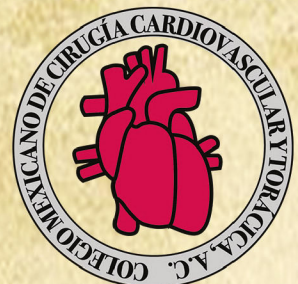


ISSN 2954-3320

CIRUGÍA CARDIACA *EN* MÉXICO

Volume 11 - Issue 3 - July - September 2026

*Official Journal of the
Sociedad Mexicana de Cirugía Cardíaca, A.C.
Colegio Mexicano de Cirugía Cardiovascular y Torácica, A.C.*



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CIRUGÍA CARDIACA EN MÉXICO

Official Journal of the Sociedad Mexicana de Cirugía Cardíaca, A.C.
and the Colegio Mexicano de Cirugía Cardiovascular y Torácica, A.C.



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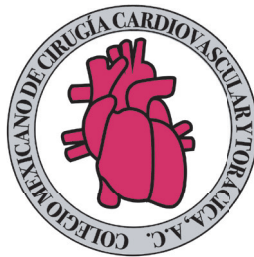
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Prosthetic valve endocarditis: a complex and lethal disease

Endocarditis valvular protésica: una enfermedad compleja y letal

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Keywords: cardiac surgery, complications, clinical outcomes, infective endocarditis, prosthetic heart valves, prosthetic valve endocarditis, reoperation.

Palabras clave: cirugía cardíaca, complicaciones, resultados, endocarditis infecciosa, válvulas cardíacas protésicas, endocarditis valvular protésica, reintervención quirúrgica.

There is something that needs to be said from the outset, without rhetorical embellishment: if an institutional series of prosthetic valve endocarditis doesn't raise concerns, it's probably being misinterpreted. Because this condition doesn't lend itself to complacent interpretations. It is, in essence, a challenge for medicine, as it implies a failure in the treatment, the device, and, sometimes, the healthcare system as a whole.

The work presented by Cueva-Tuttillo et al.¹ in this issue of *Cirugía Cardíaca en México* has a virtue that is not always recognized. It does not attempt to soften or minimize the problem, but rather presents it as it is: a serious and lethal condition. An incidence of 2.9% in a contemporary surgical cohort, with a predominance of mechanical prostheses, a virtually equal distribution between early and late endocarditis, and—above all—a mortality rate of 36.8%, remind us that we continue to operate in the realm of high lethality, not of a treatable complication.

Indeed, prosthetic valve endocarditis (PVE) constitutes the most severe form of infective endocarditis (IE), affecting between 1 and 6% of patients with prosthetic valves.²

The importance of the study lies not only in the numbers, but also in how it helps us understand the disease. PVE is not

just an infection of the valve itself, but a disease that affects the surrounding tissue, where surgery was intended to repair it. PVE accounts for 20 to 30% of all IE cases.³ This is why we see complications such as dehiscence, abscesses, and fistulas; these are not just complications, but rather the way the disease manifests. When the infection reaches this level, the problem ceases to be microbiological and becomes structural.

This study shows that surgery for prosthetic valve endocarditis is not an option; it is a necessity. When the infection spreads around the valve, surgery is unavoidable. It is not a matter of being “aggressive” or “conservative”; rather, it is about confronting the severity of the disease. The idea of doing something less invasive does not reflect the reality of the situation. Surgery is the response to the aggressiveness of the infection. The 2023 European Society of Cardiology guidelines for the management of endocarditis present surgery as a Recommendation Class 1, Level of Evidence C for early PVE, by means of new prosthetic replacement and debridement of the infected site.⁴

Another point that deserves attention is the microbiological profile. The predominance of staphylococci (73.6%) is neither a minor nor anecdotal finding. In line with the findings of

How to cite: García-Villarreal OA. Prosthetic valve endocarditis: a complex and lethal disease. *Cir Card Mex.* 2026; 11 (3): 81-82. <https://dx.doi.org/10.35366/123480>

© 2026 by the Sociedad Mexicana de Cirugía Cardíaca, A.C.

Received: 09-05-2026. Accepted: 30-05-2026.

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Berisha et al.,⁵ patients with mechanical valves are more likely to suffer from PVE caused by *S. aureus*, compared to bioprosthetic valves (36% vs. 17%, $p < 0.001$). This is, in fact, a reflection of an epidemiological transition that should no longer surprise us: prosthetic valve endocarditis is increasingly an infection associated with the healthcare environment, invasive manipulation, and the device itself.³ *Staphylococcus* not only colonizes; it invades, adheres, and destroys. And it does so with an efficiency that turns every hour of delay into a lost opportunity.

This is where the narrative of “early surgery” takes on a deeper meaning. It is not simply about operating earlier; it is about understanding that time in prosthetic valve endocarditis is not linear. There are anatomical points of no return. The abscess that is contained today may become a fistula tomorrow. Partial dehiscence is the unstable prosthesis of the following week. The window for intervention is not measured in calendar days, but in tissue progression.

The series also identifies classic risk factors—advanced age, diabetes, history of endocarditis. Factors associated with worse outcomes include advanced age, diabetes, healthcare-associated infections, and early PVE.⁶ Notwithstanding, it would be a mistake to interpret these as mere adjustment variables. Rather, they are markers of biological vulnerability. In these patients, endocarditis not only occurs more frequently but also progresses more destructively. This necessitates a more nuanced interpretation: not all patients with prosthetic valves have the same threshold for monitoring, nor should they have the same threshold for intervention.

However, the data point that might lead to an optimistic interpretation—a 63.1% survival rate—deserves to be examined critically. Is this high? It depends on the frame of reference. In absolute terms, it remains a pathology with unacceptably high mortality. It is a well-known fact that PVE has a high in-hospital mortality rate, between 20 and 40%.⁶

In addition, compared to mechanical prostheses, the risk of prosthetic valve endocarditis has typically been reported to be higher in patients with bioprostheses, both in unadjusted analysis (hazard ratio [HR], 1.51; 95%CI, 1.31-1.74) and in adjusted multivariable analysis (HR, 1.54; 95%CI, 1.29-1.83).⁶ Conversely, in this series by Cueva-Tuttillo et al., it was reported that the majority of prosthetic valve endocarditis (PVE) cases developed on mechanical prostheses (84.2%, $n = 16$), while only 15.8% ($n = 3$) were observed on previously placed bioprostheses.¹ This could be due to differences in the study population (e.g., age, comorbidities), surgical and local management practices, and patient selection for prosthesis type (mechanical vs. bioprosthesis). In other words, mechanical prostheses were more common in the studied population, which could explain the higher number of PVE in this group.

And that brings us to the core editorial point: the validation of an aggressive and early surgical strategy stems not from its success, but from the absence of viable alternatives.

Prosthetic valve endocarditis left untreated, in the presence of perivalvular invasion, has a predictable course. Surgery, on the other hand, introduces uncertainty... but also possibility. It is in this trade-off—certainty of deterioration versus uncertainty with the possibility of survival—that intervention is justified.⁴

This article, although descriptive and with a limited sample size, fulfills a more relevant function than simply “reporting experience”: it establishes a clinical position. And it does so from the perspective of a Mexican center, with its own timelines, resources, and microbiology. That matters. Because one of the most frequent distortions in cardiovascular surgery is the uncritical adoption of results from contexts we do not share.

Finally, the study conveys a lesson that transcends its own findings: PVE is, in many cases, the culmination of a chain of events that began long before the operating room. Suboptimal metabolic control, repeated exposure to healthcare, fragmented clinical follow-up. Thinking of it solely as a surgical problem is to be late to the conversation.

But when the patient is already in the operating room, when the valve ring is destroyed and the infection has spread beyond its boundaries, surgery ceases to be just one option among several. It is the only possible course of action.

And this study makes that clear, with numbers that are not reassuring, but do offer guidance: in PVE, early surgery is not an aggressive approach; it is simply a refusal to be complacent in the face of the disease.

REFERENCES

1. Cueva-Tuttillo R, Flores-Calderón O, Salazar-Hernández I, et al. Prosthetic valve endocarditis: surgical complexity, microbiological profile, and intermediate-term survival outcomes. *Cir Card Mex*. 2026 (ahead of print).
2. Vongpatanasin W, Hillis LD, Lange RA. Prosthetic heart valves. *N Engl J Med*. 1996;335(6):407-416. doi: 10.1056/NEJM199608083350607.
3. Selton-Suty C, Célard M, Le Moing V, et al; AEPEI Study Group. Preeminence of *Staphylococcus aureus* in infective endocarditis: a 1-year population-based survey. *Clin Infect Dis*. 2012;54(9):1230-1239. doi: 10.1093/cid/cis199.
4. Delgado V, Ajmone Marsan N, de Waha S, et al; ESC Scientific Document Group. 2023 ESC Guidelines for the management of endocarditis. *Eur Heart J*. 2023;44(39):3948-4042. doi: 10.1093/eurheartj/ehad193.
5. Luciani N, Mossuto E, Ricci D, et al. Prosthetic valve endocarditis: predictors of early outcome of surgical therapy. A multicentric study. *Eur J Cardiothorac Surg*. 2017;52(4):768-774. doi: 10.1093/ejcts/ezx169.
6. Glaser N, Jackson V, Holzmann MJ, Franco-Cereceda A, Sartipy U. Prosthetic valve endocarditis after surgical aortic valve replacement. *Circulation*. 2017;136(3):329-331. doi: 10.1161/CIRCULATIONAHA.117.028783.

Funding: none.

Disclosure: the author has no conflict of interest to disclose.

Prosthetic valve endocarditis: surgical complexity, microbiological profile, and medium-term survival outcomes

Endocarditis valvular protésica: complejidad quirúrgica, perfil microbiológico y resultados de sobrevida a mediano plazo

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ABSTRACT

Introduction: prosthetic valve endocarditis is the most severe form of endocarditis. Due to its high complexity, it is imperative to analyze clinical experience and outcomes, as well as the local microbiological profile and survival rates using current surgical techniques. **Objective:** to evaluate the surgical management of prosthetic valve endocarditis and its clinical outcomes, incidence, microbiological profile, clinical characteristics, echocardiographic findings, surgical techniques employed, and short- and medium-term survival of patients operated on at our institution during the period from January 1, 2022, to December 31, 2024. **Material:** a retrospective, observational, and descriptive study. It included all patients undergoing cardiac surgery at the Hospital during the specified period ($n = 655$). Categorical variables are expressed as absolute frequencies and percentages. Survival was estimated using the Kaplan-Meier method. **Results:** incidence was 2.9%, with 19 cases of prosthetic endocarditis. Mechanical prosthetic valves were the most affected structure, with 84.2% of cases. Early endocarditis accounted for 52.6%, and late endocarditis for 47.4%. Risk factors identified included diabetes mellitus, advanced age, and a history of native valve endocarditis. Staphylococci were isolated in 73.6% of cases. Echocardiographic findings included

RESUMEN

Introducción: la endocarditis de válvula protésica es la forma más grave de endocarditis. Por su alta complejidad es imperativo analizar la experiencia y los resultados clínicos, así como el perfil microbiológico local y tasas de sobrevida con las técnicas quirúrgicas actuales. **Objetivo:** evaluar el manejo quirúrgico de la endocarditis de válvula protésica y sus resultados clínicos, incidencia, perfil microbiológico, características clínicas, hallazgos ecocardiográficos, las técnicas quirúrgicas empleadas y sobrevida a corto y mediano plazo de los pacientes operados en nuestra institución durante el periodo 01 de enero 2022 al 31 de diciembre 2024. **Material:** estudio retrospectivo, observacional y descriptivo. Incluyó a todos los pacientes sometidos a cirugía cardíaca en el Hospital durante el periodo señalado ($n = 655$). Las variables categóricas se expresan en frecuencias absolutas y porcentajes. La sobrevida se estimó por Kaplan-Meier. **Resultados:** la incidencia fue del 2.9%, con 19 casos de endocarditis protésica. La prótesis mecánica fue la más afectada, con 84.2% de los casos. El 52.6% fueron endocarditis temprana y 47.4% tardía. Los factores de riesgo detectados fueron diabetes mellitus, edad avanzada y antecedente de endocarditis de válvula nativa. Se aisló estafilococos en 73.6%. Hubo hallazgos ecocardiográficos como dehiscencia de prótesis, abscesos y fistulas.

How to cite: Cueva-Tutillo R, Flores-Calderón O, Salazar-Hernández I, Ferreyro-Espinosa K, Ramírez-Castañeda S. Prosthetic valve endocarditis: surgical complexity, microbiological profile, and medium-term survival outcomes. Cir Card Mex. 2026; 11 (3): 83-89. <https://dx.doi.org/10.35366/123481>

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Received: 27-11-2025. Accepted: 04/12/2025.

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prosthesis dehiscence, abscesses, and fistulas. Mortality was 36.8%, and survival was 63.1%. **Conclusions:** prosthetic valve endocarditis treated surgically at our center represents one of the most lethal pathologies in cardiovascular surgery. Perivalvular invasion (dehiscence, abscesses, and fistulas) constitutes a primary surgical indication. The high survival rate validates the strategy of aggressive and early surgical intervention.

Keywords: cardiac surgical procedures, complications, bacterial endocarditis, endocarditis, prosthetic valve, heart valve prosthesis, infections, survival rate.

Abbreviations:

IE = infective endocarditis

PVE = prosthetic valve endocarditis

Infective endocarditis (IE) is a cardiovascular pathology with significant morbidity and mortality. Within its clinical spectrum, prosthetic valve endocarditis (PVE) represents the most severe and challenging form of the disease, constituting a major public health problem in the context of valvular heart surgery.¹⁻³

PVE occurs in 1 to 6% of patients with a valvular implant, with an annual incidence of 0.3 to 1.2% per patient-year. In various international registries (such as the Euro Heart Survey and the ICE-PCS study), PVE is responsible for 20 to 30% of all cases of infective endocarditis. PVE is classified as early or late based on the time elapsed since the valve replacement surgery, being defined as less than or equal to 12 months for early PVE and greater than 12 months for late PVE. This distinction is crucial due to significant differences in microbiological profiles and pathogenesis.³⁻⁶

The etiological profile of PVE varies according to temporality. Early PVE, which develops in the perioperative period, is associated with nosocomial infections and is primarily caused by *Staphylococcus aureus*, *Staphylococcus epidermidis*, or Gram-negative pathogens and fungi. These microorganisms have a high affinity for implanted surfaces and are capable of producing an antibiotic-resistant biofilm; coagulase-negative staphylococci susceptible to novobiocin are particularly frequent. Conversely, late PVE tends to mimic the pattern of native valve endocarditis, with a predominance of streptococcal infections. Additional risk factors include the type of prosthesis used, postoperative infections affecting the sternum or urinary tract, and performing the initial valve replacement surgery during active endocarditis.^{1,2,7-9}

The treatment of PVE requires a multidisciplinary approach, where surgery is considered the best therapeutic option for complicated cases. The main indications for surgical re-intervention include severe congestive heart failure, the presence of large vegetations (> 10 mm) with embolism risk, periprosthetic dehiscence, the formation of perivalvular abscesses or fistulas, and persistent sepsis despite effective

La mortalidad fue 36.8% y sobrevivió de 63.1%. **Conclusiones:** la endocarditis de válvula protésica tratada quirúrgicamente en nuestro centro representa una de las patologías más letales en cirugía cardiovascular. La invasión perivalvular (dehiscencia, abscesos y fistulas) son indicación quirúrgica primaria. La alta supervivencia valida la estrategia de intervención quirúrgica agresiva y temprana.

Palabras clave: procedimientos quirúrgicos cardíacos, complicaciones, endocarditis bacteriana, endocarditis, válvula protésica, prótesis valvular cardíaca, infecciones, tasa de supervivencia.

antibiotic treatment. Surgical options range from simple valve replacement to complex reconstruction of the aortic root with allografts, especially in the presence of extensive periannular involvement.^{2,5,6,10-13}

Despite technical advances, PVE is associated with high morbidity and a mortality rate of 20 to 40%. Poor prognostic factors include advanced age, diabetes mellitus, staphylococcal or fungal infections, multi-valve involvement, and hemodynamic instability. It has been established that the most significant risk factor for infection recurrence and mortality is denying surgery when a clear indication exists.^{2,3,6,11}

MATERIAL

A retrospective, observational, and descriptive study was conducted. The population included all patients undergoing cardiac surgery at our institution during the period from January 1, 2022, to December 31, 2024 (n = 655). The specific sample for data analysis consisted of 19 patients diagnosed with prosthetic valve endocarditis (PVE) who underwent surgical management during the 2022 to 2024 period.

Patients of any age and sex with a diagnosis of PVE by modified Duke criteria (2023) and patients undergoing cardiac surgical re-intervention for PVE were included. Patients with NVE and incomplete medical records were excluded.

Categorical variables (e.g., pathogen, affected valve) are expressed as absolute frequencies and percentages (proportions). Continuous variables (e.g., age, vegetation size) are expressed as medians. The incidence and frequency of PVE were estimated relative to the total number of cardiac surgeries. The Kaplan-Meier method was used for survival analysis.

RESULTS

Findings were obtained from the retrospective analysis of the cohort of 19 patients undergoing surgery for PVE between 2022 and 2024.

PVE had an incidence of 2.9% of total cardiac surgeries and represented 32.2% of all infective endocarditis cases operated

on at the hospital. The mean age of the cohort was 51.8 years. 68.4% (n = 13) of the patients were men. The majority of PVE cases developed on mechanical prostheses (84.2%, n = 16), while only 15.8% (n = 3) were observed on previously placed bioprosthesis. 52.6% of patients presented with early PVE and 47.4% with late PVE (*Table 1*).

Regarding identified risk factors potentially associated with the development of PVE, eight patients had diabetes mellitus, six were of advanced age, and one patient had chronic renal failure. Seven patients had a history of prior surgery for native valve endocarditis, suggesting a highly susceptible population. The most common precursor infections were pneumonia (n = 3), wound infection (n = 2), and periodontal infection (n = 2). Clinically, fever was present in 16 patients, and NYHA functional class III was the most common, at 84.2%. Six patients presented with an ischemic cerebrovascular event, the origin of which was determined to be vegetation embolism. Four patients with PVE arrived in septic shock.

Regarding microbiological etiology results, staphylococci were the predominant pathogens, constituting 73.6% of cases (n = 14). Seven patients grew *Staphylococcus aureus* in blood cultures, seven grew *Staphylococcus epidermidis*, and five grew *Enterococcus faecalis*.

Regarding echocardiographic findings, all patients underwent transesophageal echocardiography to confirm the diagnosis. The aortic valve was the most affected (n = 10, 52.6%), followed by combined aortic and mitral involvement (n = 4). Vegetations were detected in 84.2% of patients (n = 16), and the average vegetation size was 12 mm. All patients

Table 1: Results by category (N = 19).

Characteristics	n (%)
Demographics	
Male	13 (68.4)
Female	6 (31.6)
Type of prosthesis	
Mechanical	16 (84.2)
Bioprosthetic	3 (15.8)
Pathogen	
<i>Staphylococcus aureus</i>	7 (36.8)
<i>Staphylococcus epidermidis</i>	7 (36.8)
<i>Enterococcus faecalis</i>	5 (26.3)
Type of prosthetic valve endocarditis	
Early	10 (52.6)
Late	9 (47.4)
Location	
Aortic (isolated)	10 (52.6)
Aortic and mitral	4 (21.0)
Aortic and tricuspid	1 (5.3)
Mitral (isolated)	3 (15.8)
Tricuspid (isolated)	1 (5.3)

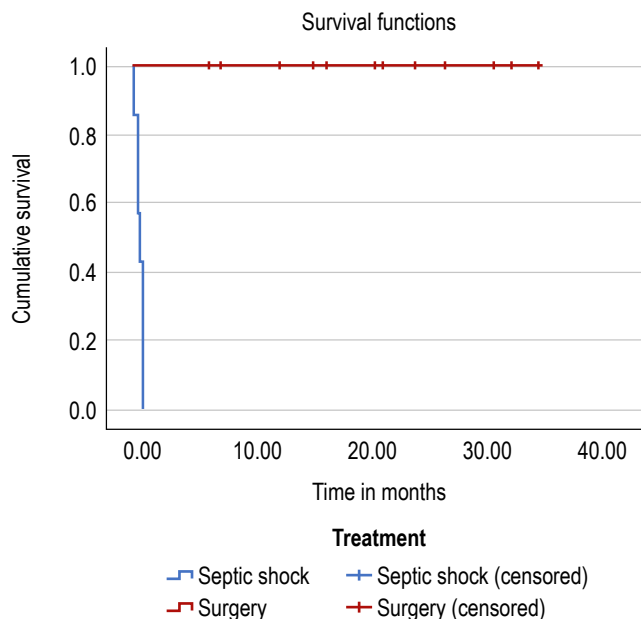


Figure 1: Survival analysis. An abrupt drop in the curve (light blue) is evident in patients who arrived in septic shock, whose survival did not exceed six days. The red curve represents patients who underwent surgery and whose hemodynamic status was not severely compromised, evidencing notable medium-term survival.

presented some degree of prosthetic dysfunction. Perivalvular findings, indicative of uncontrolled local infection, were common and severe: prosthesis dehiscence and paravalvular leak (n = 10), abscesses (n = 7), and intercavitary fistulas (n = 6), with the most frequent being between the aorta and the right atrium. Other findings like pannus (n = 4) and thrombus (n = 3) were less common. The average left ventricular ejection fraction was found to be decreased to 45%, associated with the fact that the vast majority of patients were in NYHA functional class II-III at diagnosis.

Thirty-day hospital mortality was high. Seven patients died: two died in the ward and five patients in the immediate postoperative period (i.e., in less than 30 days), representing a hospital mortality of 36.8%. Survival analysis (Kaplan-Meier) showed an abrupt drop in the curve in the first six days, stabilizing thereafter. The estimated cumulative survival probability at medium term (until the end of follow-up) was 63.1% (*Fig. 1*).

DESCRIPTION OF FINDINGS AND SURGICAL TECHNIQUE

The surgical indication was based on the presence of vegetations and prosthetic valve dysfunction leading to clinically significant heart failure. The hemodynamic status was evaluated and considered, particularly for the four patients who arrived in septic shock; they were given support with

vasopressors and broad-spectrum antibiotic coverage prior to surgical resolution, to achieve minimum hemodynamic stability before surgery to tolerate the surgical event. However, echocardiographic findings suggestive of uncontrolled local infection, such as abscesses, prosthetic dehiscence, and fistulas, ratified the need for emergent surgery as the first line of treatment. It is important to note that all patients, from readmission and suspicion of PVE, received prophylactic antibiotic coverage with vancomycin until culture results and respective antibiograms were available.

As a preventive measure for surgery, the availability of femoral cannulas and vascular grafts was guaranteed for eventual peripheral cannulation if necessary. In this cohort, all patients were approached via median sternotomy, and femoral cannulation was performed in only two of them.

One of the most significant challenges in cardiac reoperation is successfully releasing adhesions and adequately exposing the heart and great vessels to achieve good vascular control (*Fig. 2*). The risk of bleeding, myocardial wall rupture, dissection, and vascular catastrophe is very high in a reoperation, particularly during the release of adhesions.

Among the surgical findings, all patients had grade III and IV adhesions according to the Zuhke classification. In two patients who experienced greater hemodynamic deterioration, ventricular fibrillation occurred during opening and adhesion release. Upon attempting to free tissue for electrical cardioversion, dehiscence of the previous aortorrhaphy occurred, necessitating femoral cannulation; however, despite vascular control, the ventricular fibrillation was refractory, and the tissue was not viable for repair due to poor quality, resulting in death.

Once exposure is achieved, it is important to manipulate the heart as little as possible to avoid embolization of vegetations attached to the valvular prostheses. Once in cardiac arrest, the affected prosthesis was identified, and as a first step, visible vegetations were extracted, followed by annular release of the prosthesis, preserving the greatest amount of fibrous tissue if possible, while simultaneously resecting infected, necrotic, or dehiscent tissue.

In all patients, the previous prosthetic valve was extracted and replaced with a new one (*Fig. 3*) (*Fig. 4*).

Before placing sutures for the new prosthesis, the residual tissue in the annulus must be evaluated, and ventricular cavities explored as appropriate to avoid leaving residual thrombi or vegetations. In cases where abscesses were present, they were drained, tissue was cleaned, and the area was plicated with a pericardial patch, which aided in anchoring the sutures of the new valve (*Fig. 3*).

There were situations where abscesses existed in the mitral-aortic continuity and the aortic root, as well as fistulas towards atrial cavities (*Fig. 4*). In the case of fistulas, the defect is similarly repaired with a pericardial patch, isolating the cavities (*Fig. 3*). In our case series, there was no need to

perform surgery for resection and repair of the mitral-aortic continuity (Commando procedure), under the precept of maintaining the greatest amount of viable tissue and avoiding being more invasive with the myocardium.

