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#### Echocardiography and mortality in Ebstein's anomaly

Juan Francisco Fritche-Salazar, Héctor Herrera-Bello,  
Jorge Kuri Alfaro, Manuel B.A. Gaxiola Macías,  
Jorge Cossio Aranda, Nydia Ávila-Vanzzini

#### Prevalence of coronary artery disease in valvular patients

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#### Contrast echo in right ventricle infarction

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### Clinical case

#### Acute dislocation of a Perceval valve treated with TAVI valve-in-valve

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#### Cardiotoxicity and strain

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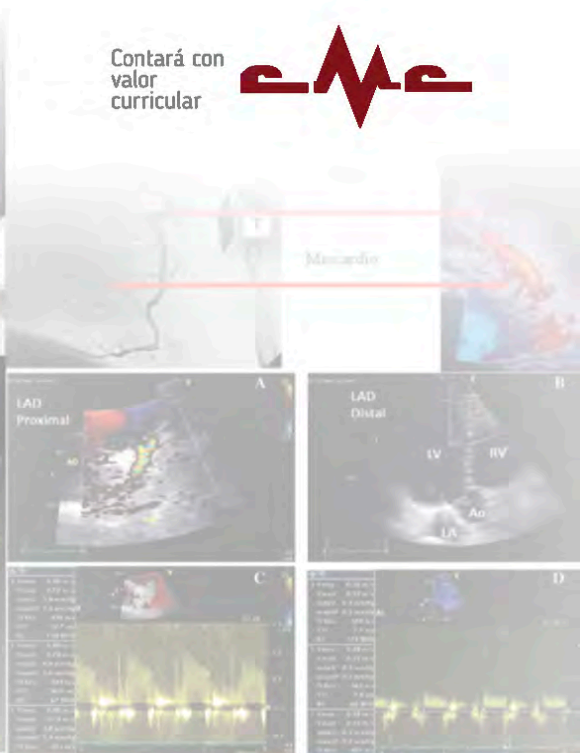
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## Original Research

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*Cardiotoxicidad, strain: evidencia actual*

Zuilma Yurith Vásquez Ortiz,  
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# Echocardiographic findings associated with mortality in adult patients with Ebstein's anomaly

*Hallazgos ecocardiográficos asociados con mortalidad en pacientes adultos con anomalía de Ebstein*

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**ABSTRACT. Introduction:** Ebstein's anomaly (EA) is a right ventricular cardiomyopathy, current echocardiographic severity criteria are not helpful to predict disease progression or identify patients at higher risk of mortality. **Objective:** We analyzed clinical and echocardiographic data of adult patients with EA to identify predictors of mortality. **Methods and results:** Clinical and echocardiographic parameters were analyzed from historical data of 60 adult patients with EA, six of them have died. Variables were compared between groups. Among clinical characteristics only the presence of cyanosis was statistically higher in the mortality group (100 vs 26%,  $p < 0.001$ ). When analyzing the echocardiographic parameters, both the right and left ventricular function were worse in the mortality group (LVEF:  $56.7 \pm 7$  vs  $49 \pm 2.2\%$ ,  $p < 0.015$ , RV FAC:  $27.1 \pm 11$  vs  $15.3 \pm 7.8\%$ ,  $p < 0.022$ ). Nor the severity of the EA, nor the pulmonary systolic pressure were different between groups. **Conclusion:** EA is a cardiomyopathy and not a disease limited to the tricuspid valve. Both right and left systolic dysfunction are predictors of mortality.

**Keywords:** Ebstein's anomaly, adult patient, echocardiographic parameters, mortality.

**RESUMEN. Introducción:** La anomalía de Ebstein (AE) es una miocardiopatía del ventrículo derecho, los criterios ecocardiográficos de severidad no correlacionan con la evolución clínica y pronóstico de los pacientes. **Objetivo:** Se analizaron las variables clínicas y ecocardiográficas de pacientes adultos con AE en búsqueda de factores asociados con mortalidad. **Métodos y resultados:** Los parámetros clínicos y ecocardiográficos fueron analizados de una cohorte histórica de 60 pacientes adultos con AE, seis de ellos murieron en el seguimiento. Las variables fueron comparadas entre grupos de sobrevida y mortalidad. De las características clínicas sólo la presencia de cianosis fue significativamente mayor en el grupo de mortalidad (100 vs 26%,  $p < 0.001$ ). Al analizar los parámetros ecocardiográficos la función sistólica tanto del ventrículo derecho como del izquierdo fueron menores en el grupo de mortalidad (FEVI:  $56.7 \pm 7$  vs  $49 \pm 2.2\%$ ,  $p < 0.015$ , FAC VD:  $27.1 \pm 11$  vs  $15.3 \pm 7.8\%$ ,  $p < 0.022$ ). Ni la severidad de la AE ni la presión sistólica pulmonar fueron diferentes entre grupos. **Conclusión:** Al ser una miocardiopatía, tanto la disfunción sistólica ventricular derecha como la izquierda se ven afectadas y correlacionan con mortalidad, estos datos deben ser útiles para el seguimiento y la toma de decisiones en pacientes con AE.

**Palabras clave:** Anomalía de Ebstein, patología congénita del adulto, parámetros ecocardiográficos, mortalidad.

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## INTRODUCTION

Ebstein's anomaly (EA) is considered a cardiomyopathy that usually involves the right ventricle, but the left ventricle could also be affected.<sup>1,2</sup> It is a rare disease, with a wide clinical spectrum and commonly associated with other cardiac abnormalities such as atrial septal defect. Its diagnosis is based on the hallmark of septal and/or posterior tricuspid valve leaflet adherence to the right myocardium.<sup>3,4</sup> Tricuspid regurgitation is common, it contributes to right chamber dilation acting as a substrate for atrial or ventricular arrhythmia. Echocardiographic severity criteria do not correlate with clinical evolution and prognosis.<sup>3,4</sup> Hemodynamics and clinical patterns of presentation depend on anatomical severity, degree of right-to-left atrial shunting, age at presentation and ventricular dysfunction.<sup>5-7</sup> We reviewed the clinical and echocardiographic data of adult patients with EA looking for factors associated to mortality.

## MATERIAL AND METHODS

We analyzed an historical observational cohort of adult patients with EA from 1980 to 2015. We obtain information from the clinical

records. Any patient older than 18 years, with the diagnosis of EA who had follow-up in the National Institute of Cardiology «Ignacio Chavez», Mexico, was included.

The diagnosis of EA was made through echocardiographic assessment (attachment of the septal and/or posterior tricuspid valve leaflets to the underlying myocardium, with respect to the anterior mitral valve leaflet  $>8$  mm/m<sup>2</sup>).<sup>8</sup> The severity of the anomaly was classified according to percent of septal valve as mild when it was less than 44%; moderate when it was between 44-61%; and severe when this was more than 61%.<sup>6</sup> As recommended for this pathology, ventricular function was evaluated through right ventricular fraction area change (RV FAC) of the functional right ventricle, left ventricular function was evaluated by Simpson's method.<sup>9</sup> Other echocardiographic variables were the diastolic dimension of the right and left ventricle, the severity of tricuspid regurgitation and the pulmonary systolic pressure. The surgical status was recorded. New York Heart Association (NYHA) functional classification was used to assess heart failure severity symptoms at the follow-up. Cyanosis was assessed at first evaluation. We excluded those patients who had incomplete clinical record and with

**Table 1: Clinical characteristics.**

	Survival group n=54 (%)	Mortality group n=6 (%)	p
Age (years)	34.8±12.4	38±8.4	0.555
Female gender	35 (64.8)	5 (83.3)	0.361
Surgery	12 (22.2)	3 (50)	0.136
Cyanosis	14 (26)	6 (100)	0.001
NYHA functional class			
I	12 (22.2)	0	0.083
II	38 (70.4)	4 (66.7)	
III	4 (7.4)	2 (33.3)	
WPW syndrome	19 (35.2)	1 (16.7)	0.361
AF/flutter	9 (16.7)	0	0.578
AV blocks	9 (16.7)	1 (16.7)	0.997
Ablation procedure	17 (31.5)	1 (16.7)	0.453
Pacemaker	4 (7.4)	1 (16.7)	0.436
Ventricular tachycardia	1 (1.8)	1 (16.7)	0.192
Stroke	3 (5.6)	1 (16.7)	0.351

NYHA = New York Heart Association; WPW = Wolff Parkinson White; AF = atrial fibrillation; AV = atrioventricular.

absence of late follow-up (all patients should have at least one-year of follow up in our institution).

### Statistical analysis

Information was stored on an electronic Excel spreadsheet and processed using STATA 12.1 statistical software package. All continuous variables were assessed for normality with the Shapiro-Wilk test, they are expressed as mean value and standard deviation and comparison

between groups was made with t-Student test. Categorical variables are expressed as number and percentage in relation to the population at risk, comparison between groups was made with  $\chi^2$  test. We considered a p value <0.05 as indicative of statistical significance.

### RESULTS

Sixty patients met our inclusion criteria, 40 patients were female (67%), the average age at diagnosis was 36.1 years old (25-42.4).

**Table 2: Echocardiographic characteristics.**

	Survival group (n=54)	Mortality group (n=6)	p
Severity (%)			0.217
Mild	18 (33.3)	0	
Moderate	15 (27.8)	3 (50)	
Severe	21 (39)	3 (50)	
LVEDD (mm)	38.3±5.7	35.6±5.7	0.146
LVESD (mm)	25.3±5.6	22.3±5.5	0.238
RVEDD (mm)	56±9.4	64.3±12.1	0.058
RV FAC (%)	27.1±11	15.3±7.8	0.022
TR (%)			0.221
Mild	3 (5.5)	0	
Moderate	17 (31.5)	0	
Severe	34 (63)	6 (100)	
PSP (mmHg)	29.5±11.6	30.5±7.8	0.846
ASD (%)	24 (44.4)	3 (50)	0.795
Tricuspid annulus (mm)	49.2±9.7	53.5±11.38	0.461
LVEF (%)	56.7±7	49±2.2	0.015

LVEDD = left ventricle end-diastolic dimension; LVESD = left ventricle end-systolic dimension; RVEDD = right ventricle end-diastolic dimension; FAC = fractional area change; TR = tricuspid regurgitation; PSP = pulmonary systolic pressure; ASD = atrial septal defect; LVEF: left ventricle ejection fraction.



**Figure 1:** Patient con severe EA. **A)** Severe septal valve attachment (orange line) to the right ventricular septum (blue line). **B)** Diastolic area of the functional right ventricle (26.8 cm<sup>2</sup>). **C)** Systolic area of the functional right ventricle (12.8 cm<sup>2</sup>). In this specific case the RV FAC was 52.2%.

Cyanosis was found in 33% of patients during the first follow-up. Four patients had stroke (6.6%). According to echocardiographic criteria for severity, 18 patients were classified as having mild EA (33.3%), 15 as moderate EA (27.8%) and 21 patients as severe EA (39%) (*Figure 1*). Fifteen patients underwent corrective or palliative surgery. Ablation procedure was made in 18 patients. Six patients died at follow-up.

When comparing the clinical characteristics between the mortality and the survival groups we only found difference in the prevalence of cyanosis, being more frequent in the mortality group, there were no difference in age, gender, surgical status, functional class or prevalence or rhythm disorders (*Table 1*).

The echocardiographic findings showed no difference for the severity of EA between groups, nor the severity of tricuspid regurgitation or the systolic pulmonary pressure were different. The end-diastolic and end-systolic dimensions of the left ventricle showed no difference, the end-diastolic dimension of the right ventricle showed a tendency to be larger in patients who died. Systolic dysfunction of both ventricles was more prevalent in the mortality group, with a mean RV FAC of 27.1 vs 15.3%,  $p < 0.022$ , and a mean LVEF of 56.7 vs 49%  $p < 0.015$  for the survival and mortality group respectively (*Table 2*).

