Coronary artery ectasia prevalence and clinical characteristics: experience from a single medical center

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RESUMEN

Antecedentes. La ectasia de arterias coronarias se define como un segmento de arteria coronaria dilatada 1.5 veces más en diámetro que los segmentos cercanos. Su presentación clínica va desde angina inestable, espasmo coronario e infarto agudo del miocardio. **Objetivo.** Conocer las características clínicas, presentación clínica, desenlaces y tratamiento de los pacientes con ectasia de arterias coronarias documentada. **Material y métodos.** Un estudio retrospectivo se realizó revisando expedientes clínicos de pacientes atendidos en la Unidad Coronaria del Hospital Médica Sur entre enero 2009 a diciembre 2010 con diagnóstico de síndrome coronario agudo o infarto del miocardio, y que fueron sometidos a coronariografía y diagnosticados con ectasia de arterias coronarias. **Resultados.** Durante un periodo de dos años, 283 pacientes se diagnosticaron con síndrome coronario agudo con elevación del segmento ST o angina inestable/síndrome coronario agudo sin elevación del segmento ST. De ellos, 118 fueron llevados a coronariografía y 13 pacientes se diagnosticaron con ectasia de arterias coronarias, con una incidencia de 4.59%, de los cuales 11 fueron varones. La mayoría de los pacientes tuvieron síntomas al diagnóstico (92.3%). La localización de los infartos fueron cara inferior (71.43%), anteroseptal (14.29%). **Conclusiones.** La incidencia de ectasia de arterias coronarias fue similar a la reportada por la literatura internacional y la forma de presentación más común de ectasia de arterias coronarias fue con síndrome coronario agudo con elevación del segmento ST.

Palabras clave. Ectasia. Angina. Infarto. Coronarias. Coronariografía. México.

ABSTRACT

Background. Coronary artery ectasia is defined as a segment of coronary artery that is 1.5 times more dilated in diameter than the nearby segments. Its clinical presentations are unstable angina, coronary vasospasm, and acute myocardial infarction. **Objective.** To determine the clinical characteristics, presentation, outcomes and treatment in patients with documented coronary artery ectasia in a single Medical Hospital Center at Mexico City. **Material and methods.** A retrospective study was conducted using the clinical records of all patients that underwent coronarography and were diagnosed with coronary artery ectasia, ACS or acute myocardial infarction attending the coronary unit at Medica Sur Medical Center in Mexico City between January 2009 and December 2010. **Results.** During the two-year period, 283 patients were diagnosed with ST elevation myocardial infarction or UA/non-STEMI. Of these, 118 underwent coronariography and 13 patients were diagnosed with coronary artery ectasia, i.e., an incidence of 4.59%, of which 11 were male. Most of the patients were symptomatic at the time of diagnosis (92.3%). The ST elevation myocardial infarction localizations of patients were inferior (71.43%), anteroseptal (14.29%), and posterior inferior (14.29%). **Conclusions.** The incidence of coronary artery ectasia was similar to that reported in the international literature, and the main mode of coronary artery ectasia presentation was ST elevation myocardial infarction.

Key words. Ectasia. Angina. Infart. Coronary. Coronariography. Mexico.

INTRODUCTION

Coronary artery ectasia (CAE), or aneurysm of coronary ectasia, is defined as a segment of coronary artery that is 1.5 times more dilated in diameter than nearby segments.[1] The first reference to CAE was made by Morgagni in 1761.[2] Subsequent studies were made by Bougon in 1812[3] and the term "ectasia" was coined by Björk in 1966.[4] The incidence of CAE is variable, ranging from 0.3 to 5.3%, but it may be increasing. CAE has been related to many agents, such as herbicides, Takayasu's disease, polyarteritis nodosa, trauma,

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and vascular lesions. It is related to atherosclerosis in most cases.[5] Its prognosis, clinical presentation, and treatment are unclear.[6] CAE congenital causes include diseases such as polycystic kidney disease[7] and Ehlers-Danlos syndrome.[8] They must be differentiated from atherosclerotic disease, Kawasaki disease,[9] syphilis, Takayasu's disease,[10] trauma, and aortic dissection.

