The findings of this small series of patients with angiographically insignificant lesions suggested destabilization of vulnerable plaques as the most probable cause of ACS. OCT has been shown to be a useful technique in the characterization of substrates causing ACS, as it can detect vulnerable plaques, plaque rupture, thrombi, superficial calcified nodules, and plaque erosion. Identification of these substrates could have important prognostic and therapeutic implications.

One limitation of this study is its small sample size. Further study limitations include the lack of OCT studies of the other coronary arteries not considered as the cause of the clinical manifestations and the lack of a control group. Furthermore, we did not perform coronary vasomotor tests and, finally, we did not definitively identify the cause of ACS in 6 patients with identification of stable plaques only. In these patients, the manifestations may have been the result of coronary vasospasms, embolism, or even acute myocarditis. Nevertheless, when coronary angiography fails to clearly detect any causative lesions in patients with ACS despite clinical suspicion, imaging techniques such as OCT can identify unstable coronary substrates in a substantial proportion of individuals (66.7% of our series). In such cases, the technique could be used as an additional imaging technique to try to clarify the cause of ACS.

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Balloon Pulmonary Angioplasty for Inoperable Patients With Chronic Thromboembolic Pulmonary Hypertension. Preliminary Experience in Spain in a Series of 7 Patients

Angioplastia pulmonar con balón en la hipertensión pulmonar tromboembólica crónica no operable. Experiencia inicial en España en una serie de 7 pacientes

To the Editor,

Chronic thromboembolic pulmonary hypertension (CTEPH) is caused by recurrent, unresolved pulmonary embolisms. The thrombi form intraluminal walls and membranes that replace the normal intima of the pulmonary arteries and cause obstruction.
candidates for surgery because of distal location or poor clinical and/or hemodynamic status, despite optimized medical treatment. This case series describes our experience of using BPA. To our knowledge, this is the first such case series published in Spain.

We performed 22 BPs in 7 patients (5 women; mean age, 61 years), all of whom had New York Heart Association (NYHA) functional class III-IV, despite receiving triple-combination therapy, which included systemic prostanoids in 6 patients. A multidisciplinary team confirmed inoperability and then made a joint decision to perform BPA. A mean of 3 procedures was performed per patient and each procedure involved treatment to a mean of 2.4 segments and 1.2 lobes. In 6 patients, there was significant hemodynamic improvement during follow-up (mean, 6 months [range, 1–18 months]), with a decrease in mean pulmonary artery pressure (mPAP) and pulmonary vascular resistance, and an increase in cardiac index (CI). In addition, right ventricular wall stress decreased, leading to lower N-terminal pro-B-type natriuretic peptide (NT-pro-BNP) levels, and improved NYHA functional class in all patients. We were able to discontinue prostanoid treatment in 3 out of 6 patients. These data are shown in the Table, and in Tables 1 and 2 of the supplementary material. Two patients had acute reperfusion pulmonary edema as a complication after their first BPA procedure. In 1 patient, the episode was subclinical and was managed with diuretics. The other patient required mechanical ventilation and circulatory support with venoarterial extracorporeal membrane oxygenation (ECMO). She died 8 days post-BPA from a brain hemorrhage (Figures 1 and 2 of the supplementary material). There were no BPA complications involving pulmonary arterial rupture.

In 2001, Feinstein et al. demonstrated improved hemodynamics and exercise tolerance after performing BPA in 18 patients with inoperable CTEPH, although 11 had post-BPA acute reperfusion pulmonary edema and 1 patient died as a result. However, despite these findings, the technique was not widely accepted as alternative or coadjuvant therapy in selected patients with CTEPH until 3 years ago, after the publication of some case series, most of which were Japanese. In some series, the technique has been refined by using intravascular ultrasound or optical coherence tomography and, more importantly, by treating only 1 or 2 segments per session, which reduces the onset of acute reperfusion pulmonary edema. Published hemodynamic results show a decrease of as much as 47% in mPAP and of 65% in pulmonary vascular resistance. In our series, we achieved a mean decrease of 28% in mPAP and of 41% in pulmonary vascular resistance, with unequal distribution among treated patients. The improvement obtained with BPA is similar to that achieved with pulmonary thromboendarterectomy (42% reduction in mPAP and 64% reduction in pulmonary vascular resistance) and is significantly better than the reported outcome of medical treatment, with a 9% reduction in mPAP and a 25% reduction in pulmonary vascular resistance. Acute reperfusion pulmonary edema is the commonest complication of BPA, and the leading cause of death (1.4%–10%). This complication has a high subclinical incidence of 60%, but mechanical ventilation is required in only 6% of cases. Variables showing a high correlation with the onset of acute reperfusion pulmonary edema are the number of lobes and segments treated per procedure, pre-BPA mPAP > 35 mmHg, and poor clinical and hemodynamic status preprocedure. The patient in our series who died from acute reperfusion pulmonary edema had NYHA class IV, despite receiving triple-combination therapy with systemic prostanoids. She had a poor hemodynamic profile, with a CI of 2.02 L/min/m² and a pre-BPA mPAP of 62 mmHg, and only 2 segments were treated in a single lobe. The other complication associated with BPA is pulmonary artery wall perforation or rupture, which is a life-threatening, albeit rare, event.

In our experience, and in agreement with the literature, we can confirm that BPA is an effective therapeutic alternative in selected patients with inoperable CTEPH, because it improves hemodynamics, functional capacity, and biomarkers and reduces the need for prostanoid therapy. However, because of the significant incidence of serious peri-procedural complications, BPA should be used appropriately and in carefully selected patients.

