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Type 1 Giant Coronary Aneurysm

CASE REPORT

Tahir Durmaz[1], Cemal Köseoğlu[2], Hüseyin Ayhan[1], Telat Keleş[1], Engin Bozkurt[1]

ABSTRACT

Coronary artery aneurysm is a rare anomaly and is defined as the expansion of the diameters of normal coronary arteries by ≥1.5 times. Aneurysms reaching 4 times the normal size or 8 mm in diameter are defined as giant coronary aneurysms. Giant aneurysms involved in all of the left main coronary artery (LMCA), left anterior descending artery (LAD), and right coronary artery (RCA) are very rare. Here we report a case of a 47-year-old man presenting with non-ST elevation myocardial infarction and LMCA, RCA, and LAD coronary giant aneurysm as well as spontaneous dissection of the distal LAD.

Keywords: Coronary Artery Aneurysm, non-ST elevation myocardial infarction, giant aneurysm

INTRODUCTION

Coronary artery aneurysms (CAAs) are a rarely seen anomaly and its angiographic incidence has been reported to vary from 1.5% to 4.9% in the literature (1). It is described as the expansion of the coronary artery diameter by 1.5 times or more and is classified into two types: fusiform and saccular (1, 2). Its most commonly located in the right coronary artery (RCA), followed by the circumflex artery (Cx), and left anterior descending artery (LAD) (1, 3, 4).

Here we present a case of a 47-year-old man whose coronary angiography (CAG), which was performed because of non-ST elevation myocardial infarction, revealed giant aneurysmal dilatation in the left main coronary artery (LMCA), LAD, and RCA.

CASE REPORT

A 47-year-old man with the risk factors for diabetes and hypertension was admitted to the coronary intensive care unit because of the complaint of chest pain radiating to the neck as a pressure sensation lasting for 2 h. No pathological finding was found on physical examination and electrocardiography. The patient was thought to have non-ST elevation myocardial infarction because of progressive increase in troponin I levels. In the echocardiography, hypokinesia was detected in the middle of the anterior septum and the middle and apex of the anterior region. Ejection fraction was measured at a rate of 45%. The patient was taken to the coronary angiography unit and selective right and left coronary angiography was performed. Coronary aneurysms were observed in the LMCA, LAD, and RCA (comparable with a diagnostic catheter). Spontaneous dissection without inhibiting the flow after LAD diagonal 2 was found (Figures 1-3). No intervention was planned because he was hemodynamically stable; LAD flow was well but his thrombus embolization risk was high. The patient's follow-up examinations in the hospital revealed no additional pathological findings and he was discharged from the hospital after having been decided that he would be given anticoagulant therapy following a dual antiplatelet therapy for 1 year.

DISCUSSION

CAA has been described as the expansion of the diameter of the artery by ≥1.5 times (1). In some sources in the literature, aneurysms reaching 4 times the normal size or 8 mm in diameter have been defined as giant coronary aneurysms. Post-mortem CAA was first detected by Morgagni in 1760 and was defined by Bourgen in 1812 (5, 6).

Regarding CASS studies, one of the greatest studies conducted till date reported the frequency of CAA as 4.9% (7). Baron et al. (8) reported that CAA was often located in RCA at the rate of 96%, in Cx at the rate of 75%, and in LAD at the rate of 57%. Aneurysms, which are classified as fusiform and saccular generally, were classified into four groups by Markis et al. (9) type 1, with diffuse aneurysmal dilatation in 2-3 veins; type 2, with diffuse involvement in a single vein and local involvement in other veins; type 3, with diffuse involvement in a single vein; and type 4, with local involvement in a single vein. The present case was consistent with type 1.

