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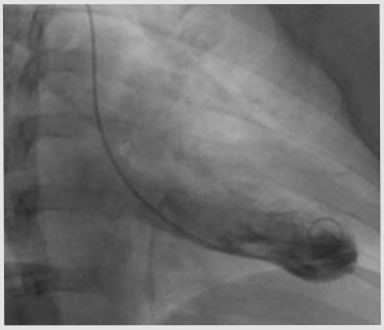

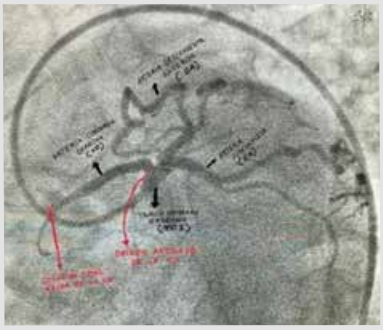

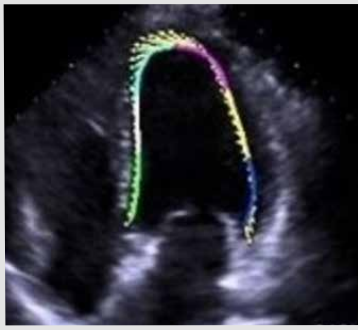

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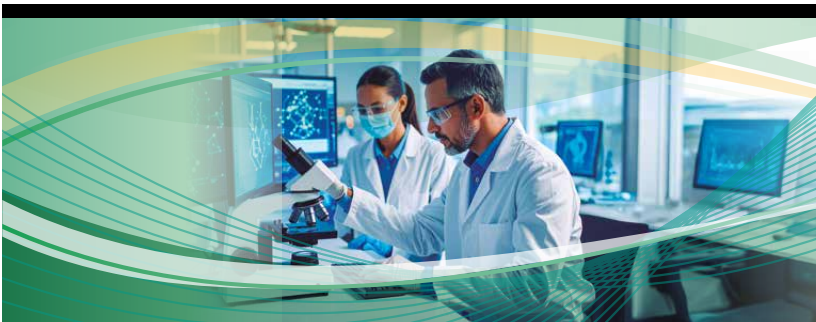

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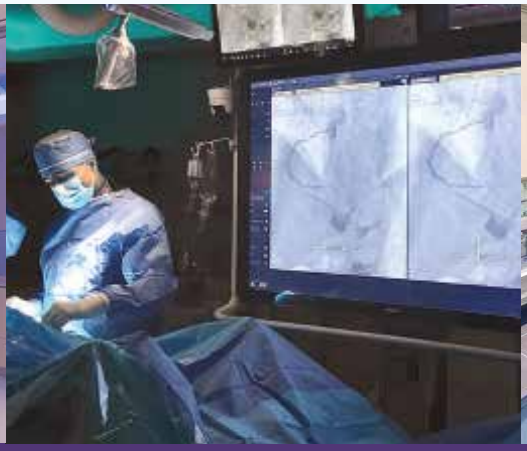

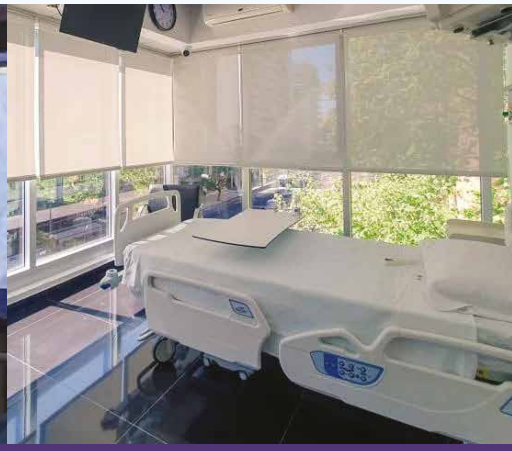

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Editorial

SCREENRA: A window to the best diagnosis, monitoring, and management of Abdominal Aortic Aneurysms in emerging economies.

Fernando Stuardo Wyss Quintana

Servicios y Tecnología Cardiovascular de Guatemala. S.A.

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Abdominal aortic aneurysm,
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screening.

ABSTRACT

In the study Prevalence of Abdominal Aortic Aneurysm through Echocardiographic Screening in a Population over 50 Years of Age: Argentine Multicenter Registry (SCREANRA) – Argentine Federation of Cardiology, the following question was raised: since it is an incidental diagnosis or one that is not detected until complications occur, can ultrasound identify aneurysms in early stages and favor their clinical follow-up?, showing interesting results that are applicable to emerging economies in countries with low–medium and medium-high economic incomes.

In 1996, during my first year of residence in Internal Medicine in the Emergency Unit of the Hospital General de Enfermedades del Seguro Social, I had a male, 54-year-old patient with acute abdominal pain of sudden onset, sharp and radiating to the back. The patient was admitted in hemodynamic shock and developed fatal cardiorespiratory arrest six minutes after arrival, with abdominal aortic dissection being determined as cause of death.

This experience was my first meeting with a pathology of catastrophic course. In the health care systems of emerging economies, this scenario poses two critical issues: the technical feasibility of a timely diagnosis in ER services (nonexistent in our nineties' reality, I'm not sure if even today) and the urgent need to implement efficient cardiovascular prevention strategies. Answering these questions is still a priority challenge for the low-medium income countries, and even for the medium-high ones.

