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 ustable 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 [1, 2]. 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 [3,4,5]. It is a diffuse affection that must be differentiated from aneurysms [3, 6, 7], and its prevalence varies between 0.3-5.3% in different series [3, 8,9,10]; although a recent study [11] reports until 6.7%. CE is suffered mainly by males [4, 9, 12,13,14,15], and may be produced by different causes; however in most of the patients it seems to be related to coronary atherosclerosis [3]. In fact, some authors consider it like a variant of the atherosclerotic disease of coronary arteries [1, 2]. Others say that CE is a rare form of coronary artery disease [15].

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 [7, 16]. Guérios et al [7]said that its etiology, treatment and prognosis remain dark.

Coronary ectasia (CE) is a common angiographic finding [1, 2]. It is defined as a dilation 1.5 fold the diameter of the normal adjacent segments of the same artery, or other arteries of comparable size. [3,4,5]. It is a diffuse affection that must be differentiated from aneurysms [3, 6, 7], and its prevalence varies between 0.3-5.3% in different series [3, 8,9,10] although a recent study [11] reports until 6.7%. CE is mainly encountered in males [4, 9, 12,13,14,15], and may be produced by different causes; however in most of the patients it seems to be related to coronary atherosclerosis [3]. In fact, some authors consider it like a variant of the atherosclerotic disease of coronary arteries [1, 2]. There has been an increasing recognition of this anomaly primarily due to a n exponential growth in the number of coronary angiography performed in recent years[7, 16].

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 acronysm), and the proximal portion of left anterior descending artery (DA by its spanish acronysm). Right anterior oblique.

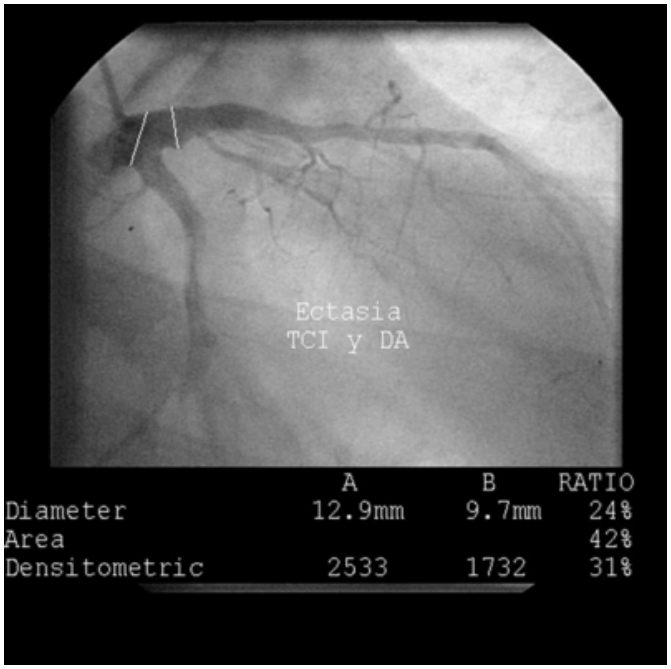
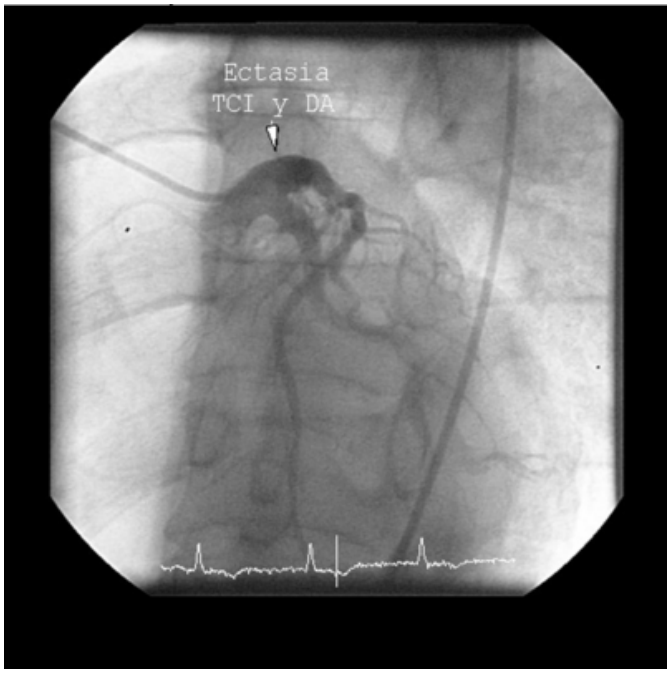