The characteristic lesions of CAE are similar to coronary atherosclerosis. It is not surprising that some hypothesis links both diseases. A previous study describes CAE pathological characteristics including lipid deposits with foam cells, loss of muscle wall elasticity, and chronic vascular inflammation.[11]

A probable mechanism that may predispose to CAE may involve the chronic overstimulation of the endothelium by NO or NO donors. Enhanced NO production via the iNOS pathway after an increase in the local interstitial concentration of acetylcholine has been documented previously.[12] Clusters of CAE were observed in Vietnam War veterans after exposure to Agent Orange, which suggests a close link between NO overstimulation and thinning of the intima-media in the etiology of CAE. The chemical components of Agent Orange may antagonize with acetylcholinesterase leading to high levels of acetylcholine and increased NO production.[13]

It has been reported that angiotensin II has an important role in abnormal ventricular remodeling. Daugherty et al. detected a strong relationship between angiotensin II in mice and apolipoprotein E deficiency, which led to a dramatically remodeled vascular pathology including an increase in atherosclerosis and the formation of aortic aneurysms.[14] Another important factor in vascular remodeling is matrix metalloproteinase 2, which is the main enzyme that contributes to the degradation of the extracellular matrix. An imbalance between metalloproteinases and endogenous tissue inhibitors has been demonstrated in patients with CAE.[15]

OBJECTIVE

To determine the clinical characteristics, outcomes and treatment in patients with documented CAE in a single Medical Hospital Center at Mexico City.

MATERIAL AND METHODS

We conducted our study at Medica Sur Medical Center in Mexico City. It was a retrospective study that analyzed all patients who attended the coronary unit from January 2009 to December 2010 with a diagnosis of acute coronary syndrome (ACS), who were subsequently diagnosed with CAE by coronarography.

CAE was defined as ectasia of the coronary artery or coronary aneurysm, with a diameter greater than 1.5 times that of nearby segments of artery.[16]

- **Inclusion criteria.** Patients older than 16 years with a diagnosis of ACS (ST elevation or non-ST) subjected to electrocardiography and coronarography with a final report of CAE between January 2009 and December 2010.
- **Exclusion criteria.** Patients under 16 years old. Patients who received coronarography but with insufficient evidence of ectasia or aneurysm in their coronary arteries. Patients with a history of congenital cardiopathy, valvulopathy, or cardiomyopathy.

We used SPSS 17.0 for all statistical analyses.

This retrospective study was approved by the ethics committee of Medica Sur, all patients signed informed consent before coronariography procedure.

RESULTS

During the two-year period, 283 patients were admitted with a diagnosis of ST elevation myocardial infarction (STEMI) or unstable angina (UA)/non-STEMI and 118 underwent coronarography (Table 1). CAE was diagnosed in 13 patients with a prevalence of 4.59%. Of the 210 male patients who underwent coronarography, 11 (5.23%) were diagnosed with CAE. In contrast, only two of 73 females had CAE (2.73%) (Table 1). The mean age of patients with CAE was 59 years (range 45-89 years) and 38.46% of the patients had a family history of coronaropathy. Male patients predominated (84.62%) compared with women (15.38%) and 69.23% were smokers (30.77% nonsmokers). In the CAE group, 53.85% did not drink alcohol, while 46.15% were alcohol consumers. We found that 38.46% of the CAE group was obese. Other comorbidities found in our study group were type 2 diabetes (38.46%), hypertension (46.15%), dyslipidemia (30.77%), and coronary artery disease (7.69%).

The presence of an abdominal aneurysm was documented in one patient (7.96%). However, it should be noted that 30.77% of the study group had no disease symptoms at the time of hospitalization. The median hospitalization period was 6.38 days (range 3-14 days) (Table 2).

Table 1. Results of two years of coronary angiography.

	Total	Men	Women
• Overall patients	283	210	73
• All patients with diagnosis if ACE	13	11	2

ACE: coronary artery ectasia.

Table 2. Basal characteristics.

Patients	13
Age (mean)	59 years (45-89)
History of familiar coronariopathy	5 (38.46%)
Gender (%)	
Male	84.62
Female	15.38
Smoking status (%)	
Non smoker	30.77
Current smoker	69.23
Alcohol intake (%)	
Yes	46.15
No	53.85
Obesity (IMC ≥ 30)	38.46
Co-morbilities (%)	
None	30.77
Type 2 diabetes	38.46
Hypertension	46.15
Lipid disorders	30.77
Coronary artery disease	7.69
Abdominal aortic aneuryms	7.69
Days of hospitalization (mean)	6.38 days (3-14)

At the time of diagnosis, 92.3% of patients were symptomatic. Patients underwent coronarography after their first acute myocardial infarction (AMI) in 84.61% of cases and because of UA in 7.69% of cases. No patients were admitted with only dyspnea (Table 3).

The right coronary artery (RCA) was involved with CAE in 56.52% of cases, the left anterior descending artery (LAD) in 26.09%, the circumflex artery (Cx) in 13.04%, and the left main coronary (LMC) in 4.35% of cases (Figure 1, Table 4).

After hospitalization, the final diagnoses of the group of patients with CAE were AMI in 84.61% of cases, one patient with UA (7.69%), and one patient (7.69%) with aortic stenosis plus ischemic-positive markers (Table 5).

Of the 12 patients diagnosed with STEMI, 58.33% had STEMI and 41.66% had UA/non-STEMI. The STE-

Table 3. Cause of coronariography in the population with diagnosis of CAE.

