Aneurysm of the Left Aortic Sinus Causing Acute Myocardial Infarction

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SUMMARY  A 30-year-old female had an acute myocardial infarction complicated by congestive heart failure. Angiography demonstrated an aneurysm in the area of the left aortic sinus. The aneurysm compressed and displaced the main trunk of the left coronary artery and the proximal portion of the left anterior descending artery. This aneurysm was considered to be the cause of the infarction. Aortic valve replacement and removal of the aneurysm were performed. Postoperative studies revealed good function of the replaced valve, good antegrade filling of the left coronary artery, and improved left ventricular function.

AN ANEURYSM of the aortic sinus is uncommon. The most frequent complications of the aneurysm of the aortic sinus are rupture and development of aorto-cardiac fistula, but until they occur, clinical manifestations are not usually recognized. Single aneurysms originating from the left aortic sinus are rare. An aneurysm of the left aortic sinus is usually combined with aneurysmal dilatation of other sinuses, as in Marfan’s syndrome.

Recently we treated a patient who had an aneurysm originating from the left aortic sinus. This aneurysm compressed and displaced the adjacent left coronary artery, which produced effort angina and resulted in acute myocardial infarction.

Case Report

The patient was a 30-year-old female who had been in good health until 1 year before this episode, when she first exhibited an abnormal chest x-ray on the routine physical check-up. Four months before, she had a single episode of precordial discomfort when she climbed up the stairs; she was otherwise asymptomatic. On March 23, 1982, after she had run about 100 meters, anterior chest pain, cold sweating, nausea and vomiting afflicted her suddenly. She consulted a local practitioner and her condition was diagnosed as acute myocardial infarction by the serial enzymatic and electrocardiographic changes (fig. 1). After she had recovered from the acute stage of myocardial infarction, she was transferred to Kurume University Hospital for further evaluation on May 17, 1982.

Physical examination on admission revealed a slightly emaciated female in mild respiratory distress. The blood pressure was 88/66 mm Hg and the pulse was 82 beats/min and regular. An S, gallop was audible, a grade 2/6 mitral regurgitant murmur was noted at the apex, and a blowing aortic regurgitant murmur was noted at the third left sternal border. Bilateral basal crepitant rales were present. Prolonged circulation time suggested the presence of congestive heart failure. Laboratory data revealed no abnormalities. C-reactive protein and serologic tests for syphilis were also negative. A chest x-ray showed a localized prominent bulge at the upper portion of the left cardiac border and the pulmonary vascular markings were increased (fig. 2). On the ECG, abnormal Q waves were noted in leads I, aV_L, and precordial leads V_1 to V_3, suggesting the presence of old myocardial infarction. Cardiac catheterization showed normal cardiac oxygen saturations and pressures, except for a moderate increase in the left ventricular end-diastolic pressure of 17 mm Hg. Cardiac index obtained by the thermodilution method was 2.8 l/min/m^2. The left coronary cusp injection demonstrated a large, globular aneurysm whose entry was located just below the ostium of the left coronary artery (fig. 3). According to the left coronary artery angiograms, the main trunk of the left coronary artery and the proximal portion of the left anterior descending coronary artery were stretched and displaced posterolaterally. Because the lumens of these displaced vessels were markedly compressed and

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FIGURE 1. The ECG 9 hours after the onset of the chest pain suggested anteroseptal myocardial infarction.
stretched by the aneurysm, injected contrast medium in these areas was thinned and the distal vessels were filled late (fig. 4). The distal vessels of the left coronary artery were also filled retrogradely by the right coronary artery injection (fig. 5). Grade II aortic regurgitation was noted on the aortogram. The left ventriculogram revealed severe anterolateral and apical hypokinesis. The left ventricular ejection fraction was 0.35.

Operative repairs were performed through a median sternotomy. The aneurysm, which was 6 cm in diameter, bulged toward the left side of the aorta, posterior to the main trunk of the pulmonary artery (fig. 6). The aortic valve consisted of two normal-shaped aortic cusps; the third cusp was composed of a small amount of scar tissue, which we considered to be the remnant of the left coronary cusp. The two normal-shaped cusps were of equal size, one on the left and the other on the right. The ostium of the left coronary artery and the mouth of the aneurysm were located above the third cusp, which was located on the posterior part of the aorta (fig. 7). The right coronary artery originated from its normal position. The poor coaptation of the
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Aortic cusps resulted in aortic regurgitation. No thrombus was detected in the aneurysmal cavity.

Operative corrections consisted of aortic valve prosthesis and aneurysmectomy.

The removed tissues were examined microscopically. The wall of the aneurysm was composed of nonspecific fibrous tissues, collagen fibers and fragmented elastic fibers. Normal structure of an arterial wall was not noted. There was no evidence of inflammation or mucoid degeneration (fig. 8).

Postoperative angiograms 2 months after the operation showed normal filling of the coronary arteries and adequate function of the replaced valve. Improved left ventricular function was manifested by the increased cardiac index of 3.9 l/min/m² and the left ventricular ejection fraction of 0.45.

**Discussion**

Thurnam' described an aortic sinus aneurysm in 1840. He named aortic sinuses, according to their relationship to the coronary arteries, as right, left and posterior (noncoronary) sinuses. In this case, the aortic valve consisted of two normal-shaped cusps and the scar tissue between them. The mouth of the aneurysm and the ostium of the left coronary artery consisted of scar tissue, so we assumed that the aneurysm originated from the left aortic sinus.

Aneurysms of the aortic sinus are generally syphilitic, atherosclerotic, mycotic or, rarely, congenital ori-
gin. In the present case, no inflammatory evidences were noted in the aneurysmal wall by the microscopic examinations. The serologic test for syphilis was also negative. Because this aneurysm was accompanied by the congenital abnormality of the aortic valve, we considered this aneurysm to be congenital.

Edwards and Burchell\(^2\) mentioned that the mechanism of the aneurysmal formation of the aortic sinus was considered to result from avulsion of the aortic media from the annulus fibrosa. The theory seems to be the same in all aneurysms of the aortic sinus, regardless of which sinus is involved; but the pathophysiologic characteristics of the aneurysm do differ depending on the sinus involved. Most aneurysms of the right or posterior sinus rupture into a cardiac chamber according to the relationship between the aortic cusp and its related intracardiac structure.\(^3, 5\) However, a small area of the aortic valve occupying the left aortic cusp is exposed to the epicardium, so the aneurysms of the left aortic sinus may protrude to extracardiac area and may rarely reveal the oppressive signs of the left coronary artery.

Reports of coronary arterial compression by aneurysm of the aortic sinus have been rare. Chipps\(^6\) reported this complication in 1941, and recent reports of similar cases were presented by Eliot et al.\(^5\) and Olsen.\(^7\) They reported autopsy findings of the aneurysms and the associated coronary lesions. Garcia-Rinaldi et al.\(^7\) also showed a similar case. In that case, surgical repairs were successful and the patient recovered after the operation.

In our case, the symptom of exertional angina and the presence of delayed antegrade filling of the left coronary artery and retrograde filling of the left coronary artery by the right coronary artery injection suggested that the compression of the left coronary artery by the aneurysm produced significant stenosis of the left coronary artery and apparently resulted in coronary ischemia. On exertion, running in this case, the elevated blood pressure would have caused expansion of the aneurysmal cavity, aggravated the coronary ischemia, and eventually would have produced myocardial infarction.

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