Figure 2

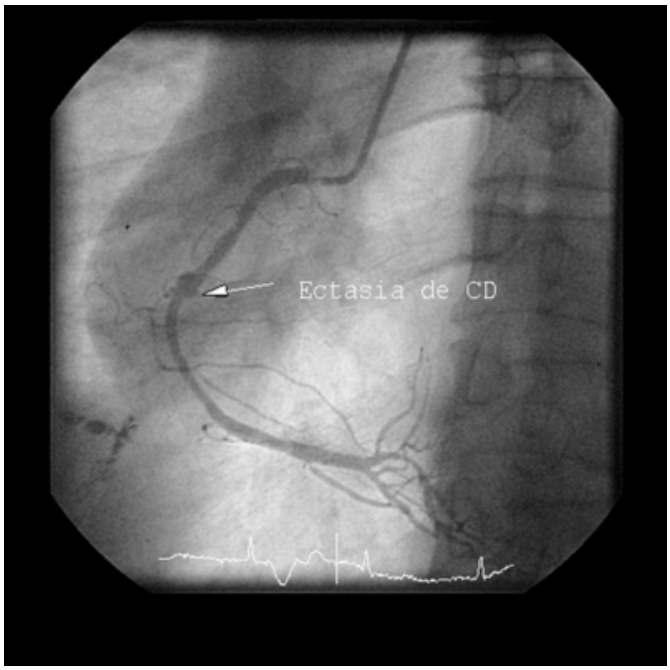
Figure 2: Severe coronary ectasia.



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 acronysm) 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 scientifics are still investigating [7], 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 [3, 7, 13, 17],

[18].

Atherosclerosis is, probably, the main cause of primary CE [7, 16, 19, 20]. Percutaneous transluminal coronary angioplasty may favor secondary CE [7, 16, 19] ; however, the alterations produced in the vascular wall by coronary dilations are rather recognized as producers of aneurysms, because the damage is, strictly, focal [5,7]. Another cause recently accepted is the prolonged anabolic steroid use, because it is considered that it produces structural damage in blood vessels [21]. In addition, Sorrel et al [19] consider that chronic exposure to vasodilator substances, such is nitric oxide may be included; in spite of being a fast local acting agent.

Androulakis et al [22] suggest that CE would have some relation with diabetes mellitus and Gulec et al [23]stated that alterations of angiotensin converting enzyme gene could constitute a potent risk factor for CE formation. Androulakis et al [24] 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 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 that in those who did not present it, which suggests the possible existence of a generalized defect of all vessel walls. Oomman et al [25] and Triantafillidi and coworkers [26] also agree this suggestion and reported cases with CE and aortic dissection, and CE with aneurysm of the basilar artery and phlebectasia. Altinbas et al [27] 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 phenomenon of arterial remodeling [28], and it may play an important role on 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 [7]. 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 [7].

We tried to find the etiology of this illness in our patient but venereal disease research was negative, as same as other specific laboratory tests that discard such diseases. We also performed an aortography, and looked for other ectatic arteries, however none positive result was found. That was why we inferred that his CE could be produce by atherosclerosis.

CLINICAL AND ANGIOGRAPHIC ASPECTS

Markis et al [29] were first in evaluating patients with CE prospectively. They described the clinical aspects of the evolution, during 24 months, of 30 selected patients out of 2457 consecutive coronary angiographies, and they observed that those patients, without obstructive coronary disease, had a greater prevalence of arterial hypertension and myocardial infarction than those from the control group; and presented a mortality rate of 15% in 2 years, equivalent to those with three-vessel disease. Palomo et al [9] found that all their patients with CE presented antecedents of angina or infarction, and left anterior descending artery was the most frequently affected (48%) by severe stenosis.

Coronary disease of the transplanted heart is one of the presentation types of CE [12]. More than 70% of the transplanted patients had some expanded coronary segment, which was associated, in more than half of the cases, to thickening of intima layer [12].