Diagnosis	n = 13	(%)
Acute myocardial infarction	11	(84.61)
Unstable angina	1	(7.69)
Coronariography in patients with positive ischemic biomarker	1	(7.69)

Table 4. Artery related with ectasia.

Artery	Lesion	(%)
Right coronary artery	13	(56.52)
Left anterior descending artery	6	(26.09)
Circumflex artery	3	(13.04)
Left main coronary	1	(4.35)

Table 5. Final diagnosis of the patients with CAE.

Final diagnosis	n = 13	(%)
Infarction	11	(84.61)
Unstable angina	1	(7.69)
Aortic stenosis plus a is chemic biomarker positive undertaken to coronariography	1	(7.69)

MI localizations in patients were inferior (71.43%), anteroseptal (14.29%), and posterior inferior (14.29%).

In five of the patients with UA/non-STEMI, the electrocardiogram changes indicated ventricular tachycardia (20%), incomplete left bundle branch block (20%), and subepicardial ischemia (20%). The remaining 40% exhibited no changes in their electrocardiograms.

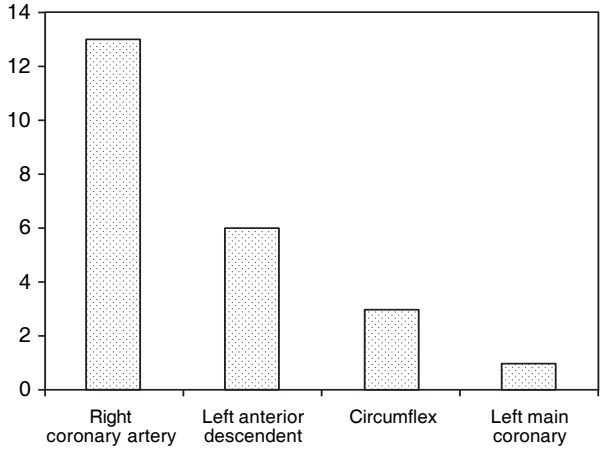

Figure 1. Number of aneurisms for each vessel studied.

The median values of the ischemic biomarkers at the time of admission of the STEMI patients were CPK (creatine phosphokinase) = 1,112.5 U/L, CPK-MB = 119.6 U/L, troponin I = 17.1 ng/mL, and myoglobin = 192.1 ng/mL. The median values for the UA/non-STEMI patients were CPK = 796.4 U/L, CPK-MB = 62.66 U/L, troponin I = 4.36 ng/mL, and myoglobin = 264.68 ng/mL.

In patients with STEMI who were tropinin I-positive, the region involved had a posterior-inferior localization in 6 of 7 cases with a tropinin I median level of 18.9 ng/mL (0.02-91.04), while one patient had an anteroseptal AMI with a troponin I level of 11.28 ng/mL. In the patient group with UA/non-STEMI (five patients; non-STEMI = 4 and UA =1), the troponin I mean level was 4.36 ng/mL.

The TIMI risk score of patients with STEMI had a mean of 3.57 points (range, normal 0 to 9 points), while the mean TIMI risk score for UA/non-STEMI patients was 3.23 (range, 1 to 5 points).

Of the patients diagnosed with ACS, 66.6% (n = 12) underwent percutaneous coronary intervention (PCI), 25% received PCI plus stent, whereas 8.33% could not pass through the guide. All patients received clopidogrel and aspirin whereas only 92.3% received statin. During hospitalization, 100% of the patients received a parenteral anticoagulant, 8.3% received conventional heparin, and 91.6% received enoxaprin.

The median length of stay was 7.14 days for the STEMI group and 6 days for the UA/non-STEMI group.

Two complications were documented, i.e., a rupture of the right ventricle and congestive heart failure. The patient with the rupture of the right ventricle died. Only one patient was discharged with dabigatran.

DISCUSSION

This is the first investigation of CAE in a nongovernmental institution in Mexico. We described the patient characteristics, clinical presentation, electrocardiographic changes, and outcomes during hospitalization. In Mexico, only one published abstract (Muñoz, *et al.*, 1996) from the VII Meeting of the National Association of Cardiology in Mexico has addressed CAE. Muñoz, *et al.* (1996) reported that 25,000 coronarographies were conducted over 25 months (January 1993 to February 1995) and 59 patients had CAE (incidence = 2.36%).[17] There have been no subsequent publications on this subject in Mexico.

In our study, the incidence of CAE was 4.59%, which was similar to that reported in other series. The incidence of CAE ranged from 1.2 to 4.9% and the highest level was reported by a coronary artery surgical study (CASS).[18] Differences in gender were described and partially attributed to women having a lower incidence of coronary disease.[19] A previous study described the higher prevalence of CAE in patients with familial hypercholesterolemia.[20] In our study, the frequency of dyslipidemia was 30.7% while that of hypertension was 46.1%, and both levels are similar to those reported by other authors.[21]

There is a known association between CAE and aortic abdominal aneurysms, descending aorta, popliteal artery, and pulmonary artery.[22] In our study group, only one patient had an aortic abdominal aneurysm at the time of coronarography (7.69%).

