echocardiography (TEE) examination was performed 45 days after the procedure.

Baseline and procedural characteristics of the study population are shown in **Table 1**. All patients but 1 had TEE at 45 ± 6 days post-LAAC. One patient (3.3%) had a device-related thrombus on the surface of the device, without clinical consequences. The patient received low molecular weight heparin for 4 weeks, and repeat TEE showed the disappearance of the thrombus.

Median follow-up was 19 (interquartile range: 12 to 24) months and no patient was lost to follow-up. Five patients (16.1%) died during the follow-up period, none of them related to the device or to cardioembolic or bleeding causes. There was no stroke or systemic embolism during the follow-up period (expected annual rates of stroke or thromboembolism according to CHA_2DS_2-VAS_C score of 8.1% and 11.2%, respectively). One patient (3.2%) had a major gastrointestinal bleeding (expected annual rate of bleeding according to HAS-BLED score of 8.6%).

The present study showed the preliminary safety and efficacy of single antiplatelet therapy following LAAC. In addition to the absence of thromboembolic events at close to 2-year follow-up, the rate of device-related thrombus at 45 days post-LAAC (3.3%) was similar to that found in the PROTECT-AF trial (4.2%) (1), and ASAP (ASA Plavix feasibility study with Watchman left atrial appendage closure technology) trial (4%) (2) using short-term anticoagulation and DAPT, respectively. The patients in the present study had a high risk of bleeding determined by a mean HAS-BLED score >4. The bleeding rate in the present study was lower than expected based on the HAS-BLED score, suggesting that single antiplatelet therapy could be the optimal therapy for patients at high risk of bleeding undergoing LAAC.

In conclusion, single antiplatelet therapy after LAAC in patients with a high risk of bleeding is associated with a low rate of cardioembolic and bleeding complications at a median follow-up of 19 months. Future randomized trials should be carried out to confirm the safety and efficacy of single antiplatelet therapy following LAAC.

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http://dx.doi.org/10.1016/j.jacc.2016.08.016

Please note: Drs. Rodriguez-Gabella and Regueiro are supported by a grant from the Fundación Alfonso Martin Escudero (Madrid, Spain). The authors have reported that they have no relationships relevant to the contents of this paper to disclose. Deepak L. Bhatt, MD, MPH, served as Guest Editor for this article.

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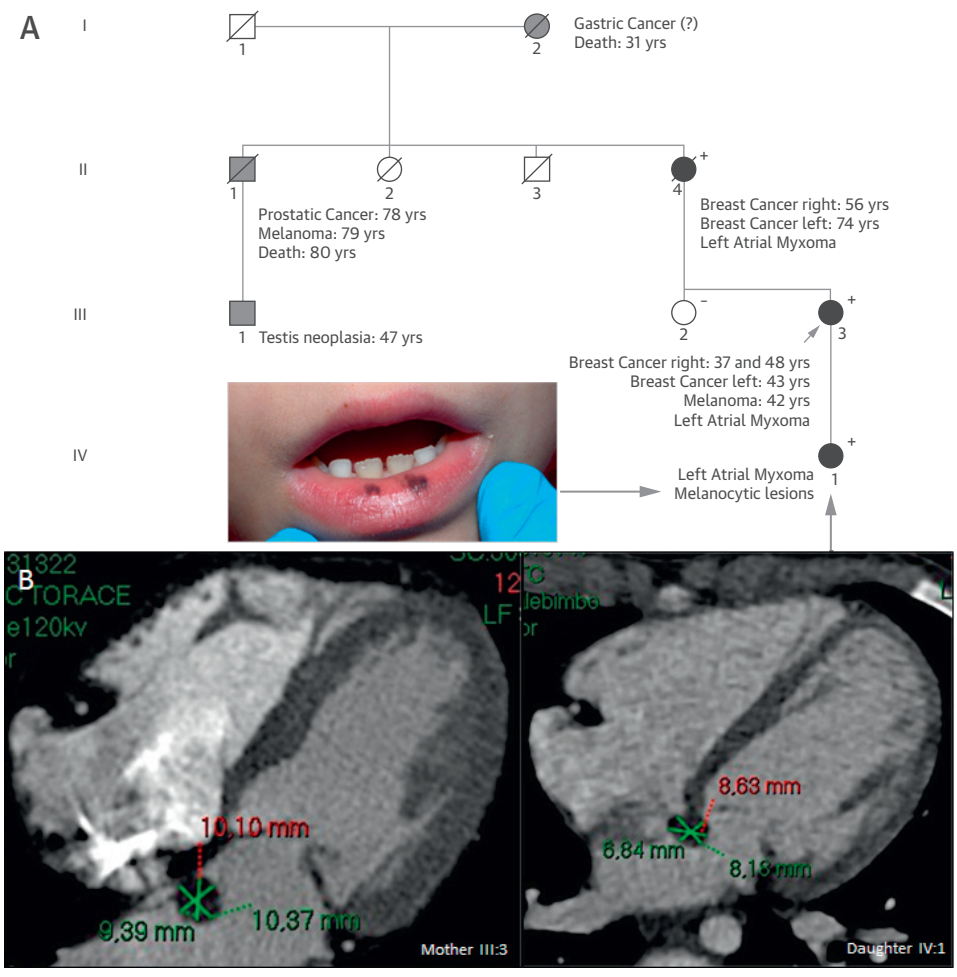
Cardio-Oncology