Abdominal aortic aneurysm (AAA) is a located dilatation in the inferior part of the aorta, characterized by aortic diameter ≥1.5 times greater than the normal diameter at the level of the renal arteries (approximately a minimum diameter of 30 mm).[1,2] In spite of the previous reports suggesting an increasingly greater load of AAA, in terms of new cases and deaths, the data from the last two decades suggest the contrary, with marked decreases in incidence and mortality, particularly in Denmark, Sweden, New Zealand, the United Kingdom and the United States.[2,3,4,5] The decrease in the prevalence of smoking, observed in some high income countries, could have driven this decrease.[6] Besides the lower exposition to risks, using antihypertensive drugs and cardioprotective medications in many high income countries may have also contributed to this decrease.[7] However, in many low and middle income countries, the growing prevalence of smoking, hypertension, harmful consumption of alcohol and many cardiovascular risk factors are continuously notified, suggesting a possible growing load of AAA, particularly with a general response of relatively deficient public health.[6,7]

Epidemiological reports about AAA vary according to age, sex and geographical location.[7,8] The reports of programs of population screening reveal a significantly greater prevalence in men, with rates ranging from 1.9% to 18.5% and between 0.1% and 4.2% in women.[9] Most studies have identified an advanced age, male sex, smoking, hypertension and family history of AAA as the most significant risk factors driving the load of this disease.[10,11] Mortality also varies between countries and is usually related with pre-hospital response, care in the ER, hospital capacity and the type of intervention, and repair after AAA rupture.[7,12,13]

Argentina is classified as a medium-high income economy, a group of countries that live with high levels of poverty (affecting more than 50% of the population).[14] Therefore, seeking accessible strategies is indispensable in these environments.

In the prevalence study of abdominal aorta aneurysm by echocardiographic screening in a population older than 50 years: Argentine multicenter registry (SCREANRA) – Argentine Federation of Cardiology, the following issue was raised: as this is an incidental diagnosis or is detected only when complications arise, can ultrasound identify aneurysms in early stages and favor their clinical monitoring?

By cardiovascular or abdominal echographic studies made in multiple centers in Argentina, a multicenter, observational, cross-sectional and descriptive study of a screening program was carried out. In it, the anteroposterior and cross-sectional diameters of the abdominal aorta were measured in people older than 50

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years with risk factors for abdominal aorta aneurysm (AAA). There were 2148 patients included with an adequate distribution by gender, among which 77 cases of AAA were detected, representing a global prevalence of 3.58%, with most presenting in people older than 65 years. One the most relevant hypotheses was that echography or ultrasound, in their different modalities, is usually cost-effective, repeatable, safe, free from radiation and harmless. Most of all, it can be applied in different levels of care and it is very useful for follow-up and surveillance of AAA.

Color Doppler echocardiogram being the study that with greater frequency allowed to perform the abdominal aorta screening (1489 patients – 69.3%) was within the most important conclusions. In scenarios where there are no national screening programs, a timely detection during imaging studies represents a cost-effective strategy, so the evaluation of the abdominal aorta during transthoracic echocardiogram becomes particularly relevant, mainly in patients with a cardiovascular profile similar to those associated with AAA. Therefore, its results endorse the incorporation of the abdominal aorta evaluation as a supplement to the usual echographic tests; even in the field of primary care by trained physicians, improving early detection of AAA by a simple and cost-effective method, which in general we all have available.

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Review Article

From new biomarkers to emerging lipid-lowering therapies. What do the new 2026 American dyslipidemia guidelines tell us?

Mónica Acevedo[1], Paola Varleta[2], Ignacio Bezama[1], Samia Mora[3].

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ABSTRACT

Atherosclerotic cardiovascular disease is the leading cause of death. The new ACC/AHA dyslipidemia guidelines, endorsed by a total of eleven societies, provide the latest evidence and recommendations for evaluating and treating dyslipidemia. The guidelines highlight the importance of early promotion of cardiovascular health by a healthy lifestyle, early lipid screening, and precision-based primary and secondary prevention. Key takeaways include the early screening for dyslipidemia in childhood for identifying hereditary dyslipidemias, one-time screening for lipoprotein(a), and increased testing of apolipoprotein B in selected individuals. For improved decision making in individuals with borderline and intermediate risk, the guidelines recommend coronary artery calcium scoring and evaluating other high-risk conditions, such as family and reproductive history, cardiovascular-kidney-metabolic syndrome, and others. There is greater emphasis on treatment goals, not only for LDL but also for non-HDL cholesterol and apolipoprotein B. Statins remain the cornerstone of treatment, in addition to broader use of other evidence-based non-statin therapies in individualizing discussions between healthcare providers and patients.

Statement of conflict of interests

Mónica Acevedo has participated in Advisory Boards for AstraZeneca, Novo Nordisk, Boehringer Ingelheim, and Novartis. She has received fees for lecturing for: Adium Pharma, AstraZeneca, Boehringer, Novo Nordisk, Axon Pharma, Bayer, and Abbott.

Paola Varleta has participated in Advisory Boards for Axon Pharma, Novo Nordisk, Boehringer, and Novartis. She has received fees for lecturing for: Boehringer Ingelheim, AstraZeneca, Novo Nordisk, Axon Pharma, Abbott, Bayer and Adium. Investigator in clinical studies: Horizon (Novartis) / Victorion Prevent (Novartis).

