



Clinical features, echocardiographic findings and surgical aspects related to mortality in complicated endocarditis

Características clínicas, hallazgos ecocardiográficos y aspectos quirúrgicos relacionados con la mortalidad en endocarditis infecciosa complicada

Jessica Mariel Bazo-Medina,^{*,‡} Rodolfo de Jesus Castaño-Guerra^{*,§}

The journal *Cardiovascular and Metabolic Science* and the material contained therein are under the Creative Commons Attribution-NonCommercial-NoDerivatives (CC BY-NC-ND) license.



Keywords:

Infective Endocarditis, echocardiogram, complications, mortality.

Palabras clave:

endocarditis infecciosa, ecocardiograma, complicaciones, mortalidad.

ABSTRACT

Infective Endocarditis is a serious public health problem with high morbidity and mortality, and a considerable prevalence in our setting. International guidelines for the diagnosis and treatment of endocarditis consider clinical, laboratory, and imaging criteria to define this entity and establish recommendations for its management. Global mortality is estimated around 20%; however, there is no information available on the epidemiology or prognosis in Mexico. An important finding among patients at our centre is the incidence of cases with local complications. Naturally, as anatomical complexity increases, surgery becomes more challenging, so local complications are expected to directly influence the risk of perioperative death. The purpose of this study was to identify risk factors associated with echocardiographic complications in patients diagnosed with Infective Endocarditis (IE) at tertiary level public hospital, as well as to evaluate their postoperative outcome. This information could contribute to a better understanding of IE and facilitate more timely treatment. Over a five-year period, 60 patients were included, with an incidence of local complications of 73.3%. No variables were significantly associated with the development of local complications. However, type 2 diabetes ($p = 0.03$), heart failure ($p = 0.01$), and prosthetic valves ($p = 0.03$) were risk factors associated with mortality. Regarding clinical scenario, uncontrolled sepsis ($p = 0.02$), septic shock ($p = 0.01$) and multiple organ failure ($p = 0.001$) at the time of IE diagnosis and after surgery, had higher mortality ($p = 0.02$).

RESUMEN

La endocarditis infecciosa es un grave problema de salud pública con alta morbilidad, mortalidad y una prevalencia considerable en nuestro país. Las guías internacionales para el diagnóstico y tratamiento de la endocarditis consideran criterios clínicos, de laboratorio e imagenológicos que definen esta entidad y establecen recomendaciones para el algoritmo de estudio. Globalmente la mortalidad se estima en torno al 20%; sin embargo, no se dispone de información sobre la epidemiología ni el pronóstico en México. Un hallazgo importante en los pacientes de nuestro centro es la incidencia de casos con complicaciones locales. Naturalmente, cuanto más compleja es una lesión, más retadora puede tornarse la cirugía, por lo que se espera que las complicaciones locales influyan directamente el riesgo de muerte perioperatoria. El propósito de este estudio fue identificar los factores de riesgo asociados con complicaciones ecocardiográficas en pacientes con diagnóstico de Endocarditis Infecciosa (EI) en un hospital público de tercer nivel, así como evaluar su pronóstico postoperatorio. Esta información podría contribuir al mejor entendimiento de la EI y favorecer un tratamiento más oportuno. Se incluyeron sesenta pacientes durante un periodo de cinco años, con una incidencia de complicaciones locales del 73.3%. Ninguna de las variables evaluadas se asoció significativamente al desarrollo de complicaciones locales. Mientras que la diabetes tipo 2 ($p = 0.03$), la insuficiencia cardíaca ($p = 0.01$) y las prótesis valvulares ($p = 0.03$) fueron factores de riesgo asociados con la mortalidad. En cuanto al contexto clínico, la sepsis no controlada ($p = 0.02$), el choque séptico ($p = 0.01$), así como la falla multiorgánica ($p = 0.001$) en el momento del diagnóstico de EI ($p = 0.001$) y después de la cirugía se asociaron con una mayor mortalidad ($p = 0.02$).

* MD Cardiology Physician. Cardiology Department, General Hospital of Mexico «Dr. Eduardo Liceaga». Mexico.
ORCID:
[‡] 0000-0002-2765-8078
[§] 0000-0003-1335-9672

Received:
09/17/2025
Accepted:
03/12/2026

How to cite: Bazo-Medina JM, Castaño-Guerra RJ. Clinical features, echocardiographic findings and surgical aspects related to mortality in complicated endocarditis. *Cardiovasc Metab Sci.* 2026; 37 (1): 9-20. <https://dx.doi.org/10.35366/122889>

Abbreviations:

CHD = Congenital Heart Disease
CIE = Complicated Infective Endocarditis
IE = Infective Endocarditis
IVDU = Intravenous Drug Users
TOE = Transesophageal Echocardiography
TTE = Transthoracic Echocardiography

INTRODUCTION

Infective Endocarditis (IE) is a life-threatening disease characterized by inflammation of the valves, endocardium, and vascular intima caused by pathogenic microorganisms. It represents a major public health problem, with an estimated incidence of 13.8 cases per 100,000 people per year in 2019. To date, the increasing population at risk and the emergence of new clinical scenarios have raised these numbers.^{1,2}

In response to this more challenging landscape, recommendations for the diagnosis and treatment of IE have been updated, starting with the identification of susceptible patients with high and intermediate risk features^{2,3} such as previous IE, surgically implanted prosthetic valves or other materials including filters, grafts, closure devices and ventricular assist devices, and patients with Congenital Heart Disease (CHD). Likewise for right-sided IE, different vulnerable groups can be distinguished based on patients' characteristics, including Intravenous Drug Users (IVDU); IE in patients with pacemakers, implantable cardiac defibrillators or central venous catheters and IE in patients with right-heart congenital abnormalities.⁴

Transthoracic (TTE) and Transesophageal (TOE) echocardiography play a key role in the evaluation and prognostic assessment of patients with IE. Echocardiography is the imaging modality of choice for diagnosing IE (as it constitutes a major Duke criterion), and is essential for monitoring disease progression (particularly for the detection of complications) and guiding treatment. Findings must be interpreted with caution, always considering the patient's clinical presentation. Notably, a negative echocardiographic examination does not rule out IE, and repeating TTE and TOE may be necessary in certain situations.⁴

The echocardiographic diagnosis of IE is primarily based on vegetations finding,

assessment of the degree of valvular and perivalvular damage, evaluation of resulting hemodynamic abnormalities, and identification of associated complications.⁵

