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PREVENIR ES NUESTRA META

- The triglyceride-glucose index
- WHO 2025 HIV guideline
- Mortality in complicated infective endocarditis
- Brugada type 2 pattern
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EDITORIAL

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The triglyceride-glucose index and its usefulness in clinical practice as a cardiovascular risk assessment tool

El índice de triglicéridos y glucosa y su utilidad en la práctica clínica como herramienta de evaluación del riesgo cardiovascular

Héctor Martínez-Ruiz,* Nayelli Nájera,‡ Guillermo Ceballos‡

Abbreviations:

T2DM = Type 2 Diabetes Mellitus
TyG = Triglyceride-Glucose

Cardiovascular diseases are the leading cause of morbidity and mortality worldwide, making the early identification of associated risk factors crucial.[1] Insulin resistance is recognized as a pathophysiological precursor to the development of Type 2 Diabetes Mellitus (T2DM), hypertension, and its complications: ischemic heart disease, stroke, and kidney failure.

In patients without a clinical diagnosis of T2DM, insulin resistance can be subclinical for years, progressively contributing to vascular damage. This process is associated with abnormal glucose and lipid metabolism that alters vascular wall structure and remodeling, endothelial function and nitric oxide synthesis (the main vasodilator), activation of the sympathetic nervous system, and renal sodium and water handling.

Several approaches exist for assessing insulin resistance. The euglycemic clamp is the gold standard for this assessment; however, its implementation is complex and impractical on a large scale. Indirect alternatives have also been proposed, such as the Homeostasis Assessment Model for Insulin Resistance (HOMA-IR) and the Quantitative Insulin Sensitivity Check Index (QUICKI);[2] however, these approaches remain limited by their high cost and limited availability in our context.

An alternative readily available to most healthcare systems is the Triglyceride-Glucose (TyG) index, considered an indirect marker of insulin resistance. Its availability in routine clinical practice, based on standard clinical determinations, facilitates its application.

The TyG index is calculated using the following formula:

$$TyG = Ln\,[TG\,(mg/dL) \times fasting\,glucose\,(mg/dL)]/2$$

Current evidence suggests that elevated TyG index values are associated with a high risk of major adverse cardiovascular events, supporting its potential use as a cardiovascular risk marker.[3,4] Also observational studies link the TyG index to the development of hypertension and cerebrovascular events in middle-aged and older adults, reflecting early cardiometabolic alterations that precede the development of cardiovascular complications.[4,5]

Recently, Lanfranco D'Elia et al.[6] reported that elevated TyG values (greater than 4.91) exhibit high predictive capacity for the development of hypertension.

The prognostic value of the TyG index, including its association with all-cause mortality, even after adjusting for traditional risk factors, reinforces its potential as a marker to identify

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individuals at greater long-term clinical vulnerability.[7]

The TyG index is not intended to replace more complex methods, but it can assist in assessing metabolic risk through a simple, reproducible, and low-cost formula. This is relevant because in primary care, most patients present without overt cardiovascular disease but with multiple risk factors. Identifying metabolic dysfunction using readily available parameters will enable earlier risk stratification, timely preventive interventions, and closer clinical follow-up. Its routine use is increasingly important for early detection of cardiovascular risk and enables comprehensive assessment in routine clinical practice.[6,7]

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LETTER TO THE EDITOR

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WHO 2025 HIV guideline: Integrating cardiometabolic care into HIV services

Guía de la OMS 2025 sobre el VIH: integración de la atención cardiometabólica en los servicios de VIH

Victor Abiola-Adepoju,* Abdulrakib Abdulrahim,‡ Safayet Jamil§

Keywords:
HIV care, diabetes, health policy, health equity, global health.

Palabras clave:
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The World Health Organization (WHO)'s 2025 human immunodeficiency virus (HIV) service delivery guideline recognizes that the epidemic is entering a new era marked by ageing, multimorbidity, and limited resources.[1] Although global Antiretroviral Therapy (ART) coverage now reaches 31.6 million people, only 73% of People living with HIV (PLHIV) achieve viral suppression.[2] Increasing life expectancy increases vulnerability to chronic comorbidities. In sub-Saharan Africa, 5-10% of PLHIV have diabetes, and 20-25% have hypertension. A meta-analysis of 10,916 participants across East Africa found hypertension prevalence at 19.75%, with alcohol use, diabetes, and longer HIV duration as key predictors.[3] Traditional HIV clinics, structured for infectious disease management, remain poorly equipped for lifelong chronic care. Without addressing hypertension and diabetes, cardiovascular risk will escalate, ultimately threatening viral suppression and the hard-won gains of the HIV response.

Evidence from sub-Saharan Africa demonstrates that integrating HIV and Non-Communicable Disease (NCD) care is both feasible and cost-effective. In South Africa, the Central Chronic Medicine Dispensing and Distribution program, launched in 2014, enables stable clients to collect ART, along with medicines for hypertension and diabetes, at community pick-up points.[4] Patients benefit from reduced travel time, shorter queues, and less stigma, while providers note decongested clinics and greater efficiency. In Uganda and Tanzania, the INTE-AFRICA pragmatic cluster-randomized trial followed more than 7,000 patients with HIV, diabetes, or hypertension.[5] Integrated management achieved retention rates above 89% among NCD patients and maintained HIV viral suppression above 95%, demonstrating non-inferiority to vertical care. The economic evaluation confirmed significant cost savings, as single-clinic management of multiple conditions reduced staffing demands and overheads. Patients also valued the convenience of fewer clinic visits. Together, these findings highlight integrated care as an efficient, patient-centered strategy for resource-limited settings.

To scale integrated care, investments are needed in equipment, supply chains, and human resources. Policies enabling task sharing with nurses and community health workers must expand beyond HIV services to include blood pressure and glucose management. Economic analyses suggest that integration can improve efficiency and equity for clients and health systems. Yet HIV funding is shrinking: 2024 resources for the AIDS response were US$18.7 billion, 17% below the US$21.9 billion needed.[2] As donors retreat and national budgets dwindle, integrated chronic disease services can maximize impact per dollar spent. Now is the time for ministries, donors, and implementers to align HIV programs with universal health

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coverage and non-communicable disease strategies, thereby protecting the hard-won gains of the HIV response.

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ORIGINAL RESEARCH

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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*,§

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 período 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).

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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

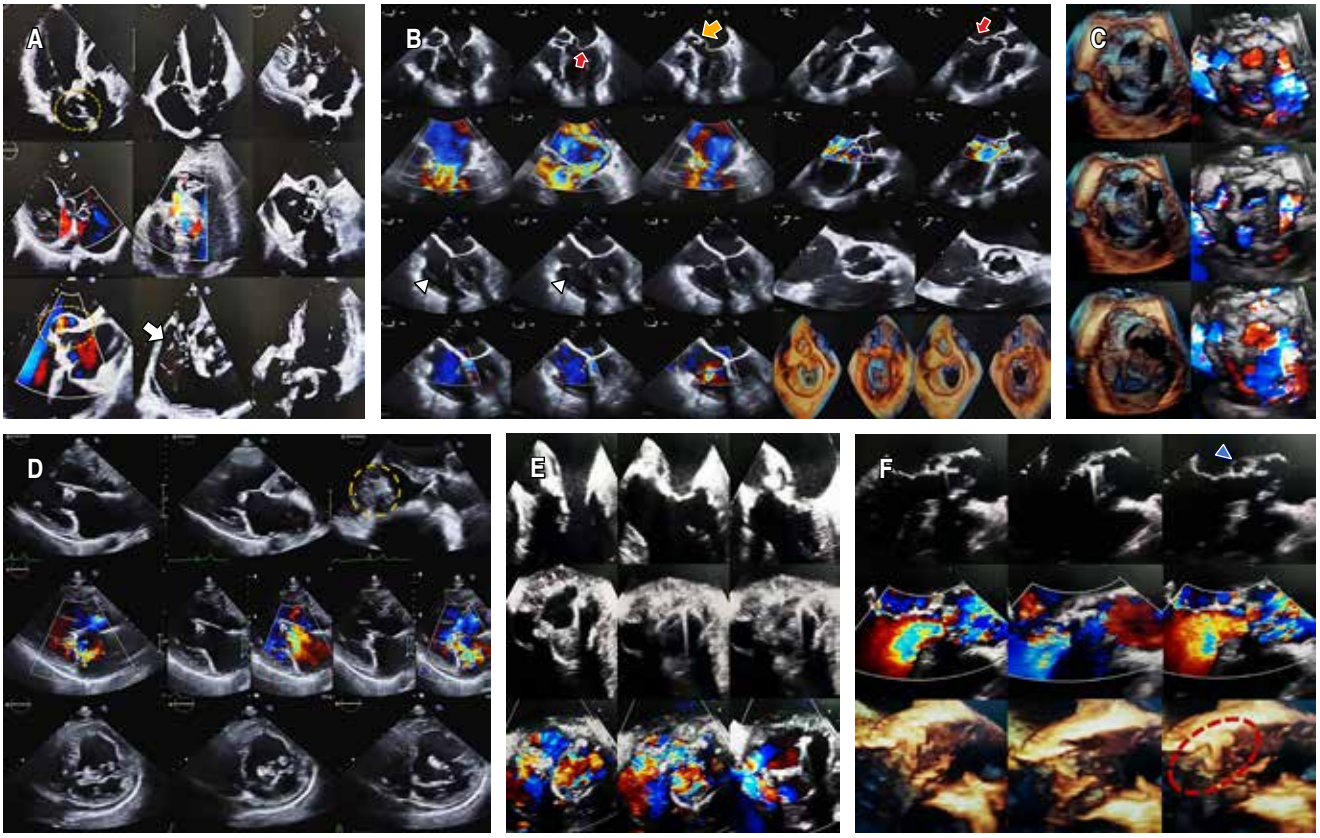

Figure 1: A) Apical and parasternal short-axis views of a case of bioprosthetic aortic valve endocarditis complicated by aortic root abscess (dotted circle), fistula draining into the right atrium, and native valve endocarditis of the tricuspid valve, which presents a large, non-mobile vegetation dependent on the septal leaflet (white arrow). **B)** TOE projections and 3D reconstruction. In the first row A3 at 0º, 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 0º, shows thin and elongated TV leaflets with anterior leaflet prolapse (white triangle). Right side A3 at 145º 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 supravalvular 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]

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.

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

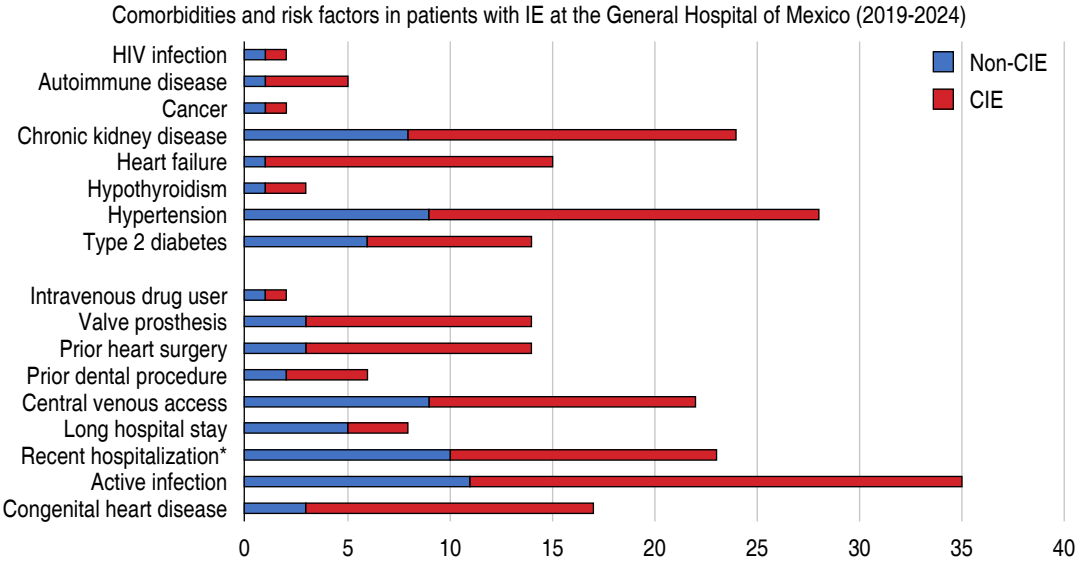

Figure 2: Graphical representation of the most prevalent comorbidities and predisposing conditions in patients with uncomplicated and complicated IE.
* Within the last 3 months. CIE = Complicated Infective Endocarditis. IE = Infective Endocarditis.

