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-

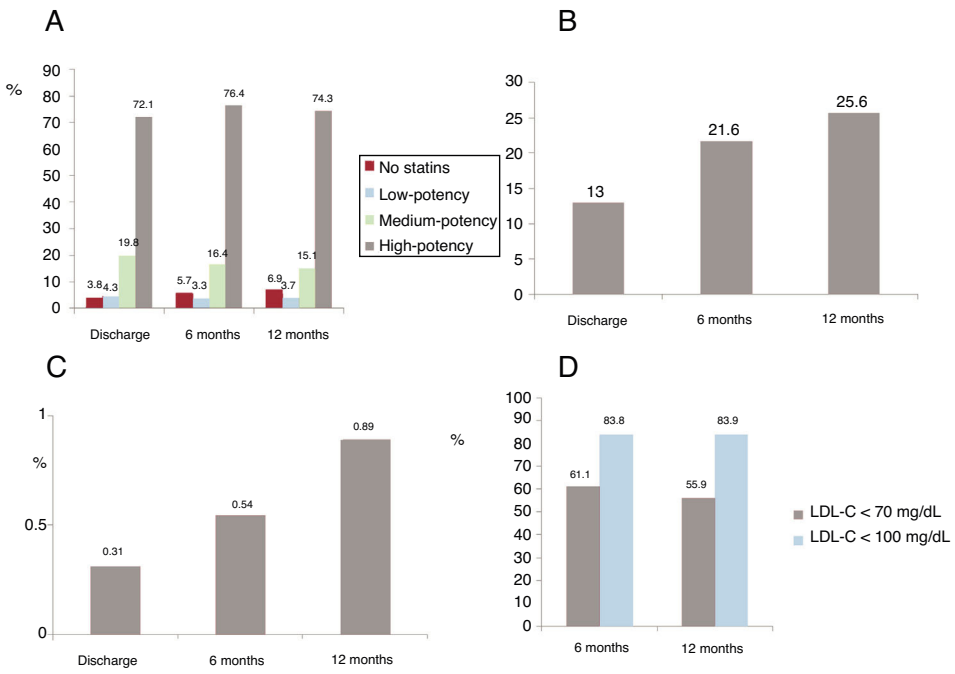

Figure 1. Lipid-lowering treatment received at discharge and at 6 and 12 months after acute coronary syndrome (ACS) in our study (A: statins; B: ezetimibe; C: PCSK9 inhibitors), and proportion of patients with low-density lipoprotein cholesterol (LDL-C) < 70 mg/dL and < 100 mg/dL at 6 and 12 months (D).

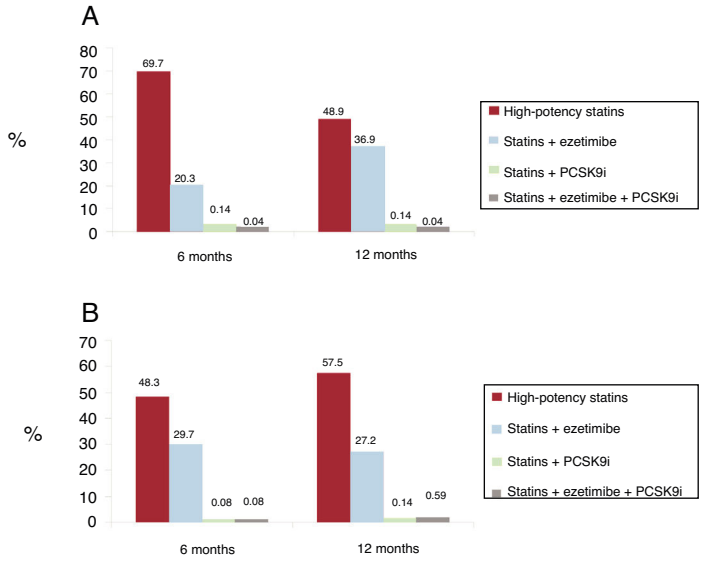

Figure 2. Lipid-lowering treatment received by patients with low-density lipoprotein cholesterol (LDL-C) > 70 mg/dL (A) or > 100 mg/dL (B) at 6 and 12 months after discharge following acute coronary syndrome (ACS). PCSK9i, proprotein convertase subtilisin/kexin type 9 inhibitors.

potency statins, 79.9% were not on ezetimibe and only 0.14% were on PCSK9i; at 12 months, 51.5% of patients with LDL-C > 70 mg/dL were not on high-potency statins, 63.1% were not on ezetimibe and only 0.04% were on PCSKi (figure 2A).

