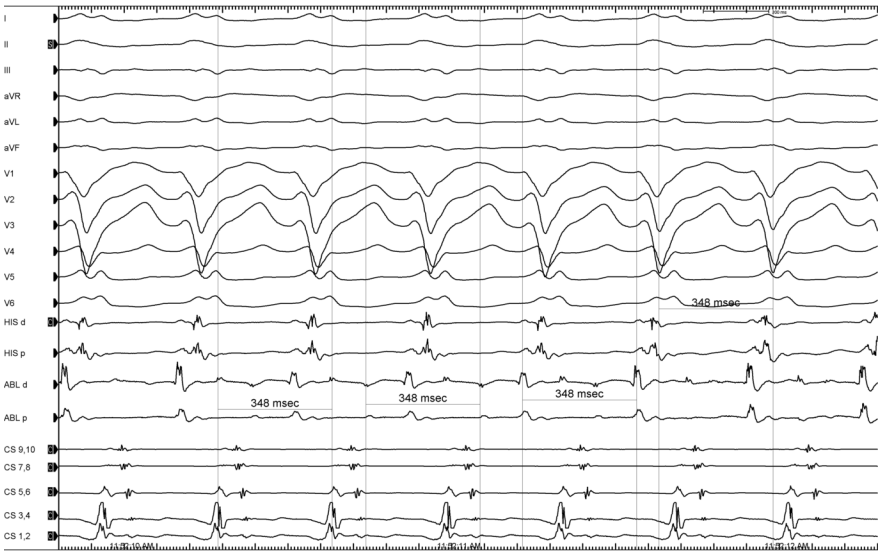


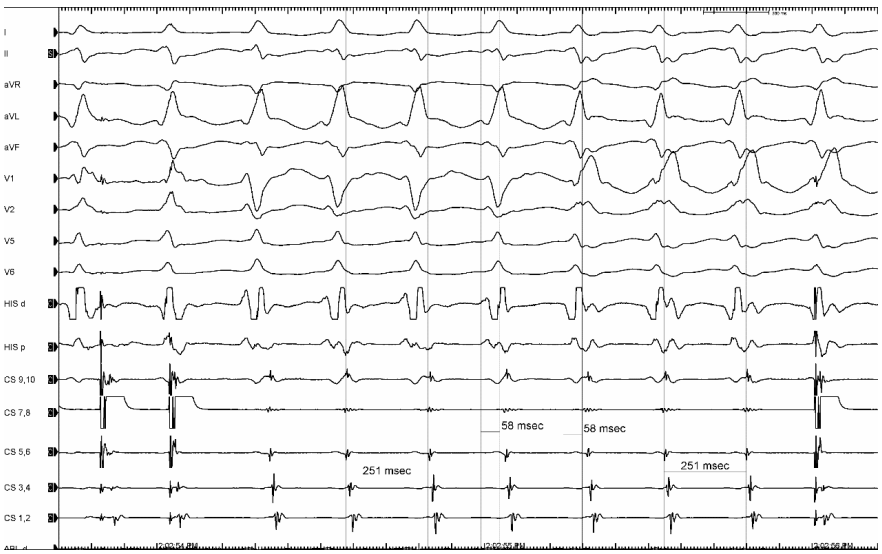
Figure legends:

Figure 1: Twelve lead electrocardiogram of the clinical arrhythmia demonstrating wide complex tachycardia of LBBB morphology at a rate of 200bpm.

Figure 2A: Induction of LBBB tachycardia by incremental CS pacing following AH prolongation and HV shortening. Note that the last 4 paced beats are non-captured beats during LBBB tachycardia. CS 1,2: distal coronary sinus; CS 9,10: proximal coronary sinus; His d: distal His bundle. His p: proximal His bundle. HRA: high right atrium. H: His deflection.

Figure 2B: Atrial extra-stimuli (arrow) during His refractoriness. Baseline tachycardia cycle length is 310ms. His deflection is marked on the distal His channel as H. The atrial electrogram is marked as A and the ventricular electrogram as V. CS 1,2: distal coronary sinus; CS 9,10: proximal coronary sinus; His d: distal His bundle. His p: proximal His bundle. RV: Right ventricle.

Figure 3A: Wide complex tachycardia at a CL of 348ms. Ablation catheter is placed at the ablation site. **3B** : CS pacing at 280ms after successful ablation. A: Atrium, V: Ventricle. CS 1,2: distal coronary sinus; CS 9,10: proximal coronary sinus; ABL d: distal ablation; ABL p: proximal ablation.



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