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

	Uncomplicated IE N = 3 (18.8%) n	Complicated IE N = 13 (31.8%) 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,

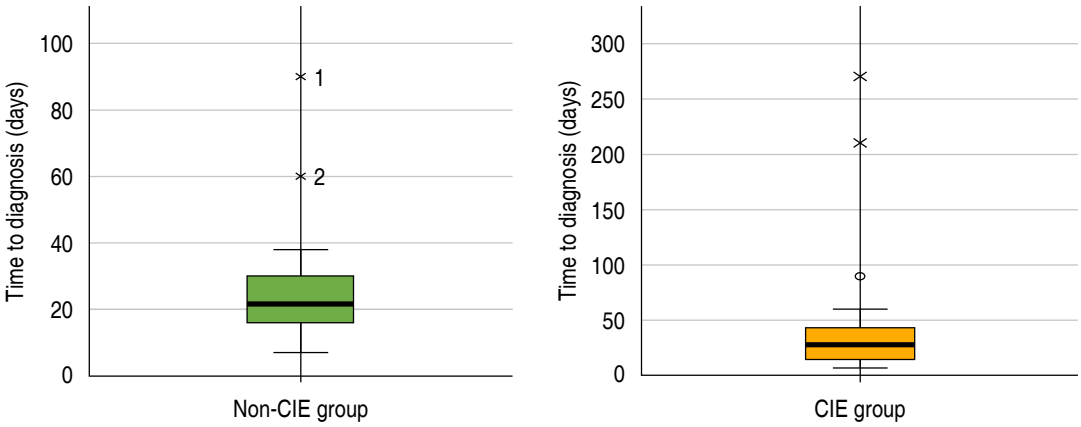

Figure 3: Comparison of time to diagnosis between uncomplicated and Complicated Infective Endocarditis: graphical representation.
CIE = Complicated Infective Endocarditis.

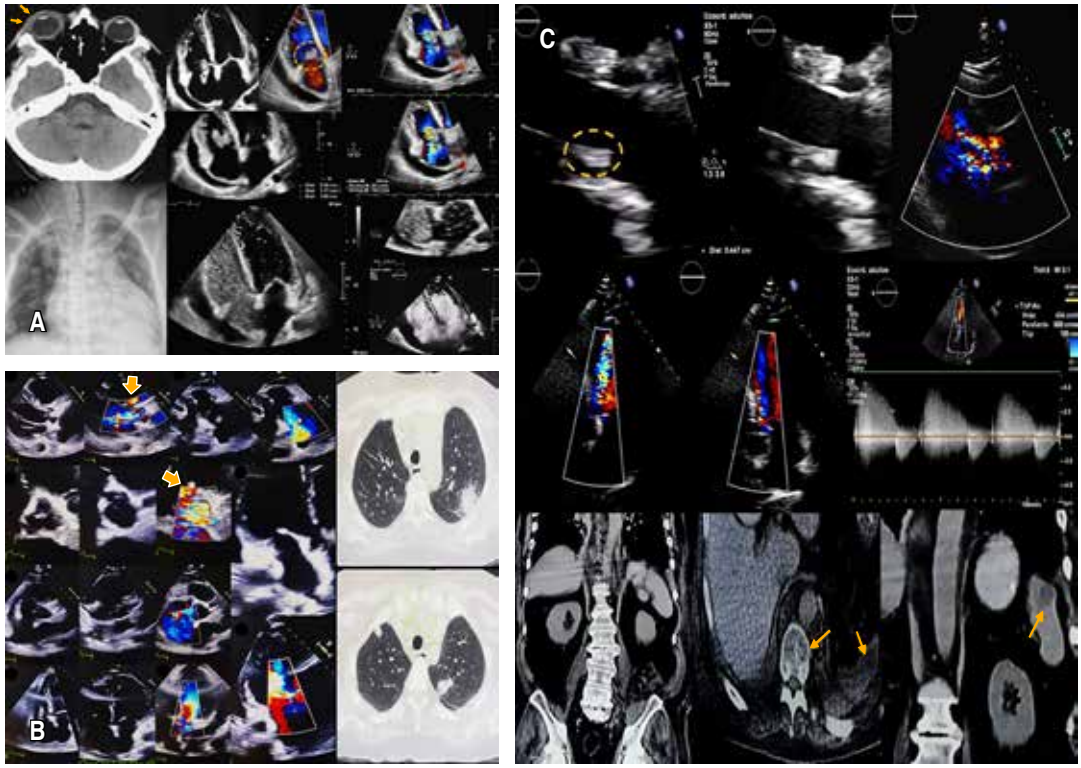

Figure 4: A) Case of native tricuspid valve Infective Endocarditis with a previously undiagnosed atrial septal defect. The infection spread to the central nervous system, including the ophthalmic artery and also embolizing into the pulmonary circulation the patient presented to the emergency department 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)*.

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

Isolated microorganism	Uncomplicated IE n	Complicated IE n
Staphylococcus aureus MR	2	3
Staphylococcus aureus MS	1	10
Staphylococcus epidermidis	2	3
Staphylococcus haemolyticus	0	1
Staphylococcus lugdunensis	1	0
Streptococcus anginosus	0	1
Streptococcus mitis	1	0
Enterococcus faecium MR	1	0
Enterococcus faecium MS	0	1
Enterococcus faecalis	0	2
Escherichia coli (ESBL)	1	1
Enterobacter cloacae	1	1
Haemophilus parainfluenzae	0	1
Klebsiella spp.	1	0
Salmonella spp.	1	0
Pseudomonas	1	1
Stenotrophomonas maltophilia	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.

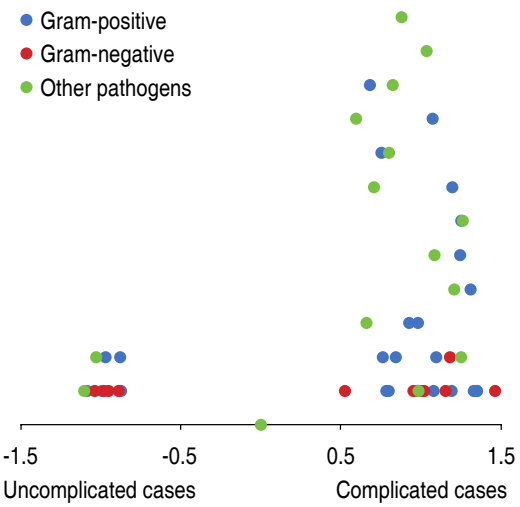

Figure 5:

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

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

Isolated microorganism	Native n	Prosthetic n
Staphylococcus aureus MR	5	0
Staphylococcus aureus MS	9	2
Staphylococcus epidermidis	1	4
Staphylococcus haemolyticus	1	0
Staphylococcus lugdunensis	1	0
Streptococcus anginosus	0	1
Streptococcus mitis	1	0
Enterococcus faecium MR	1	0
Enterococcus faecium MS	1	0
Enterococcus faecalis	2	0
Escherichia coli (ESBL)	1	1
Enterobacter cloacae	1	1
Haemophilus parainfluenzae	1	0
Klebsiella spp.	1	0
Salmonella spp.	1	0
Pseudomonas	2	0
Stenotrophomonas maltophilia	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
Staphylococcus aureus MR	2	3
Staphylococcus aureus MS	3	8
Staphylococcus epidermidis	1	4
Staphylococcus haemolyticus	0	1
Staphylococcus lugdunensis	0	1
Streptococcus anginosus	0	1
Streptococcus mitis	1	0
Enterococcus faecium MR	1	0
Enterococcus faecium MS	0	1
Enterococcus faecalis	2	0
Escherichia coli (ESBL)	2	0
Enterobacter cloacae	1	1
Haemophilus parainfluenzae	1	0
Klebsiella spp.	1	0
Salmonella spp.	0	1
Pseudomonas	0	2
Stenotrophomonas maltophilia	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

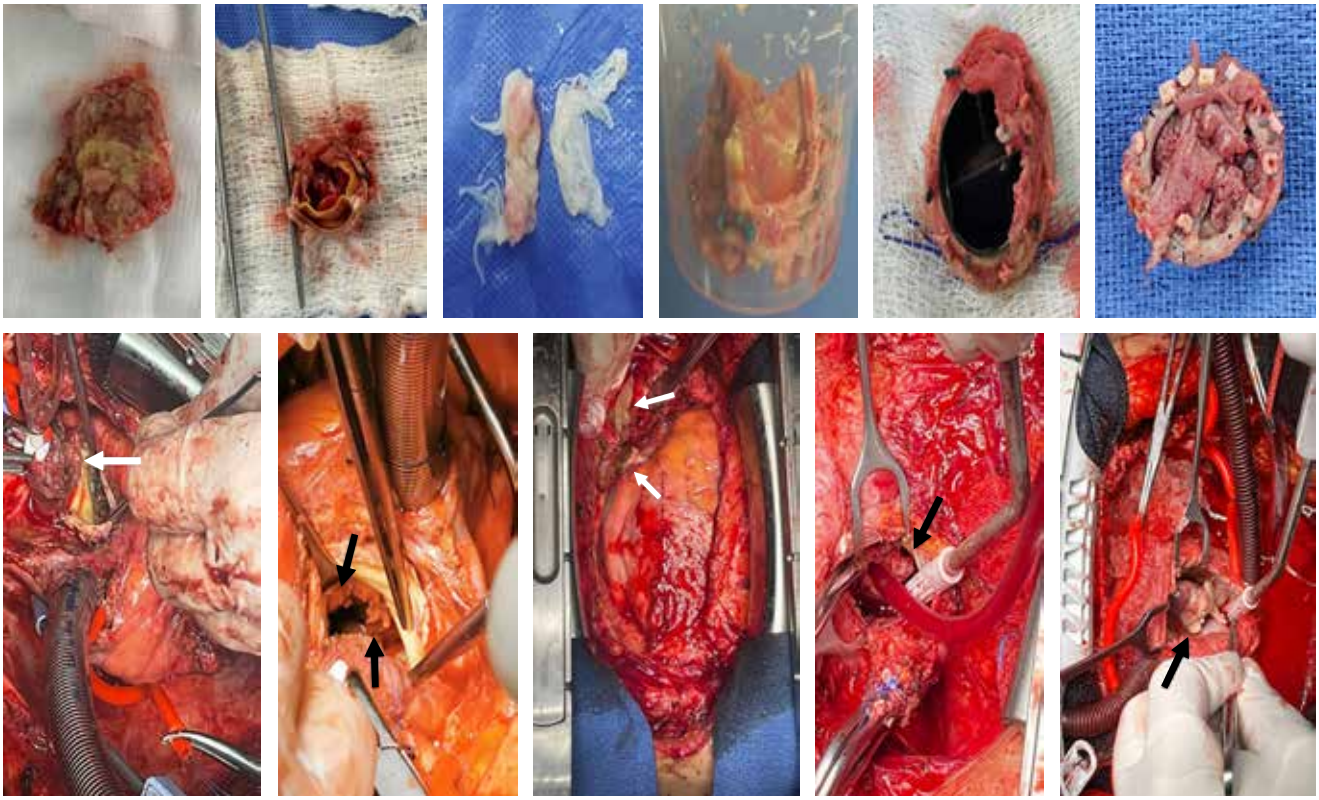

Figure 6: Top row, from left to right: infected thrombus; infected biological prosthetic valve with vegetations adhering to the leaflets; tricuspid valve showing a perforated anterior leaflet and a large vegetation on the posterior leaflet; bioprosthesis with purulent material and vegetations on the 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.