## DISCUSSION

This study showed that in adult patients with EA right ventricular systolic dysfunction measured by FAC and left ventricular systolic dysfunction measured by LVEF correlate with mortality regardless of the severity of the disease or the surgical status. The pre-excitation and WPW syndrome are more frequently in EA than in general population,<sup>10</sup> the downward displacement of the septal leaflet, associated with discontinuity of the central fibrous body and septal atrioventricular ring, generates direct muscular connections, allowing persistence of accessory atrioventricular pathways.<sup>8,11</sup> Complete heart block is rare in EA,<sup>4,7</sup> when this occurs it is explained by compression of the AV node and the abnormal formation of the central fibrous body. Early age at presentation, right ventricular outflow obstruction, right ventricular

systolic dysfunction, hemoglobin/hematocrit values, male sex and severe EA have been associated with higher mortality.<sup>5,6,12</sup> In our study, only the ventricular systolic dysfunction of both ventricles were associated to higher mortality. No difference in age, gender or severity of the pathology was found.

Right ventricular dysfunction has been previously associated to mortality.<sup>12</sup> The myopathic nature of this disease, affecting the entire anatomy of the right ventricle,<sup>8</sup> leads to ventricular dysfunction.

**Study limitations:** The retrospective character of the study is the principal limitation. New techniques for the evaluation of RV systolic function were not assessed.

## CONCLUSION

Right ventricular and left ventricular systolic dysfunction are importantly associated to higher mortality in adult patients with Ebstein's anomaly regardless of the severity of the pathology or the surgical status.

## REFERENCES

1. Dearani JA, Mora BN, Nelson TJ et al. Ebstein anomaly review: what's now, what's next? *Expert Rev Cardiovasc Ther.* 2015; 13: 1101-1109.
2. Monibi AA, Neches WH, Lennox CC et al. Left ventricular anomalies associated with Ebstein's malformation of tricuspid valve. *Circulation.* 1978; 57: 303-306.
3. Brown ML, Dearani JA. Ebstein malformation of the tricuspid valve: current concepts in management and outcomes. *Curr Treat Options Cardiovasc Med.* 2009; 11: 396-402.
4. Attenhofer CH, Connolly HM, Edwards WD, et al. Ebstein's anomaly - review of a multifaceted congenital cardiac condition. *Swiss Med Wkly.* 2005; 135: 269-281.
5. Celermajer DS, Bull C, Till JA et al. Ebstein's anomaly: presentation and outcome from fetus to adult. *J Am Coll Cardiol.* 1994; 23: 170-176.
6. Attie F, Rosas M, Rijlaarsdam M et al. The adult patient with Ebstein anomaly. Outcome in 72 unoperated patients. *Medicine.* 2000; 79: 27-36.
7. Giuliani ER, Fuster V, Brandenburg RO et al. Ebstein's anomaly: the clinical features and natural history of Ebstein's anomaly of the tricuspid valve. *Mayo Clin Proc.* 1979; 54: 163-173.
8. Edwards WD. Embryology and pathologic features of Ebstein's anomaly. *Prog Pediatr Cardiol.* 1993; 2: 5-15.
9. Warnes CA, Williams RG, Bashore TM et al. ACC/AHA 2008 guidelines for the management of adults with congenital heart disease: a report of the American



- College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Develop Guidelines on the Management of Adults with Congenital Heart Disease). J Am Coll Cardiol. 2008; 52: e143-e163.
10. Sarubbi B, D'Alto M, Vergara P et al. Electrophysiological evaluation of asymptomatic ventricular pre-excitation in children and adolescents. Int J Cardiol. 2005; 98: 207-214.
11. Frescura C, Basso C, Thiene G et al. Anomalous origin of coronary arteries and risk of sudden death: a study based on an autopsy population of congenital heart disease. Hum Pathol. 1998; 29: 689-695.
12. Sarris GE, Giannopoulos NM, Tsoutsinos AJ et al. Results of surgery for Ebstein anomaly: a multicenter study from the European Congenital Heart Surgeons Association. J Thorac Cardiovasc Surg. 2006; 132: 50-57.

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# Prevalence of coronary artery disease evaluated by computed tomography coronary angiography in preoperative assessment of cardiac valvular surgery

*Prevalencia de enfermedad arterial coronaria evaluada por angiotomografía coronaria en la evaluación preoperatoria de cirugía cardíaca valvular*

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**ABSTRACT. Introduction:** Rheumatic valvular disease is predominant in developing countries. The prevalence of coexisting coronary artery disease (CAD) is based on other populations, and could be lower than reported, so that the indication of pre-operative invasive coronary angiography (ICA) could be over evaluated. The aim of the study is to demonstrate the prevalence of CAD diagnosed by Computed Tomography Coronary Angiography (CTCA) in a population of severe valvular heart disease (VHD) that requires surgery. **Material and methods:** Observational study that included subjects with severe VHD with surgical indication who underwent pre-operative CTCA. **Results:** We included 487 patients, 37% men and average age of 53 years. The prevalence of CAD was 4.1%. The independent predictors of CAD are age older than 60 years and male sex. **Conclusion:** In patients of our institution with severe VHD, the prevalence of CAD was low and CTCA could be the pre-operative tool of choice.

**Keywords:** Heart valve disease, prevalence, coronary artery disease, computed tomography coronary angiography, preoperative care.

**RESUMEN. Introducción:** La enfermedad valvular reumática es la predominante en países en vías de desarrollo. La prevalencia de enfermedad arterial coronaria coexistente está basada en otras poblaciones, y podría ser menor a la reportada, por lo que la indicación de coronariografía preoperatoria podría ser supervalorada. El objetivo del estudio es demostrar la prevalencia de enfermedad arterial coronaria obstructiva diagnosticada por angiotomografía en una población con valvulopatías severas que requieren cirugía de cambio valvular quirúrgico. **Material y métodos:** Estudio observacional que incluye sujetos con enfermedad valvular severa con indicación quirúrgica a los que se les realizó angiotomografía de coronarias preoperatoria. **Resultados:** Se incluyeron 487 pacientes, 37% hombres y promedio de edad de 53 años. La prevalencia de enfermedad arterial coronaria fue 4.1%. Los predictores independientes de enfermedad arterial coronaria son edad mayor de 60 años y sexo masculino. **Conclusión:** En pacientes de nuestra institución con enfermedad valvular severa, la prevalencia de enfermedad arterial coronaria fue baja, por lo que la angiotomografía podría ser la herramienta de elección preoperatoria.

**Palabras clave:** Enfermedad valvular cardíaca, prevalencia, enfermedad arterial coronaria, angiotomografía coronaria, evaluación preoperatoria.

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## INTRODUCTION

In industrialized countries, valvular heart disease (VHD) affects 2.5% (95% CI 2.2-2.7%) of adults and degenerative is the main cause. These patients are older than 65 years and with multiple cardiovascular risk factors.<sup>1</sup> The coexistence with coronary artery disease (CAD) was previously reported at 22%.<sup>2</sup> In developing countries, rheumatic VHD is predominant and could affect up to 20-30 per 1,000 subjects.<sup>3</sup> However, rheumatic patients are younger and with low prevalence of CAD. European and U.S. guidelines recommendation is to perform preoperative invasive coronary angiography (ICA) previous surgery in men over 40 years, postmenopausal women or with at least one cardiovascular risk factor.<sup>4,5</sup> Recently, computed tomography coronary angiography (CTCA) is an option in low to intermediate pre-test probability of CAD. Current guidelines only recommend CTCA when ICA is a risk. In populations with mainly rheumatic VHD this could be the tool of choice, because the low prevalence of CAD. The aim of the study is to demonstrate the prevalence of obstructive CAD diagnosed by CTCA in a population of severe VHD in the preoperative assessment of cardiac valvular surgery.

## MATERIAL AND METHODS

This is an observational cross-sectional study. The study was carried out at the *Instituto Nacional de Cardiología «Ignacio Chávez»* in Mexico City from 2014 to 2017. Patients of both sexes, older than 18 years, with severe VHD diagnosed by echocardiography, were included. All patients underwent CTCA in 256-slice Siemens equipment. Prior to the study, they were premedicated with beta-blockers and/or nitrates. Iodinated contrast dye with a concentration of 350 mEq/L was injected with a caudal rate of infusion of at least 5 mL/sec. All CTCA studies were interpreted in a workstation with software for axial, curvilinear, MPR and MIP analysis. All subjects signed informed consent prior the CTCA. Obstructive CAD was defined as a coronary stenosis >50%. Statistical analysis was performed with Student's t test for quantitative variables,  $\chi^2$ /Fisher's test

for qualitative variables. The independent risk factors of CAD were determined by logistic regression. A two tails  $p < 0.05$  value is considered as statistical significance. All the calculations were made using SPSS software version 22. The present study is part of a prognostic study, previously endorsed by local ethics and research committees.

## RESULTS

A total of 487 patients were included. Baseline characteristics are presented in *table 1*. Average age is  $53 \pm 10$  years, predominance of the female sex (women:men 1.6:1), with low prevalence of cardiovascular risk factors, the most frequent was hypertension (29%). Only 20 patients had obstructive CAD (4.1%), and were older ( $63 \pm 11$  vs  $53 \pm 10$ ,  $p < 0.0001$ ), predominance of males and with high prevalence of hypertension and smoking. No differences were observed in the rest of risk factors. Sub-analysis by aortic or mitral valve disease showed differences in male sex (49 vs 22%,  $p < 0.0001$ ) and dyslipidemia (16 vs 9%,  $p = 0.02$ ), and no differences in CAD. Bicuspid aortic valve patients showed very low prevalence of CAD (1 of 51 patients,  $45 \pm 9$  years, 66% men, 33% hypertension).

**Table 1: Baseline characteristics.**

Variable	Value n=487 (%)
Age (years)	$53.5 \pm 10.8$
Male sex	182 (37.4)
Body mass index (kg/m <sup>2</sup> )	$26.87 \pm 4.2$
Obesity	98 (20.1)
Diabetes	116 (23.8)
Hypertension	142 (29.2)
Dyslipidemia	60 (12.3)
Smoking	38 (7.8)
Valve diagnosis:	
• Aortic stenosis	202 (41.5)
• Aortic regurgitation	42 (8.6)
• Mitral stenosis	39 (8)
• Mitral regurgitation	204 (41.9)
Bicuspid aortic valve	51 (10.5)
Calcium score (UA)	0 (0, 0.9)
CAD (>50% stenosis)	20 (4.1)

ROC curve calculate 60.5 years as a cutoff point to predict CAD (sensitivity=70% and specificity=77%). Only seven female patients had CAD (2%), and these were older ( $68 \pm 14$  vs  $54 \pm 9$  years,  $p < 0.0001$ ), without any other differences. Adjusted predictors of CAD are age older than 60 years (OR: 8.33, 95% CI: 2.95-23.4,  $p < 0.001$ ) and male sex (OR: 4.14, CI 95%: 1.5-11.4,  $p = 0.006$ ).

## DISCUSSION

The evaluation of CAD evolved with the advent of CTCA, which is a lower-cost, non-invasive and safe method. Previous studies demonstrated the diagnostic performance in preoperative assessment of VHD, showing sensitivity of 100% and specificity of 78-80% when compared with ICA.<sup>6,7</sup> Our study shows a very low prevalence of CAD, which does not justify the performance of invasive studies as the first choice. Historical and more recent studies show very different prevalences of CAD,<sup>2,3</sup> based on smaller and older populations, and these are the basis of guidelines. Previously in a study of our institution, a prevalence of 12.5% was reported in a small group of subjects.<sup>8</sup> Our study suggests that CAD predictors are age and male sex, so ICA would be reserved for the following populations: (i) men over 60 years old, (ii) women older than 65 years, (iii) ischemic heart disease known; (iv) aortic stenosis with angina. The use of these new criteria reduces ICA indications recommended in current guidelines. The study presents some limitations: (a) the unicentric nature may represent an inclusion bias that must be taken into account when generalizing the data; (b) there is a selection bias, since the decision to perform CTCA rests on the clinical judgment

of physicians involved in the medical care of patients, so we could underestimate the prevalence in patients with multiple risk factors or the history of angina.

