**INTRODUCTION**

Coronary artery ectasia (CE) is defined as a ≥1.5-fold dilation with respect to the diameter of adjacent normal segments of the same or other arteries. A dif-
fuse condition, it must be distinguished from discrete
aeurysms or localized dilations that appear in areas
adjacent to coronary artery stenosis in some cases of
Kawasaki disease and other conditions.

Prevalence of CE varies between 0.3% and 5.3% de-
pending on series, but appears to have increased in re-
cent years. The cause of CE has been linked to va-
dious agents such as exposure to herbicides, Takayasu
aortitis, polyarteritis nodosa, vascular trauma or le-
isons. In most cases it seems to be related to coronary
arteriosclerosis. Most commonly, patients present
with associated coronary artery stenosis, even in cases
when lesions to coronary arteries are absent. Patients
suffer angina, vasospasm and myocardial infarction. The
real prevalence in our setting is unknown, as are
its clinical presentation and evolution.

Our objectives are:
- To study the prevalence of CE in the population of
  patients referred to our hemodynamics laboratory for
coronary angiography.
- To describe clinical characteristics of patients with
  CE, analyzing presentation and cardiovascular risk.
- To study angiographic characteristics of patients
  with CE.
- To compare clinical and angiographic variables in
  patients with and without CE.

PATIENTS AND METHOD

This was a cross-sectional study of patients under-
going coronary angiography and presenting with some
Patients with valvular heart disease, cardiomyopathy
or congenital heart problems were excluded as CE
could develop as a secondary consequence of these
conditions.

From October 1998, patients with CE were iden-
tified and followed clinically with twice-yearly check-
ups. Coronary artery ectasia was defined as a dilation
of the coronary artery longer than 20 mm and a ≥1.5-
fold in diameter when compared to the adjacent nor-
mal segment. The hemodynamic specialist and one of
the physicians in charge of the study agreed on an esti-
imated "normal" caliber when no such segment existed
(Figure 1). Similarly, patients were included in the
study as a result of a joint decision. After visual as-
essment, quantitative information was obtained using
Inturis Cardio Image 1.1 (Philips Medical Systems)
software. In each case, we established the maximum
diameter of the dilated artery and that of the healthy
segments. Blood supply to the heart was calculated
using the TIMI classification and the TIMI Frame
Count calculation. We recorded patients’ clinical
characteristics including details of age, sex, history of
angina, heart attack and revascularization procedures,
and cardiovascular risk factors (hypertension, dia-
abetes, hyperlipemia, cigarette smoking), as they appea-
red in their case histories. Clinical and angiographic
variables of patients with CE were later compared
with those of patients without CE. We described sig-
ificant angiographic lesions of patients in both groups.
We defined these as stenosis causing a ≥70% luminal
reduction in comparison to that of the normal referen-
ced segment.

Statistical analysis

Continuous variables were expressed as mean±SD,
and categorical variables as absolute value and percen-

ABBREVIATIONS

RCA: right coronary artery
CX: circumflex artery
ADA: anterior descending artery
CE: coronary artery ectasia
HT: hypertension
TIMI flow: classification of blood flow according to
TIMI (Thrombolysis in Myocardial Infarction) system
We compared means of independent samples using Student’s t test, and proportions using the chi-squared test. Statistical significance was defined as $P \leq 0.05$. We carried out multivariate analysis with logistical regression to analyze independent clinical predictors of the presence of CE. Data were analyzed with SPSS 10.0 software for Windows.

RESULTS

Between October 1998 and June 2001, 4709 patients underwent coronary angiographies. We included 4332 of them in the study, and 147 of these presented with CE, which represents a prevalence of 3.39% (95% CI, 2.87-3.97) (Figure 2).

Clinical characteristics

Characteristics of patients with and without CE appear in Table 1. Most of those with CE were men, smokers, with an average age markedly below that of patients without CE. They presented a lower prevalence of both diabetes and prior revascularization. Most patients were referred for coronary angiography because of unstable angina or chest pain (63% of patients with CE; 63.3% without CE) (Figure 3). In the remaining cases, angiography was carried out after myocardial infarct (30.1% and 25.6%, respectively), or to investigate stable angina or a ventricular dysfunction caused by ischemia (6.8% and 11.1%, respectively).