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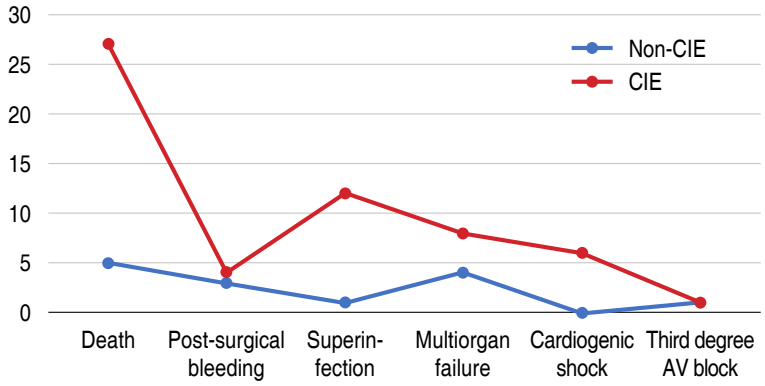

Figure 7: Incidence of postoperative complications in uncomplicated and locally Complicated Infective Endocarditis, graphical comparison.
CIE = Complicated Infective Endocarditis.

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Brugada syndrome with type 2 pattern. Case report

Síndrome de Brugada con patrón tipo 2. Reporte de un caso

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Keywords:
Brugada, electrocardiogram, Holter, dizziness, palpitations.

Palabras clave:
Brugada, electrocardiograma, Holter, mareos, palpitaciones.

ABSTRACT

Brugada syndrome is an inherited cardiac channelopathy that leads to malignant ventricular arrhythmias and sudden cardiac death, despite the absence of signs of structural heart disease. The syndrome's characteristic electrocardiographic pattern is rare and may be the only clinical manifestation, which complicates identification. This syndrome accounts for between 4 and 12% of cases of sudden cardiac death and up to 20% in patients with structurally normal hearts. It predominantly affects men, with a frequency of eight to 10 times compared to women, and an estimated prevalence of one to five cases per 10,000 individuals. The *SCN5A* gene is the most associated mutation, identified in 11 to 28% of cases. Syncope is the most relevant clinical sign, although it is not always present. Currently, diagnosis and risk stratification remain a clinical challenge due to the lack of consensus on reliable prognostic markers. Studies such as FINGER and PRELUDE have questioned the usefulness of inducibility in electrophysiological studies as a prognostic criterion, limiting its applicability in clinical practice. This report describes the case of a young asymptomatic male patient who has no relevant personal or family medical history and presents an electrocardiographic pattern consistent with Brugada syndrome. This finding poses a significant clinical challenge regarding risk assessment, highlighting the need for more precise criteria for the management of these patients.

RESUMEN

El síndrome de Brugada es una canalopatía cardiaca hereditaria sin signos de enfermedad estructural del corazón que conduce a arritmias ventriculares malignas y muerte súbita cardiaca. Su patrón electrocardiográfico característico, aunque raro, puede ser la única manifestación clínica, lo que dificulta la identificación. Este síndrome representa entre 4 y 12% de los casos de muerte cardiaca súbita y hasta 20% en pacientes con corazones estructuralmente normales. Afecta predominantemente a hombres, con una frecuencia de ocho a 10 veces en comparación con las mujeres, y una prevalencia estimada de uno a cinco casos por cada 10,000 individuos. El gen SCN5A es la mutación más asociada, identificada en 11 a 28% de los casos. El síncope es el signo clínico más relevante, aunque no siempre está presente. Actualmente, el diagnóstico y estratificación del riesgo siguen siendo un desafío clínico debido a la falta de consenso sobre marcadores pronósticos confiables. Estudios como FINGER y PRELUDE han cuestionado la utilidad de inducibilidad en los estudios electrofisiológicos como criterio pronóstico, limitando su aplicabilidad en la práctica clínica. Este informe describe el caso de un hombre joven y asintomático que no tiene antecedentes médicos personales o familiares relevantes y presenta un patrón electrocardiográfico consistente con el síndrome de Brugada. Este hallazgo plantea un desafío clínico significativo en cuanto a la evaluación del riesgo, subrayando la necesidad de criterios más precisos para el manejo de estos pacientes.

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Abbreviations:
BS = Brugada Syndrome
ECG = Electrocardiogram
SD = Sudden Death
TCAs = Tricyclic Antidepressants

INTRODUCTION

The Brugada-type electrocardiographic pattern is a rare manifestation that indicates a predisposition to ventricular arrhythmias. It is a cardiac channelopathy without structural heart disease. Its clinical

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relevance lies in its ability to cause sudden death in young patients with healthy hearts. It is known to be inherited in an autosomal dominant manner, with the *SCNA5* gene being the most common.[1]

Brugada Syndrome (BS) accounts for between 4 and 12% of sudden cardiac deaths and up to 20% in structurally healthy hearts.[2] It is eight to 10 times more common in men, with an estimated global prevalence of one to five cases per 10,000 people. Genetic variants are identified in 11 to 28% of cases, with the *SCNA5* gene being the most common.[3,4] Syncope is the most prominent feature in the clinical history. Family history is extremely important, particularly the presence of type 1 BS in relatives or a family history of sudden death in individuals ≤ 45 years of age.[5]

Studies such as FINGER have been reported, whose main contribution was to establish that medical history is the most powerful predictor. It was determined that the rate of arrhythmic events is significantly higher in patients with previous syncope or sudden cardiac arrest, while patients who do not report symptoms have a notably low risk (0.5% per year). In addition, it was the first to point out that variables such as gender, family history or inducibility in the electrophysiological study did not have solid positive predictive value. In addition, the PRELUDE study, which focused specifically on the usefulness of programmed electrical stimulation, recorded decisive results by demonstrating that the inducibility of arrhythmias in the laboratory does not reliably identify patients at risk of spontaneous arrhythmic events, clearly corroborating the low predictive value observed in the FINGER study. This study also proposed new and more promising risk markers, such as a short ventricular refractory period (< 200 ms) and QRS fragmentation potential on baseline electrocardiogram (ECG).[6,7]

As risk factors for BS, given that it is an autosomal dominant hereditary disease, the most common mutation is in the *SCN5A* gene, which encodes the α subunit of the cardiac sodium channel INa. Mutations reduce sodium flow, especially in the epicardium of the right ventricle, causing voltage gradients that manifest as ST-segment elevation. Recent studies have highlighted the importance of non-modifiable risk factors that are intrinsic to the patient, specifically the type of *SCN5A* mutation, regardless of clinical symptoms. This evidence underscores that the genetic substrate may be a predictor of malignant arrhythmic events. The most consistent non-modifiable risk factor is male sex. Arrhythmic events and Sudden Death (SD) also manifest predominantly in males, suggesting that male sex hormones (androgens) may have a modulatory effect on sodium channel function, exacerbating the phenotype. In the geographical context, although cases have been reported worldwide, the highest prevalence rates have been identified in Southeast Asian populations, where the disease is endemic and known by local names, e.g., Lai Tai in Thailand, which means Sudden Unexplained Nocturnal Death Syndrome (SUNDS).[8,9]

The diagnosis of BS is based on the identification of the distinctive electrocardiographic pattern and its correlation with clinical risk factors. The Brugada consensus established a precise classification of ECG patterns. Only type one has definitive diagnostic value. The patterns observed on the ECG are type 1 (dome or shark fin): concave or straight descending ST segment elevation of ≥ 2 mm (≥ 0.2 mV) in V1 and/or V2, followed by a negative T-wave. This pattern is the only diagnostic one. Type 2 (saddleback): J-point elevation ≥ 2 mm (greater than the PR segment), followed by a descending ST-segment that remains ≥ 1 mm above the isoelectric line and ends with a positive or biphasic T-wave. This pattern is suspicious. Type 3: type 1 or type 2 morphology, but with ST-segment elevation < 1 mm. Nonspecific pattern.

Type two pattern, known as saddleback morphology, is of clinical interest. It is characterized by a J point elevation ≥ 2 mm in V1 and/or V2, and an ST-segment that descends toward the isoelectric line but remains elevated ≤ 1 mm, creating the initial convex appearance followed by the characteristic saddleback concavity. This finding, although suggestive, has a low positive predictive value on its own, as it can be confused with normal repolarization variants, especially in athletes. Likewise,

as associated clinical criteria, it has been documented to have survived an episode of cardiac arrest secondary to ventricular fibrillation or polymorphic ventricular tachycardia, a history of unexplained syncope understood as a self-limiting arrhythmic event, nocturnal agonal respiration, which are episodes of abnormally noisy or labored breathing during sleep, and a family history of type 1 Brugada pattern in relatives or a family history of sudden death ≤ 45 years of age.[10,11]

This article presents the case of a symptomatic patient with no family or personal history who presents the characteristic electrocardiographic pattern of the syndrome. This finding poses a challenge in risk stratification and clinical decision-making.

CASE PRESENTATION

This is a 26-year-old man, originally from Chiapas, who currently resides in Villahermosa, Tabasco, Mexico. Occupation: undergraduate student. He sought medical services at the Juárez Autonomous University of Tabasco (UJAT) due to frequent spontaneous palpitations at rest, occurring three to five times per month, occasionally associated with mild dizziness but without syncope. In the physical examination, the vital signs were: blood pressure 111/67; heart rate 87 bpm; respiratory rate 18 bpm; temperature 36.6 ºC; oxygen saturation ($SatO_2$) 98%; weight 66.5 kg and height 1.69 m. Relevant personal and family history: denies any cardiovascular disease. No family history of sudden death or hereditary heart disease. Current illness: palpitations began approximately three months ago, with no apparent triggering factors. The episodes are short-lived and not related to physical exertion. The patient also reports generalized fatigue and occasional episodes of dizziness without loss of mental clarity. He denies chest pain, dyspnea, syncope, diaphoresis, fever in the last six months, or neurological symptoms. He has not received previous medical treatment. General condition: patient conscious, alert, oriented in all three neurological spheres. Cardiovascular examination: rhythmic heart sounds, no murmurs or rubs, peripheral pulses present and symmetrical.

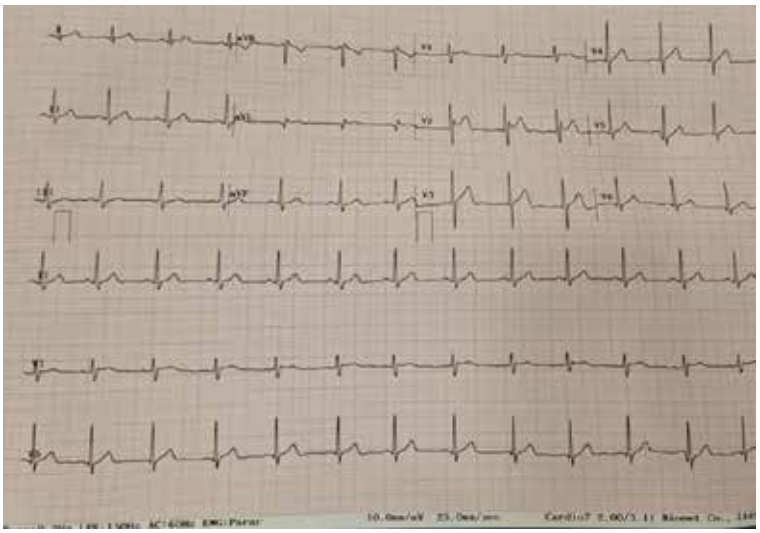

Figure 1: Standard 12-lead ECG calibrated at 25 mm/sec and 10 mm/mV; 3 channels + 3 rhythm channels. A standard 12-lead ECG is observed at rest in sinus rhythm, with a normal cardiac axis at 60º.

Studies

Baseline ECG: ST-segment elevation with saddleback morphology in leads V1-V2, with positive T-wave, suggestive of type 2 Brugada pattern.

The ECG documented ST-segment elevation in leads V1-V2 with a 1 mm notch and a positive T-wave consistent with the «saddleback» pattern characteristic of type 2 BS. The remainder of the ECG showed no evidence of atrioventricular block, bundle branch block, or atrial or ventricular hypertrophy *(Figure 1)*.

24-hour Holter monitoring

24-hour Holter monitoring, which recorded: a) a minimum heart rate of 46 bpm; b) a maximum heart rate of 137 bpm; c) a maximum RR interval of 1.422 s; and d) isolated premature atrial contraction.

