

Letter to the Editor

Incomplete left ventricular unloading following left ventricular assist device implantation**Descarga incompleta del ventrículo izquierdo tras el implante de un dispositivo de asistencia ventricular izquierda****To the Editor,**

The implications of abnormal hemodynamics following left ventricular assist device implantation is currently a cause of concern. Our team recently demonstrated the prognostic implications of abnormal hemodynamics in this cohort.¹ Ruiz-Cano et al.² demonstrated that several parameters were associated with such abnormal hemodynamics, particularly incomplete left ventricular unloading defined as pulmonary capillary wedge pressure > 15 mmHg. Several concerns have been raised.

The first concern is a 15-mmHg cutoff of pulmonary capillary wedge pressure.² There is no gold standard to define incomplete left ventricular unloading, but a promising way might be to statistically calculate a cutoff associated with clinical outcome.

Second, a key to explaining the prognostic impact of incomplete left ventricular unloading might be right ventricular failure. In the study by Ruiz-Cano et al.,² incomplete left ventricular unloading was associated with elevated central venous pressure and a decreased pulmonary artery pulsatility index. Their study would be strengthened by analysis of further echocardiographic parameters associated with right ventricular function, including right ventricular fractional area change, tricuspid annular systolic excursion velocity, and right ventricular longitudinal strain.

For the time-to-event analysis, each event would be affected by the timing of day 0. The timing of right heart catheterization (ie, day 0) varied in each patient in their study.² To minimize bias, it might be better to add outcome data stratified by the timing of right heart catheterization.

The authors propose the level of B-type natriuretic peptide as an alternative to incomplete left ventricular unloading. It might be of interest to analyze the prognostic impact of B-type natriuretic peptide level. Of note, the level of B-type natriuretic peptide might be affected by several parameters, including right ventricular failure, age, renal impairment, and obesity.

The last concern is intervention in incomplete left ventricular unloading. Could the authors propose any appropriate intervention tools? In addition to the hemodynamic and echocardiographic

ramp test to optimize device speed,³ diuretics including tolvaptan, sacubitril/valsartan, and SGLT2 inhibitor might be promising.

FUNDING

None.

CONFLICTS OF INTEREST

None.

Teruhiko Imamura

Second Department of Internal Medicine, University of Toyama, Toyama, Japan

E-mail address: teimamu@med.u-toyama.ac.jp

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Incomplete left ventricular unloading following left ventricular assist device implantation. Response**Descarga incompleta del ventrículo izquierdo tras el implante de un dispositivo de asistencia ventricular izquierda. Respuesta****To the Editor,**

We read with great interest the comments by Teruhiko Imamura on our study.¹

A pulmonary capillary wedge pressure $\leq$ 15 mmHg was found to be a reliable indicator of normal left ventricular (LV) filling pressure and is a reference value in clinical guidelines to diagnose postcapillary pulmonary hypertension and to guide prognosis and management in selected patients with heart failure. Other authors have considered higher cutoff values of pulmonary capillary wedge pressure to define a normal LV filling pressure in patients with chronic heart failure treated with a left ventricular assist device (LVAD). However, there is currently no evidence that a different cutoff value could better predict prognosis in these patients.

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/m²) were only mildly elevated.

Ours was a noninterventonal 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.

Maria J. Ruiz-Cano

Herz-und Diabeteszentrum NRW, Ruhr-University Bochum, Germany

E-mail address: mruiz-cano@hdz-nrw.de

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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.¹ 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.²

Current cardio-oncology strategies recommend individualized assessment of the cardiovascular toxicity risk in all patients who may be receiving potentially cardiotoxic anticancer therapies.³ 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.^{4,5}



We consider highly pertinent the publication of this late example of cardiovascular disease related to anthracyclines and radiotherapy^{1,2} because it reminds clinicians that they should consider cancer treatment to be a cardiovascular risk factor³ 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.^{3,6} 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.^{4,5} In this regard, monitoring of biomarkers,⁷ electrocardiography, and imaging techniques⁸ before, during, and 12 months after treatment completion could have detected subclinical changes in cardiac function requiring a more detailed long-term follow-up.^{5,6} Based on the authors' report, the electrocardiographic changes could have predicted the cardiac