Case report
Olgu sunumu

KORONER ARTER EKTAZİSİ: OLGU SUNUMU

CORONARY ARTERY ECTASİA: A CASE REPORT

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Öz

Amaç: Koroner arter ektazi (KAE), koroner arterlerin lokal veya yaygın dilatasyonu olarak tanımlanır. Ektatik segment, komşu normal koroner arter segmentine kıyasla en az 1,5 kat genişlemelidir. Biz bu vakamızda 2 damarda ve en geniş yerinde 6,8mm ölçülen KAE vakası sunduk. Bu vakada ateroskleroza sekonder KAE olduğunu düşündük. KAE tedavisinde kesin bir öneri olmamasına rağmen, uygun durumlarda girişimsel tedavi ve tıbbi tedavi önerilmektedir. Anjiyografi sonucuna göre medikal tedavi kararı aldık. Tedavi kararı KAE etiyolojisine, hastanın özelliklerine ve anjiyografi sonuçlarına göre verilmelidir.

Anahtar Kelimeler: Ateroskleroz, koroner arter hastalığı, koroner arter ektazi

Abstract

Aim: Coronary artery ectasia (CAE) is defined as the local or diffuse dilatation of the coronary arteries. The ectatic segment should expand at least 1.5 times compared to the adjacent normal coronary artery segment. In this case, we presented a case of CAE measuring 6.8mm at its widest point with two coronary arteries involved. In this case, we considered CAE secondary to atherosclerosis. Although there are no definitive suggestions in CAE treatment, in appropriate cases, interventional treatment and medical treatment are recommended. According to the angiography result, we made a medical treatment decision. The treatment decision should be made according to the etiology of CAE and the properties of the patient and angiography results.

Keywords: Atherosclerosis, coronary artery disease, coronary artery ectasia

Introduction

Coronary artery ectasia (CAE) is defined as the local or diffuse dilatation of the coronary arteries. Ectatic segment should be at least 1.5 times dilated compared to the adjacent normal coronary artery segment. CAE prevalence is between 1.2-4.9%[1] .Proximal and middle segments of the right coronary artery (RCA) are the most common locations of the CAE. This is followed by the left anterior descending (LAD) artery and the circumflex (Cx) artery[1-4]. In its etiology, 50% atherosclerosis, 20-30% congenital, 10-20% inflammatory or connective tissue disease is responsible[5]. Mostly due to atherosclerosis, excessive remodeling occurs as a response to atherosclerotic plaque growth[6]. CAE has also been shown to be associated with coronary artery disease risk factors such as hypertension, hyperlipidemia and smoking[7].

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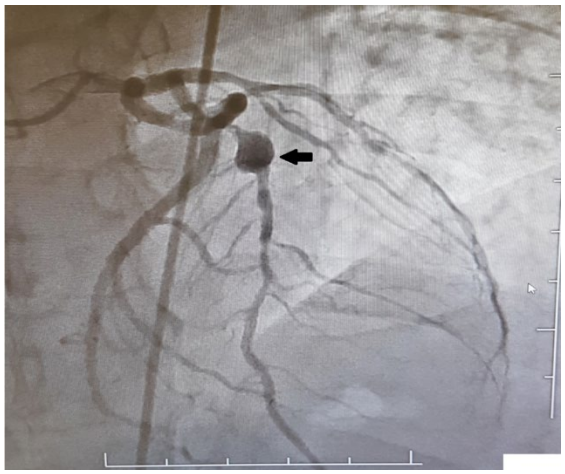


Figure 1 The ectatic segment of the LAD artery (black arrow). The diameter of the segment is 6,8 mm at the widest part.

In this case report; we present a male patient with very wide segmental left anterior descending artery (LAD) and circumflex (Cx) artery ectasia possibly due to atherosclerosis.

Case Report

A 52-year-old male patient admitted to cardiology outpatient clinic with angina symptoms. Informed consent was obtained from the patient. The patient had no known systemic disease. The patient was an active smoker and had a family history of coronary artery disease. The electrocardiogram was at 80 bpm with sinus rhythm and there was no pathology. The blood pressure was 125/80 mmHg and his physical examination was normal. Cardiac auscultation was normal with no additional heart sounds. In echocardiography; the ejection fraction was 65% and no stenosis or regurgitation was seen at color Doppler ultrasound. The treadmill stress test was positive and coronary angiography was planned.

There was a very large ectatic lesion at the proximal part of the LAD artery (Figure 1). The largest diameter of the ectatic lesion was 6.8 mm. There was 50% stenosis at the proximal and mid part of the LAD artery. There were atherosclerotic plaques all along the LAD artery and its branches.

