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## Case Report

# Multiple Giant Coronary Arterial Aneurysms Leading to Stable Angina

## Case Report

Coronary artery aneurysm (CAA) is defined as abnormal dilatation of a coronary artery luminal diameter to 1.5 to 2 times wider than the adjacent normal segment. Giant coronary artery aneurysms are rare, with a reported prevalence of 0.02% to 0.2% [1]. Most of the giant coronary artery aneurysms are asymptomatic, but some patients present with angina pectoris, sudden death, fistula formation, pericardial tamponade, compression of surrounding structures, or congestive heart failure.

A 61-year-old man referred to our outpatient clinic with stable angina pectoris lasting approximately 3 months, rising with exercise and relaxing with resting. He had no cardiovascular risk factor except smoking a pack/ day for 10 years. ECG revealed right bundle branch block and no signs of acute ischemia. Transthoracic echocardiography showed normal LV wall motion and a normal ascending aorta in diameter. Optimal medical treatment (OMT) for stable angina pectoris including acetyl salicylic acid, metoprolol and rosuvastatin was initiated immediately. However, angina continued despite a short course of OMT. Then coronary angiography was planned to identify options for revascularization. Coronary angiography revealed giant coronary aneurysms on the proximal left anterior descending artery (20 X 18 mm) and circumflex artery (16 X 20 mm) and accompanying atherosclerotic stenosis adjacent to the aneurysms (Figure 1). Hs-CRP was measured 4.5 mg/L. Antinuclear antibody, anti-ds-DNA level, and p-ANCA and c-ANCA levels were in normal range. Because our patient had multivessel CAD and SYNTAX score was 30, heart team considered CABG was more beneficial for this patient and accordingly he was transferred to cardiovascular surgery (CVS) department for CABG.

Current data are limited in clearly identifying the precise mechanism leading to luminal dilatation in patients with CAA [2]. Behçet's disease and Takayasu arteritis are responsible in some of the cases [3,4]. In addition, coronary atherosclerosis is present in almost 50% of the patients with CAA, suggesting a common causative mechanism in the formation of both CAD and CAA. Cytological studies have shown that histology of arterial wall is similar in both CAA and atherosclerosis except the loss of muscle elastic arterial wall that is seen in CAE. Infectious diseases, toxic, traumatic causes and genetic effect may also play the role in the development of CAA. Ectatic coronary arteries may be associated with potentially significant complications due to distal embolization as a result of



**Figure 1:** Coronary angiography image showing giant aneurysms on left anterior descending artery (20X18 mm) and left circumflex artery (16x20 mm) and atherosclerotic stenosis proximal and distal to the aneurysms.

stasis in the dilated segments and impaired coronary flow. Treatment options vary from aggressive surgical ligation of the aneurysm, in union with distal bypass surgery, to percutaneous implantation of a covered stent or conservative medical management with continued antiplatelet therapy. Surgical correction is generally accepted as the preferred treatment for giant coronary artery aneurysms [5].

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