Coronary Ectasia In A Patient With Unstable Angina: Case Report And Literature Review

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Abstract

Coronary ectasia is a little common angiographic finding. Its prevalence varies between 0.3-5.3% in different series, although a recent study reports until 6.7%. Coronary ectasia is suffered mainly by males, and may be produced by different causes; however in most of the patients it seems to be related to coronary atherosclerosis. In fact, some authors consider it like a variant of the atherosclerotic disease of coronary arteries. We report the case of a 54-year-old white male who was admitted on intensive care unit because of unstable angina with electrocardiographic changes. Coronary angiography revealed severe coronary ectasia, mainly of the left main coronary artery and the proximal portion of left anterior descending artery. The greatest ectasia of the left main coronary artery that we have found in reviewed literature had 15.0 millimeters and ours sized 12.9 millimeters. Angiographic images are shown.

INTRODUCTION

Coronary ectasia (CE) is a little common angiographic finding. It is defined as a dilation 1.5 fold the diameter of the normal adjacent segments of the same artery, or other which could be comparable. It is a diffuse affection that must be differentiated from aneurysms, and its prevalence varies between 0.3-5.3% in different series, although a recent study reports until 6.7%. CE is mainly encountered in males, and may be produced by different causes; however in most of the patients it seems to be related to coronary atherosclerosis. In fact, some authors consider it like a variant of the atherosclerotic disease of coronary arteries. Others say that CE is a rare form of coronary artery disease. The increasing number of coronary angiographies has allowed the diagnosis of greater number of cases with CE, due to the development and diversification of this technique. Guérios et al. said that its etiology, treatment and prognosis remain dark.

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CASE REPORT

We report the case of a 54-year-old white male who was in a penitentiary center and suffered from chronic coronary artery disease with an old noncomplicated myocardial infarction that received thrombolytic therapy. He was admitted on intensive care unit this time because of a typical chest pain that did not alleviate with sublingual nitroglycerin, reason why it was necessary to use the intravenous infusion. In spite of it, the symptoms persisted; that was why coronary angiography was prescribed.

The electrocardiograms of the last 24 hours showed evident ischemic signs (ST and T alterations), but had the characteristic of being electrical disturbances that returned to normality and reappeared in different topographies.
Coronary angiography revealed severe CE, mainly of the left main coronary artery, and the proximal portion of left anterior descending artery (Figures 1 and 2).

**Figure 1**
Figure 1: Severe coronary ectasia of left main coronary artery (TCI by its spanish acronym), and the proximal portion of left anterior descending artery (DA by its spanish acronym). Right anterior oblique.

The right coronary artery had a small and focal expansion, located on its medial third compatible with an aneurysm formation (Figure 3).

**Figure 3**
Figure 3: Right coronary artery (CD by its spanish acronym) focal expansion compatible with an aneurysm formation. Left anterior oblique view.

Just before the end of angiography, the patient experienced loss of muscle strength in the left side of the body accompanied by entangled language. Anticoagulating therapy was begun, and left hemiparesia totally disappeared in the first 24 hours of evolution in the intensive care unit.

All serologic and specific tests were normal, as same as his lipid profile.

The patient was withdrawn without symptoms within the fifth day of admission. We motivated ourselves to present this case because the greatest ectasia of the left main coronary artery that we have found in reviewed literature had 15.0 millimeters and ours sized 12.9 millimeters.

**DISCUSSION**

**ETIOLOGY**

Although the etiology of CE is an open field where scientists are still investigating, several possible causes are recognized such as chronic exposure to herbicides, disease of Takayasu, polyarteritis nodosa, diseases of Kawasaki and Ehlers-Danlos, and other less important diseases like the syndrome of Marfan, rheumatic fever and syphilis.
Atherosclerosis is, probably, the main cause of primary CE. Percutaneous transluminal coronary angioplasty may favor secondary CE; however, the alterations produced in the vascular wall by coronary dilations are rather recognized as producers of aneurysms, because the damage is, strictly, focal. Another cause recently accepted is the prolonged anabolic steroid use, because it is considered that it produces structural damage in blood vessels. In addition, Sorrel et al consider that chronic exposure to vasodilator substances, such as nitric oxide may be included; in spite of being a fast local acting agent.

Androulakis et al suggest that CE would have some relation with diabetes mellitus and Gulec et al stated that alterations of angiotensin converting enzyme gene could constitute a potential risk factor for CE formation. Androulakis et al say that CE is associated with the presence of aneurysms in other sites of the arterial tree, including the aorta, and they also say that although the relation between arterial and venous ectasia is not established, they found in their study that the presence of varicose veins or phlebectasia in lower limbs was more frequent in patients with CE than in those who did not present it, which suggests the possible existence of a generalized defect of all vessel walls. Oomman et al and Triantafillidi and coworkers also agree this suggestion and reported cases with CE and aortic dissection, and CE with aneurysm of the basilar artery and phlebectasia. Altimbas et al describe the case of a woman who went to emergency room complaining of chest pain, and angiography revealed CE, enlargement of the ascending aorta and aortic coarctation.

