“Atrial Fibrillation and Premature Ventricular Complexes” – is there a unifying diagnosis?

Luis Quininir¹, Emily Hodkinson¹, Kim Chan¹, and Raymond Sy¹

¹Royal Prince Alfred Hospital

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Authors:
Luis Quininir MD a
Emily Hodkinson MD a
Kim H Chan MBBS, PhD a, b
Raymond W Sy MBBS, PhD a, b

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Affiliations:
a Royal Prince Alfred Hospital, Sydney, Australia
b University of Sydney, Australia

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Address for correspondence:
A/ Prof Raymond Sy, Department of Cardiology, Level 6, Royal Prince Alfred Hospital NSW, Australia
Tel: +612 -9515 7929 Fax: +612 – 9550 6262 Email: Raymond.sy01@gmail.com

Case:
A 64-year-old male presented with a 2-year history of palpitations and light-headedness. Referral for electrophysiological assessment was prompted by a Holter monitoring report of “frequent ventricular ectopic beats (>21,000/24hr) with repetitive runs of atrial fibrillation” (Fig 1A). Resting echocardiogram suggested low-normal left ventricular function, and stress testing was normal. Electrocardiogram (ECG) at baseline was normal. A 12-lead rhythm strip was recorded during a brief burst of tachycardia in clinic (Fig 1B). What are the differential diagnoses for the arrhythmia?

Commentary
Careful examination of the Holter monitoring showing frequent extrasystoles revealed sinus rhythm marching though the tracing without perturbation (Figure 2A). Differential diagnoses for the extrasystoles would
include ventricular ectopy, His-Purkinje ectopy, or supraventricular ectopy conducting with aberrancy, with the latter including atrial ectopy, atrio-ventricular nodal echo beats and more rarely dual AV nodal physiology with ‘double fire’ response. Similarly, examination of irregular narrow complex tachycardia initially reported as “atrial fibrillation” revealed putative P waves consistent with sinus rhythm, with supraventricular extrasystolic beats in a grouped beating pattern.

The 12-lead rhythm strip during tachycardia is helpful (Figure 2B). Again, sinus rhythm is observed without perturbation in rate. The extrasystoles exhibited variable degrees of aberrancy. The 12-lead analysis also allows one to be reasonably confident that there are no P waves preceding the extrasystolic beats making atrial bigeminy much less likely. Single AV nodal echo beats would also be unlikely in the absence of significant PR perturbation when one compares the first 2 sinus beats on the tracing. The remaining differential diagnoses would include: 1) ectopic beats arising from the His-Purkinje system; 2) dual AV nodal pathways with ‘double fire’ response, conducting with varying aberrancy.

An electrophysiological study (EPS) was then performed under conscious sedation. Quadripolar catheters were positioned at the high right atrium, and right ventricular apex. Decapolar catheters were positioned in the coronary sinus, and His bundle and right bundle region to assess conduction through the His-Purkinje conduction system.

The clinical arrhythmia was reproducible with slow atrial pacing in the range of 700 – 900 ms. Again, the extrasystolic beats exhibited either normal QRS morphology, right bundle branch block (RBBB), or RBBB and left anterior fascicular block, all with the same coupling interval (Figures 3A). Examination of the intracardiac electrograms revealed abrupt changes in AH interval between short AH and very long AH intervals (680ms), suggestive of dual AV nodal physiology, as well as potential ‘two for one’ AV response (Figure 3A).

Although it was difficult to definitively exclude junctional extrasystoles as an alternative explanation for the extrasystoles, retrograde His activation with shortening of the HV interval is often described with junctional extrasystoles. In our case, the His activation was always proximal to distal and the HV interval was unchanged compared to that during sinus rhythm. Also, the coupling interval of the extrasystolic beat appeared consistent with both decremental atrial pacing and atrial extra-stimuli (Figure 3B). The extrasystoles were only observed during a specific range of atrial pacing intervals and they were suppressed with faster atrial pacing and during isoprenaline. Taken together, these features are most compatible with a dual AV nodal non-re-entrant tachycardia or ‘double fire’ response, where sinus beats are simultaneously conducted anterogradely over both a slow and fast pathway. This produces a long-short coupling of the His-Purkinje bundle, resulting in aberrancy pattern (RBBB and/or LAFB) on the second beat. Notably, AV nodal re-entrant tachycardia was not inducible.

Ablation of the slow pathway is the treatment of choice for double fire tachycardia with reports of 100% acute success and 94% long term success. One patient was treated by increasing the lower rate pacing of his implanted device from 40 bpm to 70 bpm to suppress slow pathway conduction. In our case, radiofrequency energy was applied in the slow pathway region. Occasional junctional extrasystole was observed during application of RF energy. Following ablation, there was no further evidence of extrasystolic beats.

Dual AV nodal response conduction is a rare phenomenon. For it to occur, the effective refractory period of the His-Purkinje system must be shorter than the difference in AH intervals between fast and slow pathway conduction. Usually retrograde ventriculo-atrial conduction is poor or absent, as in the present case.

Dual AV node physiology with “double fire response” can mimic a variety of other arrhythmias, including PVCs and atrial fibrillation as was the case in our patient. It can cause symptomatic palpitations and also tachycardia-induced cardiomyopathy. These findings emphasize the importance of carefully examining the ECG of an irregular arrhythmia, with close attention to the presence of grouped beating, and include double response in the differential diagnosis.

Figure 1
A) Holter traces. Top panel shows sinus rhythm with “PVCs”. Bottom panel shows “apparent AF”

B) 12-lead rhythm strip during tachycardia

Figure 2

A) Holter traces. Top panel shows sinus rhythm (vertical arrows) with interpolated wide-QRS complex extrasystoles (annotated +). The coupling interval of the extrasystolic beats is consistent. Bottom panel shows the irregular narrow complex tachycardia with sinus p waves annotated by vertical arrows.

B) 12-lead rhythm strip during tachycardia. Sinus P waves are observed with a regular rate approximately 65 bpm (red arrows). The extrasystolic beats have several different morphologies: Right bundle branch block (RBBB) with left anterior fascicular block (LAFB), normal QRS, RBBB (+) and LAFB ( ).
Figure N°3 Electrophysiology study

A) Atrial pacing at 810 ms. The second and the fourth beats have 2:1 AV response (blue arrows). Note that both extrasystolic beats have proximal to-distal His-RBB activation and the same HV interval as sinus rhythm. Also, the second extrasystolic beat has RBBB + LAFB (annotated) consistent with the clinical arrhythmia. The third beat fails to conduct to the ventricle (red crossed arrow), possibly due to retrograde concealed penetration of the fast pathway by the preceding beat. The 5th beat is conducted with an abrupt prolongation in the AH interval, reflecting a change from fast to very slow AV nodal pathway conduction (green arrow) HBE= His bundle electrogram; HRA=high right atrium; RVA= Right ventricular apex; CS= coronary sinus

B) Atrial extrastimulus pacing 900/810ms: Double response is observed with consistent AH₂ interval during extrastimulus testing (blue arrows) HBE= His bundle electrogram; HRA=high right atrium; RVA= Right ventricular apex; CS= coronary sinus
REFERENCES


