With the advances in the development of devices for closure of various congenital heart defects, such as persistent ductus arteriosus and ventricular or atrial septal defects, it is surprising that transcatheter APW closure still plays a very small role. This approach has several attractive advantages: extracorporeal circulation is avoided during the procedure and the postoperative hospital stay is shorter. However, the excellent outcome achieved with surgery and the technical complexity of transcatheter closure are the 2 main reasons why most centers prefer surgical treatment. Regardless of these considerations, the candidates for transcatheter closure should have relatively small defects located at a point equidistant between the bifurcation of the pulmonary artery and the semilunar valves, and far from the left coronary artery ostium and the aortic valve; that is, type I defects according to the classification of Mori et al.[6] It is important to appropriately characterize the defect through the use of several angiographic views or even measurement balloons to precisely determine the dimensions of the window.[3–5] It is also a challenge to choose the appropriate device, and to date, there is no consensus on the choice of an optimal device for APW closure. Ductus arteriosus occluders tend to protrude toward the main pulmonary artery and carry a risk of obstructing the vessel, whereas atrial septal defect occluders are bulky for this purpose and may injure or obstruct the semilunar valves or left coronary ostium.[5] Trehan et al.[3] considered that the perimembranous ventricular septal defect occluder device may be the most appropriate. It has a relatively flat profile (waist diameter, 1.5 mm), which could cause less obstruction. Furthermore, because the discs are asymmetrical, they can be used to close an APW with a relative deficiency of one of the borders.

In selected patients, percutaneous APW closure can be considered a viable, effective procedure. Nonetheless, the potential risks should be considered, such as device embolization and residual shunts. Because of its excellent results, surgical management remains the method of choice for treating these defects.

Aldo Campos-Quintero,[a] José Antonio García-Montes,[a] Carlos Zabal-Cerdeira,[a] Jorge Luis Cervantes-Salazar,[b] Juan Calderón-Colmenero,[a] and Juan Pablo Sandoval[a,*]

[a]*Departamento de Cardiología Intervencionista en Cardiopatías Congénitas, Instituto Nacional de Cardiología Ignacio Chávez, Mexico City, Mexico*
[b]*Departamento de Cirugía Cardiovascular en Cardiopatías Congénitas, Instituto Nacional de Cardiología Ignacio Chávez, Mexico City, Mexico*

* Corresponding author:
E-mail address: juanpablo.sandoval@cardiologia.org.mx (J.P. Sandoval).

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Tako-tsubo Cardiomyopathy Complicated With Cardiac Tamponade and Cardiogenic Shock

Síndrome de tako-tsubo *complicado con taponamiento cardiaco y* shock *cardiogénico*

To the Editor,

Although clinical progress is favorable in most patients with tako-tsubo syndrome with resolution of ventricular dysfunction, complications leading to shock sometimes occur.[1]

We present the case of an 83-year-old woman, with no medical history of interest, who consulted for a 12-hour history of oppressive central chest pain with no clear triggering factor. An electrocardiogram showed ST-segment elevation in V_3-V_6, DII, DIII, and aVF (Figure 1A). An ST-segment elevation acute coronary syndrome was suspected, and a loading dose of dual antiplatelet therapy was administered (300 mg aspirin and 600 mg clopidogrel). Emergent coronary angiography showed no significant lesions in the epicardial arteries (Figure 1C). The study was completed with ventriculography, which showed apical akinesia (Figure 1D), and transthoracic echocardiography, which depicted akinesia of the left ventricular middle and apical segments (Figure 1E). Left ventricular ejection fraction was 35%, with hypercontraction of the basal segments, and the mitral valve showed a systolic anterior movement (SAM), without significant flow acceleration in the left ventricular outflow tract (LVOT), and a mild (< 10 mm), circumferential pericardial effusion (PE).

