Table 2
Degree of Blood Pressure Control, Type of Treatment, and Approach to Poor Blood Pressure Control According to Patient Age (<45 Years, 45-54 Years, and >54 Years)

<table>
<thead>
<tr>
<th>Variable</th>
<th>&lt;45 years (n=440; 3.4%)</th>
<th>45-54 years (n=1672; 12.9%)</th>
<th>&gt;54 years (n=10 849; 83.7%)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Good control</td>
<td>62.3</td>
<td>54.8</td>
<td>44.0</td>
<td>.0001</td>
</tr>
<tr>
<td>Drug therapy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Monotherapy</td>
<td>56.6</td>
<td>50.1</td>
<td>33.6</td>
<td>.0001</td>
</tr>
<tr>
<td>Combination therapy</td>
<td>43.4</td>
<td>49.9</td>
<td>66.4</td>
<td></td>
</tr>
<tr>
<td>Approach to poor control</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Change in treatment</td>
<td>36.3</td>
<td>35.1</td>
<td>27.5</td>
<td>.0001</td>
</tr>
<tr>
<td>Association of another drug</td>
<td>80.0</td>
<td>78.6</td>
<td>75.3</td>
<td>ns</td>
</tr>
<tr>
<td>Increase in dose</td>
<td>16.0</td>
<td>19.6</td>
<td>23.5</td>
<td>ns</td>
</tr>
<tr>
<td>Drug substitution</td>
<td>4.0</td>
<td>1.8</td>
<td>1.2</td>
<td>ns</td>
</tr>
</tbody>
</table>

ns, not significant. The data are expressed as %.
' Chi-square test.

younger than 55 years of age were receiving combination therapy, a rate that is appreciably lower than that found among hypertensive patients aged 55 years or older. Moreover, the therapeutic regimen was modified in only slightly more than one-third of the hypertensive patients younger than 55 years with poor BP control (36% of those under the age of 45 years). All these data are probably the result, in part, of an underestimation of cardiovascular risk among young hypertensive individuals, because risk is usually assessed over the short- or medium-term, rather than over the long-term.6

In short, approximately 16% of the hypertensive patients receiving drug therapy and being managed in primary care centers in Spain are younger than 55 years of age (3.4% are younger than 45 years). Approximately 40% to 45% of the patients younger than 55 years do not achieve BP targets. In the light of all these findings, we conclude that improvement in the overall control of cardiovascular risk factors in young individual is essential and that such improvement obviously includes hypertension.

Acknowledgements

We wish to thank all the primary care physicians participating in the PRESCAP 2010 study for their collaboration in the PRESCAP 2010 study and all the members of the HTA/SEMERGEN Group (list available in supplementary material) for providing the data necessary for its performance. We also thank Almirall, S.A., for providing the infrastructure necessary for carrying out this study.

Almirall, S.A., did not influence the collection or interpretation of the data.

SUPPLEMENTARY MATERIAL

Supplementary material associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.jrecesp.2013.04.015.

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Available online 10 July 2013

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http://dx.doi.org/10.1016/j.jrec.2013.04.014

Patient With Angina and “Congenital Bypass”. A New Case of Aortocoronary Fistula

Paciente con angina y «bypass congénito». Caso inédito de fistula aortocoronaria

To the Editor,

A 58-year-old man, who had quit smoking 16 years previously and had no other modifiable cardiovascular risk factors, presented with a several-month history of episodes of anginal chest pain triggered by emotional stress. The physical examination was unremarkable.

Electrocardiography revealed a sinus rhythm of 75 bpm, with normal atrioventricular conduction, incomplete right bundle branch block, and no signs of ischemia or necrosis.

Echocardiography showed normal-sized chambers, with good biventricular contractility and dilatation of the tubular portion of the ascending aorta (44 mm), with mild aortic regurgitation of the tricuspid valve but no other noteworthy abnormalities.

A clinical diagnosis of stable angina was made and conventional stress testing was performed, which was positive due to the
Dilatation ordered showed repolarization disrupted an (Fig. 826 immediately)

Figure 1. Computed tomography angiography. A coronary artery fistula was seen between the Valsalva sinus of the right coronary artery and the left main coronary artery. Notice a first fistulous tract running anterior to the pulmonary artery and a second fistulous tract near to its termination, which seems incomplete. AR, aortic root; F1, first fistulous tract; F2, second fistulous tract; LMCA, left main coronary artery; PA, pulmonary artery; RCA, right coronary artery.

presence of pain at a submaximal heart rate, without associated repolarization abnormalities.

