Peripheral cannulation was used in 94.9% of RLMT cases (208/219). To achieve intracardiac repair, a cross clamp was adopted in 103 patients (47%) and alternatively induced ventricular fibrillation (IVF) in 116 patients (53%). Median CPB, cross clamp and IVF time were 47 minutes [IQR, 33-69 minutes), 40 minutes [IQR, 33-55 minutes], and 17 minutes [IQR, 13-25 minutes], respectively. The median intensive care unit (ICU) and hospital length of stay were 1 day [IQR, 1-1 day] and 4 days [IQR, 4-5.5 days], respectively. Blood transfusion was required in 8% (18/219) of the patients. The major complications rate was 2.7% (6/219) and comprised postoperative bleeding requiring reoperation (3/6) and vascular access complications (3/6). None of the patients in this group required conversion to traditional median sternotomy.

Logistic and linear regression models were used to identify risk factors for postoperative complications, blood transfusion requirement, and ICU or hospital stay. Peripheral cannulation was significantly associated with a shorter ICU stay in the multivariable analysis (coefficient: −0.44; 95% confidence interval, −0.79 to −0.98, $P = .012$) (table 1). None of the underlying treated CHD was associated with an increased risk of postoperative complications.

We began regularly using RLMT as a MICS strategy in 2013. In addition to comparable outcomes compared with median sternotomy and other approaches, we have also shown that in long term follow-up, the vast majority of patients treated with MICS were satisfied with the cosmetic result, and satisfaction was higher in patients undergoing RLMT or right anterior minithoracotomy than in patients with MS. While other centers have incorporated the use of RLMT for ASD repair,[3,5] we have successfully expanded the application of this strategy to numerous diagnoses of varying complexity without sacrificing clinical outcomes.[1] The lateralization process of our surgical approach has consequently evolved successfully over time.

Peripheral cannulation was associated with a shorter ICU stay. Early in our MICS experience, peripheral cannulation was used selectively for patients with a body weight greater than 30 kg, largely due to the anatomic limitations of the femoral vessels in smaller patients. We have since expanded the use of peripheral cannulation as stated above.

While MICS is able to reduce postoperative morbidity, this would not be possible without a coordinated multidisciplinary approach among all members of the operative and postoperative team.[2] Surgical outcomes for CHD are continuously improving, and MICS is currently emerging as the next developmental phase to maintain excellent outcomes while reducing the psychological and physical trauma of surgery, aspects that are particularly important to pediatric patients. Morbidity and mortality will certainly continue to be the most important outcomes for patients with CHD. However, the potential for emerging strategies, such as MICS via a RLMT to reduce postoperative trauma, and improve secondary considerations such as aesthetic outcomes, while maintaining quality standards, will play an increasingly important role in the implementation of cardiac surgery in the future.

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Apical ventricular hypertrophy in the transplanted heart: a 20-year single-center experience

Hipertrofia ventricular apical en el corazón trasplantado: experiencia de un centro en 20 años

To the Editor,

Left ventricular hypertrophy (LVH) is a common finding after heart transplant (HTx)[1] and is associated with a less favorable prognosis. Several variables have been related to a higher risk of severe LVH following transplant, such as patient age, obesity, prior diabetes or hypertension,[1] immunosuppressive therapy, and the possible presence of a primary graft disease that manifests after transplant.

Apical hypertrophic cardiomyopathy is a relatively uncommon variant, associated with increased wall thickness of the left ventricular apical segments and a reduction in the ventricular cavity.[3] The symptoms are nonspecific and diagnosis is often delayed.[3,4] The objective of our study was to describe the characteristics and clinical course of a cohort of patients with apical LVH following HTx.