Endoh et al [30] found that 65% of their patients with CE had myocardial infarction, 91% had coronary artery disease, and 66% had a single vessel affected with the ectatic illness. Major coronary events were seen in 37% of patients; and 72% of these events, that appeared during the follow up, happened in the first 4 years after the cardiac event that promoted the first angiography. Seventy eight percent of these events were acute coronary syndromes. There were no significant differences for age, nor for the number of arteries with CE. This authors [30], as same as Guérios et al [7], and Nyamu et al [2] raise, that CE is not benign and must be carefully taken care.

Mrdovic et al [31] showed a case, similar to ours, of a 47-year-old man with several admissions due to acute coronary syndromes where the electrocardiographic alterations appeared in different regions; coronary angiography showed the presence of diffuse ectasia of the coronary arteries without significant estenosis. The patient received thrombolytic therapy in the first admission and in the second he needed defibrillation because of cardiac arrest in ventricular fibrillation. In patients with CE and acute

coronary syndrome there is an activation of the fibrinolytic system, probably induced by the formation of thrombus in the ectatic segments of coronary arteries [14]. Therefore it may stimulate the appearance of chest pain that occasionally appears in patients with CE, without concomitants obstructive injuries [2,32].

Nyamu et al [2] 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 [33] 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 estenosis 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 [34]. 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 [34]. The ectatic coronary segments have a media layer significantly thinner than the normal segments. Triantafyllidi et al [35] 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 [35]. In fact, Senen et al [5] suggest that the physiopathologic mechanism of CE is not focal and they are, probably, right; however, this mechanism has not been well established yet [7]. Sorrel et al [19] 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 [10]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 [7,20]. Spasms, microembolisms and the alterations of the blood flow, that appear in CE, are common causes of episodes of angina [7].

DIAGNOSTIC AND THERAPEUTIC ASPECTS

Echocardiography may be useful to detect CE and may even detect the presence of intracoronary thrombus [36]. Tan et al [37] 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 a demonstrated effectiveness for follow up of patients with CE [4], and positron emission tomography is also useful for the diagnosis of coronary anomalies [38]. The suitable management and treatment of CE are not well established yet [16]. Nevertheless, it is known that cumarinic anticlotting therapy is a standard regime in this disease [1], and trimetazidine may improve the exercise-induced angina, and also improve tolerance to physical activity in patients with CE [15]. In addition, antiplatelet therapy may be useful in the preservation of left ventricular function in patients with CE, as well as in diminishing symptoms [39,40]. In cases of thrombotic occlusions, thrombolysis has shown effectiveness [5,41].