Vegetations remain the hallmark lesion of IE. They typically appear as oscillating or non-mobile masses attached to valvular structures and may be located anywhere on the valvular apparatus, prosthetic intracardiac materials, or mural endocardium. Identification of vegetations can be challenging in the presence of pre-existing valvular lesions (such as mitral valve prolapse, degenerative calcified leaflets, or calcified mitral annulus), prosthetic materials, or small vegetation size. Embolization before echocardiographic examination is another potential cause of a false-negative result.^{6,7}

Perivalvular extension of infection leads to the development of abscesses, pseudoaneurysms, fistulae and new prosthetic dehiscence (*Figure 1*).^{6,7}

Abscesses typically present as thickened, heterogeneous perivalvular areas with echodense or echolucent appearances, without detectable color Doppler flow within. In contrast, pseudoaneurysms appear as pulsatile, perivalvular echo-free spaces containing color Doppler flow.⁴ Fistula formation may occur as a complication of abscesses or pseudoaneurysms; however, in some cases, fistulous tracts develop as a direct consequence of infection due to tissue necrosis and rupture without prior abscess formation. Echocardiographically, they are documented when a color Doppler flow jet is observed communicating two adjacent cavities, and they may be misinterpreted as perforated valve aneurysms.^{8,9}

Leaflet perforation is practically pathognomonic of leaflet infection, while prosthetic valve dehiscence may present clinically with hemolytic anemia. IE should also be suspected in cases of new perivalvular regurgitation, even in the absence of visible vegetations or other periannular complications.⁴

The in-hospital prognosis of IE is influenced by four main factors: patient characteristics, the causative microorganism, and the presence or absence of cardiac and non-cardiac complications.^{10,11} Within this context, the most important factors affecting clinical outcomes include congestive

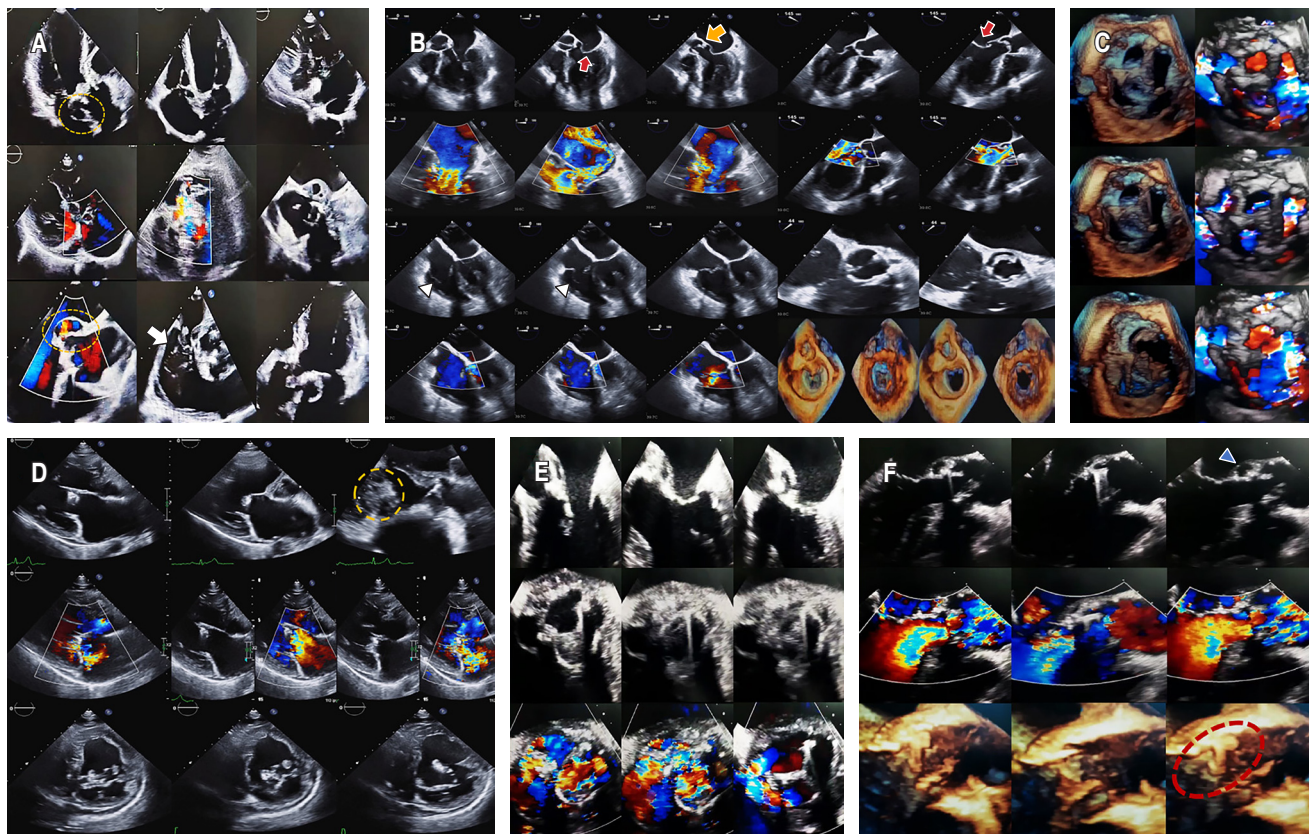


Figure 1: **A)** Apical and parasternal short-axis views of a case of bioprosthetic aortic valve endocarditis complicated by aortic root abscess (dotted circle), fistula draining into the right atrium, and native valve endocarditis of the tricuspid valve, which presents a large, non-mobile vegetation dependent on the septal leaflet (white arrow). **B)** TOE projections and 3D reconstruction. In the first row A3 at 0°, excursion of the thin mitral valve leaflets and prolapse of A2, as well as prolapse of the right coronary cuspid of the aortic valve (yellow arrow) can be seen. At second row colour Doppler shows 2 mitral regurgitation jets, the largest of which runs in a posterolateral direction towards the left atrial wall, reaching its roof, and the aortic valve regurgitation jet spanning the length of the LVOT. Below A4 at 0°, shows thin and elongated TV leaflets with anterior leaflet prolapse (white triangle). Right side A3 at 145° focused on the LVOT with colour and PSAX with zoom on the aortic bicuspid valve, which has sclerotic leaflets and an ovoid lesion suggestive of vegetation. 3D reconstruction of the atrial view and ventricle view where a perforation is observed in the anterior veil of the left ventricle. **C)** 3D reconstruction from the atrial view of a mitral bioprosthesis during the cardiac cycle. Color Doppler imaging reveals the presence of an anteroseptal paravalvular leak and mitral annulus dehiscence in the posterolateral region. **D)** A case of congenitally corrected transposition of the great arteries complicated by pulmonary valve endocarditis with ventricular septal defect. The patient was characterized by situs solitus with a morphologically right ventricle, Tricuspid atrioventricular Valve (TV) with severe regurgitation, and muscular Ventricular Septal Defect (VSD) with adherent vegetation extending toward the tricuspid septal leaflet. Also, discordant ventriculoarterial connection with multiple vegetations in the pulmonary artery trunk and bifurcation, leading to severe supra-valvular pulmonary stenosis. **E)** Mitral and aortic endocarditis, with multiple vegetations causing severe regurgitation in both cases. A close-up of the A3 and bicommissural projection of a transesophageal echocardiogram in two-dimensional and color Doppler mode is shown. **F)** Native aortic valve endocarditis complicated by aortic root abscess (blue triangle). Multiple jets associated with valve perforation and the presence of several vegetations are seen (dotted oval).