A search was carried out in patients who had undergone HTx since 1988, in active follow-up. Apical LVH was defined by myocardial thickness > 15 mm in the apex of the left ventricle, or > 13 mm with a ratio of basal to apical segments > 1.5.[5] Eight

Table 1
Patients with apical ventricular hypertrophy following heart transplant

	Reason for HTx	Age at diagnosis, years	Sex	CVRF	Immunosuppression*	NYHA	GVD	History of acute rejection	Apical LVH, mm	History of atrial arrhythmias	Years since the diagnosis	Cardiac MR	BNP	NT-proBNP
1	Ischemia	68	Man	Hypertension dyslipidemia diabetes	Cyclosporine + Azathioprine	II	CAV 1	No rejection	15	No	13	—	145	2481
2	Peripartum	46	Woman	Hypertension	Tacrolimus + mycophenolate	I	CAV 1	No rejection	14	Flutter	8	Apical hypertrophy, 14 mm; no delayed gadolinium enhancement	116	269
3	Peripartum	39	Woman	Hypertension	Tacrolimus + mycophenolate	II	CAV 0	No rejection	15	No	8	Apical hypertrophy, 15 mm; delayed enhancement in apical mesocardium	108	483
4	Myocarditis	71	Man	Hypertension	Everolimus + mycophenolate + prednisone	II-III	CAV 1	2R	19	No	3	Apical hypertrophy up to 20 mm; mesocardial enhancement in the area with greatest hypertrophy	458	4390
5	Idiopathic dilated	65	Man	Hypertension	Tacrolimus + mycophenolate + prednisone	II-III	CAV 0	2R	23	No	1	—	384	17 895
6	Ischemia	54	Woman	Hypertension	Tacrolimus (change to everolimus) + mycophenolate	II	CAV 0	No rejection	19	No	3	Apical hypertrophy, 18 mm; inferolateral mesocardial gadolinium enhancement	145	903
7	Ischemia	39	Woman	No	Tacrolimus (change to everolimus) + mycophenolate + prednisone	II	CAV 3	No rejection	18	Flutter	14	—	254	4655
8	Idiopathic dilated	65	Woman	No	Tacrolimus + mycophenolate	II	CAV 0	No rejection	20	No	6	—	283	2930

BNP, brain natriuretic propeptide; CAV, cardiac allograft vasculopathy, nomenclature of the International Society for Heart and Lung Transplantation: CAV 0 (not significant), CAV 1 (mild), CAV 2 (moderate), and CAV 3 (severe); CVRF, cardiovascular risk factors; GVD, graft vascular disease; HTx, heart transplant; LVH, left ventricular hypertrophy; MR, magnetic resonance; NT-proBNP, amino-terminal fraction of brain natriuretic propeptide; NYHA, New York Heart Association.

* The tacrolimus doses used were < 15 mg/mL in all cases.

