症例報告

一過性高度房室ブロックと失神の既往を持つ若年患者へのジソピラミド投与

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Case report

Disopyramide for Transient High-degree Atrioventricular Block in a Young Patient with a history of Syncope

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Short title

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Key words

Atrioventricular block; Syncope; Therapy

要約

失神の既往がある患者に一過性に高度房室ブロックを認めた場合、ペースメーカー植え込みを考慮するが、その治療方針決定には個々で深慮する必要がある。症例は 35 歳男性、3 度の失神の既往を持ち、動悸と嘔気を主訴に来院した。ホルター心電図で動悸を伴う心室性期外収縮と嘔気、おくび、味覚異常を伴う体位に依存しない 5：4 までの一過性高度房室ブロックを認めた。Head-up tilt 試験では血管抑制型の反応陽性で前失神状態を呈した。心臓電気生理検査で刺激伝導系に異常を認めず、房室ブロックは機能性ブロックと診断した。迷走神経系の関与を疑い、ペースメーカーの植え込みは行わず抗コリン作用を持つⅠ群不整脈薬ジソピラミドを 300mg/日で投与すると、症状は軽快し心室性期外収縮と房室ブロックも消失した。5 ヶ月後のホルター心電図で心室性期外収縮も房室ブロックも認めず、退院後 20 ヶ月間経過良好である。

Abstract

Although high-degree atrioventricular (AV) block in patients with a history of syncope usually requires pacemaker implantation, therapeutic strategies should also be considered. A 35-year-old man presented with complaints of palpitations, nausea and dysgeusia. Since aged 30, the patient had experienced 3 episodes of syncope. Holter monitoring showed transient high-degree AV block (up to 5: 4 block) associated with nausea, eructation and dysgeusia irrelevant to posture as well as ventricular ectopic beats with palpitation. A Head-up tilt test revealed neurally-mediated vasodepression but electrophysiological study showed no abnormalities. These results indicated that his transient high-degree AV block was functional, and syncope would have been because of neurally-mediated vasodepression, not bradycardia. After administration of disopyramide at 300 mg daily, the symptoms subsided and ventricular ectopic beats and AV block disappeared. He has been well for 20 months.

Key word; Atrioventricular block; Syncope; Therapy

Case report

A 35-year-old man presented with complaints of palpitations, nausea and dysgeusia. Since aged 30, he had experienced 3 episodes of syncope that had followed discomfort during his day-time work in a standing position. Physical examinations, chest X-ray, 12-lead electrocardiogram, echocardiography and exercise test were all normal, while Holter monitoring showed several occurrences of transient high-degree AV block (up to 5: 4 block) mostly in the morning (figure). The longest ventricular pause was 4.4 seconds; associated symptoms were nausea, eructation and dysgeusia. His sinus rate was not slow and no Wenckebach-type AV block was seen. He also complained of palpitations, but only when ectopic ventricular beats occurred. During a head-up tilt test after a 3-minute loading of isoproterenol, he felt presyncope developing with a sudden 33 mmHg drop of systolic blood pressure (121/49 to 88/29 mmHg) and a moderate decrease of heart rate but without AV block (116 to 91 bpm). Carotid massage test was negative. Electrophysiological study showed no abnormalities in his conduction system

(table 1). These results indicated that his transient high-degree AV block was functional, and syncope would have been because of neurally-mediated vasodepression, not AV block. After administration of disopyramide at 300 mg daily, his symptoms disappeared and the frequency and degree of AV block decreased. Holter ECG repeated 5 months after discharge showed neither AV block nor ventricular ectopic beats. He has been well for 20 months.

Discussion

We reported a case of a patient with transient high-degree AV block, palpitation, nausea and a history of syncope successfully treated by disopyramide, a class I anti-arrhythmic drug. The chief complaints were of palpitation and nausea. The former was related to ventricular premature contraction. The most important aspect of this case is whether the AV block was related to his episodes of syncope. Although the high-degree AV block might be considered as linked to syncope, prudent evaluation should be taken to decide a suitable strategy. In the present

case, head-up tilting test provoked vasodepressive presyncope (strictly, mixed type), the symptom resembling that of his clinical syncope. As well, he complained of nausea, eructation and dysgeusia around the time when AV block occurred. However, the electrophysiological study findings showed no abnormalities in his conduction system. Thus his syncope would have been related to neurally-mediated vasodepression, not AV block. Even if his syncope were the result of a mixed type of neurally mediated syncope, pacemaker therapy is unlikely to have been helpful [1]. Although little is known about the mechanism and treatment of transient AV block, several case reports have illustrated the relationship between transient AV block and vagal hypersensitivity. According to these reports, such functional AV block can cause the sudden onset of syncope, but an anti-cholinergic agent precludes such a functional AV block [2-5]. Disopyramide has a strong anti-cholinergic effect and after its administration, not only ventricular ectopic beats but also AV block and associated symptoms disappeared. These findings also support that the transient AV block was related to vagal nerve activity. However, his heart rate was not significantly slow and