Ignacio Bezama does not report conflicts of interest.

Samia Mora has participated as consultant in Medscape Education. She has received financing for personal research from the NHLBI (National Heart, Lung, and Blood Institute) and from the NIDDK (National Institute of Diabetes and Digestive and Kidney Diseases), and for her AHA (American Heart Association) Institution/Organization, the Health Effects Institute, Medscape Education and the International Atherosclerosis Society (with the latter there was no financial involvement). Co-inventor in a patent granted to Labcorp, related to the use of a glycosylation biomarker for MRI (GlycA) in relation to the risk of colorectal cancer, and co-inventor in a requested patent for a method for future risk prediction of cardiovascular disease by analysis of IgG glycome, assigned to GENOS d.o.o., and the Brigham and Women's Hospital. She was a member of the Advisory Board of AstraZeneca.

INTRODUCTION

Atherosclerotic cardiovascular disease (ASCVD) remains the first cause of mortality in the world,[1] in spite of advancements in pharmacological treatments and percutaneous intervention.

Dyslipidemia is still the main risk factor (RF) for ASCVD. Currently, it is known that not just LDL cholesterol is a cause for the disease, but there are also other atherogenic particles, such as lipoproteins rich in TG (TRLP) and lipoprotein(a) [Lp(a)], which may go through the intima and contribute to the atherogenic process.[2] This wider view of atherosclerotic CV risk, and the assessment of using new biomarkers and new tools for measuring CV risk have been clearly defined in the new 2026 US guidelines for the management of dyslipidemia, which also include the ACC and AHA (American College of Cardiology and American Heart Association, respectively) leadership, and another 11 societies involved in health care and with ASCVD patients.[3]

In this article, we will review what, from the point of view of the authors, are the main messages in the guidelines. The essential points to discuss are:

1. LDL cumulative exposure hypothesis.
2. Significance of healthy lifestyle, both in primary (PP) and secondary prevention (SP).
3. PREVENT-ASCVD risk score.
4. Use of the advanced biomarkers for the evaluation of CV risk and/or to determine the aims of the treatment, Lp(a) and apolipoprotein B (ApoB).

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5. Aims for the treatment for LDL, non-HDL cholesterol and ApoB.
6. New therapies for the management of dyslipidemia.

We invited Doctor Samia More, a US cardiologist expert in Lipidology, future president of the International Atherosclerosis Society, and one of the authors of the recently published guidelines, to participate in order to communicate as best as possible, the importance of these new guidelines.

1. CONCEPT OF LDL-CHOLESTEROL BUILDUP DURING LIFE

It is known nowadays that atherosclerosis occurs due to the accumulation of LDL cholesterol and lipoproteins containing ApoB on the arterial wall (VLDL, IDL, chylomicron remnants), including Lp(a), over life.[2] The greater the buildup of these particles on the arterial wall, the greater the risk of ischemic CV events. In other words, risk does not depend only on how elevated is LDL, but also on the extent of time of such exposition. This explanation has been called the theory of LDL cumulative exposure in terms of all ApoB-containing lipoproteins. However, it nearly always refers to LDL buildup, as 90% of these lipoproteins correspond to LDL. These lipoproteins that contain ApoB may go through the endothelium by transcytosis. The greater the accumulation in the intima, the more the atherosclerotic plaque will grow. If the plaque breaks, it produces a plaque rupture. Thus, accumulated LDL can be used to monitor plaque progression and the absolute risk of an acute atherogenic CV event. The risk of such event is not linear, but logarithmic, after an individual crosses the threshold of accumulated LDL from which plaque ruptures may occur. Therefore, if exposure to LDL is reduced from an early age, progression of atherosclerosis and event occurrence could be slowed and delayed.[4] Obviously, the predisposition of a person to an event depends on "their" accumulated load, hereditary predisposition, and the existence of other arterial wall damage factors, such as hypertension, diabetes and smoking. The LDL buildup threshold from which CV events start occurring can be estimated by the level of LDL cumulative exposure with the corresponding rate of events for each age: in men with average levels of LDL, the accumulated risk of acute CV events is ~1% after the accumulation of 130 years-plaque (in mmol/l) and 10% after 200 years-plaque (mmol/l). In women, it reaches 1% after 150 years-plaque (mmol/l) and 10% after 240 years-plaque (mmol/l). After menopause, years-plaque in women tend to be equal to men, as endogenous estrogens in the perimenopause period reduce LDL transcytosis in the intima. Therefore, currently the message about LDL cholesterol is: *"the lower the better, the earlier the better, and the longer the better"*.

2. SIGNIFICANCE OF HEART-HEALTHY LIFESTYLE

A heart-healthy lifestyle remains the cornerstone of both primary and secondary CV prevention. Guidelines emphasize again the need to promote and reinforce, since childhood to maturity, healthy habits that would contribute to reducing the risk of dyslipidemia and ASCVD. Among them, an appropriate diet, regular physical activity, healthy weight maintenance, proper sleep hygiene, stress management and abstinence from smoking and limited alcohol consumption stand out.