The Holter review documented the following: basal sinus rhythm with an average of 74 bpm, a minimum of 46 bpm, and a maximum of 137 bpm; no atrial fibrillation was documented. Negative study for complex arrhythmias. No pauses longer than 2 seconds *(Figure 2)*.

Figure 2:

24-hour HOLTER, which recorded: **A)** the lowest HR of 46 beats per minute (bpm), **B)** the highest Heart Rate (HR) of 137 bpm, **C)** the longest RR interval of 1.422 sec, and **D)** the isolated premature atrial contraction.

Laboratory tests

Blood cytometry: leukocytes 7,400/µL, rest of white blood cell count normal. Red blood cell count: Hb: 15.4 g/dL, HCT: 46.1%, MCV: 99.8 fL, MCH: 33.3 pg, RBC: 4.9 × 10^6/mm^3, Platelets: 316 × 10^3/mm^3, MPV: 6.8% (low, range: 7.4-10.4). Serum electrolytes: sodium: 147.5, Potassium: 4.40, Serum chloride: 101.9 (unit: mmol/L). Lipid profile: cholesterol: 189.9, LDL: 99, HDL: 56.5, Triglycerides: 171 (high) (unit: mg/dL). Liver profile: total serum proteins: 7.23 g/dL, Globulin: 2.455 serum albumin: 4.77 g/dL, A/G ratio: 1.943, total bilirubin: 0.477 mg/dL, indirect B: 0.399 mg/dL, direct B: 0.078 mg/dL, SGOT: 34.7 U/L, SGPT: 51.7 U/L (elevated, 0-40.0), GGT: 21.4 U/L, ALP: 208.5 U/L, LDH: 308.54 U/L. Blood chemistry: glucose: 97.6, UREA: 22.1, BUN: 10.3, creatinine: 0.91, uric acid: 3.3 (low, 3.6-7.7) (unit: mg/dL). Urinalysis: performed as part of the initial approach. No relevant findings were found.

Diagnostic impression

Probable *de novo* type 2 Brugada syndrome (electrocardiographic pattern) under investigation. Frequent palpitations and dizziness without evidence of hemodynamic compromise or loss of consciousness.

DISCUSSION

A convex ST-segment elevation of more than 2 mm in the precordial leads (V1-V3) followed by a positive or biphasic T-wave that creates a «saddleback» appearance on the electrocardiogram, suggests a type 2 Brugada pattern, which has a lower prevalence than type 1. It has been reported that the effect of mutated sodium channels can lead to the expression of the pattern in conditions of increased risk, such as fever, electrolyte imbalances, vagal stimulation, psychosis, medications that act on sodium ion channels, and drug use. It is understood that BS can be dynamic, presenting with a type 2 pattern accompanied by significant symptoms and without any physiological condition, such as fever or potassium disturbances (hyperkalemia), that may be sufficient to manifest the pattern and progress to the more dangerous type 1 pattern. Therefore, it is important to closely monitor patients to avoid a more unfavorable prognosis. The role of electrophysiological studies remains controversial, as evidence in reports indicates that they have high sensitivity for identifying patients at higher risk of arrhythmic events, while their specificity remains low.[12]

Although this pattern is highly suggestive of BS, it is essential to establish a thorough differential diagnosis, as numerous entities, including primary pathological conditions and transient or drug-induced phenomena (Brugada phenocopies), can mimic this morphology.[13] Differentiation is crucial, given that the prognosis and therapeutic management vary dramatically. The Brugada pattern is classified as a channelopathy without obvious structural heart disease. However, other cardiomyopathies and channelopathies may present ST-segment elevation in V1-V2: Early Repolarization Syndrome (ERS): ERS, especially the variant affecting the right precordial leads, may manifest with J-point and ST-segment elevation, which can be confused with the type 2 pattern. The differential diagnosis is based on the specific morphology and the absence of other clinical criteria for Brugada syndrome. Wolff-Parkinson-White (WPW) syndrome: although it is a ventricular pre-excitation, the presence of delta waves can mask or influence the morphology of the ST-segment, creating false elevations in the right precordial leads. Sick sinus syndrome: although it is primarily a conduction disorder, its presence or the influence of related drugs can complicate the ECG and generate patterns like saddleback. And Arrhythmogenic Right Ventricular Dysplasia (ARVD): this condition is characterized by the replacement of myocardial tissue with adipose and fibrous tissue, predominantly in the right ventricle. It can cause repolarization abnormalities that resemble the Brugada pattern.[14] However, ARVD is distinguished by epsilon waves and/or right ventricular dilation/dysfunction.

Brugada phenocopies and acquired (reversible) causes. Brugada phenocopies are ECG patterns that meet the Brugada criteria (type 1, 2, or 3) but are secondary to reversible or induced conditions, disappearing once the underlying cause is resolved. It is estimated that these phenocopies constitute a significant percentage of initially diagnosed patients. Electrolyte disturbances: Elevated potassium levels can affect repolarization and depolarization, creating patterns that resemble ST elevation. Similarly, elevated calcium levels can also alter action potentials, contributing to ECG abnormalities.[12] Fever/Febrile states: increased body temperature can unmask a type 1 or type 2 pattern, especially in silent BS carriers. Fever amplifies sodium channel dysfunction.

In the context of a symptomatic patient with a type 2 pattern, active management of reversible factors such as fever and immediate discontinuation of inducing drugs (sodium blockers, tricyclic antidepressants (TCAs), cocaine) is strongly recommended before performing the pharmacological provocation test for a definitive diagnosis of BS. Reversibility of the ECG pattern after elimination of the acquired cause would confirm a phenocopy and not underlying genetic BS.[15,16]

CONCLUSIONS

This clinical case provides valuable lessons in both clinical and educational settings on how to approach Brugada syndrome.

From a clinical perspective, it highlights the importance of maintaining a high index of diagnostic suspicion in patients with seemingly nonspecific symptoms such as dizziness and palpitations, especially when a type 2 Brugada electrocardiographic pattern is identified. The absence of physiological factors that explain these findings reinforces the need for a thorough evaluation using 24-hour Holter monitoring, intentional search for malignant ventricular arrhythmias, and complete biochemical analysis to rule out secondary causes or precipitating factors.

In the educational context, this case exemplifies the complexity of differential diagnosis in cardiac channelopathies and highlights that type II electrocardiographic patterns, although less specific than type I, should not be underestimated. It provides an opportunity to reinforce knowledge about risk stratification in Brugada syndrome, emphasizing that the presence of associated symptoms significantly increases the risk of fatal arrhythmic events.

The systematization of the diagnostic approach presented serves as an educational model for the methodical analysis of similar cases, promoting clinical practice based on the integration of electrocardiographic findings, clinical manifestations, and complementary studies. Finally, this case reinforces the need for structured follow-up protocols and early consideration of preventive measures, including the possible indication of an implantable cardioverter defibrillator in symptomatic patients with a strong suspicion of Brugada syndrome.

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Traditional and/or clinical epidemiology. One pillar of modern medicine

Epidemiología tradicional o clínica. Un pilar de la medicina moderna

José Luis Moragrega-Adame*

Keywords: traditional epidemiology, medicine practice, scientific method.

Palabras clave: *epidemiología tradicional, práctica de la medicina, método científico.*

ABSTRACT

Traditional epidemiology has been neglected by some clinicians, as bothersome and of little use in daily medical practice. However, as two fundamental pillars of science applied to medicine are biostatistics and epidemiology, their study is essential for clinicians to make informed decisions when reading medical papers or attending lectures and be able to decide about the quality of the evidence for the best patient's evaluation and treatment. Many medical schools around the world, as is the case in Mexico, do not include the scientific method in their curriculum, and the mathematical preparation that students acquire in previous years is usually deficient. To stimulate doctors to learn the importance of epidemiology and statistics, they must be immersed in these fields well before entering medical school or, at the very least, during residency and specialty training.

RESUMEN

La epidemiología tradicional ha sido despreciada por algunos médicos, que la consideran molesta y de poca utilidad en la práctica médica diaria. Pero, ya que dos pilares fundamentales de la ciencia aplicada a la medicina son la bioestadística y la epidemiología, su estudio es fundamental al leer artículos médicos o atender a las conferencias y así poder evaluar adecuadamente la calidad de los datos para la toma de decisiones informadas, y lograr un mejor estudio y tratamiento de los enfermos. Muchas escuelas de medicina alrededor del mundo y es el caso de México, no incluyen el método científico en su currículum y la preparación en matemáticas adquirida en los años previos también es usualmente deficiente. Para estimular a los médicos a aprender la importancia de la epidemiología y la estadística, deben ser inmersos en estas disciplinas antes de entrar a la escuela de medicina o al menos, durante los años de residencia y especialidad.

The world as we have created it is a process of our thinking. It cannot be changed without changing our thinking.
Albert Einstein

It has been mentioned that there are 15 types of thinking[i] but for today's dissertation, some definitions are relevant to the scientific method.

1. Everyday thinking is the act driven by both the brain and the individual's personal needs to interact with the environment.
2. Scientific thinking is the act of seeking systematic and controllable explanations regarding a question. It refers to both thinking about the content of science and the set of reasoning processes that permeate the field of science.
3. Philosophical thought is the impulsive act that human beings possess in the search to differentiate themselves. This thinking seeks to provide explanations for events that science cannot. Anyhow, science is considered a derivation of philosophy, so that the differentiation can be complex.

[i] 15 Types of thinking and their characteristics (https://exploringyourmind.com/types-of-thinking/).

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The point of comparison among the three thoughts is the desire to meet a need: the everyday focuses on basic needs, the scientific on the factual, and the philosophical on the transcendental.

Although at its inception epidemiology was the earliest form of preventive medicine, it is currently an important component of the science of medicine. The history of epidemiology dates back to ancient Greece, where Hippocrates observed patterns of disease in human populations. Over the centuries, epidemiology has evolved, and in the nineteenth century, it was established as a formal scientific discipline.

In 1854, there was the most violent outbreak of cholera in London. John Snow, observing the distribution of cases and the proximity to different water sources, was able to determine which were contaminated and successfully applied preventive measures to the point of being considered the father of modern epidemiology.[1]

Among contemporary clinical epidemiologists with pioneering contributions[2] are Sir Austin Bradford Hill («Causality Rules»), Archie Cochrane («Evidence relevant to the patient»), and David Sackett («Clinical epidemiology»). Sir Michael Marmot was consecrated for his epidemiological studies of the relationship between social status and health. My teacher, the late Jeremiah Stamler, disseminated knowledge of epidemiology, statistics, and prevention among health professionals in many countries, including Mexico.

Biostatistics, the scientific method, and clinical epidemiology should be the basis of medicine, instead of only using personal experience or following the clinical guidelines which have become, in the words of E. Meaney, «The Guidelines Disease»[3] and have led to a «guidemanía». Instead of being published by accredited institutions, elaborated according to approved standards, and actualized frequently, in many cases, clinical guides lack scientific methodology. There is a need for their elaboration to follow the published «Criteria for trustworthy clinical practice guidelines».[4] Meanwhile, medical professionals have to be instructed in the critical analysis of scientific papers to decide if they are appropriate to be included in clinical settings.

During the «dark ages», there was scientific and medical progress in Asia, where, additionally, were exiled scientific personalities from Europe. One example was Maimonides/Ramban. (1135 Córdoba-1204 Egypt), who arrived in 1148 at the University of al-Qarawiyyin, Fez, Morocco. While he was physician to the vizier Alfadhil and Sultan Saladin, he applied the foundations of preventive medicine that would reach Europe until the Renaissance. Clinical Epidemiology can be defined as the application of epidemiological principles and methods to the problems encountered in clinical medicine, in order to provide better care, conduct research, and critically understand the medical literature.[5] Many ideas and many epidemiologists have contributed to the basis of this concept. John R. Paul, in 1938, used the term «Clinical Epidemiology» for the first time when he gave the lecture «A new philosophy for old diseases» at the American Society for Clinical Investigation in order to propose the use of Epidemiology in the clinical area. Dr. Peter P.H. De Bruyn, a physician and professor at the University of Chicago, taught a course to students of Public Health and Medicine that they called «Principles of Epidemiology» or commonly called «Clinical Epidemiology».[6]

When Dr. John R. Paul published the first known book on Clinical Epidemiology in 1958, he recognized that he owed the idea of it to Dr. Peter P.H. De Bruyn, a physician and professor at the University of Chicago, and that part of the text is based on an instructional course that a dozen years earlier, was offered to students of Public Health and Medicine as an elective course at Yale University.

