CASE REPORT

Left main coronary artery aneurysm with chronic total occlusion of both left coronary arteries in a young athlete

S-H Park, S-E Kim, S K Ryu

Abstract

Aneurysms of the left main coronary arteries are found in 0.1% of angiograms. This case involves an athlete with a left main coronary artery aneurysm, which was combined with chronic total occlusion of the proximal left anterior descending and proximal left circumflex coronary arteries. The extraordinary clinical presentation in this patient may be associated with good coronary collaterals, which may have developed in the patient in response to chronic total occlusion of the coronary artery by the aneurysm, and repeat myocardial hypoxia during high levels of performance as a soccer player.

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Left main (LM) coronary artery aneurysm is very rare, occurring in 0.1% of adults undergoing coronary angiography.¹ Although some of these cases had associated multivessel coronary artery disease, to the best of our knowledge there has been no previous report of an LM coronary aneurysm associated with chronic total occlusion of the left anterior descending (LAD) and left circumflex (LCx) arteries at the ostium. Such findings in a young soccer player,

together with a review of the possible causes, are presented here.

Case report

A 31 year old man was admitted to the coronary care unit following a non-Q wave myocardial infarction which had occurred two hours earlier. He had been a soccer player since childhood and had played regularly up until a year ago. He had no risk factors for coronary artery disease and no unusual history. His physical examination was normal. Laboratory examinations, including erythrocyte sedimentation rate, C reactive protein, complete blood count, serologic test for syphilis, rheumatoid factor, antinuclear antibody, antithrombin III, protein C, and protein S, were normal. Echocardiography showed a greatly dilated LM coronary artery and mild hypokinesia at the left ventricular apex. Fluoroscopy and coronary angiography revealed a calcified giant aneurysm of the LM artery with total occlusion of the LAD and LCx arteries at the ostium (fig 1), with good collaterals from the normal right coronary artery (RCA). Angiographies, including the cerebrovascular and peripheral arteries, were normal. Electron beam computed tomography (EBCT) confirmed the presence of a 3.0 × 1.5 cm lobulated cystic lesion with a calcification rim from the LM to the LAD and LCx coronary arteries (fig 2A). Intravascular ultrasonography (IVUS) revealed a fusiform giant aneurysm of the LM artery (fig 2B) and a severe intimal thickening at the ostium of the LAD artery with calcification (fig 2C). The patient underwent coronary artery bypass surgery and was discharged in a good condition.

Discussion

Aneurysms of the LM coronary artery are rare.¹ Most reported cases of LM artery aneurysms were incidentally noticed at coronary angiography for evaluation of myocardial ischaemic symptoms. As our case presented with a small myocardial infarction, considering the regional wall motion abnormality detected at echocardiography, the infarction seemed to be caused by inadequate flow to the mid to distal LAD artery through the collaterals. LM aneurysms in older patients are more likely to be caused by coronary atherosclerosis. Other possible causes include Kawasaki disease,

Division of Cardiology,
College of Medicine,
Ewha Womans
University, Seoul,
Korea
S-H Park
S-E Kim

Division of Cardiology,
College of Medicine,
Yonsei University,
Seoul
S K Ryu

Correspondence to:
Dr Si-Hoon Park, Ewha
Mokdong Hospital
Cardiovascular Center, Ewha
Womans University
MokDong Hospital, 911-1,
MokDong, Yangcheon-Ku,
Seoul, Korea
sihoon@mm.ewha.ac.kr

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Figure 1 (A) Angiogram showing giant aneurysm of the LM coronary artery with total occlusion of the LAD and LCx coronary arteries. (B) Angiogram showing good collateral flows (double arrow) from the RCA to LAD (single arrow) and LCx (arrow head) coronary arteries.

Figure 2 (A) EBCT reveals 3.0 × 1.5 cm sized giant aneurysm with calcification rim from the LM to LAD (black arrow) and LCx coronary arteries at the ostium (white arrow). Intravascular ultrasonography shows enlarged external elastic laminae of LM artery (B) with some intimal plaque and calcification, and severe intimal thickening and calcification at the ostium of the LAD coronary artery (C).

arteritis, mycotic-embolic disease, dissection, congenital malformation, and connective tissue disorders. Our patient's young age, lack of coronary risk factors, and absence of coronary lesions except the bifurcation of the LM artery did not point to atherosclerosis as a cause. With no systemic involvement Takayasu's disease was excluded, and negative serologic tests ruled out connective tissue disease. Lack of a history of any obvious condition excluded traumatic, infectious, or inflammatory causes. In patients with an aneurysm caused by Kawasaki disease the LM coronary artery is more frequently involved compared to patients with atherosclerotic coronary aneurysms (42% *v* 4%).² Serial coronary angiography studies have suggested that coronary aneurysms could regress or persist, or develop into stenosis of the vessel at the outlet of the aneurysm as a result of myointimal proliferation provoked by an inflammatory insult during the acute phase of Kawasaki disease.³ Collaterals and calcification are frequently associated with coronary lesions in Kawasaki disease. Although we could

not determine a history of childhood Kawasaki disease in our patient, we believe that it might have been responsible for the insult to the bifurcation site involving the LM, proximal LAD, and proximal LCx coronary arteries, which led to aneurysmal change of the LM artery and total occlusion of the proximal LAD and LCx arteries, and concurrent formation of a collateral circulation over the years.⁴ IVUS and EBCT findings for Kawasaki disease included enlarged vessel size—that is, increased external elastic laminae, myointimal hyperplasia, and superficial calcification.⁵ IVUS and EBCT findings suggest chronic occlusion of the lesion site in our patient.

This is the first case of an athlete with chronic total occlusion of the LAD and LCx coronary arteries. The extraordinary clinical presentation in this patient may be associated with good coronary collaterals. Chronic total occlusion would have contributed to the formation of good collaterals.⁴ Conversely, repeated myocardial hypoxia during high performance as an athlete might have contributed to coronary collateralisation, although the effects of exercise on coronary collateralisation are controversial.⁶ Our case highlights the fact that coronary artery disease can be present in any individual, even young, fit sportsmen used to performing to the highest levels.

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