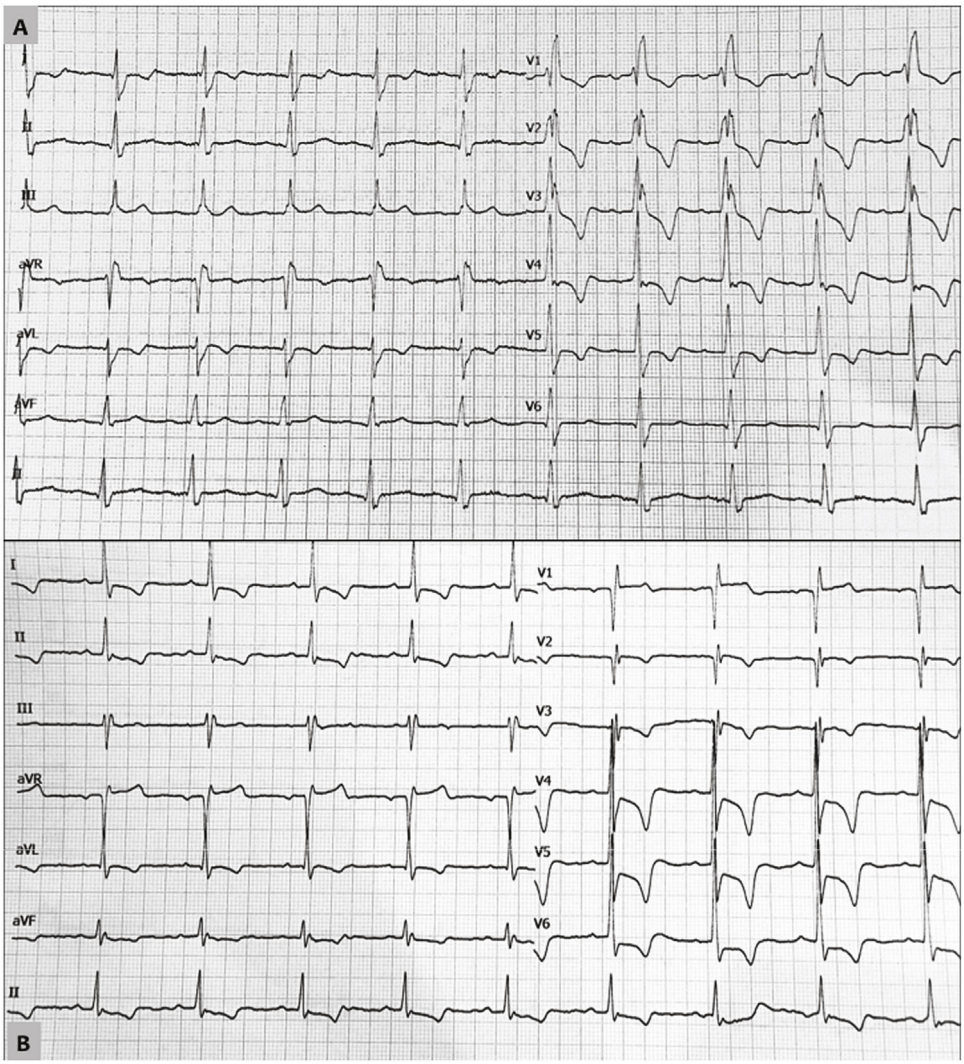

Figure 1. Electrocardiograms showing characteristic features of apical ventricular hypertrophy in 2 patients following heart transplant. A: complete right bundle branch block with increased voltages and criteria indicating left ventricular hypertrophy. B: increased voltages with criteria of left ventricular hypertrophy and deep T-wave inversion in the precordial leads.

cases of apical LVH were identified in the total of 233 transplant recipients alive and under follow-up (prevalence 3.4%). Mean age at diagnosis was 56.4 ± 8.8 years, and there was a higher percentage of women in this group than among patients without LVH (62.5% vs 26.2%, respectively).

The most common reason for transplant in patients with apical LVH was dilated cardiomyopathy: 5 patients (62.5%) (table 1). None of the donor hearts showed significant LVH (on echocardiography performed in the donor's center of origin), and ventricular function was within the normal limits. Following transplant, all patients received an immunosuppressive regimen that included a calcineurin inhibitor, but in 1 case this was replaced by everolimus before the LVH diagnosis due to graft vascular disease in a patient with renal failure. In 2 other patients a change to everolimus was made after the LVH diagnosis, but no significant regression of the condition has been observed to date (1 and 2 years, respectively, of follow-up). In all cases, electrocardiograms showed a characteristic LVH pattern, with increased voltages and giant negative T waves in the precordial leads (figure 1). Cardiac magnetic resonance confirmed the diagnosis in the 4 patients who underwent this examination. Apical aneurysms were not observed in any of the patients. Two patients had "mixed" septal and apical hypertrophy, with no differences in the form of presentation or clinical course of the condition. In the follow-up endomyocardial biopsy specimens after HTx, there were no features indicating endomyocardial fibrosis, eosinophilia, or significant fibrosis.

None of the patients with apical LVH following transplant had to be hospitalized for heart failure, malignant ventricular arrhythmias, or sudden cardiac death. The predominant symptom was exertional dyspnea, (7 patients, 87.5%). One previously reported case of LVH following HTx also had a benign clinical course.[6] Among the total, only 1 patient had repeated episodes of syncope; head trauma occurring in 1 of the episodes led to intracranial bleeding and death.