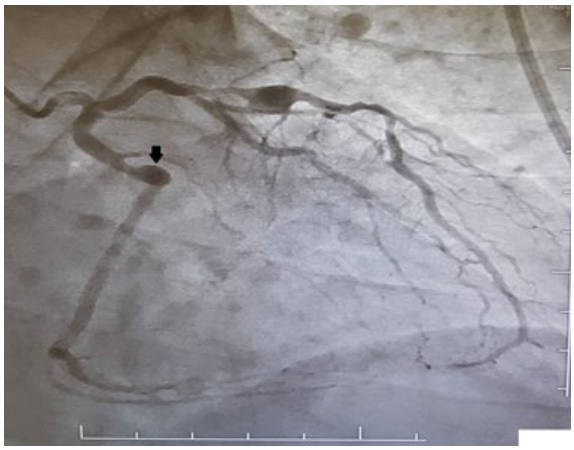
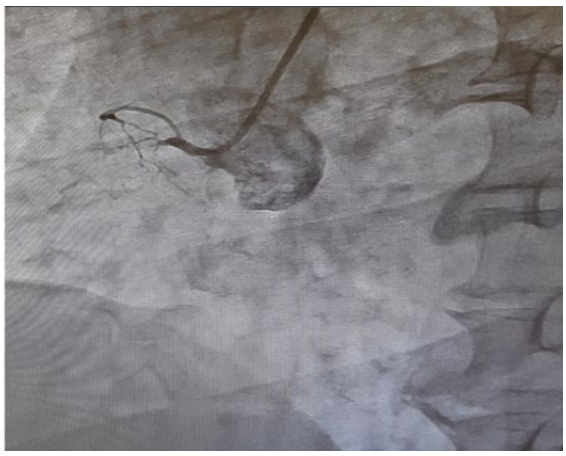


Figure 2 The ectatic segment of the Cx artery (black arrow). The diameter of the segment is 4.13 mm at the widest part.

There was an ectatic lesion after OM1 branch at Cx artery. The ectatic part's diameter was 4.13mm at the most dilated section (Figure 2).

There were atherosclerotic plaques at Cx and all its branches. There was %90 stenosis at the proximal part of the OM1 branch but the diameter of the vessel was <2mm.

There was total occlusion at the proximal part of the RCA (Figure 3).



Figüre 3 Total occlusion was present at the proximal part of the RCA (black arrow).

After the council with cardiology and cardiovascular surgery, medical follow-up decision was taken and medical treatment of the patient was arranged. The patient was started on acetylsalicylic acid, angiotensin converting enzyme inhibitor, beta blocker and statin therapy.

Discussion

CAE is defined as the ectatic segment diameter being more than 1.5 times larger than an adjacent healthy reference segment[1]. In CAE, progressive dilatation of the coronary artery segment is observed with the destruction of the arterial media and thinning of the arterial wall[8]. Most CAE cases are considered a variant of coronary artery disease[9]. At 50% of cases, atherosclerosis is responsible for the dilatation of the coronary artery[5].In our case the patient did not have a history of congenital, inflammatory or connective tissue disease. All coronary arteries have prevalent atherosclerotic plaques and stenosis. The ectasia was most probably caused by the atherosclerosis underneath.

CAE can be classified according to shape or the extent of involvement of the coronary arteries. Classification based on shape can be saccular or fusiform. In saccular CAE transverse diameter is greater than the longitudinal diameter and in fusiform CAE, transverse diameter is less than longitudinal diameter[4]. According to this classification our case is an example of saccular CAE.

Table 1 Markis Classification for coronary artery ectasia

Types	Definition
Type 1	Diffuse ectasia of two or three vessels
Type 2	Diffuse ectasia in one vessel and localized disease in another
Type 3	Diffuse ectasia in one vessel only
Type 4	Localized and segmental involvement

Markis classification classifies the CAE according to the coronary artery involvement (Table 1)[10]. According to this classification, our case is an example of Type 4.

Although there are recommendations for the treatment of CAE, studies related to its treatment are not sufficient. Since CAE is usually accompanied by atherosclerotic lesions, aspirin use is recommended[10]. There are situations where anticoagulation is recommended for ectatic segments due to slow coronary flow but the evidence is not sufficient[11]. Angiotensin converting enzyme inhibitors and statin may be useful to prevent disease progression[12,13]. Nitrates and nitroglycerin treatments are not recommended in these patients because they may induce angina pectoris[14]. Invasive treatment is also recommended with caution because there is not enough proof for its success[15]. In symptomatic patients who are not suitable for percutaneous coronary intervention, ligation or excision of ectatic segment combined with bypass grafting of the affected coronary arteries may be the treatment of choice[16,17].

Recommendations

Atherosclerosis is largely responsible for the etiology of CAE. In our case report, we observed coronary ectasia measured at 6.8 mm at its widest point due to atherosclerosis. Although there are no definitive suggestions in its treatment, in appropriate cases, interventional treatment and medical treatment are recommended. Our case was not amenable to interventional treatment and therefore only medical treatment decision was taken.

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None.

Conflict of Interest

None.

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