CASE REPORT

Congenital Absence of the Left Circumflex Artery Associated with Inferior Myocardial Infarction

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Abstract

Congenital absence of the left circumflex artery is a rare anomaly of the coronary arteries. A 52-year-old man who developed acute inferior myocardial infarction underwent coronary angiography which revealed the absence of the left circumflex artery and that the surrounding structures were supplied by the infarct-related super-dominant right coronary artery. Two stents were implanted into the right coronary artery and one stent into the mid portion of the left anterior descending artery. Follow-up coronary angiography at 67 months showed no detectable restenosis, and 64-slice multidetector computed tomography confirmed the absence of the left circumflex artery. The circumflex artery as a terminal extension of a culprit right coronary artery has not been previously reported.

Key words: coronary artery, congenital anomaly, coronary angiography, computed tomography

(Intern Med 51: 71-74, 2012)
(DOI: 10.2169/internalmedicine.51.6141)

Introduction

An anomalous course of coronary arteries is observed in 0.3–1.3% of patients undergoing diagnostic coronary angiography (1, 2). Congenital absence of the left circumflex coronary artery (LCx) is a very rare vascular anomaly (3, 4). We report a case of inferior acute myocardial infarction (AMI) in a 52-year-old man in whom the absence of the LCx arose as an extension of the distal culprit super-dominant right coronary artery (RCA).

Case Report

A patient with a history of hypertension and heavy smoking presented to a local hospital within 4 h of symptom onset. The complaint was substernal chest pain associated with shortness of breath, diaphoresis and nausea. Electrocardiography (ECG) showed ST-segment elevation in leads V3 through V6, and a Q wave in leads II, III and aVF without ST-segment elevation (Fig. 1A). Maximum values of creatine kinase (CK), CK-MB and troponin T were 6,258 IU/L, 332 IU/L and >2.0 ng/mL (normal <0.03 ng/mL), respectively.

Coronary angiography performed with a 6Fr diagnostic catheter via the right femoral artery revealed 90% stenosis in the mid-portion of the left anterior descending artery (LAD) and retrograde filling of the LCx territory (Fig. 1C). The LCx could not be visualized in its typical location. The RCA originated from the right sinus of Valsalva. This was a large super-dominant artery with 90% stenosis in mid-vessel and 90% narrowing of the posterior descending artery (PDA) with TIMI grade 2 flow. It crossed the crux of the heart that ascends to the left atrioventricular groove and perfused the zone extending to the LCx territories (Fig. 1C).

Upon hospital admission, his blood pressure was 90/60 mmHg with a pulse rate of 100 beats/min. ECG showed ST-segment elevation in leads V6 through V6, and a Q wave in leads II, III and aVF without ST-segment elevation (Fig. 1B). Maximum values of creatine kinase (CK), CK-MB and troponin T were 6,258 IU/L, 332 IU/L and >2.0 ng/mL (normal <0.03 ng/mL), respectively.

Coronary angiography performed with a 6Fr diagnostic catheter via the right femoral artery revealed 90% stenosis in the mid-portion of the left anterior descending artery (LAD) and retrograde filling of the LCx territory (Fig. 1C). The LCx could not be visualized in its typical location. The RCA originated from the right sinus of Valsalva. This was a large super-dominant artery with 90% stenosis in mid-vessel and 90% narrowing of the anterior descending artery (PDA) with TIMI grade 2 flow. It crossed the crux of the heart that ascends to the left atrioventricular groove and perfused the zone extending to the LCx territories (Fig. 1C).

There was no angiographic evidence of a separate ostium of the LCx after aortography. The patient was given 300 mg clopidogrel and 300 mg aspirin before percutaneous coronary intervention. A bare metal stent (2.5×25 mm) was deployed to the posterior descending artery with TIMI grade 1 flow. Subsequently, another bare metal stent (4.0×20 mm)
was implanted into the mid-portion of the RCA with TIMI grade 2 flow. As the patient was hemodynamically unstable, another bare metal stent (2.75×25 mm) was implanted into the mid-LAD. TIMI grade 3 flow was achieved at the end of the procedure (Fig. 2A-D).

