

segment in the remaining 3. In all patients, enhancement was confirmed in the area of mitral valve contact with the septum. There was no spread to distal territories (Figure B). The septal artery-dependent myocardial mass was 20.21 g (range, 14.03 g-28.00 g), corresponding to 9.56% (5.2%-13.8%) of total myocardial mass. Troponin I concentration was 42.1 ± 27.2 ng/mL (range, 18-89 ng/mL).

In most patients, 3D TTE with contrast allowed precise localization and quantification of myocardial tissue dependent on the selected septal artery. It also allowed immediate confirmation of the real extent of contrast, whereas with 2D TTE with contrast, we cannot always be certain that all segments have been analyzed. In our cases, septal artery myocardial distribution appeared similar to necrosis distribution on post-ASA cardiac magnetic resonance (CMR). This distribution was noted predominantly at the junction of the anterior and inferior septa in the basal left ventricle and extended to the inferior portion of the midventricular septum.² Without the availability of CMR after ASA, this concordance could not have been validated. Furthermore, 3D TTE with contrast allowed quantification of the septal artery-dependent myocardial mass in a similar way to CMR study of necrotic mass. The value obtained for contrast mass (15.6 g-33.6 g) was similar to the necrotic mass values on post-ASA CMR published by Valeti et al³ (16 g \pm 7 g with 1.7 mL \pm 0.4 mL of alcohol) and Yuan et al⁴ (27.9 g \pm 13.1 g with 2.6 mL \pm 1.3 mL). It is possible that the pre-ASA contrast study showed the entire septal artery-dependent vascular network, while post-ASA CMR showed only the necrotic area. There would undoubtedly have been dependent areas that did not necrose, and, in fact, as shown in the literature, necrosis also depends on the volume of alcohol injected.³ Using 3D TTE with contrast, information can be obtained similar to that from CMR, but prior to ASA, allowing selection of the most appropriate septal artery branch and prediction of the subsequent infarct area. In addition, CMR is unsuitable for patients with pacemakers.

Three-dimensional TTE requires a machine with a 3D transthoracic probe. Image acquisition and intraprocedural analysis of the localization and extent of contrast with 3D TTE did not take longer than with 2D TTE. Quantification of the septal artery-dependent mass was done manually on an external work station and took approximately 15 to 20 minutes. Although this can be done during the procedure, it would be desirable to have a specific quantification program that reduced estimation time.

The main limitation of this study was the small sample size, due to the infrequency of ASA. This prevented us from establishing correlations between mass and enhancement and other variables.

In this study, we demonstrate the ease and usefulness of 3D TTE with contrast during ASA, as it allowed precise estimation of the target septal artery distribution and its dependent myocardial tissue size.

Further studies are needed to evaluate the potential usefulness of 3D TTE with contrast in selecting the target septal artery in complex cases with various possible branches, and in determining alcohol volume according to the dependent myocardial mass, which could reduce the need for permanent pacemakers.

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Acute Coronary Syndrome in Patients With Normal Coronary Arteries: An Optical Coherence Tomography Study



Síndrome coronario agudo en pacientes con arterias coronarias normales: estudio con tomografía de coherencia óptica

To the Editor,

Acute coronary syndrome (ACS) in patients with normal coronary arteries has a prevalence of between 8% and 25%.^{1,2} Several mechanisms have been implicated in its etiopathogenesis, including coronary vasospasm, hypercoagulable states, and embolization.² Coronary angiography can assess the extent of stenosis but not the state of atheromatous plaques. The technique is therefore of limited use when complicated plaques are present, particularly in the absence of significant lesions.³ Atherosclerotic plaques can, however, be characterized by optical coherence tomography (OCT).^{2,3} Previous studies with this technique in ACS have focused on characterizing the substrates of angiographically

Table

Baseline Characteristics of the Study Patients

Demographic data and concurrent diseases	
Patients	21
Age, y	54.7 \pm 14.79
Female	8 (38.1)
Hypertension	10 (47.6)
Diabetes mellitus	2 (9.5)
Dyslipidemia	11 (52.4)
Smoker	7 (33.3)
Exsmoker	5 (23.8)
Diagnosis on admission	
NSTEACS	14 (66.7)
STEACS	7 (33.3)
Coronary angiography findings	
LAD involvement	14 (66.7)
Cx involvement	4 (19.05)
RCA involvement	3 (14.3)

Table (Continued)

