Acute Myocardial Infarction Caused by “Malignant” Anomalous Right Coronary Artery Detected by Multidetector Row Computed Tomography

Minoru Ichikawa, MD; Sei Komatsu, MD*; Hiroshi Asanuma, MD; Akio Iwata, MD; Tamayo Ishiko, MD; Atsushi Hirayama, MD*; Young-Jae Lim, MD; Kazuhisa Kodama, MD*; Masayoshi Mishima, MD

Anomalous coronary arteries are usually identified incidentally by angiography or autopsy, but some “malignant” coronary anomalies are associated with a high incidence of syncope, arrhythmia, myocardial infarction, and sudden death. So far, the pathogenesis of the coronary events in such cases has only been revealed by autopsy. In the present case report, a patient with anomalous origin of the right coronary artery from the left sinus of Valsalva developed acute myocardial infarction, and visualization of the anomaly and assessment of the culprit plaque in the artery were done by multidetector row computed tomography and intravascular ultrasound. (Circ J 2005; 69: 1564–1567)

Key Words: Acute myocardial infarction; Anomalous coronary artery; Multidetector row CT; Plaque map

The incidence of coronary artery anomalies is reported to be about 1% in patients undergoing angiography.1 Origination of the right coronary artery (RCA) from the left sinus of Valsalva is a rare anomaly with an incidence of 0.05–0.10% among all patients having coronary angiography (CAG)2,3 and it has been suggested that such an anomaly with the RCA coursing between the ascending aorta and the pulmonary trunk could be a cause of ischemia or sudden death.4 There are some previous reports of incidentally detecting coronary anomalies during multidetector row computed tomography (MDCT)5–7 but acute coronary syndrome (ACS) associated with “malignant” anomalies has not been reported. We describe a patient with anomalous origin of the RCA in whom ACS occurred. We evaluated the plaque texture using a plaque analyzing system (“Plaque Map”), and compared the results with those from intravascular ultrasound (IVUS).

Case Report

A 47-year-old man with no prior history of cardiac disease developed severe chest pain that persisted for 2 h while he was drinking alcohol. His cardiovascular risk factors included current smoking. The 12-lead electrocardiogram showed ST segment elevation in leads II, III and aVF, so emergency cardiac catheterization was performed. Multiple attempts to catheterize the RCA were unsuccessful, so the possibility of anomalous origin of the RCA was suspected, but aortography failed to reveal the ostium (Fig 1). The left coronary artery (LCA) showed no significant stenosis, and there were no collaterals to the RCA. The patient was treated with recombinant tissue plasminogen activator (1,600,000 IU of Monteplase). His peak creatine kinase level was 5,480 IU/L and the clinical course was uneventful.

To confirm the anomalous origin and course of the RCA, MDCT was performed using a 16-slice scanner (LightSpeed 16, GE Systems, USA) at 2 weeks after admission. The patient was pretreated with metoprolol (20 mg po) 1 h before scanning and with sublingual nitroglycerin 10 min before the procedure. For determination of the circulation time, 15 ml of contrast medium (Optiray 320; Tyco Healthcare Co, Ltd, Japan) was administered into an antecubital vein at 3.5 ml/s and 80 ml of contrast medium was used for MDCT at 3.5 ml/s. The slice thickness was 0.625 mm, pitch 0.3:1, rotation time 0.5 s, temporal resolution 125 ms and the trigger point was 70–80% (R-R). Reconstruction was done by the snapshot burst reconstruction method (retro-gating reconstruction). Cross-sectional images of the coronary arteries were obtained by applying the curved multi-planar reformation technique at intervals of 5 mm and these were analyzed by Plaque Map8

Axial images showed the RCA originating from the left sinus of Valsalva, anterior to the origin of the LCA. Volume-rendered images that were reconstructed on an Advantage Workstation 4.2 (GE Healthcare, WI, USA) showed an anomalous RCA arising from the left sinus of Valsalva and coursing between the aortic root and the pulmonary trunk (Fig 2). The anomalous RCA did not show any kinks or sharp bends and it branched from the aorta at angle of 33°. There was significant stenosis at the midpoint of the RCA.

Three weeks after admission, invasive CAG was attempted again based on the information obtained by MDCT. CAG confirmed that the RCA arose from the left sinus of Valsalva and was contiguous anterior with the LCA ostium (Fig 3A). The culprit lesion showed 75% stenosis. There was Thrombolysis in Myocardial Infarction...
Fig 1. Selective angiogram of the left coronary artery (A) and aortic angiogram (B). The right coronary artery and its origin are poorly visualized.

Fig 2. Multislice computed tomography coronary angiography. The anomalous right coronary artery (white arrows) originates from the left coronary sinus and the culprit region is indicated by the yellow arrow (A). The anomalous right coronary artery runs between the aorta and the pulmonary trunk (B). Ao, ascending aorta; PA, pulmonary artery; RCA, right coronary artery; LCA, left coronary artery.

