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Case Report

Paroxysmal atrioventricular block induced during paroxysmal intra-atrial reentrant tachycardia



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ABSTRACT

Paroxysmal atrio-ventricular (AV) block is a relatively rare form of bradyarrhythmia that may be caused by vagal reflex, intrinsic His-Prukinje system (HPS) disorder, or idiopathic mechanisms. We report a case with paroxysmal AV block and syncopal episodes that appeared only during intra-atrial reentrant tachycardia (IART) after an ablation procedure. Syncope did not occur under sinus rhythm with stable 1:1 AV conduction. An HPS disorder was proven in an electrophysiological study. It was suggested that paroxysmal AV block was induced via a tachycardia-dependent mechanism with an exacerbation of latent HPS disorder. The occurrence of the IART was only transient, and there was no recurrent syncope during one-year follow-up. Pacemaker implantation could be avoided.

<Learning objective: Paroxysmal atrio-ventricular block is a relatively rare form of bradyarrhythmia and can occur only during atrial tachyarrhythmia. Indications for a cardiac implantable electrical device should be carefully considered if the attributed tachycardia is treatable.>

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Introduction

Paroxysmal atrioventricular (AV) block is an uncommon cause of syncope that can be overlooked because of its unfamiliarity and unpredictability. It occurs in cases without apparent evidence of cardiac abnormalities nor AV conduction disturbances in a usual condition. Vasovagal reaction mediated, intrinsic, and idiopathic mechanisms are thought to be associated [1, 2]. It has been reported that some premature contractions trigger paroxysmal AV block [3, 4].

We here report a case of paroxysmal AV block that was induced during intra-atrial re-entrant tachycardia (IART) but not induced during sinus rhythm (SR).

Case report

The patient was a 68-year-old male with paroxysmal AF [atrial fibrillation] and IART. He had a history of pre-syncope after taking anti-arrhythmic drug (pilsicainide hydrochloride hydrate), but had no family history of sudden death or cardiac disease. He had a history of catheter ablation (CA) and bilateral pulmonary vein isolation for persistent AF 8 years before.

He was admitted to our hospital due to symptomatic recurrent paroxysmal AF and IART while taking bepridil. The CHA2DS2-VASc score was 2 due to hypertension and his age. He underwent a 2nd session ablation. There was no residual electrical activity in the pulmonary veins (PV). Low voltage areas in the anterior and septum wall, and the antrum of right PVs could be identified using an HD grid mapping catheter (Abbott, St Paul, MN, USA) under the navigation of the Ensite Navx system (Abbott). Planar ablation in those low voltage areas, superior vena cava isolation, and cavotricuspid isthmus ablation were performed.

On post-procedural day 1, paroxysmal AF and paroxysmal IART recurred with palpitation (Fig. 1A). Episodes of syncope with tran-

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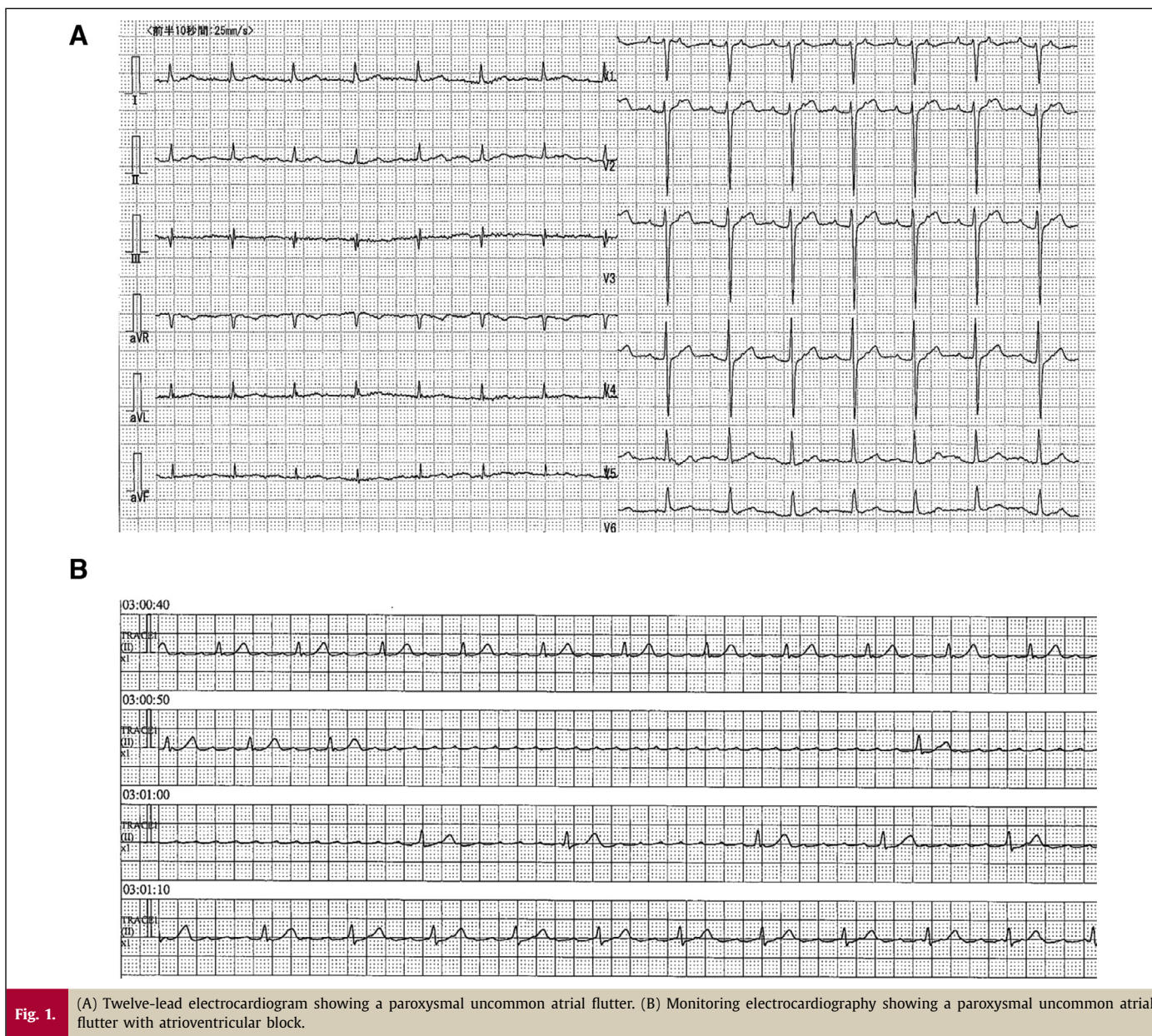


