Postprandial Atrioventricular Block in a Patient with Diabetes Mellitus

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Postprandial atrioventricular (AV) block and hypotension occurred in an 80-year-old man with a 13-year history of diabetes mellitus. Two weeks before admission, he experienced syncope on two occasions after lunch. Master's two-step test induced second degree AV block (2:1 block). Four days after admission, syncope recurred while walking 1 hour after eating; electrocardiogram (ECG) showed complete AV block following 2:1 block. PP interval analysis suggested phase 3 block as the mechanism of AV block. Electrophysiologic study revealed 2:1 HV block. Postprandial syncope ceased after permanent pacemaker implantation. Postprandial AV block and hypotension merit close attention in diabetics.

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Introduction

Postprandial hypotension is well known in patients with diabetes mellitus (1), idiopathic Parkinson’s disease (2), and uremia treated with hemodialysis (3) and in the normal elderly (4). However, in the recent literature, there does not appear to be any case report of atrioventricular (AV) block after meals. We report a diabetic patient with Adams-Stokes syndrome due to complete AV block and hypotension after meals.

Case Report

An 80-year-old man with diabetes mellitus visited the outpatient clinic of Hiroshima Prefectural Hospital. He had been taking oral hypoglycemic agents for ten years and insulin for the recent 3 years. Two weeks prior to admission, he experienced syncope on two occasions after lunch. His electrocardiogram (ECG) at rest showed sinus rhythm of 60 beats/min with right bundle branch block, normal electrical axis with PQ interval of 0.20 second, QRS duration of 0.12 second and QT interval of 0.44 second (Fig. 1). Master’s two-step test induced second degree AV block (2:1 block) (Fig. 2). After admission ECG was monitored. About thirty minutes after each meal, 2:1 AV block occurred and the patient sometimes felt slightly faint. On the third admission day, blood pressure was measured before and after lunch; it decreased from 138/74 to 79/36 mmHg thirty minutes after lunch (Table 1). Between 5 and 60 minutes after lunch, 2:1 AV block was observed. Walking one hour after supper on the fourth admission day induced transient complete AV block (Fig. 3). Consciousness was restored by cardiac massage and intramuscular injection of atropine sulphate. Based on analysis of monitoring ECG, the PP interval was 0.72 second in complete AV block, 0.72–0.84 second in 2:1 block and 0.90–1.12 second in 1:1 AV conduction.

A pacing catheter with bipolar electrodes was inserted from the right subclavian vein and placed in the right ventricle for temporary pacing. Coronary angiography was also performed and revealed no significant coronary stenosis. Electrophysiologic study showed 2:1 HV block with an AA interval of 0.88 second (Fig. 4). A permanent pacemaker was implanted, and his syncope disappeared.

Discussion

Food ingestion causes important hemodynamic adjustments. Blood flow is redistributed towards the mesenteric and hepatic circulation while total peripheral resistance decreases (5). However, such vasodilatation is compensated by a sympathetic reflex, and both heart rate and stroke volume increase. As a result, under normal conditions, blood pressure remains unchanged or falls very slightly after meals (5, 6). Ryan et al reported from spectral analysis of heart rate dynamics in elderly
Okamoto et al

Table 1. Hemodynamic Changes after Meal

<table>
<thead>
<tr>
<th></th>
<th>before</th>
<th>5min</th>
<th>30min</th>
<th>60min</th>
<th>180min</th>
</tr>
</thead>
<tbody>
<tr>
<td>BP (mmHg)</td>
<td>138/74</td>
<td>98/45</td>
<td>79/36</td>
<td>122/72</td>
<td>141/80</td>
</tr>
<tr>
<td>HR (b/min)</td>
<td>53</td>
<td>39</td>
<td>40</td>
<td>63</td>
<td>62</td>
</tr>
<tr>
<td>Rhythm</td>
<td>NSR</td>
<td>2:1AVB</td>
<td>2:1AVB</td>
<td>NSR</td>
<td>NSR</td>
</tr>
</tbody>
</table>

BP: blood pressure (systole/diastole), HR: heart rate, NSR: normal sinus rhythm, AVB: atrioventricular block.

This compensatory response, patients with impaired autonomic nerves may display postprandial hypotension. Several investigators found that postprandial hypotension (1-4) occurs in patients with diabetes mellitus, Parkinson's disease, Shy-Drager syndrome, alcoholic neuropathy, and uremia treated with hemodialysis and even in normal elderly persons.

The present patient was 80 years old and had a long history of treated diabetes mellitus. Advanced age and diabetic neuropathy may be related to postprandial hypotension. Moreover, in the present patient, AV block occurred after meals. Master's two step test also induced second degree AV block. It is reported that transient AV block may occur immediately after swallowing (8, 9). However, AV block in the present patient occurred about fifteen to thirty minutes after meals. Several mechanisms of AV block were considered in this patient. One
Postprandial AV Block

possible mechanism is predominant parasympathetic tone after meals or exercise. However, intracardiac ECG revealed infra-His block. Usually, autonomic activities do not exert an influence on the HV interval compared with the AH interval. Therefore, a predominant parasympathetic tone does not seem to be a major factor responsible for AV block in this patient. A second possible mechanism of AV block is a reduction in coronary flow with decreasing blood pressure after meals. The minimum blood pressure after meals in this patient was 79/36 mmHg. Hypotension may cause a deterioration in AV conduction in patients with an impaired His-Purkinje system. However, when blood pressure decreases gradually, coronary flow is usually kept constant by means of coronary autoregulation until systolic blood pressure decreases to 80 mmHg or less. Moreover, significant stenosis was not evident in the coronary angiogram. Accordingly, coronary blood flow reduction is not considered a major factor responsible for AV block in this patient. A third possible mechanism is phase 3 block (10). A conduction disturbance occurs when the heart rate is accelerated. It is assumed that the blocked impulse reaches the injured fibers of the involved region during phase 3 of abnormally prolonged action potentials. Based on the analysis of monitoring ECG, AV conduction was maintained at 1:1 when PP intervals were more than 0.9 second. The observed PP intervals of less than 0.84 second caused 2:1 or complete AV block. These data may support phase 3 block (tachycardia-dependent) as the mechanism of transient AV block in this patient. It is speculated that acceleration of the sinus rate (shortening of PP intervals) may be induced by compensatory sympathetic activation following hypotension after a meal or exercise.

In conclusion, diabetic patients with fainting episodes after meals merit attention not only for postprandial hypotension but also for AV block.

References