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Coronary Lithoplasty: Initial Experience in Coronary Calcified Lesions

Litoplastia coronaria: experiencia inicial en lesiones calcificadas

To the Editor,

Coronary lithoplasty is a novel treatment that uses high-energy mechanical pulses to break up calcium deposits in atherosclerotic heart disease. These pulses, known as *shockwaves*, are also used to break up calcium in other locations, especially the kidneys (lithotripsy). Their use in the heart is very new and there have been very few reported cases.[1,2]

Coronary lithoplasty involves the use of a nylon ball that, once positioned at the target site, is connected to an external unit that generates pulsatile mechanical waves lasting microseconds. The pulses are emitted on demand and deliver an intermittent pressure of 50 to 60 atm to the vessel wall. This pressure is 3 to 4 times higher than that achieved using other devices. Balloon size must be such to achieve a balloon to artery ratio of 1:1, as the mechanical energy is generated and transmitted along the vessel wall when the balloon makes contact with the arterial intima.[1] As the waves travel along the wall and through the connective tissue, they cause microfractures in the calcified tissues, allowing good lesion expansion and correct stent placement.[2]

We present the cases of 3 patients with multivessel coronary artery disease in whom 6 severely calcified lesions were successfully treated with coronary lithoplasty. The 3 patients all had characteristics that typically call for interventional treatment: advanced age and functional class, high surgical risk, previous revascularization, and a high probability of rotational atherectomy.

The lithoplasty balloon was successfully used to treat all

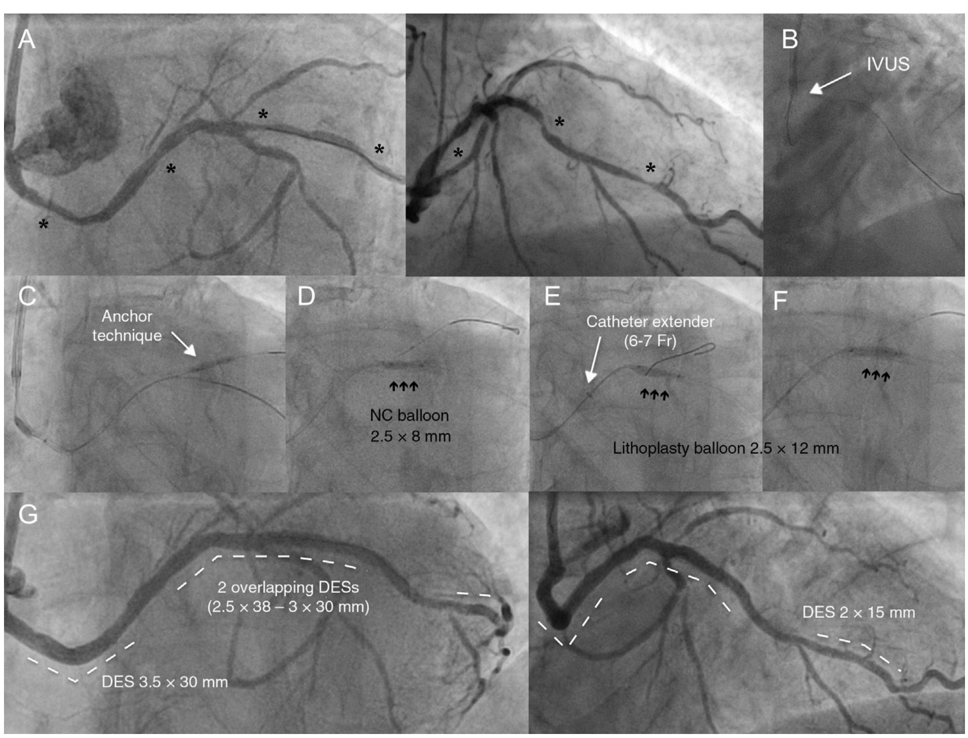

Figure 1. A: Calcified, tortuous circumflex artery with distal, medial, and proximal lesions (asterisks). B: The intravascular ultrasound (IVUS) catheter could not be advanced. C: Application of the side-branch anchor technique did not allow advancement of the lithoplasty balloon. D: Underexpansion of the 2.5-mm noncompliant (NC) balloon in the medial area helped advance the catheter extender and move the lithoplasty balloon forward (E). E: Unsuccessful dilation with 40 pulses. F: Opening of lesion after 60 pulses. G: Outcome after placement of 4 drug-eluting stents (DES).