heart failure, valvular dysfunction, and thromboembolic events.¹²⁻¹⁴

Furthermore, perivalvular extension of IE is the most common cause of uncontrolled infection and is associated with poor prognosis and a high likelihood of requiring surgery.⁴

It stands to reason that the more complex the injury, the more difficult the surgery may be. Additionally, such complexity implies longer cardiopulmonary bypass times, which in turn exacerbates the inflammatory response, promote greater bleeding, and increase the risk of coagulopathy and associated metabolic disorders. Therefore, local complications are

expected to directly influence perioperative mortality risk.

Despite advances in medical therapies and surgical techniques, the morbidity and mortality of IE remain high, with up to one in five deaths occurring during the initial hospitalization.^{15,16} Given its poor prognosis and high mortality, early diagnosis and timely intervention are of paramount clinical importance.⁵

MATERIAL AND METHODS

This was a retrospective cohort study conducted at a tertiary-level public hospital, covering a five-year period from January 1, 2019, to December 31, 2023.

The study included adult patients (≥ 18 years), both men and women, who had been hospitalized at our institution with a confirmed or suspected diagnosis of Infective Endocarditis. All information was obtained exclusively from existing medical records and surgical reports. No direct patient contact or intervention was performed. The data were used to compare echocardiographic descriptions with intraoperative findings and to verify the presence of local complications.

Because this study involved only the review of anonymized clinical records, no informed consent was required.

All patients included in the study had previously undergone transthoracic echocardiography, and in selected cases, transesophageal echocardiography, performed during their hospitalization using a Philips 7C model 01800 253 0446. Surgical indications in each case had been determined according to the 2015 and 2023 ESC guidelines for the diagnosis and treatment of Infective Endocarditis.

Information regarding intraoperative findings, hemodynamic conditions, and outcomes was extracted from surgical and postoperative records. For patients with fatal outcomes, intraoperative events were reviewed as documented in the clinical files. Postoperative courses and complications were compared between the two groups based on the information available in the medical records.

Statistical analyses were performed using SPSS version 25. Qualitative variables are expressed as frequencies and percentages, while quantitative variables with non-normal distributions are

Table 1: Demographic and clinical features of patients with Infective Endocarditis (N = 60).

	Uncomplicated IE n (%)	Complicated IE n (%)
Sex		
Men	9 (56.3)	35 (79.5)
Women	7 (43.8)	9 (20.5)
Age group (years), %		
18 to 30	17.6	34.9
31 to 43	17.6	20.9
44 to 56	23.5	11.6
57 to 69	23.5	23.3
≥ 70	17.6	9.3
Clinical presentation		
Uncontrolled sepsis	15 (93.8)	38 (86.4)
Systemic embolism	8 (50.0)	20 (45.5)
Septic shock	6 (37.5)	17 (38.6)
Acute heart failure	5 (31.3)	19 (43.2)
Cardiogenic shock	–	1 (2.3)
Acute coronary syndrome	–	1 (2.3)
AV block/arrhythmia	–	3 (6.8)
Stroke	1 (6.3)	6 (13.6)
Multiorgan failure	3 (18.8)	19 (43.2)
Diagnosis of Infective Endocarditis		
Duke criteria		
Two major criteria	12 (75.0)	13 (29.5)
One major and 3 minor criteria	4 (25.0)	31 (70.5)
Native valve	13 (81.3)	32 (72.7)
Prosthetic valve	3 (18.8)	12 (27.3)

IE = Infective Endocarditis.

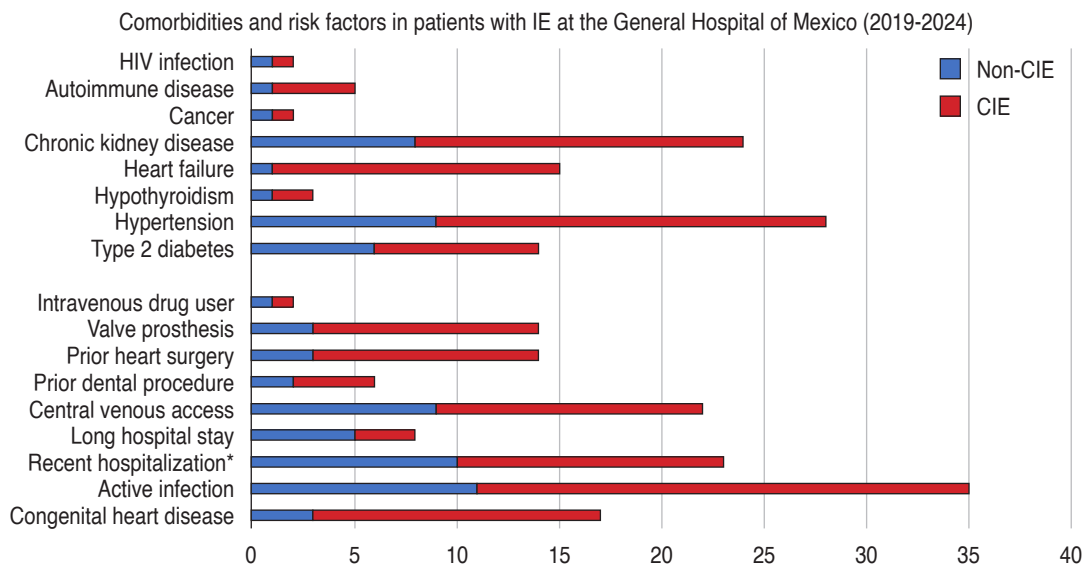


Figure 2: Graphical representation of the most prevalent comorbidities and predisposing conditions in patients with uncomplicated and complicated IE.

* Within the last 3 months. CIE = Complicated Infective Endocarditis. IE = Infective Endocarditis.

Table 2: Congenital heart disease identified in patients with Infective Endocarditis.

