Giant Organized Thrombus in the Left Sinus of Valsalva
Causing Intermittent Left Coronary Obstruction
— An Unusual Case of Acute Myocardial Infarction —

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A 48-year-old Japanese man was admitted to hospital for acute myocardial infarction associated with a giant organized thrombus occupying the left sinus of Valsalva. Cardiac catheterization revealed no organic stenosis in either coronary artery, but left ventriculography and aortography showed a filling defect above the left coronary cusp. Transesophageal echocardiography was immediately performed and showed a round mass filling the left sinus of Valsalva. A solid, round mass approximately 2.5 cm in diameter was removed during emergency surgery and determined to be a thrombus on the basis of microscopic findings. This is the second report of a giant organized thrombus occupying the entire left sinus of Valsalva, obstructing the ostium of the left coronary artery intermittently, and leading to acute myocardial infarction. (Circ J 2004; 68: 795–798)

Key Words: Angiography; Myocardial infarction; Sinus of Valsalva; Thrombus; Transesophageal echocardiography

A cute myocardial infarctions (AMI) are usually caused by the rupture of coronary atheromatous plaque, which can evolve to a completely occlusive thrombus. There have been a few reports of thrombus located close to the ostium of the coronary artery leading to myocardial infarction2–7 and we report an unusual case of AMI caused by a giant organized thrombus occupying the entire left sinus of Valsalva and intermittently obstructing the ostium of the left coronary artery (LCA).

Case Report

A 48-year-old Japanese man came to the emergency room by ambulance in December 1999 because of an initial episode of left anterior chest pain lasting 2 h. He had a history of smoking (30 cigarettes per day) and diabetes mellitus (hemoglobin A1c 7.1%), but he did not have a history of heart disease. He was not taking any medications. In the emergency room, the patient’s pulse was regular at 100 beats/min, and his blood pressure was 124/91 mmHg. Auscultation of the heart and lungs indicated nothing abnormal. Chest X-ray examination showed mild enlargement of the heart, with a cardiothoracic ratio of 54%, but no apparent pulmonary congestion. Electrocardiography showed sinus rhythm, left axis deviation, and ST-segment depression in leads I, II, aVL and V3–6, indicative of an acute coronary syndrome (Fig 1).

Emergency cardiac catheterization revealed neither organic stenosis nor thromboembolism in either coronary

Fig 1. Electrocardiogram obtained in the emergency room shows regular sinus rhythm, left axis deviation, and ST-segment depression in leads I, II, aVL and V3–6.
Fig 2. Emergency cardiac angiography findings. (a) Left coronary arteriogram (left anterior cranial projection). The left coronary artery has no organic stenosis, but there is a filling defect (arrow) in the left sinus of Valsalva. (b) Left ventriculogram (right anterior oblique projection), showing non-opacification (arrow) above the aortic valve and hypokinetic movement at the anterior and apical walls. (c) Ascending aortogram (left anterior oblique projection), showing the same filling defect (arrow). There is no aortic regurgitation.

Fig 3. Multiplane transesophageal echocardiographic findings. (a) Long-axis section at 112° rotation. The mass (arrow) can be seen, just behind the left coronary cusp, with its inhomogeneous, slightly cavernous interior. (b) Oblique short-axis view of the aortic valve showing the mass filling the left sinus of Valsalva (arrow). LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle; Ao, ascending aorta; N, noncoronary cusp; R, right coronary cusp; Th, thrombus.

Fig 4. (a) Surgical findings (right anterior cranial view). A solid mass fills the left sinus of Valsalva. (b) Macroscopically, the removed mass is 2.5 cm in diameter and solid with a rough surface.
artery. However, a filling defect of the sinus of Valsalva was detected during left coronary arteriography (Fig 2a). Left ventriculography (LVG) showed non-opacification above the left coronary cusp (Fig 2b). Aortography (AoG) showed this same defect and no aortic regurgitation (Fig 2c). Multiplane transesophageal echocardiography (TEE) was performed immediately in the catheterization laboratory for further evaluation of the coronary cusp and a slightly mobile, round mass filling the left sinus of Valsalva (Fig 3a,b) was clearly seen. Other than the left coronary cusp being motionless, there were no abnormalities of the aortic valves or cardiac chambers. During catheterization, chest pain recurred intermittently with transient ST-segment deviation and aortic pressure depression.