Clinical epidemiology is ideologically linked to evidence-based medicine (EBM), whose origins some authors place in France in the mid-nineteenth century. In 1834, a movement called «*Médecine d'observation*» contributed to the eradication of useless treatments, such as bloodletting. For the first time, the observation, quantification, and careful measurement of phenomena related to health and disease were opposed to the deductive and intuitive method prevailing until then, to generate valid knowledge. «Observational medicine» has

become the first basis of all medical science; it gives the diagnosis, the prognosis, and the prediction of what will happen according to the knowledge of the evolutionary law of diseases. Nevertheless, pure observational medicine has never been able to suffice either. As a treatment is considered in clinical decisions, in the 1980's, the studies looked at the effects and determinants of those clinical decisions. The term 'Evidence-Based Medicine' (EBM) was first used in 1991, in an article by Gordon Guyatt published in the ACP Journal Club.[7] Accordingly to Alvan Feinstein, one of the pillars of modern epidemiology, John R. Paul's approach led to the development of the methods of analytical epidemiology and their extension to the field of non-communicable diseases. In 1992, the first working group on EBM was created in Canada.[8] In the words of Sackett et al., EBM is «... the integration of the best research evidence with the clinical expertise and values of the patient...».[9]

For many medical professionals, epidemiology has been considered «the ugly duckling» among the subjects taught in medical school. If we add the usual disgust for mathematics, we will see why statistics is another component of the lack of interest in the scientific method. To see the other side of a coin toss where one side is «the arid epidemiology and mathematics», the other one should be the description of science by Max Born –Nobel laureate in physics– «Science is one of the great adventures of the human race, as fantastic and demanding as the tales of heroes and gods, nations and states, writers and poets. That is my conviction, and I think that science can and should be taught in such a way as to transmit a suspicion of that spirit to the mind of the student».

Overall, critical appraisal plays a pivotal role in ensuring that healthcare professionals can make well-informed decisions based on high-quality, reliable, and relevant research evidence. By mastering critical appraisal skills, medical professionals can effectively navigate the extensive landscape of medical literature and apply the best available evidence to patient care.

Clinical guidelines cannot be used as if they were the absolute truth and should not be followed without taking into account the particular conditions of each case and information from other sources. There are currently so many guidelines developed by multiple medical groups in different parts of the world and of very different quality and applicability, that their revision requires the use of the recommendations for critical analysis of the medical literature.[10]

The evolution to evidence-based medicine, especially through the contribution of David Sacket, has allowed blending traditional epidemiology with clinical medicine.[11] Clinical epidemiology and Evidence-Based Medicine (EBM) can be our allies to take advantage of research studies and incorporate them into the practice of medicine, taking into account the importance of the doctor's experience and the individuality and preferences of each patient.[12]

It has been mentioned that evidence-based medicine has some weaknesses, but we will review some of them. One is the misuse of statistical values, which are improperly used instead of clinical significance, frequently trying to hide the lack of utility of some procedure or drug. Another problem is the definition of evidence, which can have many levels and is not always properly stated in the publications. The other is that EBM is not a substitute for the physician's experience but has to be seen as a complement.

Modern treatises on clinical epidemiology agree that there are two major applications: clinical practice and clinical research. According to Jeniseck,[13] in both, the user will devote themselves to:

1. Defining the concept of normality or abnormality.
2. Choose or evaluate diagnostic tests.
3. Control etiological factors.
4. Predict the evolution of the disease.
5. Carry out therapeutic tests.
6. Select the necessary information.
7. Evaluate the quality of medical care.

As an example of the usefulness of evidence-based medicine, in a study where two treatment strategies are compared, when we are told that a result is significant, we will understand that:

1. The data reflect with high probability that the differences found in the study represent the data of the universe where the sample comes from ($p < 0.05$).
2. That the size of the sample and its characteristics make it efficient and representative.
3. The results are important for the patient, and the recommendations can be incorporated into clinical practice.[14]

If any other method that you use to practice medicine has all these items, it is clinical epidemiology and evidence-based medicine by any other name!

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Cardiac arrest corner

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Challenges in Mexico in the management of out-of-hospital cardiac arrest

Retos en México en el manejo del paro cardiaco extrahospitalario

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ABSTRACT

Sudden cardiac death (SCD) and out-of-hospital cardiac arrest (OHCA) are global health problems that are responsible for almost 30% of cardiovascular deaths. The implementation of the chain of survival, including hands-only cardiopulmonary resuscitation (CPR) and early defibrillation, has improved survival and prognosis for OHCA patients. This work reviews the challenges and opportunities in Mexico regarding various strategies applied in recent decades to improve the survival rate of sudden cardiac arrest through the timely administration of CPR and early defibrillation. Interventions such as Public Access Defibrillation (PAD), volunteer responder programs, mobile dispatch of Automatic External Defibrillators (AED), drone delivery of AEDs, or new technologies for early detection of cardiac arrest are analyzed in the Mexican context.

RESUMEN

La muerte súbita cardiaca (MSC) y el parocardiaco extrahospitalario (PCEH) son un problema de salud mundial, responsables de casi el 30% de las muertes por causa cardiovascular. La implementación de la cadena de supervivencia, incluida la reanimación cardiopulmonar (RCP) sólo con las manos y la desfibrilación temprana, ha mejorado la supervivencia y el pronóstico de los pacientes con OHCA. El presente trabajo revisa los desafíos y oportunidades actuales en México respecto de diferentes estrategias que se han aplicado en diferentes países en las últimas décadas, para mejorar la supervivencia del paro cardíaco súbito mediante la administración oportuna de RCP y desfibrilación temprana. Se analizan en el contexto mexicano intervenciones como la desfibrilación de acceso público (PAD), los programas de respuesta voluntaria, el envío de desfibrilador externo automático (DEA) móvil, la entrega de DEA con drones o las nuevas tecnologías para la detección temprana de paro cardiaco.

Keywords:
sudden cardiac death, out of hospital cardiac arrest, hands only cardiopulmonary resuscitation, public access defibrillation, automatic external defibrillators.

Palabras clave:
muerte súbita cardiaca, paro cardiaco extrahospitalario, resucitación cardiopulmonar sólo con las manos, desfibrilación de acceso público, desfibrilador externo automático.

Abbreviations:
AED = Automatic External Defibrillators
ByDef = Bystander Defibrillation
CA = cardiac arrest
CPA = Cardio Protected Areas
CPR = Cardiopulmonary Resuscitation
CRUM = Centro Regulador de Urgencias Médicas
EMS = Emergency Medical System
OHCA = Out-Of-Hospital Cardiac Arrest
PAD = Public Access Defibrillation
RENAPACE = Registro Nacional de Paros Cardiacos Extra Hospitalarios
ROSC = Return to Spontaneous Circulation
SCD = Sudden Cardiac Death
T-CPR = Telephone-guided CPR
VF = Ventricular Fibrillation
VRP = Volunteer Responder Program

INTRODUCTION

Out-of-Hospital Cardiac Arrest (OHCA) and Sudden Cardiac Death (SCD) are world health problems, accounting for around 30% of total cardiovascular mortality and nearly 20% of all deaths in adults. The one-year survival rate for OHCA is low, around 8-10%.[1-3] In México, an estimated 33,000 SCD cases occur each year, although the lack of precise information

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may underestimate this figure. At least half of these events happen outside a hospital or medical facility.[4,5]

Early cardiopulmonary resuscitation (CPR) by laypersons (bystander CPR, B-CPR) and the use of an Automatic External Defibrillator (AED) before the arrival of the Emergency Medical System (EMS) may increase the survival rate.[6-9] The implementation of the survival chain in any SCD event is vital. This survival chain implies simple and useful maneuvers and concepts that require coordination in each action. To be successful, CPR during an OHCA involves the intervention of a community trained in hands-only CPR.

They can activate the first three steps of the survival chain by detecting the OHCA, notifying the EMS, starting hands-only CPR, and, if necessary, using the AED.[10,11] Hands-only CPR and early on-site use of AED may increase survival rates from 50 to 74%,[12-14] thus reducing the incidence of SCD; nonetheless, most OHCAs occur at the patient's home or working places, where the victim might be alone, and access to early defibrillation is low.[15-18]

The creation of Cardio Protected Areas (CPA) in Mexico requires early public access to defibrillation as a priority mechanism for treating SCD and involves training laypersons and other non-medical personnel, who are usually the first to intervene during a cardiac arrest (CA). They must be able to start the chain of survival, establishing basic and efficient hands only CPR until they or the EMS use an AED on the scene.[19,20]

In this paper, we analyze international recommendations and various approaches to achieve early defibrillation and improve OHCA survival. A perspective on the current state of the matter is offered, and the challenges in OHCA and SCD in Mexico, and what is currently being implemented, are depicted.

PUBLIC ACCESS DEFIBRILLATOR

Public access defibrillator (PAD) is probably the first and simplest strategy to prevent SCD and treat OHCA in many countries.[21-26] In Mexico, the first PAD program began in San Miguel de Allende, Guanajuato, in 2013.[27] Since then, several cities and states around the country have installed AEDs in public areas.[28-30] Latin American countries, such as Colombia, Panamá, Uruguay, and Chile, which are considered low- to medium-income, have also made scattered efforts to achieve successful PAD programs. However, perhaps Brazil pioneered a large-scale intervention program in Sao Paulo. They installed AEDs in the city's subway and trained the station personnel to administer CPR and use the AED. The initial return to spontaneous circulation (ROSC) did not increase significantly, but one-year survival reached 43% in treated patients.[31,32] In Spain, a high-income country, the concept of PAD involves areas with a high risk of OHCA. AED installation is mandatory in towns with more than 50,000 inhabitants and in public facilities, such as sports centers or complexes, with a daily capacity of 500 or more persons. It is also required by law for public establishments with a capacity of 5,000 or more people and subway stations with an average daily influx of 5,000 or more people. Other venues include large commercial centers, airports, bus and railway stations, and establishments with an average daily influx equal to or greater than 1,000 users, and centers or complexes with 500 users. In schools, work centers with more than 250 people, hotels with more than 100 beds, and residential centers for the elderly with more than 200 places, AEDs are also required.[33] This PAD strategy, along with a high rate of witnessed OHCA, By-CPR, and early defibrillation has increased survival rates up to 74 to 100% in patients with a witnessed arrest and an initial shockable rhythm such as ventricular tachycardia or fibrillation.[34,35] In such scenarios, the probability of bystander defibrillation (ByDef) can be increased by up to three times, and if the OHCA occurs near an accessible AED, survival rates can double.[36,37] The distribution of resources in Mexico and similar countries is hardly attainable due to geographic, economic, and social limitations. A targeted program like the one developed in Sao Paulo, based on population studies identifying high-risk areas and facilities, can optimize the distribution of AEDs and hands-only CPR training efforts.

Another issue that must be addressed is that most OHCA do not occur in high-person-flux areas but in places without a nearby AED or easy access.[38] In contexts such as residential areas, where OHCA and SCD occur more often, the probability of ByDef is very low, even when an AED is on-site.[39,40] A possible solution is the inclusion of other first responders, such as hands-only CPR-trained police officers with AED-equipped patrol cars, that might have an early arrival on-scene,[41] (discussed later), and telephone dispatch-guided CPR by family or on-site persons.

Although the Centro Regulador de Urgencias Médicas (CRUM) of Querétaro City has begun implementing that assistance, implementation has been inconsistent, and no measurable results have been achieved (non-published information).

It has been proven that PAD with on-site AED offers high survival rates, but also a very low coverage of patients since OHCA is more likely to occur in the victim's house. In countries like México, the financial cost of providing large numbers of AEDs will likely lead to excessive expenses and low efficacy. Only a few CA or OHCAs are covered by PAD programs with an AED on site, and private homes, where most events take place, are poorly covered by these devices. Efforts must be directed towards early OHCA recognition and EMS activation, community hands-only CPR training, and the use of AEDs. A smartphone application to identify the location of the nearest available AED can be helpful in residential areas to shorten response times.