	Uncomplicated IE	Complicated IE
	N = 3 (18.8%)	N = 13 (31.8%)
	n	n
Bicuspid aortic valve	1	7
Patent ductus arteriosus	0	1
Atrial septal defect	0	3
Ventricular septal defect	2	1
Transposition of the great arteries	0	1

IE = Infective Endocarditis.

described as medians and interquartile ranges. Group comparisons were carried out using the χ^2 test for categorical variables and the Mann-Whitney U test for continuous variables. A 95% confidence level was used, and p-values < 0.05 were considered statistically significant.

RESULTS

A sample of 60 patients was collected, ensuring at least one case of IE each was reported per

month. The demographic and clinical features of our sample are summarized in *Table 1*.

There were nearly three times as many male cases, and in 80% of these, men were also the most affected by local complications. Notably, the largest age subgroup among patients with CIE was that of young patients under 30 years of age (34.9%), although this group represented only 35% of non-survivors. Complicated cases were three times more common in patients with prosthetic valves.

Information on comorbidities and known predisposing factors was recorded (*Figure 2*). The most prevalent chronic conditions were hypertension, chronic kidney disease, heart failure, and type 2 diabetes. Other immunosuppressive conditions such as HIV infection, cancer, and autoimmune diseases, were also present but albeit in only 15% of patients.

As outlined in the 2023 ESC guidelines, several cardiac and non-cardiac risk factors may increase susceptibility to Infective Endocarditis. In a targeted search for such risk factors, the most frequently observed were recent hospitalization (within one month prior to diagnosis), the presence of central venous access, and ongoing infections with gastrointestinal, respiratory,

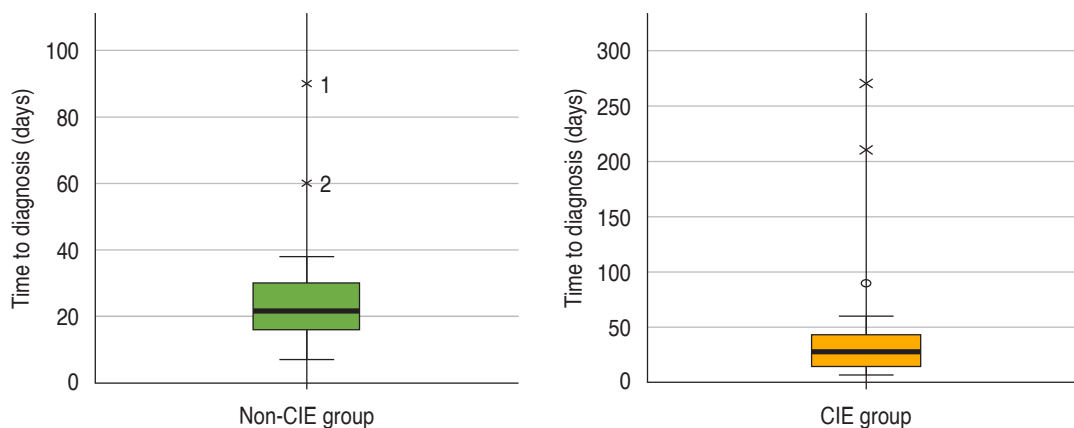


Figure 3: Comparison of time to diagnosis between uncomplicated and Complicated Infective Endocarditis: graphical representation.

CIE = Complicated Infective Endocarditis.

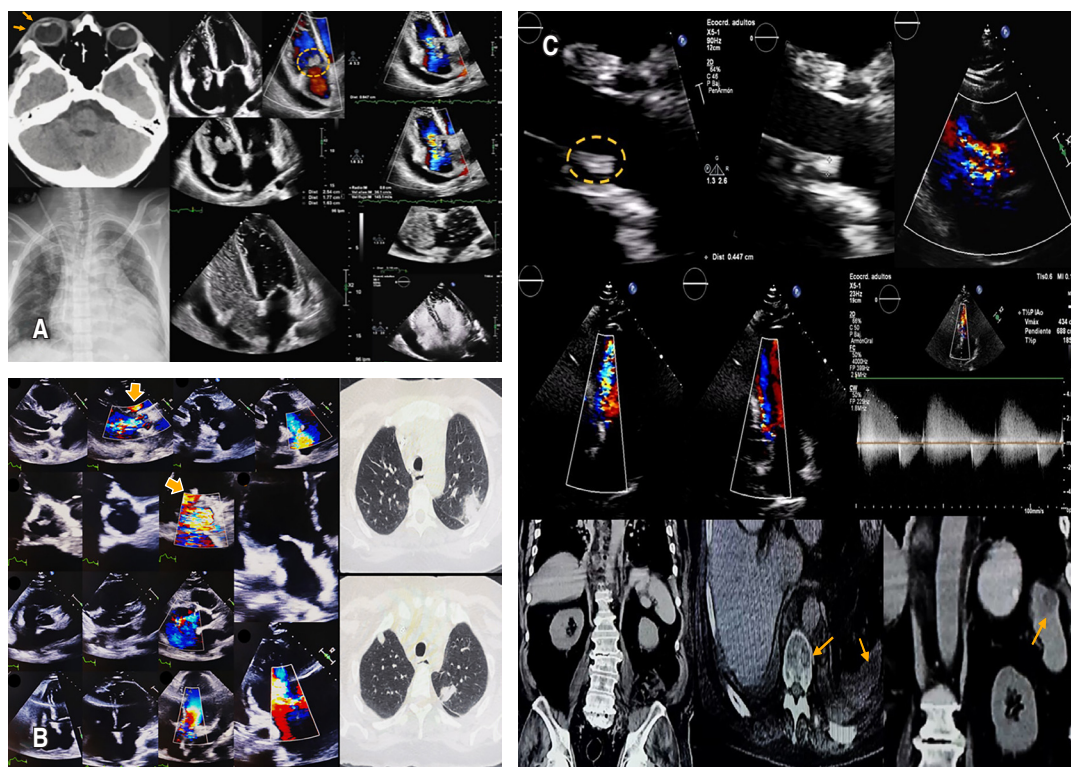


Figure 4: **A)** Case of native tricuspid valve Infective Endocarditis with a previously undiagnosed atrial septal defect. The infection spread to the central nervous system, including the ophthalmic artery and also embolizing into the pulmonary circulation the patient presented to the emergency department from the hemodialysis unit with fever and right eye proptosis (yellow arrows). **B)** Infective Endocarditis of the aortic and tricuspid valves in a patient with a subaortic ventricular septal defect (yellow arrows), who developed multifocal pneumonia secondary to septic embolism. **C)** Native aortic valve Infective Endocarditis in an elderly patient presenting with acute abdomen due to mesenteric ischemia. During diagnostic evaluation, and in the context of persistent sepsis, multiple splenic emboli and infectious spondylodiscitis were identified (yellow arrows).

genitourinary, or soft tissue foci. Although CHD was present in 28.3% of all patients, it had not been previously diagnosed in 94% of

these cases and was only discovered upon the diagnosis of endocarditis (Table 2).