One of the surgical decisions made concerns the depth of sutures in an already weakened annulus. Within our experience, each case and each annulus is individualized; however, in mitral replacements, greater care is taken not to injure the circumflex artery. Regarding rhythm disorders, principally atrioventricular blocks that could be generated, the risk and benefit of placing a definitive epicardial pacemaker versus not being able to reseat a new prosthesis in the patient are weighed. Therefore, if viable, the greatest amount of healthy tissue must be taken to anchor the new prosthesis. In our cohort, four patients presented complete AV block requiring placement of an epicardial pacemaker.

Finally, one of the variables also evaluated was the cardiopulmonary bypass time and aortic cross-clamp time. The mean CPB time was 210 minutes, and the aortic cross-clamp time was 130 minutes. The hospitalization time for patients who survived ranged between 42 to 50 days, associated with compliance with antibiotic therapy.

DISCUSSION

Our findings confirm the significant burden that PVE imposes on surgical practice. The incidence of 2.9% represents a high result, exceeding the upper limit of the reported incidence range (0.3 to 1.2%) per patient-year. In our center, PVE constituted 32.2% of all operated endocarditis cases, which is higher than the 20-30% generally reported in large registries such as the Euro Heart Survey and the ICE-PCS study. This high proportion could reflect a cohort with

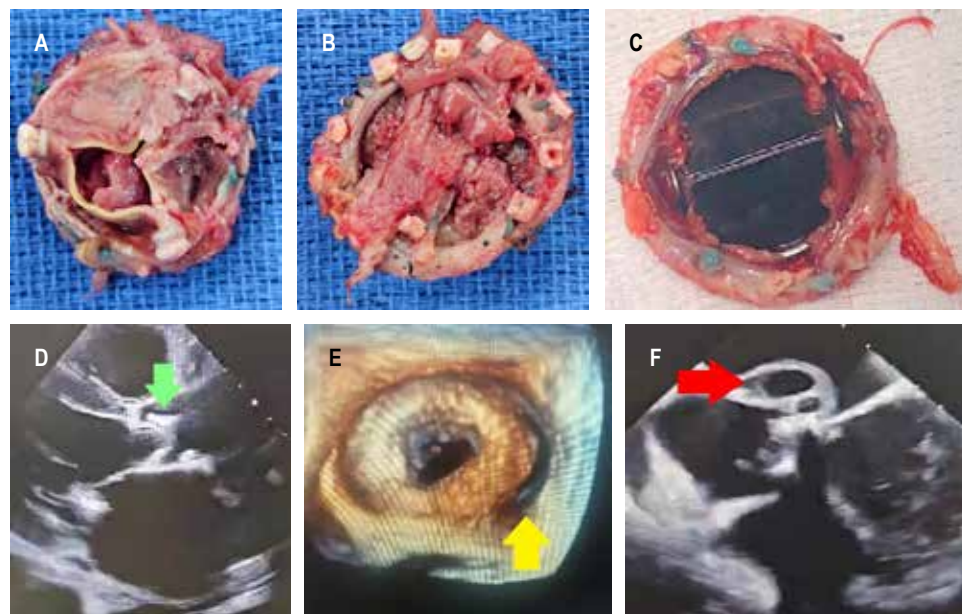
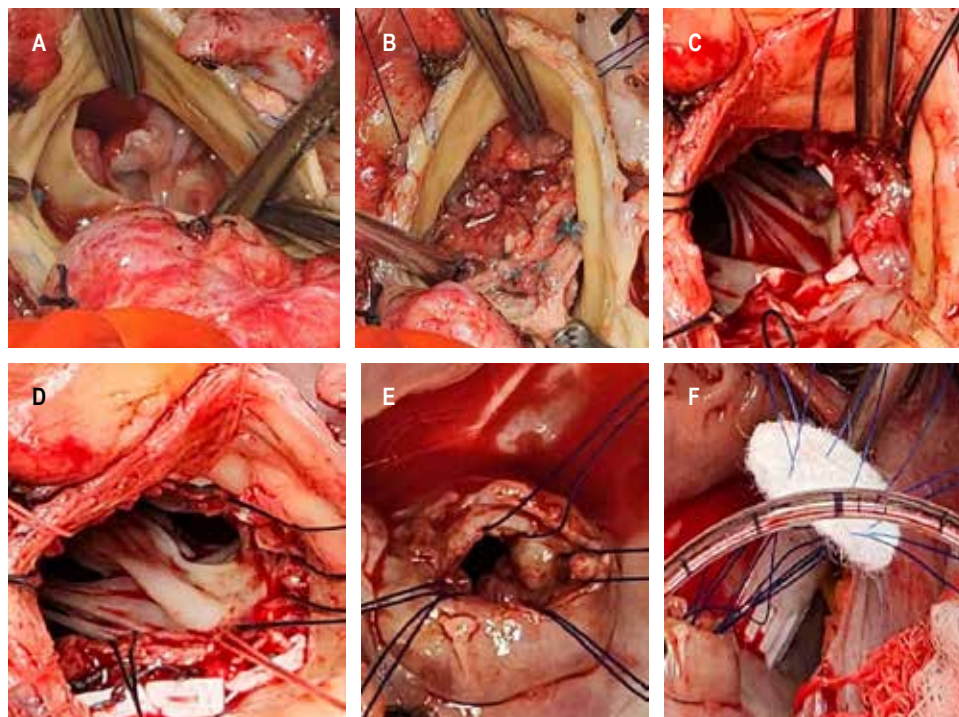


Figure 2:

Heart and great vessels exposed following adhesion release.

Figure 3:

A) Prosthetic valve endocarditis of a biological valve prosthesis in the aortic position; severe obstruction is evident. **B)** Prosthesis with dehiscence; extraction begins while preserving fibrous annular tissue. **C)** Residual aortic ring following valve prosthesis resection. Exploration of the left ventricle is performed to ensure no residual vegetations remain. **D)** The residual ring is cleaned of necrotic and infected tissue. At tear sites, it is plicated with a pericardial patch to reinforce the ring. Wide and loose sutures are placed to grasp the maximum amount of healthy ring. These sutures may pass through the previously placed pericardial patch for reinforcement. **E)** Fistula to the right atrium. Necrotic tissue is resected. **F)** Polytetrafluoroetileno (PTFE) patch is placed for fistula closure.

**Figure 4:**

A) Aortic surface. **B)** Ventricular surface of biological valve prosthesis with endocarditis. Abundant vegetations are observed obstructing and limiting opening. Fibrin tissue on leaflets and prosthesis cuff. **C)** Mechanical prosthesis with abundant pannus and vegetations limiting leaflet opening and closing. **D)** Vegetation attached to the arterial aspect of a mechanical prosthesis in the aortic position (green arrow). **E)** 3D transesophageal echocardiogram reconstruction of a biological mitral valve. Abundant pannus and prosthesis dehiscence with severe paravalvular leak are observed (yellow arrow). **F)** Abscess (red arrow) in the aortic root that caused a fistula towards the right atrium.

greater complexity or a more aggressive surgical policy at our center.^{2,5,6,11,13}

Temporal classification revealed a slight predominance of Early PVE (52.6%). This form is invariably associated with a worse prognosis, as infections are nosocomial in origin, highly virulent, and develop on a myocardium still recovering from the initial surgery. Nevertheless, this result compels us

to review and safeguard the correct execution of asepsis and antisepsis protocols followed within the institution. Regarding late PVE, there were cases where the presence of thrombi led to a higher risk of microbial seeding and development on the prostheses; during the review of medical records, incomplete adherence to anticoagulant treatment was evidenced. Another aspect identified in outpatient assessments, although not

evaluable in all patients due to incomplete follow-up notes, was neglect in home wound cleaning, as well as untreated dental infections. In particular, these were the patients who presented with septic shock upon admission and whose surgical findings were devastating. These findings suggest that sociocultural conditions may contribute to wound infection, poor clinical evolution, and consequently, a portal of entry for endocarditis development.^{2,3,6,8,9,11}

The etiological profile of our cohort is consistent with the PVE pattern, especially early PVE. The predominance of staphylococci (73.6%), divided equally between *Staphylococcus aureus* (36.8%) and *Staphylococcus epidermidis* (36.8%), underscores the high burden of nosocomial pathogens. In the literature, coagulase-negative staphylococci (*S. epidermidis*) are the most frequent in early PVE due to their ability to form antibiotic-resistant biofilms on prosthetic material. The presence of *Enterococcus faecalis* in 26.3% of cases is another sign of the complexity of our cohort. While streptococci are more common in late PVE in some series, our results lean towards pathogens with high intrinsic or acquired resistance, explaining the high rate of antibiotic treatment failure associated with worse perioperative findings and greater cardiac tissue destruction.^{3,5-9,12}

Echocardiographic findings evidence the destructive nature of the infection. Almost all patients had a vegetation attached to the prosthesis, and the average size of 12 mm is above the threshold of 10 mm considered a criterion for high embolic risk and an indication for surgery. A critical finding has been the high rate of perivalvular complications, such as dehiscence (52.6%), abscesses (36.8%), and intercavitary fistulas (31.6%). These complications are the result of local invasion by uncontrolled infection and are directly associated with the need for complex surgical procedures, such as annulus reconstruction or aortic root replacement. The predominant involvement of the aortic valve (73.6%) is especially concerning, as infection in this position tends to extend to the fibrous skeleton of the heart, complicating surgery and affecting prognosis.^{3-6,10,11}

The analysis of perioperative findings demonstrated extensive tissue destruction secondary to infection. While an attempt was always made to be as conservative as possible, the removal of the greatest amount of infected tissue was guaranteed to prevent recurrence. All patients underwent prosthesis replacement, and in more severe cases of intercavitary fistulas, defects were repaired with bovine pericardial patches. One of the main complications in the perioperative period was conduction system blocks, which in some cases required definitive epicardial pacemaker placement. A common surgical finding in all patients was firm grade III and IV adhesions based on the Zuhlke classification, which led to longer surgical times. In two cases of death, femoral cannulation had to be performed; however,

the myocardial tissue was friable and of poor quality, not viable for repair. The cardiopulmonary bypass time was somewhat prolonged; however, the aortic cross-clamp time was shorter, reflecting the myocardial stunning that occurs following reoperation and extensive myocardial manipulation. Specifically, after aortic unclamping, we maintained the patient on circulatory assistance for a considerable time until achieving adequate myocardial contractility and tolerance to weaning from cardiopulmonary bypass.^{1-3,5,6,12,13}

Our hospital mortality of 36.8% is at the upper end of the range reported in the literature (20-40%). In this context, it is important to mention its relationship with pre-existing risk factors in our cohort, such as cases of septic shock and cerebral embolism upon admission, which are predictors of poor outcomes. The Kaplan-Meier analysis offers a crucial perspective. The rapid drop in survival in the first six days confirms that mortality is dominated by the acute surgical phase and postoperative multi-organ failure. However, the subsequent stabilization of survival at 63.1% suggests that surgical intervention, although carrying a high initial risk, is effective in controlling the infection and its complications. This sustained survival highlights the clinical maxim that “denying surgery when an obvious indication exists is the most important risk factor for mortality.” Patients who manage to overcome the perioperative phase benefit from the radical debridement that only surgery can offer.^{1-3,5,6,10,11,13}

The results of this study reinforce the need for aggressive diagnostic evaluation (including TEE and cardiac CT, according to guidelines) and early surgical decision-making in the face of any sign of medical treatment failure or perivalvular complication. The high number of perivalvular complications in our series demands that surgical protocols emphasize radical debridement and the use of complex reconstruction techniques, such as the use of homografts or aortic root replacement, when the annulus is compromised.^{2,3,6,11-14}

CONCLUSIONS

PVE treated surgically at our center during the 2022-2024 period reaffirms its status as one of the most lethal pathologies in cardiovascular surgery. The analysis demonstrated that our cohort of 19 patients presented high complexity, characterized by a predominance of early PVE (52.6%) and an etiology marked by highly virulent pathogens (staphylococci and enterococci in almost 100% of cases). Echocardiographic findings confirmed extensive perivalvular invasion (dehiscence, abscesses, and fistulas) in the majority of cases, which constituted the primary surgical indication.

Hospital mortality was high (36.8%), aligning with the upper ranges of international literature. However, the Kaplan-Meier survival analysis revealed that all mortality was concentrated in the immediate postoperative period

(first six days). Patients who overcame this critical phase presented robust medium-term survival (63.1%), validating the strategy of aggressive and early surgical intervention as the fundamental rescue treatment in the face of medical management failure and local infectious complications.

REFERENCES

- Galadí J. Guía ESC 2023 sobre el diagnóstico y el tratamiento de la endocarditis [Internet]. Sociedad Española de Cardiología. [citado 2 Diciembre 2025]. Disponible en: <https://secardiologia.es/publicaciones/catalogo/guias/14870-guia-esc-2023-sobre-el-diagnostico-y-el-tratamiento-de-la-endocarditis>
- Nappi F. Advancements and challenges in the management of prosthetic valve endocarditis: a review. *Pathogens*. 2024;13(12):1039. doi: 10.3390/pathogens13121039.
- Ko S, Uno S, Kohsaka S. Prosthetic aortic valve endocarditis. *Intern Med*. 2019;58(21):3201. doi: 10.2169/internalmedicine.3015-19.
- Emperador CR, Gorrín MG, Ramírez ML, Trelles JO, Abiz-Reck MN, Ayllón MH. Endocarditis infecciosa en válvulas protésicas. *CorSalud*. 2020;12(2):146-154. Available in: <https://dialnet.unirioja.es/servlet/articulo?codigo=8106556>.
- Olmos C, Vilacosta I, López J, Sarriá C, Ferrera C, San Román JA. Actualización en endocarditis protésica. *Cir Cardiovasc*. 2017;24(1):33-40. doi: 10.1016/j.circv.2016.11.001.
- Revilla A, López J, Sevilla T, et al. Pronóstico hospitalario de la endocarditis protésica tras cirugía urgente. *Rev Esp Cardiol*. 2009;62:1388-1394. doi: 10.1016/S0300-8932(09)73124-0.
- Li JS, Sexton DJ, Mick N, et al. Proposed modifications to the Duke criteria for the diagnosis of infective endocarditis. *Clin Infect Dis*. 2000;30(4):633-638. doi: 10.1086/313753.
- Berisha B, Ragnarsson S, Olaison L, Rasmussen M. Microbiological etiology in prosthetic valve endocarditis: a nationwide registry study. *J Intern Med*. 2022;292(3):428-437. doi: 10.1111/joim.13491.
- Rajani R, Klein JL. Infective endocarditis: a contemporary update. *Clin Med (Lond)*. 2020;20(1):31-35. doi: 10.7861/clinmed.cme.20.1.1.
- Glaser N, Jackson V, Holzmann MJ, Franco-Cereceda A, Sartipy U. Aortic valve replacement with mechanical vs. biological prostheses in patients aged 50-69 years. *Eur Heart J*. 2016;37(34):2658-2667. doi: 10.1093/eurheartj/ehv580.
- Cuervo G, Quintana E, Regueiro A, et al. The clinical challenge of prosthetic valve endocarditis: JACC Focus Seminar 3/4. *J Am Coll Cardiol*. 2024;83(15):1418-1430. doi: 10.1016/j.jacc.2024.01.037.
- Akowuah EF, Davies W, Oliver S, et al. Prosthetic valve endocarditis: early and late outcome following medical or surgical treatment. *Heart*. 2003;89(3):269-272. doi: 10.1136/heart.89.3.269.
- Mahesh B, Angelini G, Caputo M, Jin XY, Bryan A. Prosthetic valve endocarditis. *Ann Thorac Surg*. 2005;80(3):1151-1158. doi: 10.1016/j.athoracsur.2004.11.001.
- Wang A, Athan E, Pappas PA, et al. Contemporary clinical profile and outcome of prosthetic valve endocarditis. *JAMA*. 2007;297(12):1354-1361. doi: 10.1001/jama.297.12.1354.

Funding: none.

Disclosure: the authors have no conflict of interest to disclose.

Johari Window: heart transplant following donation after circulatory death.

Expanding the donor pool in Mexico

Ventana de Johari: trasplante de corazón tras la donación después de la muerte circulatoria. Aumentando el número de donantes en México

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ABSTRACT

This statement discusses how Donation after Circulatory Death (DCD) can expand the heart donor pool and improve access to transplantation, particularly in Mexico. It reviews key barriers to DCD implementation, including unavoidable warm ischemia, cost and logistical complexity, and ethical/legal concerns surrounding certain procurement strategies. Two established approaches are summarized: Direct Procurement and Perfusion (DPP) using *ex situ* normothermic perfusion platforms, and Thoracoabdominal Normothermic Regional Perfusion (TA-NRP), which allows *in situ* assessment but may raise “dead donor rule” concerns. Using the Johari Window framework, the author argues that some feasible solutions remain in a national “blind spot”. The paper highlights Rapid Recovery with Extended Ultra-Oxygenation Preservation (REUP) as a promising, lower-cost, reanimation-less strategy that may avoid expensive technology and ethical issues tied to reperfusion or *ex situ* systems. Early reported outcomes are presented as comparable to DPP and TA-NRP, including feasibility with prolonged “no-touch” periods and extended ischemic times. The author concludes that REUP could enable broader DCD adoption in Mexico, while emphasizing the need for further comparative and long-term studies.

RESUMEN

Este documento analiza cómo la donación tras la muerte circulatoria (DCD, por sus siglas en inglés) puede ampliar el grupo de donantes de corazón y mejorar el acceso al trasplante, particularmente en México. Se revisan las principales barreras para la implementación de la DCD, incluyendo la inevitable isquemia caliente, la complejidad logística y de costos, y las preocupaciones éticas y legales que rodean a ciertas estrategias de obtención. Se resumen dos enfoques establecidos: la procuración directa y perfusión (DPP, por sus siglas en inglés) mediante plataformas de perfusión normotérmica ex situ, y la perfusión regional normotérmica toracoabdominal (TA-NRP, por sus siglas en inglés), que permite la evaluación in situ, pero puede plantear dudas sobre la “regla del donante muerto”. Utilizando el marco de la ventana de Johari, el autor sostiene que algunas soluciones viables permanecen en un “punto ciego” nacional. El artículo destaca la procuración rápida con preservación por ultraoxigenación extendida (REUP, por sus siglas en inglés) como una estrategia prometedora, de menor costo y sin necesidad de reanimación, que podría evitar la tecnología costosa y los problemas éticos vinculados a la reperusión o a los sistemas ex situ. Se presentan informes de resultados tempranos comparables a los de DPP y TA-NRP, incluyendo su factibilidad con periodos prolongados

How to cite: Orozco-Hernández EJ. Johari Window: heart transplant following donation after circulatory death. Expanding the donor pool in Mexico. *Cir Card Mex*. 2026; 11 (3): 90-97. <https://dx.doi.org/10.35366/123482>

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Received: 26-03-2026. Accepted: 01-05-2026.

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Keywords: death by circulatory determination, thoracoabdominal normothermic regional perfusion, direct procurement and perfusion, rapid recovery with extended ultra-oxygenation preservation, heart transplantation, Johari Window.

Abbreviations:

CPB = Cardiopulmonary Bypass
 DBD = Donation after Brain Death
 DCD = Donation after Circulatory Death
 DPP = Direct Procurement and Perfusion
 DPP = Direct Procurement Protocols
 ECMO = Extracorporeal Membrane Oxygenation
 NRP = Normothermic Regional Perfusion
 OCS = Organ Care System
 REUP = Rapid Recovery with Extended Ultra-Oxygenation Preservation
 TA-NRP = Thoracoabdominal Normothermic Regional Perfusion

This document explores the transformative potential of Donation after Circulatory Death (DCD) for expanding the heart donor pool in Mexico. Using the Johari Window framework, the author analyzes how institutional blind spots and knowledge asymmetries hinder the adoption of innovative DCD techniques. The paper reviews established procurement methods like Direct Procurement and Perfusion (DPP) and Thoracoabdominal Normothermic Regional Perfusion (TA-NRP),¹ while highlighting the novel Rapid Recovery with Extended Ultra-Oxygenation Preservation (REUP) technique. REUP enables reanimation-less recovery of DCD hearts, offering comparable early outcomes to traditional approaches while reducing costs and logistical complexity.² The paper argues that integrating REUP could help Mexico overcome technical, ethical, and legislative barriers, transforming hidden institutional limitations into opportunities for equitable access to heart transplantation. Ultimately, the piece calls for greater transparency, collaboration, and openness to innovation, positioning REUP as a promising solution for countries facing resource constraints and prolonged ischemic times.

Approximately 6.7 million Americans are afflicted with heart failure, of whom roughly 5% progress to refractory, end-stage disease (American College of Cardiology/American Heart Association stage D/New York Heart Association class IV [ACC/AHA Stage D/NYHA IV]), yielding a subset of roughly 335,000 patients grappling with advanced heart failure at any given time. This Stage D cohort, though numerically small, represents an exceptionally high-risk population for whom advanced therapeutic interventions (including left ventricular assist devices and transplantation) become imperative.

de observación (“no-touch”) y tiempos de isquemia extendidos. El autor concluye que la técnica REUP podría permitir una adopción más amplia de la DCD en México, enfatizando la necesidad de realizar más estudios comparativos y de largo plazo.

Palabras clave: donación por muerte circulatoria, perfusión regional normotérmica toracoabdominal, procuración directa y perfusión, procuración rápida con preservación por ultraoxigenación extendida, trasplante de corazón, ventana de Johari.

The most up-to-date publicly available data show that the United States heart transplant waiting list includes approximately 3,289 patients nationwide.³

The number of heart transplants performed in the United States annually continues to increase, reaching approximately 4,000 in 2024. Altogether, despite increases in heart transplantation, the current incidence of surgical treatment of end-stage heart failure does not meet the need of patients suffering from advanced heart failure.^{4,5} This increase was mostly driven by the 68% increase in the use of DCD donors with a smaller increase of 4.6% in Donation after Brain Death (DBD) donors.^{6,7} Some of this increase is also related to liberalization of qualifications of potential recipients, such as increased age and comorbidities that were previously prohibitive (human immunodeficiency virus [HIV], hepatitis C) but now are treatable.^{8,9}

DCD refers to organ procurement from donors who, though not meeting brain death criteria, suffer irreversible cessation of circulatory and respiratory function following withdrawal of life-sustaining therapy, a practice distinct from donation after brain death.¹⁰ Though pioneered by Christiaan Barnard in 1967, DCD heart transplantation lay largely dormant until its revival in the 2000's, spurred by successful transplants in Australia¹¹ and the United Kingdom,¹² and later adopted in the US in 2019.¹³ While DCD donors have expanded the cardiac allograft pool (a critical step given transplant demand) their utilization introduces a clinical challenge: inevitable warm ischemic injury during therapy withdrawal, demanding meticulous donor-recipient matching and logistical coordination.

To ensure graft viability, DCD heart procurement employs distinct strategies, each with unique procedural nuances and trade-offs: DPP, TA-NRP, and select modified protocols. These approaches aim to mitigate ischemic injury, though optimal techniques remain under evaluation.

Before exploring potential adaptations to expand DCD heart procurement in Mexico (a country with unique healthcare constraints) let's examine current strategies and their applicability. Given the technology and investment required, leveraging international experience via the Johari Window framework may illuminate pathways to increase donor utilization and transplants locally.

The Johari Window (a four-quadrant model of knowledge distribution) proves useful beyond interpersonal dynamics. In DCD transplantation, it highlights knowledge asymmetries: open (known to all: brain death vs. DCD), hidden (local expertise undisclosed), blind (unseen barriers), and unknown (untapped innovations). Disclosure and collaboration expand the “open area”, informing strategies to increase donor utilization.

The blind area (those truths known to others but obscured to oneself) contracts as feedback refines self-perception. Within this quadrant reside overlooked techniques, latent knowledge, and procedural alternatives that remain invisible to dominant paradigms. For instance, a DCD heart procurement method exists that bypasses both Direct Procurement Protocols/Organ Care System (DPP/OCS) and Normothermic Regional Perfusion (NRP). It is simpler, more cost-effective, and reproducible, yet remains largely unrecognized in certain national programs. This gap is not merely technical, it reflects a deeper institutional blind spot, where innovation is constrained not by feasibility but by visibility. Bridging this divide requires not only procedural awareness but philosophical openness to what others already see. The hidden area reflects intentional modulation of information; we found here things that you know about yourself, but are unknown to others, like the Mexican Legislation about DCD, and the

unknown area represents latent capacities or unrecognized patterns that may emerge under new conditions. As its name suggests, in this area we found the things that are unknown to you and by everyone else (what is the best technique for DCD heart procurement?).