Evidence shows that reaching targets related to CVRF and healthy behaviors, including Life's Essential 8 from AHA, can reduce ~50% of relative risk of CV events, even in people with a genetic predisposition to ASCVD.[5,6]

Adopting a healthy diet since childhood is associated to a lower chance of developing dyslipidemia in adulthood. In general terms, a diet rich in fruits, vegetables, nuts, seeds, high-fiber foods, and monounsaturated and polyunsaturated fats are recommended. Between the different alternatives, the Mediterranean diet is the most recommended one due to its impact on reduction of CV mortality.[7] However, although healthy food patterns generate multiple CV benefits, such as weight loss, less inflammation, and blood pressure and glycemia control, its direct effect on LDL is usually modest.[3]

In regard to the impact of specific interventions on LDL, a vegan diet may reduce LDL in average 14.8 mg/dL, while the Mediterranean diet does not usually produce reduction of this magnitude. Daily consumption in three daily portions of fiber (28 g each) can reduce LDL ~5 mg/dL. The Portfolio diet, which combines nuts, soy protein, fiber and margarine enriched with plant sterols, can achieve reductions of ~26 mg/dL.[8]

Diet can also play a significant role in the treatment of hypertriglyceridemia (HTG). TRLP contribute to the development and progression of the atherosclerotic plaque, and therefore, to an increase in the risk of ASCVD.[2] For any degree of HTG, reducing consumption of sugar, total fat, and alcohol is a fundamental measure. Furthermore, it is advised to improve the quality of carbohydrates, favoring those with higher fiber content and lower glycemic index.[9] The risk of acute pancreatitis increases considerably when TG levels are ≥1000 mg/dL. In patients with genetic syndromes, such as the familial chylomicronemia syndrome, restricting carbohydrates is essential. In them, supplementing with liposoluble vitamins, minerals and medium-chain TG is usually required. Any person with TRLP elevation should supplement their diet with moderate to intense aerobic activity, plus resistance exercises for the upper and lower limbs, favoring a higher sensitivity to insulin, and contributing to weight maintenance and TG reduction.[9]

Obesity constitutes a great epidemiological problem in South America. Besides increasing CVRF, it may be considered a CV disease per se. It is frequently associated to atherogenic dyslipidemia. For this reason, a Mediterranean, a vegetarian or a vegan diet is recommended, always accompanied by aerobic and resistance exercises. In all people with dyslipidemia and overweight/obesity, a weight loss of at least 5% is recommended. This loss is mainly associated to a reduction in TG; the impact on LDL is limited. The glucagon-like peptide-1 (GLP-1) receptor agonists and dual GLP-1/GIP receptor agonists have shown significant effects on weight loss, TG and LDL reduction, glycosylated hemoglobin decrease, and improvement in atherosclerotic CV events.[10] Likewise, metabolic bariatric surgery has shown consistent benefits on TG and LDL, besides a reduction in CV risk.

Finally, in regard to exercise in people with dyslipidemia, moderate to intense aerobic exercise ≥150 minutes per week is advised, associated with resistance exercises twice a week. This favors the metabolism of lipids and increases the use of fatty acids

as an energy source. Exercise reduces TG and may increase HDL, besides contributing to controlling weight, improving sensitivity to insulin and improving RF[11]

3. ADDITION OF A NEW RISK STRATIFICATION TOOL, THE PREVENT-ASCVD, WITH BETTER CALIBRATION AND MORE ACCURATE THAN THE PREVIOUS POOLED-COHORT EQUATION, CLASSIFYING PEOPLE SINCE AGE 30

Atherosclerotic CV risk estimation is essential for decision-making to treat dyslipidemia, both for the indication of pharmacological therapy and to define the desired therapeutic target. International recommendations for the management of dyslipidemia always add some CV risk score that would meet validation criteria and applicability to their population. In the United States, the score used was the Pooled-Cohort Equation (PCE), recommended in 2013 by the AHA/ACC, and promoted because it adds multiple US cohorts in its validation. Another great strength of the PCE was having included atherosclerotic stroke in the risk estimation. Nevertheless, over the years it became evident that the PCE tended to overestimate the risk of ASCVD. Thus, in 2024, a group of AHA experts led by Sadiya Khan reviewed the prediction models and generated a new sex-specific CV risk equation, not differentiated by race, with better discrimination and calibration than the PCE, representing all the US population.

This new tool called PREVENT (Predicting Risk of CVD Events) seems to be an attractive and contemporary proposal, with modifications in regards to its predecessor.[12] PREVENT grants the chance to estimate CV risk within 10 and 30 years. In turn, it allows to estimate atherosclerotic CV risk, and the risk of heart failure and/or total CVD (ASCVD + heart failure). It also stands out due to the age range width, from 30 to 79 years. This is favorably compared with the SCORE (Systematic Coronary Risk Evaluation) of the European Society of Cardiology with two age ranges: the SCORE2, in apparently healthy individuals from 40 to 69 years and the SCORE2 OP in ≥70 years.[13,14] The PREVENT equation also integrates the concept of cardiovascular-kidney-metabolic syndrome (CKM), adding glomerular filtration rate and body mass index. As options, it adds the urine albumin/creatinine ratio and hemoglobin A1c. Finally, it introduces the social variable through the social deprivation index, providing a more equitable approach for the implementation of CV prevention. Due to the strength of the PREVENT, the AHA and the ACC decided to add it in this new guideline.

Along with the risk estimation by the PREVENT-ASCVD, the guideline recommends personalizing and possibly reclassifying the risk of individuals if necessary. Personalization is based on including particular conditions of the CV risk of some people, which should be taken into account, such as: family history of atherosclerotic CVD, being a carrier of some inflammatory disease, or having a gynecologic history of risk. Within these risk enhancing factors, elevated serum biomarkers are added, such as Lp(a) and high-sensitivity C-reactive protein *(Figure 1)*.