The patient was hemodynamically stable, showed no evidence of heart failure, and had a minimal troponin I elevation (peak 0.823 ng/mL); nonetheless, at 24 hours she began to show hemodynamic deterioration. Transthoracic echocardiography detected an increase in the dynamic LVOT obstruction[2] and progression of the PE (18 mm in the right ventricular free wall) (Figure 2A), with no echocardiographic signs of cardiac tamponade. Based on these findings, fluid therapy was increased and phenylephrine and esmolol infusion was started. These measures led to a decrease in the dynamic LVOT obstruction and improvement of the patient's hemodynamic status. In light of the PE increase, cardiac computed tomography was performed, but there was no evidence of cardiac rupture. However, an aneurysmal dilatation of the left ventricular apical region was detected, with preserved myocardial thickness and a thrombus adhering to the inferoapical segment (Figure 2B).

In the following hours, the patient's clinical course was unfavorable, with the development of severe cardiogenic shock (hypotension, anuria, elevated lactate), worsening of the dynamic LVOT obstruction (Figure 2C), progression of PE (21 mm), and evidence of tamponade (partial collapse of the right chambers in diastole and a change in the transtricuspid flow > 50%).

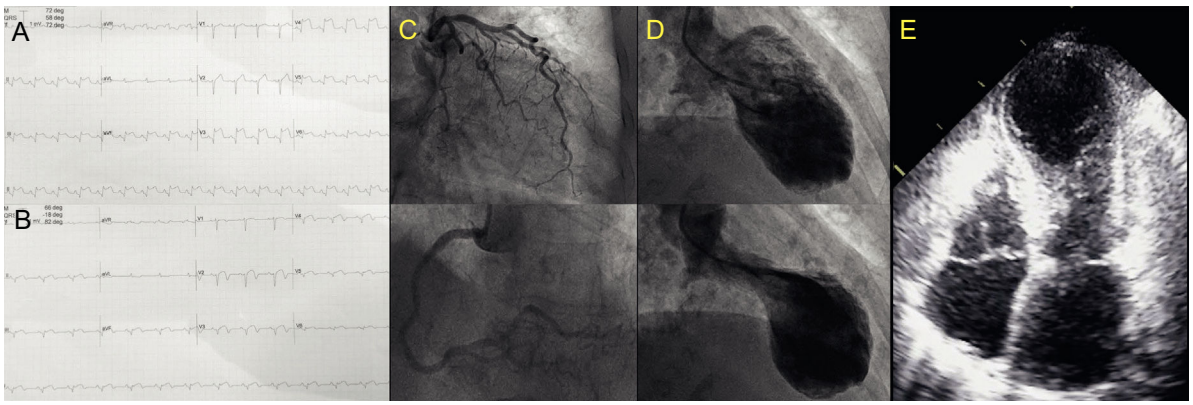

Figure 1. A. Electrocardiogram at admittance. B. Follow-up electrocardiogram. C. Coronary angiography. D. Ventriculography. E. Transthoracic echocardiography at admittance.