[1]Department of Cardiology, Yıldırım Beyazıt University Faculty of Medicine, Ankara, Turkey

[2]Department of Cardiology, Ankara Atatürk Training and Research Medicine, Ankara, Turkey

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Correspondance
Hüseyin Ayhan MD, Department of Cardiology, Yıldırım Beyazıt University, Ankara, Turkey
Phone: +90 312 291 25 25
e.mail: huseyinayhan44@yahoo.com

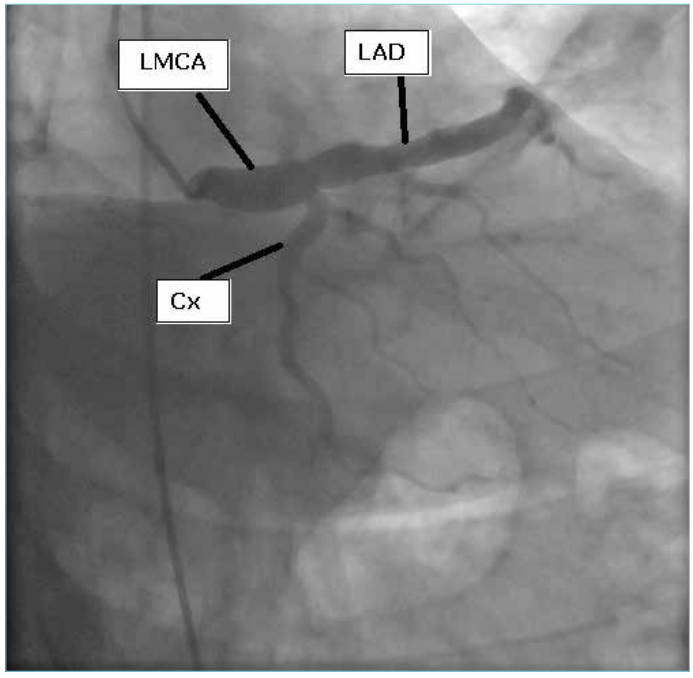

Figure 1. Right caudal angiographic image of a giant coronary aneurysm in the LMCA and LAD (LMCA; Left main coronary artery, LAD; left anterior descending artery)

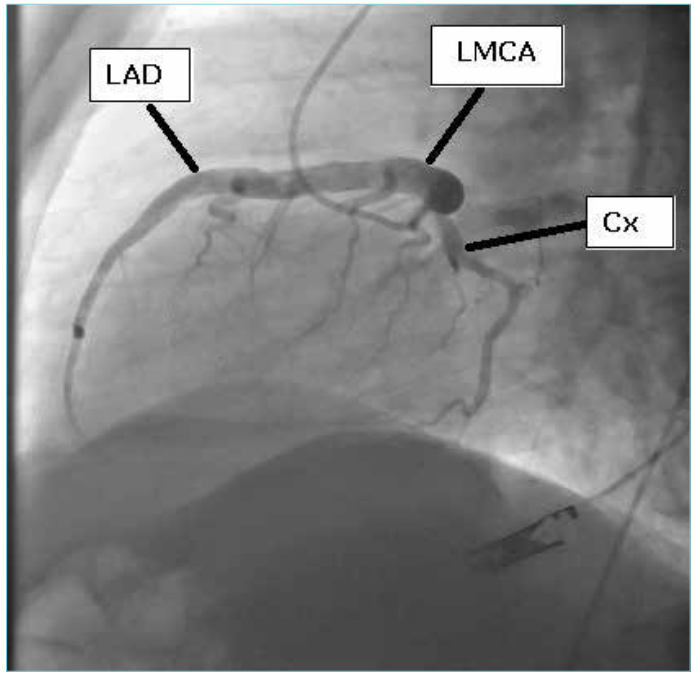

Figure 2. Left lateral angiographic image of a giant coronary aneurysm in the LMCA and LAD (LMCA; Left main coronary artery, LAD; left anterior descending artery)