Fig 3. Selective angiography of the right coronary artery (RCA). Culprit lesion (arrow) (A). Intravascular ultrasound image of the culprit lesion in the RCA shows soft plaque (arrow) (B). Short-axial image of the culprit lesion converted into Plaque Map showing soft plaque of less than 50 Hounsfield unit (HU) (arrow) (C).
grade 3 flow and no thrombus was detected by IVUS. A 6Fr left Amplatz II guide catheter was advanced into the anomalous RCA.

On the IVUS images, the intramural segment showed a reduction in area of approximately 75% compared with the distal reference vessel (Fig 3B). The short-axial image of the culprit lesion was converted into Plaque Map (Fig 3C) and soft plaque of less than 50 Hounsfield unit was detected.

Four months after the onset of infarction, we performed stress/rest single-photon emission computed tomography with technetium-99m tetrofosmin (740 MBq), which revealed reversible ischemia in the inferior left ventricular segments. Six months after onset, we repeated the CAG and IVUS. Because the stenosis had progressed, he underwent percutaneous coronary intervention (PCI: stenting of the anomalous RCA) (Fig 4). One year after the onset, CAG was repeated with provocation testing for coronary artery spasm. Administration of 80±g acetylcholine into the RCA induced spasm in the proximal portion, which was located between the aorta and pulmonary trunk (Fig 5A, B). Therefore, we prescribed calcium-channel blocker to prevent vasospasm, and he remained free from recurrent angina. After discharge, a treadmill exercise test was performed, but no myocardial ischemic change was detected.

**Discussion**

It is quite important for interventional physicians to consider the possibility of coronary anomalies. In the present patient, the ostium of the RCA was not detected by aortography, although thrombolysis might have revealed the masked ostium.

The incidence of an anomalous RCA arising from the left coronary sinus is 0.05–0.10% among patients undergoing CAG, and it is a potentially serious anomaly that can cause syncope, exercise-induced arrhythmia, myocardial infarction (MI), or sudden death. However, the pathophysiologic mechanisms underlying the sudden onset of myocardial ischemia in these patients have not been clarified.

There are 3 subtypes of anomalous origin of the RCA from the left sinus of Valsalva, based on the path of the anomalous artery; that is, interarterial, retro-aortic, and anterior to the pulmonary trunk. The present patient had the interarterial subtype, which is a so-called “malignant anomaly” associated with a high risk of exercise-induced ischemia, MI, and sudden cardiac death. Several theories have been proposed to explain the occurrence of these associated adverse events:

1. Coronary spasm in the proximal portion of the anomalous coronary artery located between the aorta and pulmonary trunk could occur if there is a critical reduction in coronary arterial flow, resulting in ACS.
2. Compression of the proximal anomalous coronary artery by the aorta and pulmonary artery may occur, especially while drinking alcohol or during exercise because of the aortic and pulmonary dilatation in response to the increased cardiac output, combined with increased coronary flow caused by myocardial demand.
3. Limitation of blood flow by a kink or bend at the aberrant origin of the artery.
4. Narrowing of the slit-like, small ostium of the aberrant artery.
5. Intramural course of the proximal portion of the...
RCA within the aortic wall.\textsuperscript{14} In the present patient, the results of IVUS and CAG suggested that the first mechanism may have been responsible. The culprit lesion consisted of a soft plaque, but there was no sign of rupture on either MDCT or IVUS. Moreover, the anomalous RCA did not show any kinks or sharp bends and it branched from the aorta at an angle of 33°. It is possible that the ostium of the RCA was compressed while the patient was drinking alcohol, because of the marked augmentation of cardiac contractility and cardiac output, so that RCA flow slowed and acute thrombosis occurred at the stenosis in association with dehydration. This thrombus might have masked the ostium of the RCA.

Furthermore, the culprit lesion was remote from the RCA orifice and pulmonary artery – ascending aorta portion, and as there was no evidence of plaque rupture we speculate that vasospasm at the culprit lesion rather than compression of the proximal RCA may have cause the acute MI.

In this patient, the anomalous artery ran between the aortic root and the pulmonary trunk. Although the culprit lesion was not located at the ostium, the clinical course suggests that this type of anomaly is “malignant” and therefore associated with a high risk of MI. Accordingly, aggressive treatment was justified, including the PCI. The presence of viable myocardium was suggested by the existence of reversible ischemia in the territory of the RCA on myocardial perfusion single-photon emission computed tomography. There have been other reports of PCI in patients with an anomalous RCA arising from the left sinus of Valsalva.\textsuperscript{15,16} and we selected this treatment because of the occurrence of ischemia.

In conclusion, MDCT was effective for both examining the plaque morphology and characterizing the coronary artery anomaly. It is a non-invasive method of determining the PCI strategy by MDCT “angiologist” and, moreover, pixel analysis \textit{in silico} by Plaque Map it can assist in anticipating coronary events or sudden death associated with malignant coronary anomalies.

References