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Paroxysmal Complete Atrioventricular Block: Who is the True Culprit?

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***Corresponding Author:** Hai-Ying Li, Department of Cardiology, Shenzhen University General Hospital, Nanshan District, Shenzhen, People's Republic of China. # - Contributes Equally to this Work.*

Abstract

A 60-year-old woman was presented with unexpected syncope in the emergency department. After performing a serious of examinations, we obtained that intact paroxysmal atrioventricular block (AVB) was the culprit. Moreover, 24h Holter monitoring showed complete paroxysmal AVB occurred in no special conditions, having nothing to do with pause-dependent 4 phase block, 3 phase block, premature beats, Mobitz type II AVB and rate-related heart beats. Our report is entirely different from the present submitted research. We performed electrophysiological study to testify our thought. The intracardiac electrogram showed that AVB existed in mid-distal location of His-Purkinje system, which had a decreased voltage-gated sodium channel availability and then sinus impulse conducted to ventricle was prevented.

The most outstanding feature of our study different from other reports is that the whole probable factors contributing to intermittent AVB occurred in the same patient. Nevertheless, other reports revealed the above phenomena referred to different patients. It is reasonable for us to regard variable factors inducing paroxysmal AVB as a coincidence.

Case Presentation

A 60-year-old woman, without a history of hypertension, diabetes mellitus and heart disease, presented with unexpected syncope about a half hour ago in the emergency department. She recovered consciousness spontaneously and there was no complaint of palpitation and fatigue. On arrival in the emergency department, her heart rate was 50 beats per minute (bpm), and respiratory rate was 18 bpm. Oxygen saturation was 99% under room air. This patient underwent a full cardiovascular, pulmonary and neurological work-up that excluded structural diseases. The levels of electrolytes, glucose, cardiac enzyme, tests of coagulation, D-dimer, renal and liver functions as well as complete blood count were all normal. An ECG obtained at bedside indicated sinus bradycardia (50bpm) without other obvious abnormality (Figure 1). Standard 24h Holter monitoring showed 65 periods of ventricular asystole (paroxysmal third-degree atrioventricular block, AVB) ranging from 3.5 seconds to 12.3 seconds, which occurred both in day and light. We could also find repeatedly Mobitz II type AVB, 2:1 AVB and premature beats during in the 24h monitoring (Figure 2). Finally, the patient underwent permanent cardiac pacing with selective His-bundle pacing without recurrence of syncope during hospital.

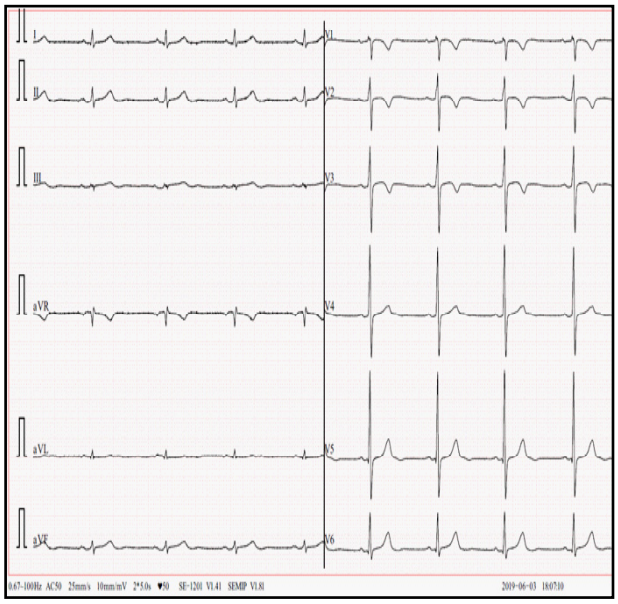

Fig1. *EDAN SE-1201(made in LiBang of Shenzhen, China) The above leads were recorded simultaneously. The above rhythm strip shows sinus bradycardia (50bpm) with P wave interval 100ms, PR interval 170ms, QRS complexes interval 80ms, inverted and asymmetric T waves in V1-V3 leads and slightly inverted U waves in V4-V5 leads.*

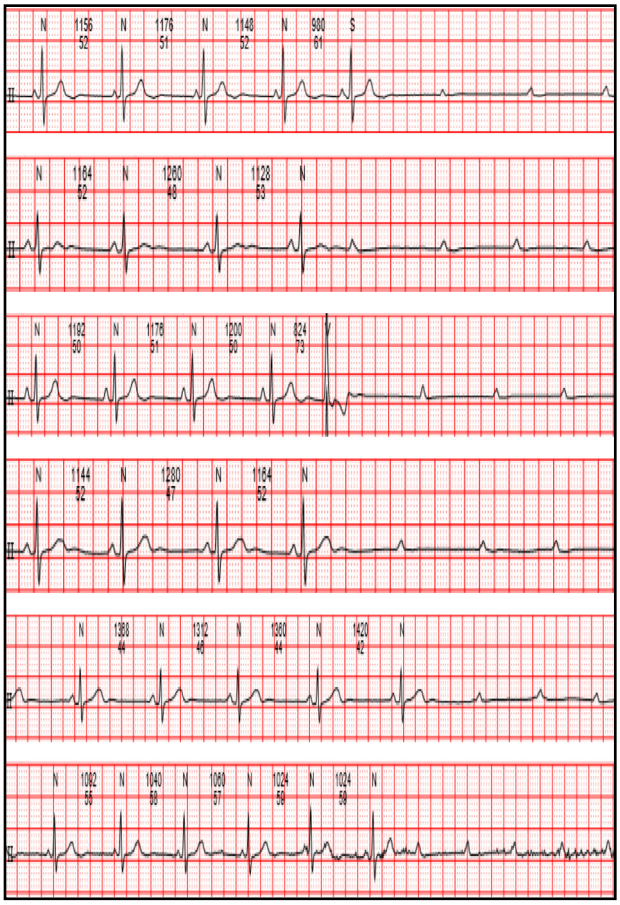

Fig2. *The above rhythm strip shows standard II lead only. From top to bottom, the six rhythm strips indicate: atrial premature beat with conducted QRS complex contributes to paroxysmal third-degree AVB; atrial premature beat with nonconducted QRS complex contributes to paroxysmal third-degree AVB; ventricular premature beat results to paroxysmal AVB; slow rate-related paroxysmal third AVB; fast rate-related paroxysmal third-degree AVB; stable rhythms (rate-unrelated) accompany with unexpectedly AVB.*