Fig. 1. (A) Twelve-lead electrocardiogram showing a paroxysmal uncommon atrial flutter. (B) Monitoring electrocardiography showing a paroxysmal uncommon atrial flutter with atrioventricular block.

sient AV block were observed in electrocardiogram (ECG) monitor recordings only during IART with about 16 s pause at maximum (Fig. 1B; This figure is from a different time because the ECG during the 16-second pause is not available). A temporary pacemaker was introduced and he was observed for 5 days. Paroxysmal AV block did not recur after then during SR (Fig. 2). We considered that paroxysmal AV block was induced by transient post-procedural effects, and he was discharged. However, he was readmitted to our hospital due to a presyncope episode that night. ECG monitoring again demonstrated paroxysmal AV block occurring only during IART. We thus considered that the IART was related to the transient AV block, and performed CA to the IART. An EPS [electrophysiological study] suggested the IART was LA [left atrium] roof-dependent and a linear ablation to the LA roof was performed. At the end of this procedure, we confirmed that the atrio-His-interval was prolonged whereas the effective refractory period of AV conduction was 200 msec, which is within normal range. Although he had first degree AV block before the 2nd CA, the monitoring ECG

before and after the session showed stable 1:1 AV conduction during SR.

Further, we performed a pharmacological stress test to the His-Purkinje system (HPS) using pilsicainide hydrochloride hydrate, because he had a history of pre-syncope after taking pilsicainide. His-ventricular (HV) interval was prolonged in the same way as atrio-His-interval (Fig. 3A,B). Therefore, we did not prescribe additional anti-arrhythmic drug treatment. Moreover, we suggested an implantation of pacemaker against paroxysmal AV block, however, the patient requested an observation period after the treatment of tachyarrhythmia, understanding the risk of cardiac sudden death as well. Paroxysmal AV block did not recur during the admission. During 12 months of follow-up, SR has been maintained and the patient did not experience a recurrent syncope.

Discussion

Paroxysmal AV block is recognized with sudden and unpredictable onset of complete AV block. The true incidence of this