A few Mexican states have approved laws to support and accelerate the establishment of cardio-protected areas, with limited success so far, despite the publication of a certification protocol two years ago.[42-44] The states of Sonora, Coahuila, Sinaloa, Jalisco, Morelos, and Yucatán have passed a Cardioprotection Law, and bill initiatives have been presented to Congress in Chiapas, Guerrero, Mexico City, Oaxaca, and Querétaro. There are other states and cities where isolated efforts have been made to implement cardio-protected spaces (for example, Aguascalientes, Durango, Hidalgo, Nayarit, Puebla, San Luis Potosí, Tamaulipas, and Tijuana).[5] Regarding the federal legislation on cardio-protected areas, on March 14th, 2013, the General Health Council issued an agreement published in the Official Gazette of the Federation, which urges owners and those responsible for establishments to have an AED in their facilities.[43-45]

On September 21st, 2021, after approval by the United Commissions on Health and Legislative Studies, the initiative was presented and approved in the Senate of the Republic as a reform to the General Health Law to prevent SCD. The draft decree was sent to the Chamber of Deputies for the purposes of Article 72 of the Constitution.[46] In this regard, another challenge is to optimize and clarify legislative work and resolutions (in the form of laws, codes, or regulations) at the municipal, state, and federal government levels. It needs a coordinated effort from society, medical societies, and non-government organizations to reach political representatives and establish proper communication channels. Furthermore, the same civil organizations and the public must have the legal certainty and protection to teach and implement nationwide hands-only CPR programs that include PAD. The Asociación Nacional de Cardiólogos de México (ANCAM) has just started a free Hands-only CPR training program for laypersons and the public interested in the topic. In this regard, scientific societies with an educational vocation might also join efforts to reach a broader population and raise awareness.

Figure 1 summarizes the key points of a PAD program.

THE REGISTRY

An official report in Mexico by the National Institute of Statistics and Geography (INEGI, its acronym in Spanish) indicates a mortality rate for cardiovascular disease, not linked to gender, close to 20% in our country. This report demonstrates that from the first week of 2020 to week 25 of 2021, the expected mortality from cardiovascular diseases was 232,658. Unfortunately, in Mexico, due to the lack of statistics and information, many SCDs are not reported in the death certificate as the leading cause and appear, instead, as if acute myocardial infarction was the direct cause

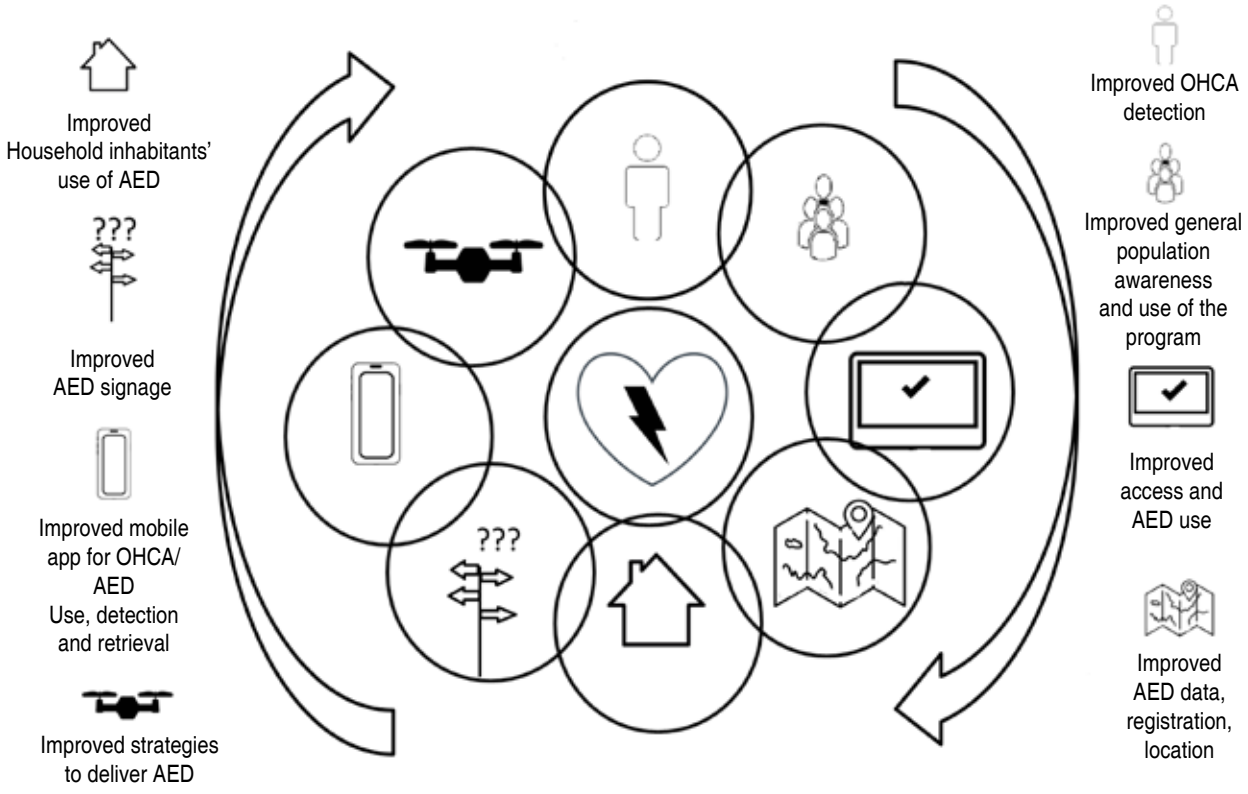

Figure 1: Public access defibrillation scheme.
Modified from: Folke F et al.[73]
AED = Automatic External Defibrillators. OHCA = Out-Of-Hospital Cardiac Arrest.

of death. These are two related but different clinical entities, especially in the case that concerns us (SCD), for treatment, statistics, research, and improvement of public health. In general, if statistics do not reveal SCD as the real cause of death, SCD will not be recognized as a public health problem. Thus, it will not have a tangible impact on public decision-making regarding intervention. The death certificate must be correctly completed so it can be used to evaluate health needs, as previously mentioned. They consist of research, planning, and monitoring of public health policies.[47,48]

A suggested protocol for certifying cardio-protected areas in Mexico has been published as described earlier.[42] An Out-of-Hospital Cardiac Arrest Registry (Registro Nacional de Paros Cardiacos Extra Hospitalarios, RENAPACE, its acronym in Spanish) should be the first step toward improving SCD mortality in Mexico.[42] RENAPACE should be a national public health policy involving complete pre-hospital EMS. Not only does it measure the outcome (life or death) of the OHCA event but also the aspects related to the initial CPR treatment, for instance, if B-CPR was performed, the number of pauses and quality of chest compressions, the initial heart rhythm, and successful cardiac shock by an AED, this information will tell what parts of the CPR and chain of survival could be improved. The program should have sufficient resources and the full support of the public and private health system administration, politicians, and medical and paramedical associations.[49] Resources, including administrative and medical personnel, will collect information from OHCA reports, ideally including dispatch center records, AED records database, hospital records, and the new entity, SCD, to be included in the death

certificates. Emory University did an example of this on the CARES Project.[18]

Other important variables to include in the RENAPACE would be total population, OHCA survival rate for all the rhythms, with special focus on patients treated for ventricular fibrillation (VF), discharged alive after an OHCA, neurological outcome evaluation, witnessed OHCA and B-CPR, Telephone-guided CPR (T-CPR), time of CA to call to dispatch center, time from CA to first compression for T-CPR, and time from collapse to EMS arrival (estimated or witnessed). Other variables can be included, among them, AED's location, time of EMS to start CPR and PAD, and first responder CPR with the use of AED before the EMS arrival, including the police department, first rhythm obtained, if it was a shockable rhythm, and time to first defibrillation. Trauma patients should be excluded from the registry.

IMPLEMENT A TELEPHONE-GUIDED CPR (T-CPR) CENTER WITH ONGOING TRAINING

This intervention relies entirely on the municipal government and should be coordinated with the 911 service. Several municipalities or states have recently tried implementing a CRUM to optimize emergency medical services deployment. In 2022, there were 156 CRUM nationwide; however, there are several disparities in the required degrees and experience to work in such centers. A telephone dispatch center with dispatcher-assisted CPR protocols should be implemented. It is difficult for telephone dispatchers to recognize a CA in the context of an OHCA by telephone, and it is even more challenging and demanding to provide CPR instructions. Ideally, this telephone call should be recorded and reviewed to gather feedback from individual dispatchers and the entire team, identifying how the service could be improved (e.g., how OHCA recognition could be completed faster and more effectively).[50,51] The dispatcher should have a professional and psychological profile (or training) to fill this position. The national medical associations could assess the dispatch center's training to improve the center's quality, evaluation, and service. Some initiatives and municipalities have tried to start this type of program in México; however, there is no proposal at the federal level that includes the entire country for its implementation. Special legislation to support this program is needed in the country.

VOLUNTEER RESPONDER PROGRAM (VRP)

Another strategy that has shown benefits in locations where geographic or financial reasons might limit PAD programs is the Volunteer Responder Program (VRP). Volunteer personnel from different organizations are contacted via a mobile app or smartphone from the dispatch center and directed to the victim, with information about the nearest available AED. This kind of intervention, initiated 15 years ago in Scandinavian countries, is trying to reduce gaps with PAD.[52,53]

The VRP tried to increase CPR and Bystander defibrillation administration before the EMS arrived at the scene. Several studies have found that VRP is associated with increased B-CPR and early defibrillation rates.[54,55] VRP significantly reduced the time to first defibrillation, increased B-CPR, and improved survival for patients with a shockable rhythm.[56] Activation of the VRP could have a greater impact on patient survival in areas with long EMS response times (e.g., rural areas or crowded cities such as Mexico City, with an average response time of 30 to 50 minutes, according to several public sources).

Local volunteers can get to the victim more quickly with an AED. Nonetheless, the positive results will rely on addressing different aspects: the EMS dispatcher must recognize the emergency call as a potential OHCA, activate the professional EMS system, and activate the VRP. The VRP needs to have enough volunteer responders and precisely know the location of the nearest available AEDs in all areas where OHCAs occur.[57]

Implementing a VRP requires an AED location registry accessible to the emergency dispatch center and specialized software or communication tools to activate volunteer responders in a timely manner. This program can potentially increase B-CPR and defibrillation,

but further investigation is needed, especially to determine survival rates.[58]

No VRP program has been implemented in Mexico. If it was to be done, it should probably be done after PAD had succeeded. Several barriers are perceived in this matter: legislation must be implemented, as with the PAD programs, to establish VPR reach and limits, protect volunteer personnel, determine the certifications they need, and so on.

Social issues must be faced: safety is a primary concern in many regions in Mexico. Some paramedics have tactical formation, but volunteers and laypersons do not: they should be able to administer first aid without concern for their own safety, and legal and social measures must be in place.

A variant of VRP could include other emergency service providers in the system, such as firefighters or police officers. A study by Aguilera et al. used the Hauswald methodology[59] to estimate the costs of shifting the first response to an OHCA from EMS to police units.[41] According to the city study, the yearly cost of saving a life with an ambulance might be from 5.8 to 60 million pesos. If the term «ambulance» is changed to «police vehicle», the estimated costs go to 0.5 to 5.5 million pesos for a saved life yearly.

Training police officers as OHCA first responders is already a worldwide recommendation,[60-65] positively impacting early B-CPR administration, survival, and cost reduction. Training the police might be a challenge in Mexico for several social and practical reasons: the EMS system is fragmented, and there is little continuity between the actions of first responders, ambulance personnel, and hospital staff. The implementation of the CRUM is slowly improving the coordination between EMS components and other emergency services, but there is still a long way to go to avoid the influence of government changes and other political conditions.[66]

There is little information on OHCA survival in Mexico, but a rough estimate is that it is nearly 0%.[41] These numbers are due to prolonged emergency services response times, especially when activating the EMS (time from collapse to EMS call), the reduced number of medical interventions, even CPR, and a general lack of knowledge about SCD and CPR. A continuous improvement program for pre-hospital emergency services and implementation of permanent educational programs for the public regarding hands-only CPR, OHCA recognition, and EMS activation are essential interventions.