Interestingly, none of these factors appeared to be associated with an increased risk of local complications. However, with regard to mortality in global population, diabetes (p = 0.03), heart failure (p = 0.01), and prosthetic valves (p = 0.03) were statistically significant predictors. In fact, patients with prosthetic valve Infective Endocarditis had five-fold higher odds of in-hospital mortality compared to those with native valve endocarditis (OR 5.0, 95%CI 1.24-20.18; p = 0.017).

We also recorded the time from the onset of initial symptoms to the formal diagnosis of IE. Longer progression periods were observed in complicated cases, with a maximum duration of 270 days; however, no statistically significant differences were found when compared to patients without local complications (Figure 3).

Clinical presentation at diagnosis was also analyzed. In both groups, uncontrolled sepsis and systemic embolism were the most frequent presentations. Among patient with locally complicated IE, acute heart failure, septic shock, and multi-organ failure were also prominent diagnostic indicators (Table 1).

Regarding systemic embolism, affected sites included the lungs, spleen, kidneys, central nervous system, eyes, musculoskeletal system, pancreas, and mesenteric circulation (Figure 4). Stroke was the cause of hospitalization in 11.6% of the overall cohort.

Other cardiac complications were documented exclusively in patients with local complications, including third-degree atrioventricular block, acute coronary syndrome, cardiogenic shock, and even cardiac tamponade.

Most cases met 1 major and 3 minor Duke criteria for diagnosis, particularly embolic phenomena, persistent fever and less frequently autoimmunity manifesting Roth spots and glomerulonephritis in a couple of cases. Interestingly, in the uncomplicated subgroup, three times as many cases were diagnosed using both major criteria.

The microbiological profile of patients with Infective Endocarditis is displayed in Table 3 and Figure 5, as well as in Tables 4 and 5. Microbiological isolation was obtained

Table 3: Microbiological distribution according to echocardiographic findings in Infective Endocarditis (N = 60).

Isolated microorganism	Uncomplicated IE	Complicated IE
	n	n
<i>Staphylococcus aureus</i> MR	2	3
<i>Staphylococcus aureus</i> MS	1	10
<i>Staphylococcus epidermidis</i>	2	3
<i>Staphylococcus haemolyticus</i>	0	1
<i>Staphylococcus lugdunensis</i>	1	0
<i>Streptococcus anginosus</i>	0	1
<i>Streptococcus mitis</i>	1	0
<i>Enterococcus faecium</i> MR	1	0
<i>Enterococcus faecium</i> MS	0	1
<i>Enterococcus faecalis</i>	0	2
<i>Escherichia coli</i> (ESBL)	1	1
<i>Enterobacter cloacae</i>	1	1
<i>Haemophilus parainfluenzae</i>	0	1
<i>Klebsiella</i> spp.	1	0
<i>Salmonella</i> spp.	1	0
<i>Pseudomonas</i>	1	1
<i>Stenotrophomonas maltophilia</i>	0	2
Non microbiological identified	2	12
Data not available*	1	5

* Since some patients were already receiving treatment for suspected endocarditis at hospital admission, culture samples were not available in all cases, and microbiological data could not be retrieved from clinical records. IE = Infective Endocarditis.

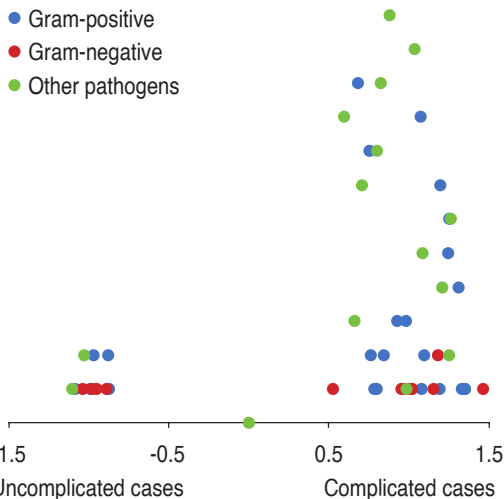


Figure 5:

Scatter plot showing the microbiological distribution between groups of complicated and uncomplicated Infective Endocarditis by gram stain phenotype.

Table 4: Distribution of isolated microorganisms in native and prosthetic valve Infective Endocarditis (N = 60).

Isolated microorganism	Native n	Prosthetic n
<i>Staphylococcus aureus</i> MR	5	0
<i>Staphylococcus aureus</i> MS	9	2
<i>Staphylococcus epidermidis</i>	1	4
<i>Staphylococcus haemolyticus</i>	1	0
<i>Staphylococcus lugdunensis</i>	1	0
<i>Streptococcus anginosus</i>	0	1
<i>Streptococcus mitis</i>	1	0
<i>Enterococcus faecium</i> MR	1	0
<i>Enterococcus faecium</i> MS	1	0
<i>Enterococcus faecalis</i>	2	0
<i>Escherichia coli</i> (ESBL)	1	1
<i>Enterobacter cloacae</i>	1	1
<i>Haemophilus parainfluenzae</i>	1	0
<i>Klebsiella spp.</i>	1	0
<i>Salmonella spp.</i>	1	0
<i>Pseudomonas</i>	2	0
<i>Stenotrophomonas maltophilia</i>	2	0
Non microbiological identified	10	4
Data not available*	5	1

* Since some patients were already receiving treatment for suspected endocarditis at hospital admission, culture samples were not available in all cases, and microbiological data could not be retrieved from clinical records.

from blood cultures; tissue cultures were not routinely performed due to logistical and resource limitations, and therefore were unavailable in a substantial number of cases. While the latest 2017-2023 report from the PUCRA network of hospitals in Mexico reported *E. coli* as the most frequently isolated microorganism,¹⁷ this bacterium appeared in only 6% of the total cases, while *Staphylococcus aureus* had a prevalence of 26%.

The empirical treatment until identification of the etiological agent in each case was based on the epidemiological profile of our centre and the management algorithm established by the Infectious Diseases Service (Table 6).

Echocardiographic findings of Infective Endocarditis are summarized in Table 7. The aortic valve was the most commonly affected. In 26.6% of cases, two or more valves were involved,

sometimes simultaneously on both left and right sides in the presence of structural defects. Other sites of involvement included the right atrium, right ventricular outflow tract, Eustachian valve, and moderator band. Vegetation size exceeded 20 mm in 20% of cases. Valvular regurgitation—rather than obstruction—was the predominant dysfunction, ranging from moderate to severe in 86.6% of patients. Echocardiographic signs of local complications extended to the interatrial and interventricular septum, aortic root, mitral-aortic continuity, sinuses of Valsalva, and even the pulmonary artery trunk.