Reclassification is recommended in individuals in intermediate risk, with PREVENT score between 5% to <10%. Reclassification is made by the evaluation of subclinical atherosclerosis by coronary artery calcium test (CAC). This recommendation is based on the high percentage of reclassification yielded by CAC. It has been observed that in some studies, up to ~40% of patients in intermediate risk would present CAC = 0, and 25% CAC ≥100, which would lead to a reclassification into low and high risk, respectively. CAC is a test with a low dose of radiation and very cost-effective.[15]

4. A GREATER USE OF ADVANCED BIOMARKERS FOR THE EVALUATION OF RISK AND/OR TO DETERMINE THE GOALS OF THE TREATMENT, INCLUDING A SINGLE TEST OF LIPOPROTEIN(A) [LP(A)] FOR ALL INDIVIDUALS, AND A MORE WIDESPREAD USE OF APOB MEASUREMENT

The traditional lipid profile, both in a fasting state or not, is still the most useful tool to assess CV risk, guide the treatment, monitor the therapeutic response and verify targets are met. It is recommended to measure since age 9 to research familial dyslipidemias. This profile includes the direct determination of total cholesterol, HDL and TG; while LDL is estimated by mathematical formulas. Currently, it is advised to carry out the lipid profile in a fasting state in patients with TG ≥400 mg/dL and/or familial history of premature ASCVD or genetic dyslipidemias.

FIGURE 1.
Cardiovascular risk enhancers

New suggestions to determine LDL cholesterol

One of the main changes in the 2026 guidelines is the recommendation to use the Martin/Hopkins or Sampson/NIH equations instead of the Friedewald formula, for the estimation of LDL.[16,17] This is due to its greater accuracy in a wider range of LDL and TG concentrations. This suggestion is particularly relevant in patients with TG ≥150 mg/dL, LDL <70 mg/dL or non-HDL cholesterol <100 mg/dL. Both equations use LDL measurement as reference by β-quantification, and offer a more accurate estimation than the Friedewald formula, especially in patients with elevated TG and low LDL.

Apolipoprotein B

Another novel aspect is the recommendation of measuring ApoB in selected patients. Although LDL cholesterol is still the main parameter for CV risk evaluation and to define therapeutic targets, it only shows the amount of cholesterol transported by LDL particles. ApoB instead quantifies the total number of circulating atherogenic particles, as each LDL, VDLD and Lp(a) particle contains a single ApoB molecule.[18] The determination of ApoB becomes particularly relevant when there is a mismatch between LDL cholesterol and ApoB. This happens when LDL is within the control targets, but ApoB remains elevated, indicating a residual load of atherogenic particles, and the possible need to intensify the treatment. This mismatch is more frequent in patients with diabetes (DM), CKM syndrome, ASCVD or elevated TG. In this groups, LDL could be "controlled" while there remains a significant residual CV risk. ApoB in these cases would allow to research this residual risk as it is a direct measurement of the total number of atherogenic particles, including LDL, VLDL and Lp(a). The guideline, however, does not support a widespread use of ApoB in primary prevention of events, because there is no prospective evidence that measuring it would improve the prediction of CV events. In other words, ApoB should be considered as a supplementary tool, that improves but does not replace the treatment based on LDL cholesterol.

Lipoprotein(a)

A relevant incorporation to this guideline, also included in the last European guidelines of dyslipidemia, is the recommendation of measuring Lp(a), which is a particle similar to LDL, containing an apolipoprotein(a) bound to ApoB. Its concentration is mainly determined by the LPA gene, and it varies according to ascendancy.

The evidence from prospective and genetic tests shows a causal association between elevated concentrations of Lp(a) and a greater risk of ASCVD, calcified aortic valve stenosis, CV events and mortality. The risk of high Lp(a) is independent from LDL and other CVRF. It is considered high when an Lp(a) concentration is ≥125 nmol/L (50 mg/dL), which would be associated to an increase in the risk of ASCVD of 40%. The pathophysiological mechanisms of damage by Lp(a) include proatherogenic, proinflammatory and oxidative effects.[19]

Lp(a) levels are genetically determined and remain stable through life, so one measurement is usually enough to assess CV risk. Inheritance is autosomal codominant, and cascade genetic testing is recommended in first-degree relatives when elevated. The main limitation for measurement is the variability of Apo(a) isoforms. Currently, it is advised to use methods independent from isoform, and expressing results preferably in nmol/L.[20] *Figure 2* shows the steps to follow if Lp(a) is high.