In view of these findings, we considered the probability of the presence of major coronary lesions to be low. Accordingly, we ordered a multidetector computed tomography (CT) scan, which showed no coronary lesions but revealed the presence of an irregular fistula that, from its origin in the right Valsalva sinus, cranial to the ostium of the right coronary artery, passed in front of the trunk of the main pulmonary artery before terminating in the left main coronary artery; at this site, another possible fistulous tract was observed that passed cranially and anteriorly to the pulmonary artery. However, visualization of this fistula was disrupted at this point by defects in image processing (Fig. 1).

The above findings were confirmed by coronary angiography (Fig. 2), but an additional fistulous tract was identified that shared an origin and termination with the fistula detected by CT. Dilatation of the ascending aorta was also seen, without visualization of stenosis of the coronary arteries.

Because the symptoms were mild, and their relationship with the fistula was unclear, we opted for medical treatment and, taking into account the aneurysm of the ascending aorta, also prescribed beta-blockers. Moreover, the risk of coronary artery fistula rupture has rarely been described, and there is no evidence that would have justified a more invasive approach. The patient showed marked clinical improvement and only experienced sporadic anginal episodes that were exclusively associated with sexual intercourse. These episodes were resolved by prescription of oral nitrates.

Congenital coronary fistulas are rare and generally isolated.¹ These anomalies are described as a connection between one or various coronary arteries and a cardiac chamber or large vessel.² ³ However, we found no cases in the literature describing a fistulous tract that connected a coronary artery with the aorta. The mechanism by which coronary fistulas usually cause angina is ischemia due to “coronary steal”,⁴ ⁵ which does not explain the symptoms in our patient, given that it would involve an excessive flow from the aorta to the left coronary artery. A likely cause of the ischemia was endothelial dysfunction secondary to excessive flow or thrombosis of the angulated and tortuous fistulous tracts. This shape favors blood stasis, which is a

Figure 2. Coronary angiography. The aortic root and a fistula originating in the right Valsalva sinus, independent of the orifice of the right coronary artery, which immediately bifurcates into 2 fistulous tracts (F1 and F2) that reconnect before terminating in the left main coronary artery. AR, aortic root; F1, first fistulous tract; F2, second fistulous tract; LMCA, left main coronary artery; RCA, right coronary artery.
documented cause of infarction in conventional coronary artery fistulas, although our patient had angina, not infarction. It is also important to stress the accuracy of multidetector CT in precisely defining the abnormal origin and path of the anomalous coronary arteries and their relationship with other structures, because it is sometimes difficult to visualize the course of the coronary arteries though conventional coronary angiography. CT can be considered the diagnostic method of choice in cases of clinical angina that are not explained by coronary artery lesions and which could be secondary to congenital abnormalities of the coronary artery. Nevertheless, we consider the use of coronary angiography to be appropriate because, in addition to confirming the diagnosis of a fistula, this technique provides complementary information about its connections, the bidirectional nature of the flow, and the coexistence of other tracts that escape the spatial resolution of CT, either due to their reduced diameter or to their omission from the sample volume imaged.

The use of image-based ischemia detection, whose usefulness would probably have been enhanced by the administration of vasodilator stress agents, could also have provided useful information on the presence of ischemia and its location and severity.

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Available online 10 July 2013

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Totally Subcutaneous ICD Implantation as an Alternative to the Conventional ICD in a Patient With a Congenital Cardiopathy

Implante de un DAI completamente subcutáneo como alternativa al DAI convencional en un paciente con una cardiopatía congénita

To the Editor:

Implantable cardioverter-defibrillators (ICD) are the standard treatment to prevent sudden death from ventricular arrhythmias. 1 Traditional devices require vascular access for electrode implantation, with the consequent risk of complications such as electrode dislodgement or fracture, thrombosis, or infection. 2,3 However, vascular access can prove difficult or even impossible in certain patients, such as those with certain congenital conditions that limit the use of traditional devices.

The subcutaneous ICD (S-ICD, Cameron Health, San Clemente, California, USA) avoids the need for intracardiac electrode implantation, facilitates the removal procedure, and does not require fluoroscopic guidance.

We present the clinical case of a man aged 32 years with a single-ventricle congenital heart disease, with transposition of the great arteries, atrial septal defect, and pulmonary stenosis. In 1998 he underwent a De Leval right subclavian pulmonary fistula 4 and, in July 2001, complete cavopulmonary shunt and closure of the fistula.

Since 2009 he had experienced several sudden episodes of syncope. Echocardiography revealed a dilated left ventricle with very depressed systolic function (30% ejection fraction) and right ventricular hypoplasia. The aorta was dilated, with wide interventricular septal defect override and substantial pulmonary artery hypoplasia. We found no flow through the valve but did observe distal venous flow proceeding from the bidirectional cavopulmonary shunt.

Figure 1. View of the patient’s chest after implantation of the automatic subcutaneous implantable device (A) and anteroposterior X-ray at 24 h post-implantation (B).