Overall post-surgical mortality was 53.3%, with 31.3% in the uncomplicated group and 61.4% in the CIE group. Among patients who underwent surgery (Figure 6), postoperative outcomes were categorized and compared. Among non-survivors, emergency surgery was required in 32.1% of cases, at least two valves were replaced in 32.1%, additional procedures beyond valve replacement were performed in 42.9%, intraoperative defibrillation was needed in 42.9%, and 57.1% experienced hemodynamic instability (Table 8). These findings reflect a highly adverse clinical scenario before and during surgery, which severely reduced the chances of survival.

No significant association was found between CIE and postoperative complications aside from mortality. Multi-organ failure as a postoperative complication was associated with an 83.3% mortality rate ($p = 0.02$). Cardiogenic shock and superinfection were more common among fatal cases, though these did not reach statistical significance (Figure 7).

Finally variables significantly associated with mortality in univariate analysis and clinically relevant baseline characteristics were included in a multivariable logistic regression model. The analysis showed that diabetes remained independently associated with in-hospital mortality (adjusted OR 4.4, 95%CI 1.0-18.9; $p = 0.049$), whereas prosthetic valve endocarditis was no longer significantly associated with mortality after adjustment.

DISCUSSION

Although our analysis did not allow us to identify specific demographic or clinical characteristics

Table 5: Microbiological distribution according to in-hospital mortality in Infective Endocarditis (N = 60).

Isolated microorganism	Survivors n	Non-survivors n
<i>Staphylococcus aureus</i> MR	2	3
<i>Staphylococcus aureus</i> MS	3	8
<i>Staphylococcus epidermidis</i>	1	4
<i>Staphylococcus haemolyticus</i>	0	1
<i>Staphylococcus lugdunensis</i>	0	1
<i>Streptococcus anginosus</i>	0	1
<i>Streptococcus mitis</i>	1	0
<i>Enterococcus faecium</i> MR	1	0
<i>Enterococcus faecium</i> MS	0	1
<i>Enterococcus faecalis</i>	2	0
<i>Escherichia coli</i> (ESBL)	2	0
<i>Enterobacter cloacae</i>	1	1
<i>Haemophilus parainfluenzae</i>	1	0
<i>Klebsiella spp.</i>	1	0
<i>Salmonella spp.</i>	0	1
<i>Pseudomonas</i>	0	2
<i>Stenotrophomonas maltophilia</i>	2	0
Non microbiological identified	7	7
Data not available*	3	3

* Since some patients were already receiving treatment for suspected endocarditis at hospital admission, culture samples were not available in all cases, and microbiological data could not be retrieved from clinical records.

that clearly predisposed patients to the development of intracardiac complications, we did observe a direct association between the presence of such complications and a higher risk of perioperative mortality. This finding underscores the clinical relevance of early recognition of locally Complicated Infective Endocarditis (CIE), particularly through echocardiographic evaluation, as these complications significantly affect prognosis.

With regard to comorbidities, type 2 diabetes and heart failure were more frequently observed among deceased patients. However, it is important to emphasize that these conditions were not independently associated with the development of local complications. Rather, the majority of fatal cases corresponded to patients who already presented echocardiographic evidence of periannular extension or other structural damage at the time of diagnosis. This may suggest that comorbidities exert their impact indirectly, primarily by worsening systemic decompensation once complications emerge, rather than functioning as direct primary risk factors for CIE itself.

Although prosthetic valve Infective Endocarditis was associated with a five-fold increase in mortality, the relatively small prosthetic subgroup (n = 15) may have led to instability in the effect estimate. The wide confidence interval suggests limited precision,

Table 6: Empirical antimicrobial therapy for Infective Endocarditis (IE) without an identified pathogen.

Infection type	First-line treatment	Second-line treatment
Native valve non-IV drug user	Penicillin G 20 million IU IV daily or Ampicillin 12 g IV daily plus Dicloxacillin 2 g IV q6h Gentamicin 1 mg/kg IV or IM q8h	Vancomycin 15 mg/kg q12h plus Gentamicin 1 mg/kg or IM q8h
Native valve IV drug user	Dicloxacillin 2 g IV q6h	Vancomycin 15 mg/kg q12h plus Gentamicin 1 mg/kg or IM q8h
Prosthetic valve early IE (< 2 months postoperative)	Vancomycin 15 mg/kg q12h plus Gentamicin 1 mg/kg IV or IM q8h plus Rifampin 600 mg daily	
Prosthetic valve late IE (> 2 months postoperative)	Ampicillin 12 g IV daily plus Dicloxacillin 1 g IV q6h Gentamicin 1 mg/kg q8h	Vancomycin 15 mg/kg q12h plus Gentamicin 1 mg/kg or IM q8h

Table 7: Echocardiographic findings in patients with complicated Infected Endocarditis (IE).

	Survivors N = 28 n (%)	Non- survivors N = 32 n (%)
Affected valve (isolated involvement)		
Aortic	5 (17.9)	6 (18.7)
Mitral	4 (14.2)	3 (9.4)
Tricuspid	7 (25.0)	4 (12.5)
Pulmonary	–	3 (9.4)
Non-valvular involved structures*	7 (25.0)	3 (9.4)
Multiple valves affected	5 (17.9)	14 (43.7)
Regurgitation or paravalvular leak		
Moderate	11 (39.3)	15 (47.0)
Severe	12 (43.0)	14 (43.7)
Obstruction or stenosis		
Severe	2 (7.1)	2 (6.2)
Haemodynamic disturbances not related to IE		
AV moderate to severe regurgitation	3 (10.7)	13 (40.6)
Presence of multiple vegetations	10 (35.7)	14 (43.7)
Local complications**		
Valve perforation	8 (28.5)	10 (31.2)
Chord tendon rupture	2 (7.1)	1 (3.1)
Papillary muscle rupture	–	1 (3.1)
Abscess	2 (7.1)	6 (18.7)
Extension to adjacent structures	2 (7.1)	7 (21.9)
Fistula	1 (3.6)	3 (9.4)
Prosthetic valve dehiscence	1 (3.6)	6 (18.7)
Vegetations size***		
Size between 10 to 20 mm	19 (68.0)	21 (65.6)
Greater than 20 mm	7 (25.0)	5 (15.6)

* In one patient we found non-valvular structure and valve involvement simultaneously.