References

1. Ohno H, Higashidate M, Yokosuka T. Natural evolution of coronary artery ectasia after coronary artery bypass grafting. Kyobu Geka 2003; 56(13): 1092-4 [Abstract].
2. Nyamu P, Ajit MS, Joseph PK, Venkitachalam L, Sugirtham NA. The prevalence and clinical profile of angiographic coronary ectasia. Asian Cardiovasc Thorac Ann 2003; 11(2): 122-6.
3. Pinar E, López R, Lozano I, Cortés R, Carrillo P, Rodríguez R, et al. Ectasia coronaria: prevalencia, características clínicas y angiográficas. Rev Esp Cardiol 2003; 56: 473-9.
4. Mavrogeni SI, Manginas A, Papadakis E, Foussas S, Douskou M, Baras P, et al. Correlation between magnetic resonance angiography (MRA) and quantitative coronary angiography (QCA) in ectatic coronary vessels. J Cardiovasc Magn Reson 2004; 6(1): 17-23.
5. Senen K, Yetkin E, Turhan H, Atak R, Sivri N, Battaloglu B, et al. Increased thrombolysis in myocardial infarction frame counts in patients with isolated coronary artery ectasia. Heart Vessels 2004; 19(1): 23-6.
6. Lekuona I, Laraudogoitia E, Palomar S, Salcedo A, Zumalde J, Morillas M. ¿Ectasia coronaria aterosclerótica o síndrome linfomucocutáneo del adulto (enfermedad de Kawasaki)? Rev Esp Cardiol 1999; 52: 63-6.
7. Guérios EE, Bueno RRL, Andrade PMP, Nercolini DC, Pacheco ALA, Tarastchuk JCE, et al. Aneurisma de Tronco de Coronária Esquerda. [Consulted 24 August 2004]. Available at: http://esquina.cardiol.br/UDT/casos_clinicos/016.htm
8. Cokkinos DV, Demopoulos VP, Voudris V, Manginas A, Cotileas P, Foussas SG. Coronary artery ectasia: aspects of fitness to fly. Eur Heart J 1999; 1: D53-8.
9. Palomo VJA, Necoechea AJC, Ruesga ZE, Díaz DE, Muñoz RME, Jáuregui AR, et al. Ectasia de las arterias coronarias. Prevalencia y significancia clínica. Rev Mex Cardiol 1997; 8 (1): 15-20.
10. Harikrishnan S, Sunder KR, Tharakan J, Titus T, Bhat A, Sivasankaran S, Francis B. Coronary artery ectasia: angiographic, clinical profile and follow-up. Indian Heart J 2000; 52(5): 547-53.
11. Altinbas A, Nazli C, Kinay O, Ergene O, Gedikli O, Ozaydin M, et al. Predictors of exercise induced myocardial ischemia in patients with isolated coronary artery ectasia. Int J Cardiovasc Imaging 2004; 20(1): 3-17.
12. Coma-Canella I, Calabuig J, Díaz I, Rábago G, Maceira A, Mejía S. Dilatación de arterias coronarias en receptores de trasplante cardíaco. Estudio con ultrasonidos intravasculares. [Consulted 27 Julio 2004]. Available at: http://www.cfnavarra.es/salud/anales/textos/vol24/n1/resumen2.html
13. Posnik-Urbanska A, Szymanowska Z, Kawecka-Jaszcz K. The probability of Kawasaki diseases in young patients with cardiac disorders - retrospective studies. Przegl Lek 2003; 60(12): 792-6. [Abstract].
14. Dogan A, Tunc B, Ergene O, Ozaydin M, Nazli C, Altinbas A, et al. Evaluation of overall fibrinolytic activity in patients with coronary artery ectasia: global fibrinolytic capacity. Int J Cardiovasc Imaging 2003; 19(6): 465-71.
15. Dogan A, Ozaydin M, Gedikli O, Altinbas A, Ergene O. Effect of trimetazidine on exercise performance in patients with coronary artery ectasia. Jpn Heart J 2003; 44(4): 463-70. [Abstract].
16. Pagel A, Horovitz M, Michovich Y, Rapoport M. Coronary artery ectasia: a therapeutic dilemma. Harefuah 2002; 141(12): 1055-8, 1089, 1090.
17. Dieter RS, Murtaugh T, Black J, Russell DC. Coronary arteriomegaly in a patient with Ehlers-Danlos syndrome and multiple aneurysms - a case report. Angiology 2003; 54(6): 733-6.
18. Chakrabarti S, Thomas E, Wright JG, Vettukattil JJ. Congenital coronary artery dilatation. Heart 2003; 89(6): 595-6.
19. Sorrell VL, Davis MJ, Bove AA. Current knowledge and significance of coronary artery ectasia: chronologic review of the literature, recommendations for treatment, possible etiologies and future considerations. Clin Cardiol 1998; 21: 157-60.
20. Fleites H, Moreno R, Perez J, Alfonso F, Hernandez-Altolin R, Macaya C. Dilated coronariopathy and acute myocardial infarction. Rev Port Cardiol 2003; 22(4): 583-6.
21. Tischer KH, Heyny-von Haussen R, Mall G, Doenecke P. Coronary thrombosis and ectasia of coronary arteries after long-term use of anabolic steroids. Z Kardiol 2003; 92(4): 326-31.
22. Androulakis AE, Andrikopoulos GK, Kartalis AN, Stougiannos PN, Katsaros AA, Syrogiannidis DN, et al. Relation of coronary artery ectasia to diabetes mellitus. Am J Cardiol 2004; 93(9): 1165-7.
23. Gulec S, Aras O, Atmaca Y, Akyurek O, Hanson NQ, Sayin T, et al. Deletion polymorphism of the angiotensin I converting enzyme gene is a potent risk factor for coronary artery ectasia. Heart 2003; 89(2): 213-4.
24. Androulakis AE, Katsaros AA, Kartalis AN, Stougiannos PN, Andrikopoulos GK, Triantafyllidi EI, et al. Varicose veins are common in patients with coronary artery ectasia. Just a coincidence or a systemic deficit of the vascular wall? Eur J Vasc Endovasc Surg 2004; 27(5): 519-24.
25. Oomman A, Ramachandran P, Kumar NS. Combined presence of coronary artery ectasia and descending aortic dissection in polycystic kidney disease presenting as acute coronary syndrome. Indian Heart J 2003; 55(6): 646-8.
26. Triantafillidi H, Rizos I, Androulakis A, Stratos K, Arvaniti C, Toutouzas P. Coronary artery ectasia, aneurysm of the basilar artery and varicose veins: common presentation or generalized defect of the vessel wall? A case report. Angiology 2001; 52(4): 287-91.
27. Altinbas A, Ozaydin M, Kahraman H, Dogan A. A case with coarctation of the aorta accompanied by coronary ectasia and dilatation of the ascending aorta. Int J Cardiovasc

