Our study has the limitations derived from its design and the small number of patients included, as it evaluates an uncommon treatment. Nonetheless, it may be an indication of typical clinical practice.

In conclusion, this study is the first to provide data on patients initially treated with i2PY12 monotherapy. This therapeutic strategy is not commonly used in clinical practice but is a reasonable choice for patients who cannot receive DAPT containing aspirin, as the outcome at 36 months was similar to that of patients receiving DAPT. Furthermore, the new i2PY12 agents could be an option when APM is needed, particularly after the acute phase has passed.

CONFLICTS OF INTEREST

I.J. Núñez-Gil has participated in lectures for AstraZeneca and Lilly, and has served as an advisor for AstraZeneca. E. Cerrato is a speaker for AstraZeneca Italy and has received research grants from AstraZeneca Spain.

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Functional and Morphological Assessment of Left Anterior Descending Artery in Patients With Tako-tsubo Syndrome

Análisis morfológico y funcional de la arteria descendente anterior de pacientes con síndrome de tako-tsubo

To the Editor,

Tako-tsubo syndrome (TKS) is a clinical syndrome characterized by reversible left ventricular dysfunction in the absence of epicardial coronary obstruction. Although several pathogenic mechanisms have been proposed (ie, multivessel epicardial spasm, catecholamine-induced myocardial stunning, spontaneous coronary thrombus lysis, and acute microvascular spasm), its causes are still unknown.1,2 Possible causes of TKS are the presence of vulnerable plaques or flow alteration, but they have not been well elucidated.

In our study, we sought to perform a functional and morphological assessment of the left anterior descending artery (LAD) in TKS patients by using optical coherence tomography (OCT) and pressure-temperature wire.

From January 2016 to May 2017, 14 consecutive TKS patients, admitted to 2 institutions and defined accordingly to Mayo Clinic diagnostic criteria,1 were included. The study was approved by the ethics committee of our center and each patient provided written informed consent. A pressure-temperature guidewire (Certos, St Jude) was introduced in the LAD at the level of the second diagonal branch. After induction of hyperemia with adenosine (140 μg/kg/min), fractional flow reserve and the index of microcirculatory resistance (IMR) were measured as previously shown.3 Fractional flow reserve and IMR were considered abnormal if < 0.80 and > 22, respectively.3 OCT acquisition was then performed using a commercially available system for intracoronary imaging (C7XR-Fourier-Domain System; LightLab Imaging, Westford, Massachusetts, United States) on the LAD (at least 50 mm) during continuous injection of contrast medium (3 ml/s, iodixanol 370, visipaque, GE Health Care, Cork, Ireland) through the guide catheter with an injection pump. The presence of coronary plaque on OCT pullback was analyzed offline by 2 independent investigators (LightLab Imaging, Westford, Massachusetts, United States).

Thirteen patients (92.8%) were women, with a mean ± standard deviation age of 66.1 ± 11.5 years. Coronary angiography showed no significant stenosis of at least 50% in the LAD. OCT and pressure-temperature wire analysis were performed in 14 and 12 patients, respectively. OCT analysis showed a normal 3-layer vessel wall, without atherosclerotic plaque, images of plaques rupture, plaques erosion, or intraluminal thrombus. None of the patients had fractional flow reserve ≤ 0.80, with a mean value of 0.96 ± 0.18, while 10 (83.3%) patients had microvascular dysfunction with IMR ± standard deviation of 33.8 ± 11.4 (Table).

The physiopathology of TKS is unknown, but can be related to intracoronary thrombus, either emerging from a ruptured thin-cap fibroatheroma or plaque erosion in the LAD, which is responsible for the typical left ventricle appearance. Coronary thrombus or at least the coronary plaque, which has led to thrombus formation, may be undetectable by coronary angiography, but may be seen on OCT.1 Nevertheless, our OCT analysis did not show any coronary plaque or thrombotic remnants on LAD, also excluding the presence of any atherosclerotic coronary plaque. This is in contrast with a recent study that showed a high prevalence of atherosclerotic plaques in these patients without any plaque rupture or thrombi.1 Unlike that study, our patients were younger with fewer
Table
Baseline Clinical Characteristics of the Tako-tsubo Syndrome Patients