** In some cases, more than one local complication was detected in echocardiographic studies in same patient.

*** Dimension from the major axis of the vegetation was taken into consideration for each case. Also, smaller vegetations were documented on few cases.

and larger studies are needed to confirm this association.

An additional aspect that deserves attention is the frequent delay in diagnosis observed in our cohort. In many cases, patients were initially managed under the suspicion of alternative conditions due to the clinical presentation dominated by systemic embolism

(e.g., stroke, splenic or renal infarction). This often postponed the recognition of IE as the underlying cause, thereby extending the time from symptom onset to definitive diagnosis. Such diagnostic delays may have contributed to the advanced stage at which local complications were identified, further worsening prognosis and limiting surgical outcomes.¹²

Several limitations must be acknowledged. First, the retrospective design of this study carries an inherent risk of information bias, as data collection relied on medical records and echocardiographic reports. Second, the relatively small sample size limited the statistical power to detect additional associations between predisposing factors and outcomes. Third, the absence of advanced imaging modalities such as cardiac CT or PET/CT, which are increasingly used to improve diagnostic accuracy in Infective Endocarditis, may have led to an underestimation of the true prevalence of local complications.⁷

Despite these limitations, our findings highlight the prognostic significance of echocardiographic complications in IE and reinforce the importance of multidisciplinary management.¹⁶ Early diagnosis and timely surgical intervention remain crucial strategies to improve outcomes, particularly in resource-limited settings such as large public hospitals, where delays in referral and treatment are frequent.

CONCLUSIONS

In this cohort, patients who developed uncontrolled sepsis, septic shock, or multiple organ failure at the time of IE diagnosis had significantly higher mortality. No additional risk factors demonstrated statistical significance as predictors of local complications. However, patients with Complicated Infective Endocarditis (CIE) presented a 3.4-fold higher risk of death compared with those without local complications.

Despite the absence of statistically significant predictors for the development of CIE, mortality was notably influenced by pre-existing comorbidities such as diabetes, heart failure, and prosthetic valves. Moreover, echocardiographic findings confirmed the

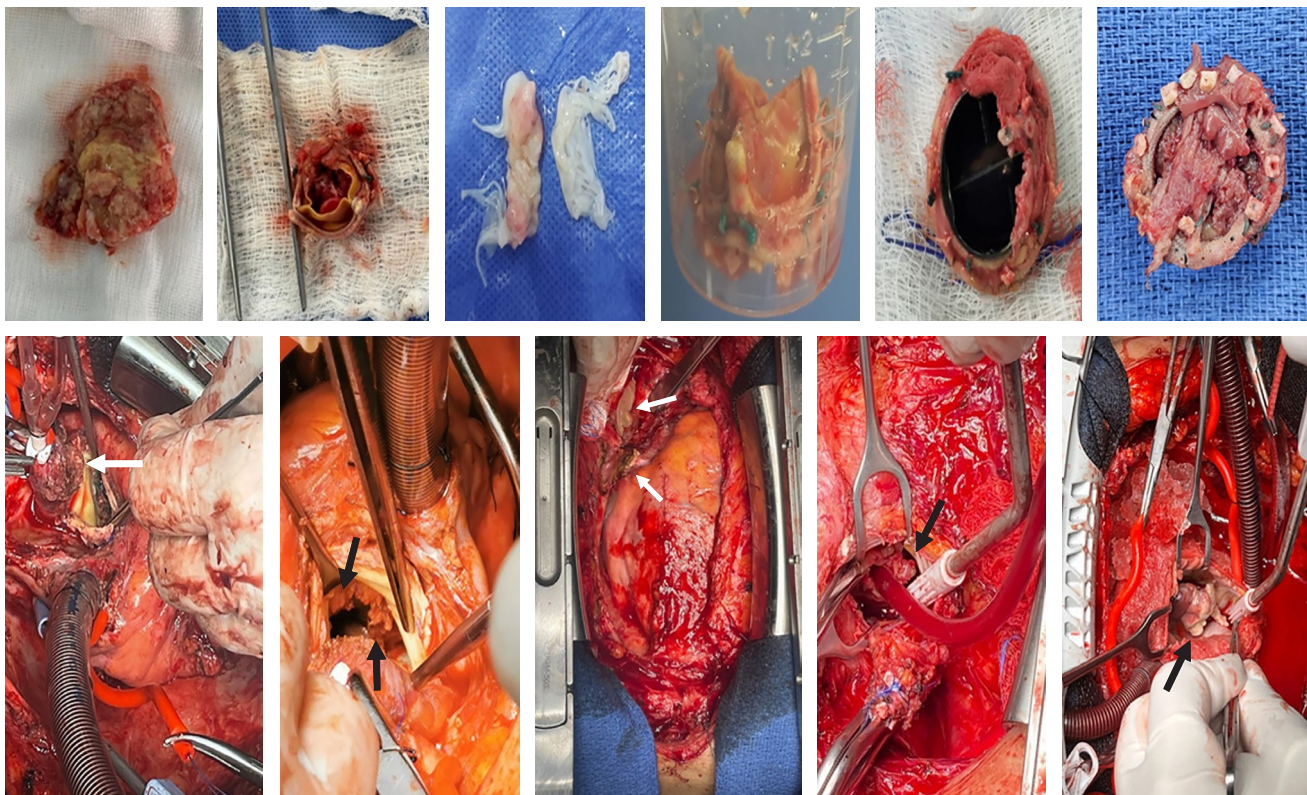


Figure 6: Top row, from left to right: infected thrombus; infected biological prosthetic valve with vegetations adhering to the leaflets; tricuspid valve showing a perforated anterior leaflet and a large vegetation on the posterior leaflet; bioprosthesis with purulent material and vegetations on the suture ring; infected double-disc mechanical valve prosthesis; bioprosthesis covered with granulation tissue, vegetations, and pus. Bottom row: intraoperative photographs taken during cardiopulmonary bypass, showing friable tissue, purulent discharge, and vegetations on the atrioventricular and aortic valves.

Table 8: Most relevant characteristics of valve replacement surgery in mortal patients with Infective Endocarditis (N = 28).

	n (%)
Type of intervention	
Emergency surgery	9 (32.1)
Urgent surgery	19 (67.9)
Valve replacement	
Two or more valves	9 (32.1)
Additional procedure	12 (42.9)
Time in cardiopulmonary bypass (min)*	153.8 ± 73.9
Transoperative bleeding (mL)**	925 [P50]
Need for cardiac defibrillation	12 (42.8)
Use of vasopressors prior to surgery	16 (57.1)
Use of vasopressors after surgery	16 (57.1)
Deaths during surgery	2 (7.0)

* Data expressed in mean ± SD.