Angiographic characteristics

Thirty-three patients (22.4%) (95% CI, 16-30.1) presented with CE but without significant lesions to coronary arteries. In contrast, results of the angiographies indicated that most patients with CE, (77.6%) (95% CI, 69.9-84) presented significant stenosis in one (47%), two (37%) or three vessels (16%). Normally, CE affected one artery (49.7%), but two arteries were affected in 23.1% of the patients, and three were affected in 27.2%. The RCA was most frequently affected (132 patients), followed by the ADA (72 patients), and the CX.
(50 patients). The RCA presented the greatest degree of dilation, and higher levels of stasis of blood flow, and of contrast retention (Tables 2 and 3).

**Differences between patients with CE and without CE**

Men were in the majority in both groups. However, the proportion of men was significantly greater among patients with CE (91.2% vs 72%; \(P < .001\)). Proportions of the cardiovascular risk factors hypertension, hyperlipemia, family antecedents and history of infarct were similar. A history of smoking was more frequent among patients with CE (56.5% vs 40.2%; \(P < .001\)), whereas diabetes was less common (22.4% vs 35.1%; \(P < .001\)). Patients with CE showed a lower prevalence of prior revascularization, whether by angioplasty (8.2% vs 18%; \(P < .001\)) or surgery (1.4% vs 6.4%; \(P < .01\)).

In our series, 22.4% of patients with CE did not present coronary artery stenosis. Nineteen percent of the patients with angiographically significant lesions presented ischemic heart disease.

Despite the absence of stenosis, some patients with CE presented functional alteration, with slowing and stasis of the blood flow (Table 3).

A logistical regression model of the variables studied (age, gender, diabetes, hypertension, hyperlipemia, cigarette smoking, family history) showed that male sex was the only variable associated with the presence of CE (hazard ratio [HR]=3.33; 95% CI, 1.81-6.13). Diabetes was the only variable independently associated with the absence of CE (HR=0.65; 95% CI, 0.43-0.98) (Table 4; Figure 4).

### Follow-up of patients with CE

Patients with CE were evaluated clinically with an average follow-up of 25.6±9.3 months. The 33 patients with CE but without lesions received medical treatment. Two died (6.1%) during the follow-up period. Of the remaining 31 patients, seven continued to be completely asymptomatic (22.5%); 22 patients (71%) improved despite occasional angina; two patients (6.1%) reported symptoms identical to those recorded prior to the coronary angiography. During follow-up, two of the 31 surviving patients (6.5%) were readmitted for angina. Eight of the 114 patients with CE and associated coronary artery lesions received pharmacological treatment (7%). Nine underwent revascularization surgically (8%) and 97 by angioplasty (85%). During the long-term follow-up, eight patients died (7%), 56 of the 106 survivors were classified as asymptomatic (52%), 44 showed clear signs of improvement (41.5%), and six were considered stable (5.7%). Six survivors were readmitted for angina during the follow-up (5.7%) and angioplasty was repeated in 5 cases.

### DISCUSSION

The prevalence of CE in our series (3.39%) lies in the middle of the range described in the literature. It is greater than that reported in the earliest series,

<table>
<thead>
<tr>
<th>Artery</th>
<th>Diameter ectasia/normal vessel (mm)</th>
<th>Flow (TIMI/TFC)</th>
<th>Blood flow stasis (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADA</td>
<td>5.20±1.1/2.61±0.4</td>
<td>2.6±0.4/33.1±23</td>
<td>54.2</td>
</tr>
<tr>
<td>CX</td>
<td>4.76±1.3/2.10±0.4</td>
<td>2.8±0.2/25.7±23</td>
<td>31.3</td>
</tr>
<tr>
<td>RCA</td>
<td>5.61±0.9/2.54±0.4</td>
<td>2.8±0.2/23.1±15</td>
<td>67.7</td>
</tr>
</tbody>
</table>

TFC indicates TIMI Frame Count; ADA, anterior descending artery; CX, circumflex artery; RCA, right coronary artery.