In this sense, the model parallels adaptive biological systems, where transparency, feedback, and environmental interaction govern functional coherence (Fig. 1).

Philosophically, the Johari Window evokes the notion of relational epistemology -the idea that knowledge of oneself is neither static nor solitary, but co-constructed through interaction. It highlights the inherent asymmetry between how we see ourselves and how we are seen, and it underscores the humility required to navigate that gap. The model suggests that self-understanding is not merely introspective but contingent upon the willingness to engage with others, to tolerate vulnerability, and to accept that parts of the self remain undiscovered until circumstances reveal them.

In this dual scientific-philosophical framing, the Johari Window becomes more than a communication tool; it becomes a reminder that human insight is iterative, relational, and perpetually incomplete. It affirms that growth (whether personal, organizational, or clinical) emerges from the interplay between what we reveal, what we learn, and what we have yet to uncover.

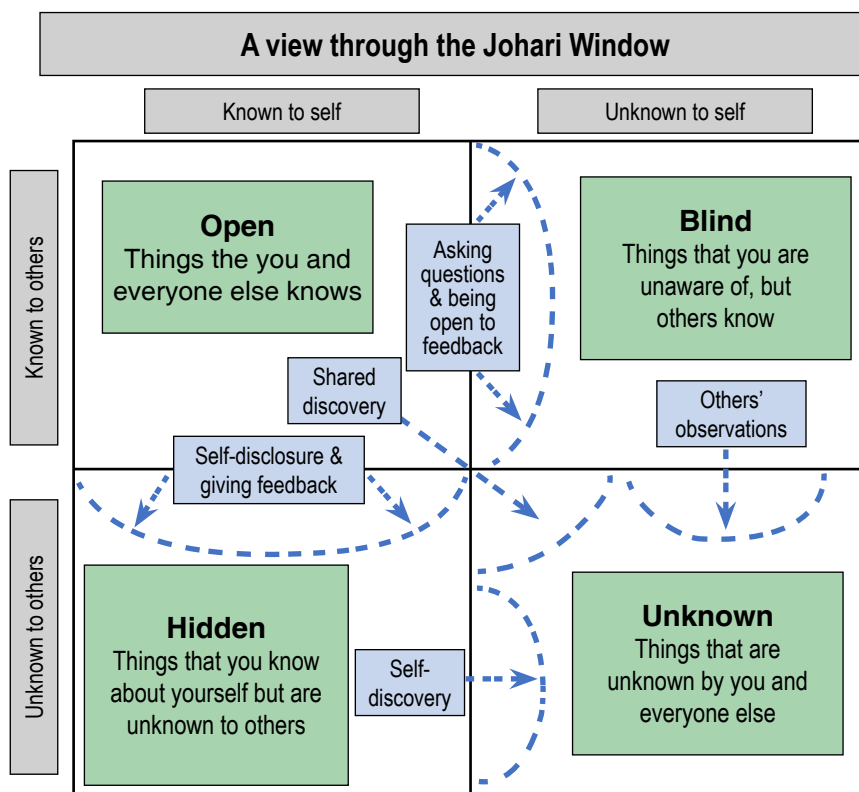


Figure 1:

The Johari Window. A four-quadrant framework illustrating the interaction between self-knowledge and external perception. The model comprises the open (known to self and others), hidden (known to self only), blind (known to others only), and unknown (known to neither) areas, highlighting how feedback and disclosure shape the evolution of personal insight.

The dynamic and essential logistics required for progress in the field of transplantation and, in truth, in nearly every area of science and art.

Let us now examine the different techniques employed in DCD heart procurement:

Direct Procurement and Perfusion (DPP)

Procedure: after circulatory death is confirmed and a standoff period is observed, the donor heart is rapidly procured and immediately placed on an *ex situ* normothermic perfusion platform, such as the OCS, for reanimation and functional assessment. This process typically involves draining 1.2–1.5 liters of donor blood to prime the perfusion system before aortic cross-clamping and heart explantation. The heart is then transported to the recipient center on the perfusion device (*Fig. 2*), allowing for continued assessment and preservation during transport.^{14,15}

Advantages and limitations: allow for *ex situ* functional assessment of the heart.

Can be resource-intensive, costly, and operationally complex due to the need for specialized equipment and trained personnel¹⁶ and may delay abdominal organ perfusion, potentially increasing warm ischemia time for other organs.¹⁷

Thoracoabdominal Normothermic Regional Perfusion (TA-NRP)

Procedure: after circulatory death is declared, Extracorporeal Membrane Oxygenation (ECMO) is established via central aortic and right atrial cannulation. The cranial branches of the aortic arch are clamped to prevent cerebral reperfusion, and circulation is restored to the thoracoabdominal organs. The heart is reanimated *in situ*, and its function is assessed under physiological conditions using transesophageal echocardiography and pulmonary artery catheterization before procurement (*Fig. 3*). The heart is then preserved, usually using cold storage for transport.^{15,18}

Advantages and Limitations: enables *in situ* functional assessment of the heart in a loaded state, which may improve graft selection and outcomes.^{18,19}

Allows for more rapid reperfusion of all transplantable organs, potentially reducing warm ischemic injury to abdominal organs.¹⁷ Less costly than *ex situ* perfusion, but has raised ethical and legal concerns regarding the dead donor rule and restoration of circulation after death.^{14,19}

A modification that could significantly expand its use in developing countries is the substitution of conventional Cardiopulmonary Bypass (CPB) for ECMO, as this would dramatically reduce procedure-related costs, and current knowledge and experience with CPB are far more widespread and disseminated than those concerning ECMO.

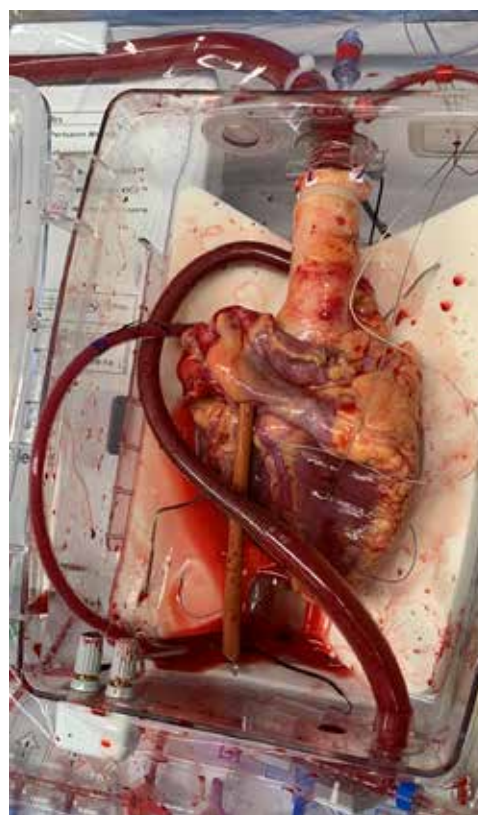


Figure 2: Heart in organ care system after direct procurement and perfusion.

Rapid Recovery with Extended Ultra-Oxygenation Preservation (REUP) [BLIND AREA]

Entering the subject directly, this technique represents the cornerstone for achieving the objective described in the title of the manuscript.

Though nascent and unconventional, this approach underscores global potentialities, a promise tempered by systemic hurdles: entrenched donation cultures, socioeconomic disparities, and policy constraints variably stifle DCD expansion worldwide.

The REUP technique importantly highlights the issue of cardiac reanimation. Discard rates using *ex situ* perfusion are 8–10%, as visual assessment of cardiac reanimation in an unloaded state and lactate levels may not necessarily correlate with allograft performance, and the fact that the associated mortality of DCD remains comparable to that of DBD loses impact.²⁰ *In situ* cardiac reanimation with TA-NRP provides a more physiologic assessment, and discard rates are much lower at around 1–3%.¹⁸

With REUP, cardiac reanimation may be unnecessary and contribute to added costs. REUP uses Del Nido as

a preservation solution based on ensuring an excellent diastolic arrest and avoiding additional myocardial energy depletion with cardiac reanimation. This additionally eliminates the ethical issue of cardiac reanimation in the donor, which is problematic for NRP. Furthermore, the REUP technique is simple to set up with a small team (one perfusionist), requires little time and setup (30 min), and is much less costly (less than \$2K for disposables) than these other techniques.²¹

This novel, reanimation-less rapid recovery technique using extended oxygenated preservation and cold storage, designed to avoid the need for *ex situ* perfusion systems or TA-NRP, is a technique developed for the recovery and preservation of cardiac allografts from DCD donors. This method is characterized by the rapid, ultra-oxygenated recovery of DCD hearts for transplantation, notably without the need for pre-implant reanimation, which distinguishes it from traditional approaches such as *ex situ* perfusion or TA-NRP.²²

Key features and application of REUP

Reanimation-less recovery: unlike conventional methods that require reanimating the heart before implantation to assess graft viability, REUP enables transplantation without this step; you don't need the use of ECMO, CPB, or TEE, simplifying the process and reducing associated costs.¹⁶

REUP advantages: by providing ultra-oxygenation and eliminating the need for reanimation, REUP may reduce the

risk of organ damage and expand the applicability of DCD heart transplantation, especially in settings with mandatory prolonged "no touch" periods.²¹

Extended ultra-oxygenation: the technique involves providing ultra-oxygenated conditions during the rapid recovery phase, which is intended to preserve organ viability even after prolonged periods of circulatory arrest.

Before withdrawal of life-sustaining care, a modified NRP flush circuit with reservoir was constructed (Fig. 4). The components of the preservation solution included two units of packed red blood cells, two liters of del Nido cardioplegia solution, heparin (30,000 U), mannitol (3,125 mg), solumedrol (125 mg), N-acetyl-L-cysteine (30 g), and levothyroxine (50 mg). The addition of valproic acid, as well as oxygenation of the perfusate, has helped contribute to the favorable outcomes of the REUP technique. Valproic acid has been shown to promote cardioprotection. After mixing the components, the circuit was started at two liters per minute, and the perfusate was oxygenated. However, a perfusate temperature of 10-12 °C was targeted. A pH of 7.5 and a partial pressure of oxygen of 500 mmHg were targeted. Arterial blood gases were analyzed serially until temperature-correct values were achieved.²¹

The premise for the technique is that the slow delivery of an oxygenated preservation solution with additional cardioprotective additives may help resuscitate the heart and reverse the myocardial energy debt incurred during circulatory death.

There is an interesting theory with respect to the concept of reanimation, independently of the protocol (DPP-OCS,

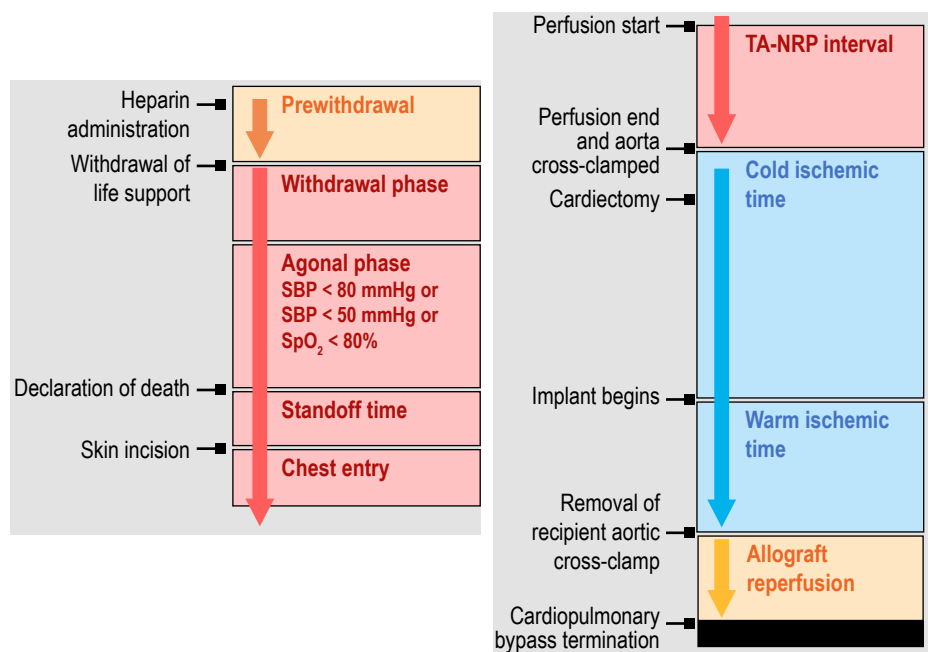


Figure 3:

Algorithm for Toracoabdominal-Normothermic Regional Perfusion (TA-NRP).



Figure 4: Modified normothermic regional perfusion flush circuit with reservoir.

TA-NRP), there may be a deleterious impact introduced by the additional arrest of the DCD donor heart that is necessarily introduced by reanimation techniques. Namely, that the “second hit” of cardiac arrest imposed on the donor heart following reanimation creates additional insult and non-physiologic consequence to the allograft that may impact its function after implantation.^{21,22}

Following the protocol for DCD, when the right time was, A sternotomy was performed, the left and right sides were vented, and an ascending cross clamp was placed. A bifurcated ascending root vent was used to administer the perfusate with an aortic root pressure of 80 mmHg and a flow rate of 200 ml/minute. The donor cardiectomy is performed in standard fashion, and the heart is placed in the 10 °C cooler.

The premise for this technique is that the slow delivery (aortic root pressure of 80 mmHg) of an oxygenated preservation solution may help resuscitate the heart and reverse the myocardial energy debt incurred during circulatory death, and hypothesized that the use of 10 °C preservation would allow successful transplantation of REUP allografts even in the setting of prolonged ischemia time.²²

In the last several years, cardiac allografts from DCD donors have been used in adult heart transplantation with excellent early and midterm outcomes comparable to those from donation after brain death. TA-NRP and DPP with commercial *ex situ* platforms have shown success in countries with short asystolic “no-touch” periods (five minutes or less), including the United States, England, and Australia. However, countries such as Italy have some of the longest known periods, requiring up to 20 minutes of asystole prior to final declaration of death. These prolonged periods of circulatory arrest have been considered major barriers to the uptake of DCD adult heart transplant in these settings. Early studies demonstrated a 10% 30-day mortality rate and a 25% severe PGD rate, and larger follow-up studies showed similar results. Compared to the United States, where “no-touch” periods are typically less than five minutes, the rates of mortality and severe PGD remain significantly higher.²²

Williams et al. reported the longest total ischemic time in literature involving the rapid recovery of a DCD cardiac allograft (eight hours) with static cold storage using the REUP technique with optimal results. Also, the same group described recovery of a 32-year-old donor cardiac allograft with a prolonged “no touch” period of circulatory arrest. Despite 37 minutes of circulatory arrest before donor heart preservation, the recipient underwent successful heart transplantation with normal biventricular function and excellent outcomes postoperatively.²¹ This case challenges the concerns regarding the REUP technique in prolonged “no-touch” periods.

Just recently, Williams et al. reported the first series of DCD hearts using REUP. In this case series of 24 adult patients who underwent heart transplant with REUP-recovered DCD cardiac allografts, 38% were older than 40 years. The mean time from initial declaration of donor death to flush was nine minutes. 60% had a total ischemic time longer than four hours, including one that was eight hours. Among recipients, 30-day survival was 96%. Only one patient (4%) had severe primary graft dysfunction, and one other patient (4%) had secondary graft dysfunction. On initial endomyocardial biopsy, one patient (4%) had acute cellular rejection grade 2R; no cases of antibody-mediated rejection were observed, highlighting the safety, feasibility, and efficacy of this novel DCD recovery technique.²²

The 30-day survival of 96% remains similar to TA-NRP and DPP of 96 and 97%, respectively. Several studies have highlighted a lower rate of biopsy-proven acute cellular rejection grade 2R with TA-NRP compared with a DPP (15 vs 35%) over the first year. The present study also observed low rates of clinically significant acute cellular rejection grade 2R at 4%; although this is low and promising, additional data out to one year are needed.

REUP does not prevent the recovery of any other organ, or use of abdominal NRP, the data in this study reflect similar

abdominal organ and lung utilization rates, compared with the literature.²²

At present, it exists a big deal of controversy internationally regarding the costs of *ex situ* perfusion systems and ethical concerns with TA-NRP. As such, this manuscript highlights how the REUP technique has potential for broad application to allow more patients access to DCD heart transplantation, and will be appealing to programs with long travel times, limited resources (financial and technologic) without cardiac reanimation, with ethical issues that create legislative barriers to advancing the fundamental goal of better balancing the demands of the waiting list with the donor pool available in response. Undoubtedly, REUP is a highly promising technique (particularly in Mexico and other developing countries) for achieving the balance previously mentioned, and it carries substantial potential for worldwide implications for DCD hearts, even in the setting of prolonged pulseless electrical activity and agonal warm ischemic time, geographic distance, and extremely high ischemic times. Although REUP-recovered DCD hearts from younger donors (aged 16-30 years) with shorter ischemic times (< 4 hours) have demonstrated favorable early outcomes, the last report highlights that REUP is safe, feasible, and effective for the recovery of older donor cardiac allografts, for use in high-risk recipients, and for allografts with prolonged ischemic time.

Notwithstanding the excellent early outcomes have been achieved using the REUP technique, additional studies are required to further validate its use. This includes evaluation of long-term patient outcomes, and direct comparisons with TA-NRP and DPP will be needed. Just as further basic and translational science studies are required before widespread adoption, additional prospective trials will be required to evaluate REUP.

ACKNOWLEDGMENTS

The author acknowledges Dr. Nelly J. Mar-Bautista for her contribution to this work. Her expert analysis using the Johari Window framework provided critical insight into the dimensions of heart transplantation following donation after circulatory death.

REFERENCES

- Williams AM, Ahmad A, Bommarreddi S, Lima B, Nguyen D, Quintana E, et al. Normothermic regional perfusion in donation after circulatory death compared with brain dead donors: single-center cardiac allograft outcomes. *J Thorac Cardiovasc Surg.* 2025;170(2):585-593. doi: 10.1016/j.jtcvs.2025.02.023.
- Williams AM, Trahanas JM, Bommarreddi S, Lima B, DeVries SA, Lowman J, et al. Rapid recovery of donor hearts for transplantation after circulatory death. *N Engl J Med.* 2025;393 (3):267-274. doi: 10.1056/NEJMoa2500456.
- Colvin MM, Smith JM, Ahn YS, Lindblad KA, Handarova D, Israni AK, et al. OPTN/SRTR 2023 Annual data report: heart. *Am J Transplant.* 2025;25(2S1):S329-S421. doi: 10.1016/j.ajt.2025.01.024.
- Organ Procurement and Transplantation Network (OPTN). National Data Report: Heart Transplant Waiting List. *OPTN Annual Report*. 2024. Available at: <https://optn.transplant.hrsa.gov> [(optn.transplant.hrsa.gov)]
- Khush KK, Cherikh WS, Chambers DC, Harhay MO, Hayes D Jr, Hsieh E, et al. The International Thoracic Organ Transplant Registry of the ISHLT: Forty-first adult heart transplantation report—2024. *J Heart Lung Transplant.* 2024;43(10):1121-1138.
- Kharawala A, Nagraj S, Seo J, Pargaonkar S, Uehara M, Goldstein DJ, et al. Donation after circulatory death heart transplant: current state and future directions. *Circ Heart Fail.* 2024;17(7):e011678. doi: 10.1161/CIRCHEARTFAILURE.124.011678.
- Bobba CM, Whitson BA, Henn MC, Mokadam NA, Keller BC, Rosenheck J, et al. Trends in donation after circulatory death in lung transplantation in the United States: impact of era. *Transpl Int.* 2022;35:10172. doi: 10.3389/ti.2022.10172.
- Woolley AE, Singh SK, Goldberg HJ, Mallidi HR, Givertz MM, Mehra MR, et al. Heart and lung transplantation from HCV-infected donors to uninfected recipients. *N Engl J Med* 2019;380(17):1606-1617. doi: 10.1056/nejmoa1812406.
- Durand C, Bowring M, Tobian A, Brown D, Desai N, Bagnasco S, et al. Outcomes after HIV+ kidney transplant: a HOPE in action study. *ATC Abstracts.* 2018. Available from: <https://atcmeetingabstracts.com/abstract/outcomes-after-hiv-kidney-transplant-a-hope-in-action-study/>
- Jawitz OK, Devore AD, Patel CB, Keenan JE, Milano CA, Schroder JN. Regional variation in donation after circulatory death heart allograft utilization. *JTCVS Open.* 2024;21:191-196. doi: 10.1016/j.xjon.2024.07.004.
- Dhital KK, Iyer A, Connellan M, Chew HC, Gao L, Doyle A, et al. Adult heart transplantation with distant procurement and *ex-vivo* preservation of donor hearts after circulatory death: a case series. *Lancet.* 2015;385(9987):2585-2591. doi: 10.1016/S0140-6736(15)60038-1.
- Messer SJ, Axell RG, Colah S, White PA, Ryan M, Page AA, et al. Functional assessment and transplantation of the donor heart after circulatory death. *J Heart Lung Transplant.* 2016;35(12):1443-1452. doi: 10.1016/j.healun.2016.07.004.
- Schroder JN, Patel CB, DeVore AD, Bryner BS, Casalnova S, Shah A, et al. Transplantation outcomes with donor hearts after circulatory death. *N Engl J Med.* 2023;388(23):2121-2131. doi: 10.1056/NEJMoa2212438.
- Kaffka Genaamd Dengler SE, Vervoorn MT, Brouwer M, de Jonge J, van der Kaaij NP. Dilemmas concerning heart procurement in controlled donation after circulatory death. *Front Cardiovasc Med.* 2023;10:1225543. doi: 10.3389/fcvm.2023.1225543.
- Benkert AR, Jawitz OK, Lobo AA, Casalnova S, Goel D, Dewan KC, et al. Five-year experience of heart transplantation following donation after circulatory death. *J Thorac Cardiovasc Surg.* 2025;170(6):1687-1696. doi: 10.1016/j.jtcvs.2025.08.013.
- Li X, Ding R, Cai J. Organ transplantation: current status, challenges, and future prospects. *MedComm (2020).* 2026;7(1):e70567. doi: 10.1002/mco2.70567.
- Thomas J, Chen Q, Roach A, Wolfe S, Osho AA, Sundaram V, et al. Donation after circulatory death heart procurement strategy impacts utilization and outcomes of concurrently procured abdominal organs. *J Heart Lung Transplant.* 2023;42(7):993-1001. doi: 10.1016/j.healun.2023.02.1497.
- Williams AM, Ahmad A, Bommarreddi S, Lima B, Pasirja C, Nguyen D, et al. Two hundred cases of cardiac donation after circulatory death utilizing normothermic regional perfusion: the 4-year Vanderbilt experience. *J Heart Lung Transplant.* 2025;44(11):1703-1711. doi: 10.1016/j.healun.2025.05.007.

19. Bashian EJ, Gergen AK, Park SY, Cain MT, Hoffman JRH, Campbell D. A novel technique for innominate artery cannulation in thoracoabdominal normothermic regional perfusion. *JTCVS Tech.* 2024;29:94-96. doi: 10.1016/j.xjtc.2024.11.013.
20. Kwon YIC, Keller M, Elhigzi K, Adibi I, Jones HC, Lai A, et al. Early outcomes of primary graft dysfunction comparing donation after circulatory and brain death heart transplantation: an analysis of the UNOS registry. *Clin Transplant.* 2025;39(7):e70222. doi: 10.1111/ctr.70222.
21. Williams AM, Ahmad A, McGann K, Wang CC, Bommarreddi S, Lima B, et al. Reanimation-less rapid recovery of a donor heart after circulatory death with prolonged 8-hour ischemic time. *J Heart Lung Transplant.* 2026;45(4):575-578. doi: 10.1016/j.healun.2025.09.009.
22. Williams AM, Trahanas J, Bommarreddi S, McGann KC, Ahmad A, Lima B, et al. Donation after circulatory death heart transplant without preimplant reanimation using rapid ultraoxygenated recovery. *JAMA.* 2026;335(10):885-893. doi: 10.1001/jama.2025.25169.

Funding: none.

Disclosure: the author has no conflict of interest to disclose.