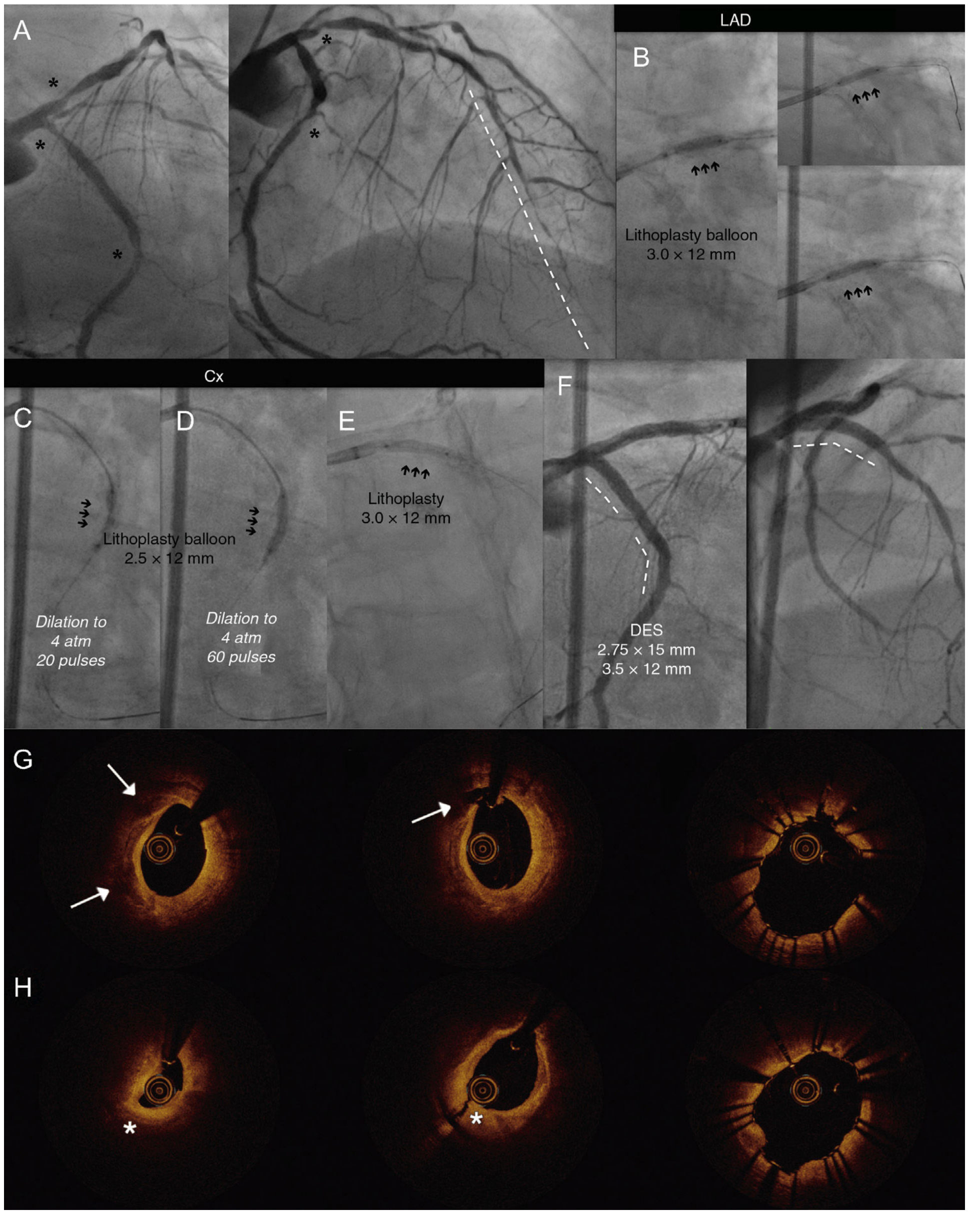

Figure 2. A: Calcified lesions in the ostial and medial-distal circumflex artery (Cx) and ostial left anterior descending artery (LAD) (asterisks) with diffuse disease in the distal LAD (dotted line). Coronary lithoplasty with a 3-mm balloon in the ostial DA (B) and the medial-distal Cx. Dilation to 4 atm and delivery of 20 pulses (C) and 60 pulses (D). E: Coronary lithoplasty with a 3-mm balloon in the ostial LAD. F: Placement of 3 drug-eluting stents, with good results; optical coherence tomography images before coronary lithoplasty; fracture of calcified plaque (arrows) and dissection of fibrous plaque (asterisks). G and H: Images after coronary lithoplasty and placement of stent in the ostial (G) and medial-distal Cx (H).

6 lesions and there were no intraprocedural complications, such as dissections or perforations.

The first patient was a 73-year-old man with severe kidney failure who had undergone bilateral iliofemoral bypass. He had triple-vessel heart disease. Of note, the circumflex artery (Cx) arose in the right sinus and had 3 features that contraindicated percutaneous treatment: calcification, severe tortuosity, and diffuse distal disease (Figure 1A, Video 1 of the supplementary data). The left anterior descending artery (LAD) was treated first, using coronary lithoplasty followed by drug-eluting stent (DES) placement. The Cx was treated in the same procedure using a multipurpose catheter and 2 guide catheters. Following unsuccessful advancement of the catheter via intravascular ultrasound (Figure 1B) and failure to successfully apply the side-branch anchor technique (Figure 1C), the medial lesion was partially dilated with a 2.5-mm noncompliant balloon (Figure 1D) (Video 2 of the supplementary data). This maneuver allowed us