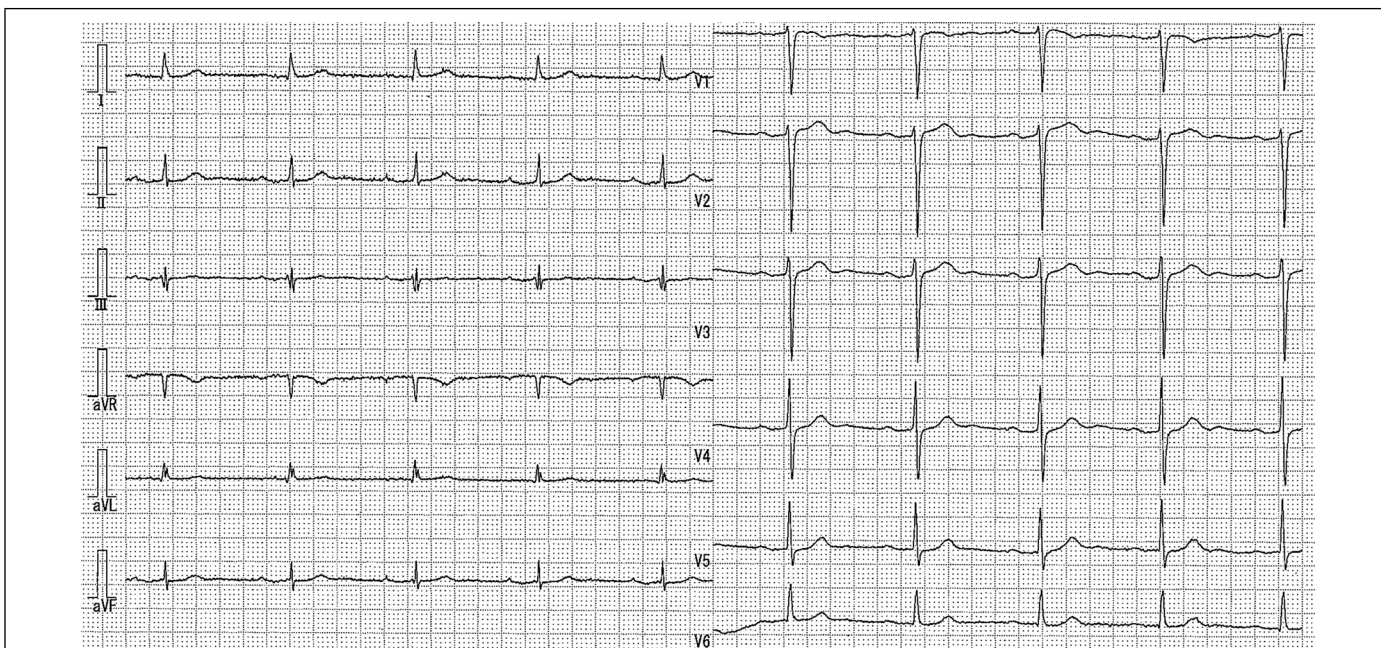


Fig. 2. Twelve-lead electrocardiogram showing a PR interval prolongation and 1:1 atrioventricular conduction.