Apical approach with double-lumen cannula for extracorporeal membrane oxygenation as a bridge to ventricular assist device. Alea Iacta est

Abordaje apical con cánula de doble lumen para oxigenación por membrana extracorpórea como puente a dispositivo de asistencia ventricular. Alea Iacta est

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ABSTRACT

Temporary mechanical circulatory support (MCS) is used in the treatment of cardiogenic shock. options for temporary MCS typically include intra-aortic balloon pump placement (IABP), impella, left ventricular assist device (LVAD) placement, and veno-arterial extracorporeal membrane oxygenation (VA-ECMO). Cannulation for VA-ECMO can be done either peripherally, or centrally. Central cannulation allows for antegrade arterial return and can be used when adequate flow cannot be achieved via peripheral cannulation, but it must be performed surgically. However, minimally-invasive techniques, such as transapical placement under the guidance of transesophageal echocardiography, have been described using the dual lumen cannula ProtekDuo®. This approach may be associated with decreased hemolysis, improved mobility, and early extubation. This technique has been used as a bridge to LVAD, as well as bridge to recovery, IABPs and Impella® both require access via either axillary or femoral artery. Our case describes the successful

RESUMEN

El soporte circulatorio mecánico temporal (SCM) se utiliza en el tratamiento del choque cardiogénico. Las opciones para el SCM temporal incluyen comúnmente el balón de contrapulsación intra-aórtico (BCIA), el Impella®, el dispositivo de asistencia ventricular izquierda (LVAD) y la oxigenación por membrana extracorpórea venoarterial (VA-ECMO). La canulación para VA-ECMO puede realizarse de manera periférica o central. La canulación central permite el retorno arterial en sentido anterógrado y puede emplearse cuando no se logra un flujo adecuado por vía periférica, aunque su colocación requiere intervención quirúrgica. Sin embargo, se han descrito técnicas mínimamente invasivas, como el abordaje transapical guiado por ecocardiografía transesofágica, utilizando la cánula de doble lumen ProtekDuo®. Este enfoque puede asociarse con menor hemólisis, mayor movilidad y extubación temprana. La técnica se ha utilizado como puente hacia el implante de un VAD, así como puente a la recuperación. Tanto el BCIA como el

How to cite: Orozco-Hernández EJ, Cozette KA, Restrepo-Cárdenas J, Wong R, Tallaj J, Tyndal JCM et al. Apical approach with double-lumen cannula for extracorporeal membrane oxygenation as a bridge to ventricular assist device. Alea lacta est. Cir Card Mex. 2026; 11 (3): 98-103. <https://dx.doi.org/10.35366/123483>

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Received: 16-09-2025. Accepted: 02-11-2025.

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minimally invasive placement and use of a Spectrum® cannula for central VA-ECMO in a patient who did not have adequate arterial access, as a bridge to LVAD, after which the patient achieved cardiac recovery.

Keywords: cardiogenic shock, dual lumen cannula, extracorporeal membrane oxygenation, heart failure.

Abbreviations:

CPB = cardiopulmonary bypass
IABP = intra-aortic balloon pump placement
LVAD = left ventricular assist device
MCS = mechanical circulatory support
TEE = transesophageal echocardiography
VA-ECMO = veno-arterial extracorporeal membrane oxygenation

Use of mechanical circulatory support (MCS) for cardiogenic shock has rapidly increased.¹ Most common initial MCS strategies entail institution of veno-arterial extracorporeal membrane oxygenation (VA-ECMO). For patients with anatomically small peripheral arteries or insufficient circulatory support, central cannulation techniques may be necessary. These invasive approaches are associated with increased risk of bleeding and other significant complications.² To minimize complications, many different techniques have been described including a mini left thoracotomy and direct apical cannulation for VA-ECMO using a ProtekDuo Cannula (Cardiac Assist Inc., LivaNova). This technique allows early ambulation and avoids peripheral artery access complications but has only been described in small case series.³ However, to our knowledge, this is the first case describing the use of the Spectrum® cannula (Spectrum Medical) for apical central ECMO as bridge to left ventricular assist device (LVAD).

CASE DESCRIPTION

A 27-year-old female with a history of dilated cardiomyopathy, peripartum cardiomyopathy resulting in heart failure with reduced ejection fraction, hypertension, and obesity with Body Mass Index (BMI) 40, presented with pneumonia and acute on chronic systolic heart failure. She experienced dyspnea, rapid labored breathing, and hypoxia. She was placed on nasal cannula. Chest X-ray showed bilateral infiltrates and pulmonary edema. The patient was intubated due to worsening respiratory distress and started on intravenous Lasix, milrinone, heparin infusion, propofol, and fentanyl. An arterial line connected to a FloTrac device recorded a cardiac output of 6.0 l/min, cardiac index of 3.3 l/min/m² and central venous pressure of 12 mmHg. Fevers developed, vancomycin and piperacillin/tazobactam were initiated. The patient was transferred for further management.

Impella® requieren acceso por la arteria axilar o femoral. Nuestro caso describe la colocación exitosa y mínimamente invasiva de una cánula Spectrum® para VA-ECMO central en una persona que no tenía acceso arterial adecuado, utilizándolo como puente hacia LVAD, tras lo cual se logró la recuperación cardíaca.

Palabras clave: choque cardiogénico, cánula de doble lumen, oxigenación por membrana extracorpórea, insuficiencia cardíaca.

Upon arrival, the patient was in cardiogenic shock and maintained on propofol and milrinone. Temperature was 38.4 °C, blood pressure 122/80 mmHg, with low ventilator settings and no acidosis and/or hypoxemia. Laboratory studies showed total bilirubin 7.4 mg/dl, direct bilirubin 4.3 mg/dl, ALT 74, LDH 356, BNP 135, troponin 67, WBC 14.4, PLT 134, D-dimer 2362, fibrinogen 575, and urinalysis negative for infection.

The patient initially declined transplant consideration due to negative family experience. Barriers included BMI, severity of illness with multi-organ dysfunction with possible pneumonia, and segmental/subsegmental pulmonary thromboembolism. In subsequent days, multiple vasopressors were required. Diminished lower extremity pulses bilaterally raised concern for acute limb ischemia. Echocardiography revealed marked stasis in the left ventricle “smoke” (*Fig. 1*) and femoral and axillary vessels measuring less than 5 mm. Given the lack of suitable peripheral cannulation sites, including axillary Impella® (Abiomed, Johnson & Johnson, Massachusetts, United States America), and in order to avoid sternotomy, the decision was made to transition the patient to apical central ECMO.

SURGICAL TECHNIQUE

The apex of the heart was identified. A small left anterior thoracotomy was performed. The pericardium was incised. A 14 mm Dacron Graft® (Maquet, New Jersey, United States America) was anastomosed with several mattress full thickness 4-0 Pledgeted Prolene® suture (Ethicon, Johnson & Johnson, New Jersey, United States America) at the apex. Heparin was administered, an 8 mm Dacron Graft® (Maquet, New Jersey, United States America) was anastomosed to the small right axillary artery, simultaneously, the right femoral vein was cannulated with Seldinger Technique with a 25 Fr long venous cannula Multihole® (Medtronic, Minneapolis, United States America) in preparation for cardiopulmonary bypass (CPB). Under transesophageal echocardiography (TEE) guidance, the apical myocardium inside the graft was accessed with a needle. Using the Seldinger technique, a 0.035” Glidewire® (Terumo Cardiovascular Systems Corp., Colorado, United States America) was placed into the left

ventricle and passed antegrade through the aortic valve into the ascending aorta. A 5 Fr Berenstein exchange catheter® (Cordis, Florida, United States America) was placed and was exchanged for an 0.035" Lunderquist guidewire® (Cook Medical, Indiana, United States America). After serial dilations until the dilator 29 Fr, A 32F chest tube was placed inferior to the thoracotomy at the level of the anterior axillary line, making a muscular tunnel to allow passage of the Lunderquist Guidewire® (Cook Medical, Indiana, United States America). The chest tube was removed. A 31 Fr Spectrum® cannula (Spectrum Medical, South Carolina, United States America) was advanced over the guidewire (across the tunnel). Cannula positioning was guided by TEE, the two sets of inflow drainage orifices were visualized within left ventricle under TEE (Fig. 2). The most proximal drainage orifice was located immediately above aortic valve (Fig. 3) After adequate positioning of the inflow and outflow ports (aortic arch area) (Fig. 4) was confirmed, MCS was initiated successfully. Adequate biventricular function on central ECMO via the Spectrum® cannula (Spectrum Medical, South Carolina, United States America) was verified on TEE and the CPB was weaned.

She was now in INTERMACS 1 on central VA Apical ECMO (Fig. 5) (Fig. 6). After eight days, LVAD Heartmate 3® (Abbot, Illinois, United States America) was implanted, complicated by right ventricular failure and acute kidney injury, she required two days later a Protekduo® (Cardiac Assist, LivaNova, London, United Kingdom) as "oxy-right ventricular assist device", and decannulated for improvement nine days later. For the acute kidney injury she was on hemodialysis, with renal recovery. She has been stable, without complications related to the LVAD, now as a bridge to decision, according with her evolution, bridge to transplant can be the next step.

COMMENT

This case report introduces a novel, minimally invasive surgical technique for central VA-ECMO, using the apical placement of a Spectrum cannula (Spectrum Medical) via a left thoracotomy. This approach successfully served as a bridge to a durable implantable LVAD in a patient for whom conventional temporary mechanical circulatory support devices, such as the Impella® platform or peripheral VA-ECMO, were not viable.

MCS is used in the treatment of cardiogenic shock in order to improve hemodynamics when initial therapy has failed to achieve adequate tissue perfusion.⁴ Cannulation for VA-ECMO can be done either peripherally, with venous drainage from the femoral or internal jugular vein and



Figure 2: Spectrum cannula with the aortic valve open, showing drainage orifices located beneath the valve.

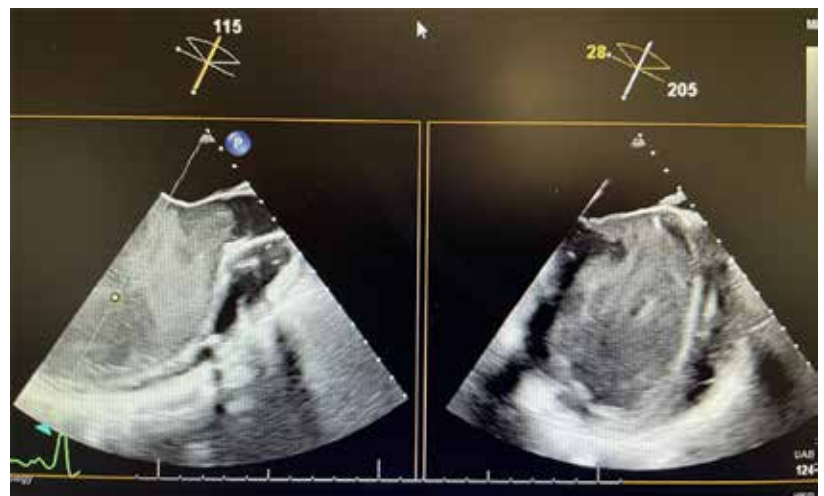


Figure 1:

Pronounced stasis on left ventricle with imminent thrombus formation.

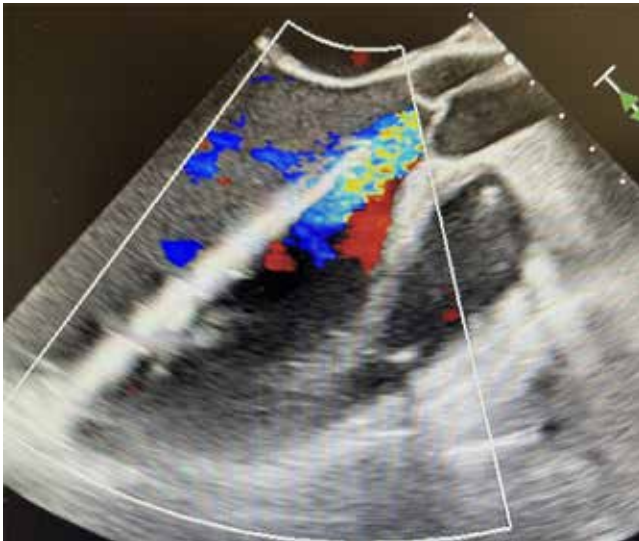


Figure 3: Doppler color imaging of drainage orifices beneath the aortic valve.



Figure 4: Doppler color imaging of distal delivery orifices in the aortic arch.

arterial return access through the femoral or axillary artery, or centrally, with direct cannulation of the aorta and the right atrium.^{5,6} Central cannulation allows for antegrade arterial return and can be used when adequate flow cannot be achieved via peripheral cannulation, but it must be performed surgically, and is usually done through median sternotomy.⁷ However, minimally invasive techniques, such as transapical placement under the guidance of TEE, have been described for central cannulation using devices such as the ProtekDuo® (Cardiac Assist, LivaNova, London, United Kingdom).⁸⁻¹⁰ Additionally, the use of transapical dual-lumen cannula may be associated with decreased hemolysis, improved mobility,

and earlier freedom from mechanical ventilatory support.¹¹ This technique has been used successfully as a bridge to durable implantable LVAD placement as well as a bridge to recovery.^{12,13} Other short-term mechanical circulatory support devices include IABPs, as well as intravascular microaxial left ventricular assist devices, such as Impella® platform devices.^{14,15} However, IABPs and Impella® both require access via either the axillary or the femoral artery. Our case describes the successful minimally invasive placement and use of a Spectrum® cannula (Spectrum Medical, South Carolina, United States America) for central VA-ECMO in a patient who did not have adequate access, as a bridge to LVAD, after which the patient achieved cardiac recovery.

Capelli published the largest case series to date documenting the safety and efficacy of transapical VA-ECMO cannulation with the ProtekDuo cannula® (Cardiac Assist, LivaNova, London, United Kingdom). Our surgical technique followed the same steps and principles.⁸ As the ProtekDuo cannula® (Cardiac Assist, LivaNova, London, United Kingdom), the Spectrum® cannula (Spectrum Medical, South Carolina, United States America) was designed for Right ventricular Support. However, it introduces an important modification, this cannula is designed with an additional set of orifices to ensure more complete and more effective right ventricular decompression. This is particularly relevant given that the ProtekDuo cannula® (Cardiac Assist, LivaNova, London, United Kingdom) is no longer available, making our technique a critical alternative for managing complex cases. Some multiples reports showed the use of the spectrum® cannula (Spectrum Medical, South Carolina, United States America) as Right Ventricular Support.¹⁶⁻¹⁸



Figure 5: Postoperative chest X-ray.

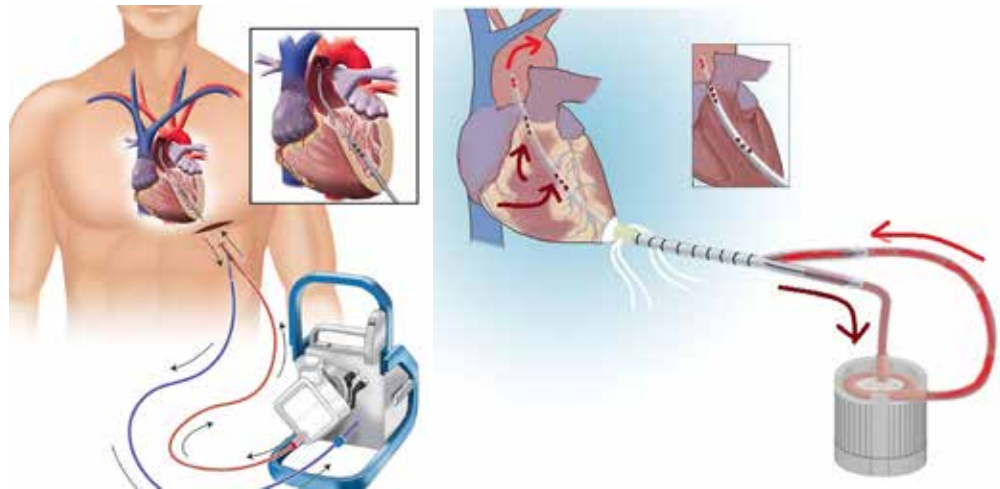


Figure 6:

Diagram illustrating the fundamental operation of central extracorporeal membrane oxygenation using the apical dual lumen spectrum cannula.

However, the apical technique provides Left ventricular decompression with optional biventricular support and respiratory support. The successful application of this method allowed the patient to be supported for approximately one week, a timeframe that allowed for clinical stabilization and decision-making regarding destination therapy. This highlights the viability of this minimally invasive central VA-ECMO approach as a durable bridge to either recovery or long-term mechanical support. Furthermore, the technique avoids the risk of aortic insufficiency and intravascular hemolysis often associated with other support devices. By facilitating ambulation and early freedom from mechanical ventilation, it may also contribute to improved patient outcomes and quality of life. To our knowledge, this represents the first reported case of using a Spectrum® cannula (Spectrum Medical, South Carolina, United States America) for minimally invasive central VA-ECMO as a bridge to a durable mechanical circulatory support device. This technique should be considered in the therapeutic repertoire of temporary circulatory support strategies, especially in situations where other methods are not feasible due to unfavorable anatomy, inadequate flow, or significant hemolysis. In complex and seemingly intractable clinical scenarios. “*Alea iacta est*”, this quote by Julius Caesar describes the moment when an irrevocable decision is made and a point of no return is crossed, implying that there is no turning back and one must face whatever comes next. However, this approach offers a viable and effective solution.

CONCLUSIONS

This novel central cannulation technique using an apical placement of a Spectrum cannula for VA-ECMO provides LV decompression with optional biventricular support and respiratory support to facilitate ambulation while avoiding

many of the risks of traditional cannulation, this unique case was bridge to LVAD, however, bridge to decision, transplant or recovery are some potential therapeutic goals. It has been demonstrated that this technique with ProtekDuo cannula was used safely when there is not any other option for MCS. Further rigorous studies are warranted to continue documenting outcomes using this novel Spectrum cannula before its use can become widespread.

REFERENCES

1. Shah M, Patnaik S, Patel B, et al. Trends in mechanical circulatory support use and hospital mortality among patients with acute myocardial infarction and non-infarction related cardiogenic shock in the United States. *Clin Res Cardiol.* 2018;107(4):287-303.
2. Takeda K, Garan AR, Ando M, et al. Minimally invasive CentriMag ventricular assist device support integrated with extracorporeal membrane oxygenation in cardiogenic shock patients: a comparison with conventional CentriMag biventricular support configuration. *Eur J Cardiothorac Surg.* 2017;52(6):1055-1061.
3. Rao P, Alouidor B, Smith R, Khalpey Z. Ambulatory central VA-ECMO with biventricular decompression for acute cardiogenic shock. *Catheter Cardiovasc Interv.* 2018;92(5):1002-1004.
4. Nakata J, Yamamoto T, Saku K, Ikeda Y, Unoki T, Asai K. Mechanical circulatory support in cardiogenic shock. *J Intensive Care.* 2023;11(1):64. Available in: <https://doi.org/10.1186/s40560-023-00710-2>
5. Dangel M, Albosta M, Butros H, Loebe M. Temporary mechanical circulatory support: left, right, and biventricular devices. *Curr Cardiol Rev.* 2023;19(5):27-42. Available in: <https://doi.org/10.2174/1573403X19666230314115853>
6. Jayaraman AL, Cormican D, Shah P, Ramakrishna H. Cannulation strategies in adult veno-arterial and veno-venous extracorporeal membrane oxygenation: Techniques, limitations, and special considerations. *Ann Card Anaesth.* 2017;20(Supplement):S11-S18. Available in: <https://doi.org/10.4103/0971-9784.197791>
7. Pavlushkov E, Berman M, Valchanov K. Cannulation techniques for extracorporeal life support. *Ann Transl Med.* 2017;5(4):70. Available in: <https://doi.org/10.21037/atm.2016.11.47>
8. Cappelli J, Emling J, Edwards A, Babu A. Direct apical cannulation with protek duo rapid deployment cannula via mini

- thoracotomy for ambulatory venoarterial-extracorporeal membrane oxygenation. *ASAIO J.* 2024;70(7):565-569. doi: 10.1097/MAT.0000000000002157. Available in: <https://doi.org/10.1097/MAT.0000000000002157>
9. Ravipati HL, Nooli NP, Hoopes CW, et al. A novel method of transapical ProtekDuo rapid deployment cannula as temporary left ventricular assist device. *J Surg Case Rep.* 2023;2023(6):rjad246. Available in: <https://doi.org/10.1093/jscr/rjad246>
 10. Belani K, Saikus CE, Schroder JN, Klinger RY. Transapical ProtekDuo rapid deployment cannula as temporary left ventricular assist device in a Jehovah's witness patient. *J Cardiothorac Vasc Anesth.* 2021 Dec;35(12):3735-3742. Available in: <https://doi.org/10.1053/j.jvca.2020.12.009>
 11. Singh R, Chandel A, Paras J, et al. Transapical cannulation with a dual lumen cannula for mechanical circulatory support in cardiogenic shock. *ASAIO J.* 2022;68(11):e215-e219. Available in: <https://doi.org/10.1097/MAT.0000000000001683>
 12. Ghannam AD, Babu AN, Jeng EI. Protek Duo Rapid Deployment cannulation utilized to bridge to durable heartware left ventricular assist device. *J Card Surg.* 2020;35(8):2067-2069. Available in: <https://doi.org/10.1111/jocs.14775>
 13. Lima B, Hufton AD, Hussain ST. Minimally invasive off-pump technique for temporary left ventricular support. *ASAIO J.* 2022;68(1):e1-e4. Available in: <https://doi.org/10.1097/MAT.0000000000001404>
 14. Wong ASK, Sin SWC. Short-term mechanical circulatory support (intra-aortic balloon pump, Impella, extracorporeal membrane oxygenation, TandemHeart): a review. *Ann Transl Med.* 2020;8(13):829. doi: 10.21037/atm-20-2171.
 15. Zein R, Patel C, Mercado-Alamo A, Schreiber T, Kaki A. A review of the Impella devices. *Interv Cardiol.* 2022;17:e05. doi: 10.15420/icr.2021.11.
 16. Usman AA, Spelde AE, Cevasco M, et al. Technical considerations for percutaneous pulmonary artery cannulation for mechanical circulatory support. *JTCVS Tech.* 2023;18:65-73.
 17. Usman AA, Spelde AE, Olia SE, et al. J. First-in-man successful use of the SPECTRUM percutaneous dual-stage right ventricle and right atrium to pulmonary artery ventricular assist device. *J Card Surg.* 2022;37(10):3403-3407.
 18. Spelde AE, Hernandez-Morgan ME, Augoustides JG. Advances in mechanical support for right ventricular failure. *J Cardiothorac Vasc Anesth.* 2022;36(8):3289-3291.

Funding: none.

Disclosure: the authors have no conflict of interest to disclose.

Cardiac intimal sarcoma in the right atrium.

Case report and literature review

Sarcoma intimal cardíaco en aurícula derecha. *Reporte de caso y revisión de la literatura*

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ABSTRACT

Primary cardiac intimal sarcoma is a very rare subtype of cardiac tumor that is often misdiagnosed. It can present in several ways and is known to mimic other conditions. Due to the advances in immunohistochemistry of the cardiac tumors each day is more frequent the diagnosis of cardiac intimal sarcoma. We present the case of a 42-years old patient, who was found to have a right atrial mass during the protocol of acute dyspnea in the emergency room, and after further investigation with enhanced computed tomography imaging confirmed the presence of right atrial mass that rule out the diagnosis of myxoma or cardiac thrombus. The objective of this article is to present the first case report in the country of the authors of an intimal cardiac sarcoma in the right heart and provide a comprehensive analysis of the medical literature about these rare primary cardiac sarcomata, including details on its pathogenesis, clinical presentation, current treatments, prognosis, and follow-up.

Keywords: heart surgery, dyspnea, cardiac sarcoma, immunohistochemistry, Murine Double Minute 2 Protein, genetic testing.

RESUMEN

El sarcoma intimal cardíaco primario es un subtipo muy raro de tumor cardíaco, que a menudo se diagnostica erróneamente porque este tumor cardíaco puede presentarse de diversas maneras y se sabe que es un excelente imitador de otras afecciones. Debido a los avances en la inmunohistoquímica de los tumores cardíacos, el diagnóstico de sarcoma intimal cardíaco es cada vez más frecuente. Presentamos el caso de un paciente de 42 años, que se encontró con una masa auricular derecha durante el protocolo de disnea aguda en el servicio de urgencias, y tras una investigación adicional con tomografía computarizada mejorada confirmó la presencia de una masa auricular derecha que descarta el diagnóstico de mixoma o trombo cardíaco. El objetivo de este artículo es presentar el primer caso diagnosticado de sarcoma cardíaco intimal del corazón derecho en el país de los autores y proporcionar una revisión exhaustiva de la literatura médica sobre estos sarcomas cardíacos primarios raros, incluyendo detalles sobre su patogénesis, presentación clínica, tratamientos actuales, pronóstico y seguimiento.

Palabras clave: cirugía cardíaca, disnea, sarcoma cardíaco, inmunohistoquímica, proteína MDM2, pruebas genéticas.