Discussion and Conclusion

In conclusion, the most persuasive reason for the syncope is intact AVB which lead to cerebral ischemia. However, paroxysmal total AVB, characterized by its episodic nature, is frequently underdiagnosed and neglected. The underlying conduction system abnormality of our patient highly likely exists in His-Purkinje system.

So far, some reports have discussed that paroxysmal AVB is associated with pause-dependent 4 phase block, 3 phase block, premature beats, Mobitz type II AVB and rate-related heart beats.[1,2] As we known, diseased His-Purkinje system characterized by a proclivity for spontaneous diastolic depolarization. Prasada S et al suggested that compensatory pause generated by ventricular premature beat provided extra time for depolarization of His-Purkinje myocytes, which had a decreased voltage-gated sodium channel availability and then sinus impulse conducted to ventricle was prevented.[2]

Several reports regard that intermittent AVB is associated with extrinsic vagal AV block and lower baseline adenosine plasma level.[3] However, extrinsic vagal AV block is usually characterized by gradual prolongation of PR and PP intervals[2], and when heart rate increases, vagal AV block will improve. But this situation cannot be found in our report.

Surprisingly, our report uncovers thoroughly contrary opinion. In our study, paroxysmal AVB is most frequently to be seen after atrial premature beats (APBs), especially after the non-conducted APBs. Nevertheless, paroxysmal AVB is not seen in the whole APBs with incomplete compensatory pauses. Furthermore, paroxysmal AVB has no causal relationship with gradually accelerated sinus rhythm or sharp decelerated rhythm. In the same way, ventricular premature beats (VPB) do not always induce third AVB. Therefore, paroxysmal AVB related to 4 phase block, 3 phase block or unstable heart beats previously reported is not fully accepted now. Just as the study of Nabil EI-Sherif, MD et al said[1], they suggested that paroxysmal AVB was not related to phase 3 block or phase 4 block. As we know, we have still held the viewpoint that phase 3 and phase 4 block are equal to tachycardia-dependent and pause-dependent block, respectively.[1] So it is the high time for us to change our inappropriate standpoint.

To sum up, complete paroxysmal AVB occurs in no special conditions Figure2. We agree with the opinion of Prasada S et al.[2] Our explanation for this phenomenon is the diseased His-Purkinje system with decreased availability of sodium channels, instead of other special factors. Hence normal PR interval and QRS complexes are not enough to exclude diseased His-Purkinje system. We performed electrophysiological study to testify our thought. The intracardiac electrogram showed that AVB existed in mid-distal location of His-Purkinje system (Figure 3).

The most outstanding feature of our study different from other reports is that the whole probable factors contributing to intermittent AVB occurred in the same

patient. Nevertheless, other reports revealed the above phenomena referred to different patients. It is reasonable for us to regard variable factors inducing paroxysmal AVB as a coincidence.

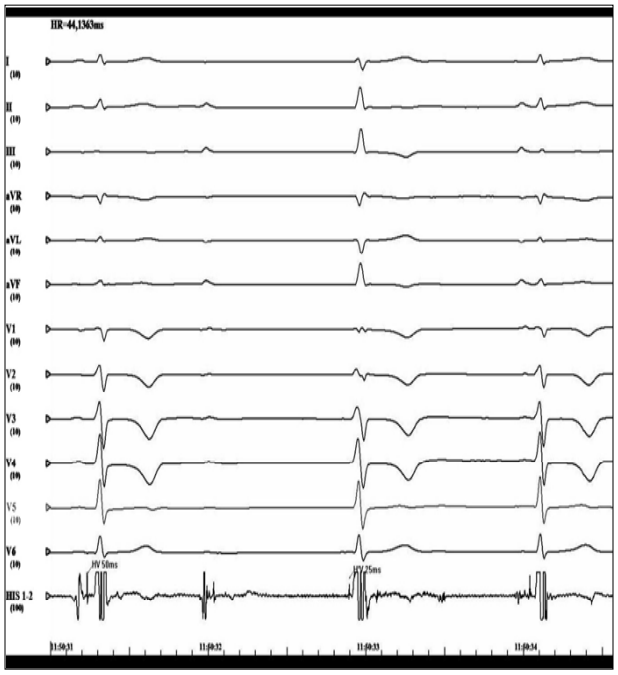

Fig3. *Mapping of the location of AVB: The above rhythm strips show 12 leads recorded simultaneously and His potential mapped by 3830 pacing electrode (Medtronic). The first and the last QRS complexes are conducted by sinus P waves, with normal PR interval and stable HV interval (50 milliseconds). The second sinus impulse fails to conduct to ventricle, with the constant AH interval. So we obtain that the location of AVB occurs in mid-distal His bundle. The second aberrant QRS complex is preceded by H wave, with 25 milliseconds HV interval. Therefore, the escape rhythm originates in the distal His-Purkinje system of AVB location*

References

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