to advance the catheter extender (6-7 Fr) and correctly position the 2.5-mm lithoplasty balloon; the lesion was successfully expanded with 60 pulses (Figure 1E-F). The proximal and medial lesions were treated with a 3-mm lithoplasty balloon and placement of 4 DES (Figure 1G and Video 3 of the supplementary data).

The second patient was a 63-year-old diabetic man with triple-vessel disease. Surgical revascularization had not been possible in 2014 because of the poor condition of the distal bed of the LAD. The patient's condition was initially treated with rotational atherectomy applied to the dominant CX and placement of 3 DESs. In 2018, a coronary angiogram performed to investigate refractory angina showed calcified lesions in the Cx (an ostial lesion and a lesion distal to the stent) and the LAD (an ostial lesion) (Figure 2A and Video 4 of the supplementary data). The 3 lesions were treated with coronary lithoplasty (Figure 2B-E) and the results were excellent (Figure 2F and Video 5 of the supplementary data).

Optical coherence tomography showed several fracture points in the intimal and even the medial plaque (Figure 2G and H).

The third patient was an 81-year-old woman with heart disease not amenable to surgical treatment due to distal disease. In an initial step, the proximal right coronary artery was treated with rotational atherectomy. The distal truncus arteriosus and the proximal-medial LAD were both treated in a subsequent step (Video 6 of the supplementary data). The distal LAD was treated with a noncompliant balloon and a 2-mm cutting balloon. The proximal LAD and distal truncus arteriosus were treated with coronary lithoplasty and 2 overlapping DESs. The distal truncus arteriosus required postdilation with a double-layered balloon (Video 7 of the supplementary data).