Baseline Characteristics of the Study Patients

<i>Quantitative coronary analysis (QCA)</i>	
Reference diameter, mm	3.20 ± 0.81
Minimum luminal diameter, mm	2.37 ± 0.90
Stenosis, %	27.03 ± 14.84
Length, mm	8.6 ± 4.07
<i>Quantitative coronary analysis (OCT)</i>	
Reference area, mm ²	9.85 ± 4.29
Stenotic area, mm ²	6.93 ± 4.3
Mean stenotic diameter, mm	2.87 ± 0.84
Stenosis, %	32.9 ± 20.62
Length, mm	8.19 ± 4.75
<i>Qualitative coronary analysis (OCT)</i>	
Stable plaque	9 (42.9)
Ruptured plaque	10 (47.6)
Eroded plaque	2 (9.5)
Thrombus	8 (38.1)
Calcified nodules	3 (14.3)
Normal	1 (4.8)

Cx, circumflex artery; LAD, left anterior descending artery; NSTEACS: non-ST-segment elevation acute coronary syndrome; RCA, right coronary artery; STEACS: ST-segment elevation acute coronary syndrome.

significant lesions.^{4,5} None, however, have studied unstable substrates in patients with insignificant findings on angiography. In a recent study of patients who were resuscitated after sudden death and had lesion-free coronary arteries, intracoronary ultrasound allowed identification of unstable plaque substrates in all patients.⁶ OCT has demonstrated better resolution than intracoronary ultrasound in the characterization of atheromatous plaques.^{4,5} The objective of our study was to identify the presence of unstable substrates using OCT in patients with ACS and angiographically normal coronary arteries or angiographically insignificant lesions (< 50%).

Between April 2012 and September 2014, 1178 patients with high-risk ACS underwent coronary angiography. Of these, 53 (5.14%) had normal coronary arteries and 58 (5.63%) had angiographically

insignificant lesions. Of these 111 patients, 21 met the following criteria: a) clinical manifestations of angina or equivalent; b) electrocardiographic findings indicative of ischemia; c) elevated levels of biomarkers of myocardial damage (troponin I or ultrasensitive troponin I), and d) wall motion abnormalities on echocardiography, magnetic resonance imaging, or ventriculography. Based on angiographic irregularities and electrocardiographic and wall motion abnormalities, the artery considered the cause of ACS was selected and OCT (Dragonfly™ Duo OCT Imaging Catheter, St Jude Medical; St. Paul, Minnesota, United States) performed. Patients with ST-segment elevation ACS underwent emergency catheterization. None received fibrinolytic agents. In patients with non-ST-segment elevation ACS, an early invasive strategy was followed with coronary angiography in the first 24 to 48 hours. Only 1 patient had a Q-wave infarction and all had elevated troponin I or ultrasensitive troponin I and creatine kinase.

The baseline characteristics of the patients are summarized in Table. The mean age was 54.7 ± 14.79 years and 7 patients (33.3%) presented with ST-segment elevation ACS. The artery most frequently considered the cause of the event was the left anterior descending artery (66.7%). Quantitative coronary angiography and OCT showed an acceptable correlation (Table). Qualitative analysis with OCT (Table) showed that 20 patients had signs of arteriosclerosis and 9 had stable plaques (Figure A). Of these 9 plaques, 2 were considered vulnerable as they had a very thin fibrous cap with a large necrotic core. In 8 patients, residual thrombotic material was found (Figure B), associated with the presence of ruptured plaques. Ten patients had ruptured plaques (Figure C), 2 had plaque erosion (Figure D), and 3 had superficial calcified nodules with thrombus (Figure E) or rupture of a thin fibrous cap. Eleven patients had several remarkable findings, 6 had only stable plaques, and 1 had normal coronary arteries with no findings on OCT; 5 patients (22.7%) had anterior ST-segment elevation ACS and apical akinesis with subsequent normalization of ventricular function, indicative of tako-tsubo syndrome. Only 1 patient had a normal left anterior descending artery on OCT and 4 had unstable substrates. In summary, of the 21 patients studied, 20 showed signs of coronary arteriosclerosis and 14 (66.7%) had signs of complicated atherosclerotic plaque that could cause ACS, despite the absence of angiographically significant lesions on coronary angiography.