The etiology of CAE remains unclear and it would be interesting to determine the risk factors that might influence the origins of its pathology in this group of patients. Bermudez, *et al.* concluded that male gender and the absence of diabetes were the only variables associated with CAE as independent factors.[23] In our study, we found that the gender correlation was comparable with Bermudez, *et al.*'s conclusions, although we only found diabetes in 38.4% of the patients and it was less than the presence of hypertension.

Twelve patients (92.3%) clinically presented with angina or AMI, which was a much higher percentage than that reported in other series.[24] We suggest that this may mean that CAE is not a benign condition. The most common reported symptom of CAE is angina,[25] whereas other series indicate that uncommon events include STEMI, non-STEMI, arrhythmias, or sudden death.[26] In our study, 84.6% of the patients had AMI but only 7.7% had UA.

Angiographic findings were similar to those found in other publications, where RCA was the most commonly affected artery, with LMC being less affected.[27] The cause of a high predisposition to ectasia in the RCA is unknown. At present, no studies have reported the analysis of anatomical changes with CAE over a long time period. In a small series of nine patients, the CAE diameter was reported to have stabilized during a 36-month follow-up.[28]

In our study, the CAE findings were documented after the clinical event. The TIMI risk scores did not differ when the clinical presentation was ACS, because the median score for STEMI was 3.57 while that for UA/non-STEMI was 3.20, indicating that both groups had a mild risk according to the TIMI risk scale. The literature is unclear on the prognosis for patients with CAE. In our study, only one patient death was reported (7.7%).

It is known that CAE patients are prone to thrombosis, spasms, and dissection.[29,30] It has been shown that a low flow could be calculated based on the TIMI flow.[31] CASS detected no difference in the survival of patients with or without CAE.[18] Sadr-Ameli and Sharifi reported no significant differences in the AMI or death rate in patients with both CAE and CAD compared with either CAE or CAD.[32]

All the patients in our study received oral anti-aggregants and statins, as well as anticoagulants according to the guidelines for this type of event.[33,34] However, questions remain about the optimum CAE treatment. Warfarin, aspirin, diltiazem, and beta-blockers have all been suggested as treatments.[35-38] However, the literature on these management protocols is currently poor and they are only recommendations based on experience. Case-control studies are needed to prove the therapeutic efficacy of these drugs with this disease. There is also some controversy about the use of oral anticoagulants with CAE patients.

It is hypothesized that patients with angina experience a paradoxical ischemic response after the use of nitrates, probably via a mechanism known as coronary steal. In our study, only one patient was discharged with an oral anticoagulant, dabigatran. The source of this recommendation was the experience of one patient reported by Swanton, *et al*. who commented on the tendency to mural thrombosis in the bottom of ectatic vessels, cerebral arteries, and probably coronary arteries.[39] However, a series that tested oral anticoagulants did not prove any benefit after two- and five-year follow-ups.[40,41] This recommendation must be reviewed in larger cohorts because of the high risk of bleeding/hemorrhaging when using oral anticoagulants.

Most of the patients in our institution (Medica Sur) were discharged with dual antiaggregant therapy (aspirin and clopidogrel). If the diagnosis of CAE was incidental and the course of the disease was asymptomatic, the recommendation was oral antiaggregation in the form of aspirin, or clopidogrel for those allergic to aspirin.[35]

Management using beta-blockers and/or calcium channel blockers reduces the arterial flow but their use must be considered for each individual patient. There are no specific recommendations for this strategy and the same applies to statins.[39]

CONCLUSIONS

Our investigation of CAE in the Medica Sur Medical Clinic and Foundation detected a similar incidence to that reported in the medical literature. The main clinical presentation was ACS such as STEMI. In most cases, it was difficult to release a Stent in the affected artery because of the diameter of the vessel. There is no consensus on the ongoing management of CAE after an acute event has been controlled.

ABBREVIATIONS

- **ACS:** acute coronary syndrome.
- **AMI:** acute myocardial infarction.
- **CAE:** coronary artery ectasia.
- **CPK:** creatine phosphokinase.
- **CV:** coronary vasospasm.
- **Cx:** circumflex artery.
- **iNOS:** inducible nitric oxide synthase.
- **LAD:** left anterior descending artery.
- **LMC:** left main coronary.
- **NO:** nitric oxide.
- **Non-STEMI:** Non-ST elevation myocardial infarction.
- **PCI:** percutaneous coronary intervention.
- **RCA:** right coronary artery.
- **STEMI:** ST elevation myocardial infarction.
- **UA:** unstable angina.

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