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, y</th>
<th>Sex</th>
<th>Cardiovascular risk factors</th>
<th>Stressful event</th>
<th>Ballooning pattern</th>
<th>Initial EF %</th>
<th>Discharge EF%</th>
<th>Time since symptom to pressure-temperature wire measurement (hours)</th>
<th>FFR</th>
<th>IMR</th>
<th>OCT</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>67</td>
<td>M</td>
<td>Smoking</td>
<td>Physical</td>
<td>Apical</td>
<td>40</td>
<td>56</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>Normal</td>
</tr>
<tr>
<td>2</td>
<td>71</td>
<td>F</td>
<td>None</td>
<td>Physical</td>
<td>Apical</td>
<td>40</td>
<td>55</td>
<td>4</td>
<td>0.96</td>
<td>51.8</td>
<td>Normal</td>
</tr>
<tr>
<td>3</td>
<td>50</td>
<td>F</td>
<td>Smoking</td>
<td>Emotional</td>
<td>Apical</td>
<td>60</td>
<td>59</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>Normal</td>
</tr>
<tr>
<td>4</td>
<td>55</td>
<td>F</td>
<td>Smoking</td>
<td>Emotional</td>
<td>Apical</td>
<td>34</td>
<td>43 (55, 1 month after discharge)</td>
<td>12</td>
<td>0.96</td>
<td>43.3</td>
<td>Normal</td>
</tr>
<tr>
<td>5</td>
<td>70</td>
<td>F</td>
<td>Hypertension, hypercholesterolemia</td>
<td>Emotional</td>
<td>Apical</td>
<td>40</td>
<td>51 (62, 1 month after discharge)</td>
<td>8</td>
<td>0.97</td>
<td>39.2</td>
<td>Normal</td>
</tr>
<tr>
<td>6</td>
<td>86</td>
<td>F</td>
<td>Hypertension</td>
<td>Physical</td>
<td>Apical</td>
<td>35</td>
<td>55</td>
<td>18</td>
<td>0.94</td>
<td>33</td>
<td>Normal</td>
</tr>
<tr>
<td>7</td>
<td>72</td>
<td>F</td>
<td>None</td>
<td>Emotional</td>
<td>Apical</td>
<td>50</td>
<td>60</td>
<td>8</td>
<td>0.96</td>
<td>37.2</td>
<td>Normal</td>
</tr>
<tr>
<td>8</td>
<td>68</td>
<td>F</td>
<td>Hypertension, hypercholesterolemia</td>
<td>Emotional</td>
<td>Apical</td>
<td>45</td>
<td>50 (65, 1 month after discharge)</td>
<td>120</td>
<td>0.92</td>
<td>26.9</td>
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</tr>
<tr>
<td>9</td>
<td>63</td>
<td>F</td>
<td>Hypertension</td>
<td>Emotional</td>
<td>Midventricular</td>
<td>35</td>
<td>45 (55, 1 month after discharge)</td>
<td>18</td>
<td>0.98</td>
<td>19</td>
<td>Normal</td>
</tr>
<tr>
<td>10</td>
<td>47</td>
<td>F</td>
<td>Hypertension, hypercholesterolemia</td>
<td>Emotional</td>
<td>Apical</td>
<td>30</td>
<td>60</td>
<td>4</td>
<td>0.95</td>
<td>25.6</td>
<td>Normal</td>
</tr>
<tr>
<td>11</td>
<td>61</td>
<td>F</td>
<td>Hypertension</td>
<td>Physical</td>
<td>Apical</td>
<td>40</td>
<td>60</td>
<td>254</td>
<td>0.96</td>
<td>10.9</td>
<td>Normal</td>
</tr>
<tr>
<td>12</td>
<td>83</td>
<td>F</td>
<td>Hypertension, hypercholesterolemia</td>
<td>Emotional</td>
<td>Apical</td>
<td>40</td>
<td>60</td>
<td>27</td>
<td>0.98</td>
<td>37</td>
<td>Normal</td>
</tr>
<tr>
<td>13</td>
<td>59</td>
<td>F</td>
<td>Hypertension, hypercholesterolemia</td>
<td>Emotional</td>
<td>Apical</td>
<td>35</td>
<td>65</td>
<td>11</td>
<td>0.98</td>
<td>39</td>
<td>Normal</td>
</tr>
<tr>
<td>14</td>
<td>64</td>
<td>F</td>
<td>Hypercholesterolemia</td>
<td>Emotional</td>
<td>Apical</td>
<td>55</td>
<td>60</td>
<td>12</td>
<td>0.96</td>
<td>43</td>
<td>Normal</td>
</tr>
</tbody>
</table>