Echocardiography showed severe hypokinesis of the lateral wall, inferior left ventricular wall thinning and akinesis and slightly reduced left ventricular systolic function with left ventricular ejection fraction (LVEF) 52.8% and moderate mitral regurgitation 2 days after percutaneous coronary intervention. The patient was discharged on day 7 with prescription medication: aspirin, clopidogrel, Fosinopril, metoprolol and atorvastatin. Follow-up coronary angiography at 67 months showed no detectable restenosis. The absence of the LCx was confirmed on 64-slice multidetector computed tomography (MDCT) (Fig. 3A-D).

**Discussion**

Studies have shown that the LCx can arise anomalously from the RCA, right sinus of Valsalva, separately from the left sinus of Valsalva or, very rarely, from the pulmonary artery (5). The absence of the LCx is a very rare congenital anomaly with a frequency of only 0.003% in all patients who undergo coronary angiography (2).

In the absence of LCx with superdominant RCA, when acute occlusion of RCA occurs, it usually results in inferior, posterior and lateral wall myocardial infarction which is equivalent to two-vessel coronary artery disease involving both the RCA and LCx. However, in the present case, the first ECG showed ST-segment elevation in leads III and aVF with concomitant ST depressions in leads I and aVL. This pattern of ECG can be explained by the following reasons: (a) Conventional ECG findings used to distinguish infarct-related artery are less useful in patients with simultaneous occlusion of RCA and LCx. Pollak et al described ECG characteristics of simultaneous occlusions of the RCA and LCx, two-thirds of the patients had isolated inferior ST-segment elevation (6). (b) Good collateral from LAD to the territory of LCx may explain the absence of ST-segment elevation in leads V1 and V2. The second ECG revealed that ST-segment elevation in leads V1 through V6, and a Q wave in leads II, III and V5. The following findings may explain this ECG pattern: coronary angiography revealed restored...
Figure 2. A: Coronary angiography with a 6Fr diagnostic catheter reveals an intraluminal thrombus and a severe stenotic lesion in the mid-RCA with TIMI grade 2 flow. B: 7Fr Judkins right 4.0 guide catheter was used in RCA, final results of RCA post stenting with TIMI grade 1 flow. C: left coronary angiography with a 6Fr diagnostic catheter in the anteroposterior projection shows severe stenosis in the mid-LAD. D: 7Fr Judkins left 4.0 guide catheter was used in the left coronary artery, TIMI grade 3 flow was achieved in mid-LAD after stent implantation.

Figure 3. Follow-up coronary angiography with a 5Fr diagnostic catheter via the right radial artery at 67 months shows no significant restenosis at the site of stenting in the LAD (A) and RCA (B). The angiographic image of 64-slice MDCT reveals the absence of the left circumflex artery and a super-dominant RCA with large posterolateral branches (C and D). MDCT: Multidetector computed tomography.
flow in the RCA that lead to ST resolution of leads III and aVF. In addition to the culprit RCA, LAD also had a severe stenosis in the mid portion; LAD flow and collateral flow from LAD to the territory of “LCx” was reduced by hemodynamic instability and hypotension and resulted in ST elevation in leads V1 through V6.

In patients with coronary artery disease, if the LCx cannot be visualized during coronary angiography, an ostial total occlusion or congenital agenesis may be suspected. In the present case, collateral filling of the LCx territory contributed the distal portion of RCA. The absence of the stump of the ostium of the LCx could not support the notion that the LCx was occluded. Absence of the LCx was also confirmed by MDCT angiography. Coronary MDCT is a useful tool to depict anomalies of the coronary artery with a high accuracy. MDCT provides high-resolution three-dimensional (3D) data sets that allow precise definition of 3D spatial relationships of the anomalies (7). Unfortunately, we could not carry out intravascular ultrasound to support the hypothesis of congenital agenesis.

Heavy smoking and hypertension probably contributed to the development of coronary atherosclerosis in the present patient. In addition, depressed coronary flow reserve of the distal end of the RCA may also be a risk factor for ischemic heart disease (8).

The absence of the LCx, and continuation of the dominant RCA as the LCx along the entire length of the atrioventricular groove is, in general, thought to have a benign outcome unless atherosclerotic coronary artery disease is superimposed (9). However, in the present case, RCA was the culprit vessel and a branch of RCA covered the territory of the LCx. It is crucial to recognize this condition when undertaking coronary angiography to determine the appropriate treatment since the patients are critically ill.

To the best of our knowledge, this is the first report of a large RCA as a culprit vessel supplying the LCx territory with the absence of the LCx.

The authors state that they have no Conflict of Interest (COI).

References


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