How to cite: Rendón-Elías FG, Castillo-Romero C, Leyva-Villegas J, Salas-Ríos CM, Puentes U, Carranza-Hernández A et al. Cardiac intimal sarcoma in the right atrium. Case report and literature review. Cir Card Mex. 2026; 11 (3): 104-110. <https://dx.doi.org/10.35366/123484>

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Received: 06-10-2025. Accepted: 27-11-2025.

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Abbreviations:

CT = Computed Tomography
MDM2= Murine Double Minute 2 Protein
TEE = Transesophageal Echocardiography
TTE = Transthoracic Echocardiography
UEAs = Ultrasound-Enhancing Agents

PPrimary cardiac tumors are very uncommon, can be primary or metastatic, occurring in about 0.001-0.3% of autopsies¹ and diagnosis may be difficult, as symptoms and cardiac imaging may mimic other cardiac diseases. They can be classified into three clinicopathological groups: 1) benign congenital tumors; 2) benign acquired tumors; and 3) malignant tumors.² Nearly 75-90% of primary cardiac tumors are benign (myxoma the most common), and 10-25% are malignant (angiosarcoma is the most common type of sarcoma). Cardiac sarcomas account for 75-95%, with a severe prognosis, having a five-year survival rate of 14%.³ Generally, sarcomas represent a heterogeneous group underlying a variety of aetiologies and anatomical features.⁴ Intimal sarcomas are extremely rare mesenchymal tumors and is a subtype of sarcoma that occurs more commonly in the great vessels and pulmonary veins but rarely involves the heart and less common in the right heart.⁵

The objective of this article is to present the first case report in the country of the authors of an intimal cardiac sarcoma in the right heart and provide a comprehensive analysis of the medical literature about these rare primary cardiac sarcomata, including details on its pathogenesis, clinical presentation, current treatments, prognosis, and follow-up.

CASE REPORT

A 42-year-old male patient with past medical history of hypertension, non-insulin dependent diabetes mellitus, obesity, dyslipidemia, history of smoking (eight packs/year), and family history of colon and breast cancer, was

referred to our emergency room for evaluation of sudden rest dyspnea without thoracic pain accompanied by constitutional symptoms. The patient referred a two-month history of progressive worsening effort-related dyspnea, fatigue and dry cough. On admission, he was unstable, with an arrhythmic heart rate (150 bpm), respiratory rate of 30 breaths/minute, and body temperature of 36.5 °C, the blood pressure and the peripheral oxygen saturation were not possible measure (because of the arrhythmia). The electrocardiogram showed atrial fibrillation with high ventricular response that was treated by cardioversion. With the patient hemodynamically stable a transthoracic echocardiography was performed, which revealed presence of a well-defined right atrial mass of approximately 7 × 5 cm, attached to the auriculo-ventricular septum prolapsing to the right ventricle (*Fig. 1*). The patient was subsequently referred for a contrast enhanced computed tomography (CT) scan to further visualize the mass and rule out pulmonary embolism. The CT revealed a well-circumscribed mass within the right atrium, measuring 7 × 5 cm fixed in the auriculo-ventricular septum near the tricuspid valve which was partially occluded by the mass and pass through it to the right ventricle (*Fig. 1*), pericardial and peritoneal effusion accompanied it.

The patient was subsequently translated to intensive care unit and presented to cardiac surgery team that decided to program the patient to realized resection of the cardiac mass. An intraoperative transesophageal echocardiography performed prior to placing the patient on Cardiopulmonary Bypass (CPB) revealed a 6 × 10 cm, right atrial mass attached to the septum interatrial that prolapsed the tricuspid valve.

In the operating room after a median longitudinal sternotomy, cardiopulmonary bypass under normothermia was routinely instituted and Del Nido cardioplegic solution was administered via the aortic root after aortic cross clamping. A right atriotomy was used to expose and realize the resection of the cardiac tumor. The tumor was glistening, white, hard,



Figure 1: Preoperative echocardiogram showing the tumor in the right atrium (crisscrossing lines) prolapsing to the right ventricle (A). Computed tomography image demonstrating a large non-enhancing, irregularly shaped mass with homogenous central hypodensity, originating from the fossa ovalis (asterisk) and protruding into the ventricle (B). Intraoperative photograph of the excised specimen (C).

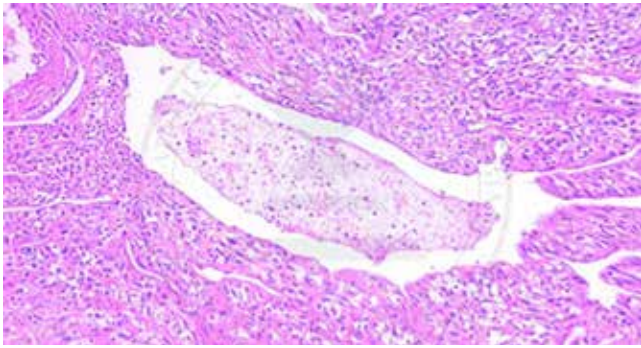


Figure 2: A delineated endothelial space is observed, surrounded by pleomorphic epithelioid and spindle cells (hematoxylin and eosin, $\times 200$).

smooth, not lobed, adherent to auriculo-ventricular septum, and prolapsing to tricuspid valve. The tumor was resected circumferentially *en bloc*. The tumor did not appear to invade another cardiac chamber, the myocardium and any major vessel. The patient during the postoperative period showed an atrial fibrillation and right heart failure that was treated without complications.

The patient's resected cardiac tumor was sent to pathologic examination in formalin. The macroscopy appearance of the resected tumor was ovoid, smooth surface, light brown color, hard consistency, measured $7 \times 5 \times 5$ cm and weight of 100 grams. On section, the tumor was solid-cystic, light brown and areas of hemorrhage (Fig. 1).

Microscopically, the mass was composed of atypical spindle cells disclosing the presence of sarcoma with areas of necrosis and chondroid differentiation. Immunohistochemical analysis revealed positive expression of Murine Double Minute 2 Protein (MDM2) and focal positivity for smooth muscle actin and sarcomeric actin in neoplastic cells. Combined morphological, immunological and molecular studies performed on the resection specimen were consistent with a diagnosis of primary cardiac intimal sarcoma (Fig. 2) (Fig. 3).

The postoperative course was uneventful, and the patient was discharged home on the 11th postoperative day and immediately started the oncologic protocol. Seven months after the surgery the patient does not present tumoral activity, Eastern Cooperative Oncology Group (ECOG) 1; Visual Analogue Scale for pain (VAS) 0, and received six doses of chemotherapy with Adriamycin.

COMMENTARY

Cardiac tumors have been reported since the 16th century however due to the incidence of malignant cardiac tumors are a very rare, they still pose a unique diagnostic and therapeutic challenge. Cardiac masses and tumors have

always fascinated pathologists and cardiovascular physicians/surgeons alike. Before the mid-1950s, primary cardiac tumors were usually reported at autopsies. With the development of echocardiography, the imaging and diagnosis of tumors became a possibility. With advancements in cardiopulmonary bypass, their resection and thus a cure became an exciting reality. The first successful resection of a cardiac tumor was carried out by Crafoord in 1954,⁶ but the experience in treating these aggressive and lethal tumors is minimal and management protocols are not well defined.

Primary cardiac tumors are rare; the reported prevalence is between 0.0017 and 0.028%⁷ that has increased in recent decades. Primary cardiac tumors occur along a pathologic spectrum that includes benign, malignant, and intermediate with uncertain biological behavior. Cardiac sarcomas affect more men than women at a 2.5:1 ratio, usually presenting in the third and fourth decades of life.⁸

In 2015, the World Health Organization (WHO) wrote the classification of cardiac neoplasms including benign tumors, tumor-like lesions, malignant tumors, and pericardial tumors. Cardiac tumors are divided into primary and secondary forms.⁹ Updates to each of the main categories of cardiac tumors were made in the fifth edition in 2021,¹⁰ resulting in some entities being combined with others and some being newly established. Malignant primary cardiac neoplasms have been an area of growing controversy in the past decade. As detailed subsequently, the entities of intimal sarcoma and undifferentiated pleomorphic sarcoma have been formally separated, reflecting a better understanding of their biology and a better reflection of their anatomical sites of occurrence. Intimal sarcoma has been relocated to the Lung Section of the Thoracic Volume and the Intimal Sarcoma chapter of the WHO Classification of Soft Tissue and Bone Tumors.¹¹ Although many sarcomas have been described in the heart, only a few are encountered with regularity.

Approximately 10% of primary cardiac tumors are malignant and 90% benign.¹² Myxomas account for

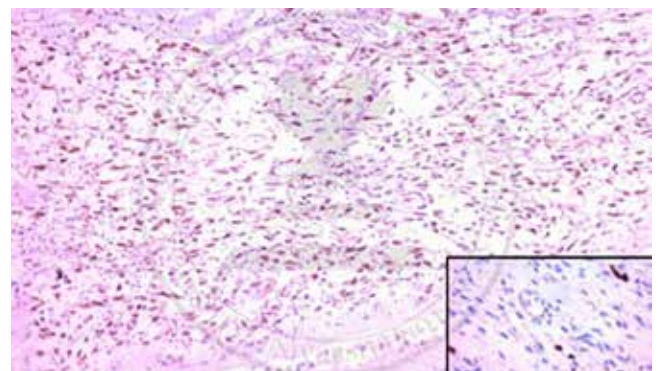


Figure 3: Murine double minute 2 protein immunohistochemistry marker positive.

approximately 50% of all benign cardiac tumors in adults and only for a small percentage in children. Rhabdomyoma is the most common benign tumor in children, accounting for 40 to 60% of the cases. Other benign cardiac tumors that have been described include fibromas, lipomas, hemangiomas, papillary fibroelastomas, cystic tumors of the atrioventricular node, and paragangliomas. The remaining 10-20% of primary cardiac tumors are malignant and usually are pathologically described as sarcomas.¹³

Primary cardiac sarcomas constitute approximately 1% of all soft tissue sarcomas and are the most common malignant primary cardiac tumor.¹⁴ Angiosarcomas and unclassified sarcomas account for approximately 76% of all cardiac sarcomas, of which angiosarcomas are the most common. Rhabdomyosarcoma is the most common form of cardiac sarcoma in children. Leiomyosarcoma, synovial sarcoma, osteosarcoma, fibrosarcoma, myxoid sarcoma, liposarcoma, mesenchymal sarcoma, neurofibrosarcoma, and malignant fibrous histiocytoma are other cardiac sarcomas observed. Most cardiac sarcomas have been published as single case reports or institutional small series, with only a few studies examining large sample sizes.¹⁵

Different histological subtypes of cardiac sarcomas include, but are not limited to angiosarcoma, leiomyosarcoma, fibrosarcoma, rhabdomyosarcoma, synovial sarcoma, osteosarcoma, undifferentiated pleomorphic sarcoma and intimal sarcoma.

Intimal sarcoma is a mesenchymal tumor that is more commonly encountered in the great vessels and pulmonary veins. It rarely involves the heart and less frequent the right atrium. To our knowledge, our case is the first cardiac intimal sarcoma reported in our country and around the world no more than thirty cases have been reported till date.¹⁶

Intimal sarcoma are poorly differentiated malignant tumors characterized by the formation of tightly packed spindle shaped cells with fascicular growth pattern, and often show areas of necrosis. These tumors can frequently resemble smooth muscle neoplasms such as leiomyosarcoma. They exhibit variable degrees of necrosis, cellular atypia and nuclear polymorphisms and rarely show areas with morphological features like angiosarcoma, rhabdomyosarcoma, and osteosarcoma. It was believed that angiosarcoma and undifferentiated pleomorphic sarcoma were the most common cardiac sarcomas but in recent study, intimal sarcoma has been reported as the most frequently occurring primary cardiac sarcoma (42%) with MDM2 gene amplification being its characteristic pathological feature. Immunohistochemical analyses of these tumors typically show positive reactivity for MDM2, osteopontin and vimentin. Variable positivity is seen for SMA, desmin, CD117, CD68, P53 and BCL-2. CD31, CD34, and factor VIII are typically negative, but may be positive in areas with angiosarcomatous differentiation.¹⁷⁻¹⁹

The cardiac tumors may be symptomatic or found incidentally during evaluation for seemingly unrelated problem or physical finding. Symptoms are usually related to its cardiac location producing mass effect interfering with myocardial function or blood flow, causing arrhythmias, cardiac valves dysfunction, pericardial effusion with or without tamponade, and these are manifested by dyspnea, chest discomfort, pre-syncope, or syncope. Also, may be present like systemic signs and symptoms (fever, arthralgias, weight loss, fatigue among others) and thromboembolic pulmonary or systemic. Cardiac sarcoma can present in several ways and are known to be great mimickers of other conditions. Patients may often remain asymptomatic until advances stages or present with dyspnea, orthopnea, paroxysmal nocturnal dyspnea, or lower extremity edema. In our case, the patient had a history of dyspnea, and dry cough and the mass was seen on transthoracic echocardiography that was performed as a part of the emergency protocol for acute dyspnea and chest pain.^{20,21}

For an appropriate diagnostic approach, the first step is differentiating between cardiac tumors and other cardiac masses such as thrombi or vegetations. Four steps should be followed to orient the diagnosis:²¹ 1) the clinical setting provides critical diagnostic clues in helping establish the etiology of a cardiac mass or lesion and may be able to realize the differential diagnosis (cardiac thrombi secondary to atrial fibrillation, myocardial infarction or infective endocarditis);²²⁻²⁷ 2) the tumor location, the intracardiac tumors can arise in every cardiac cavity or in every cardiac wall, even though each type of cardiac tumor prefers a well-defined location; 3) the histology-based likelihood and the age of the patient at the time of presentation; 4) the morphological and functional characteristics: size, shape, mobility, tissue characterization, vascular supply, and metabolic activity. Points 3 and 4 may be investigated using different imaging techniques: echocardiography, CT, magnetic resonance imaging, and positron emission tomography/computed tomography. Each technique has advantages and disadvantages in terms of costs, availability, and diagnostic power: often they must be used in combination.^{28,29}

The role of multimodality imaging is very important and crucial in planning further evaluation and management. The goals of the initial evaluation are to ascertain the presence of a cardiac tumor, the location of the lesion within cardiac structures, and when possible, whether a tumor is benign or malignant.

Two-dimensional Transthoracic Echocardiography (TTE) is often the first diagnostic approach in terms of imaging modality because of its wide availability, low cost, and portability. TTE allows for the assessment of size, location, mobility, understanding the hemodynamic pattern provoked by the tumor and the pericardial involvement of a tumor.

The limitations of TTE include poor acoustic windows, particularly in obese patients (like our patient) and those with chronic lung disease,³⁰ and lack of potential for tissue characterization. In addition, the extent and origin of the mass may not be distinguishable by echocardiography if it arises from outside of standard imaging views (e.g., in the superior vena cava or inferior vena cava or branch pulmonary vessels).

Transesophageal Echocardiography (TEE) is commonly used when a valvular lesion is suspected, particularly in patients with atrial masses or with mobile valvular lesions and is necessary to better characterize a cardiac tumor in terms of size, morphology, attachment site, extension, and hemodynamic affects. When used in combination with real-time three-dimensional echocardiography, TEE offers incremental value for the evaluation of intracardiac masses by providing a more accurate assessment of the anatomical relationships, size, and shape of the mass.³¹

The use of Ultrasound-Enhancing Agents (UEAs) in echocardiography is an important tool for the assessment of myocardial perfusion and can also be used for the evaluation of the relative perfusion of a cardiac mass. It allows for improved definition of intracavity structures and assessment of vascularity.³² The difference in perfusion of cardiac masses may help distinguish between vascular and nonvascular tumors or thrombus because there are both qualitative and quantitative differences in perfusion among the various pathologies.³³ For example, malignant tumors, which are highly vascular due to abnormal neovascularization, demonstrate greater enhancement than the adjacent myocardium. In contrast, benign tumors (myxomas) often have poor blood supply and therefore demonstrate lower perfusion on visual inspection and have quantitatively less perfusion than the surrounding myocardium. Because thrombi are avascular, they do not perfuse with UEAs echocardiography. Therefore, UEAs echocardiography perfusion imaging may aid in the early identification of thrombi and guide subsequent diagnostic and/or treatment strategies.

Once a cardiac mass or tumor is suspected, patients may be referred for cardiovascular magnetic resonance, imaging study that offer a complete multiplanar and noninvasive evaluations of the mass (vascularity, adjacent infiltration) and its potential involvement with cardiac chambers and pericardium; because it also provides information about extracardiac structures and its surrounding anatomy, this often proves useful in surgical planning.³⁴

Cardiac computed tomography has become increasingly used modality for the assessment of cardiac masses, especially when other imaging modalities are nondiagnostic or contraindicated, and is also useful in tumor staging because of its ability to detect metastases in cases suspected malignancies.³⁵

In our case the diagnostic approach was to discard pulmonary embolism because of the clinical presentation

(acute exacerbation of dyspnea and atrial fibrillation), and the most practical approach was to realize a TTE in the emergency and when the patient was more stable was taken to perform an CT. with this modality of images study the medical and surgical plan was designed. After the surgical procedure and with the histopathological diagnosis of intimal cardiac sarcoma the patient was sent to realize and CT and positron emissions tomography for staging malignancies.

Currently, there are no specific treatment guidelines for primary cardiac intimal sarcomas because of the rarity of these cases and their heterogeneous spectrum.³⁶ Surgical removal of the tumor (potentially associated with auto-transplantation or heart transplant) is still the best treatment with radical intent, in small tumors without loco-regional/distant spreading or severe impairment of cardiac function.³⁷⁻⁴¹ Nonetheless, the lack of symptoms may delay the diagnosis as the patient might remain for a prolonged period only under primary health care surveillance and thus, the opportunity window for a successful intervention might be missed. Additionally, chemotherapy, radiotherapy, immunotherapy, or molecular treatments with genomic agents are used as adjuvant/neoadjuvant lines, as an alternative to surgery, in addition to complete/incomplete tumor removal or in recurrent postoperative cases.⁴²⁻⁴⁵

The prognosis of cardiac intimal sarcoma is generally poor. Factors that are associated with worse prognosis include necrosis, high mitotic count, and metastasis. Another important factor determining the prognosis is the anatomical location of the tumor within the heart (intracavitary vs intramural). It has also been found that the right-side tumors carry a worst prognosis than left sided tumors.^{20,46,47}

Due to the frequent involvement of vital structures and early metastasis (up to 50% of patients at diagnosis), cardiac sarcomas are associated with high mortality and morbidity. Intimal sarcomas are highly aggressive with the mean survival being three months to one year, although survival up to 11 years has been reported. Patients with complete tumor resection are known to live twice as long as those without the surgical resection. Thus, early diagnosis and complete resection are of paramount importance in the management of intimal sarcomas.⁴⁸⁻⁵⁰

Although the achievement of tumor free margins with surgical resection is the mainstay of treatment in cardiac sarcomas and associated with improved survival, it is important to understand that surgical resection may not be possible in almost 50% of the cases due to the involvement of vital structures. However, in a recent study, about 65% of the patients with planned surgical management could undergo complete resection. Furthermore, local recurrence and metastasis occur frequently and early, usually within one year. Thus, there is a role for other treatment modalities such as chemotherapy and radiation therapy in the management of cardiac sarcomas. Ifosfamide-epirubicin

(or doxorubicin) and CyVADIC (cyclophosphamide, vincristine, doxorubicin, and dacarbazine) are the two main regimens used in adult soft tissue sarcomas and may confer survival advantage in patients with cardiac sarcomas as well.⁵¹ Two meta-analysis have demonstrated that adjuvant chemotherapy improves the time to local and distant recurrence as well as the overall survival.^{52,53} The addition of ifosfamide further improves these benefits, but these should be weighed against the toxic effects arising from its use. Gemcitabine based chemotherapy regimen (single agent or in combination with docetaxil) may also be tried in patients with cardiac sarcomas based on the favorable response to these drugs in various trials evaluating their role in the treatment of soft tissue sarcomas.⁵⁴ Apart from the conventional chemotherapeutic regimens, there could also be a role for tyrosine kinase inhibitor pazopanib in the treatment of advanced disease, especially in those with poor response to the abovementioned drugs. Pazopanib was approved in the United States in 2012 for treating patients with advanced soft tissue sarcomas who had received prior chemotherapy, based on the data from PALETTE trial.⁵⁵

CONCLUSIONS

Cardiac intimal sarcoma is a rare, but aggressive disease entity with poor prognosis. Owing to its variable presentation, diagnosing requires a high degree of suspicion while encountering patients with characteristic findings on the imaging studies. Although surgical excision with tumor free margins is the mainstay of treatment, complete surgical excision may often not be possible because of the involvement of the vital structures. Owing to this entity could be very challenging, the high rates of recurrence and metastasis, adjuvant chemotherapy must be used in all the patients diagnosed with this disease. Finally, it cannot be stressed enough that early diagnosis and prompt treatment are extremely crucial in improving outcomes in patients with this tumor.

REFERENCES

- Raphael KL, Martinez AP, Clements SD, Isiadinso I. Role of multimodal cardiac imaging in diagnosing a primary intimal sarcoma of the left atrial appendage. *Tex Heart Inst J*. 2019;46(1):28-31. doi: 10.14503/THIJ-16-5896.
- Moeri-Schimmel R, Pras E, Desai I, Krol S, Braam P. Primary sarcoma of the heart: case report and literature review. *J Cardiothorac Surg*. 2020;15(1):104. doi: 10.1186/s13019-020-01157-4.
- Thuraisingam A, Fletcher N, McKay G. Primary cardiac intimal sarcoma. *Ann Surg Case Rep*. 2019;2(1):1013. doi: 10.1016/j.hlc.2019.02.113.
- Valecha G, Pau D, Nalluri N, Liu Y, Mohammad F, Atallah JP. Primary intimal sarcoma of the left atrium: an incidental finding on routine echocardiography. *Rare Tumors*. 2016;8(4):6389. doi: 10.4081/rt.2016.6389.
- Nomoto N, Tani T, Konda T, Kim K, Kitai T, Ota M, et al. Primary and metastatic cardiac tumors: echocardiographic diagnosis, treatment and prognosis in a 15-years single center study. *J Cardiothorac Surg*. 2017;12(1):103. doi: 10.1186/s13019-017-0672-7.
- Chitwood WR Jr. Clarence Crafoord and the first successful resection of a cardiac myxoma. *Ann Thorac Surg*. 1992;54(5):997-8. doi: 10.1016/0003-4975(92)90676-u.
- Patel J, Sheppard MN. Pathological study of primary cardiac and pericardial tumors in a specialist UK centre: surgical and autopsy series. *Cardiovasc Pathol*. 2010;19:343-352. doi: 10.1016/j.carpath.2009.07.005.
- Oliveira GH, Al-Kindi SG, Hoimes C, Park SJ. Characteristics and survival of malignant cardiac tumors: a 40-year analysis of > 500 patients. *Circulation*. 2015;132:2395-2402. doi: 10.1161/CIRCULATIONAHA.115.016418.
- International Agency for Research on Cancer, World Health Organization (WHO), International Academy of Pathology. WHO classification of tumours of the lung, plura, thymus and heart. 4a ed. Travis WD, editor. IARC; 2015.
- Maleszewski JJ, Basso C, Bois MC, Glass C, Klarich KW, Leduc C, et al. The 2021 WHO Classification of Tumors of the Heart. *J Thorac Oncol*. 2022;17(4):510-518. doi: 10.1016/j.jtho.2021.10.021.
- Thoracic tumours: WHO Classification of Tumors. WHO Classification of Tumours Editorial Board (editor). WHO editorial, 5th edition; 2021.
- Basso C, Valente M, Poletti A, Casarotto D, Thiene G. Surgical pathology of primary cardiac and pericardial tumors. *Eur J Cardiothorac Surg*. 1997;12:730-737. doi: 10.1016/s1010-7940(97)00246-7.
- Ekmektzoglu KA, Samelis GF, Xanthos T. Heart and tumors: location, metastasis, clinical manifestations, diagnostic approaches and therapeutic considerations. *J Cardiovasc Med (Hagerstown)*. 2008;9:769-777. doi: 10.2459/JCM.0b013e3282f88e49.
- Simpson L, Kumar SK, Okuno SH, Schaff HV, Porrata LF, Buckner JC, et al. Malignant primary cardiac tumors: review of a single institution experience. *Cancer*. 2008;112(11):2440-2446. doi: 10.1002/cncr.23459.
- Bussani R, De-Giorgio F, Abbate A, Silvestri F. Cardiac metastases. *J Clin Pathol*. 2007;60:27-34. doi: 10.1136/jcp.2005.035105.
- Nistor C, Carsote M, Cucu AP, Stanciu M, Popa FL, Ciuche A, et al. Primary cardiac intimal sarcoma: multi-layered strategy and core role of MDM2 amplification/co-amplification and MDM2 immunostaining. *Diagnostics (Basel)*. 2024;14(9):919. doi: 10.3390/diagnostics14090919.
- Ibrahim A, Luk A, Singhal P, Wan B, Zavodni A, Cusimano RJ, et al. Primary intimal (spindle cell) sarcoma of the heart: a case report and review of the literature. *Case Rep Med* 2013;2013:461815. doi: 10.1155/2013/461815.
- Butany J, Nair V, Naseemuddin A, Nair GM, Catton C, Yau T. Cardiac tumors: diagnosis and management. *Lancet Oncol* 2005;6:219-228. doi: 10.1016/S1470-2045(05)70093-0.
- Neuville A, Collin F, Bruneval P, Parrens M, Thivolet F, Gomez-Brouchet A, et al. Intimal sarcoma is the most frequent primary cardiac sarcoma: clinicopathologic and molecular retrospective analysis of 100 primary cardiac sarcomas. *Am J Surg Pathol* 2014;38(4):461-469. doi: 10.1097/PAS.0000000000000184.
- Gupta A. Primary cardiac sarcomas. *Expert Rev Cardiovasc Ther*. 2008;6(10):1295-1297. doi: 10.1586/14779072.6.10.1295.
- Bruce C. 42-cardiac tumors. En: Otto CM, editor. *The practice of clinical echocardiography*. 5th ed. Elsevier; 2016.
- Kraft C, Schuettfort G, Weil Y, Tirneci V, Kasper A, Haberichter B, et al. Thrombosis of the inferior vena cava and malignant disease. *Thromb Res*. 2014;134(3):668-673. doi: 10.1016/j.thromres.2014.07.012.
- Kojiro M, Nakahara H, Sugihara S, Murakami T, Nakashima T, Kawasaki H. Hepatocellular carcinoma with intra-atrial tumor growth. A clinicopathologic study of 18 autopsy cases. *Arch Pathol Lab Med*. 1984;108(12):989-992. doi: 10.1007/978-4-431-68177-9_4.