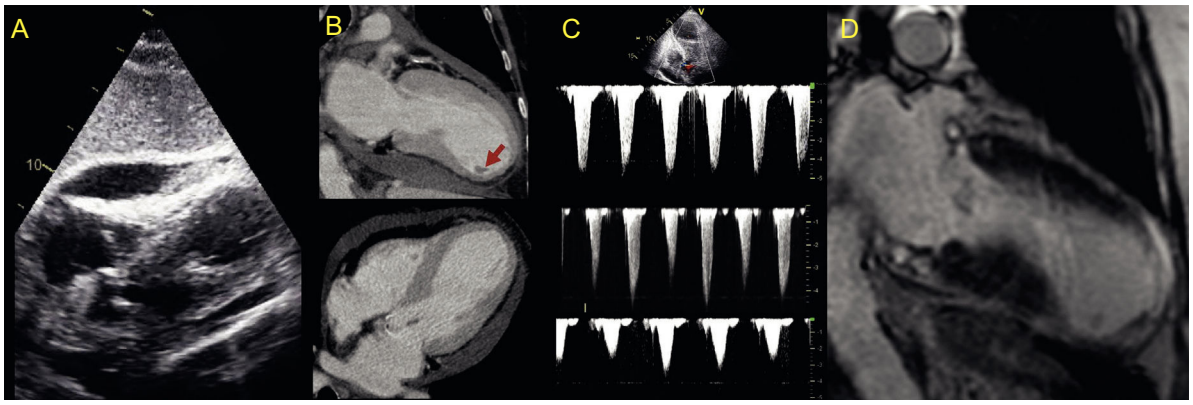

Figure 2. A. Pericardial effusion on transthoracic echocardiography. B. Cardiac computed tomography depicts a thrombus adhering to the inferoapical segment (arrow). C. Continuous Doppler signal in the left ventricular outflow tract. D. Cardiac magnetic resonance.

Pericardiocentesis was performed, yielding 350 mL of hematic fluid (hemoglobin, 9.9 g/dL). Because the increase in PE was gradual and cardiac computed tomography showed no evidence of cardiac rupture, we adopted an expectant attitude regarding the etiology of the hemopericardium. Nonetheless, aspirin administration was discontinued (opting for single antiplatelet treatment), as well as the enoxaparin 40 mg/d. A slight improvement in the patient's hemodynamic status was achieved following pericardiocentesis, but she remained in cardiogenic shock and consequently initiation of circulatory support was evaluated. Use of an Impella support device (Abiomed) was contraindicated because of the intraventricular thrombus, and extracorporeal membrane oxygenation was rejected due to the patient's advanced age. An intraaortic counterpulsation balloon was implanted and the amines were optimized to maintain mean blood pressure at $>$ 60 mmHg, but the tissue hypoperfusion and anuria persisted, requiring the start of continuous venovenous hemofiltration. The patient's condition slowly improved without the application of additional measures, and we were able to discontinue the counterpulsation balloon, hemofiltration, and intravenous drugs at 72 hours after the onset of the clinical situation. The patient's evaluation was completed with a magnetic resonance study, which showed no signs of myocardial necrosis on delayed enhancement sequences; we identified only a focal pericardial hypersignal in the anteroapical region (Figure 2D). After 10 days of hospitalization, the patient was discharged with preserved left ventricular systolic function. At the 3-month follow-up examination, she was in New York Heart Association functional class I, with preserved overall and segmental left ventricular systolic function and persistence of the electrocardiography changes (Figure 1B).

The development of PE is not unusual in tako-tsubo cardiomyopathy, but it rarely requires drainage. We found only one case of hemopericardium in the literature, and the authors suggested that inflammation and high doses of antithrombotic treatment may have had an impact on the genesis of the effusion.[3] This theory is supported by the clinical course of our patient and the magnetic resonance findings.[4]. In addition, our patient is the first showing dynamic LVOT obstruction, cardiac tamponade, and intraventricular thrombosis simultaneously. The reversibility of the syndrome, as well as the potentially deleterious effect of inotropic amines and vasopressors, would support prompt use of short-term circulatory assistance if there is hemodynamic instability. Of note, the mean age at the onset of tako-tsubo cardiomyopathy (70 $\pm$ 12.5 years in the RETAKO national registry)[5] should lead to a reconsideration of circulatory assistance protocols, which often exclude patients of advanced age.

J. Nicolás López Canoa,[a,*] Alfredo Redondo Diéguez,[a] Anaberta Bermúdez Naveira,[b] Pedro Rigueiro Veloso,[a] J. María García Acuña,[a] and J. Ramón Gónzalez Juanatey[a]

[a]*Servicio de Cardiología y Unidad Coronaria, Hospital Clínico Universitario de Santiago de Compostela, Santiago de Compostela, A Coruña, Spain*

[b]Servicio de Radiodiagnóstico, Hospital Clínico Universitario de Santiago de Compostela, Santiago de Compostela, A Coruña, Spain

* Corresponding author:
E-mail address: jnicolas.lopez.canoa@gmail.com (J.N. López Canoa).