## CONCLUSIONS

In patients with severe VHD the prevalence of obstructive CAD diagnosed by CTCA is 4.1%, and predictor of CAD are age  $> 60$  years and the male sex. In countries with predominance rheumatic VHD, CTCA is the method of choice for preoperative assessment.

## REFERENCES

1. Lung B, Vahanian A. Epidemiology of acquired valvular heart disease. *Can J Cardiol.* 2014; 30 (9): 962-970.
2. Lacy J, Goodin R, McMartin D et al. Coronary atherosclerosis in valvular heart disease. *Ann Thorac Surg.* 1977; 23 (5): 429-435.
3. Emren ZY, Emren SV, Kiliçaslan B et al. Evaluation of the prevalence of coronary artery disease in patients with valvular heart disease. *J Cardiothorac Surg.* 2014; 9: 153.
4. Baumgartner H, Falk V, Bax JJ et al. 2017 ESC/EACTS Guidelines for the management of valvular heart disease. *Eur Heart J.* 2017; 38: 2739-2791.
5. Nishimura RA, Otto CM, Bonow RO et al. 2014 AHA/ACC Guideline for the management of patients with valvular heart disease. *J Am Coll Cardiol.* 2014; 63 (22): e57-185.
6. Gilard M, Cornily JC, Pennec PY et al. Accuracy of multislice computed tomography in the preoperative assessment of coronary disease in patients with aortic valve stenosis. *J Am Coll Cardiol.* 2006; 47: 2020-2024.
7. Meijboon WB, Mollet NR, Van Mieghem CAG et al. Pre-operative computed tomography coronary angiography to detect significant coronary artery disease in patients referred for cardiac valve surgery. *J Am Coll Cardiol.* 2006; 48: 1658-1665.
8. Trevethan-Cravioto S, Cossio-Aranda J, Martinez-Rios MA et al. Predictive value of multi-sliced computed tomography to evaluate obstructive coronary vessel, in the preoperative assessment of non-coronary cardiac surgery. *Arch Cardiol Mex.* 2011; 81 (2): 75-81.

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# Correlation of contrast echocardiography with location of coronary stenosis in myocardial infarction with right ventricle extension

*Correlación de ecocardiografía de contraste con la localización de la estenosis coronaria en infarto de miocardio con extensión al ventrículo derecho*

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**ABSTRACT. Introduction:** Contrast echocardiography (CE) is an important diagnostic tool for routine clinical use in the assessment of the patient with acute coronary syndrome (ACS). Its utility in elucidating the functional status of the right ventricle (RV), is hypothesized to be of use when evaluating the prognosis and hence the need for a prompt intervention. Used in the setting of an ACS, CE can predict which patients are likely to have proximal obstruction of right coronary artery (RCA). Based on that premise, when the clinical suspicion of proximal RCA obstruction is suspected, we can confidently proceed to an earlier intervention. The aim of the study was to determine if the magnitude of the RV perfusion defects correlates with the stenosis site of RCA. **Methods and results:** We studied 24 patients with ACS, in which a CE was performed previous to a coronary angiography. A statistical correlation was made to demonstrate the severity of the obstruction in terms of location; with the walls affected, seen with CE. A direct correlation was found when more than two walls were involved. Those patients had proximal RCA obstructions, and therefore a worse prognosis. **Conclusion:** There is a good correlation between the magnitude of the perfusion defect observed by CE and the stenosis site of the responsible coronary artery.

**Keywords:** Contrast echocardiography, perfusion defect, right ventricle myocardial infarction, coronary stenosis.

**RESUMEN. Introducción:** La ecocardiografía de contraste (EC) es una importante herramienta de diagnóstico para el uso clínico de rutina en la evaluación del paciente con síndrome coronario agudo (SCA). Su utilidad para dilucidar el estado funcional del ventrículo derecho (VD) podría usarse para evaluar el pronóstico y, por lo tanto, la necesidad de una intervención rápida. Utilizado en el contexto de un SCA, la EC puede predecir qué pacientes tienen probabilidad de tener una obstrucción proximal de la arteria coronaria derecha (CD). Sobre la base de esa premisa, cuando existe sospecha clínica de obstrucción de la CD proximal, podemos proceder con confianza a una intervención más temprana. El objetivo del estudio fue determinar si la magnitud de los defectos de perfusión del VD se correlaciona con el sitio de estenosis de CD. **Métodos y resultados:** Se estudiaron 24 pacientes con SCA, en los que se realizó un EC previo a una angiografía coronaria. Luego se realizó correlación estadística para demostrar la gravedad de la obstrucción en términos de ubicación con las paredes afectadas. Se encontró una correlación directa cuando se involucraron más de dos paredes. Esos pacientes tenían obstrucciones de la CD proximal y, por lo tanto un peor pronóstico. **Conclusión:** Existe una buena correlación entre la magnitud del defecto de perfusión observado por la EC y el sitio de la estenosis de la arteria responsable.

**Palabras clave:** Ecocardiografía con contraste, defecto de perfusión, infarto de miocardio de ventrículo derecho, estenosis coronaria.

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## INTRODUCTION

The assessment of ventricular function is very important in the clinical course of some cardiovascular diseases such as acute myocardial infarction with extension to the right ventricle (RV). In these cases, functional assessment at different times is crucial, due to its prognostic and therapeutic implications.<sup>1-3</sup> The proximal occlusion of the right coronary artery (RCA) compromises the perfusion of the ventricular branches, which leads to RV dysfunction, whereas distal occlusion rarely produces such an effect.

The intravenous administration of contrast substances that cross the pulmonary barrier allows the obtention of an adequate visualization of the endocardium and therefore an excellent delimitation of the three portions of the RV, such as the tract of entry, the trabecular portion and the outflow tract. This facilitates the measurement of RV areas in diastole and systole, even in patients with poor acoustic window and also allows an accurate assessment of the coronary microcirculation.<sup>4-6</sup> There are many studies in the literature that have evaluated the coronary microcirculation in patients with left ventricular infarction,<sup>6,7</sup> but there are still no studies in humans that assess the coronary microcirculation of the RV in the presence of a posteroinferior infarction with extension to the right cavities.

## MATERIAL AND METHODS

**Design and population:** This is a prospective, double blind and observational study. Performed from October 2002 to July 2003, 24 consecutive patients of the Coronary Care Unit of *Instituto Nacional de Cardiología «Ignacio Chávez»* (Mexico City) were included, with a diagnosis of myocardial infarction of the posteroinferior wall of the left ventricle with extension to the RV. We included patients with acute myocardial infarction presenting with precordial oppressive pain of more than 30 minutes duration and ST segment elevation greater than 1 mm in at least two contiguous leads of the inferior wall of the left ventricle and enzymatic elevation of creatine phosphokinase at twice higher than its normal value. To determine the extent of

the infarction to the RV, the following criteria were considered: elevation of the ST segment greater than 1 mm in the right leads V3r and/or V4r. We excluded patients with a history of: old myocardial infarction, severe chronic lung disease that would have required hospitalization or oxygen therapy, congenital heart disease, cardiomyopathies, constrictive pericarditis or pulmonary arterial hypertension. All patients underwent conventional transthoracic CE and coronary angiography. All patients received follow-up during their hospital stay. The end points during the follow-up were death, development of cardiogenic shock, low cardiac output and rhythm disorders.

**Echocardiogram:** The conventional transthoracic echocardiogram was performed with a Hewlett Packard Sonos 5500, equipped with an S3 transducer. In the 4-chamber apical plane, the mobility of both ventricles was assessed and it was considered normal when parietal mobility and systolic thickening were symmetric; hypokinesia when decreased parietal mobility and systolic thickening were present; akinesia when parietal mobility and systolic thickening were absent and dyskinesia when there was outward systolic bulging of a certain segment. The shortening fraction of areas of the right ventricle (SFA-RV) was determined in the apical four chamber view, delimiting the area of the RV in the end diastole and in the telesystole with the following formula:  $(ADV-ASVD/ADVD) \times 100$ . The SFA-RV was considered normal when the value was equal to or greater than 40%. The right ventricle ejection fraction (RVEF) was calculated with the values of the descent of the tricuspid annulus in diastole and in systole, in the apical four chamber view. The formula  $DAnTD-DanTS$  was used and the difference obtained was multiplied by 3.2. The RVEF was considered normal when the value was equal to or greater than 44%. For CE, a Sonos 5500 instrument with harmonic perfusion and Doppler angio software was used. For the assessment of the coronary microcirculation of the RV, the images were used in the short parasternal axis at the level of both ventricles and apical four chamber view. Optison (perfluoropropane with diameter of the microbubbles of 3.7 microns) was applied intravenously per ulnar vein, in bolus at a rate of 0.4 mL/minute, with a 3 mL

**Table 1: Contrast echocardiogram BEFORE reperfusion treatment.**

Number of patients	Number of walls with absence of perfusion	Site of coronary obstruction	>1 vessel affected	Collateral circulation	Clinical evolution	End point
2	3	Proximal RCA	Yes	No	Cardiogenic shock, rhythm disturbances	Dead
1	2	Proximal RCA	Yes	Yes	Cardiogenic shock, AV block	Alive
1	2	Proximal RCA	Yes	Yes	Cardiogenic shock, AV block	Alive
1	1	Proximal RCA	No	Yes	Low output, A-Fib	Alive
1	1	Distal RCA	No	No	Stable	Alive
2	Normal	Medial Cx	No	No	Low output	Alive
1	Normal	Proximal RCA	No	Yes	Low output	Alive

RCA = Right coronary artery; Cx = Circunflex artery.

saline solution bolus and subsequently at a rate of 1 mL/minute. Microvascular myocardial perfusion with harmonic and angio Doppler was evaluated, using the triggered modality in telesystole with shots of 1: 1, 1: 5. The contrast image was considered adequate when the gray scale was greater than 70 pixels. The coronary microcirculation was measured qualitatively in the walls of the RV and it was determined to be normal when the distribution of the microbubbles was homogeneous in the three walls of the RV: anterior, free and inferior wall. Hypo perfusion was determined when heterogeneous distribution of the microbubbles was observed in some of the walls and absence of perfusion; when there was absence of microbubbles in any of the walls of the right ventricle. Perfusion images with contrast, RV function and mobility were correlated with coronary angiography at the site of the lesion and coronary flow. The CE was evaluated qualitatively by two echocardiographers and the site of stenosis and flow in the coronary artery related to the infarction were assessed by an interventional cardiologist based on the TIMI classification.

**Statistic analysis:** The data were represented in a contingency table and the

Spearman correlation coefficient was calculated to determine the degree of correlation between the CE and coronary angiography. The intra- and inter-observer concordance was assessed with the Kappa index in the perfusion images of the walls of the RV.