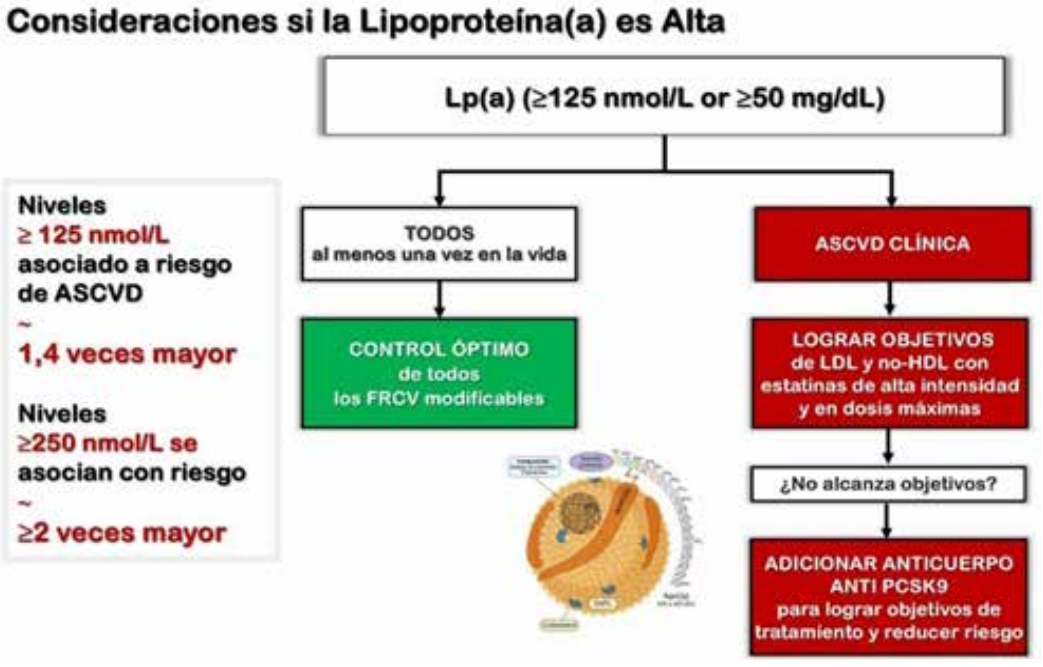

FIGURE 2. Considerations if Lp(a) is elevated

5. REINCORPORATION OF TREATMENT TARGETS, NOT JUST FOR LDL, BUT ALSO FOR NON-HDL CHOLESTEROL AND APOLIPOPROTEIN B (APOB) *(Table 1)*

The 2026 guideline reintroduces numerical and specific percentage treatment targets for LDL and non-HDL cholesterol in all risk categories, and for ApoB individuals in whom it was measured.

Table 1 summarizes control targets for the different risk categories. The recommendations in primary prevention in adults with ages from 30 to 79 years with LDL levels from 70 to 189 mg/dL are summarized in *Table 2.*

After estimating the PREVENT-ASCVD risk within 10 years, the risk-benefit balance should be evaluated to decide to start a lipid-lowering therapy, considering co-morbidities, life expectancy and preferences of the patient.

Primary prevention is based on a healthy lifestyle. When a pharmacological treatment is required, statins are the therapy of choice, with LDL reduction targets of ≥30% and ≥50% in people with greater CV risk. If the response is not enough, adherence should be optimized and the treatment should be intensified, with ezetimibe being the first drug to associate.

Cardiovascular risk estimation within 30 years supplements the 10-year evaluation, and it allows identifying individuals with PREVENT-ASCVD risk <3% within 10 years that may benefit from the treatment, particularly when LDL ≥160 mg/dL. Lipid-lowering therapy should be installed after optimizing lifestyle, in people

TABLE 1
Lipoprotein control targets for the different risk categories

Category of risk	LDL target (mg/dL)	Non–HDL target l (mg/dL)	ApoB target (mg/dL)
Borderline to intermediate risk	< 100	<1 30	< 90
High risk	< 70	< 100	< 70
Very high risk	<55	<85	<55

TABLE 2

Patients	LDL <100 mg/dL non-HDL<130 mg/dL	LDL<70 mg/dL non-HDL<100 mg/dL	LDL<55 mg/dL non-HDL<85 mg/dL
Primary prevention	PREVENT-ASCVD < 10% if TG ≥ 150 to 499 mg/dL - target apoB <90mg/dL	PREVENT-ASCVD ≥ 10% If TG ≥ 150 to 499 mg/dL - target apoB <70mg/dL	No data
Severe hypercholesterolemia	WITHOUT clinical ASCVD WITHOUT FH, WITHOUT CVRF WITHOUT subclinical atherosclerosis	WITHOUT clinical ASCVD WITH FH, WITH additional CVRF, or WITH subclinical atherosclerosis	FH WITH clinical ASCVD
Diabetes	WITHOUT CVRF for ASCVD or WITHOUT specific RF modifiers-of-DM - target apoB: <90 mg/dL	WITH CVRF for ASCVD WITH specific RF-modifiers of-DM- - target apoB: <70 mg/dL	No data
Subclinical atherosclerosis	CAC ≥1 to 99 and < p75 for age, sex and race	CAC ≥100 to 299 or ≥Pc75th for age, sex and race CAC ≥ 300 to 999 AU Optional targets: - LDL< 55 mg/dL - non-HDL < 85 mg/dL. - apoB <55 mg/dL.	CAC ≥ 1000 AU
Hypertriglyceridemia	<50 years WITHOUT additional risk enhancers	WITH clinical ASCVD and NO very high risk - ApoB: < 70 mg/dL 40 -75 years WITH ≥1 RF for ASCVD - ApoB: <70 mg/dL	WITH clinical ASCVD of very high risk -ApoB: <55 mg/dL
Clinical ASCVD	No data	WITHOUT very high risk Optional targets: - LDL< 55 mg/dL - non-HDL < 85 mg/dL. - ApoB <55 mg/dL	WITH very high risk -ApoB: <55 mg/dL WITH CKD

CVRF: cardiovascular risk factors; **ASCVD:** atherosclerotic cardiovascular disease; **DM:** diabetes mellitus; **p:** percentiles; **FH:** familial hypercholesterolemia; **CKD:** chronic kidney disease; **CAC:** coronary artery calcium score.

with PREVENT-ASCVD within 30 years of ≥10%, and in those with high levels of atherogenic lipids, multiple CVRF or moderate CAC. Given the relative reduction of risk with statins is relatively constant, the absolute benefit increases according to the baseline risk. For this reason, if the risk within 10 years is ≥3%, therapy with a statin of moderate intensity can be started. In those with PREVENT-ASCVD between 3% and <10%, the target is an LDL reduction of ≥30%. In this group, the targets are: LDL <100 mg/dL and non-HDL cholesterol <130 mg/dL.