In this regard, the need for a national cardiac arrest and sudden cardiac death registry in Mexico has already been addressed, along with the use of recorded AED data. In conjunction, this information might allow us to establish proven strategies to address OHCA or to design novel ones adapted to local needs.

Volunteer Responder Programs have great potential to improve B-CPR initiation and AED use, but they need technological tools to make them more efficient and to measure their results objectively.

DISPATCH OF MOBILE AND DRONE DELIVERY AEDS

The goal of reducing OHCA mortality and, thus, the burden of SCD requires, among other measures, substantially reducing response times. Professional first responders (PFRs), such as police officers, firefighters, or off-duty healthcare personnel, represent a valuable means to increase the likelihood of CPR and early defibrillation.

PFR and VRP can provide AED coverage in areas where an AED is not on site, and they can also arrive at an OHCA scene before the EMS, with an average survival rate to hospital discharge of 28.6% (range 9 to 76%).[67] *Figure 1* addresses this issue. Some European countries have implemented PFR programs and have found higher rates of return of spontaneous circulation (ROSC) and survival to hospital discharge,[62] but the effect on 30-day survival has not been demonstrated.[54]

A trial that recruited taxi drivers found very low acceptance of messages asking to act as first responders: 0.4% of the alerted ones arrived on the scene before EMS.[68]

In Mexico, this program is unavailable, and only local municipal efforts (Querétaro) have explored recruiting PFRs, with promising initial results. However, the general implementation has seen little success.[69] This probably relies on

the legal voids beginning to be filled with the above-mentioned legislation.

Figure 2 shows the success and survival rates for different CPR and AED strategies.

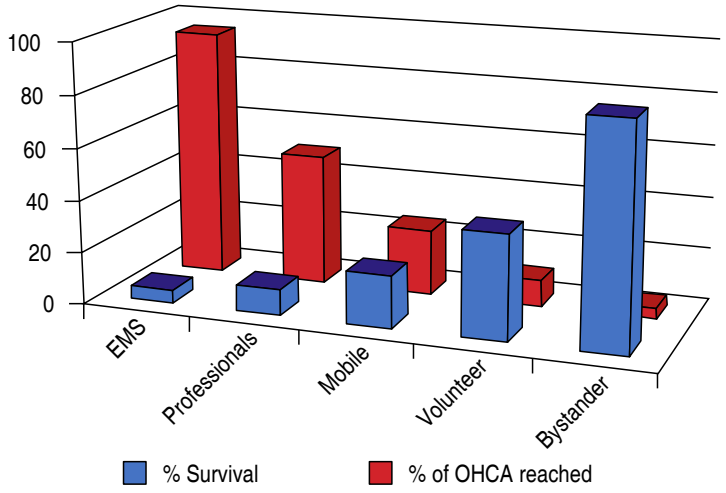

Figure 2: Accessibility and successful rates for different strategies of cardiopulmonary resuscitation and the use of automatic external defibrillators. EMS = Emergency Medical System. OHCA = Out-Of-Hospital Cardiac Arrest.

Table 1 summarizes the advantages and disadvantages of the different CPR and AED use strategies.

Implement a mandatory program to teach CPR and AED use from elementary school to the general population.

Mandatory CPR/AED training is part of the school curriculum in Norway and Denmark. Some states in the USA have legislation requiring compulsory high school CPR/AED training, but a community program, including public employees, is needed to teach CPR to the general population.[70] To obtain a driving license, a CRP course should be required, and it could be directed and supervised by Mexican Medical Organizations like ANCAM, following international medical standard protocols and guidelines. The current practices are based on international guidelines issued by ILCOR and its associates (the American Heart Association and the American College of Cardiology), although they are partial and incomplete. However, the local implementation of these programs does not require the endorsement of a foreign

Table 1: Summary of the different strategies of cardiopulmonary resuscitation and use of automatic external defibrillators.

Strategy	Advantages	Disadvantages
• Public access defibrillation • AEDs on site	• Rapid defibrillation • High survival rates	• Small impact on survival in private homes • Difficult predict high-risk areas to place AED • Need a big number of AED
• Volunteer responders AEDs on site	• Better defibrillation rates when arriving before EMS • Better defibrillation rates in private homes • More use of on-site AEDs because been transported to the OHCA	• Big number of volunteer responders and AEDs • Need an AED register and dispatched center
• Professional first-responders (e.g. police officers) firefighters	• Stations widely spread throughout the community • Trained in CPR and AED • Able to enter private homes	• Can be occupied by other jobs and may leave the scene if dispatched to another incident • Only effective if arrival before EMS
• Drone-delivered AEDs	• AED drone can cover a large area • Volunteers can move directly to the OHCA location while the drone delivers the AED • Fast AED delivery before EMS arrival • Very useful in in rural/and difficult access areas	• Legislation problems • Needs a trained pilot • High cost • Limit used in in with strong winds or • Heavy rain

AED = Automatic External Defibrillators. CPR = Cardiopulmonary Resuscitation. EMS = Emergency Medical System. OHCA = Out-Of-Hospital Cardiac Arrest. Modified from: Folke F et al.[73]

society. National medical associations have the authority to advocate for these programs and legislation to enable state and federal authorities to establish the necessary legal frameworks. The more people know how to perform CPR and feel protected by the law, the higher the reduction in SCD/OHCA mortality rate could be expected.[71] As mentioned earlier, in Mexico, the ANCAM is starting a free educational program to teach hands-only CPR to the general population.[72]

INTERDISCIPLINARY WORK

SCD involves a complex interplay of factors; therefore, an urgent call for a clinical multidisciplinary approach, including the three levels of government, private initiative, medical associations (at all levels of education), and EMS (including police), are essential to address this national health problem. The development of specialized sudden cardiac arrest care teams is required to ensure care, teaching, and research on SCD. There is a lot to do in Mexico, where international recommendations cannot practically be applied because the required public health programs to prevent sudden death and OHCA are not yet in place or are only beginning to be implemented in specific areas where isolated efforts have been made.

Above all, legislation that supports the eight points mentioned is needed. The implementation might be complex, and joint efforts are required from government agencies, NGOs, various authorities, and medical associations specializing in the area overseeing their regulation and implementation. It requires hard work that will take time and strong determination to complete, as well as direct financial support. Without the law's mandatory nature, these efforts might prove futile. An ongoing implementation can use the examples from other countries and adapt these points to the possibilities of our country and population. Otherwise, efforts will remain isolated and not be implemented.

CONCLUSIONS

Many efforts worldwide have been made to establish cardio-protected areas through various strategies that employ different tools and imply different approaches tailored to specific cultures, societies, geographies, politics, medicine, and other considerations. However, every approach relies on early CPR and AED use, known measures to improve OHCA survival.

Every intervention depicted in the present paper requires a precise sequence of actions based on the «Chain of survival» and almost perfect timing to achieve recovery from sudden cardiac arrest. If a PAD program is not in place, VRP and mobile AED systems will have limited success. Other interventions (drone delivery of AEDs or OHCA detection using electronic devices) will also need to have a solid PAD program to increase the chances of success in reversing cardiac arrest.

Thus, PAD in Mexico, with emphasis on hands-only CPR and early defibrillation combined with other CPR strategies, as mentioned before, needs significant efforts from the government and civil institutions to set and implement the proper legislation, define and implement educational and training activities, initiate, protect and supervise the corresponding actions, and follow up the results to establish continuous improvement programs so the medical and social burden of sudden cardiac arrest and death can be reduced to a minimal.

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Consensus Statement

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The 2026 Mexican Consensus on Arterial Hypertension: Expert Group on Arterial Hypertension (GREHTA)

Consenso Mexicano sobre Hipertensión Arterial 2026: Grupo de Expertos en Hipertensión Arterial (GREHTA)

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Abbreviations:

ACS = acute coronary syndrome
AF = atrial fibrillation
BP =blood pressure
CKD = chronic kidney disease
COPD = chronic obstructive pulmonary disease
CPAP = continuous positive airway pressure
eGFR = estimated glomerular filtration rate
HF = heart failure
MASLD = metabolic dysfunction–associated steatotic liver disease
MI = myocardial infarction
OSAS = obstructive sleep apnea syndrome
RAAS = renin-angiotensin-aldosterone system
SGLT2 = sodium-glucose cotransporter-2

EXECUTIVE SUMMARY

OPERATIONAL SUMMARY FOR PRIMARY CARE PHYSICIANS[1-7]

I. CORE PRINCIPLES (THE «GOLDEN RULE» OF THE CONSENSUS)

1. Arterial hypertension is NOT an isolated disease

It is one of the central components of a complex cardio–renal–metabolic phenotype (MACARENHA, see below).

All elements of MACARENHA must be addressed and treated.

2. Accurate blood pressure (BP) measurement is the most important clinical act

«If I measure incorrectly → I diagnose incorrectly → I treat incorrectly → I fail».

Without proper measurement: office, home, ambulatory blood pressure monitoring (ABPM), or home blood pressure monitoring (HBPM), there is no patient benefit.

3. If I do not assess cardio–reno–metabolic risk, I cannot construct an appropriate treatment plan

Comprehensive management depends on global cardiovascular risk.

4. If I initiate therapy with monotherapy, the probability of failure is very high (≈ 70%)

Most Mexican patients require dual or triple therapy from the outset, ideally in a single-pill combination.

5. My objective is to bring the patient to < 130/80 mmHg within three months

These BP targets should be maintained lifelong.

6. Beyond blood pressure, I must control glucose, lipids, and body weight

In Mexico, hypertension is almost always cardio–reno–metabolic and atherosclerotic simultaneously.

II. GENERAL GUIDELINES FOR THE MANAGEMENT OF ARTERIAL HYPERTENSION

1. Diagnosis must be based on accurate blood pressure measurement

1.1 In-office measurement

- Appropriate cuff size.
- Patient seated for five minutes.
- Two measurements separated by two minutes.
- Repeat if a discrepancy exists.
- Measure in both arms at the first visit and use the arm with the higher reading.
- A discrepancy of > 10 mmHg between arms warrants further evaluation.

1.2 Out-of-office measurement

Mandatory in adult patients with office BP values of 120-159/85-109 mmHg (for patients with > 160/110 mmHg, office measurement alone is sufficient).

1.2.1 HBPM

- The most useful instrument in daily practice for diagnosis and follow-up.
- Three measurements each morning and evening for seven consecutive days using the correct technique.

- Allows assessment of BP variability and stability.

1.2.2 24-hour ABPM

Indicated in:

- Suspected nocturnal hypertension.
- Marked BP variability.
- Resistant hypertension.
- Obstructive sleep apnea syndrome (OSAS).
- Non-dipping hypertension.

BP thresholds for clinical action

Condition	Immediate action
BP ≥ 140/90 mmHg	Diagnosis and initiation of treatment
BP ≥ 130/80 mmHg + high or very high risk	Diagnosis and initiation of treatment
Suspected white-coat or masked hypertension	Confirm with HBPM/ABPM

III. CARDIO–RENO–METABOLIC RISK STRATIFICATION (MACARENHA CONNECTION AS THE NEW INTEGRATIVE AXIS): HYPERTENSION IS ONE COMPONENT

1. Key components of the MACARENHA phenotype

- **Metabolic:** type 2 diabetes (T2D), dyslipidemia.
- **Adipose:** pathological visceral adiposity.
- **Cardiovascular:** ischemic heart disease, atrial fibrillation (AF), venous insufficiency.
- **Arterial:** arterial stiffness, microvascular damage, endothelial dysfunction.
- **Renal/respiratory:** chronic kidney disease (CKD), albuminuria/chronic obstructive pulmonary disease (COPD), OSAS, infections.
- **Enterohepatic:** microbiota alterations, metabolic dysfunction–associated steatotic liver disease (MASLD).
- **Neurological:** stroke, cognitive decline, mental health disorders.
- **Arterial hypertension.**

2. Rapid evaluation using indices derived from routine laboratory tests with simple calculations

- Estimated glomerular filtration rate (eGFR) using serum creatinine for estimating renal damage.
- Albuminuria/proteinuria (urinalysis dipstick).
- Urinary albumin-to-creatinine ratio (morning spot sample).
- Non-high-density lipoprotein cholesterol (Non-HDL-c cholesterol, primary marker of dyslipidemia).
- Fasting glucose.
- Triglyceride–glucose index (marker of glucose intolerance).
- FIB-4 index calculated from complete blood count and chemistry panel.