## RESULTS

Twenty-four patients were studied: 21 (87.5%) were males, with an average age of  $58 \pm 13$  years (35-76). Nine patients had dyslipidemia, 9 diabetes mellitus type II, 9 arterial hypertension and 7 exogenous obesity. At admission, 18 patients (75%) had ST segment elevation in V3r and V4r. The average value of creatine phosphokinase was  $2,032 \pm 1,407$  U/dL (11-4,100). The average MB fraction was  $142 \pm 102$  U/dL (18-400). Fifteen patients (62.5%) arrived within the therapeutic window. CE was performed in 9 patients before reperfusion treatment, whether they were in a therapeutic window (TW) or outside it (OTW). Two patients (one OTW and the other TW) presented three-wall hypoperfusion or absence of perfusion of the RV; coronary angiography showed affection of the proximal segment of the right coronary artery (RCA); angioplasty

failed in both patients and they died. In one TW patient, there was hypoperfusion of the three walls and perforation of RV. He presented atrioventricular (AV) block and cardiogenic shock; coronary angiography revealed involvement of the proximal segment of the RCA; after revascularization, hemodynamic state improved. In other OTW patient, hypoperfusion was detected in two walls of the RV and presented AV block and cardiogenic shock, after revascularization the patient remained stable; coronary angiography showed obstruction of the proximal segment of the RCA. In 1 OTW patient, hypoperfusion was found in one RV wall, presented with atrial fibrillation and low cardiac output; its coronary angiography showed proximal RCA lesion with improvement after revascularization. All these patients mentioned above have decreased RVEF and SFA-RV. One TW patient presented with hypoperfusion of RV wall and normal RVEF and SFA-RV, with a good evolution. Coronary

angiography showed slow flow and thrombus in the distal segment of the RCA. In 2 patients the RV perfusion was normal (one TW and another OTW) and mid dominant circumflex segment lesion was observed. The EC showed normal perfusion in one patient with proximal RCA lesion (Table 1).

The EC performed in 9 patients after reperfusion treatment OTW or TW demonstrated: 3 patients presented perfusion disorders; (1) TW patient with hypoperfusion of free and posterior wall with decreased RVEF and SFA-RV, and ostial RCA lesion with a failed reperfusion therapy, the patient died. (2) OTW patient with posterior wall hypoperfusion, normal RVEF and SFA-RV, and proximal RCA lesion with a failed reperfusion therapy, showed hemodynamic stability. (3) One patient after failed thrombolysis, anterior hypoperfusion was found in the EC and lesion in the middle RCA. In 6 patients, the EC of the RV was normal, one with proximal RCA, five with mid RCA (Table 2).

**Table 2: Contrast echocardiogram AFTER reperfusion treatment.**

Number of patients	Number of walls with absence of perfusion	PTCA and Site of coronary obstruction	> 1 vessel affected	Collateral Circulation	Clinical evolution	Endpoint
1	3	Proximal RCA, PTCA failed	Yes	No	Cardiogenic shock	Death
1	1	Proximal RCA, PTCA failed	No	Yes	Stable	Alive
1	1	Medial segment Trombolysis failed	No	Yes	Stable	Alive
1	Normal	Proximal RCA, PTCA success	No	No	Stable	Alive
2	Normal	Medial RCA, PTCA success	No	No	Stable	Alive
3	Normal	Medial RCA, PTCA success	No	No	Stable	Alive

RCA = Right coronary artery; PTCA = Percutaneous transluminal coronary angioplasty.



The correlation between CE and coronary angiography was 0.77. The intra- and inter-observer concordance in the perfusion images of the walls of the right ventricle was 0.84 and 0.83, respectively.

## DISCUSSION

Patients who present with a diagnosis of infarction of the posteroinferior myocardium with extension to RV, outside of a therapeutic window for thrombolysis or primary angioplasty, the EC will provide information on how severe the ischemia is, determining the site of obstruction of the lesion and therefore leading us to take the decision of urgent or elective revascularization in these patients.<sup>7-9</sup> The EC can help during the first week of the infarction, since it allows assessing the right ventricular function without the need to transfer the patient to the nuclear cardiology study.<sup>10</sup> Alterations in RV perfusion are related to RV dysfunction and unfavorable clinical evolution. When two or more walls of the right ventricle are affected, obstruction is found in the proximal segment of the RCA, there are important obstructive lesions in more than two vessels and there is no collateral circulation. When the RV perfusion remained normal due to probable spontaneous thrombolysis or presence of collaterals before reperfusion, the clinical evolution was favorable.

Now if we relate the clinical state and RV perfusion, we observed that there were conduction disorders and cardiogenic shock in absence of perfusion or hypoperfusion of two or more RV walls. The EC shows that when the perfusion is normal or there is hypoperfusion of a single wall, it must be related to involvement of either the middle segment of the RCA or the circumflex and/or the presence of collateral circulation. When the reperfusion treatment fails and there is hypoperfusion in more than one RV walls, it is related to a lesion in the proximal segment and involvement in more than two vessels, with absence of collateral circulation. In presence of collateral circulation prognosis is better.

## CONCLUSIONS

The clinical evolution is not favorable when there are defects of myocardial perfusion in two or more walls of the RV and lesions in other vessels without collateral circulation. The absence of perfusion of the RV and its dysfunction is mainly due to involvement of the proximal segment and is related to higher mortality. The proximal occlusion of the RCA causes RV dysfunction. There is a good correlation between the magnitude of the perfusion defect observed by CE and the stenosis site of the responsible coronary artery.

## REFERENCES

1. Bowers TR et al. Patterns of coronary compromise resulting in acute right ventricular ischemic dysfunction. *Circulation*. 2002; 106: 1104-1109.
2. Shiraki H, Yoshikawa T, Anzai T et al. Association between preinfarction angina and a lower risk of right ventricular infarction. *N Engl J Med*. 1998; 338 (14): 941-947.
3. Goldstein JA, Tweddell JS, Barzilai B et al. Importance of left ventricular function and systolic interaction to right ventricular performance during acute right heart ischemia. *J Am Coll Cardiol*. 1999; 18: 1564-1572.
4. Borrayo G et al. Valoración de la función ventricular derecha mediante ecocardiografía de contraste en pacientes con infarto agudo de miocardio. *Rev Esp Cardiol*. 2003; 56: 175-180.
5. Tei C, Sakamaki T, Shah P. Myocardial contrast echocardiography: a reproducible technique of myocardial opacification for identifying regional perfusion defects. *Circulation*. 1983; 67: 585-593.
6. Fernández J, García M, Moreno M et al. Utilidad de las nuevas técnicas de imagen, segundo armónico y contraste en la visualización del borde endocárdico. Análisis de la reproducibilidad en la valoración de la contracción segmentaria. *Rev Esp Cardiol*. 2000; 53: 1459-1466.
7. Steven B, Lasterm SB, Shelton TJ et al. Determinants of the recovery of right ventricular performance following experimental chronic right coronary artery occlusion. *Circulation*. 1993; 88: 696-708.
8. Jacobs AK, Leopold JA, Bates E et al. Cardiogenic shock caused by right ventricular infarction. *J Am Coll Cardiol*. 2003; 41: 1273-1279.
9. Zeymer U, Neuhaus KL, Wescheider K et al. Effects of thrombolytic therapy in acute inferior myocardial infarction with or without right ventricular involvement. *J Am Coll Cardiol*. 1998; 32: 876-881.
10. Lepper W, Kamp O, Vanoverschelde JL et al. Intravenous myocardial contrast echocardiography predicts left ventricular remodeling in patients with acute myocardial infarction. *J Am Soc Echocardiogr*. 2002; 15: 849-856.

# Acute dislocation of a Perceval valve treated with TAVI valve-in-valve: a literature review and case report

*Dislocación aguda de una válvula Perceval tratada con una válvula en válvula TAVI: una revisión de la literatura y reporte de caso*

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**ABSTRACT.** Dislocation of a Perceval valve is an extremely rare situation, with only a handful of proven cases entailing migration or dysfunction of the valve. A 76-year-old male with severe dyspnea with a severe aortic stenosis revealed by transthoracic echocardiography (TTE); surgical procedure with a Perceval valve was made without any complications. Within the first 24-hours in the Intensive Care Unit, the patient presented acute heart failure. The transesophageal echocardiography (TEE) showed severe paravalvular leak, then a fluoroscopy confirmed the dislocated and dysfunctional Perceval valve. An emergency transcatheter aortic valve-in-valve procedure with a CoreValve 29 mm was performed to correct the dislocated valve. To the best of our knowledge, this is the first documented case in the world of an acute dislocated Perceval valve treated with a TAVI valve-in-valve.

**Keywords:** Perceval valve, valve-in-valve, dislocation.

**RESUMEN.** La dislocación de una válvula Perceval es una situación extremadamente rara, unos pocos casos probados que implican migración o disfunción de la válvula. Varón de 76 años con disnea severa y estenosis aórtica severa revelada por ecocardiografía transtorácica (ETT); el procedimiento quirúrgico con una válvula Perceval se realizó sin complicaciones. En las primeras 24 horas en la Unidad de Cuidados Intensivos, el paciente presentó insuficiencia cardíaca aguda. La ecocardiografía transesofágica (ETE) mostró una fuga para-valvular severa, luego una fluoroscopia confirmó la dislocación y disfunción de la válvula Perceval. Se realizó un procedimiento de emergencia de válvula aórtica transcáteter con una válvula CoreValve de 29 mm para corregir la válvula dislocada. Hasta donde sabemos, éste es el primer caso documentado en el mundo de una válvula Perceval aguda dislocada tratada con una válvula en válvula TAVI.

**Palabras clave:** Válvula ascendente, válvula en válvula, dislocación.

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## INTRODUCTION

Aortic stenosis is the most prevalent valvular heart condition in the world, and its prevalence has constantly increased over the years.<sup>1,2</sup> Surgical aortic valve replacement (SAVR) stands as the «gold standard» treatment for patients with aortic valve stenosis (AVS).<sup>3</sup> SAVR with sutureless Perceval valve has emerged as the new biological aortic valve bearing easy

implantation potential, reduced myocardial ischemic time, surgical trauma; moreover, it has proven safe and compares favorably to conventional SAVR<sup>3</sup> with a less invasive approach for high-risk patients.<sup>4</sup> Its safety and efficacy have been reported in intermediate-risk population in a 1-year follow-up.<sup>5</sup>

An alternative to SAVR is the transcatheter aortic valve implant (TAVI) that has recently become widely accepted for patients with



intermediate-high risk for conventional SAVR.<sup>6</sup> TAVI is emerging above AVS as a valuable procedure in patients with dysfunctioning biological aortic valves, who are deemed inoperable using conventional surgery as a valve-in-valve (ViV) procedure.<sup>7,8</sup> There are no reports regarding the use of TAVI as an emergency procedure to treat an acute dislocation of a Perceval valve.

### CASE REPORT

A 76-year-old male with history of hypertension presented with severe dyspnea (NYHA-III), without fever, syncope or chest pains. A transthoracic echocardiography (TTE) revealed a severe aortic stenosis with a left ventricle ejection fraction (LVEF): 62%, aortic valve area (AVA): 0.59 cm<sup>2</sup>, mean gradient (MG): 25 mmHg; peak velocity (PV): 3.5 m/s; paradoxical low flow low gradient; coronary arteries not compromised, STS score 4%. The patient was assessed by our multidisciplinary cardiology team, and it was determined that he was suitable for surgical aortic valve replacement (SAVR) with a Perceval valve.

A surgical procedure was carried out with a medium size Perceval (Sorin, Saluggia, Italy) valve based on surgical technique for sizing, placed without any complications, MG: 12 mmHg. The patient was transferred to the intensive care unit, but twenty-four hours after the procedure, the patient presented acute heart failure and his clinical condition

deteriorated rapidly despite inotropic support. A transesophageal echocardiography (TEE) was performed documenting severe paravalvular leak; therefore, the heart team assessed the patient in a cath lab. A fluoroscopy was performed to confirm the situation of the dysfunctional Perceval valve seen in the TEE, and a dislocation of the Perceval valve was found (*Figure 1*, *Video 1* [Supplementary File]). A TEE confirmed the dislocation and the severe paravalvular leak (*Figure 2*); as a result of which, an emergency TAVI-ViV procedure with a CoreValve 29 mm was placed (*Videos 2 to 4* [Supplementary File]). The fluoroscopy confirmed proper release and functioning of the valve (*Video 5* [Supplementary File]) and TEE: MG: 10 mmHg, PV: 2.2 m/s, LVEF: 52%, without central or paravalvular leak. The clinical status of the patient improved rapidly, and the patient was discharged without any complications one week after procedure.