Individuals with PREVENT-ASCVD ≥10% within 10 years should receive high-intensity statins for an LDL reduction of ≥50%, with LDL targets <70 mg/dL and non-HDL <100 mg/dL. If the target is not reached, ezetimibe could be added. Bempedoic acid has also proven to reduce CV events in patients in high risk who are intolerant to statins. PCSK9 inhibitors and inclisiran, although strong LDL reducers, still lack definitive evidence of CV event reduction in primary prevention. There is no neat benefit proven to start statins when LDL is <70 mg/dL and non-HDL <100 mg/dL.

The recommendations for adults with diabetes and no established atherosclerotic cardiovascular disease are shown in *Table 2.*

Individuals with DM present an elevated risk of ASCVD; in them, an intensive control of LDL is a priority in primary prevention. In most adults from 40 to 75 years of age, it is recommended to start with statins of moderate intensity to reduce LDL of 30.49%.

In patients with additional CVRF or with PREVENT-ASCVD ≥10% within 10 years, it is recommended to reduce LDL ≥50%, with high-intensity statins. If the target is not met or there is intolerance to statins, ezetimibe can be added, or even PCSK9 inhibitors or inclisiran, powerful drugs to reduce LDL. In patients with DM and additional RF and persistent elevation of TG (150-499 mg/dL), in spite of treatment with statins, eicosapentaenoic acid (EPA) can be considered to reduce residual risk.

In adults >75 years, use of statins of moderate or high intensity should be individualized according to life expectancy, comorbidities and risk of adverse effects in patients.

In young people with DM with ages from 20 to 39, evidence of treatment and reduction of ASCVD is limited. Statins of moderate intensity can be considered in people with T2D ≥10 years or T1D ≥20 years coursing, particularly if it coexists with hypertension, smoking or microvascular complications. The goal is a reduction in LDL ≥30%, intensifying the treatment if the risk is higher. In all cases with DM >30 years, PREVENT-ASCVD within 10 and 30 years is recommended to guide the intensity of the lipid-lowering treatment and the control targets.

The recommendations for the secondary prevention of ASCVD are shown in *Table 2*.

In individuals with ASCVD that do not present a very high CV risk, treatment should be started with high-intensity statins for a reduction ≥50% in LDL cholesterol, to reach a target of <70 mg/dL and non-HDL cholesterol <100 mg/dL to reduce the risk of recurrent CV events. Patients with ASCVD with very high risk also require high-intensity statins to reduce LDL ≥50%. The LDL target is <55 mg/dL and non-HDL cholesterol <85 mg/dL, with an optional ApoB <55 mg/dL. Patients with ≥2 major ASCVD (acute coronary syndrome <12 months, myocardial infarction different from the previous one, ischemic stroke, and symptomatic peripheral vascular disease) or a major CV event associated to 2 or more high risk conditions, are considered to be in very high risk *(Figure 3)*.

In patients with ASCVD with very high risk, who do not reach the established targets, it is possible to add ezetimibe, PCSK9 inhibitors, inclisiran or bempedoic acid (reduction of ~20%, ~60%, ~50% and ~20%, respectively). Evidence has shown safety, even with LDL ~30 mg/dL in the follow-up of patients up to 8 years. In patients with ASCVD without very high risk, the target is LDL <70 mg/dL and non-HDL cholesterol <100 mg/dL; but, in some cases, LDL <55 mg/dL and non-HDL <85 mg/dL can be requested.

The recommendations for the management of adults with subclinical coronary atherosclerosis (men ≥40 or women ≥45 years) are shown in *Table 2.*

FIGURE 3.
Criteria to define very high cardiovascular risk

Subclinical atherosclerosis has acquired great relevance in the prevention of ASCVD. It has been proven that CAC provides sound prognostic information, with an almost linear relationship between CAC load and the risk of events. In men ≥40 and women ≥45 years and people with PREVENT-ASCVD borderline or intermediate, CAC can help to define the onset of lipid lowering drugs.

A CAC ≥1000 AU identifies patients in very high CV risk, comparable to that observed in secondary prevention. In them, it is recommended to reduce LDL ≥50%, with LDL goals >55 mg/dL and non-HDL <85 mg/dL. A combination of high-intensity statins, ezetimibe, PCSK9 inhibitors, inclisiran and/or bempedoic acid may be required to reach the targets. People with CAC between 300 and 999 AU also present a risk that is similar to that of patients with ASCVD. In them, LDL-C <70 mg/dL and non-HDL <100 mg/dL is proposed, and LDL-C <55 mg/dL, non-HDL <85 mg/dL and ApoB <55 mg/dL can be considered. Patients with CAC ≥100 AU show rates of events similar to those observed in secondary prevention, and clearly benefit from statins, with a reduction in major events and less progression to the atherosclerotic plaque. For this reason, the same therapeutic goals are suggested than with CAC ≥300 AU. Finally, in adults with CAC between 1 and 99 AU, it is considered reasonable to start statins, seeking to reduce LDL-C ≥30% and a target of LDL-C <100 mg/dL.

