

Regarding the involvement of right ventricular (RV) failure in patients with incomplete LV unloading, our results add to accumulating evidence underlining the impact of RV failure after LVAD on LV hemodynamics and supports the evidence of the strong interaction between the left and right filling pressures that occur during long-term LVAD support. Regarding our methods to evaluate RV function, we used the echocardiographic variables that are commonly employed in clinical practice, including RV dimensions, tricuspid annular plane systolic excursion, and tricuspid regurgitation. We agree that the new evidence addressed by our study, regarding the association between LV unloading and RV hemodynamics, merits further investigation with specific echocardiographic methods to evaluate RV function.

Due to our limited cohort, we evaluated a small number of variables in the multivariable analysis, including age. Brain natriuretic peptide emerged as an independent factor for LV unloading. Although we cannot rule out the influence of renal failure and obesity in the predictive value of brain natriuretic peptide, the mean creatinine (1.3 mg/dL) and body mass index (26 kg/m<sup>2</sup>) were only mildly elevated.

Ours was a noninterventive clinical study and we did not perform right-heart catheterization with the intention to optimize LVAD rotor speed setting or medication if patients were otherwise clinically stable. Therefore, the events would not be affected by the timing of the day 0 that we chose. We considered that global surveillance from the time of LVAD implantation was of greater clinical interest for survival analysis. In this line, our study cannot address the question of whether changes in medications or rotor speed setting based on right-heart catheterization might impact hemodynamics. Although worse hemodynamics after LVAD seem to be associated with more adverse events, there is still a clear knowledge gap regarding the clinical implications of a strategy guided by hemodynamics on quality of life and event-free survival in clinically stable patients.

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## CONFLICTS OF INTEREST

The author has no conflicts of interests to declare regarding this manuscript.

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## Cardio-oncology at present: a pending challenge



### Cardio-oncología en la actualidad: un reto pendiente

#### To the Editor,

We have read with considerable interest the scientific letter by Caballero Valderrama et al.<sup>1</sup> regarding anthracycline-related onset of ventricular dysfunction associated with familial dilated cardiomyopathy. The early diagnosis and management of the cardiovascular toxicity associated with anticancer drugs is an ever-growing challenge, from both clinical and research perspectives. Undoubtedly, the risk of cardiovascular toxicity is the result of a complex interaction among the characteristics of both the patient (eg, age, genetics, and cardiovascular risk) and the tumor itself, as well as the type and length of the proposed treatment.<sup>2</sup>

Current cardio-oncology strategies recommend individualized assessment of the cardiovascular toxicity risk in all patients who may be receiving potentially cardiotoxic anticancer therapies.<sup>3</sup> Based on this risk, prevention and monitoring protocols have been established for during and after the cancer treatment, as well as recommendations for optimizing the management of related cardiovascular events.<sup>4,5</sup>

We consider highly pertinent the publication of this late example of cardiovascular disease related to anthracyclines and radiotherapy<sup>1,2</sup> because it reminds clinicians that they should consider cancer treatment to be a cardiovascular risk factor<sup>3</sup> and because it exemplifies the need for multidisciplinary teams coordinating among the different levels of care.

We agree with the authors that the performance of a genetic study in patients with a family history compatible with heart disease could improve the prevention of cardiotoxicity risk. However, in terms of genetics and personalized medicine in cardio-oncology, there is still a long way to go.<sup>3,6</sup> Until then, cardiovascular risk should be stratified before, during, and after cancer treatment to optimize the control of cardiovascular risk factors and detect subclinical phases of myocardial damage. In line with the current consensus, the reported patient had an intermediate risk of anthracycline-related toxicity, and the combination of anthracycline with radiotherapy would increase this risk in the mid-to-long-term.<sup>4,5</sup> In this regard, monitoring of biomarkers,<sup>7</sup> electrocardiography, and imaging techniques<sup>8</sup> before, during, and 12 months after treatment completion could have detected subclinical changes in cardiac function requiring a more detailed long-term follow-up.<sup>5,6</sup> Based on the authors' report, the electrocardiographic changes could have predicted the cardiac

damage.<sup>1</sup> The cardiovascular monitoring of cardiotoxic treatments<sup>4</sup> is a class I recommendation with level of evidence B in the 2021 European guidelines on cardiovascular disease prevention<sup>3</sup> and most of the tests mentioned are easily accessed.

Ultimately, all of this underlines the importance of the creation of multidisciplinary programs and pragmatic intervention protocols permitting the optimal screening and follow-up of cardiovascular disease in cancer patients to improve their outcomes and facilitate their treatment.

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## AUTHORS' CONTRIBUTIONS

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