Histological studies have demonstrated that there are certain atherosclerotic plaques that do not reduce the diameter of the lumen, presumably due to the relation of arterial remodeling, and it may play an important role on the physiopathologic mechanism of CE. Diffuse hyalinization and lipid deposition, and the appearance of alterations in the intima and media layers, as well as calcifications, fibrosis and intramural hemorrhage are findings which appear on histopathologic exam of aneurysms. Some of these findings can also be observed in CE. The presence of expanded segments in arteries with remarkable atherosclerosis and in those with structural changes as in the syndrome of Marfan supports the hypothesis that these are the result of a weakening of the media layer of the artery.

We tried to find the etiology of this illness in our patient but...
coronary syndrome there is an activation of the fibrinolytic system, probably induced by the formation of thrombus in the ectatic segments of coronary arteries. Therefore it may stimulate the appearance of chest pain that occasionally appears in patients with CE, without concomitants obstructive injuries.

Nyamu et al. reviewed 6938 coronary angiographies and they found that 134 (2%) had isolated CE; 118 were symptomatic, and of them, 34 (25%), had a history of or presented with myocardial infarction, with correlation between the affected territory and the ectatic artery in 32 patients. Sixty two patients presented dyslipidemia, and 65% of this were hypertriglyceridemia. Left anterior descending was the most commonly affected artery. Diffuse ectasia most commonly involved the right coronary artery, and one patient had spontaneous coronary dissection. There was a greater prevalence of isolated CE that frequently affected the right coronary artery, whereas diffuse CE and involvement of left anterior descending artery were discrete.

Nishikawa et al. showed the case of a 69-year-old woman who debuted with disnea on exertion, and echocardiogram revealed global hypokinesia of the left ventricle, which was dilated. Myocardial perfusion scintigraphy showed hypocaptation of the tracer which suggested the presence of a coronary artery disease rather than dilated cardiomyopathy, reason why coronary angiography was performed and no organic stenosis was observed; but diffuse CE was noted in three vessels. Intravascular ultrasound showed a severe CE with a maximal diameter of 8.2 mm (smaller than our patient). Coronary flow velocity was reduced, and spasm was not caused with the ergonovine test. These findings suggested the presence of alterations of microcirculation, probably by microthrombi, which could have produced myocardial ischemia and the later dilation. The patient was treated with nicorandil and ticlopidine and the left ventricle wall motion, as well as ischemia seen in the perfusion test, improved.

Coronary flow reserve is significantly reduced in patients with diffuse CE when comparing with control group. Although volumetric coronary blood flow is higher in the patients with CE, the dysfunction of microcirculation, reflected by the reduction of the coronary flow reserve, may be the cause of myocardial ischemia induced by exercise. The ectatic coronary segments have a media layer significantly thinner than the normal segments. Triantafyllidi et al. found that the pulsating changes of the arterial wall were increased in the ectatic segments and the ectatic coronary segments due to atherosclerosis showed a diminution of the thickening of the media layer, and an increase of the pulsating changes of the coronary arterial wall, which may be indicative of mechanical and structural weakness of the artery that prone to aneurysms formation. In fact, Senen et al. suggest that the physiopathologic mechanism of CE is not focal and they are, probably, right; however, this mechanism has not been well established yet. Sorrel et al. suggest that there is an association between the chronic stimulus of endogenous nitric oxide and the relaxation of the vascular wall which favors the sprouting of CE.

In the study of Harikrishnan et al. 144 patients with CE were diagnosed out of 3200 coronary angiograms (4.5%), and 122 presented coronary stenosis more than 50% (group A). Group B was formed by the 22 patients with CE who did not have obstructive coronary disease, and group C was formed by comparable patients who only had coronary disease without CE. All patients with CE presented similar epidemiologic features, however, the patients from group B showed better ventricular function and stress tests were negative. Nevertheless, in patients from group A, the incidence of myocardial infarction, coronary risk factors, and severity of coronary disease, as well as the stress test status were similar to those from group C.

On a mean follow up of 3±1.2 years, all patients (from 3 groups) had similar rates of cardiac events. In this study right coronary artery was the most commonly affected vessel, followed by circumflex, left anterior descending and, finally, the left main coronary artery. Diffuse CE was found more frequently in right coronary artery, and focal or localised CE was more common in left anterior descending artery.

It is considered that the ectatic areas, which were associated to coronary stenosis, are very prone to spasms, thrombosis and spontaneous dissection; being, therefore, potential causes of myocardial infarction. Spasms, microembolisms and the alterations of the blood flow, that appear in CE, are common causes of episodes of angina.

**DIAGNOSTIC AND THERAPEUTIC ASPECTS**

Echocardiography may be useful to detect CE and may even detect the presence of intracoronary thrombus. Tan et al. raise that the echocardiographic coronary-aorta index (greater coronary diameter in diastole divided between the
diameter of the aortic annulus) could be useful for detecting coronary dilation.

Magnetic resonance angiography, in spite of being a noninvasive method, has demonstrated effectiveness for follow up of patients with CE, and positron emission tomography is also useful for the diagnosis of coronary anomalies.

The suitable management and treatment of CE is not well established yet. Nevertheless, it is known that cumarinic anticlotting therapy is a standard regime in this disease, and trimetazidine may improve the exercise-induced angina, and also improve tolerance to physical activity in patients with CE. In addition, antplatelet therapy may be useful in the preservation of left ventricular function in patients with CE, as well as in diminishing symptoms. In cases of thrombotic occlusions, thrombolysis has shown effectiveness.

References

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[Resumen].
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