The Carney Complex Type I

Cardio-oncology is an important, expanding discipline. Although most developing programs address the cardiotoxicity of old and novel anticancer drugs, cardio-oncology expertise and experience are also needed for rare multiorgan diseases, which require interdisciplinary evaluation to provide optimal, timely care for patients and families. Carney complex type I is the paradigmatic example that cardiologists should consider when diagnosing cardiac myxoma and oncologists should consider when diagnosing endocrine, cutaneous, and neural myxomatous neoplasms, especially in patients demonstrating pigmented lesions of the skin and mucosae (1). The disease was first described as a "complex including myxomas, spotting pigmentation and endocrine over-reactivity" (Carney Complex 1 [CNC1], OMIM #160980). The diagnosis is made by the presence of 2 major criteria confirmed by histology, imaging, or biochemical testing, or 1 major and 1 supplemental criterion (2). The disease gene is

FIGURE 1 Family Pedigree and Cardiac Mixoma



(A) Family pedigree. The proband (III:3), her 9-year-old daughter (IV:1), and her mother (II:4) fulfill the clinical diagnostic criteria of Carney syndrome type 1 and are carrier of the *PRKAR1A* mutation. The proband's sister (III:2) was clinically healthy and tested negative. The lower lip of the youngest affected family member (IV:1) shows the melanocytic lesion. **(B)** Cardio-AngioCT scans show the left atrial myxoma of the proband (III:3) **(left)** and of the proband's daughter (IV:1) **(right)**. Family members I:2, II:1, and III:1 had history of neoplasms, but could not be tested.

protein kinase cyclic adenosine monophosphate-dependent regulatory type I alpha (PRKAR1A) that maps at the 17q24.3 locus (3); genetic testing confirms the clinical diagnosis in probands and provides early diagnosis in younger relatives.

In the previous scenario, we observed a family (Figure 1A) with autosomal-dominant cardiac myxoma, breast cancer and pigmented cutaneous lesions. The proband's mother (II:4) was reported as having dextroposition of the aorta, metachronous breast cancer (at 55 and 74 years of age), hepatic cysts, dolichosigma, and thalassemia trait; she had 2 surgeries for left atrial myxoma at 64 and 67 years of age. The proband (III:3) herself is a 50-year-old female with a long clinical history, since 32 years of age, of breast myxoid fibroadenomas, intraductal papillomas, and intraductal carcinoma ending in left mastectomy at 43 years of age and right mastectomy at 48 years of age. In the midst, she also developed a melanoma. In 2015, echocardiographic study demonstrated presence of a left atrial myxoma (Figure 1B). Her daughter (IV:1) underwent her first cardiologic evaluation at 8 years of age: echocardiography demonstrated presence of an atrial myxoma

(Figure 1B). The girl had lentigo of the lips (Figure 1A). The genetic defect identified in the family (*PRKAR1A* c. 761_762delTC p. Ser 254Tyr fsx15) predicts a truncated protein, and was identified in the proband and her daughter and in the affected mother. The proband's sister tested negative for the mutation (III:2).

This typical example of Carney syndrome highlights the multidisciplinary diagnostic work-up in patients with myxoma (cardiology perspective), extracardiac myxomatous neoplasms (oncology perspective), and melanoma (dermatology perspective). In syndromes affecting more than 1 organ, the first clinical episode can fall within either specialty. Therefore, all specialists exploring traits pertinent to this syndrome should include Carney complex in the list of differential diagnoses. The only differential diagnosis for cardiac myxoma is the "isolated" or "sporadic myxoma," whereas the differential diagnosis of noncardiac traits is more complex and should include all syndromes with lentigines and tumors similar to those that recur in Carney complex, reviewed in Kirschner et al. (3). The precise diagnosis can save a life, especially when complex syndromes include the risk of malignancies and helps management. The youngest member of the family (IV:1) is affected by myxoma and melanocytic skin lesions: the myxoma shows a large implant and smooth borders, encouraging careful monitoring rather than early surgery, given that future surgeries, both cardiac and noncardiac, are expected to be needed in the course of her life. In fact, cardiac myxomas tend to recur in Carney complex (4). Similarly, the high risk of developing malignancies calls for personalized clinical monitoring and need for close longitudinal follow-up. Up to 10% of cardiac myxomas occur in Carney syndromes (4). The number of missed diagnoses is likely high. Cardiologists and cardiac surgeons caring for these patients should be encouraged to organize multidisciplinary evaluation. We suggest that Carney complex should be excluded in all patients presenting with cardiac myxomas.

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http://dx.doi.org/10.1016/j.jacc.2016.07.764

Please note: Dr. Bertherat has served as a board member for Novartis, HRA, and Shire. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose. Renu Virmani, MD, served as Guest Editor for this article.

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T1 and T2 Mapping in Nonischemic Cardiomyopathies and Agreement With Endomyocardial Biopsy

With interest we read the study by Lurz et al. (1), investigating concordance between quantitative tissue characterization techniques and Lake Louise criteria in patients with myocarditis and endomyocardial biopsy. We would like to congratulate the authors for contributing an increasingly rare piece of evidence, as well as for performing multiple contrasts and contributing the histological correlate in a large number of patients. We would like to share a few further observations drawn from the presented data.

Despite the seemingly different groups in terms of duration of symptoms, defined by an interval of 14 days between the onset and hospitalization, there is very little difference between the acute and chronic groups in terms of native T1 or T2 (Figure 1A). This observation contrasts our previous study in highly selected patients with clinical diagnosis of myocarditis, which reported considerably higher native T1 values in acute stage, with subsequent decline of values in a longitudinal study (Figure 1B) (2). Indeed, endomyocardial biopsy results reveal that only 8% of