24. Wang Y, Yuan L, Ge RL, Sun Y, Wei G. Survival benefit of surgical treatment for hepatocellular carcinoma with inferior vena cava/right atrium tumor thrombus: results of a retrospective cohort study. *Ann Surg Oncol.* 2013;20(3):914-922. doi: 10.1245/s10434-012-2756-0.
25. Wassef J, Xu S. Hepatocellular carcinoma with tumor thrombus to the hepatic veins and the right atrium: a case report and review exploring various presentations and treatment options. *Cureus.* 2020;12(6):e8405. doi: 10.7759/cureus.8405.
26. Lee JM, Lee KW, Hong SK, Yoon KC, Cho JH, Yi NJ, et al. Unusual techniques for preserving surgical and oncologic safety in hepatectomy of advanced adrenal malignancy with vena cava and liver invasion. *Ann Surg Oncol.* 2018;25(11):3324-3325. doi: 10.1245/s10434-018-6545-3.
27. Sun N, Zhang J, Li B, Li A, Lv M, Zhang C. Favorable response to multimodal treatment in hepatocellular carcinoma with inferior vena cava and right atrial tumor thrombus and left adrenal gland metastasis: a case report and literature review. *Medicine (Baltimore).* 2021;100(49):e27987. doi: 10.1097/MD.00000000000027987.
28. Lestuzzi C. Primary tumors of the heart. *Curr Opin Cardiol.* 2016;31(6):593-598. doi: 10.1097/HCO.0000000000000335.
29. Tyebally S, Chen D, Bhattacharyya S, Mughrabi A, Hussain Z, Manisty C, et al. Cardiac tumors: *JACC CardioOncology* state-of-the-art review. *JACC CardioOncol.* 2020;2(2):293-311. doi: 10.1016/j.jacc.2020.05.009.
30. Buckley O, Madan R, Kwong R, Rybicki FJ, Hunsaker A. Cardiac masses, part 1: imaging strategies and technical considerations. *AJR Am J Roentgenol* 2011;197(5):W837-W841. doi: 10.2214/AJR.10.7260.
31. Zaragosa-Macias E, Chen MA, Gill EA. Real time three-dimensional echocardiography evaluation of intracardiac masses. *Echocardiography.* 2012;29(2):207-219. doi: 10.1111/j.1540-8175.2011.01627.x.
32. Bhattacharyya S, Khattar R, Senior R. Characterisation of intra-cardiac masses by myocardial contrast echocardiography. *Int J Cardiol.* 2013;163(1):e11-e13. doi: 10.1016/j.ijcard.2012.06.098.
33. Kirkpatrick JN, Wong T, Bednarz JE, Spencer KT, Sugeng L, Ward RP, et al. Differential diagnosis of cardiac masses using contrast echocardiographic perfusion imaging. *J Am Coll Cardiol.* 2004;43(8):1412-1419. doi: 10.1016/j.jacc.2003.09.065.
34. Beroukhim RS, Prakash A, Buechel ER, Cava JR, Dorfman AL, Festa P, et al. Characterization of cardiac tumors in children by cardiovascular magnetic resonance imaging: a multicenter experience. *J Am Coll Cardiol.* 2011;58(10):1044-1054. doi: 10.1016/j.jacc.2011.05.027.
35. Kassop D, Donovan MS, Cheezum MK, et al. Cardiac masses on cardiac CT: a review. *Curr Cardiovasc Imaging Rep.* 2014;7(8):9281. doi: 10.1007/s12410-014-9281-1.
36. Alama L, Agrawala K, Kankanala K, Fishberg R, Powell D. Primary cardiac undifferentiated high-grade intimal pleomorphic sarcoma: a case series report. *Cardiol Res.* 2020;11(2):129-133. doi: 10.14740/cr1029.
37. Tamaki T, Suzuki M, Umezumi R, Utanohara Y, Mahara K, Takanashi S, et al. Clinicopathological features of bi-ventricular cardiac intimal sarcoma-Report of an autopsy case. *J Cardiol Cases.* 2017;16(4):113-115. doi: 10.1016/j.jccase.2017.06.001.
38. Hudzik B, Szkodziniski J, Zubelewicz-Skodziniska B, Gasior M. Primary sarcoma of the heart. *Pol Arch Intern Med.* 2017;127(10):694-695. doi: 10.20452/pamw.4121.
39. Lennerz C, O'Connor M, Schunkert H, Deutsch MA. A case report of primary cardiac sarcoma: A diagnostic and therapeutic challenge. *Eur Heart J Case Rep.* 2018;2(4):1-4. doi: 10.1093/ehjcr/tyt143.
40. Beller JP, Maddalo S, Zamuco R, Axel L, DeAnda A, Balsam LB. Right ventricular undifferentiated pleomorphic sarcoma: a case report. *J Cardiol Cases.* 2015;13(2):60-62. doi: 10.1016/j.jccase.2015.10.008.
41. Muturi A, Kotecha V, Ruturi J, Muhinga M, Waweru W. High-grade spindle cell sarcoma of the heart: a case report and review of literature. *J Cardiothorac Surg.* 2015;29(10):46. doi: 10.1186/s13019-015-0245-6.
42. Mito JK, Mitra D, Doyle LA. Radiation-associated sarcomas: an update on clinical, histologic, and molecular features. *Surg Pathol Clin.* 2019;12(1):139-148. doi: 10.1016/j.path.2018.10.010.
43. Chen E. Pathogenesis of sarcomas. *Diagn Pathol.* 2016;01(01). doi: 10.4172/2476-2024.1000e103.
44. Salminen SH, Sampo MM, Bohling TO, Tuomikoski L, Tarkkanen M, Blomqvist CP. Radiation-associated sarcoma after breast cancer in a nationwide population: increasing risk of angiosarcoma. *Cancer Med.* 2018;7(9):4825-4835. doi: 10.1002/cam4.1698.
45. Orlandi A, Ferlosio A, Roselli M, Chiariello L, Spagnoli LG. Cardiac sarcomas: an update. *J Thorac Oncol.* 2010;5(9):1483-1489. doi: 10.1097/JTO.0b013e3181e59a91.
46. Burke AP, Cowan D, Virmani R. Primary sarcomas of the heart. *Cancer.* 1992;69(2):387-395. doi: 10.1002/1097-0142(19920115)69:2<387:aid-cncr2820690219>3.0.co;2n.
47. Ye N, Lan L, Hu H, Liu J, Xu H. Case report: The diagnostic challenge of primary cardiac intimal sarcoma. *Front Cardiovasc Med.* 2023;10. doi: 10.3389/fcvm.2023.1089636.
48. Blackmon SH, Patel A, Reardon MJ. Management of primary cardiac sarcomas. *Expert Rev Cardiovasc Ther.* 2008;6(9):1217-22. doi: 10.1586/14779072.6.9.1217.
49. Modi A, Lipnevicius A, Moorjani N, Haw M. Prolonged survival with left atrial spindle cell sarcoma. *Interact Cardiovasc Thorac Surg.* 2009;8(6):703-704. doi: 10.1510/icvts.2009.203562.
50. Fu B, Yu H, Yang J. Primary intimal (spindle cell) sarcoma of the left atrium. *Echocardiography.* 2015;32(1):192-4. doi: 10.1111/echo.12777.
51. Adjuvant chemotherapy for localised resectable soft-tissue sarcoma of adults: meta-analysis of individual data. *Sarcoma meta-analysis collaboration.* *Lancet.* 1997;350(9092):1647-1654.
52. Pervaiz N, Colterjohn N, Farrokhyar F, Tozer R, Figueredo A, Ghert M, et al. A systematic meta-analysis of randomized controlled trials of adjuvant chemotherapy for localized resectable soft-tissue sarcoma. *Cancer* 2008;113:573-581. doi: 10.1002/cncr.23592.
53. Maki RG, Wathen JK, Patel SR, Priebat DA, Okuno SH, Samuels B, et al. Randomized phase II study of gemcitabine and docetaxel compared with gemcitabine alone in patients with metastatic soft tissue sarcomas: results of sarcoma alliance for research through collaboration study 002. *J Clin Oncol.* 2007;25(19):2755-2763. doi: 10.1200/JCO.2006.10.4117.
54. Pautier P, Floquet A, Penel N, et al. Randomized multicenter and stratified phase II study of gemcitabine alone versus gemcitabine and docetaxel in patients with metastatic or relapsed leiomyosarcomas: a Federation Nationale des Centres de Lutte Contre le Cancer (FNCLCC) French Sarcoma Group Study (TAXOGEM study). *Oncologist* 2012;17(9):1213-1220. doi: 10.1634/theoncologist.2011-0467.
55. Van der Graaf WT, Blay JY, Chawla SP, Kim DW, Bui-Nguyen B, Casali PG, et al. Pazopanib for metastatic soft-tissue sarcoma (PALETTE): a randomised, double-blind, placebo-controlled phase 3 trial. *Lancet.* 2012;379(9829):1879-1886. doi: 10.1016/S0140-6736(12)60651-5.

Funding: none.

Disclosure: the authors have no conflict of interest to disclose.

Large pericardial cyst mimicking a unilateral pleural effusion: a case report

Quiste pericárdico gigante mimetizando derrame pleural unilateral: reporte de caso

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ABSTRACT

Pericardial cysts are rare and usually asymptomatic. We present the case of a 65-year-old woman with progressive dyspnea and an apparent massive left pleural effusion. After unsuccessful thoracenteses, chest CT revealed a large pericardial cyst measuring 20 × 20 cm. Video-assisted thoracoscopic drainage and unroofing were performed, the presence of a benign serous cyst was confirmed by pathology. This case highlights the diagnostic challenge and the importance of advanced imaging in atypical mediastinal masses.

Keywords: pericardial cyst, dyspnea, pleural effusion, thoracoscopy.

RESUMEN

Los quistes pericárdicos son lesiones infrecuentes, habitualmente asintomáticas. Presentamos el caso de una mujer de 65 años con disnea progresiva y aparente derrame pleural izquierdo masivo. Tras toracocentesis infructuosas, la tomografía reveló un quiste pericárdico gigante de 20 × 20 cm. Se realizó drenaje y destechamiento por toracoscopia, confirmando su naturaleza benigna. El caso resalta el reto diagnóstico y la importancia de la imagen avanzada en masas mediastínicas atípicas.

Palabras clave: quiste pericárdico, disnea, derrame pleural, toracoscopia.

INTRODUCTION

Pericardial cysts are uncommon entities, accounting for approximately 33% of all mediastinal cysts and 6% of mediastinal masses. Their estimated incidence is 1 per 100,000 individuals in the general population, with no gender predilection, and a peak occurrence between 30 and 50 years of age. They are usually asymptomatic and incidentally detected on imaging studies. However, when large, they may cause symptoms such as atypical chest pain, persistent

cough, progressive dyspnea, and others, due to extrinsic compression of adjacent structures.¹⁻³ We present the case of a symptomatic giant pericardial cyst initially misdiagnosed as pleural effusion.

CASE DESCRIPTION

A 65-year-old female with a clinical history of diabetes mellitus and bilateral gonarthrosis presented to the emergency department in April 2024 with progressive dyspnea and a

How to cite: Ortiz-Betance GT, Saldivar-Landeros A, Arreola-Solís PS. Large pericardial cyst mimicking a unilateral pleural effusion: a case report. *Cir Card Mex.* 2026; 11 (3): 111-113. <https://dx.doi.org/10.35366/123485>

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Received: 14-10-2025. Accepted: 25-11-2025.

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radiographic finding suggestive of pleural effusion. She reported the onset of exertional dyspnea for six months, with fatigue after walking 500 meters or bending over. One month later, she developed orthopnea (requiring two pillows to sleep), palpitations, dizziness, and an unintentional weight loss of 5 kg over recent months. The patient also mentioned a history of COVID-19 three years ago, with residual persistent cough and sputum production. Due to persistence of symptoms, she consulted a private physician, who performed an EKG and chest X-ray revealing a massive pleural effusion, prompting referral to our institution. On arrival, her vital signs were blood pressure 143/64 mmHg, heart rate 76 bpm, and respiratory rate 25 breaths per minute. Physical examination revealed decreased chest expansion, crackles over the right hemithorax, absent breath sounds at the left base, and preserved apical vesicular murmur without wheezing. Further electrocardiographic tests and laboratory investigations were mostly negative. Chest radiography showed a large opacity occupying approximately 80% of the left hemithorax, tracheal deviation to the right, and left lung atelectasis (*Fig. 1*).

Multiple attempts of diagnostic-therapeutic thoracentesis were performed unsuccessfully, prompting a thoracic CT scan, which revealed a large fluid-filled collection within the anterior and inferior mediastinum extending laterally, causing extrinsic compression of both lungs without evidence of pleural involvement (*Fig. 2*). Video-assisted thoracoscopy identified a large pericardial cyst containing approximately 2,100 ml of serohematic fluid, located in the left hemithorax and occupying two thirds of it, displacing the lung superiorly. The cyst measured approximately 20 × 20 cm. The cyst was drained and unroofed, and an endopleural tube was placed. Cytological analysis revealed no bacterial growth. Histopathological examination of the cyst wall showed a fragmented serous wall with no evidence of malignancy, which confirmed the diagnosis. Postoperatively, the patient's course was uneventful, with marked improvement in dyspnea and no immediate complications. She was discharged on postoperative day 7 with stable hemodynamic and respiratory parameters.

COMMENTARY

Most pericardial cysts are congenital, resulting from the abnormal development of celomic cavities. They may also be acquired secondary to inflammatory, infectious, or traumatic processes. In this case, a congenital defect with progressive growth leading to clinically significant volume is the most likely etiology.⁴

Giant pericardial cysts are defined as those exceeding 10 cm in diameter. Reported sizes range from less than 3

cm to as large as 28 cm being extremely rare.^{5,6} The cyst's size is clinically relevant, as symptoms typically arise from compression of thoracic structures, explaining the clinical presentation in this case.⁷

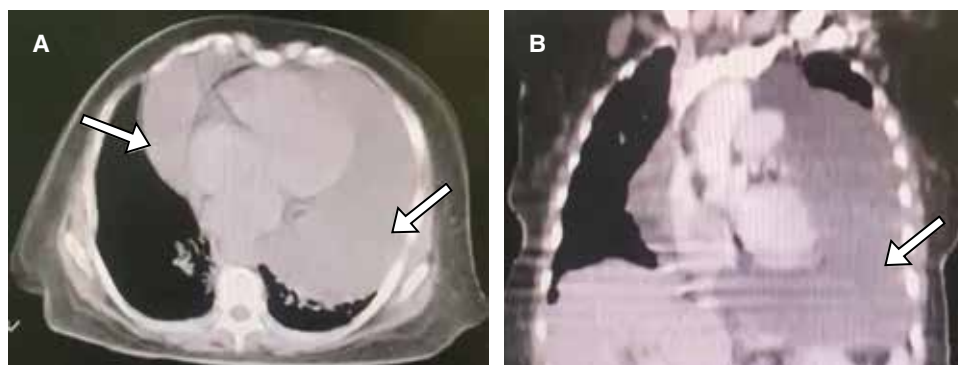
Pericardial cysts are most frequently located in the right cardiophrenic angle (70%), followed by the left (approximately 22%) and other mediastinal sites (superior or posterior) in 8% of cases.⁶ The left-sided location in our patient is atypical and contributed to the initial diagnostic confusion.

The diagnostic workup is often challenging, as several radiologic differential diagnoses must be considered, including bronchogenic cysts, pericardial effusions, teratomas, and pleural effusions. Chest radiography is non-specific. Other imaging modalities include transthoracic echocardiography, computed tomography, and magnetic resonance imaging (MRI), this last one being superior for diagnosis and follow-up due to its excellent delineation of pericardial anatomy.⁸⁻¹⁰ In this case, the absence of complementary imaging led to multiple unsuccessful thoracentesis attempts, which delayed the final diagnosis. This situation has been previously described in the literature, with persistent pleural collections mimicking pericardial cysts.

Management of pericardial cysts should be individualized based on symptoms, cyst growth, and imaging findings suggesting solid components. Asymptomatic patients may be managed conservatively with periodic imaging follow-up. However, surgical resection via thoracotomy, video-assisted thoracic surgery, or percutaneous aspiration are indicated in symptomatic patients or those with enlarging cysts.^{10,11}



Figure 1: Chest radiography showing extensive opacity in the left hemithorax with near-complete obliteration of the pulmonary parenchyma.

**Figure 2:**

Large fluid collection in the anterior and inferior mediastinum extending laterally, causing extrinsic compression of both lungs without evidence of pleural involvement (indicated by white arrows). **A)** Axial view. **B)** Coronal view.

CONCLUSIONS

Giant pericardial cysts are rare entities that can mimic more common conditions such as pleural effusion, representing a significant diagnostic challenge. The atypical left-sided location and large size in this case explained the compressive symptoms observed. This case highlights the importance of including pericardial cysts in the differential diagnosis of mediastinal masses and atypical pleural effusions, as well as the value of advanced imaging for accurate characterization. Video-assisted thoracoscopic drainage and unroofing provided a safe and effective therapeutic approach, resulting in excellent clinical outcomes and favorable prognosis.

REFERENCES

1. Qamar Y, Gulzar M, Qamar A, Sabry H, Minhas T. An incidental finding of a large pericardial cyst. *Cureus*. 2022;14(4):e23917. doi: 10.7759/cureus.23917.
2. Ugwu J, Hamilton R, Taskesen T, Osei K, Ghali M. A rapidly enlarging giant pericardial cyst resected by video-assisted thoracoscopic surgery (VATS): a case report. *J Cardiol Cases*. 2021;25(4):234-236. doi: 10.1016/j.jccase.2021.10.006.
3. Kar SK, Ganguly T. Current concepts of diagnosis and management of pericardial cysts. *Indian Heart J*. 2017;69(3):364-370. doi: 10.1016/j.ihj.2017.02.021.
4. Ershadi R, Vahedi M. Uncommon location of a giant pericardial cyst: a case report. *Iran J Med Sci*. 2021;46(4):308-311. doi: 10.30476/ijms.2021.88408.1911.
5. Noori NM, Shafighi Shahri E, Soleimanzadeh Mousavi SH. Large congenital pericardial cyst presented by palpitation and left ventricle posterior wall compression: a rare case report. *Pediatr Rep*. 2021;13(1):57-64. doi: 10.3390/pediatric13010007.
6. Lomborg ME, Wang Y, Albiniussen RR, Elpert FP. A large pericardial cyst in the left cardiophrenic causing persistent chest pain and cough: a case report. *Am J Case Rep*. 2022;23:e937785. doi: 10.12659/AJCR.937785.
7. Kar SK, Ganguly T. Current concepts of diagnosis and management of pericardial cysts. *Indian Heart J*. 2017;69(3):364-370. doi: 10.1016/j.ihj.2017.02.021.
8. Li M, Yang C, Li J, Jia D, Wang Y, Xie W, et al. A large (giant) pericardial cyst mimicking unilateral pleural effusion: VATS diagnosis and cure. *Medicine (Baltimore)*. 2023;102(15):e33540. doi: 10.1097/MD.00000000000033540.
9. Taguchi E, Oshitomi T, Kamio T, Sakamoto T. Surgical resection of a giant pericardial cyst: case report. *Eur Heart J Case Rep*. 2021;5(4):ytab116. doi: 10.1093/ehjcr/ytab116.
10. Carmona-Ruiz HA, Orihuela-Rodríguez O, Morales-Gudiño I. Quiste pericárdico gigante asintomático. *Cir Cir*. 2021;89(S2):68-71. doi: 10.24875/CIRU.21000280.
11. Hekmat M, Ghaderi H, Tatari H, Arjmand Shabestari A, Mirjafari SA. Giant pericardial cyst: a case report and review of literature. *Iran J Radiol*. 2016;13(1):e21921. doi: 10.5812/iranradiol.21921.

Funding: none.

Disclosure: the authors have no conflict of interest to disclose.

Video-assisted approach to thoracic cysts (pericardial cyst)

Abordaje videoasistido para quistes torácicos (quiste pericárdico)

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ABSTRACT

Pericardial cysts are rare, benign, and asymptomatic mediastinal masses. Most are found in the right cardiophrenic angle, they occur due to embryonic malformations and are found incidentally during complementary studies. Management may be conservative or surgical and is individualized according to the clinical presentation. We describe the case of a patient with a pericardial cyst in an unusual location, left cardiophrenic angle.

Keywords: pericardial cyst, mediastinal masses, videoassisted thoracotomy, incidental finding.

Abbreviations:

CT = computed tomography

VATS = video-assisted thoracoscopic surgery

Pericardial cysts comprise a relatively rare subset of thoracic tumors, accounting for approximately 5 to 10% of cases.^{1,2} They rank as the third most common mediastinal mass, following thymomas and thymic carcinomas.^{2,3} The estimated incidence of pericardial cysts is roughly 1 in 100,000 individuals.⁴ These cysts typically present with diameters of less than 3 cm, although their size can vary considerably, ranging from 2 to 28 cm, with

RESUMEN

Los quistes pericárdicos son infrecuentes, generalmente benignos y de curso asintomático. La mayoría se localizan en el ángulo cardiofrénico derecho, ocurren secundarios a malformaciones embrionarias y se presentan como un hallazgo en estudios complementarios. El manejo puede ser conservador o quirúrgico, y se individualiza según la clínica. Describimos el caso de una paciente con un quiste pericárdico de localización inusual, en ángulo cardiofrénico izquierdo.

Palabras clave: quiste pericárdico, masas mediastinales, toracotomía videoasistida, hallazgo incidental.

those exceeding 8 to 9 cm in diameter being classified as giant cysts.⁵

The majority of pericardial cysts are congenital in origin, resulting from embryological malformations due to defects in the development of the coelomic cavity, which occur when the pericardium becomes entrapped during embryonic development.⁶ However, acquired cases can also arise following thoracic surgery, chest trauma, or pericarditis.^{7,8} Typically, these cysts are asymptomatic, although they may present with symptoms such as dyspnea, chest pain, and persistent cough. Furthermore, compression of adjacent structures can lead to complications like superior vena cava

How to cite: Jiménez-González RJ, Ogaz-Escarpita MC, Castañón-Colunga EA, Carrera-Baca M, Guillén-Ramírez RM, Marmolejo-Rivera R et al. Video-assisted approach to thoracic cysts (pericardial cyst). Cir Card Mex. 2026; 11 (3): 114-117. <https://dx.doi.org/10.35366/123486>

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Received: 13-11-2025. Accepted: 20-11-2025.

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syndrome. Diagnosis is often incidental, with chest X-rays revealing a round radiopaque mass.^{9,10} Characteristic imaging features include a single, thin-walled, round, well-defined lesion with clear fluid content and no contrast enhancement.^{5,11} The right costophrenic angle is the most common site of presentation, often associated with dextrocardia when located in this region.¹² Although rare, potential complications include cardiac tamponade, a severe and potentially life-threatening outcome.¹³ We present a case of a patient with a pericardial cyst located in the left costophrenic angle, an uncommon site.

