Sinus Node Deceleration During Exercise Stress Testing: Bezold-Jarisch Reflex versus Sinus Node Ischemia

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Introduction

Sinus node deceleration, defined as an initial increase and subsequent decrease in heart rate with continued exercise at the same or higher work load, has been described as a marker of right coronary artery disease associated with inferior wall ischemia (1). This report describes a patient, who presented with dizzy spells and presyncopal episodes for one month. The initial workup (including Holter monitoring and transthoracic echocardiography) was negative. After having a positive exercise stress test for myocardial ischemia associated with sinus node deceleration, the patient underwent coronary angiography, which showed two vessel disease including severe ostial right coronary artery stenosis.

Case Report

A 48-year-old woman with a history of type 2 diabetes mellitus for five years presented to the outpatient cardiology clinic with dizzy spells and two episodes of presyncope on exertion for one month. She denied any chest pain or shortness of breath. Her 48-hour Holter monitoring electrocardiogram and transthoracic echocardiogram were unremarkable.

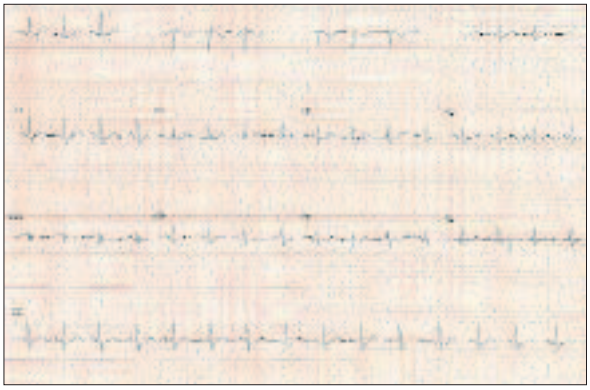

Figure 1. Baseline 12-lead electrocardiogram

An exercise stress test (Bruce protocol) was ordered to rule out significant coronary artery disease. Baseline electrocardiography (ECG) showed normal sinus rhythm with a rate of 101 beats/min (Figure 1). There was rSr' pattern in V1 with a QRS width of 80 miliseconds. There were no ST-T wave changes. Her heart rate steadily increased up to 124 beats/min towards the end of stage-1 of the protocol when she started complaining of dizziness and blurred vision. The test was stopped immediately. Her systolic blood pressure was undetectable. ECG revealed junctional rhythm of 43 beats/min with 2 to 3 mm ST segment elevation in leads III, aVF and V1 with 2 to 3 mm ST segment depression in leads I and aVL (Figure 2). There was rSr' pattern in V1 and RSr' pattern in V2-V3 with QRS width of 90 miliseconds. Her symptoms resolved completely without any intervention. Systolic blood pressure and ECG normalized within minutes. Because of the apparent evolutionary ischemic ECG changes associated with hemodynamic instability, the patient was taken to the catheterization la-

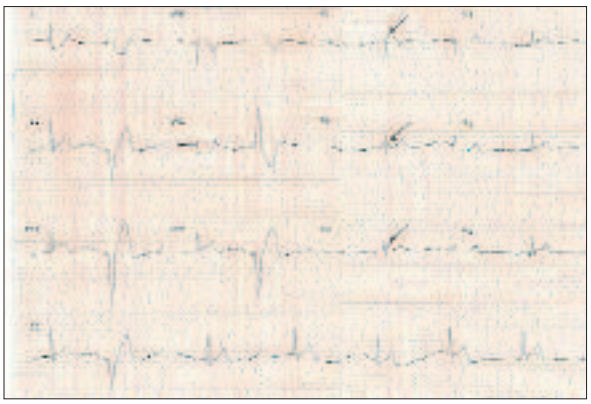

Figure 2. Twelve-lead electrocardiogram showing sinus node deceleration with junctional rhythm. Of note, in addition to ST-segment elevations in inferior leads and V1, there is slight widening of the QRS width in V1-V3 (arrow), compared to baseline probably secondary to right ventricular ischemia indicating right coronary artery stenosis proximal to right ventricular branch.

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boratory. Coronary angiography revealed 95% ostial right coronary artery (RCA) stenosis and 75% proximal left anterior descending (LAD) artery stenosis (Figure 3-A, B). Sinus node artery originated in the posterolateral segment of the left circumflex artery (Figure 3-C, D). Left ventricular ejection fraction was 64%. She underwent a successful two vessel bypass surgery (saphenous vein graft to RCA and left internal mammary artery to LAD). Exercise stress test (Bruce protocol) was repeated 6 weeks after her CABG surgery. She tolerated the test very well without any signs of ischemia or hemodynamic instability.