**TABLE 3. Angiographic characteristics of vessels with ectasia but without coronary artery stenosis**

**TABLE 4. Clinical variables included in the logistical regression model used to determine characteristics independently associated with the existence of coronary artery ectasia (CE)**

### TABLE 2. Maximum diameters of affected areas of vessels with coronary artery ectasia and of normal reference segments

<table>
<thead>
<tr>
<th>Artery</th>
<th>Ectasia (mm)</th>
<th>Normal segment (mm)</th>
<th>Ectasia/normal diameter ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior descending</td>
<td>5.36±1.1</td>
<td>2.79±0.4</td>
<td>1.9</td>
</tr>
<tr>
<td>Circumflex</td>
<td>4.91±1.3</td>
<td>2.43±0.4</td>
<td>2</td>
</tr>
<tr>
<td>Right coronary artery</td>
<td>5.71±1.1</td>
<td>2.81±0.5</td>
<td>2</td>
</tr>
</tbody>
</table>

**TABLE 4. Clinical variables included in the logistical regression model used to determine characteristics independently associated with the existence of coronary artery ectasia (CE)**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Odds ratio</th>
<th>95% CI</th>
<th>(P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>0.97</td>
<td>0.97-1.01</td>
<td>ns</td>
</tr>
<tr>
<td>Male</td>
<td>3.33</td>
<td>1.81-6.13</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Diabetes</td>
<td>0.65</td>
<td>0.43-0.98</td>
<td>.03</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1.16</td>
<td>0.83-1.64</td>
<td>ns</td>
</tr>
<tr>
<td>Hyperlipemia</td>
<td>0.94</td>
<td>0.67-1.32</td>
<td>ns</td>
</tr>
<tr>
<td>Cigarette smoking</td>
<td>1.28</td>
<td>0.89-1.84</td>
<td>ns</td>
</tr>
<tr>
<td>Family history</td>
<td>0.98</td>
<td>0.49-1.98</td>
<td>ns</td>
</tr>
</tbody>
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NS: nonsignificant.
the tendency to provoke thrombosis and vasospasm, this slower blood flow may be the cause of the symptoms of angina and might even be one of the factors involved in the alterations that cause myocardial infarct in some patients with CE.

Given that the mechanism that causes CE is not clearly understood, it is important we investigate the risk factors in these patients which could influence the appearance of this condition. In our series, after correction for other variables, male sex and the absence of diabetes were the only variables independently associated with CE.

Patients with CE are predominately men, and in our study they represent 91.2% of all cases. Sudhir et al found a higher prevalence of CE in patients with a family history of high blood cholesterol. In our study, percentages of hyperlipemia (49.7%) and hypertension (51%) were high, but similar to those of patients with ischemic heart disease and without CE, which is consistent with other findings. However, there were significantly more smokers among patients with CE.

Prior revascularization was significantly lower in the group with CE, and this may have a variety of causes. We recognize that the population was relatively young, that most patients had only recently been diagnosed, that they had undergone coronary angiography for the first time, and that CE had been diagnosed throughout this procedure. However, in some cases medical treatment would have been chosen as they presented with nonsignificant stenosis or diffuse conditions.

Although the number of patients analyzed is as yet insufficient, the ≥2 year follow-up offers a less optimistic prognosis that could have been expected. Mortality rates were 6% among patients with CE and without lesions, and 7% among patients with significant stenosis. These data coincide with the results of a 5-year follow-up described by Cokkinos et al, these authors reported mortality rates of 10% and 9.5%, respectively, similar to the 11.9% rate among patients without CE but with acute coronary artery stenosis.

One significant finding, not previously reported, was the minimal prevalence of diabetes among patients with CE. The percentage was especially low among those with CE but without lesions (3%). It was also low among patients with CE and coronary artery stenosis (28.1%). In both cases, it was significantly lower than among patients without CE, and in these groups diabetes was present in 19% and 39%, respectively. This is not easily explained. Coronary artery ectasia seems to be a distinctive form of coronary artery atherosclerosis, caused by the action of different risk factors based on a genetic predisposition. This would lead to initial endothelial damage activating a series of inflammatory mediators (macrophages, metalloproteins, etc) that cause degeneration of the medial layer of the vessel. These structural alterations, together with the action of nitric oxide and other vasodilators, lead to a dilation of the coronary artery: an extreme form of «positive remodeling». However, among diabetic patients we found more cases of coronary arteries with diffuse conditions and «negative remodeling», without large amounts of plaque, but with luminal reduction and a decrease in the size of the vessel. One explanation might lie in the association of diabetes with a reduction in endothelium-dependent vasodilation, caused by alterations in the synthesis and inhibition of nitric oxide, which seems to play a significant role in the genesis of CE. Diabetes mellitus primarily affects the in-
timal, but not the medial layer of the vessel, thus caus-
ing «negative remodeling». It seems reasonable to hypothesize that positive remodeling appears in re-
cently developed lesions, whereas negative remode-
ing occurs at a later stage, after structural changes. 
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