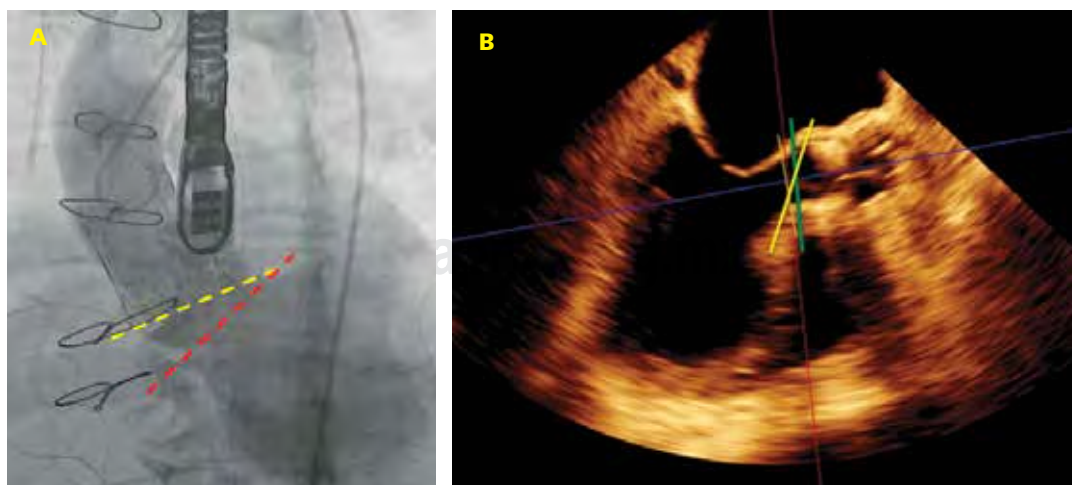
### DISCUSSION

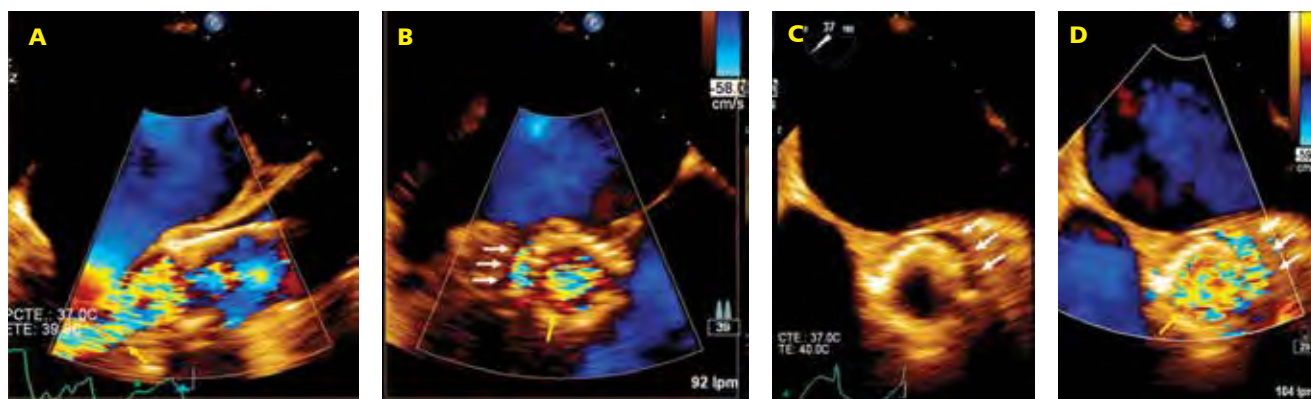
The data from three European prospective multicenter trials (Pilot, Pivotal and CAVALIER), reported that major paravalvular leak occurred in 1.4 and 1% within the 1st to 5th year follow-up. There were only 3 cases of explants due to miss-sizing, but no cases of valve migration or dislocation after surgery were reported.<sup>9</sup>

There are previous reports of failure of a Perceval bioprosthesis rescued with TAVI-ViV, but in a scenario of 3-year early failure, probably

**Figure 1:**

- A)** Fluoroscopy.  
Red line: aortic annulus. Yellow line: prosthesis upward dislocation.
- B)** Two-dimensional echocardiography of the migrated prosthesis into the ascending aorta.  
Green line: aortic annulus. Yellow line: prosthesis upward dislocation.





**Figure 2:** Transesophageal echocardiogram. **A)** Long axis: severe central aortic regurgitation (yellow arrow). **B)** Short-axis: severe central aortic regurgitation (yellow arrow) and paravalvular leak (white arrow). **C)** Short-axis view: dehiscence of the valve corresponding with the dislocated valve (white arrow). **D)** Short axis: severe paravalvular leak (yellow arrow) and central aortic regurgitation (white arrow).

### Video 1:

Fluoroscopy  
diagnosis of  
Perceval valve  
dislocation.



[www.medigraphic.com/videos/ciu/192\\_1](http://www.medigraphic.com/videos/ciu/192_1)

### Video 2:

Super stiff guidewire  
throw the Perceval  
valve inside the left  
ventricle.



[www.medigraphic.com/videos/ciu/192\\_2](http://www.medigraphic.com/videos/ciu/192_2)

due to a sudden tearing of a leaflet.<sup>10</sup> Another case exhibited a degenerated prosthesis with stenosis and severe aortic regurgitation without paravalvular regurgitation for 5 years after SAVR,<sup>11</sup> both of which were treated with an expandable Edwards SAPIEN 3 TAVI-ViV procedure.

Although migration of a Perceval prosthesis had been described, reports showed late migration after surgery (3 and 15 months), caused by a displacement at the Valsalva left sinus and a distorted and displaced valve. Both cases were dealt with a reoperation either by preserving the valve with pledgeted stitches<sup>12</sup> or by replacing the standard aortic valve after a complete calcium debridement.<sup>13</sup> Several hypotheses could explain the delayed migration such as inadequate sizing of a non-uniformly decalcified annulus.

Since the stability of these prostheses rely on radial forces, correct sizing and proper annular decalcification are crucial to avoid displacement. There is evidence that excessive oversizing of the Perceval valve is detrimental, however still there is no consensus on how to size properly the valve to be implanted, although some authors recommend surgical sizers (such as this case), others rather do sizing based on the friction of the white obturator while going through the annulus; others propose the cardiac CT as a simple solution that had proved their role with the TAVI procedure<sup>14</sup> and when hesitating between two sizes it is proposed by Baert et al<sup>15</sup> to choose the smaller size.



**Video 3:**

Move forward of the 29 mm CoreValve towards the aortic annulus.



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**Video 5:** Functional TAVI-ViV of the CoreValve 29 mm.  
[www.medigraphic.com/videos/ciu/192\\_5](http://www.medigraphic.com/videos/ciu/192_5)

**Video 4:**

Release of 29 mm CoreValve.



[www.medigraphic.com/videos/ciu/192\\_4](http://www.medigraphic.com/videos/ciu/192_4)

determine the «new» left ventricle outflow tract a CoreValve 29 mm was selected for the implant.

## CONCLUSIONS

We can conclude that this case clearly illustrates the unusual dislocation of a Perceval valve and the successful TAVI-ViV procedure as an emergency treatment notwithstanding the fact that similar procedures in patients with sutured, stented, or stentless aortic valve prostheses and even in patients with dysfunction of a Perceval valve<sup>10,16</sup> had been reported. On the one hand, there is no data reported on the feasibility of the TAVI-ViV procedure in an acute dislocated sutureless aortic valve prosthesis. On the other hand, this case demonstrates that the TAVI-ViV procedure with a CoreValve is feasible, it can be performed easily and quickly in an urgent dislocated Perceval sutureless bioprosthetic valve with a beneficial outcome, the appropriate method to determine the proper size of the valve for a TAVI-ViV should be done with a 3D transesophageal echocardiography, and the utility of the app should be avoided in acute cases since the anatomy can be distorted.

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In our case although the surgeon was familiarized with the surgical technique, we cannot assure whether or not the cause of the dislocation could be related to the valve undersizing or to a high positioning of the prosthesis. For the size selection of the ViV strategy, the valve-in-valve aortic app (version 2.0) recommends CoreValve 26 mm for a medium Perceval (true internal diameter of 19.5-21 mm). However, due to the lack of a clear view of the dislocated aortic annulus, it was necessary for us to assess a new evaluation; hence, a 3D annular size measured with transesophageal echocardiography was made to

## REFERENCES

1. Cowell S, Newby D, Boon N, et al. Calcific aortic stenosis: same old story? Age and Ageing. 2004; 33: 538-544.
2. Iung B, Baron G, Butchart EG et al. A prospective survey of patients with valvular heart disease in Europe: The Euro Heart Survey on Valvular Heart Disease. Eur Heart J. 2003; 24: 1231-1243.
3. Shrestha M, Folliguet T, Meuris B, Dibie A et al. Sutureless Perceval S aortic valve replacement: a multicenter, prospective pilot trial. J Heart Valve Dis. 2009; 18: 698-702.
4. Zannis K, Folliguet T, Laborde F. New sutureless aortic valve prosthesis: another tool in less invasive aortic valve replacement. Curr Opin Cardiol. 2012; 27: 125-129.
5. Fischlein T, Meuris B, Hakim-Meibodi K et al. The sutureless aortic valve at 1 year: A large multicenter cohort study. J Thorac Cardiovasc Surg. 2016; 151: 1617-1626.
6. PARTNER 2 Investigators. Transcatheter or surgical aortic-valve replacement in intermediate-risk patients. N Engl J Med. 2016; 374: 1609-1620.
7. Loyalka P, Montgomery KB, Nguyen TC et al. Valve-in-valve transcatheter aortic valve implantation: a novel approach to treat paravalvular leak. Ann Thorac Surg. 2017; 104: e325-e327.
8. Dvir D, Webb JG, Bleiziffer S et al. Transcatheter aortic valve implantation in failed bioprosthetic surgical valves. JAMA. 2014; 312: 162-170.
9. Shrestha M, Fischlein T, Meuris B et al. European multicenter experience with the sutureless Perceval valve: clinical and haemodynamic outcomes up to 5 years in over 700 patients. Eur J Cardiothoracic Surg. 2016; 49: 234-241.
10. Durand E, Tron C, Eltchaninoff H. Emergency transcatheter aortic valve implantation for acute and early failure of sutureless Perceval aortic valve. Can J Cardiol. 2015; 31: 1204.e13-5.
11. Mangner N, Holzhey D, Misfeld M et al. Treatment of a degenerated sutureless Sorin Perceval® valve using an Edwards SAPIEN 3. Interact Cardiovasc Thorac Surg. 2018; 26: 364-366.
12. Concistre G, Miceli A, Chiaramonti F et al. Delayed dislocation of a sutureless aortic bioprosthesis: the first case. Interact Cardiovasc Thorac Surg. 2012; 14: 892-893.
13. Amr G, Ghoneim A, Giraldeau G et al. First case of a sutureless Perceval valve delayed proximal migration. J Thorac Cardiovasc Surg. 2017; 153: e21-e23.
14. Cerillo AG, Amoretti F, Mariani M et al. Increased gradients after aortic valve replacement with the perceval valve: the role of oversizing. Ann Thorac Surg. 2018; 106: 121-128.
15. Baert J, Astarci P, Noirhomme P, de Kerchove L. The risk of oversizing with sutureless bioprosthesis in small aortic annulus. J Thorac Cardiovasc Surg. 2017; 153: 270-272.
16. Kalra A, Reyes M, Yang EY et al. Transcatheter aortic valve replacement for perceval sutureless aortic valve failure. J Invasive Cardiol. 2017; 29: E65-E66.

## Conflict of interest statement

The authors have no conflicts of interest to declare.