CASE DESCRIPTION

A 77-year-old woman with a longstanding history of systemic hypertension spanning over two decades, treated with atenolol and chlorthalidone 50/12.5 mg, presented with nonspecific chest pain and exertional dyspnea, which significantly impaired her functional capacity. A chest radiograph revealed a round radiopaque mass in the left costophrenic angle, prompting further evaluation with a plain and contrast-enhanced computed tomography (CT) scan of the chest (*Fig. 1*).

The CT scan demonstrated an extrapulmonary intrathoracic cystic lesion in the left costophrenic angle, measuring approximately 290 ml in volume. Based on the imaging findings, a differential diagnosis of pericardial mesothelial cyst versus pleural cyst of unknown etiology was considered. Following consultation with the cardiothoracic surgery department, a diagnostic and therapeutic *video*-assisted thoracoscopic surgery (VATS) was planned (*Fig. 2*).

The procedure involved thoracoscopy via the sixth left intercostal space, followed by single-port thoracoscopic resection of the pericardial cyst. Intraoperatively, an

extrapulmonary cystic lesion dependent on the parietal pericardium with a highly vascularized capsule was observed. Two feeding vessels, located anteriorly and posteriorly, were identified, ligated, and surgically excised. Subsequent puncture of the cyst evacuated approximately 290 ml of citrine fluid, which was sent for cytological and cytochemical analysis. The cyst capsule was then excised and submitted for histopathological examination. The cytochemical analysis revealed a transparent, straw-yellow fluid with a pH of 8.00, protein content of 2 g/dl, glucose level of 20 mg/dl, and absence of blood cells or microorganisms. The culture remained negative after 48 hours of incubation. The patient recovered uneventfully and was discharged after a fourth-day hospital stay (*Fig. 3*). Histopathological examination of the cyst capsule confirmed the diagnosis of a giant pericardial cyst.

COMMENT

The diagnosis of pericardial cysts is typically incidental, often detected on chest radiography, but computed tomography and cardiothoracic magnetic resonance imaging are the modalities of choice for accurately determining the size, location, and characteristics of the lesion.¹⁴

Transesophageal echocardiography is employed to assess the hemodynamic impact on right ventricular ejection flow and to evaluate potential constrictive pathophysiology affecting both ventricular chambers.^{11,15} With the advent of minimally invasive surgical techniques, video-assisted thoracoscopic surgery (VATS) has become an invaluable tool not only for diagnostic confirmation but also for therapeutic intervention.

The management of pericardial cysts spans a spectrum from conservative approaches, characterized by serial imaging

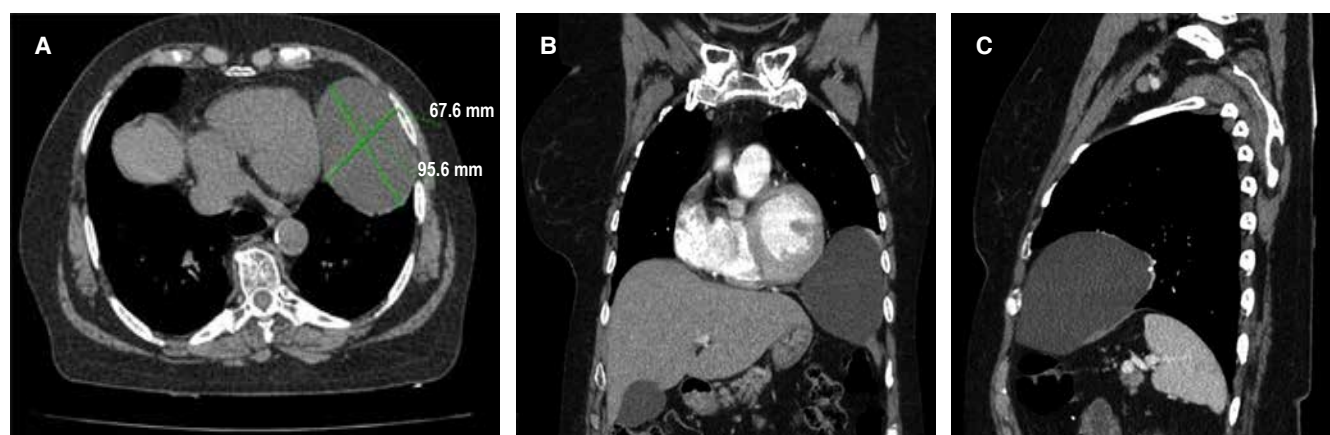


Figure 1: A) Plain axial computed tomography scan showing a cystic lesion in the left pericardial region measuring 6.76 × 9.56 cm, with an approximate volume of 290 ml. B-C) Contrast-enhanced coronal and sagittal computed tomography scans showing the presence of a pericardial cyst over the left cost diaphragmatic sinus.

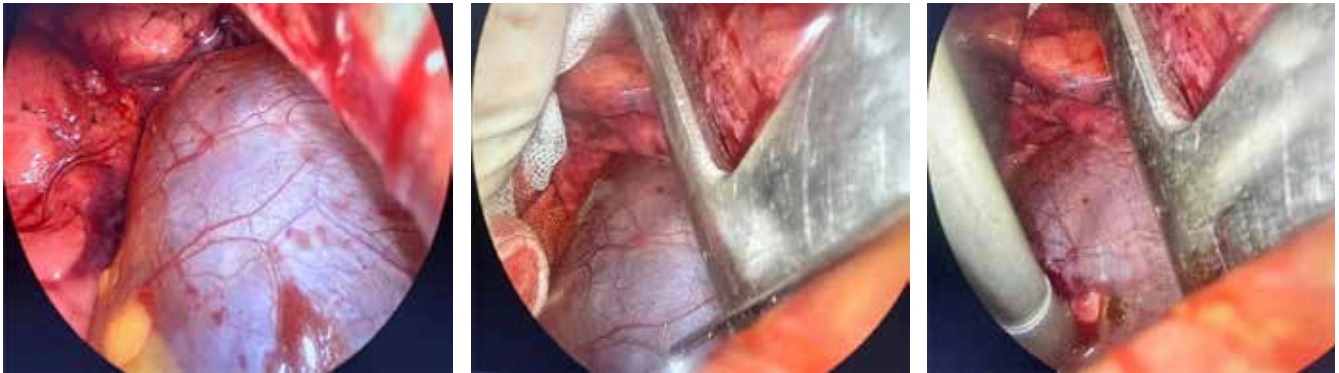


Figure 2: Intraoperative images showing the presence of an extrapulmonary cystic lesion dependent on the parietal pericardium with a highly vascularized capsule.

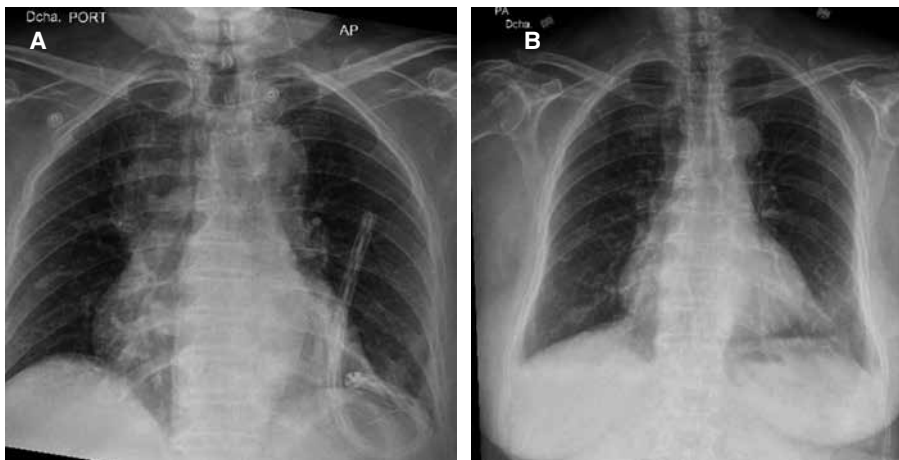


Figure 3:

A) Anteroposterior chest X-ray immediately after surgery, showing the presence of a Kardia Spiral pleural drain in the left hemithorax. **B)** Control anteroposterior chest X-ray two days after surgery.

surveillance in asymptomatic cases, to invasive procedures, including percutaneous aspiration and surgical excision, which are typically reserved for symptomatic cases or those associated with complications. In the present case, surgical removal via a minimally invasive approach was chosen for due to the cyst's size and symptomatic nature.^{14,15}

The decision-making process for managing pericardial cysts hinges on several factors, including the presence or absence of symptoms secondary to compression of adjacent structures, the size of the mass, the patient's hemodynamic status, and the behavior of the mediastinal lesion.¹⁵ According to the European Society of Cardiology guidelines, initial treatment with percutaneous aspiration and ethanol sclerotherapy is recommended for single, small cysts or in cases where surgical risk is high, while surgical resection via VATS or thoracotomy is advocated for large, symptomatic cysts as a second-line therapy.^{9,16} Notably, minimally invasive approaches offer several advantages, including reduced perioperative and postoperative complications, shorter hospital stays, and a lower risk of infection.¹⁶

CONCLUSIONS

Pericardial cysts often present with nonspecific symptoms, necessitating a thorough and meticulous evaluation to prevent diagnostic delays and ensure timely intervention. The vague chest symptoms exhibited by the patient in this case underscore the importance of considering a broad differential diagnosis when assessing chest syndromes.

Management must be tailored to the individual, as large cysts, such as the one presented, can cause compressive symptoms in adjacent structures, potentially leading to severe complications if left untreated. Currently, percutaneous aspiration and cyst resection via video-assisted thoracoscopy are the recommended treatment modalities, offering an effective and minimally invasive approach to managing these lesions.

REFERENCES

1. Hynes JK, Tajik AJ, Osborn MJ, Orszulak TA, Seward JB. Two-dimensional echocardiographic diagnosis of pericardial cyst. *Mayo Clin Proc.* 1983;58(1):60-63.

2. Adib-Hajbagheri P, Mirmohammadsadeghi M, Paknahad M, Rafiyan M. Pericardial cyst unveiling: a case of unusual chest symptoms in a young woman with a family history of cancer: a case report and review of literature. *J Med Case Reports*. 2024;18(1):459. doi: 10.1186/s13256-024-04771-1.
3. Keita IK, Nazario Dolz AM, Falcón VGC, Castillo TL, Rodríguez FZ, Romero GLI. Consideraciones en torno a los tumores del mediastino. *Rev Colomb Cir*. 2020;35(3):472-482. doi: 10.30944/20117582.460.
4. Rahman SMT, Rhaman MM, Hoque MA, Proma SB. Unusual cause of mediastinal tumor: a case of calcified pericardial cyst. *Rare Tumors*. 2023;15:20363613231177539. doi: 10.1177/20363613231177539.
5. Grigoras A, Amalinei C, Caruntu ID, Grigoras CC, Chiselita IR, Crisan-Dabija RA. Symptomatic pericardial cysts and dilemmas in their diagnosis. *Rom J Morphol Embryol*. 2023;64(4):517-525. doi: 10.47162/RJME.64.4.08.
6. Koo CW, Newburg A. Congenital absence of the right pericardium: embryology and imaging. *J Clin Imaging Sci*. 2015;5:12. doi: 10.4103/2156-7514.152338.
7. Carmona-Ruiz HA, Orihuela-Rodríguez Ó, Morales-Gudiño I. Asymptomatic giant pericardial cyst. *Cir Cir*. 2021;89(S2):68-71. doi: 10.24875/CIRU.21000280.
8. Jiménez MC, España BE, Sanz MAI, Robles VP, Olmedilla AP, Campuzano RR. Resolución completa de quiste pericárdico atípico tras pleuropericarditis aguda. *Rev Esp Cardiol*. 2021;74(12):1111-1113. doi: 10.1016/j.recesp.2021.05.001.
9. Kar SK, Ganguly T. Current concepts of diagnosis and management of pericardial cysts. *Indian Heart J*. 2017;69(3):364-370. doi: 10.1016/j.ihj.2017.02.021.
10. Tyebally S, Chen D, Bhattacharyya S, Mughrabi A, Hussain Z, Manisty C, et al. Cardiac tumors: JACC CardioOncology State-of-the-art review. *JACC CardioOncol*. 2020;2(2):293-311. doi: 10.1016/j.jacc.2020.05.009.
11. Elhakim A, Boguschewski A, Zamzow P, Saad M. A haemorrhagic pericardial cyst compressing the right side of the heart: a case report. *Eur Heart J Case Rep*. 2023;7(10):ytad497. doi: 10.1093/ehjcr/ytad497.
12. Jiménez-Serrano JA, Jiménez-González A, Esparza-Pantoja J, López-Viramontes B. Quiste pericárdico gigante: reporte de un caso. *Lux Médica*. 2016;11(34):35-39. doi: 10.33064/34lm2016713.
13. Álvarez CM, Cantarini MB, Lombardi M, Martínez L, Pagés M, Orquera D, et al. Taponamiento cardíaco como complicación de ruptura intrapericárdica de quiste celómico. *CONAREC*. 2016;31(137):282-284.
14. Wang ZJ, Reddy GP, Gotway MB, Yeh BM, Hetts SW, Higgins CB. CT and MR imaging of pericardial disease. *RadioGraphics*. 2003;23(Suppl_1):S167-S180. doi: 10.1148/rg.23si035504.
15. Lennon Collins K, Zakhariou F, Mandal AKJ, Missouris CG. Pericardial cyst: never too late to diagnose. *J Clin Med*. 2018;7(11):399. doi: 10.3390/jcm7110399.
16. Zhang WM, Maimaitiaili A, Aizezi R, Abulimiti K, Yan F, Huo Q. Surgical management of pericardial cysts: a single-center retrospective study. *Cureus*. 2023;15(11):e49298. doi: 10.7759/cureus.49298.

Funding: none.

Disclosure: the authors have no conflict of interest to disclose.

Yang-Y in reoperation: a case report

Y de Yang en reoperación: reporte de un caso

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ABSTRACT

We present the case of a 27-year-old woman with cyanotic congenital heart disease due to a ventricular septal defect that was complicated by endocarditis in 2017. Following corrective surgery and aortic valve replacement, she developed prosthetic valve dysfunction in 2025, necessitating reintervention utilizing the Yang Y-incision technique. This approach enlarges the aortic annulus and mitral-aortic continuity, which makes it easier to place a larger prosthesis. This case underscores the successful surgical management of a young patient with a complex medical history and highlights the utility of this technique in complex reoperations.

Keywords: aortic valve, aortic root enlargement, cardiac surgical procedures, reoperation, surgical aortic valve replacement, Yang Y-incision technique.

RESUMEN

Presentamos el caso de una mujer de 27 años de edad con cardiopatía congénita cianótica secundaria a un defecto del tabique interventricular que se complicó con endocarditis en 2017. Tras una cirugía correctiva y un reemplazo valvular aórtico, desarrolló disfunción valvular protésica en 2025, lo que requirió una reintervención mediante la técnica de incisión en Y de Yang. Este abordaje amplía eficazmente el anillo aórtico y la continuidad mitroaórtica, facilitando así la colocación de una prótesis de mayor tamaño. Este caso destaca el éxito del tratamiento quirúrgico de una paciente joven con antecedentes médicos complejos y destaca la utilidad de esta técnica en reoperaciones complejas.

Palabras clave: válvula aórtica, agrandamiento de la raíz aórtica, procedimientos quirúrgicos cardíacos, reoperación, reemplazo quirúrgico de la válvula aórtica, técnica de incisión en Y de Yang.

A prosthetic valve dysfunction can lead to signs or symptoms compatible with heart failure due to the improper functioning of cardiac valves, which may be biological or mechanical. Despite the benefits provided by prosthetic valves, recipients may develop new pathologies; therefore, these mechanisms can be classified as intrinsic or extrinsic. Intrinsic prosthetic dysfunction, also known as primary structural failure of the prosthesis, is mainly related to the lifespan of the valve, associated with material wear or

fracture in mechanical prostheses, and leaflet calcification in biological valves. Extrinsic dysfunction refers to any cause external to the valve itself that results in its disruption. It is generally associated with paravalvular leak, which is the most common, inappropriate prosthesis size relative to the recipient annulus, hemolytic anemia, pannus entrapment, or prosthetic thrombosis. When prosthetic dysfunction or secondary complications are suspected, a series of paraclinical tests must be performed to optimally evaluate

How to cite: Ramírez-Zedillo D, de Paz-Ocaña A, Alcalá-Gutiérrez DA, Peña-Quintero KV, Bustos-Romero M, Gómez-Manríquez J. Yang-Y in reoperation: a case report. *Cir Card Mex*. 2026; 11 (3): 118-121. <https://dx.doi.org/10.35366/123487>

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Received: 11-02-2025. Accepted: 01-09-2026.

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the patient and subsequently determine the appropriate management.^{1,2}

Aortic annulus enlargement with the Yang Y-incision was first described by Bo Yang in 2021. Initially named the “Y-incision/rectangular patch,” it was later renamed in honor of its creator and is now known as the “Yang Y-incision.” Typical indications include patients with a small aortic annulus and those undergoing surgical reintervention after prior valve replacement who present with dysfunction of the existing prosthesis, as in this clinical case report. This technique allows the implantation of a larger prosthesis without compromising adjacent structures, reducing the risk of patient-prosthesis mismatch and improving long-term hemodynamic performance. Timely decision-making and detailed planning are essential to avoid future complications.^{3,4}

CASE DESCRIPTION

We report the case of a 27-year-old woman with a complex cardiovascular clinical history of acyanotic congenital heart disease, specifically a perimembranous ventricular septal defect, complicated by infective endocarditis involving the native aortic valve during pregnancy in 2017. This condition required emergency cesarean delivery followed by concomitant mechanical aortic valve replacement and patch closure of the ventricular septal defect. The patient remained under cardiology surveillance; however, during the year preceding the current admission, she developed progressive deterioration in functional capacity, manifested by worsening exertional dyspnea, marked reduction in exercise tolerance, and recurrent syncopal episodes, consistent with advanced prosthetic valve obstruction. Transthoracic echocardiography demonstrated severe dysfunction of the mechanical aortic valve secondary to extensive pannus formation, with elevated transprosthetic gradients and significant obstruction to left ventricular outflow, warranting surgical reintervention.

Surgical reintervention was performed under balanced general anesthesia with invasive arterial and central venous monitoring. Peripheral cardiopulmonary bypass was established via left inguinal exposure. After systemic heparinization, purse-string sutures of 4-0 Prolene were placed on the femoral artery and vein. Arterial inflow was achieved using a 19-Fr femoral arterial cannula, and venous drainage was obtained with a 21-Fr femoral venous cannula. Cardiopulmonary bypass was initiated, and the patient was cooled systemically. Myocardial protection was achieved using cardioplegic arrest following cross-clamping of the ascending aorta. A transverse aortotomy was performed approximately 1 cm above the sinotubular junction, providing optimal exposure of the aortic root and prosthetic valve.



Figure 1:

Aortotomy and removal of mechanical aortic valve with pannus.

Intraoperative inspection revealed circumferential pannus overgrowth extending from the annulus into the subvalvular region, severely restricting leaflet motion. The mechanical prosthesis was carefully explanted, and complete resection of pannus tissue was performed using sharp dissection to restore native annular contours while avoiding injury to adjacent structures (*Fig. 1*).

Given the markedly small aortic annulus and concern for postoperative patient-prosthesis mismatch, annular enlargement was considered mandatory. Yang Y-incision technique was selected due to its ability to provide substantial annular and root enlargement while preserving aortic root geometry. A Y-shaped incision was created, extending from the nadir of the non-coronary sinus and bifurcating toward the left coronary sinus, traversing the annulus and entering the fibrous trigones. This incision was subsequently reconstructed using a tailored Dacron patch, sutured meticulously to enlarge both the annulus and the adjacent sinuses, thereby increasing the effective annular diameter (*Fig. 2*).

Following annular and root reconstruction, a 21-mm bioprosthetic aortic valve was implanted in the supra-annular position using interrupted, pledgeted 2-0 Prolene sutures placed evenly around the enlarged annulus to ensure uniform load distribution and optimal seating. Valve orientation was carefully adjusted to avoid coronary ostial obstruction and to ensure symmetric leaflet opening. Prior to aortic closure, the prosthesis was thoroughly inspected, confirming unrestricted leaflet mobility and absence of paravalvular gaps. The aortotomy was closed in a continuous fashion using 4-0 Prolene. After de-airing maneuvers, the aortic cross-clamp was removed, restoring coronary perfusion and spontaneous sinus rhythm.

Temporary epicardial atrial and ventricular pacing wires were placed prophylactically. The patient was successfully weaned from cardiopulmonary bypass on the first attempt, demonstrating stable hemodynamics without the need for inotropic or mechanical circulatory support. Transesophageal echocardiographic assessment confirmed appropriate prosthetic valve function, low transvalvular gradients, and no evidence of paravalvular leak.

Aortic root enlargement using Yang Y-incision technique is depicted in *Fig. 3*, illustrating the characteristic bifurcated extension through the non-coronary and left coronary sinuses with patch augmentation. This configuration allows for significant expansion of the aortic annulus and root, facilitating implantation of a larger prosthetic valve and effectively mitigating the risk of patient-prosthesis mismatch while maintaining physiologic aortic root geometry (*Fig. 3*).

Following separation from cardiopulmonary bypass, decannulation was performed in a standard fashion with meticulous hemostasis. A left-sided chest tube was positioned toward the left pleural cavity, and an additional right pleural tube was placed for adequate bilateral pleural drainage. Mediastinal closure was performed in layers. The sternum, noted to be of poor bone quality, was approximated using two crossed stainless-steel wires and one simple wire to enhance stability. Intraoperative findings included dense and loose pericardial and epicardial adhesions, consistent with the patient's history of prior median sternotomy.

COMMENTARY

In patients with previous prosthetic valve replacement, reintervention represents a therapeutic challenge due to the need to preserve hemodynamic stability as much as possible in

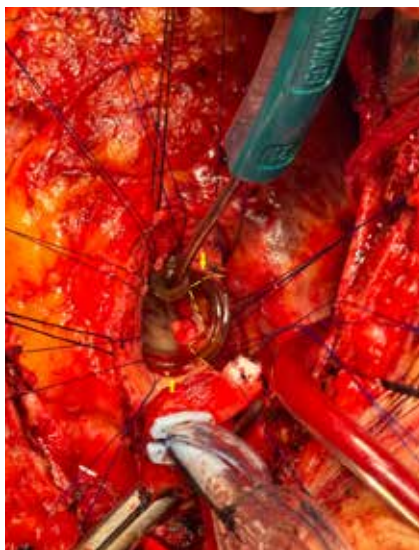


Figure 2:

Enlargement using the Yang Y-incision technique with Dacron patch.



Figure 3:

Placement of biological prosthetic aortic valve replacement.

the setting of prosthetic dysfunction requiring aortic annulus enlargement, which is essential to prevent patient-prosthesis mismatch. Although classical techniques previously described have been viable therapeutic options, the Yang Y-incision technique offers an innovative alternative that allows annular enlargement without compromising adjacent structures. The case presented highlights the usefulness of this technique in a rare and highly complex clinical scenario.^{1,5,6}

CONCLUSIONS

The Yang Y-incision technique has demonstrated safety and efficacy as an alternative in young patients with a small aortic root or the need for reintervention. This experience reinforces its value as a strategy for aortic annulus enlargement and for reducing complex clinical scenarios. Although the published experience is still limited and comes mainly from specialized centers,^{2,7,8} the initial results support its inclusion as a valid option.

REFERENCES

1. Yang B, Ghita C, Makkinejad A, Green C, Wu X. Early outcomes of the Y-incision technique to enlarge the aortic annulus 3 to 4 valve sizes. *J Thorac Cardiovasc Surg.* 2024;167(4):1196-1205.e2. doi: 10.1016/j.jtcvs.2022.07.006.
2. Yang B, Naeem A. A Y incision and rectangular patch to enlarge the aortic annulus by three valve sizes. *Ann Thorac Surg.* 2021;112(2):e139-e141. doi: 10.1016/j.athoracsurg.2021.01.072.
3. Inno G, Takahashi Y, Nishiya K, Nagao M, Kawase T, Morisaki A, et al. Aortic annular enlargement with modified Y-incision technique devised from preoperative three-dimensional computed tomography. *Ann Thorac Cardiovasc Surg.* 2024;30(1):23-00153. doi: 10.5761/atcs.nm.23-00153.

