CASE REPORT

Solitary Focal Coronary Artery Aneurysm in a Middle Aged Male with Atypical Chest Pain

C K Yeo, MRCP, Y Khalid, FACC, Department of Medicine, Faculty of Medicine, Hospital Universiti Kebangsaan Malaysia, Jalan Tenteram, Cheras, 56000, Kuala Lumpur

Summary

A 52 year old hypertensive Malay man, a smoker who presented with a one month history of mild chest discomfort not related to exertion and had a positive stress test with ST segment depression in the lateral leads. Coronary angiography showed stenosis in the right coronary artery and a coronary aneurysm in the proximal segment of his left anterior descending. The aneurysm was situated just distal to a stenotic lesion. The aneurysm is most likely related to atherosclerotic coronary artery disease. The patient was treated with oral nitrates, aspirin, angiotensin converting enzyme inhibitor and warfarin to prevent thromboembolism related to the coronary aneurysm. He remains asymptomatic one year after diagnosis.

Key Words: Coronary aneurysm, Atherosclerosis, Coronary artery disease

Introduction

Coronary artery aneurysm was found in 1% to 4% of all coronary angiograms performed in one study [1]. Before the advent of coronary angiography, coronary artery aneurysms were found on postmortem as there are no specific clinical findings suggestive of this entity.

Underlying causes of aneurysm include atherosclerosis, Kawasaki disease, syphilis, congenital anomaly and other rare diseases [1]. The pathogenesis of coronary aneurysm is not well understood. Treatment depends on symptoms, the extent of disease and the underlying disease. Antianginal drugs with or without anticoagulant therapy has been the mainstay of therapy with recourse to coronary artery bypass surgery if symptoms remain uncontrolled [1-3].

A solitary coronary artery aneurysm involving the left anterior descending artery in a middle-aged man is reported.

Case Report

A 52 year old Malay man complained of chest discomfort of one month's duration. The chest discomfort was not related to exertion, described as chest heaviness with no radiation and came intermittently with each episode lasting less than five minutes. There was no history of shortness of breath, orthopnoea or paroxysmal nocturnal dyspnoea. He had hypertension for the past ten years and was compliant with treatment. He was not diabetic and there was no family history of ischaemic heart disease.

Examination revealed a medium built middle aged man with a blood pressure of 130 / 90 mm Hg and pulse rate of 80 beats per minute. There was no stigmata of hypercholestrolemia. His JVP was not raised and there was no evidence of heart failure. Cardiovascular examination revealed a normal apex beat with normal heart sounds and no murmurs. Examination of his respiratory, abdominal and neurological systems was essentially normal.

Investigations revealed normal full blood count, renal profile, and blood sugar level and lipid profile. There was no cardiomegaly on chest X-ray and the lung fields were not congested. His ECG showed sinus rhythm and left axis deviation with no evidence of left ventricular hypertrophy or ischaemic changes.

At an exercise stress test using full Bruce protocol, there was ST segment depression of 1mm in the lateral leads during stage 3 , which persisted for 6 minutes into recovery. His blood pressure and heart rate response were normal and there was no chest discomfort. A coronary angiogram performed subsequently showed a tight proximal stenosis with aneurysmal dilatation just distal to the lesion in the left anterior descending artery (Figure 1). There were also multiple mid-right coronary artery stenosis. His left circumflex artery was normal. The left ventricular function was normal.

The patient was treated with Enalapril 5 mg daily for his hypertension. He was also treated with oral nitrates and aspirin, and anticoagulated with warfarin to reduce the risk of thromboembolic phenomenon related to the coronary artery aneurysm. He was advised to quit smoking.

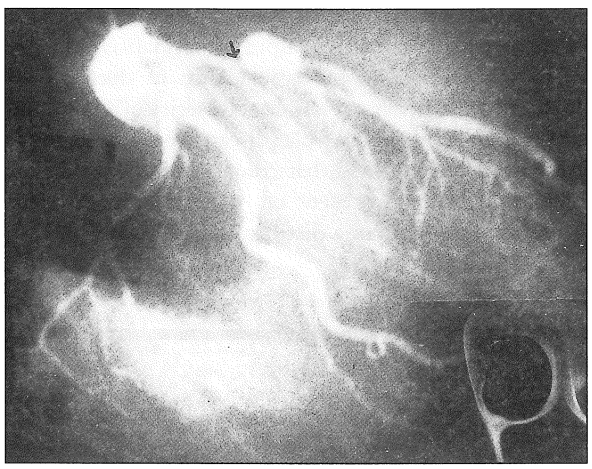

Fig. 1: Arrow showing a tight proximal stenosis with aneurysmal dilatation distal to it in the left anterior descending artery

Discussion

The first pathologic description of a coronary artery aneurysm was published by Morgagni in 1761[1]. Before 1967, all reported cases were based on post mortem studies[1]. The development of coronary angiography made in vivo diagnosis possible. The largest series reported to date is a review of the registry data of the Coronary Artery Surgery Study (CASS) in the United States. About 4.9% of a total registry population of 20,087 patients were identified as having aneurysmal coronary artery disease [2]. In other series the incidence of coronary artery aneurysm ranged between 1-4% of all coronary angiograms done. In our patient, the finding of the coronary artery aneurysm was incidental. The true incidence of coronary aneurysms is not known in Malaysia.

