Coronary Artery Ectasia: A Ten-year Experience in a Tertiary Hospital in Singapore
CSP Lam,1MBBS, MRCP, KT Ho,1MBBS, MRCP, FAMS

Abstract

Introduction: Coronary artery ectasia (CAE) is a well-recognised, albeit rare, angiographic finding of abnormal coronary dilatation. To our best knowledge, its incidence has never been described in Singapore. We aimed to determine the incidence of CAE at our teaching hospital and to describe patient and angiographic characteristics. Materials and Methods: Cases were identified through a search of our hospital’s computer database and all records were reviewed. Results: Over the 10-year study period (1992-2001), 8641 patients underwent coronary angiography. CAE was diagnosed in 104 patients, giving an incidence of 1.2%. The majority (74%) were male. The median age was 54 years (range, 25 to 79 years). Sixty-six per cent of patients were Chinese, 19% Malays, 12% Indians and 3% other races. Concomitant diabetes mellitus was present in 31%, hypertension in 58% and dyslipidaemia in 63% of patients. Co-existent obstructive coronary artery disease was present in 82% of cases. The frequency of arterial involvement was: right coronary artery (RCA), 65%; left anterior descending artery (LAD), 48%; circumflex artery (CX), 43%; and left main artery (LM), 2%. CAE affected only 1 major vessel in 62% of cases and all 3 vessels in 20%. Eighteen patients were advised to undergo bypass surgery, while percutaneous coronary intervention was recommended in 26 patients. Conclusions: The incidence of ectasia was 1.2%. The majority of patients were males in their sixth decade with underlying dyslipidaemia or hypertension. CAE was associated with obstructive coronary artery disease in more than 80% of cases. The RCA was most commonly affected and most patients had single vessel involvement.

Key words: Atherosclerosis, Dilatation, Myocardial ischaemia

Introduction

Coronary artery ectasia (CAE) is the abnormal dilatation of a segment of the coronary artery to a diameter of at least 1.5 times that of normal adjacent segments1 (Fig. 1). It is a well-recognised, albeit uncommon, finding at cardiac catheterisation. Although largely attributed to atherosclerosis, the mechanism behind the occurrence of dilatation in some, with stenosis in other, individuals with atherosclerotic coronary artery disease, remains obscure. Equally unclear are the prognosis and best form of treatment for the disease.

The condition was first described by Bourgon in 1812,2 while the term “ectasia” was first coined by Bjork in 1966.3 One of the earliest retrospective studies was by Markis in 1976,4 who also proposed the classification system still used today. Ectasia was classified according to the extent of involvement of the coronary vessels, with type I representing diffuse ectasia of 2 or more major vessels; type II diffuse ectasia in 1 vessel and localised disease in another; type III diffuse ectasia of 1 vessel only and type IV localised involvement only.

Several reviews have been done worldwide,1,4-7 but to our best knowledge, none has been conducted in Singapore. We sought to determine the incidence of CAE at our University teaching hospital in Singapore and to describe patient and angiographic characteristics.

Materials and Methods

Cases of CAE were identified through a search of our hospital computer database. All patients undergoing coronary angiography have the findings recorded in the database. The search extended from January 1992 to December 2001. Cases were reviewed and details regarding patient characteristics and angiographic findings were

1 Cardiac Department
The Heart Institute at National University Hospital, Singapore
Address for Reprints: Dr KT Ho, Cardiac Department, National University Hospital, 5 Lower Kent Ridge Road, Singapore 119074.
recorded. CAE was defined as dilatation of a segment of the coronary artery to a diameter of at least 1.5 times that of normal adjacent segments. Obstructive coronary artery disease (obstructive CAD) was defined as stenosis of more than 50% of the coronary lumen. Diabetes mellitus was defined by the documented clinical diagnosis or treatment with oral hypoglycaemic agents or insulin. Dyslipidaemia was defined by the documentation of the diagnosis, use of lipid-lowering agents, fasting total cholesterol ≥6.2 mmol/L (240 mg/dL) or low density lipoprotein cholesterol level ≥4.1 mmol/L (160 mg/dL).

Dichotomous variables were summarised as percentages, while continuous variables were summarised by their mean values (for normally distributed data) or median values (for skewed data).

Results

Over the 10-year study period, 8641 patients underwent coronary angiography. Of these, 104 patients were found to have CAE. This gave an incidence of 1.2%.

Of 3814 males undergoing cardiac catheterisation, 77 (2.0%) had CAE. This contrasted with only 27 (0.6%) of 4827 women studied (Table 1).

The median age of patients with CAE was 54 years (range, 25 to 79 years). Almost three quarters were males (74% males, 26% females). There were 69 (66%) Chinese, 20 (19%) Malay and 12 (12%) Indian patients, while the remaining 3 (3%) patients were of other races. Concomitant diabetes mellitus was present in 31%, hypertension in 58% and dyslipidaemia in 63% of patients.

Co-existent obstructive CAD was present in 85 cases (82%). Isolated CAE was found in only 19 (18%) cases (Table 1).

The frequency of arterial involvement, in descending order, was: right coronary artery (RCA) in 65%, left anterior descending artery (LAD) in 48%, circumflex artery (CX) in 43% and left main artery (LM) in 2% (Fig. 2). Ectasia affected only 1 major vessel in 64 (62%) cases and all 3 vessels in 21 (20%) cases. By Markis’ classification, the incidence of types I, II, III and IV lesions was 35%, 3%, 28% and 35%, respectively.