**SUPPLEMENTARY MATERIAL**

Supplementary material associated with this article can be found in the online version available at doi:10.1016/j.rec.2015.02.004.

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**Satisfaction With Medical Care in Patients With Atrial Fibrillation Treated With Vitamin K Antagonists Versus New Oral Anticoagulants**

Satisfacción con el cuidado médico de pacientes con fibrilación auricular anticoagulados con antagonistas de la vitamina K o nuevos anticoagulantes

To the Editor,

Oral anticoagulants (OAC) significantly reduce the risk of thromboembolism in patients with nonvalvular atrial fibrillation. In addition to oral anticoagulation with the traditional vitamin K antagonists (VKA), for the past few years, new oral anticoagulants (NOAC) have been available, whose efficacy and safety are at least similar to those of VKA. 1 One of the advantages of these drugs lies in the stability of their anticoagulant action, obviating the need for systematic follow-up and thus making them more convenient for patients to use. It is important to determine patients’ opinion of the treatment and care provided to them, but this is often overlooked. The objective of our study was to analyze satisfaction among patients with nonvalvular atrial fibrillation with OAC-related medical care and to compare those receiving VKA or NOAC. To do this, we studied the first 1247 patients included in the FANTASIA registry. This per-protocol analysis included consecutive patients treated with VKA and NOAC (at a proportion of 4:1) who had received OAC for at least 6 months prior to the inclusion

**Table 1**

General Characteristics of Patients Taking Vitamin K Antagonists and New Oral Anticoagulants in the FANTASIA Study

<table>
<thead>
<tr>
<th></th>
<th>VKA (n = 964)</th>
<th>NOAC (n = 283)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>History of heart disease</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Previous heart disease</td>
<td>50.3</td>
<td>40.88</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Heart failure</td>
<td>30.68</td>
<td>21.46</td>
<td>.01</td>
</tr>
<tr>
<td>Coronary disease</td>
<td>20.02</td>
<td>14.53</td>
<td>.03</td>
</tr>
<tr>
<td>Coronary revascularisation</td>
<td>11.57</td>
<td>9.80</td>
<td>.58</td>
</tr>
<tr>
<td>Patient has coronary stents</td>
<td>10.06</td>
<td>7.09</td>
<td>.13</td>
</tr>
<tr>
<td>Dilated cardiomyopathy</td>
<td>13.48</td>
<td>8.45</td>
<td>.02</td>
</tr>
<tr>
<td>Left ventricular hypertrophy HT</td>
<td>17</td>
<td>13.85</td>
<td>.20</td>
</tr>
<tr>
<td>Other structural heart disease</td>
<td>10.36</td>
<td>9.46</td>
<td>.86</td>
</tr>
<tr>
<td>Other tachyarrhythmia, not AF</td>
<td>6.74</td>
<td>7.43</td>
<td>.68</td>
</tr>
<tr>
<td>Previous bradyarrhythmia</td>
<td>7.75</td>
<td>2.7</td>
<td>.01</td>
</tr>
<tr>
<td>Patient has a pacemaker</td>
<td>7.95</td>
<td>4.05</td>
<td>.09</td>
</tr>
<tr>
<td>Ejection fraction, %</td>
<td>58.33 ± 10.5</td>
<td>60.28 ± 10.7</td>
<td>.02</td>
</tr>
</tbody>
</table>

**Data related to AF**

Type of AF

- Paroxysmal | 27.07 | 30.75 | .08 |
- Persistent  | 21.12 | 25.67 | .06 |
- Permanent    | 51.81 | 43.58 | .05 |
- Previous electrical cardioversion | 18.51 | 20.95 | .35 |
- Previous ablation   | 3.42 | 3.38 | .97 |

**Rhythm control strategy**

- CHADS2 score | 2.31 ± 1.2 | 2.19 ± 1.1 | .12 |
- CHA2DS2-VASc score | 3.78 ± 1.5 | 3.6 ± 1.6 | .09 |
- HAS-BLED score | 1.98 ± 1.0 | 1.92 ± 1.0 | .32 |

**Sinus rhythm at baseline ECG**

- 31.76 | 42.17 | .01 |

**Pharmacological treatment**

- Diuretics | 61.87 | 51.01 | .01 |
- Aldosterone antagonists | 15.9 | 10.81 | .03 |
- ACEI | 32.29 | 27.36 | .11 |
- ARB | 40.34 | 43.24 | .37 |
- Statins | 57.44 | 52.36 | .12 |
- Antiplatelet agents | 10.36 | 8.11 | .25 |
- Beta-blockers | 60.97 | 57.77 | .32 |
- Digoxin | 20.12 | 17.23 | .27 |
- Calcium antagonists | 14.79 | 13.51 | .51 |
- Dihydropyridines | 14.79 | 13.51 | .51 |
- Verapamil | 2.52 | 2.7 | .93 |
- Diltiazem | 8.45 | 6.76 | .32 |
- Antiarrhythmic agents | 23.84 | 27.7 | .18 |

ACEI, angiotensin-converting enzyme inhibitors; AF, atrial fibrillation; ARB, angiotensin receptor blocker; COPD, chronic obstructive pulmonary disease; ECG, electrocardiogram; HT, hypertension; NOAC, new oral anticoagulants; VKA, vitamin K antagonists.

Data are expressed as mean ± standard deviation (quantitative variables) and percentages (qualitative variables).