Progress in an Adult Male Suffering from Coronary Artery Ectasia as Assessed with Exercise Stress Testing: A Nine-Year Case Report

Charles Micallef¹,²*

¹Sport Malta, Cottoner Avenue, Cospicua, Malta
²Ministry of Health, Merchants Street, Valletta, Malta

*Corresponding author: Charles Micallef, B. Pharm (Hons), M.Sc (PAPH), Sport Malta, Cottoner Avenue, Cospicua BML 9020, Malta, Tel: +356 99863324, E-mail: carmel.micallef@gov.mt, miccha@onvol.net

Abstract

A nine-year history of a young to middle-aged male who was diagnosed with coronary artery ectasia after presenting with impaired vision in one of his eyes is presented. Throughout this period, four exercise stress tests were carried out and improvements were noted: from a positive stress test indicative of myocardial ischemia, the outcome gradually progressed to negative. A distinct feature of this case is that the patient was only on moderate statin and dual antiplatelet therapy and his cholesterol levels remained significantly elevated.

Keywords

Coronary artery ectasia, Exercise stress test, Myocardial ischemia

Introduction

Coronary artery ectasia (CAE) is defined as localized or diffuse dilatation of coronary artery lumen exceeding the largest diameter of an adjacent normal vessel by more than 1.5 fold [1,2]. It is often regarded as an uncommon expression of atherosclerosis [3,4] and affects around 3 to 8% of patients undergoing coronary angiography [1,4] with male predominance [5]. Ectatic coronary arteries, even without the presence of coronary stenosis, are subject to thrombus formation, vasospasm, and spontaneous dissection [6].

Positive exercise stress tests can be associated with CAE [1]. However, more accurate testing is required to diagnose the illness. Although computed tomography (CT) angiography is increasingly used to detect coronary artery disease, the evaluation of stenosis is often uncertain [7]. On the other hand, as perfusion imaging has established a role in detecting ischemia and literature is showing that hybrid PET/CT (positron emission tomography/CT) imaging is extremely accurate in detecting coronary disease [7], this technique is however, insufficiently tried and tested on CAE patients. Hence, until now, conventional coronary angiography keeps offering a more detailed description of coronary artery anatomy and remains superior to cardiac ultrasound. One also needs to remember that the angiography case goes back to 2007 when evidence on accurate superior to cardiac ultrasound. One also needs to remember that the angiography case goes back to 2007 when evidence on accurate superior to cardiac ultrasound. One also needs to remember that the angiography case goes back to 2007 when evidence on accurate superior to cardiac ultrasound. One also needs to remember that the angiography case goes back to 2007 when evidence on accurate superior to cardiac ultrasound. One also needs to remember that the angiography case goes back to 2007 when evidence on accurate superior to cardiac ultrasound. One also needs to remember that the angiography case goes back to 2007 when evidence on accurate superior to cardiac ultrasound. One also needs to remember that the angiography case goes back to 2007 when evidence on accurate superior to cardiac ultrasound. One also needs to remember that the angiography case goes back to 2007 when evidence on accurate superior to cardiac ultrasound. One also needs to remember that the angiography case goes back to 2007 when evidence on accurate superior to cardiac ultrasound. One also needs to remember that the angiography case goes back to 2007 when evidence on accurate superior to cardiac ultrasound. One also needs to remember that the angiography case goes back to 2007 when evidence on accurate superior to cardiac ultrasound. One also needs to remember that the angiography case goes back to 2007 when evidence on accurate superior to cardiac ultrasound. One also needs to remember that the angiography case goes back to 2007 when evidence on accurate superior to cardiac ultrasound. One also needs to remember that the angiography case goes back to 2007 when evidence on accurate superior to cardiac ultrasound. One also needs to remember that the angiography case goes back to 2007 when evidence on accurate superior to cardiac ultrasound. One also needs to remember that the angiography case goes back to 2007 when evidence on accurate superior to cardiac ultrasound. One also needs to remember that the angiography case goes back to 2007 when evidence on accurate superior to cardiac ultrasound. One also needs to remember that the angiography case goes back to 2007 when evidence on accurate superior to cardiac ultrasound. One also needs to remember that the angiography case goes back to 2007 when evidence on accurate superior to cardiac ultrasound. One also needs to remember that the angiography case goes back to 2007 when evidence on accurate superior to cardiac ultrasound. One also needs to remember that the angiography case goes back to 2007 when evidence on accurate superior to cardiac ultrasound. One also needs to remember that the angiography case goes back to 2007 when evidence on accurate superior to cardiac ultrasound. One also needs to remember that the angiography case goes back to 2007 when evidence on accurate super...
regression [4,10,11]. A somewhat strange observation is the fact that stable coronary syndromes, can occur with atherosclerotic disease negative remodelling (arterial shrinkage), which is associated with is fundamental to the pathophysiology of coronary artery disease, states? As more than 50% of the reported cases of CAE are seen as a but to base the explanation on ‘arterial remodelling’ [10]. Could coronary atherosclerosis as no longer pertaining to a ‘fixed’ model where plaque formation would always lead to luminal narrowing, but to base the explanation on ‘fixed model’ meaning arterial remodelling [10]. Could coronaries remodel themselves back to their quasi-original states? As more than 50% of the reported cases of CAE are seen as a variant of atherosclerosis [9], and the process of arterial remodelling is fundamental to the pathophysiology of coronary artery disease, negative remodelling (arterial shrinkage), which is associated with stable coronary syndromes, can occur with atherosclerotic disease regression [4,10,11]. A somewhat strange observation is the fact that the subject’s cholesterol levels remained elevated (> 6.0 mmol/l).