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# Cardiotoxicity, strain: evidence to date

*Cardiotoxicidad, strain: evidencia actual*

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**ABSTRACT.** In the last decade there have been notable advances in the treatment of cancer. The introduction of targeted therapies has increased the rates of cure and remission in some of them. The net result is the emergence of a cohort of patients whose survival will be sufficient to cause cardiac side effects of the therapies used. In this review we will deal with the current knowledge about the mechanisms of cardiotoxicity, the traditional methods for its evaluation and the new strategies for its early detection.

**Keywords:** Cardiotoxicity, strain rate, echocardiography, oncology.

**RESUMEN.** En la última década ha habido avances notables en el tratamiento del cáncer. La introducción de terapias dirigidas ha aumentado las tasas de cura y remisión en algunas de ellas. El resultado neto es la aparición de una cohorte de pacientes cuya supervivencia será suficiente para causar efectos secundarios cardíacos de las terapias utilizadas. En esta revisión trataremos el conocimiento actual sobre los mecanismos de cardiotoxicidad, los métodos tradicionales para su evaluación y las nuevas estrategias para su detección temprana.

**Palabras clave:** Cardiotoxicidad, strain rate, ecocardiografía, oncología.

## INTRODUCTION

Breast cancer in Mexico is the leading cause of death in women due to malignant neoplasms since 2,006; in Mexican women it represents 14% of deaths related to cancer.<sup>1</sup> According to estimates for 2030 of GLOBOCAN in Mexico, 24,386 women will have been diagnosed and 9,778 will die from breast cancer. Survival at 5 years in patients with breast cancer and access to oncological treatment in the public sector has been reported as 82% (95% CI, 81-84%), next few years a high number of women survivors of cancer with significant cardiovascular risk basal and/or secondary to multimodal oncological treatment (chemotherapy, white therapy and/or radiotherapy) received with curative oncological intent. The economic pressure for the Mexican public health system and the

urgent need for care for cancer survivors in Mexico is a reality.<sup>2-4</sup> Estimates of disability-adjusted life years or DALYs (Disability Adjusted Life Years) from 2,006 to 2,016 have been reported in breast cancer; for women, breast cancer is the cancer with the highest incidence, mortality and DALYs (1.7 million incidents, 535,000 deaths and 14.9 million DALYs). A complex and costly picture of attention is expected in Latin American countries with limited resources.

It has been reported that breast cancer survivors have a 3-fold increased risk of developing heart failure within the first 5 years after cancer diagnosis compared to the general population.<sup>5-7</sup>

The term 'cardiotoxicity' is used to refer to LV dysfunction. The two main anticancer agents responsible for LV dysfunction are

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anthracyclines and targeted therapies (tyrosine kinase inhibitor, anti-human epidermal growth factor receptor 2, anti-vascular endothelial growth factor, proteasome inhibitors).<sup>8-10</sup> Recently, immune fulminant myocarditis was reported with the use of checkpoint immune inhibitors (anti-programmed cell death protein 1, anti-programmed cell death ligand 1, anti-cytotoxic T lymphocyte-associated protein 4), suggesting new cardiotoxicity pathways. LV dysfunction remains asymptomatic for a long time, but once symptomatic, the prognosis is one of the poorest in the heart failure population.<sup>11-14</sup>

## CARDIOTOXICITY

The concept of left ventricular dysfunction induced by cardiotoxic chemotherapies is defined by a decrease in left ventricular ejection fraction (LVEF) of >10 percentage points to a value 15% of the global longitudinal strain are useful tools. According to the European Society of Cardiology, LVEF assessment can be performed by echocardiography (attempting to favour 3D LVEF), cardiac nuclear imaging and cardiac MRI.

### Types of left ventricular dysfunction (LVD)

- **Type I:** due to cell death, whereby ventricular dysfunction and heart failure can occur years after antineoplastic treatment is concluded, it is dose dependent and implies worse prognosis (eg, anthracyclines);
- **Type II:** there is a compromise of myocyte function, without loss of these, for which ventricular dysfunction and heart failure are reversible, without leaving long-term sequelae (ex: trastuzumab).<sup>15,16</sup>

### Initial cardiac evaluation prior to treatment and stratification of cardiotoxicity risk

To avoid the cardiotoxic effects to all patients who are going to undergo oncological treatment by radio or chemotherapy, a stratification of risk prior to treatment is performed, then follow-up is scheduled during the same and according to the results arise the measures to be taken through multidisciplinary assessment.

The initial assessment should consist of a history and physical examination aimed at assessing the presence of heart disease, ECG to detect arrhythmias or alterations in the QTc interval, echocardiogram and biomarkers for functional and structural assessment. Recommend healthy lifestyles, identify and establish a correct treatment for CVRF. Patients with CVRF present a higher risk of developing cardiotoxicity. CV risk factors are considered: unfavorable lifestyle (smoking, overweight and reduced physical activity) pre-existing diseases such as dyslipidemia, hypertension, diabetes, coronary heart disease, heart failure, stroke or thromboembolic events. Patients with arrhythmias and ventricular dysfunction with EF lower than 50-55% are considered high risk group to develop cardiotoxicity with oncological treatment.

The guide of the American Society of Clinical Oncology (ASCO) in 2,016 developed guidelines for the prevention and monitoring of cardiac dysfunction in cancer survivors. This guide recommends that patients receiving the following treatment should be considered at high risk to develop cardiac dysfunction.<sup>16,17</sup>

### Risk factors for cardiotoxicity

1. High doses of anthracyclines (eg: cumulative dose of doxorubicin greater than or equal to 250 mg/m<sup>2</sup>, cumulative dose of epirubicin greater than or equal to 600 mg/m<sup>2</sup>.
  - High doses of radiotherapy (greater or equal 30 Gy) with the heart within the treatment field.
  - Low doses of anthracyclines in combination with low doses of radiation with the heart in the irradiated area.
2. Treatment with low doses of anthracyclines or trastuzumab alone and the presence of one of the following CVRF:
  - Multiple FCRCV (greater or equal 2): smoking, obesity, HTA, DBT, DSLP during or after treatment.
  - Age greater than or equal to 60 years at the time of cancer treatment.
  - Commitment of cardiac function (LVEF 50-55%, antecedent of AMI, valvular disease of moderate degree) at any time before or after treatment.



3. Treatment with low doses of anthracyclines (<250 mg/m<sup>2</sup> ASC), followed by treatment with trastuzumab.<sup>17</sup>

### Cardiovascular monitoring during treatment

#### *Left ventricular ejection fraction (LVEF)*

For years, LVEF was the only parameter monitoring method that detected cardiotoxicity, and multigated acquisition was the most common method used by oncologists. In the past 10 years, 2D and 3D echocardiography has become the standard for myocardial function assessment. 3D LVEF was shown to have the lowest temporal variability. A recent study of breast cancer patients has suggested that nadir LVEF values were identified by 3D echocardiography earlier than 2D echocardiography, suggesting that 3D measured LVEF might be a useful method to identify early cardiac injury. 3D LVEF and myocardial strain were associated with concurrent and subsequent changes in 2D LVEF, and concurrent change in diastolic function (E/e'). When adjusted for the respective 2D parameters, post-anthracycline 3D LVEF and global circumferential strain predicted subsequent 2D LVEF.<sup>18-20</sup>

Cardiac magnetic resonance (CMR) is particularly interesting in the cancer population, because of its spatial and temporal resolution, its reproducibility and accuracy for LVEF assessment. Recent evidence suggests that LV global circumferential strain and GLS measured with feature-tracking CMR may also identify early LV dysfunction. CMR also helps explain the decrease in LVEF and strain in cancer patients LV end diastolic volume due to decrease in preload (vomiting, diarrhea, sepsis leading to dehydration); therefore, LV end diastolic volume and LV end systolic volume should always be taken into account. CMR may facilitate our understanding for cardiotoxicity pathogenesis. Myocardial tissue changes, such as intracellular and interstitial edema, and fibrosis, may precede the alterations in LV volumes, reduction in LVEF, or changes in myocardial strain and may represent early markers of myocardial injury. Also, there is accumulating evidence of the presence of

diffuse interstitial fibrosis (assessed by increased T1 mapping and extracellular volume fraction in anthracycline-induced cardiomyopathy), independent of cardiovascular comorbidities and associated with impaired diastolic function. There are also many etiologies of myocellular dysfunction that lead to LV dysfunction in patients receiving cardiotoxic chemotherapies that CMR can diagnose: myocarditis, stress-induced cardiomyopathy, myocellular injury and interstitial fibrosis.<sup>21-23</sup>

#### *Deformation imaging by 2D echocardiography*

Global longitudinal strain (GLS) is a strong predictor of cardiovascular morbidity and mortality in several cardiac diseases, and seems to be a consistent marker of cardiotoxicity. The expert consensus of the American Society of Echocardiography and the European Association of Cardiovascular Imaging considered a 15% reduction of GLS as a significant change to detect cardiotoxicity. In a small study of 44 patients treated with anthracycline and trastuzumab, Sawaya et al. showed that a 10% decrease of GLS combined with an increase of TnI from baseline to 3 months had an 83% positive predictive value and an 89% negative predictive value to detect cardiotoxicity (as defined as a symptomatic decrease >5% of LVEF with LVEF <55% or an asymptomatic decrease >10% with LVEF <55%).<sup>24,25</sup> Although the early detection of myocardial changes seems to be conceptually important, the real value of these changes lies in their capacity as prognostic markers of outcomes such as the same reduction in LVEF or the development of heart failure. The prognostic value has been evaluated in 8 studies involving about 452 patients (47 to 51 years old), most of them women with breast cancer, all of them received anthracyclines and most of them trastuzumab, the duration of follow-up was 12 to 15 months on average. A fall in the GLS of 10 and 15% predicted cardiotoxicity (including asymptomatic and symptomatic dysfunction).<sup>26,27</sup> The 95% confidence interval for the optimal cut-off value of the strain was 8.3 to 14.6%. On the contrary, the radial and circumferential deformation were not statistically significant

to predict cardiotoxicity. An interesting finding is that there is a combined parameter: DLG and LV torsion, which was the best prognostic marker of cardiotoxicity; this parameter provides information on subendocardic (DLG) and subepicardial (torsion) function, therefore this most sensitive measure of early myocardial changes should be evaluated in the future. Integrated biomarkers and cardiac imaging appears as a promising approach to precisely detect and predict cardiotoxicity. After chemotherapy regimens are completed, there are limited recommendations on appropriate follow-up in these patients. However, we must make several considerations, the first one that specifically the cardiotoxicity by anthracyclines can be detected several years after having concluded the therapy. There are 9 published studies of cases and controls that evaluated subclinical myocardial damage, approximately 436 patients, but none provided results with sufficient statistical significance, however in survivors treated with anthracyclines, a reduction in strain of 6.6 to 26% was demonstrated.<sup>28</sup>