Practical office rule:

If your patient has hypertension + T2D and/or dyslipidemia and/or obesity and/or CKD → the patient is at high cardio–reno–metabolic risk.

2.1 Rapid risk stratification version

- Always count the number of risk factors (≥ 3 = high risk).
- Presence of target organ damage (retina, heart, kidney, brain, arteries).
- Previous cardiovascular event (stroke, myocardial infarction (MI), or other acute coronary syndrome (ACS), atrial fibrillation (AF), and heart failure (HF) → very high risk.
- eGFR < 60 mL/min.
- BP > 160/100 mmHg = high risk; BP > 180/110 mmHg = very high risk.

If the patient meets any of these criteria, they are automatically classified as high or very high cardio-reno-metabolic risk and do not require a digital risk calculator. Approximately 70-80% of primary care patients are already at high or very high risk at their first consultation.

IV. NON-PHARMACOLOGICAL TREATMENT

Applies to all patients and consists fundamentally of a healthy lifestyle: reduction of salt intake,

an appropriate dietary pattern (milpa diet), regular physical activity, and management of excess body weight.

V. PHARMACOLOGICAL TREATMENT: A SINGLE AND SIMPLE ALGORITHM

Key message: monotherapy is reserved only for special cases and should therefore be prescribed by a specialist. Its effectiveness in achieving BP targets is approximately 30%.

Step 1. Initiation (the vast majority)

The fundamental rule is the use of dual combination therapy in a single tablet at treatment initiation.

(Patients with systolic/diastolic blood pressure (SBP/DBP) > 160/100 mmHg at high or very high risk may require triple therapy from the outset)

- ACE inhibitor (ACEi) or angiotensin 2 receptor blocker (ARB) + calcium channel blocker (CCB)
  or
- ACEi or ARB + thiazide/thiazide-like diuretic (chlorthalidone/indapamide)

Always prefer fixed-dose single-pill combinations.

Note: Beta-blockers may be initiated at Step 1 under special conditions (sustained heart rate > 80 bpm; AF to maintain HR < 110 bpm; HF to maintain HR < 70 bpm; chronic coronary syndrome).

Step 2. Intensification

If the target BP is not achieved within one month, treatment must be intensified (~ 30% of patients on dual fixed combinations will require adjustment).

Add a third agent:

ACEi/ARB + CCB + thiazide-like/thiazide diuretic.

Step 3. Resistant hypertension

(Defined as uncontrolled BP despite three or more medications, including a full-dose diuretic.)

Remember! First, verify pseudo-resistance: improper measurement, non-adherence, BP-elevating drugs, OSAS, secondary hypertension.

Add:

- Spironolactone (first choice)
- Other options: beta-blockers, clonidine, alpha-blockers, loop diuretics, and innovative therapies depending on patient context.

Consider referral for renal denervation in selected cases to specialized centers with clear criteria.

Mandatory concomitant treatments

Because BP control alone does not guarantee the total achievable benefit:

- Statin therapy in all patients, regardless of calculated risk, individualized dosing ± ezetimibe.
- Sodium-glucose cotransporter-2 (SGLT2) inhibitors or glucagon-like peptide-1 (GLP-1) receptor agonists in T2D or high cardiovascular risk.
- Sustained weight reduction.
- Sodium restriction + cardio–renal diet.
- Mandatory vaccination (Influenza, Pneumococcus, SARS-CoV-2 virus (Coronary disease 2019, COVID-19), herpes zoster, respiratory syncytial virus).

VI. SECONDARY HYPERTENSION: RAPID AND PRACTICAL DETECTION

Red flags:

- Onset in children, adolescents, adults < 30 or > 65 years.
- Resistant hypertension.

- Hypokalemia.
- Significant albuminuria.
- OSAS.
- Abrupt BP worsening.

Main causes and basic management:

- OSAS → Continuous positive airway pressure (CPAP) therapy for sleep apnea + combination therapy.
- Kidney disease → Renin-angiotensin-aldosterone system (RAAS) blockade + CCB + thiazide-like + SGLT2 inhibitor.
- Hyperaldosteronism → spironolactone/surgery.
- BP-raising drugs → discontinue.
- Renovascular disease → multifactorial management/selective intervention.
(Other less frequent causes are discussed in the extended document).

VII. SPECIAL POPULATIONS

Hypertension and pregnancy

- Definition: BP ≥ 140/90 mmHg; may predate pregnancy and persist.
- Preeclampsia: hypertension after week 20 → multiorgan dysfunction, proteinuria, platelet dysfunction, thrombotic diathesis → Hemolysis-elevated liver enzymes-low platelet count (HELLP).
- Low-dose of acetylsalicylic acid from week 11 if no contraindication.
- Initial BP control: methyldopa, labetalol, nifedipine, hydralazine, or combinations.
- Magnesium sulfate in severe cases; mandatory hospital management.
- High future CV risk → mandatory postpartum follow-up.

All these conditions confer lifelong elevated risk in women.

Women and other disorders

- Menopause
- Polycystic ovary syndrome
- Contraceptive and hormonal therapy use
- Autoimmune diseases

Children and adolescents

- Hypertension defined by percentiles; ≥130/80 mmHg in those >13 years
- Treatment: lifestyle + ACEi/ARB + CCB
- Consider obesity
- Evaluate for secondary causes

Older adults

- Consider arterial stiffness (pulse pressure > 50 mmHg).
- Assess frailty and cognitive decline.
- Dual or triple therapy may be used if no contraindications.
- BP flexible target, preferably close to 130/80 mmHg.
- Avoid orthostatic hypotension.
- Consider monotherapy in frailty.

VIII. ARTIFICIAL INTELLIGENCE IN HYPERTENSION

- Cuffless BP measurement.
- Support for cardio–reno–metabolic risk stratification.
- Greater precision in ABPM/HBPM interpretation and remote monitoring.
- Identification and control of the MACARENHA connection.
- Detection of clinical inertia.
- Improved adherence.
- Opportunity for precision medicine.
- Research and big data management.
- Automation of medical records.
- Greater diagnostic and therapeutic precision via invasive and non-invasive methods.
- Education for physicians and patients.
- Improved decision-making in healthcare and public policy.

IX. KEY MESSAGES: THE 10 COMMANDMENTS OF HYPERTENSION

1. Remember: there is no correlation between symptoms and the severity of hypertension.
2. Measure BP correctly (in and out of the office).
3. Do not use monotherapy except in exceptional cases.

4. Initiate fixed combinations from day 1.
5. General target < 130/80 mmHg - sustain it permanently.
6. Always think MACARENHA: correct unhealthy habits, lipids, glucose, weight, and renal function.
7. Anticipate clinical inertia: adjust therapy when necessary (at least every 4-6 weeks).
8. Educate and empower patients and their families.
9. One combination pill is worth more than two or three separate tablets.
10. These commandments are lifelong.

CONCLUSIONS

The 2026 Mexican Hypertension Consensus, proposed by GREHTA, establishes a new clinical paradigm, redefining hypertension as an integral component of the MACARENHA phenomenon, which interconnects cardiovascular, renal, and metabolic health. The consensus prioritizes diagnostic precision through strict measurement protocols, both in and out of the clinical setting, to avoid diagnostic and therapeutic errors.

For pharmacological management, it recommends abandoning monotherapy and initiating fixed-dose combination therapy in a single tablet immediately, aiming to achieve BP targets below 130/80 mmHg. The guideline emphasizes that treatment success depends on simultaneously addressing lifestyle factors, obesity, glucose, and lipid abnormalities, rather than solely reducing blood pressure.

Finally, the consensus provides simplified algorithms and risk-stratification tools specifically designed to optimize primary care delivery in the Mexican population, and underscores the importance of leveraging emerging technologies and artificial intelligence.

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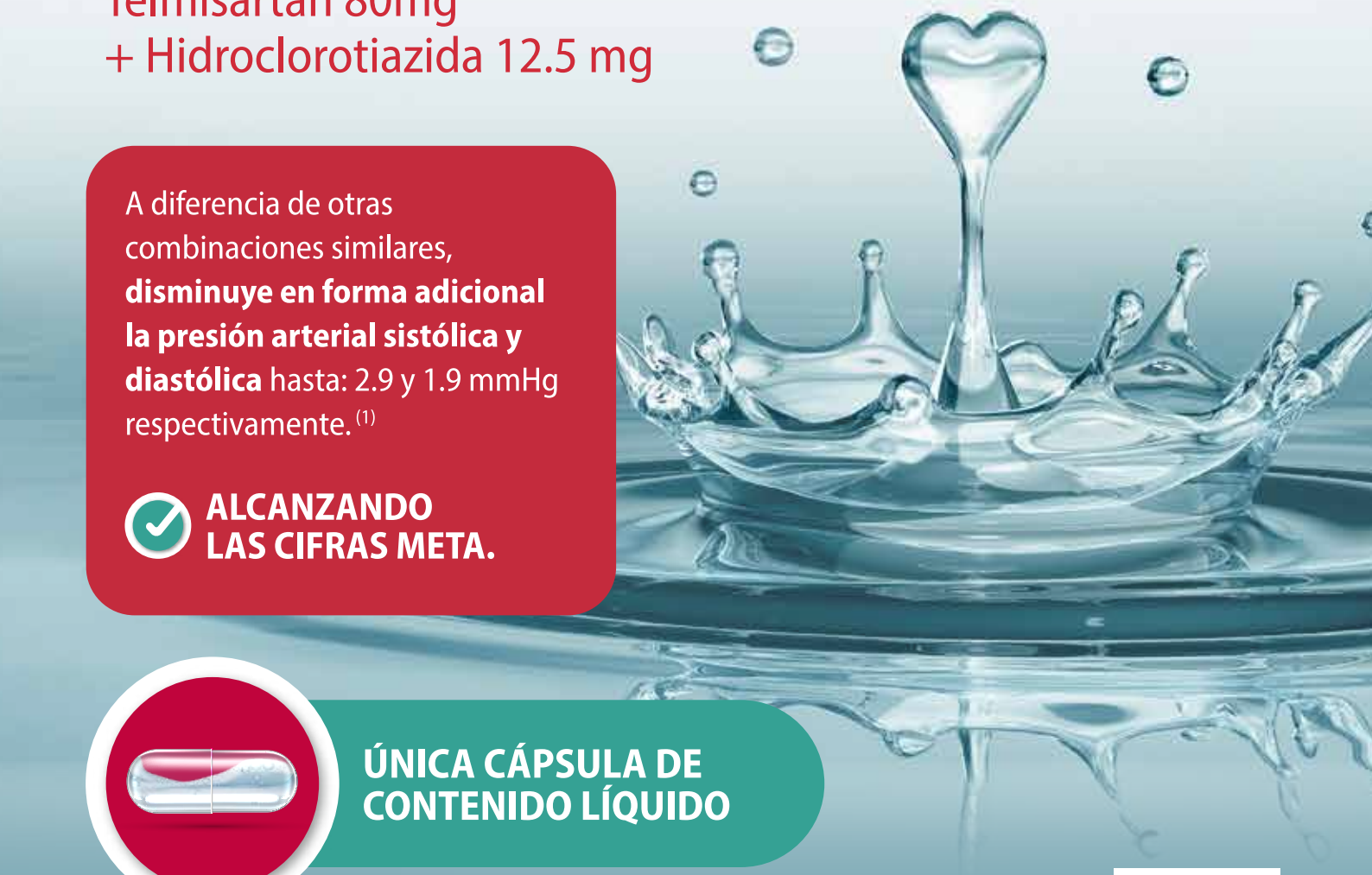

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