Discussion

Sinus node deceleration was first described during exercise stress testing (1). It has been also reported during dobutamine perfusion scintigraphy and dobutamine stress echocardiography (2-4). It occurs in < 1% of patients during exercise stress testing and 8% of patients during dobutamine stress testing. Although most often observed in patients with coronary artery disease (particularly in the presence of significant stenosis in the right coronary artery), it can occur in the absence of ischemia and coronary artery disease, and in some patients may be due to a vasodepressor reflex (in the case of sinus node deceleration this is a true vagal component of the reflex) especially during dobutamine stress testing (3,4).

The most likely mechanisms to explain sinus node deceleration in our patient were ischemia-induced provocation of the Bezold-Jarisch reflex (BJR), direct sinus node ischemia or intrinsic sinus node dysfunction. Bezold-Jarisch reflex is an inhibitory reflex originating in the sensory receptors with vagal afferents and preferentially located in the inferoposterior wall of the left ventricle. This reflex produces both an increase in parasympathetic activity and a decrease in sympathetic activity resulting in bradycardia, vasodilatation, and hypotension. It is observed most frequently or implicated during inferoposterior myocardial infarction or ischemia, neurocardiogenic syncope, or coronary angiography. Although the exact nature of the involved sensory receptors is not known, they are influenced by mechanical and/or chemical stimuli, and their impulses are conducted through the afferent pathway by non-myelinated slow-conducting fibers (C-fibers) (5). Bezold-Jarisch reflex seems to be the most plausible explanation for sinus node deceleration in our patient.

Although it was not the case in our patient, sinus node dysfunction secondary to direct ischemia of the sinoatrial node should be ruled out particularly in patients with sinus node deceleration, ST-segment elevations in inferior leads and V1, and conduction delays in right precordial leads indicating high-grade stenosis in the proximal segment of the right coronary artery where sinus node artery and right ventricular branch may originate (6). In our case, sinus node artery originated in the posterolateral segment of the left circumflex artery which did not have any obstructive lesions.

Sinus node deceleration should be distinguished from chronotropic incompetence developing secondary to intrinsic sinus node dysfunction in which heart rate shows inappropriate acceleration during exercise stress testing. Four different types of chronotropic incompetence have been described (7). In one particular type, patients may experience frequent and unpredictable fluctuations in heart rate during activity. In our case, sinus node deceleration can be differentiated from the intrinsic sinus node dysfunction by the presence of dynamic ST-segment changes during the first exercise stress test and disappearance of ST-segment changes along with completely normal chronotropic response during the second exercise stress test after the revascularization surgery.

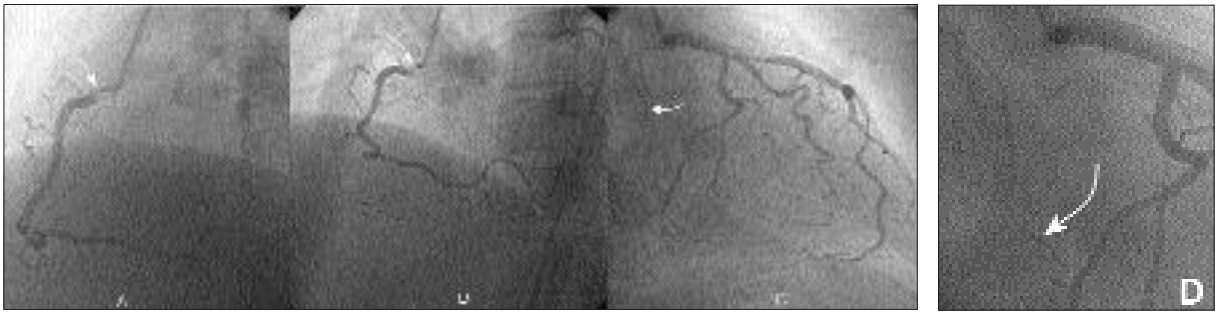

Figure 3. A. Right coronary artery angiography in left anterior oblique view showing the ostial right coronary artery stenosis (arrow). B. Shallow left anterior oblique cranial view. C. Left coronary artery angiography in right anterior obligue view. Sinus node artery (arrow) originating from the posterolateral segment of the left circumflex artery. D. Enlarged view of the sinus node artery (arrow), free of obstructive lesions.

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