Imaging 2003; 19(3): 185-7.
28. Schoenhagen P, Ziada KM, Vince DG, Nissen SE, Tuzcu EM. Arterial remodeling and coronary artery disease: the concept of "dilated" versus "obstructive" coronary atherosclerosis. J Am Coll Cardiol 2001; 38(2): 297-306.
29. Markis JE, Joffe CD, Cohn PF, Feen DJ, Herman MV, Gorlin R. Clinical significance of coronary arterial ectasia. Am J Cardiol 1976; 37(2): 217-22.
30. Endoh S, Andoh H, Sonoyama K, Furuse Y, Ohtahara A, Kasahara T. Clinical features of coronary artery ectasia. J Cardiol 2004; 43(2): 45-52.
31. Mrdovic I, Jozic T, Asanin M, Perunicic J, Ostojic M. Myocardial reinfarction in a patient with coronary ectasia. Cardiology 2004; 102(1): 32-4.
32. Gutiérrez O, Sáenz M, Vásquez G, Romero L, Esquivel L. Angina de pecho con arterias angiográficamente normales. Características epidemiológicas y clínicas. Rev Costarr Cardiol 2002; 4(3): 15-20.
33. Nishikawa S, Ito K, Takata H, Tsubakimoto Y, Yuba T, Adachi Y, et al. Assessment of microcirculation disturbance with nuclear cardiology in a patient with coronary ectasia: a case report. Kaku Igaku 2003; 40(1): 17-22.
34. Akyurek O, Berkalp B, Sayin T, Kumbasar D, Kervancioglu C, Oral D. Altered coronary flow properties in diffuse coronary artery ectasia. Am Heart J 2003; 145(1): 66-72.
35. Triantafyllidi H, Vavouranakis M, Arvaniti C, Stefanadis C, Toutouzas P. Anatomical and functional characteristics of ectatic coronary arteries. An intracoronary ultrasound study. Acta Cardiol 2004 59(2): 153-8.
36. Hatano S, Nobuoka S, Tanaka H, Nagashima J, Ikeda K, Miyake F. A case of coronary artery ectasia with thrombus detected by transthoracic two-dimensional echocardiography. Echocardiography 2002; 19(8): 693-4.
37. Tan TH, Wong KY, Cheng TK, Heng JT. Coronary normograms and the coronary-aorta index: objective determinants of coronary artery dilatation. Pediatr Cardiol 2003; 24(4): 328-35.
38. Singh TP, Muzik O, Forbes TF, Di Carli MF. Positron emission tomography myocardial perfusion imaging in children with suspected coronary abnormalities. Pediatr Cardiol 2003; 24(2): 138-44.
39. Nagata K, Kawasaki T, Okamoto A, Okano A, Yoneyama S, Ito K, et al. Effectiveness of an antiplatelet agent for coronary artery ectasia associated with silent myocardial ischemia. Jpn Heart J 2001; 42(2): 249-54. [Resumen].
40. Grigorov V, Goldberg L, Mekel J. Platelet glycoprotein IIb/IIIa receptor blockade with integrilin (eptifibatide) for early postinfarction angina in a patient with coronary arterial ectasia. Cardiovasc J S Afr 2002; 13(3): 125-8.
41. Tanabe Y, Itoh E, Nakagawa I, Suzuki K. Pulse-spray thrombolysis in acute myocardial infarction caused by thrombotic occlusion of an ectatic coronary artery. Circ J 2002; 66(2): 207-10.

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