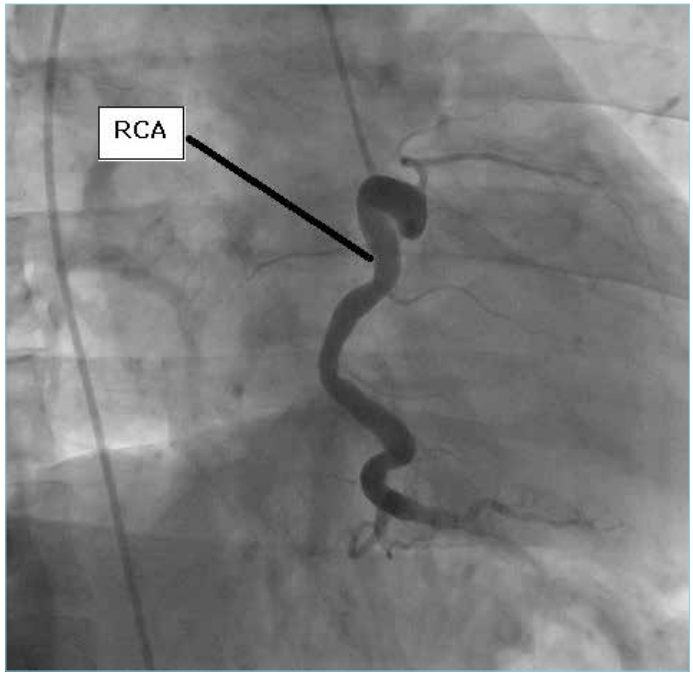

Figure 3. Right oblique angiographic image of a giant coronary aneurysm in the RCA (RCA; Right coronary artery)

Its etiology has atherosclerotic, nonatherosclerotic, and congenital causes, but atherosclerosis was thought to be the most common cause (6, 8, 9). Some causes, including congenital disorders, rheumatic fever, systemic lupus erythematosus, Kawasaki disease, syphilis, Ehlers-Danlos syndrome, Marfan syndrome, infective endocarditis, iatrogenic disease, Osler-Weber-Rendu syndrome, mycotic arthritis, and trauma, have been reported in the literature (6, 8, 9-12). Markis et al. (9) specified that increased intraluminal pressure against blood vessel walls with a thinned media layer and decreased tolerance to stress due to aterosclerosis could lead to aneurysms. Arslan et al. (13) showed the risk factors, including genetic predisposition, hypercholesterolemia, and diastolic hypertension, as an answer to the question why constriction occurs in some vessels and aneurysm in others because of atherosclerosis.

Most of patients with CAA are asymptomatic and can also present with stable angina pectoris and acute coronary syndrome. It is suggested that CAAs can cause ischemia and myocardial infarction by leading to slow coronary flow, turbulent flow, in-situ thrombosis, spontaneous dissection, and microembolus (6, 11, 13, 14). The most important predictor of myocardial infarction is the diameter of the aneurysm (15, 16). In the present case, the giant aneurysm led to non-ST elevation myocardial infarction. In general, CAAs have relatively thick walls and their rupture risks are low but their coexistence with myocardial ischemia is frequent. In addition, giant CAAs can affect the mediastinum like a mass, can cause superior vena cava syndrome, and increase rupture risks (17). The rupture risk increases with age, especially after 40 years (18).

The prognosis of giant CAAs is unclear; however, the 5-year survival rate has been reported as 71% in the literature (6). No obvious treatment is available. There are many treatment alternatives such as medical therapy, stent implantation, and surgical excision according to the symptoms, etiology, and lesion (15, 19). Antiplatelet and anticoagulant therapies are recommended as medical treatment. In the present case, we also planned to administer dual antiplatelet therapy for at least 1 year and then anticoagulant therapy, because the patient had acute coronary syndrome. There are some studies suggesting aneurysm ligation or coronary by-pass without ligation (7).

CONCLUSION

Here we present a case of giant coronary type 1 aneurysm that developed in the LMCA, RCA, and LAD as fusiform and constituted the manifestation of acute coronary syndrome. It has been emphasized that coronary angiography should be performed carefully and unnecessary interventions should be avoided for such patients. Moreover, we attempted to underline the importance of antiplatelet and anticoagulant therapies.

Informed Consent: Written informed consent was obtained from patient who participated in this study.

Peer-review: Externally peer-reviewed.

Authors' contributions: Conceived and designed the experiments or case: EB, TD. Performed the experiments or case: CK, HA. Analyzed the data: TK, TD, HA. Wrote the paper: EB, TD, CK. All authors read and approved the final manuscript.

Conflict of Interest: No conflict of interest was declared by the authors.

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