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. Pulmonary thromboendarterectomy is the treatment of choice and offers the only potential cure for CTEPH.[1] However, almost 40% of patients with CTEPH are inoperable, due to the location of the peripheral thrombus and/or comorbidities.

Patients who are not candidates for pulmonary thromboendarterectomy are prescribed specific medication for pulmonary hypertension, but many of them have persistent poor functional and hemodynamic status, despite medical treatment. For these patients, balloon pulmonary angioplasty (BPA) has been suggested as a coadjuvant therapy in recent years (Figure).

Since 1996, we have treated 188 patients with CTEPH at our unit, 100 of whom received pulmonary thromboendarterectomy and 88 medical treatment.[2] In May 2013, we started performing BPA as coadjuvant therapy in patients with CTEPH who were not

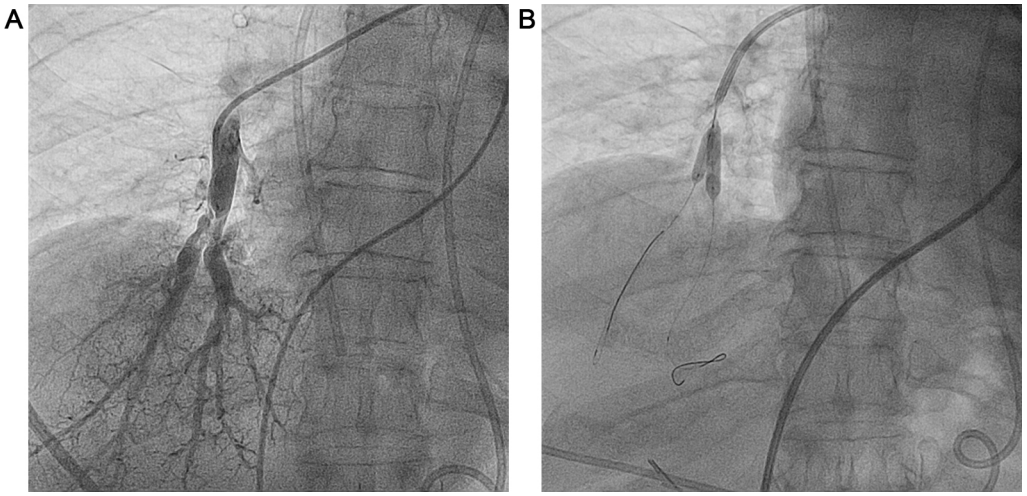

Figure. Chronic thromboembolic pulmonary hypertension. A: Membranes in right lower lobe segmental artery. B: Kissing balloon pulmonary angioplasty.

Table
Hemodynamic Parameters, Functional Class and Biomarkers at Baseline and After Balloon Pulmonary Angioplasty Procedures

	Baseline values	End values	Mean variation, %	*P*
Mean pulmonary artery pressure, mmHg	56 ± 17	36 ± 10	−28	< .06
Pulmonary vascular resistance, UW	11.78 ± 4	6.1 ± 2.2	−41	< .02
Cardiac index, L/min/m^2	2.28 ± 0.4	2.64 ± 0.6	+15.7	< .1
NYHA functional class I-IV	3.8 ± 0.2	2.3 ± 0.2	+39	< .001
NT-pro-BNP, pg/dL	1366 ± 929	646 ± 677	−52	< .1

NT-pro-BNP, N-terminal pro-B-type natriuretic peptide; NYHA, New York Heart Association.

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 BPAs 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[3] 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.[4] 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 29% reduction in pulmonary vascular resistance.[5] 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/m^2 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 periprocedural 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 FANTASIIA registry.[2] 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 FANTASIIA Study

	VKA (n = 964)	NOAC (n = 283)	*P*
Age, y	74.03 ± 9.4	72.69 ± 9.1	.03
Women	42.35	44.26	.56
Risk factors and comorbidities			
History of HT	80.78	82.43	.52
History of hyperlipidemia	55.33	50.34	.13
History of diabetes mellitus	31.08	25.33	.06
Smoking			
Current smoker	4.53	4.73	.90
Recent exsmoker, < 1 y	2.11	3.72	.14
Longstanding exsmoker, > 1 y	31.89	29.73	.61
COPD	17	17.23	.93
Renal failure	21.13	12.5	< .001
History of cancer	9.36	4.73	.01
Peripheral artery disease	7.04	6.76	.87
Past stroke	14.79	19.25	.07
Past noncerebral embolism	2.21	3.38	.26
Thyroid dysfunction	13.98	10.13	.06
Drug or alcohol abuse	4.12	3.72	.75
Previous major bleeding	2.41	6.76	.05
History of heart disease			
Previous heart disease	50.3	40.88	< .001
Heart failure	30.68	21.46	.01
Coronary disease	20.02	14.53	.03
Coronary revascularisation	11.57	9.80	.58
Patient has coronary stents	10.06	7.09	.13
Dilated cardiomyopathy	13.48	8.45	.02
Left ventricular hypertrophy HT	17	13.85	.20
Other structural heart disease	10.36	9.46	.86
Other tachyarrhythmia, not AF	6.74	7.43	.68
Previous bradyarrhythmia	7.75	2.7	.01
Patient has a pacemaker	7.95	4.05	.09
Ejection fraction, %	58.33 ± 10.5	60.28 ± 10.7	.02
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	38.73	41.55	.38
$CHADS_2$ score	2.31 ± 1.2	2.19 ± 1.1	.12
CHA_2DS_2-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			
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).