4. Yang B, Naeem A, Palmer S. “Roof” technique-a modified aortotomy closure in Y-incision aortic root enlargement upsizing 3-4 valve sizes. *JTCVS Tech.* 2022;12:33-36. doi: 10.1016/j.xjtc.2022.01.006.
5. Yazdchi F, Monaghan K, Yang B. Aortic valve replacement with Y-incision/rectangular patch aortic annular enlargement. *Indian J Thorac Cardiovasc Surg.* 2023;39(Suppl 2):341-343. doi: 10.1007/s12055-023-01606-4.
6. Yang B. Aortic valve replacement vs aortic valve replacement + annular enlargement: apples to oranges? *Ann Thorac Surg.* 2024;117(2):479-480. doi: 10.1016/j.athoracsur.2023.02.044.
7. Paredes-Acevedo FE, Martínez-Ninanqui FW, Morón-Castro J, Ríos-Ortega JC. Yang aortic root enlargement through ministernotomy in an obese patient. *Cirugía Cardiovascular.* 2024;31(3):137-139. doi: 10.1016/j.circv.2024.02.004.
8. Ghimire A. Hemodynamic assessment of Y-incision aortic root enlargement using computational simulations. *Electronic Theses and dissertations.* [Masters Thesis in Internet]. Denver: University of Denver; 2023. 55 p. Available at: <https://digitalcommons.du.edu/etd/2281>

Funding: none.

Disclosure: the authors have no conflict of interest to disclose.

Repair of type I truncus arteriosus with pulmonary artery branch preservation and use of a valved PTFE conduit

Corrección del tronco arterioso tipo I con preservación de la anatomía de las ramas pulmonares y uso de conducto valvulado de PTFE

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ABSTRACT

Truncus arteriosus type I is a rare and complex congenital heart defect, the surgical management of which is fraught with the risk of pulmonary branch obstruction. We describe a corrective approach utilizing the Alfieri technique, with a focus on preserving the native pulmonary branch architecture, complemented by the use of a handcrafted PTFE valved conduit for right ventricular outflow tract reconstruction. This strategy mitigates anastomotic tension, thereby reducing the risk of obstruction and endocarditis, and consequently, the need for early reintervention.

Keywords: type I truncus arteriosus, pulmonary artery preservation, PTFE valved conduit.

Abbreviations:

PTFE = polytetrafluoroethylene
VSD = ventricular septal defect

Truncus arteriosus is a rare congenital heart disease characterized by a single arterial trunk giving rise to the aorta, pulmonary arteries, and coronary arteries.^{1,2} The classic Collett and Edwards classification distinguishes four anatomic types (I-IV) based on the origin

RESUMEN

El tronco arterioso tipo I es una cardiopatía congénita compleja que requiere tratamiento quirúrgico, el cual conlleva un riesgo significativo de obstrucción de las ramas pulmonares. Se presenta la corrección mediante la técnica de Alfieri, enfocada en preservar la arquitectura de las ramas pulmonares. Además, se utiliza un conducto valvulado de PTFE fabricado in situ para reconstruir el tracto de salida del ventrículo derecho. Esta estrategia reduce la tensión en las anastomosis, minimizando el riesgo de obstrucción y endocarditis, lo que a su vez disminuye la necesidad de reintervenciones tempranas.

Palabras clave: tronco arterioso tipo I, preservación de ramas pulmonares, conducto valvulado de PTFE.

and arrangement of the pulmonary arteries.³ Type I (*Fig. 1*), the most prevalent and surgically pertinent variant, features pulmonary arteries arising from a short common pulmonary trunk, thereby dictating the surgical approach required to preserve pulmonary artery integrity and architecture.^{3,4} This anatomic consideration underpins the surgical technique described by Alfieri, which prioritizes preservation of the native pulmonary artery architecture during repair.⁵

How to cite: Alcántara-Noguez C. Repair of type I truncus arteriosus with pulmonary artery branch preservation and use of a valved PTFE conduit. *Cir Card Mex.* 2026; 11 (3): 122-124. <https://dx.doi.org/10.35366/123488>

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Received: 19-01-2026. Accepted: 09-02-2026.

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Standard surgical management entails separation of the pulmonary arteries from the common trunk, closure of the ventricular septal defect, and reconstruction of the right ventricular outflow tract using a valved conduit to the pulmonary arteries.^{1,6} A frequent postoperative complication is pulmonary artery branch stenosis, typically precipitated by branch division, anatomic distortion, and tension at the distal conduit anastomosis, which may precipitate early reintervention and functional decline.⁶⁻⁸

SURGICAL TECHNIQUE

With the heart under aortic cross-clamping, moderate hypothermia, bicaval cannulation, and del Nido cardioplegia, a “hockey-stick” incision is made on the truncal artery and extended toward the left pulmonary artery, allowing clear identification of the common aortopulmonary window (Fig. 2). This approach facilitates preservation of pulmonary artery architecture, a fundamental principle of the technique described by Alfieri.⁵

The defect within the truncal artery is closed using a 0.4-mm polytetrafluoroethylene (PTFE) patch with continuous 6-0 vascular polypropylene sutures, ensuring that the pulmonary artery confluence remains free of obstruction. Subsequently, a right ventriculotomy is performed to expose the ventricular septal defect (VSD), which is closed with a second 0.4-mm PTFE patch using continuous 5-0 vascular polypropylene sutures (Fig. 3).

Prior to surgery, a 10-mm valved PTFE conduit is handcrafted using two 0.1-mm PTFE leaflets. The proximal anastomosis is initiated by positioning the valve as close as



Figure 1:

Three-dimensional computed tomography angiography reconstruction demonstrating type I truncus arteriosus, with a single arterial trunk giving rise to the aorta and pulmonary arteries, including a short common pulmonary trunk and bifurcation into both pulmonary artery branches.

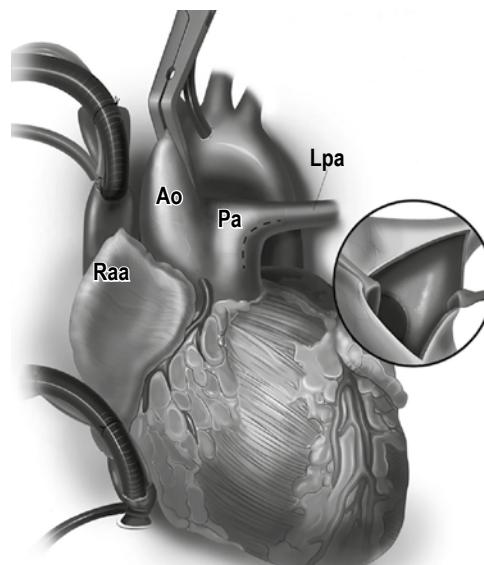


Figure 2: Surgical illustration depicting the aortopulmonary window in type I truncus arteriosus. A “hockey-stick” incision is shown on the arterial trunk, extended toward the left pulmonary artery, providing excellent exposure of the short common pulmonary trunk and facilitating preservation of pulmonary artery branch anatomy.

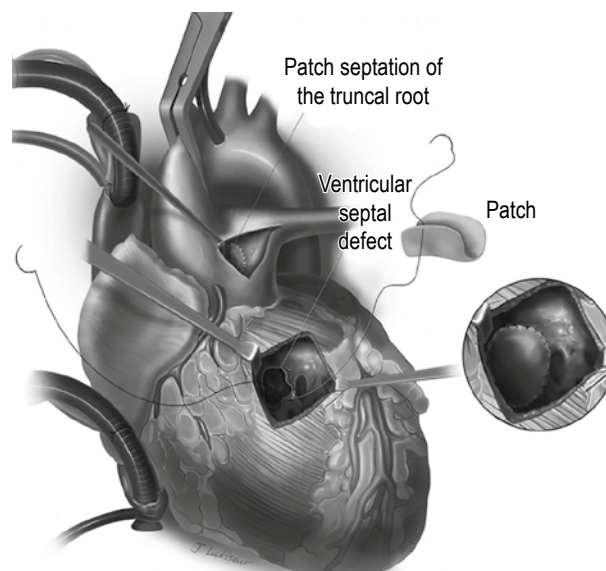


Figure 3: Surgical illustration demonstrating right ventriculotomy with exposure of the ventricular septal defect. Patch closure of the ventricular septal defect and separation of the truncal root using a patch are shown, completing intracardiac repair prior to right ventricle-pulmonary artery continuity reconstruction.

possible to the pulmonary artery confluence and suturing it with 6-0 vascular polypropylene suture. The conduit is then tailored and its distal end beveled to complete the distal anastomosis to the right ventricle using 6-0 polypropylene

suture, thereby establishing continuity between the right ventricle and the pulmonary arteries. This anastomosis is performed with the heart beating (*Fig. 4*) (*Fig. 5*), with the aim of minimizing tension and optimizing conduit alignment.

CONCLUSION

The Alfieri repair technique, which is based on preservation of the pulmonary artery architecture via a cryopreserved aortic homograft, has yielded excellent anatomic and functional outcomes, while reducing the incidence of pulmonary artery branch stenosis.⁵ However, the scarcity of homografts in our setting constitutes a significant impediment to its routine implementation. In this context, the utilization of a handcrafted valved PTFE conduit has demonstrated good outcomes in right ventricular outflow tract reconstruction, exhibiting comparable performance to homografts in terms of patency and reintervention rates.^{6,7} The combined use of the Alfieri technique, aimed at preserving pulmonary artery architecture, in conjunction with a handcrafted valved PTFE conduit, represents a viable alternative for patients with type I truncus arteriosus and, selectively, type II. This approach may reduce the risk of pulmonary artery obstruction, minimizes tension at the distal anastomosis, and attenuates the need for early reinterventions on the pulmonary arteries, without augmenting the risk of endocarditis or other major complications.⁶⁻⁸



Figure 4: Intraoperative image demonstrating the distal anastomosis of a valved PTFE conduit to the pulmonary artery confluence, highlighting the valve leaflets positioned close to the pulmonary bifurcation, with appropriate orientation and no anastomotic tension.

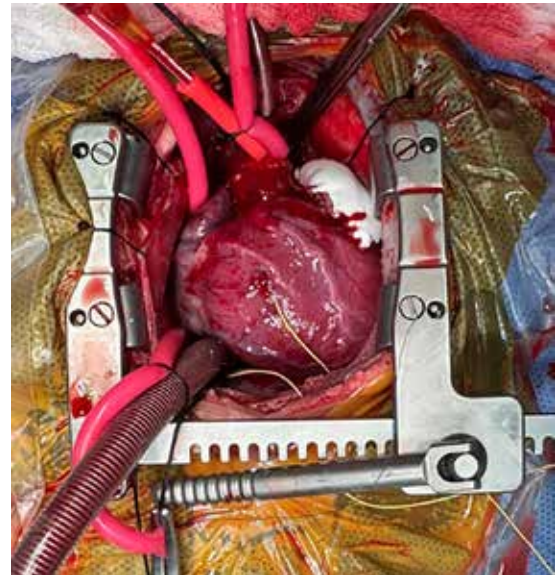


Figure 5: Final intraoperative image demonstrating the proximal anastomosis of the valved polytetrafluoroethylene conduit to the right ventricle, with a beveled conduit end, completing right ventricular outflow tract reconstruction and finalizing the surgical procedure.

REFERENCES

1. Mery C, Bastero P, Cabrera A, Hall S. Texas children's hospital handbook of congenital heart disease. Hall AGCS (eds). Texas Children's Hospital; 2020.
2. Collett RW, Edwards JE. Persistent truncus arteriosus; a classification according to anatomic types. *Surg Clin North Am.* 1949;29(4):1245-1270.
3. Van Praagh R, Van Praagh S. The anatomy of common aorticopulmonary trunk (truncus arteriosus communis) and its embryologic implications. A study of 57 necropsy cases. *Am J Cardiol.* 1965;16(3):406-425. doi: 10.1016/0002-9149(65)90732-0.
4. Russell HM, Jacobs ML, Anderson RH, et al. A simplified categorization for common arterial trunk. *J Thorac Cardiovasc Surg.* 2011;141(3):645-653. doi: 10.1016/j.jtcvs.2010.08.022.
5. Alfieri G, Swartz M. Technique for the repair of truncus arteriosus to maintain pulmonary artery architecture. *Oper Tech Thorac Cardiovasc Surg.* 2011;16(2):98-103. doi: 10.1053/j.optechstcvs.2011.06.001.
6. Mercer CW, West SC, Sharma MS, Yoshida M, Morell VO. Polytetrafluoroethylene conduits versus homografts for right ventricular outflow tract reconstruction in infants and young children: An institutional experience. *J Thorac Cardiovasc Surg.* 2018;155(5):2082-2091.e1. doi: 10.1016/j.jtcvs.2017.11.107.
7. Buckley JR, Amula V, Sassalos P, et al. Multicenter analysis of early childhood outcomes after repair of truncus arteriosus. *Ann Thorac Surg.* 2019;107(2):553-559. doi: 10.1016/j.athoracsur.2018.08.094.
8. Herrmann JL, Larson EE, Mastropietro CW, et al. Right ventricular outflow tract reconstruction in infant truncus arteriosus: a 37-year experience. *Ann Thorac Surg.* 2020;110(2):630-637. doi: 10.1016/j.athoracsur.2019.11.023.

Funding: none.

Disclosure: the authors have no conflict of interest to disclose.

Post-myocardial infarction ventricular septal defect closure: does the end justify the means?

Cierre de comunicación interventricular postinfarto: ¿el fin justifica los medios?

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Keywords: cardiogenic shock, myocardial infarction, complications, percutaneous coronary interventions, post-infarction ventricular septal defect, transcatheter procedures.

Palabras clave: choque cardiogénico, infarto de miocardio, complicaciones, intervenciones coronarias percutáneas, defecto del tabique ventricular postinfarto, procedimientos transcáteter.

Post-myocardial infarction ventricular septal defect (MI-VSD) remains one of the most catastrophic mechanical complications of acute coronary syndromes. It is characterized by a dismal natural history and an exceptionally high mortality rate, despite advances in reperfusion and critical care. While surgical repair persists as the definitive gold standard, one-year survival without intervention is exceedingly poor, and percutaneous alternatives have yielded only limited success. In clinical practice, the optimal timing and selection of candidates remain subjects of vigorous debate; although outcomes appear more favorable when delayed repair is feasible, surgical chronometry is frequently dictated by hemodynamic instability, necessitating high-risk emergent intervention.¹

I read with profound interest the report by Tuner et al.,² detailing a case of acute MI-VSD, where surgical intervention was deemed to carry a prohibitive risk. The patient's nearly 13-year survival, managed through a multifaceted percutaneous strategy, represents an extraordinary longevity that challenges conventional expectations. I concur with the authors' assertion regarding the pivotal role of the surgical and interventional teams'

expertise, a principle that is virtually axiomatic. As the adage goes, *Optima curatio, quam medicus optime novit* (the superior therapy is that which the clinician masters most proficiently). Nevertheless, beyond the subjective expertise of the Heart Team, specific nuances of the clinical course and the management strategy employed merit a more rigorous appraisal.

In a 65-year-old patient devoid of significant comorbidities or overt contraindications, surgical intervention remains the established standard of care.^{3,4} Based on the clinical vignette, surgery does not appear inherently futile; notably, the authors provide no specific exclusion criteria for a surgical approach, including advanced strategies such as the Total Artificial Heart (TAH) as a bridge to transplantation (BTT) in highly selected cohorts. TAH implantation represents a viable (and occasionally solitary) recourse for patients with post-MI VSD and extensive myocardial destruction precluding conventional tension-free repair, with published literature corroborating its role in achieving hemodynamic stabilization and subsequent recovery.^{5,6}

For patients in refractory cardiogenic shock ineligible for standard surgical or transcatheter repair, primary heart

How to cite: Orozco-Hernández EJ. Post-myocardial infarction ventricular septal defect closure: does the end justify the means?. *Cir Card Mex.* 2026; 11 (3): 125-127. <https://dx.doi.org/10.35366/123489>

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transplantation (though rarely employed and resource-intensive) remains a conceptual option. Contemporary data suggest superior in-hospital survival compared to other modalities, provided rigorous patient selection is maintained. In this context, mechanical circulatory support (MCS) serves as a critical bridge, notwithstanding the limitations of current data due to selection bias and restricted sample sizes.⁷

Regarding MCS selection, the authors utilized an intra-aortic balloon pump (IABP) in accordance with the 2023 ESC guidelines for acute coronary syndromes, which positioned IABP as the first-line support for MI-VSD. At that juncture, evidence favoring alternatives such as extracorporeal membrane oxygenation or micro-axial flow pumps (e.g., Impella 5.5) was deemed insufficient to mandate their primary use. However, evolving data now support these devices as superior initial strategies for left ventricular unloading. While definitive head-to-head evidence comparing IABP and Impella is still pending, the latter provides more robust hemodynamic support, albeit at the cost of a higher hemorrhagic risk.⁸

The decision to perform percutaneous coronary intervention (PCI) on the left main (LM) artery only two days after symptom onset raises significant clinical concerns. Such an indication appears dubious at best, likely carrying a marginal Class IIa recommendation. Given that MI-VSD is more frequently associated with right coronary artery occlusion (as in this instance) and absent any documented acute ischemia or myocardium at risk in the LM territory, the justification for stenting remains elusive. Current indications for LM intervention (including inoperability coupled with refractory angina, complex bifurcation disease, or iatrogenic dissection)^{9,10} do not appear to have been clearly met.

Furthermore, the optimal chronometry for VSD closure remains a subject of vigorous debate. The rationale for a delayed approach is well-established: allowing for infarct maturation and myocardial scarring to facilitate secure patch anchoring. The optimal chronometry for MI-VSD closure remains a subject of vigorous debate. The rationale for a delayed approach is well-established: allowing for infarct maturation and myocardial scarring to facilitate secure patch anchoring.

Current evidence advocates for an individualized strategy integrating hemodynamic status with specific defect characteristics; while elective repair within 1-2 weeks may be appropriate for stabilized patients, those refractory to medical therapy require emergent intervention.^{1,11,12} In the present case, the justification for opting for percutaneous closure at a mere 48 hours (rather than pursuing a stabilization-then-delay strategy) remains unaddressed.

Furthermore, during a protracted 38-day hospitalization characterized by persistent heart failure, the patient achieved only marginal improvement under conservative management. It is clinically pertinent to consider whether this course

could have been attenuated (and functional status optimized) through early implementation of temporary mechanical circulatory support (MCS), specifically an axillary Impella 5.5. By providing active left ventricular unloading, this device effectively mitigates pulmonary congestion while enhancing systemic perfusion.¹³

The patient's subsequent three-year clinical course, marked by recurrent heart failure exacerbations, multiple PCIs, and eventual transcatheter edge-to-edge repair, further invites scrutiny. Given the apparent absence of overt contraindications, a proactive evaluation for a left ventricular assist device as destination therapy would have been a highly appropriate consideration in this context.¹⁴

Despite these points of contention, the long-term survival reported is undeniably impressive for such a devastating mechanical complication. Nevertheless, the evolution of medical knowledge is a dynamic process; it is only through the rigorous examination of these diverse therapeutic angles that our management strategies for MI-VSD will become truly robust and refined.

In conclusion, the extraordinary long-term survival documented by Tuner et al.² underscores the inherent heterogeneity of MI-VSD and the potential for unconventional strategies to yield success in carefully selected cohorts. However, this case also exposes critical lacunae in our current decision-making frameworks—specifically regarding the stringency of surgical exclusion criteria, the strategic integration of advanced MCS, and the physiological rationale for early percutaneous closure. As clinical evidence evolves, we must remain prepared to challenge established paradigms through individualized, multidisciplinary inquiry. Ultimately, the management of MI-VSD demands more than technical proficiency; it requires a nuanced clinical judgment that acknowledges that progress in this field will continue to be refined by the passage of time and rigorous scrutiny (*veritas temporis filia*).

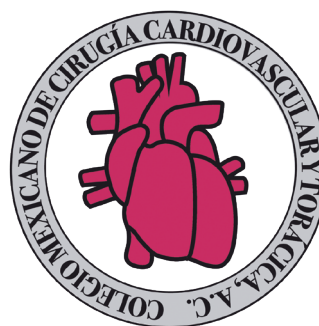
REFERENCES

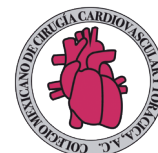
1. Goubran D, Issa H, Clarizia N, Chan V, Ruel M. Postmyocardial infarction ventricular septal rupture: optimizing surgical timing and repair. *Curr Opin Cardiol*. 2025;40(6):395-398. doi: 10.1097/HCO.0000000000001256.
2. Turner H, Townsend M, Strange J, Turner M. Long-term survival after post-myocardial infarction Ventricular Septal Defect (VSD) closure, multiple percutaneous coronary interventions and edge-to-edge repair. *Catheter Cardiovasc Interv*. 2025;106(6):3349-3354. doi: 10.1002/ccd.70197
3. Wang S, Liu H, Yang P, Wang Z, Chen S. Current understanding of timing of surgical repair for ventricular septal rupture following acute myocardial infarction. *Cardiology*. 2024;149(6):618-631. doi: 10.1159/000538967.
4. Schlöter F, Huber K, Hassager C, Halvorsen S, Vranckx P, Pöss J, et al. Ventricular septal defect complicating acute myocardial infarction: diagnosis and management. A clinical consensus statement of the

- Association for Acute CardioVascular Care (ACVC) of the ESC, the European Association of Percutaneous Cardiovascular Interventions (EAPCI) of the ESC and the ESC Working Group on Cardiovascular Surgery. *Eur Heart J*. 2024;45(28):2478-2492. doi: 10.1093/eurheartj/ehae363.
5. Fiebiger R, Dashkevich A, Nozdrzykowski M, Jozwiak-Nozdrzykowska J, Gjermeni E. Inpatient cardiac rehabilitation after implantation of a total artificial heart (Aeson device CARMAT) in case of ventricular septal defect after infarction. *Eur Heart J Case Rep*. 2025;9(10):ytaf478. doi: 10.1093/ehjcr/ytaf478.
 6. Huenges K, Panholzer B, Cremer J, Haneya A. Case report-CARMAT: the first experience with the Aeson bioprosthetic total artificial heart as a bridge to transplantation in a case of post-infarction ventricular septal rupture. *Front Cardiovasc Med*. 2023;10:1211365. doi: 10.3389/fcvm.2023.1211365.
 7. Hanna DB, Verghese D, Dakkak W, Sierra J, Navas V, Paz L, et al. Heart Transplantation in post-infarction ventricular septal rupture: contemporary outcomes from the 2016-2021 National Inpatient Database. *JHLT Open*. 2025;9:100278. doi: 10.1016/j.jhlto.2025.100278.
 8. Tariq MD, Jain H, Khan AM, Shahnoor S, Goyal P, Zulfikar E, et al. Efficacy and safety of percutaneous mechanical circulatory support in patients with cardiogenic shock following acute myocardial infarction: a meta-analysis of randomized controlled trials. *Medicine (Baltimore)*. 2024;103(46):e40595. doi: 10.1097/MD.00000000000040595.
 9. Sohail MM, Masood I, Goyal D, Rashid H. Single centre experience of rotablation-assisted left main percutaneous coronary intervention. *Am Heart J Plus*. 2025;61:100678. doi: 10.1016/j.ahjo.2025.100678.
 10. Bagh I, Patel RAG. Unprotected distal left main percutaneous intervention. *Prog Cardiovasc Dis*. 2025;88:53-59. doi: 10.1016/j.pcad.2024.12.006.
 11. Moros D, Maigrot JA, Tong MZY, Smedira NG, Soltesz EG, Bakaeen FG, et al. Temporary microaxial transvalvular left ventricular assist device for post-myocardial infarction ventricular septal rupture: bridging a paradigm shift. *JTCVS Tech*. 2024;28:97-108. doi: 10.1016/j.xjtc.2024.08.019.
 12. Ma D, Zhang Z, Zhang S, Wang Z, Zhang G, Wang C, et al. Treatment strategies for ventricular septal rupture after myocardial infarction: a single-center experience. *Front Cardiovasc Med*. 2022;9:843625. doi: 10.3389/fcvm.2022.843625.
 13. Jabri A, Kumar S, Shadid AM. A comprehensive review of left atrial venoarterial extracorporeal membrane oxygenation. *J Cardiothorac Vasc Anesth*. 2025;39(9):2468-2479. doi: 10.1053/j.jvca.2025.04.036.
 14. Saeed D, Feldman D, Banayosy AE, Birks E, Blume E, Cowger J, et al. The 2023 International Society for Heart and Lung Transplantation guidelines for mechanical circulatory support: a 10- year update. *J Heart Lung Transplant*. 2023;42(7):e1-e222. doi: 10.1016/j.healun.2022.12.004.

Funding: none.

Disclosure: the authors have no conflict of interest to disclose.





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