Right Sinus of Valsalva Aneurysm Causing Acute Myocardial Infarction

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Figure 1. (A) Right coronary artery (RCA) angiogram. Left anterior oblique view showing a saccular dilatation located at the aortic root in the right sinus of Valsalva. The lower intensity of the contrast in the aneurysmal lumen is due to the presence of thrombus. On the left side of the catheter, a conus branch is visible. (B) Coronary angiography and intravascular ultrasound after manual thrombus aspiration. Coronary angiography showed a stenosis in the RCA ostium. (C) Computed tomography showing thrombotic right sinus of Valsalva aneurysm.
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inus of Valsalva aneurysm (SVA) is a relatively uncommon condition. It may be acquired; most cases are congenital anomalies arising from a defect of the aortic media. The most common site is the right coronary sinus. Although most patients with SVA are asymptomatic, serious complications including rupture to any heart chamber and aortic regurgitation may occur. Acute myocardial infarction (AMI) is a rare complication of ruptured or unruptured SVA. Here, we describe a case of AMI caused by SVA in which the patient underwent emergency coronary intervention followed by surgical repair.

A 40-year-old man who had been in good health presented to hospital with sudden-onset chest pain at rest. Electrocardiography showed ST-segment elevation in leads II, III, aVF, V5, and V6. Echocardiography indicated mild inferior wall hypokinesis and an ejection fraction of 55%. Although a secondary cause of AMI including aortic dissection was suspected, SVA could not be detected at this time. Under a diagnosis of ST-segment elevation AMI, emergency coronary angiography was performed via right radial artery approach after loading doses of dual anti-platelet agents (aspirin 200 mg and prasugrel hydrochloride 20 mg). Because it was difficult to engage the catheter at the right coronary artery (RCA) ostium, we performed right coronary cusp angiography, which showed total occlusion of the RCA at the ostium and aneurysmal dilatation of the right coronary cusp (Figure 1A). Percutaneous coronary intervention was performed with a 6-Fr guiding catheter. Using the floating wire technique, the guide wire crossed the occluded lesion without resistance. After manual thrombus aspiration, Thrombolysis in Myocardial Infarction (TIMI) 2 flow was obtained. Repeated angiography showed thrombus-like shadow of aneurysm. Intravascular ultrasound (IVUS) showed a hematoma-like shadow that compressed the coronary artery wall from outside the artery, resulting in luminal narrowing of the proximal RCA. There was neither atherosclerotic lesion nor plaque disruption of the coronary arterial wall (Figure 1B). After dilatation with a 2.0×15-mm angioplasty balloon, TIMI 3 flow was obtained. Thoracic multi-detector row computer tomography angiography (CTA) was used to confirm the anatomy, and it showed a large SVA compressing the proximal RCA (Figure 1C). Mural thrombus was observed in the SVA. During manipulation of the guiding catheter, a small amount of contrast agent might have been injected into the thrombus, which could have produced the hematoma-like shadow on IVUS. We held a heart team conference with a cardiac surgeon. Although surgical repair is the first-line treatment for SVA, we chose elective surgery because rupture was not imminent. To maintain RCA patency, we carried out coronary stenting (Integrity™ 4.0×26 mm). At the 28th day, the patient underwent surgery with extracorporeal circulation via median sternotomy. After incision of the ascending aorta just distal to the SVA (16×19 mm), SVA with mural thrombus compressing the RCA wall was sighted. The SVA was resected, and repair with a Hemashield patch (Boston Scientific, Natick, MA, USA) was achieved. The proximal RCA was occluded with interrupted stitches and a saphenous vein graft bypass from the aorta to the distal RCA was implanted. After surgery, pathology of the resected SVA showed mural thrombus inside the SVA (Figure 2). It also showed chronic dissection of the SVA with organized thrombus containing fibro-collagenous tissue, lymphocyte infiltration and recanalized lumen.

SVAs rarely present with symptoms unless rupture occurs. SVA is usually congenital and caused by the absence of elastic lamellae of the aortic media, which weakens the aortic wall and leads to aneurysm. SVA is found most often in the right coronary sinus. Approximately 70% of aneurysms occur in the right coronary sinus, 29% in the non-coronary sinus, and only 1% in the left coronary sinus. In the present case, SVA was complicated with inferior AMI due to thrombotic occlusion of the RCA. On IVUS the proximal segment of the RCA was compressed by the large SVA from outside. There are several possible mechanisms for thrombus formation at the stenotic segment of the RCA. First, SVA compression occluded the RCA and facilitated subsequent thrombus formation. Second, the AMI might have been spontaneous and related to plaque disruption at the stenotic segment of the RCA. Although IVUS did not show plaque rupture or ulceration, it is difficult to detect erosion. Finally, mural thrombus inside the SVA might have embolized the stenotic segment of the RCA.

**Figure 1.** Pathology of resected Valsalva aneurysm: the wall of the aneurysm is composed of fibrous tissue, collagen fibers, fragmented elastic fibers and organized thrombus. Organized thrombus containing recanalized lumen and lymphocyte infiltration was located between the media and the external elastic lamina, suggesting chronic dissection that had formed at least 3 months previously. Elastica van Gieson stain.
the RCA.

Although echocardiography is useful for the diagnosis of SVA because it can visualize dilated Valsalva wall and disturbed blood flow within the SVA, we could not detect SVA on echocardiogram in the emergency room. In this case, it was difficult to engage a guide catheter at the RCA orifice, and fortunately the SVA was imaged via the right cusp. Alternatively, aortography using pig-tail catheter located at the aortic root can easily visualize SVA. If patient condition permits, CTA can provide 3-D imaging of the SVA and information on its anatomic relationship to the coronary artery. We should keep in mind that SVA can, albeit rarely, cause AMI, especially inferior AMI.

Disclosures

None.

References