There is no history or clinical findings which are diagnostic of coronary artery aneurysm [1]. This patient presented with atypical chest pain. Coronary angiography was performed because of the positive stress test in a middle aged man with coronary risk factors. Clinical examination was essentially normal.

Although coronary angiography is the most useful method of investigation, large coronary artery aneurysms may also be detected by echocardiography, computed tomography (CT) and magnetic resonance imaging.

The risk factors for acquiring atherosclerotic heart disease in this patient are hypertension and smoking. Swaye et al.[2] found that there were no significant differences in family history, number or distribution of prior myocardial atherosclerotic score, calcification, hypertension, glucose intolerance or serum cholesterol in patients with and without aneurysms.

Kawasaki disease is currently the most common cause of coronary artery aneurysm worldwide [1]. This syndrome, of unknown etiology, was first described in Japan in 1967 and is relatively common in Japan. The syndrome which is most prevalent in children under the age of 5 years is characterised by fever, lymphadenopathy, enanthema and exanthema. The coronary arteries are usually involved in a diffuse manner or in multiple skip lesions resulting in arteritis or aneurysms. This patient is unlikely to have Kawasaki disease in view of his advanced age, no previous history suggestive of the

disease and the solitary aneurysmal lesion. The most common cause of aneurysms in the United States is thought to be atherosclerosis [1-3]. In view of the atherosclerotic lesion in his right coronary artery and in the proximal part of the left anterior descending artery just proximal to the aneurysm, the most probable cause for his aneurysm is atherosclerotic. Other rare causes include congenital anomaly, bacterial infection, syphilis, trauma, polyarteritis nodosa, systemic lupus erythematosus, Ehlers Danlos syndrome, scleroderma, Marfan's syndrome and Takayasu's arteritis[1]. Angioplasty has also been reported as a cause of coronary artery aneurysm.

Swaye et al.[2] reported that the proximal and middle segments of the right coronary artery were the most common sites of coronary aneurysms, followed by the proximal left anterior descending artery and lastly by the left circumflex artery.

The mechanism of coronary artery aneurysm formation in both patients with underlying atherosclerosis and without atherosclerosis is not well understood. The histologic features of an atherosclerotic aneurysm include diffuse hyalinization, lipid deposition, disruption of intima and media, focal calcification and fibrosis, cholesterol crystals, intramural haemorrhage and foreign body giant cell reaction of the atherosclerotic process [1]. It has been postulated that the aneurysmal dilatation is generated by intraluminal pressure against an elastic vessel wall with decreased stress tolerance [1]. Berkhoff and Rowe[3] postulated that if the intima covering the plaque in an atherosclerotic coronary artery breaks, the material within may become eroded by the blood stream and the excavated plaque becomes the site of aneurysm formation. As reduced flow velocity increases lateral pressure, material from the atheroma and microthrombi of platelets and fibrin resulting from eddy currents in the forming aneurysm may lead to embolisation of small distal vessels, resulting in progressive myocardial ischaemia.

Coronary artery aneurysm may cause angina, myocardial infartion or sudden death from thrombosis, thromboembolism or rupture. As postulated by Berkhoff and Rowe [3], thromboembolisation may result in progressive myocardial ischaemia. We therefore decided to anticoagulate patient with warfarin to prevent possible thrombosis.

The preferred treatment for symptomatic aneurysmal artery disease which is not responsive to conventional antianginal and anticoagulation therapy is coronary artery bypass grafting [1]. A trial of anticoagulant is justified as we believe that his chest discomfort could partly be due to microthrombi episodes as postulated by Berkhoff and Rowe.

The prognosis of this patient is good if thromboembolisation from the aneurysm could be prevented and hypertension controlled to limit the progression of atherosclerosis. Swaye et al.[2] found that there was no significant difference in 5 year survival between aneurysmal and non aneurysmal coronary disease patients. In addition, there were no significant differences noted between aneurysmal and non aneurysmal coronary disease patients when features such as hypertension, diabetes, lipid abnormalities, family history, cigarette smoking, incidence of documented myocardial infartion, presence and severity of angina and presence of peripheral vascular disease were examined. It was therefore suggested that coronary artery aneurysms are probably a subset or variant of coronary atherosclerosis and not a distinct clinical entity [1,2].

References

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