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Clinical Acceptance of the Universal Definition of Myocardial Infarction

Aceptación clínica de la definición universal del infarto de miocardio

To the Editor,

In the emergency department, troponin determination is a useful test to differentiate between non–ST-segment myocardial infarction (MI) and unstable angina. Acute and chronic myocardial lesions due to a large spectrum of cardiac and noncardiac causes are recognizable in clinical practice.[1] In 2007, the universal definition of MI established the classification of patients according to the etiology of the condition.[2] Since then, the term type 2 MI has been used to describe clinical conditions associated with an ischemic myocardial lesion in the absence of complicated atheromatous plaques. Although several studies have reported higher mortality rates in patients with type 2 MI than in those with type 1, discrepancies remain regarding this prognosis, possibly because of the different diagnostic criteria used.[3–5] Nonetheless, there are no studies investigating the degree of acceptance of this classification or the extension of its use in clinical practice. Our aim was to evaluate the concordance between diagnosis associated with a

Table 1
Patients' Baseline Clinical Characteristics of the Patients According to the Department Issuing the Discharge Report

	Cardiology (n = 119)	Internal medicine (n = 105)	Others (n = 125)	Emergency (n = 303)	*P*
Age, y	75 [63-81]	84 [75-88]	72 [59-81]	81 [72-85]	< .001
Men	73 (61.34)	52 (49.52)	76 (60.80)	159 (52.48)	.128
Myocardial infarction	29 (24.37)	21 (20.00)	30 (24.00)	80 (26.40)	.624
Heart failure	19 (15.97)	21 (20.00)	16 (12.80)	63 (20.79)	.218
Stroke or TIA	18 (15.13)	19 (18.10)	11 (8.80)	14 (15.51)	.200
COPD	23 (19.33)	38 (36.19)	24 (19.20)	94 (31.02)	.003
Diabetes	41 (34.45)	37 (35.24)	42 (33.60)	118 (38.94)	.677
Hypertension	91 (76.47)	80 (76.19)	86 (68.80)	239 (78.88)	.175
Chronic kidney disease	27 (22.69)	17 (16.19)	34 (27.20)	71 (23.43)	.257
Charlson index	2 [1-3]	2 [1-4]	2 [0-4]	2 [1-4]	.255
Symptoms					
Chest pain	27 (22.69)	17 (16.19)	15 (12.00)	97 (32.01)	< .001
Dyspnea	44 (36.97)	65 (61.90)	30 (24.00)	103 (33.99)	< .001
Syncope	25 (21.01)	4 (3.81)	13 (10.40)	17 (5.61)	< .001
Others	30 (25.21)	29 (27.62)	72 (57.60)	127 (41.91)	< .001
Electrocardiogram[*]					
Atrial fibrillation	36 (31.30)	34 (33.66)	21 (19.09)	114 (40.43)	.001
Vital signs					
HR, bpm	90 [67-117]	100 [81-112]	87 [73-109]	87.5 [69-113]	.126
SAP, mmHg	134 [119-159]	129 [116-148]	134 [110-159]	134 [119-156]	.368
SaO_2, %	96 [93-98]	93 [89-96]	97 [92-99]	97 [94-99]	< .001
Analytical determinations					
eGFR, mL/min/1.73 m^2	58.6 [40.2-78.0]	59.3 [39.0-80.2]	45.8 [19.8-82.3]	53.7 [41.0-74.1]	.021
Hemoglobin, g/L	130 [109-140]	123 [110-134]	123 [98-139]	124 [112-140]	.282
TnI maximum, ng/mL	0.25 [0.08-1.09]	0.12 [0.06-0.46]	0.14 [0.08-0.67]	0.09 [0.06-0.17]	< .001

COPD chronic obstructive pulmonary disease; eGFR, estimated glomerular filtration rate; HR, heart rate; SAP, systolic arterial pressure; SaO_2, arterial oxygen saturation; TIA, transient ischemic attack; TnI, troponin I
The data are expressed as No. (%) or median [interquartile range].
[*] Electrocardiography data available for 115 patients admitted in cardiology, 101 admitted in internal medicine, 110 admitted in other departments, and 282 who were not admitted.