These study results, obtained in 2018, show improvements in the use of lipid-lowering drugs and LDL-C control after ACS. Only 20% to 25% of patients did not receive high-intensity statins in the first 12 months after ACS. The use of ezetimibe increased to 25% of patients at 12 months. This translated to close to 40% of patients having an LDL-C > 70 mg/dL. Although it may not be possible to extrapolate these results nationally to all patients, they do indicate improved control of this problem. However, they also indicate that there remains much to be done, since *a)* up to 40% of patients did not meet the target LDL-C of < 70 mg/dL (and this would increase substantially if we applied the current recommendations of LDL-C < 55 mg/dL[3]); *b)* there is underuse of the available treatments, both the combination of statin + ezetimibe and particularly of PCSKi, whose use was minimal, and *c)* in 40% of patients with LDL-C higher than target levels, treatment was even further from the recommendations (figure 2). Better adherence to the scientific societies' guidelines[2,3,6] for these patients remains one of our greatest unmet challenges.

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Cardiac dimensions for young adolescent athletes

Medidas de las cavidades cardiacas de jóvenes adolescentes deportistas

To the Editor,

Sudden cardiac death (SCD) in young, healthy people is an issue of medical and social concern.[1] Echocardiography has become an important diagnostic tool in this setting, as it can detect certain preventable causes of SCD. A study of 2688 Spanish athletes (67% male; mean ± standard deviation age, 21 ± 10 years) found that 4 athletes had risk factors for SCD and 3 had a heart condition requiring specific treatment or follow-up.[2] The aim of this study was to establish the percentile distribution of 6 echocardiographic measurements that could serve as reference values for assessing and monitoring competing athletes.

We studied 2574 consecutive athletes (81% male, 89.2% white) from different clubs in La Rioja, Spain who had undergone cardiological examination between September 2013 and June 2017. While 20 different sports were represented, 85.5% of the athletes were from just 4 sports: soccer (61.2%), basketball (17.3%), handball (4.1%), and athletics (2.6%). They had been training for an average of 6 years (range, 4-7 years). Forty-two athletes (2.1%) were excluded because they had a heart condition. All athletes underwent a full cardiological examination consisting of structured history taking, physical examination, a resting electrocardiogram, and a 2-dimensional echocardiogram[3] (Vivid S5; General Electric Vingmed, USA). Body mass index and body surface area (BSA), calculated as body weight $(kg)^{0.425} \times$ height $(cm)^{0.725}) \times 0.007184$, were also measured. Percentile curves (P_3 to P_{97}) adjusted for age and sex were created using the lambda-mu-sigma (LMS) method in LMSchartmaker Pro, version 2.54 (Harlow Printing Limited, Tyne & Wear, UK). The coefficient of determination (R^2) was used to assess goodness-of-fit based on the inverse logarithm of BSA and cardiac structure measurements adjusted for sex.

The athletes' general characteristics and echocardiographic measurements are shown in table 1.

We observed a linear increase in percentiles (P_3-P_{97}) according to BSA for the 6 echocardiographic measurements analyzed (table 2). The association between BSA and all 6 measurements was linear (R^2 = 0.11-0.31) but was strongest for left ventricular end-diastolic diameter (R^2 = 0.29-0.31, $P < .05$).

Our findings could contribute to the detection of potentially serious cardiac abnormalities in young athletes and identify athletes requiring cardiology follow-up or treatment. Our findings are similar to those of a study of competing child and adolescent athletes, but we analyzed a larger sample and more right ventricle echocardiographic measurements.[4] As expected, the absolute chamber dimensions observed in our study are lower than those reported for elite athletes.[5] Once adjusted for BSA, however, they are similar.

Generally speaking, the increase observed in cardiac dimensions according to BSA is consistent with the available evidence. However, discrepancies between our findings and others may partly be due to differences in type of sport, training characteristics, years of training, age, race, and sex. In addition, echocardiographic measurements, like all measurements, are prone to interobserver and intraobserver variability, and technical difficulties associated with echocardiographic image acquisition have been reported by various research centers.

One of the main limitations of our study is the absence of a control group (noncompeting adolescents) with which to compare our results. Our findings are, however, similar to those reported by several studies that did not use a control group either.[6] Because of the cross-sectional nature of our study, we are unable to establish a causal link between our findings and numerous aspects, such as type of training, sporting history, and "possible" adaptations of the heart to training. Information on hours spent training every week might have allowed us to detect differences in heart chamber structures and diameters between athletes participating in different types of sport (eg, sport involving resistance and/or strength training or individual