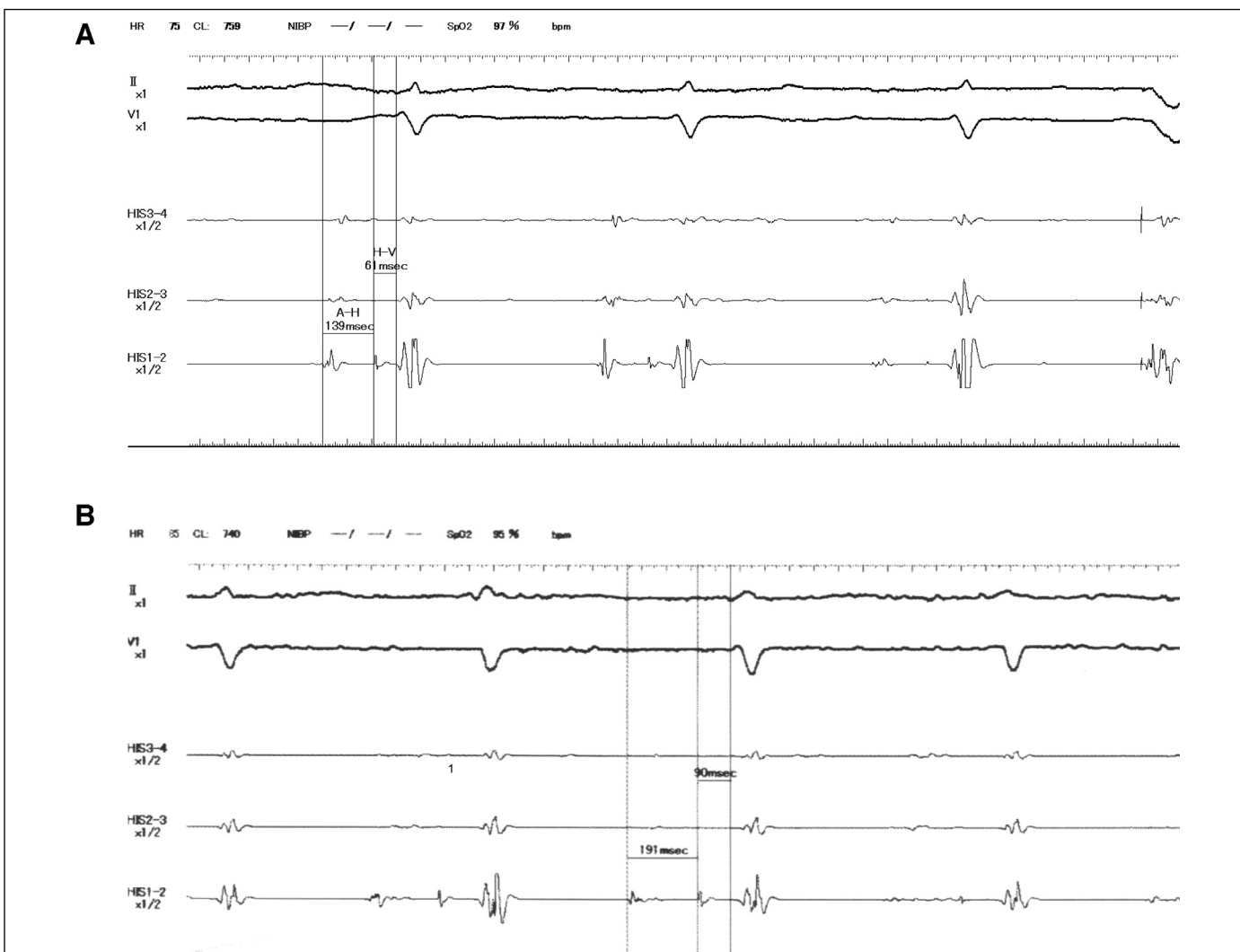


Fig. 3. (A) The intracardiac electrogram showing atrio-His-interval and His-ventricular interval before pilscainide hydrochloride hydrate administration. (B) The intracardiac electrogram showing atrio-His-interval and His-ventricular interval after pilscainide hydrochloride hydrate administration.

phenomenon is not well known. Several investigators have reported that it might be vagally-mediated, intrinsic HPS disease, or idiopathic [1, 2, 5]. Furthermore, paroxysmal AV block has been reported as both bradycardia- and tachycardia-dependent, or an abrupt change in heart rate [4].

Paroxysmal AV block can be induced by paroxysmal supraventricular tachycardia, atrial flutter or premature ventricular contraction [6, 7]. In the present case, paroxysmal AV block occurred only during IART but not during SR. The mechanism of this case can be an intrinsic HPS disorder because 1st degree AV block was observed during SR and the pharmacological stress test of HPS using pilsicainide hydrochloride hydrate further prolonged the AV and HV interval. This Vaughan Williams class I drug has been reported as a potentially useful tool in diagnosis of infra-Hisian AV block [8]. The patient previously had a presyncope after taking pilsicainide hydrochloride hydrate, which may have exacerbated the AV conduction disturbance. Our observation of paroxysmal AV block only during atrial tachyarrhythmia may mimic previously reported “fatigue” phenomenon, suppression of AV conducting by a high-frequency excitement [9]. It has been reported that fatigue phenomenon could be induced both by atrial and ventricular tachyarrhythmia in cases with pathologic HPS. Continuous AV block during atrial tachyarrhythmia could be explained by a repetitive occurrence of subthreshold depolarization of the conduction system in response to high-frequency atrial impulses with underlying electrical disorders of HPS [5]. Moreover, as paroxysmal AV block mainly occurred in the night time, increased vagal tone during the night time also could contribute to the suppression of AV conduction. We thus reasoned that the combination of exacerbating latent electrical disorder of HPS by similar fatigue and changes of autonomic nervous system is the most likely mechanism for causing paroxysmal AV block in this case.

Idiopathic AV block occurs in the absence of any cardiac or baseline ECG abnormalities, and without any known trigger. An increased susceptibility to adenosine triphosphate has been suggested as an underlying mechanism. Vagally-mediated paroxysmal AV block occurs secondary to a surge in parasympathetic activity [1]. In the present case, the syncope did not occur with an identifiable trigger or premonitory symptom, whereas baseline ECG showed a PR interval prolongation.

In the present case, maintenance of SR and withdrawal of antiarrhythmic drug resulted in no recurrence of syncope or AV block for 1 year. Therefore, pacemaker implantation could be evaded. Regardless of the type of paroxysmal AV block, indication of pace-

maker implantation should be considered [8]. Nevertheless, we cannot predict the recurrence of atrial arrhythmias, which induces AV block and syncope. Thus, careful follow-up considering pacemaker implantation has been continued.

Conclusion

We reported a case of paroxysmal AV block that occurred only during IART. This AV block may be caused by an exacerbation of intrinsic HPS disorder or transient fatigue phenomenon induced only during atrial tachyarrhythmia. Permanent pacemaker could be avoided under no recurrence of atrial tachyarrhythmia.

Declaration of Competing Interest

All authors declare no conflicts of interest related to this report.

Acknowledgement

None.

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