Sick Sinus Syndrome in a Patient with Single Coronary Artery Anomaly

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Abstract: Single coronary artery anomaly is very rare. The reported manifestations include angina pectoris and congestive heart failure. Here we describe a case of single coronary artery anomaly presenting as sick sinus syndrome, which has no literature precedence. A 47-year-old woman had complained of intermittent dizziness for years. A Holter electrocardiogram showed sinus bradycardia and junctional or ventricular rhythm with a maximal ventricular pause of up to 3.2 seconds. Electrophysiologic study revealed prolonged corrected sinus nodal recovery time. Coronary angiography showed that the left anterior descending artery had a long course with a side branch originating from the proximal part and coursing anteriorly to the territory of the proximal portion of the right coronary artery. The sinus node is usually supplied by the sinoatrial branch via the right coronary artery. Aortography showed that the right coronary artery ostium was absent. A permanent pacemaker was implanted and the patient was discharged in good condition. The present case suggests that coronary artery anomaly may lead to compromised blood supply to the sinus node, and hence sick sinus syndrome.

Case Report

A 47-year-old woman with a 10-year history of bradycardia presented with intermittent dizziness and exertional dyspnea of 1 year's duration. The dizziness was usually transient without any prodromal event. She was admitted to National Cheng Kung University Hospital for further evaluation and management.

Her blood pressure was 110/60 mm Hg; heart rate, 46 beats per minute; and respiratory rate, 20 breaths per minute. Physical examination revealed no remarkable findings except for an irregular heartbeat. The 12-lead electrocardiogram showed junctional escape rhythm (Fig. 1). The 24-hour Holter electrocardiogram also displayed sinus bradycardia, junctional escape rhythm, and intermittent atioventricular dissociation with a maximal ventricular pause of up to 3.2 seconds. Two-dimensional echocardiography revealed adequate global left ventricular performance with trivial mitral valve and tricuspid valve regurgitation. Thyroid function was normal. The results of tests for collagen diseases and virus antibodies were all normal.

An electrophysiologic study revealed prolonged corrected sinus nodal recovery time, with a maximum of 9,125 msec, and normal baseline atrium-His and His-ventricle intervals (97 msec and 48 msec, respectively) (Fig. 2). Coronary angiography showed a normal origin of the left main trunk and a long course of the left anterior descending artery reaching the inferoapical area, with a side branch originating...
from its proximal portion and coursing anteriorly to the territory of the proximal portion of the right coronary artery (Fig. 3). There was no sinoatrial branch. The distal portion of the left circumflex artery coursed posteriorly and reached the crux. The right coronary artery ostium was absent on aortography. Left ventriculography revealed adequate global performance without regional wall motion abnormality. Single coronary artery (Lipton classification L-IIA [5]) with sinus node dysfunction was diagnosed. A permanent pacemaker was implanted and the patient was discharged in good condition. She was well at subsequent follow-up for 1 year.

**Discussion**

Single coronary artery anomaly is a very rare condition, with the reported incidence ranging from 0.024% to 0.066% [1, 5, 6]. In 1979, Lipton et al proposed a useful angiographic classification [5], which was modified in 1990 by Yamanaka and Hobbs [6], according to the site of origin and anatomical distribution of the branches.
Sick Sinus Syndrome with Coronary Artery Anomaly

Fig. 3. (A) Left coronary angiogram showing abnormal origin of the right coronary artery from the proximal portion of the left anterior descending artery in the right anterior oblique projection. (B) Aortogram showing absence of right coronary artery ostium. LAD = left anterior descending artery; LCX = left circumflex artery; RCA = right coronary artery.

of the coronary arteries. Accordingly, our case was classified as LII-A; that is, the single left coronary artery originated from the left coronary sinus of the Valsalva and the right coronary artery originated from the proximal portion of the left coronary artery and coursed anteriorly into the pulmonary trunk.

The usual manifestations of single coronary anomalies include angina pectoris, sudden death, myocardial infarction, and congestive heart failure [1–4]. There has been no report of an association between single coronary anomaly and sinus node dysfunction.

Sick sinus syndrome is a generalized abnormality of cardiac impulse formation and intra-atrial and atrioventricular conduction abnormalities that may be manifested by a variable combination of bradycardias and tachyarrhythmias [7]. In children, sinus node dysfunction occurs most commonly in those with congenital heart disease, particularly following corrective cardiac surgery [8–10]. Anatomical destruction of the sinus node, areas of nodal-atrial discontinuity, inflammation, connective tissue disease, fibrous change, fatty infiltration, or degenerative changes of the nerves and ganglia surrounding the node can all lead to sinus node dysfunction [8]. The artery supplying the sinus node usually branches from the right (55%–60%) or the left circumflex (40%–45%) coronary artery, approaching the node from a clockwise or counterclockwise direction around the superior vena caval-right atrial junction [11].

Occlusion of the sinus node artery is a possible cause of sinus node dysfunction [10]. In a postmortem angiographic study [9], coronary artery disease, especially after myocardial infarction, was found in one-third of decedents with chronic sinoatrial disorder, and was probably related to the obstruction of blood flow to the sinus node. In the present case, there was no artery branching toward the sinoatrial node over the territory of the left circumflex coronary artery. In addition, the right coronary artery was absent and no visible artery could be shown to supply the sinoatrial node via the substitutional right coronary artery originating from the proximal left anterior descending artery. We postulate that this coronary artery anomaly may have led to hypoperfusion of the sinoatrial node, and hence sick sinus syndrome.

Aside from the coronary artery anomaly, our patient had no structural abnormalities of the heart. She was in euthyroid status and without evidence of virus infection or connective tissue disease that might cause sinus node dysfunction. There was no drug history that might have affected the sinus function. The relatively young age of this patient suggests that degenerative change in the conduction system is less likely to be the cause of the sick sinus syndrome. The coronary artery anomaly in this patient may have led to compromised blood supply to the sinus node, and hence sick sinus syndrome. Therefore, in patients with sick sinus syndrome who are relatively young, coronary angiography should be considered to rule out the possibility of congenital coronary artery abnormalities.
References