However, although in about 50% of the cases CAE is often attributed to atherosclerosis, 20-30% has been considered to be congenital in origin [4]. Moreover, as the cholesterol levels, albeit were always significantly above normal, were never critically elevated, it is hard to elucidate whether or not the condition had anything to do with elevated cholesterol. In other words, the author cannot assume that this incidentally discovered ectasia is due to atherosclerosis. Perhaps it was present in childhood (for example, due to undiagnosed Kawasaki disease), discovered incidentally, and is still present despite the fact that the stress tests normalized. No such past medical history was recorded in the medical file and no tests are available to confirm its presence in the past.

As regards equipment, exercise electrocardiogram is not the ideal tool for investigating CAE progression or regression; a recent cardiac catheterization could consolidate this discussion. However, the patient did not wish to undergo another angiography. As no follow up imaging was performed, the study also cannot tell if this was a case of transient ectasia or a more permanent aneuryssmal defect.

A further drawback is associated with the consistency of the exercise stress testing; the stress tests should have been conducted by the same physician and technician. The reader should also be aware that the sensitivity and specificity of stress tests in identifying ischemia is not perfect and false positive or negative results may occur.

A general limitation with all CAE interventions is that no randomised control trial has ever been conducted to prove the utility of a particular treatment because the relative rarity of the condition would prove a hindrance to any such study [10]. Therefore, as supporting literature is scant, many recommendations have been based on anecdotal evidence. As a round-up, however, the prognosis of this CAE case can be associated with (but not necessarily attributed to) a combination of moderate statin use and dual antiplatelet therapy.

Table 1: Stress test reports.

<table>
<thead>
<tr>
<th>Year</th>
<th>Age</th>
<th>Duration</th>
<th>METS</th>
<th>Intensity</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>2009</td>
<td>40 yrs</td>
<td>9.4 min</td>
<td>11.2</td>
<td>96%</td>
<td>Atrial premature beats. ST changes: depression up sloping. Equivocal stress test.</td>
</tr>
</tbody>
</table>

1 METS stands for ‘metabolic equivalents’.
2 Intensity was calculated as a percentage of the maximal, age-predicted heart rate.

Cardiac investigations

Between 2006 and 2007 the patient underwent a series of tests. The first investigations included two ultrasound tests: a carotid Doppler, which showed no signs of stenosis, and an echocardiogram, which detected no cardiac sources of emboli.

In a span of nearly ten years, four treadmill ergometer stress tests were undertaken by the patient. The outcome of the first stress test was positive, suggestive of ischemia. The results of all the stress tests in chronological order are shown in table 1.

Soon after the first stress test was carried out in 2007, femoral coronary angiography revealed severe ectasia in the proximal left anterior descending coronary artery and mild ectasia in the proximal right coronary artery. Regular blood profiles kept indicating the need for statins to lower his cholesterol as much as possible.

Prescribed treatment

The initial medical treatment involved taking 75 mg of enteric coated aspirin, high dose statins and oral anticoagulants (aiming for an international normalized ratio [INR] of 2.0 to 3.0). However, treatment with 40 mg simvastatin daily was not tolerated due to severe myalgia in his legs and hence the patient was put on fluvastatin 40 mg twice daily. Warfarin therapy also had to be abruptly discontinued due the development of pruritic rash over his forearms and posterior thighs two days after starting warfarin (INR 1.2); this was supplemented by clopidogrel 75 mg daily. Aspirin and omega-3 intake continued.

Exercise stress test reports

Between 2007 and 2015 the patient (38 to 46 years) underwent four exercise stress tests that were conducted according to the Bruce protocol. The subject always achieved his target heart rate and no chest pains were ever reported. The overall impression changed from positive to negative or normal stress test. A summary of the stress test reports is found in table 1.

Ethical issues and access to medical file

Ethical approval and acquisition of written informed consent were not required. The subject was the researcher himself who was also not registered with an academic institution. The request to access the medical file was approved by the hospital’s Data Controller.

Discussion

It is clear from the stress tests reports that there was some progress: from a positive stress test indicative of myocardial ischemia (2007), the outcome gradually progressed to negative, with the results of the final stress test (2015) confirming those of the previous test (2011). A possible mechanism that could explain these changes is to look at coronary atherosclerosis as no longer pertaining to a ‘fixed model’ meaning arterial remodelling [10]. Could coronary vessels remodel themselves back to their quasi-original states? As more than 50% of the reported cases of CAE are seen as a variant of atherosclerosis [9], and the process of arterial remodelling is fundamental to the pathophysiology of coronary artery disease, negative remodelling (arterial shrinkage), which is associated with stable coronary syndromes, can occur with atherosclerotic disease regression [4,10,11]. A somewhat strange observation is the fact that the subject’s cholesterol levels remained elevated (> 6.0 mmol/l).

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Learning Objectives

Coronary artery ectasia can be managed without anticoagulant drugs.

Conflicts of Interest

The author has no competing interests to declare.

Disclosures

The author is the subject.

References