Based on the angiographic findings of stenoses in other arterial segments, 18 patients (17%) were advised to go for bypass surgery. Percutaneous coronary intervention was recommended in 26 patients (25%), while the rest (60 patients, 58%) were advised to undergo medical therapy.

<table>
<thead>
<tr>
<th>Intended therapy*</th>
<th>No. of patients</th>
<th>No. of deaths</th>
<th>Mortality rate over follow-up period (%)</th>
<th>Mortality rate per annum (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>104</td>
<td>4</td>
<td>3.8</td>
<td>1.0</td>
</tr>
<tr>
<td>CABG</td>
<td>18</td>
<td>3</td>
<td>16.7</td>
<td>4.5</td>
</tr>
<tr>
<td>PCI</td>
<td>26</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Medical</td>
<td>60</td>
<td>1</td>
<td>1.7</td>
<td>0.4</td>
</tr>
</tbody>
</table>

CABG: coronary artery bypass grafting; PCI: percutaneous coronary intervention

* Intended therapy: please see text for comments

Table 1. Results of Coronary Angiography Over the Ten-year Study Period

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>All patients</td>
<td>8641</td>
<td>3814</td>
<td>4827</td>
</tr>
<tr>
<td>All patients with CAE</td>
<td>104</td>
<td>77</td>
<td>27</td>
</tr>
<tr>
<td>Isolated CAE</td>
<td>19</td>
<td>16</td>
<td>3</td>
</tr>
<tr>
<td>CAE with obstructive CAD</td>
<td>85</td>
<td>61</td>
<td>24</td>
</tr>
</tbody>
</table>

CAD: coronary artery disease; CAE: coronary artery ectasia

Figures represent numbers of patients
consistently reported in the literature. Our findings are in agreement with that of Hartnell et al in their large series of almost 5000 patients.

Literature is unclear with regard to prognosis of the disease. In our series, there were 4 reported deaths, which translated to a mortality rate of 3.8% over the follow-up period, or 1.0% per annum. Unfortunately, we did not have a suitable control group for the study. Using the recommended therapy as a surrogate measure of the severity of the co-existent obstructive CAD, we sub-analysed the group. The analysis was based on the assumption that the most severe cases of stenotic disease (i.e., triple vessel or severe double vessel obstructive disease) would be advised to go for surgery, while the less severe cases would be recommended medical therapy. It should be emphasised that the categories in Table 2 represent the intended therapy and this does not necessarily correspond to the actual therapy received. For example, as mentioned previously, one of the deaths in the “intended surgery” group actually occurred in a patient who refused the surgery. Also, the figures are likely to be an underestimate of the true mortality rate as they only represent the hospital-recorded deaths.

The sub-analysis suggests that patients with the more severe form of obstructive CAD have a higher mortality rate. There were too few events to allow conclusions to be drawn regarding the prognostic significance of CAE alone.

There is a growing body of literature which suggests that ectasia is not a benign condition. Bove and Vliestra showed that ectatic arteries were prone to spasm, despite the fact that vascular medial damage was seen on histology. Suzuki et al reported that the actual narrowing usually occurred in the areas adjacent to the ectatic portion of the artery. There have been reports of thrombosis and dissection occurring in ectatic vessels. More recently, Kruger et al demonstrated exercise-induced myocardial ischaemia in patients with isolated ectasia, as measured by ergometry and coronary sinus lactate studies. They reported that the severity of coronary ischaemia measured on coronary sinus lactate sampling correlated with the size of coronary luminal enlargement in the proximal LAD. Papadakis et al showed that slow coronary flow, as measured by the TIMI frame count, occurred in ectatic vessels, suggesting that a possible mechanism of ischaemia was diminished coronary flow velocity. Other retrospective studies, that have used control groups of patients with isolated obstructive disease, have shown that the long-term prognosis of patients with isolated CAE was no better than in individuals with isolated obstructive disease.

There are many questions remaining, that this and other studies have not answered: if the aetiologic mechanism of CAE is atherosclerosis, why does stenosis occur in some individuals while dilatation occurs in others? Whatever the answer, it is clear that coronary atherosclerosis can no
longer be thought of as a “fixed” model where plaque growth would always lead to luminal narrowing. A more accurate model would be that of “arterial remodelling” where coronary atherosclerosis may cause vessel obstruction or dilatation, representing points in a continuum. Another unanswered question is: how should coronary ectasia be treated? The use of warfarin, aspirin, diltiazem and beta-blockers have all been suggested in literature. However the supporting literature is scant, with many recommendations based on anecdotal evidence. No randomised control study has ever been carried out to prove the utility of any particular therapy. The relative rarity of the condition would certainly prove a hindrance to any such study.

In conclusion, the incidence of CAE was 1.2% in our series. The majority of patients were males in their sixth decade with underlying dyslipidaemia or hypertension. CAE was associated with obstructive CAD in more than 80% of cases. The RCA was most commonly affected and most patients had single vessel involvement. Though a rare condition, literature review suggests that CAE is not a benign entity and further studies are required to establish the best strategy to treat these patients.

REFERENCES