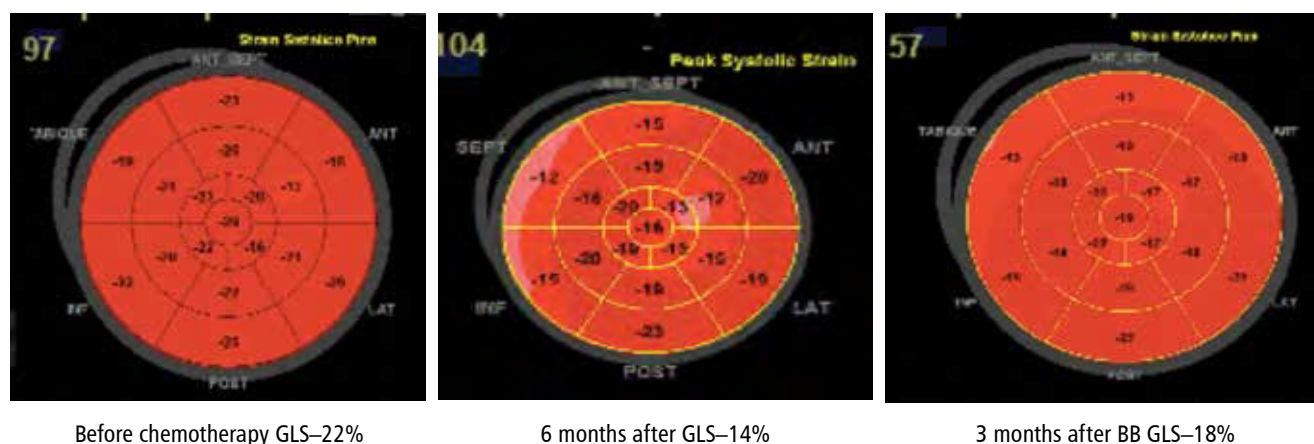
### *Biomarkers*

Cardinale and Sandri in 2003 evaluated a cohort of patients treated with high-dose chemotherapy, measuring cardiac troponin I (TropIc) before tx (basal) and at 0, 12, 24, 36 and 72 hours after the end of it. Those patients who had elevation of Troponin I (TropIc +) had a 12-month LVEF decrease of 18%, compared to the group that never had elevation of this marker (142 patients) where an average decrease of 2.5% was presented with a statistically significant difference between both. It is important to note that ventricular function gradually decreased over the 12 months that the study lasted, and that 20 of 57 patients who presented TropIc + did not show a decrease in LVEF, raising the question of whether the toxicity assessment cardiac echocardiography is a sufficiently sensitive method to distinguish those who will have a higher degree of cardiotoxicity. Early elevation (<72 h) of troponin I (TnI) (>0.08 ng/dL) has been demonstrated in up to one third of patients treated with anthracyclines.<sup>29</sup> The persistent elevation of the

TnI during cancer treatment identifies patients with worse cardiovascular prognosis, who could benefit from ACEI treatment to reduce the risk of CVD without the need to suspend or modify the antitumor treatment. The current consensus of Dr. Plana recommends the determination of troponins at baseline, at the end of 250 mg of anthracyclines or 3 months of trastuzumab, and from there after 50 mg of anthracycline or every 3 months of trastuzumab.<sup>15</sup>

### *Subclinical LV Dysfunction with Global Longitudinal Strain guide to therapy*

GLS-guided heart failure therapy is less studied. A small, observational, non-randomized study enrolled 159 patients receiving anthracycline, trastuzumab or both. Fifty-two patients showed a decrease in GLS > -11% at 6 months after baseline evaluation.<sup>30,31</sup> Of 52 patients, 24 were treated with beta-blockers and 28 with placebo. After 6 months of treatment, GLS and LVEF significantly improved in the beta-blockers group, but not in the placebo group. The Strain Surveillance During Chemotherapy for Improving Cardiovascular Outcomes (SUCCOUR) study will give some answers. Indeed, in this study in progress, patients with a relative reduction of GLS by  $\geq 12\%$  are treated with cardioprotective therapy. According to the position paper of the Working Group on Cardio-oncology of the European Society of Cardiology, ACE inhibitors and beta-blockers are recommended in patients with asymptomatic cardiac dysfunction to prevent the development of symptomatic heart failure or further dysfunction. This recommendation is based on an observational study, enrolling 2625 patients treated with anthracycline. In the population developing cardiotoxicity (n=226; defined by a decrease of 10 percentage points to a value <50%), ACE inhibitors +/- beta-blockers were initiated early. Among these 226 patients, 82% recovered from cardiotoxicity at least partially with heart failure therapy. Nevertheless, those who failed to improve LVEF had a significantly higher risk of major cardiovascular events. These findings support the fact that early detection of subclinical cardiac dysfunction by LVEF decrease could lead to an early start of heart failure therapy, thus preventing cardiac outcomes (Figure 1).<sup>32-35</sup>



**Figure 1:** Illustrates a case with subclinical left ventricular dysfunction, and comparison GLS before and after cardioprotective therapy.

## CONCLUSION

The global longitudinal deformation can be a more reproducible early method of ventricular mechanics. Much still remains to be understood about the role of different imaging methods in the identification and management of cardiotoxicity by chemotherapy. The long-term effect of those early changes we are now finding is still not understood, but certainly the prognostic significance of those abnormalities in cancer survivors or those who have received radiation therapy should be clarified to allow for early interventions that can change the natural history of cardiotoxicity. The primary objective for the cardio-oncological group is through these advanced techniques to identify patients and identify those at high risk for cardiotoxicity.

## REFERENCES

1. World Health Organization. Global Status Report on Noncommunicable Diseases 2014. 2015.
2. Knaul FM, Nigenda G, Lozano R, et al. Breast cancer in Mexico: a pressing priority. *Reprod Health Matters* 2008; 16: 113-123.
3. Bray F, Piñeros M. Cancer patterns, trends and projections in Latin America and the Caribbean: a global context. *Salud Pública Mex.* 2016; 58: 104-117.
4. Reynoso-Noverón N, Villarreal-Garza C, Soto-Pérez-de-Celis E et al. Clinical and epidemiological profile of breast cancer in Mexico: results of the Seguro Popular. *J Glob Oncol.* 2017; 3: 757-764.
5. Global Burden of Disease Cancer Collaboration, Fitzmaurice C, Allen C et al. Global, Regional, and National Cancer Incidence, mortality, years of life lost, years lived with disability, and disability-adjusted life-years for 32 Cancer Groups, 1990 to 2015: a systematic analysis for the Global Burden of Disease Study. *JAMA Oncol.* 2017; 3: 524-548.
6. Mavrogeni SI, Sfendouraki E, Markousis-Mavrogenis G et al. Cardio-oncology, the myth of Sisyphus, and cardiovascular disease in breast cancer survivors. *Heart Fail Rev.* Epub ahead of print el 27 de mayo de 2019. doi: 10.1007/s10741-019-09805-1.
7. Ewer MS, Ewer SM. Cardiotoxicity of anticancer treatments. *Nat Rev Cardiol.* 2015; 12: 547-558.
8. Yeh ETH, Chang HM. Oncocardiology-past, present, and future: a review. *JAMA Cardiol.* 2016; 1: 1066-1072.
9. Cuomo A, Rodolico A, Galdieri A et al. Heart failure and cancer: mechanisms of old and new cardiotoxic drugs in cancer patients. *Card Fail Rev.* 2019; 5: 112-118.
10. Tocchetti CG, Leppo MK, Bedja D et al. Cardiac over-expression of creatine kinase differentially affects cardiomyocyte function in ischemic and non-ischemic heart failure. *Biophys J.* 2016; 110: 599a.
11. Truitt R, Mu A, Corbin EA et al. Increased Afterload augments sunitinib-induced cardiotoxicity in an engineered cardiac microtissue model. *JACC Basic Transl Sci.* 2018; 3: 265-276.
12. Varricchi G, Marone G, Mercurio V et al. Immune checkpoint inhibitors and cardiac toxicity: an emerging issue. *Curr Med Chem.* 2018; 25: 1327-1339.
13. Love VA, Grabie N, Duramad P et al. CTLA-4 ablation and interleukin-12 driven differentiation synergistically augment cardiac pathogenicity of cytotoxic T lymphocytes. *Circ Res.* 2007; 101: 248-257.
14. López-Fernández T, Martín-García A et al. Cardio-Onco-Hematología en la práctica clínica. Documento de consenso y recomendaciones. *Rev Esp Cardiol.* 2017; 70: 474-486.
15. Plana JC, Galderisi M, Barac A et al. Expert consensus for multimodality imaging evaluation of adult patients during and after cancer therapy: a report from the ASE and the EACVI. *J Am Soc Echocardiogr.* 2014; 27: 911-939.
16. Virani SA, Dent S, Brezden-Masley C et al. Canadian cardiovascular society guide-lines for evaluation and

- management of cardiovascular complications of cancer therapy. *Can J Cardiol*. 2016; 32: 831-841.
17. Armenian SH, Lacchetti C, Barac A et al. Prevention and monitoring of cardiac dysfunction in survivors of adult cancers: American Society of Clinical Oncology Clinical Practice Guideline. *J Clin Oncol*. 2017; 35: 893-911.
  18. Zamorano JL, Lancellotti P, Rodriguez Muñoz D et al. 2016 ESC Position paper on cancer treatments and cardiovascular toxicity developed under the auspices of the ESC Committee for Practice Guidelines: The Task Force for cancer treatments and cardiovascular toxicity of the European Society of Cardiology (ESC). *Eur Heart J*. 2016; 37: 2768-2801.
  19. VonHoff DD, Layard MW, Basa P et al. Risk factors for doxorubicin-induced congestive heart failure. *Ann Intern Med*. 1979; 91: 710-717.
  20. Peng X, Chen B, Lim CC, Sawyer DB. The cardiotoxicology of anthracycline chemotherapeutics: translating molecular mechanism into preventative medicine. *Mol Interv*. 2005; 5 (3): 163-171.
  21. Ong G, Brezden-Masley C, Dhir V et al. Myocardial strain imaging by cardiac magnetic resonance for detection of subclinical myocardial dysfunction in breast cancer patients receiving trastuzumab and chemotherapy. *Int J Cardiol*. 2018; 261: 228-233.
  22. Neilan TG, Coelho-Filho OR, Shah RV et al. Myocardial extracellular volume by cardiac magnetic resonance imaging in patients treated with anthracycline-based chemotherapy. *Am J Cardiol*. 2013; 111: 717-722.
  23. Jordan JH, Todd RM, Vasu S, Hundley WG. Cardiovascular magnetic resonance in the oncology patient. *JACC Cardiovasc Imaging*. 2018; 11: 1150-1172.
  24. Tsai HR, Gjesdal O, Wethal T et al. Left ventricular function assessed by two-dimensional speckle tracking echocardiography in long-term survivors of Hodgkin's lymphoma treated by mediastinal radiotherapy with or without anthracycline therapy. *Am J Cardiol*. 2011; 107: 472-477.
  25. Yingchoncharoen T, Agarwal S, Popovic ZB, Marwick TH. Normal ranges of left ventricular strain: a meta-analysis. *J Am Soc Echocardiogr*. 2013; 26: 185-191.
  26. Mantovani G, Madeddu C, Cadeddu C et al. Persistence, up to 18 months of follow-up, of epirubicin-induced myocardial dysfunction detected early by serial tissue Doppler echocardiography: correlation with inflammatory and oxidative stress markers. *Oncologist*. 2008; 13: 1296-1305.
  27. Hequet O, Le QH, Moullet I et al. Subclinical late cardiomyopathy after doxorubicin therapy for lymphoma in adults. *J Clin Oncol*. 2004; 22: 1864-1871.
  28. Erven K, Florian A, Slagmolen P et al. Subclinical cardiotoxicity detected by strain rate imaging up to 14 months after breast radiation therapy. *Int J Radiat Oncol Biol Phys*. 2013; 85: 1172-1178.
  29. Fallah-Rad N, Walker JR, Wassef A et al. The utility of cardiac biomarkers, tissue velocity and strain imaging, and cardiac magnetic resonance imaging in predicting early left ventricular dysfunction in patients with human epidermal growth factor receptor II-positive breast cancer treated with adjuvant trastuzumab therapy. *J Am Coll Cardiol*. 2011; 57: 2263-2270.
  30. Saha SK, Kiotsekoglou A, Toole RS, Moggridge JC, Nichols KJ, Govind S et al. Value of two-dimensional speckle tracking and real time three-dimensional echocardiography for the identification of subclinical left ventricular dysfunction in patients referred for routine echocardiography. *Echocardiography*. 2012; 29: 588-597.
  31. Avi VM, Lang RM et al. Is Echocardiography reliable for monitoring the adverse cardiac effects of chemotherapy? *J Am Col Cardiol*. 2013; 61: 85-87.
  32. Negishi T, Thavendiranathan P, Negishi K et al. Rationale and design of the Strain Surveillance of Chemotherapy for Improving Cardiovascular Outcomes: the SUCCOUR Trial. *JACC Cardiovasc Imaging*. 2018; 11: 1098-1105.
  33. Yu W, Li SN, Chan GC, Ha SY, Wong SJ, Cheung YF. Transmural strain and rotation gradient in survivors of childhood cancers. *Eur Heart J Cardiovasc Imaging*. 2013; 14: 175-182.
  34. Saha S, Kiotsekoglou A, Rena S et al. Value of two-dimensional speckle tracking and real time three-dimensional echocardiography for the identification of subclinical left ventricular dysfunction in patients referred for routine echocardiography. *Echocardiography*. 2012; 29: 588-597.
  35. Bovelli D, Plataniotis G, Roila F, on behalf of the ESMO Guidelines Working Group. Cardiotoxicity of chemotherapeutic agents and radiotherapy-related heart disease: ESMO Clinical Practice Guidelines. *Ann Oncol*. 2010; 21 Suppl 5: 277-282.