Based on our preliminary experience, coronary lithoplasty is *a)* a safe and effective procedure for cases in which rotational atherectomy is not an option; *b)* a simple procedure with no learning curve that can be performed using standard guide catheters, and *c)* a procedure that permits protection of the lateral branches. The current balloon profile, however, is lower than that of state-of-the-art noncompliant balloons and needs to be improved, as it requires a catheter of at least 6 Fr. Lithoplasty balloons are currently available in just one length (12 mm) and have a diameter ranging from 2.5 to 4 mm.

In conclusion, coronary lithoplasty is a safe and effective procedure for treating severely calcified coronary lesions and will probably become an important addition to the armamentarium for modifying calcified plaque.[3]

APPENDIX. SUPPLEMENTARY DATA

Supplementary data associated with this article can be found in the online version, at https://doi.org/10.1016/j.rec.2018.11.017.

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Regression of Cardiac Amyloidosis Following Autologous Stem Cell Transplant in Patients With Atypical Magnetic Resonance Imaging Findings

Regresión de amiloidosis cardiaca tras un trasplante autólogo de células progenitoras en pacientes con hallazgos atípicos en la resonancia magnética

To the Editor,

A diagnosis of cardiac amyloidosis often requires histological evidence of amyloid deposits, either in the heart itself or in biopsies from other affected organs because prognosis and treatment vary considerably according to the type of amyloidosis.[1] The appearance of global, subendocardial late gadolinium enhancement (LGE) has been described as highly characteristic of cardiac amyloidosis, and is associated with a ~ 5-fold increase in mortality.[2] However, ~ 7% of the patients present with an atypical LGE pattern such as focal subendocardial or midmyocardial LGE for which the prognostic significance and therapeutic implications are unclear.[3] Regression of cardiac light-chain (AL) amyloidosis has been reported following autologous stem cell transplant (ASCT) or chemotherapy in patients with the characteristic LGE pattern for cardiac amyloidosis.[4,5] However, little is known about the impact of these therapies in patients with atypical patterns of LGE.

Here, we present 3 patients with AL amyloidosis with an atypical LGE pattern who underwent ASCT and serial imaging with cardiac magnetic resonance imaging (RMI) and transthoracic echocardiogram (TTE) before and after treatment (respectively 10, 21 and 23 months). TTE with global longitudinal strain (GLS) and RMI (1.5T) including cine-RMI, LGE, and T_1-mapping were performed before and after ASCT. Left ventricular ejection fraction (LVEF), native T_1-relaxation times, and extracellular volume (ECV) fraction were calculated from RMI images using commercially available software (qmass, Medis Medical Imaging systems, Leiden, Netherlands). GLS was calculated from TTE images using QLAB software (Phillip Medical Systems, Andover, Massachusetts). Clinical evaluation with electrocardiogram and N-terminal pro-B-type natriuretic peptide (NT-proBNP) were also performed. Measurements from before ASCT were compared with those after ASCT using a *t* test.

Patients were aged 63, 64 and 73 years and 1 was female. All patients had biopsy proven AL amyloidosis and underwent ASCT with complete hematological response. These patients were at low risk for ASCT according to the Mayo Clinic staging system. Native myocardial T_1-relaxation time and ECV were increased at baseline and decreased following ASCT (Figure 1, T_1: 1186 ± 30 msec vs 1119 ± 50 msec, $P = .036$ and ECV: 41 ± 6% vs 28 ± 10%, $P = .026$). LGE was present in 2 patients in the subendocardial inferior and inferolateral walls, and in 1 patient in the subendocardial anterolateral wall. LGE persisted in the post-ASCT MRI but was slightly improved following ASCT (Figure 2). An "apical sparing" pattern of longitudinal strain (ie, abnormal in the basal and mid levels of the left ventricle but relatively normal in the apical levels) was present in all cases. GLS was abnormal at baseline and improved significantly after ASCT (14 ± 1% to − 20 ± 3%, $P = .037$),