Although LVH was severe in all cases, most patients in our series showed no significant clinical consequences. The most common symptom was functional class deterioration. This outcome is comparable to that of other cohorts with apical hypertrophic cardiomyopathy, who have shown low mortality and few adverse cardiovascular events.[4,5] Although a considerable percentage of our apical LVH patients had a history of hypertension, it is unlikely that hypertension was the cause of the condition given the location of LVH and the cardiac magnetic resonance findings. The specific cause of LVH was not identified after excluding rejection and

microbiological causes such as cytomegalovirus infection. Of particular note, an uncommon adverse effect of calcineurin inhibitors is LVH,[2] as described in patients receiving these drugs for immunosuppressive therapy in autoimmune diseases, hematologic malignancies, and solid organ transplantation. The origin of apical LVH in our patients would likely be multifactorial, including the action of cytokines, hemodynamic overload, and possible sarcomeric cardiomyopathy in the donor that could develop later. The most important limitations of the present study are the small number of cases of apical LVH and the disparity in the length of follow-up (between 1 and 14 years).

This is the first study to date describing a consecutive series of patients with apical LVH following HTx, which is likely an underdiagnosed condition. The patients' clinical course was benign in most cases.

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Control of LDL-C levels after an acute coronary syndrome in Spain. Are the available treatments adequately used?

Grado de control del cLDL tras un síndrome coronario agudo en España. ¿Se utilizan adecuadamente los recursos terapéuticos existentes?

To the Editor,

In recent years, due to the therapeutic advances and the introduction of the "infarct code" for primary angioplasty, patient mortality in the acute phase of acute coronary syndrome (ACS) has decreased considerably.[1] However, the high rate of further ischemic events after discharge shows the need for optimizing secondary prevention measures in these patients. Among the objectives, it is fundamental to reduce levels of low-density lipoprotein cholesterol (LDL-C) (to < 70 mg/dL, as recommended in the 2016 European guidelines on dyslipidemia,[2] or even < 55 mg/dL according to the most recent guidelines from 2019[3]). Because of the efficacy of the available lipid-lowering drugs (high-potency statins, ezetimibe, and proprotein convertase subtilisin/kexin type 9 inhibitors [PCSK9i]), this is also now feasible.

However, recent studies continue to show that dyslipidemia control, despite improvement in recent years, remains very poor. Specifically, after an ACS, several studies in US populations have shown that the percentage of patients who received high-intensity statins in 2007-2009 was very low: 21% at discharge and just 14% at 1 year,[4] and that this proportion increased slightly in 2011 (24.8% at discharge) and in 2014 (57.5%).[5] More than a third of the patients had LDL-C > 70 mg/dL.[4] These data, as well as the release of PCSK9i, prompted the Spanish Society of Cardiology (SEC) to publish a position document on the subject in 2016.[6] The aim of our study was to analyze the attainment of lipid targets in the first year of follow-up after an ACS in Spain following the publication of this document.[6]

We selected 20 cardiology departments from secondary or tertiary hospitals (10 of each) in Spain. All the departments had catheterization laboratories and infarct code programs, and 40% had cardiac rehabilitation units. Patient follow-up and, therefore, treatment monitoring, was done either by cardiology or by primary care; 80% of the hospitals did not have established protocols. A cutoff target LDL-C < 70 mg/dL was used, as this was the recommended target in the 2016 guidelines.[2]

The study included 6364 patients (mean, 355 per hospital [range, 54-2254]),with a mean age of 73.3 ± 10.6 years; 61.5% were men and 37.3% had diabetes. Figure 1 shows the lipid-lowering therapy used (figure 1A-C). At the time of discharge, 72.1% of patients received high-dose potent steroids (rosuvastatin 20 mg or atorvastatin 80 mg) and 24.1% received low- or medium-intensity statins, while just 3.8% received no statins (figure 1A). Thirteen percent received ezetimibe (figure 1B) and only 0.31% received PCSK9i (figure 1C). At 12 months, the percentage of patients receiving ezetimibe increased to 25.6%. Less than 1% of patients received PCSK9i. With these treatments, 61.1% of patients had LDL-C < 70 mg/dL at 6 months and 55.9% at 12 months (figure 1D). Figure 2 shows the percentage of patients with LDL-C > 70 (figure 2A) or > 100 mg/dL (figure 2B) and the different lipid-lowering treatments they were receiving. At 6 months, 30.3% of patients with LDL-C > 70 mg/dL were not on high-