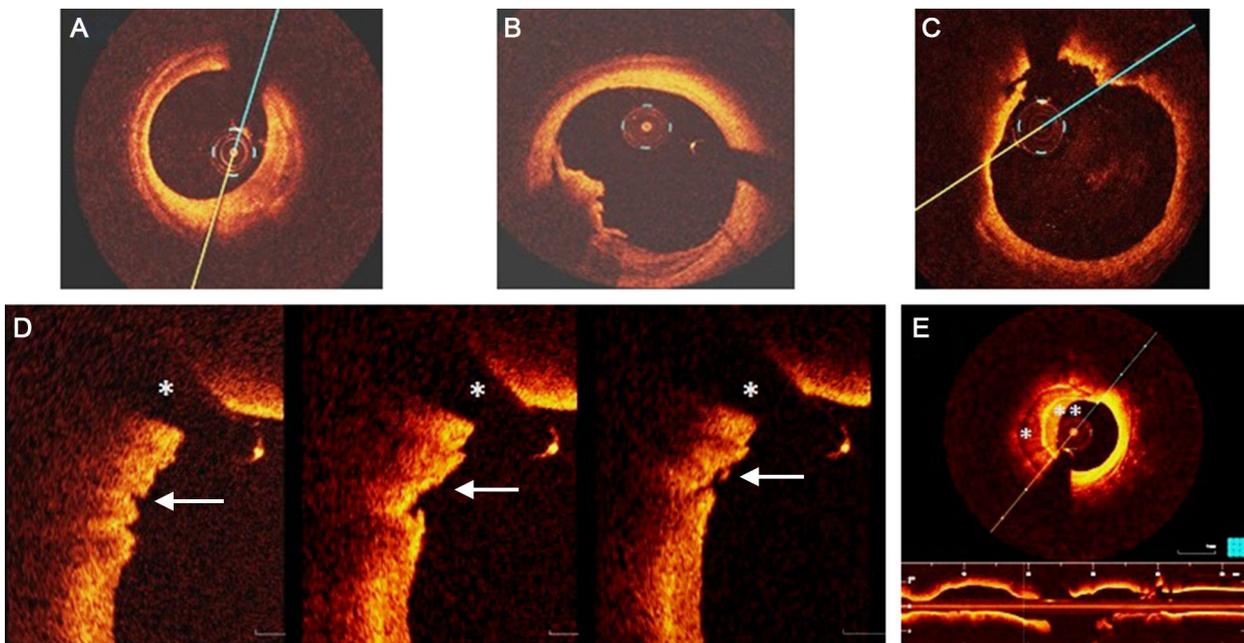


Figure. Qualitative coronary analysis (optical coherence tomography). A: Stable plaque. B: Residual ruptured plaque. C: Ruptured plaque D: Consecutive panels that show rupture of the intima, indicating plaque erosion (arrows), acoustic shadow of the guide (*). E: Superficial calcified nodule (*) with adhered laminar thrombus (**).

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

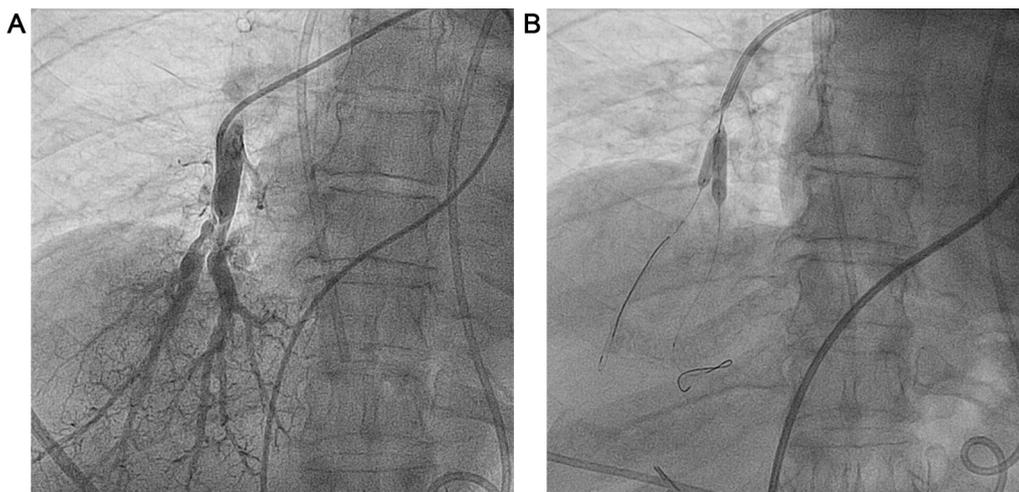


Figure. Chronic thromboembolic pulmonary hypertension. A: Membranes in right lower lobe segmental artery. B: Kissing balloon pulmonary angioplasty.