** Data expressed in median [P50].

SD = standart deviation.

central role of local complications in adverse outcomes, especially when associated with severe valvular dysfunction, systemic embolism, or conduction disorders.

These findings reinforce the importance of early recognition of systemic and cardiac complications at the time of IE diagnosis. The high perioperative mortality observed in CIE highlights the need for timely diagnosis, multidisciplinary management, and rapid surgical decision-making to improve patient prognosis.

ACKNOWLEDGEMENTS

We appreciate the collaboration from other members of the Cardiology staff specially Dr. Jhasiel Vladimir Villa Alcaraz and Dr. Pamela Milijaed Muñoz Reyes; members of the Echocardiographic Department and Cardiothoracic Surgery Department.

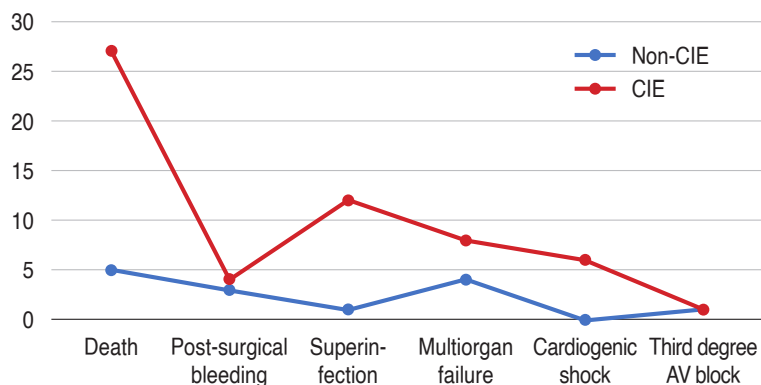


Figure 7: Incidence of postoperative complications in uncomplicated and locally Complicated Infective Endocarditis, graphical comparison. CIE = Complicated Infective Endocarditis.

REFERENCES

1. Imazio M. The 2023 new European guidelines on infective endocarditis: main novelties and implications for clinical practice. *J Cardiovasc Med (Hagerstown)*. 2024; 25 (10): 718-726.
2. Delgado V, Ajmone Marsan N, de Waha S, Bonaros N, Brida M, Burri H et al. 2023 ESC Guidelines for the management of endocarditis. *Eur Heart J*. 2023; 44 (39): 3948-4042.
3. Habib G, Lancellotti P, Antunes MJ, Bongjorni MG, Casalta JP, Del Zotti F et al. 2015 ESC Guidelines for the management of infective endocarditis: The Task Force for the Management of Infective Endocarditis of the European Society of Cardiology (ESC). Endorsed by: European Association for Cardio-Thoracic Surgery (EACTS), the European Association of Nuclear Medicine (EANM). *Eur Heart J*. 2015; 36 (44): 3075-3128.
4. Petersen JK, Østergaard L, Fosbøl EL. Role of echocardiography in the diagnosis and clinical management of infective endocarditis. *Indian J Thorac Cardiovasc Surg*. 2024; 40 (Suppl 1): 16-28.
5. Yuan XC, Liu M, Hu J, Zeng X, Zhou AY, Chen L. Diagnosis of infective endocarditis using echocardiography. *Medicine (Baltimore)*. 2019; 98 (38): e17141.
6. Habib G, Badano L, Tribouilloy C, Vilacosta I, Zamorano JL, Galderisi M et al. Recommendations for the practice of echocardiography in infective endocarditis. *Eur J Echocardiogr*. 2010; 11 (2): 202-219.
7. Galzerano D, Kinsara AJ, Di Michele S, Vríz O, Fadel BM, Musci RL, Galderisi M, Al Sergani H, Colonna P. Three dimensional transesophageal echocardiography: a missing link in infective endocarditis imaging? *Int J Cardiovasc Imaging*. 2020; 36 (3): 403-413.
8. Anguera I, Miro JM, Vilacosta I, Almirante B, Anguita M, Muñoz P et al. Aorto-cavitary fistulous tract formation in infective endocarditis: clinical and echocardiographic features of 76 cases and risk factors for mortality. *Eur Heart J*. 2005; 26 (3): 288-297.

9. Saadia S, Sultan FAT, Iqbal S, Fatimi SH, Nasir A. Case report: Aorto-left atrial fistula-A rare complication of native aortic valve endocarditis. *Egypt Heart J*. 2023; 75 (1): 58.
10. San Román JA, López J, Vilacosta I, Luaces M, Sarriá C, Revilla A et al. Prognostic stratification of patients with left-sided endocarditis determined at admission. *Am J Med*. 2007; 120 (4): 369.e1-369.e7.
11. Paixao MR, Besen BAMP, Felicio MF, Pocebon LZ, Furtado RHM, de Barros E Silva PGM et al. Prediction of hospital mortality in patients with left-sided infective endocarditis using a score in the first hours of admission. *Sci Rep*. 2025; 15 (1): 28566.
12. Sanguettoli F, Marchini F, Frascaro F, Zanarelli L, Campo G, Sinning C et al. The impact of neurological complications in endocarditis: a systematic review and meta-analysis. *J Clin Med*. 2024; 13 (23): 7053.
13. Becker JB, Moisés VA, Barbosa DA. Clinical aspects and short-term prognosis in a cohort of patients with infective endocarditis, São Paulo, Brazil. *Rev Esc Enferm USP*. 59:e20250060.
14. Motoc A, Kessels J, Roosens B, Lacor P, Van de Veire N, De Sutter J et al. Impact of the initial clinical presentation on the outcome of patients with infective endocarditis. *Cardiol J*. 2023; 30 (3): 385-390.
15. Miró JM, Ambrosioni J. Infective endocarditis: an ongoing global challenge. *Eur Heart J*. 2019; 40 (39): 3233-3236.
16. Lau L, Baddour L, Fernández-Hidalgo N, Brothers TD, Kong WKF, Borger MA et al. Infective endocarditis: it takes a team. *Eur Heart J*. 2025; 46 (24): 2275-2288.
17. Universidad Nacional Autónoma de México. Plan Universitario de Control de la Resistencia Antimicrobiana (PUCRA). Resistencia antimicrobiana en México 2017-2023: reporte de los hospitales de la Red PUCRA: resistencia antimicrobiana y consumo de antibióticos. Ciudad de México: Universidad Nacional Autónoma de México; 2024.

Declaration of confidentiality and patients consent: we declare that the entire information from patient's records is confidential and had been used with strictly academic purposes. However, data has been de-identified.

Clinical trial registration and approval number: does not apply.

Funding: this research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Declaration of interests: the authors have no conflicts of interest to disclose.

Correspondence:

Jessica Mariel Bazo-Medina

E-mail: mariel.mb@outlook.com