EF, ejection fraction; F, female; FFR, fractional flow reserve; IMR, index of microcirculatory resistance; M, male; NA, not available; OCT, optical coherence tomography.

cardiovascular risk factors, suggesting that the presence of atherosclerotic plaques could be a finding with no causal relationship with TKS. Despite the lack of epicardial lesions, we show that some degree of microvascular dysfunction may be present. In the absence of atherosclerotic alteration on epicardial vessels, this dysfunction can be caused either by thrombi or by intense and temporary spasms in the coronary microcirculation. Thrombi embolism from the epicardial vessel can be excluded by our OCT findings. A significant
negative correlation was also found between the extent of microvascular dysfunction and the time from symptom onset to IMR analysis (R - 0.69; P = .012), in line with previous data (Figure). In particular, a normal IMR value was found in a patient studied 10 days after the appearance of initial symptoms, suggesting the reversibility of this microvascular dysfunction.

In conclusion, our study shows that LAD of TKS patients may exhibit some degree of microvascular dysfunction in the absence of atherosclerotic or vulnerable plaque. Future studies are needed to further determine the causes of this dysfunction.

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Experience With the Absorb Bioresorbable Vascular Scaffold in Various Scenarios of Congenital Heart Disease

**Experiencia con el armazón vascular bioabsorbible Absorb en varios escenarios de cardiopatías congénitas**

To the Editor,

Percutaneous treatment in children with congenital heart disease has continued to evolve in recent years. Stent implantation plays an important role in some of these diseases.1 However, because vessel growth is rapid in infants, there is a need for subsequent stent redilations, surgical removal, or complicated surgical intervention. This problem could be resolved by a bioresorbable stent.2

The ideal bioresorbable stent in pediatric patients would provide sufficient radial force and acceptable flexibility for deployment into distal arteries, should be low profile, and would disappear without creating a significant local inflammatory response or systemic toxicities.

The bioresorbable vascular scaffold (BVS) (Abbott) is the most advanced, well-studied bioresorbable stent.3 The product is available in multiple sizes and it fully reabsorbs within 3 years and provides mechanical support for up to 6 to 12 months.4 Due to the features described, the Absorb-BVS, could meet the needs of pediatric patients.

The aim of our study was to describe our experience with BVS in the setting of various vascular lesions in children with congenital heart disease.

A retrospective, interventional and clinical follow-up study was conducted. The primary outcome measures were procedural success and complication rates. Informed consent was obtained in all patients. BVS implantation was performed in 8 patients as an alternative to surgical intervention or bare-metal stent implantation, bridging the patient in some cases to a future more definitive surgical intervention. The median age was 3.8 months (10 days-6.3 years) and the median weight was 3.95 kg (range, 2.3-20). The demographic data and types of vessel obstruction are summarized in the Table.

Based on the type of lesion, vascular access was gained via the femoral vein (n = 5) or the femoral artery (n = 2) and there was 1 hybrid procedure through the right ventricular free wall. Vessel lesion morphology and critical diameter were assessed angiographically. If there was pre-existing critical vessel obstruction and as a guide for proper implantation, we performed balloon predilation of the vessel in 5 patients. After the decision to implant a stent was confirmed, the Absorb-BVS was implanted. The stent sizes (mm) used were 3.5 x 12 (n = 5), 2.5 x 12 (n = 1), and 3 x 12 (n = 2). Vascular stenting was achieved in all patients. No recoil of the stent was observed. In 6 patients, subsequent stent overdilation was necessary to achieve the maximum diameter of the native vessel. The angiographic result was satisfactory in all patients (Figure). There were no procedure-related complications. Improvement in hemodynamic parameters or clinical recovery was achieved in all patients in the acute follow-up.

Patients underwent serial echocardiographic and clinical follow-up to evaluate hemodynamic status and the patency of the stented area. The median follow-up period was 82.1 days (range, 3-155). Angiographic reevaluation was performed in 2 patients. In patient No. 2, a balloon over dilation was performed 71 days after implantation; however, we were unable to achieve clear improvement and 5 days later the patient was accepted for total surgical correction. Patient No. 5, was reevaluated 142 days after the procedure to check vessel integrity and growth. Angiography showed the permeability and lumen integrity of the stented vessel. Four more patients underwent successful corrective surgery (Table).

With the concept of temporary scaffolding, biodegradable stents might solve one of the greatest challenges in pediatric congenital heart disease intervention: adaptation to growth.1 So far, there is little published information on BVS use in pediatric patients.5

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