there was no Wenckebach-type of AV block around the time when he had several episodes of transient AV block. These findings suggest that the transient AV block was because of vagal hypersensitivity rather than general vagal surge. With regard to the syncope, it is unknown if disopyramide precluded any recurrence of syncope because we did not perform a head-up tilting test after disopyramide administration and the follow-up period was relatively short. For medication of neurally mediated syncope, midodrine, an α-1 adrenergic agonist, may be recommended rather than disopyramide [6-8]. However, midodrine can increase ectopic beats causing palpitations; as well, the usefulness of α-1 adrenergic agonists has not been clearly established [9]. In the present case, disopyramide resolved palpitations involving ventricular ectopic beats, transient AV block and associated symptoms. Fortunately there has been no recurrence of syncope at this time. Although co-morbidity of syncope and high-degree AV block usually predisposes a patient to pacemaker implantation, a prudent strategy should be ascertained for each case by careful analysis of a patient's history, and after appropriate examinations and evaluations have been completed.

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Legend to Figure.

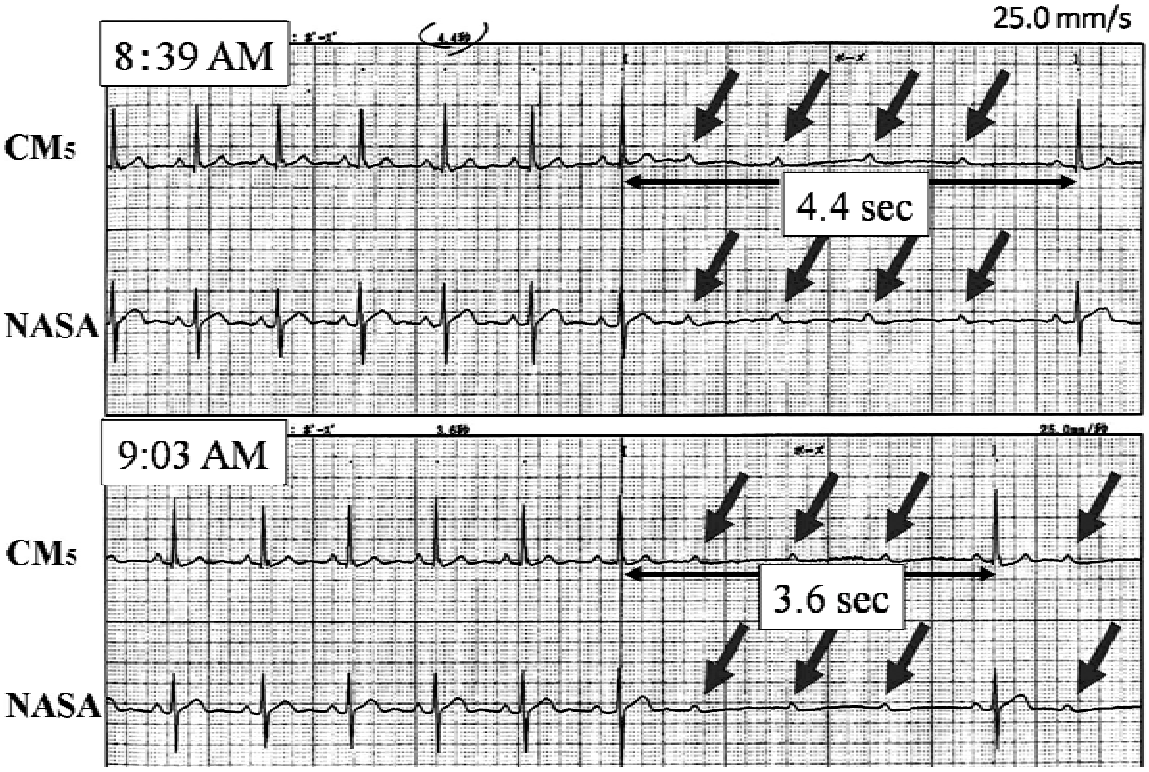

Figure. Strips of Holter monitoring showing high-degree AV block.

Table. Electrophysiological findings.

Parameters	baseline	after PAB
AA interval	772	625
AH interval	106	80
HV interval	56	54
maximal SNRT	1085	–
1: 1 AV conduction (ppm)	170	–
Wenckebach rate (ppm)	160	–
ERP of AV node (msec)	< 290 (BCL 600 msec)	–
VA conduction	none	–

Abbreviations: AA = atrio-atrio; AH = atrio-His; HV = His-ventricular; SNRT = sinus node recovery time; AV = atrio-ventricular; ERP = effective refractory period; VA = ventriculo-atrial; PAB = pharmacologic autonomic block; ppm = pacing per minute; BCL = basic cycle length.