Seven hours after admission, the patient underwent emergency surgery. A solid, round mass approximately 2.5 cm in diameter occupying the left sinus of Valsalva was seen (Fig 4a). Although the mass loosely adhered to the aortic wall and the commissure of the left coronary cusp and noncoronary cusp, we removed it from the left sinus of Valsalva without difficulty (Fig 4b). The mass did not penetrate into the LCA ostium, and no atherosclerotic lesion was seen in the proximal LCA. After removal of the mass, slight atheromatous changes were observed in the left sinus of Valsalva. The remainder of the aortic wall and valve were nonsclerotic. No other macroscopic abnormalities were found. Microscopically, the mass was determined to be a mixed thrombus consisting mostly of fibrin, erythrocytes, platelets, and neutrophils; bacterial flora and inflammatory changes were not found (Fig 5).

Laboratory data for platelet count and antithrombin III and protein C and S concentrations were normal. Anti-cardiolipin antibodies were negative. Though creatine kinase (CK) was 43 IU/L at admission, follow-up CK and CK-MB just before surgery were elevated to 776 IU/L and 127.2 IU/L, respectively, indicating myocardial infarction.

He had had a prior episode of left lower leg pain upon walking that lasted 2 weeks before admission. Two weeks after the cardiac surgery, we performed arteriography of the left leg, and a filling defect indicative of thromboembolism was observed from the distal popliteal artery to the proximal posterior tibial artery. Follow-up cardiac catheterization revealed no filling defect in the left sinus of Valsalva and no organic coronary artery stenosis. LVG showed slightly hypokinetic movement at the apical wall, and AoG showed aortic regurgitation (Sellers’ grade II), which had not been detected by TEE immediately after surgery.

The patient was discharged in January 2000 and since then he has done well under oral anticoagulation therapy.

Discussion

Thrombosis in the ascending aorta2–5 or in the sinus of Valsalva6,7 associated with AMI is a very rare condition. As much as 150 years ago, Rudolf Virchow pointed out 3 important factors necessary for thrombus formation; namely, abnormalities in blood flow, blood constituents, and the vessel wall, which are now referred to as Virchow’s triad. In recent years, eroded or disrupted atherosclerotic plaques have been recognized as a substrate for thrombus formation, leading to an ischemic coronary artery and cerebrovascular disease8 Nader et al2 and Dik et al3 ascribed the etiology of the thrombus in their respective cases to local erosive lesion and age-related degenerative change of the aortic wall. Fuster et al hypothesized that thrombus with non-disrupted plaque might depend on a hyperthrombogenic states, such as elevated low density lipoprotein, cigarette smoking, hyperglycemia, hemostasis and other conditions that had been associated with increased blood thrombogenicity.9 In the present case, we assume that the thrombus was generated on the atheromatous plaque in the sinus of Valsalva. The present patient had a history of moderate cigarette smoking and diabetes, which might be associated with thrombogenesis. Jobic et al described a similar case of giant thrombus occluding the left coronary ostium in a patient with protein S deficiency; however, the exact mechanism of formation of a giant thrombus in a high-flow environment, such as the sinus of Valsalva in the present case, is not fully known.

One diagnostic clue is the filling defect of the sinus of Valsalva seen during left coronary arteriography. Both LVG and AoG showed this same defect. We considered that filling defect to be a thrombus or tumor rather than an intimal flap because the defect’s movement was unlike that of an intimal flap and because aortic regurgitation, which often appears in ascending aortic dissection, was not detected. However, we consider now that either LVG or AoG is sufficient to rule out an intimal flap and performing a series of both procedures may increase the risk of embolization.

An important feature of the present report is the major role played by TEE in showing the presence of the mass filling the left sinus of Valsalva, as has been reported by other investigators5,6 We provided definitive echographic evidence that a mobile mass provoked intermittent left coronary occlusion. Ito et al reported the use of thrombolytic therapy for fresh thrombus in the ascending aorta1 but we did not consider the mass to be fresh thrombus on the basis of TEE findings and thus surgical treatment was employed. Our experience suggests TEE is particularly useful for showing not only the anatomical structure but also its characteristics when a thrombus or tumor is suspected in the sinus of Valsalva. The commonly accepted therapeutic strategies for thoracic aortic thrombus have not been established yet, and therefore the therapeutic approach will differ according to the characteristics of the thrombus5 The TEE findings can assist in the decision of the therapeutic approach.

The histologic characteristics of the mass were those of
thrombus. Although most often masses on the aortic valve are vegetations or papillary fibroelastoma, there was no clinical microbiologic or histologic evidence of infective endocarditis in the present case.

In conclusion, we report an unusual case of AMI caused by a giant organized thrombus occupying the entire left sinus of Valsalva and intermittently obstructing the ostium of the LCA. Intraaortic thrombus should be considered in the differential diagnosis of the causes of coronary arterial occlusion that may lead to AMI.

References