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Sólo serán considerados los manuscritos inéditos (trabajos aún no publicados **EN EXTENSO**), los cuales no podrán ser sometidos a ninguna otra revista o medio de difusión durante el proceso de evaluación (desde su recepción hasta su dictamen). La propiedad de los manuscritos será transferida a la Sociedad Nacional de Ecocardiografía de México AC, por lo que no podrán ser publicados en otras fuentes (como revistas, libros o sitios de internet), ni completos o en partes, sin previo consentimiento por escrito del Editor.

El Comité Editorial decidirá cuáles manuscritos serán evaluados por árbitros expertos en el tema y no se admitirán los manuscritos presentados de manera inadecuada o incompleta. El dictamen del Comité para publicación es inapelable y podría ser: Aceptado, Aceptado con modificaciones, No aceptado.

### TIPOS DE ARTÍCULOS

Los artículos deberán enviarse a la revista **Cardiac Image Updated**, a través del editor en línea disponible en: <http://ciu.medigraphic.com> En este sitio, el Autor podrá informarse sobre el estado de su manuscrito en las fases del proceso: recepción, evaluación y dictamen.

Los artículos pueden ser de las siguientes categorías:

**Artículo original.** Puede ser investigación básica o clínica relacionada con imagen cardiovascular, ecocardiografía, tomografía cardíaca, resonancia magnética cardíaca, medicina nuclear cardíaca y tiene las siguientes características:

- a) **Título:** representativo de los hallazgos del estudio. Agregar un título corto para las páginas internas (es importante identificar el diseño metodológico del estudio: registro, observacional de asociación cruzada, casos y controles, cohorte, ensayo clínico, estudio de prueba diagnóstica o revisión sistemática).
- b) **Resumen estructurado:** debe incluir introducción, objetivo, material y métodos, resultados y conclusiones; con palabras clave y *key words*. Máximo 1,500 caracteres sin espacios. En español e inglés.
- c) **Introducción:** describe los estudios que permiten entender el objetivo del trabajo, mismo que se menciona al final de la introducción (no se escriben aparte los objetivos, la hipótesis ni los planteamientos).
- d) **Material y métodos:** parte importante que debe explicar con todo detalle cómo se desarrolló la investigación y, en especial, que sea reproducible (mencionar



- tipo de estudio, observacional o experimental).
- e) **Resultados:** en esta sección, de acuerdo con el diseño del estudio, deben presentarse todos los resultados o figuras (gráficas o imágenes), deben incluirse aparte, en las últimas páginas con pie de figura.
  - f) **Discusión:** con base en bibliografía actualizada que apoye los resultados. Las conclusiones se mencionan al final de esta sección.
  - g) **Referencias:** deberá seguir las especificaciones descritas más adelante.
  - h) **Número máximo de caracteres sin espacio:** 8,000 (no incluye resumen).

#### **Caso clínico o quirúrgico (1-3 casos) o serie de casos (más de tres casos clínicos)**

- a) **Título:** debe especificar si se trata de un caso clínico o una serie de casos clínicos.
- b) **Resumen estructurado:** debe incluir objetivo de la presentación, descripción del caso y conclusiones, con palabras clave y *key words*. Máximo 1,200 caracteres sin espacios. En español e inglés.
- c) **Introducción:** se trata la enfermedad o causa atribuible. Señalar el objetivo de la presentación del caso clínico.
- d) **Presentación del(los) caso(s) clínico(s):** descripción clínica, laboratorio y otros. Mencionar el tiempo en que se reunieron estos casos. Las figuras o cuadros van en hojas aparte.

- e) **Discusión:** se comentan las referencias bibliográficas más recientes o necesarias para entender la importancia o relevancia del caso clínico.
- f) **Referencias:** deberá seguir las especificaciones descritas más adelante.
- g) **Número máximo de caracteres sin espacio:** 5,000 (no incluye resumen).

**Artículo de revisión.** Se admitirán para evaluación aquéllos que, por invitación del Editor y a propuesta de los miembros del Comité o del Consejo, sean relevantes para la temática de la revista.

- a) **Título:** que especifique claramente el tema a tratar.
- b) **Resumen:** en español y en inglés, con palabras clave y *key words*. Máximo 1,200 caracteres sin espacios. En español e inglés.
- c) **Introducción y, si se consideran necesarios, subtítulos:** pueden iniciarse con el tema a tratar sin divisiones.
- d) **Referencias:** deberá seguir las especificaciones descritas más adelante.
- e) **Número máximo de caracteres sin espacio:** 8,000 (no incluye resumen).

**Cartas al Editor.** Esta sección es para documentos de interés social, novedades terapéuticas, imágenes cardíacas, noticias y cartas sobre Bioética, normativos o complementarios a uno de los artículos de investigación. No tiene un formato especial. Número máximo de caracteres sin espacio: 2,500.

Los requisitos se muestran en la **Lista de Verificación**. El formato se encuentra disponible en [www.medigraphic.com/pdfs/ciu/ciu-lista-verif.pdf](http://www.medigraphic.com/pdfs/ciu/ciu-lista-verif.pdf) (PDF). Los autores deberán descargarla e ir marcando (en el PDF interactivo) cada apartado una vez que éste haya sido cubierto durante la preparación del material para publicación.

Renombre el PDF y envíelo como un archivo adjunto a través del Editor en línea de la revista *Cardiac Image Updated*, disponible en: <http://ciu.medigraphic.com>

## Llega a México tecnología de punta para revolucionar el diagnóstico de padecimientos cardíacos

Según datos de la Asociación Nacional de Cardiólogos al Servicio de los Trabajadores del Estado (ANCISSTE), el 54% de las muertes en México se debe a enfermedades cardíacas.

El problema se agrava con el paso del tiempo ya que por la falta de información entre nuestra sociedad y la ausencia de un estilo de vida saludable; los padecimientos cardíacos se están presentando en edades más tempranas, afectando cada vez más a la población económicamente activa con múltiples factores de riesgo, con complicaciones y mayores costos en el tratamiento y manejo de las mismas. La detección tardía y el mal manejo de estas enfermedades, son responsables de diversas complicaciones que repercuten en la calidad de vida y la capacidad laboral de las personas

La compleja anatomía y los sofisticados mecanismos de función de las estructuras cardíacas en los pacientes con cardiopatías requieren que la aplicación de las técnicas ecocardiográficas como método diagnóstico cubran dos aspectos básicos: Morfología y función. La ecocardiografía bidimensional (Eco2D) y Doppler solía ser una herramienta diagnóstica útil disponible en México, y en muchos casos era suficiente para la evaluación morfológica y hemodinámica. Una de las limitaciones de la Eco2D radicaba en que se realiza en un solo plano, por lo que se requiere la obtención de imágenes en diferentes aproximaciones, lo que obliga a realizar una reconstrucción mental para su mejor entendimiento. Hasta hoy, en México existían diferentes tecnologías disponibles para tener un diagnóstico de estos padecimientos como la Eco2D. Sin embargo, los ecocardiógrafos han evolucionado drásticamente y los diagnósticos están a punto de evolucionar para siempre con visualizaciones tridimensionales (Eco3D) fotorrealistas que proporcionan un mayor detalle del estado del paciente y un mayor detalle de las estructuras anatómicas. Por su parte, Philips se ha posicionado como líder en ecocardiografía de muy alto desempeño para los desafíos actuales y por venir en la prevención, diagnóstico y tratamiento de las enfermedades cardiovasculares.

Al desarrollar transductores con tecnología de cristales de onda pura, logró obtener imágenes diagnósticas aún en pacientes técnicamente difíciles en la población con sobrepeso; Philips fue pionero en el desarrollo de transductores matriciales con alta densidad de cristales que permiten obtener imágenes clínicas bidimensionales de excepcional calidad diagnóstica y alta sensibilidad Doppler y estos transductores también generan imágenes tridimensionales en escala de grises y Doppler color de las estructuras cardíacas, proporcionando una visualización de excelente resolución de la anatomía estructural del corazón y un apoyo sin precedentes en las salas de intervencionismo con imágenes tridimensionales transesofágicas.

El ecocardiógrafo Philips EPIQ CVx recientemente lanzado al mercado, mejora notablemente la atención en las enfermedades cardiovasculares gracias a las diferentes tecnologías implementadas como son: *interface de usuario más eficiente* al permitir personalizar los controles del equipo. Cuenta con *monitor OLED* para una excepcional calidad de imagen aún en entornos no convencionales como son en quirófano y la sala de intervencionismo; *imagen fotorrealista tridimensional con fuente de luz ajustable* que proporciona imagen tridimensional en tiempo real con tejidos iluminados para una apreciación más detallada y con sentido de la profundidad de las estructuras; *innovadoras herramientas y transductores en pediatría* para un mejor diagnóstico de las complejas estructuras congénitas. Está equipado con *HeartModel Dinámico*, una sofisticada herramienta automatizada de Inteligencia Anatómica e Inteligencia artificial, que permite cuantificar la función del lado izquierdo del corazón de manera rápida, robusta y reproducible a partir de una imagen volumétrica tridimensional. Además, el ecocardiógrafo Philips EPIQ CVx incorpora ahora las *tecnologías automatizadas de TomTec* como son: *autoSTRAIN* para el ventrículo izquierdo, ventrículo derecho y aurícula izquierda, función del Ventrículo derecho a partir del HeartModel Dinámico y valoración de la válvula mitral; llevando la prevención, diagnóstico y tratamiento de las enfermedades cardiovasculares a un estado del arte en beneficio de las personas.



*"Este equipo representa la evolución de los diagnósticos cardíacos en México y es el resultado de más de 15 años de investigación de Philips. Escuchamos las necesidades de miles de médicos alrededor del mundo y adaptamos toda esa información para ofrecer soluciones enfocadas a los principales padecimientos que afectan a la humanidad hoy en día como es el caso del nuevo ecocardiógrafo Philips EPIQ CVx, diseñado para los desafíos de la ecocardiografía moderna. Hoy Philips es una pieza clave en la vida de millones de personas, desde la prevención de enfermedades, su diagnóstico certero, tratamiento y cuidado en casa. La mirada está puesta no sólo en seguir desarrollando la mejor tecnología para los expertos de la salud, sino también en hacerlos cada vez más interconectados para fortalecer la toma de decisiones y mejorar la vida de más de 3 billones de personas para el año 2025."* - destacó Rodrigo Fernández, líder de la División de Ultrasonido para Philips en México.

Philips EPIQ CVx, el ecocardiógrafo que se adelantó al futuro para mejorar la salud en el presente.

Obtenga más información sobre Philips EPIQ CVx y sobre cómo realizar estudios de ecocardiografía de última generación escribiendo a [miguel.corona@philips.com](mailto:miguel.corona@philips.com)

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