6. NEW THERAPIES FOR THE MANAGEMENT OF DYSLIPIDEMIA

Emerging lipid-lowering therapies are oriented toward three great goals: a more intensive reduction of LDL, a decrease of Lp(a) and control of severe HTG.

Inclisiran: it is a small RNAi, producing the catalytic destruction of mRNA of PCSK9, decreasing LDL receptor degradation and reducing LDL ~50%. Its advantage is the six-month administration after the load dose. It is considered in patients with ASCVD or severe hypercholesterolemia that do not reach their targets.

Bempedoic acid inhibits ATP citrate lyase, an enzyme previous to HMG-CoA reductase, reducing LDL-C ~18-25%. It has shown efficacy, safety and reduction of CV events in patients intolerant to statins. Guidelines add it as additional therapy in primary prevention of high risk and secondary prevention.

Olezarsen is a new antisense oligonucleotide, that binds to the mRNA of the ApoC-III and degrades it, thus reducing ApoC-III. Therefore, it increases TG and VLDL removal. Its use has been only approved for the treatment of familial chylomicronemia. It is administered monthly subcutaneously, reaching an average reduction of TG of 50% in 6 months. Other agents being developed: plozasiran, zodasiran and solbinsiran. They all act on ApoC-III or ANGPTL3 and may reduce triglycerides in more than 70%.

Evinacumab corresponds to a 100% human monoclonal antibody that binds to the ANGPTL3 enzyme and inhibits it. ANGPTL3 is a protein that is secreted and expressed only in the liver, inhibiting lipoprotein lipase. It produces reduction of TG, ApoB and LDL. LDL reduction is independent from the LDL receptor and it would occur by reduction in liver secretion of VLDL and increase in LDL removal, reducing LDL ~49%. Evinacumab is administered intravenously every 4 weeks in doses of 15 mg/kg. Currently, its use is restricted to patients with homozygous familial hypercholesterolemia.

Therapies for Lp(a): currently there is no treatment approved specifically to reduce CV events associated to Lp(a), but studies are very promising:

Pelacarsen (antisense oligonucleotide inhibiting Apo(a)), olpasiran (RNAi for Apo(a)), lepodisiran (siRNA of prolonged action) and muvalaplin (oral inhibitor of Apo(a)-Apo(b) binding). These therapies achieve Lp(a) reductions of 80-95% in phase 2 studies. The results of cardiovascular outcomes are expected between 2026 and 2028.

Lomptapida selectively inhibits the microsomal TG transfer protein (MTP), which is essential for the assembly of lipoproteins containing ApoB in the liver. Thus, it inhibits the synthesis of chylomicrons and VLDL, reducing LDL cholesterol by 40% to 50%. Its use is also limited to patients with homozygous familial hypercholesterolemia. It is associated with significant secondary gastrointestinal effects, with hepatotoxicity and hepatic steatosis. It is an oral drug, requiring liposoluble vitamin supplements and other fats to alleviate the malabsorption produced by these liposoluble vitamins and other fatty acids.

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Original Investigation Reports

Results of subintimal and intraluminal angioplasty in chronic total occlusion of the superficial femoral artery.

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ABSTRACT

Introduction: endovascular revascularization is key in the management of chronic lower limb ischemia.

Objective: to describe the outcomes of subintimal angioplasty in the treatment of superficial femoral artery chronic total occlusion.

Materials and methods: observational, descriptive, cross-sectional, and prospective study. The study population consisted of patients diagnosed with chronic total occlusion of the superficial femoral artery, treated at a referral center in the Autonomous City of Buenos Aires during the period from January 2022 to December 2023.

Results: 42 patients were selected, median age 68 years, 16 women (38.1%), with a Leriche-Fontaine stage III in 32 patients (76.2%), and IV in 10 (23.8%), and an occlusion length of more than 10 cm in the entire sample. Pre-procedure ABI was less than 0.9 in all cases, and greater than 0.9 post-procedure ($p<.001$). Intra-procedural adverse events occurred in 28 patients (66.7%) and only three required reinterventions.

Conclusion: subintimal and intraluminal angioplasty represent viable and effective revascularization techniques in the treatment of patients with chronic lower limb ischemia in the femoral artery territory.

INTRODUCTION

Critical limb ischemia (CLI) constitutes the most advanced stage of peripheral artery disease, characterized by severe arterial obstruction in the lower limbs, with a high impact on quality of life and a high rate of morbimortality.[1] This scenario, characterized by pain in rest and ischemic lesions, represents a therapeutic challenge. Coronary chronic total occlusions (CTO) are defined as those with ischemic pain in rest (type III of the Leriche-Fontaine classification) or critical ischemia with development of ulcers or gangrene (type IV).[2] CTOs are usually found in CLI, with a frequency of up to 40% in patients with symptomatic peripheral arterial disease. The complex nature of CTOs, particularly those with long lesions and intense calcification, may negatively affect immediate results and the mid-term after endovascular treatment.

Angioplasty or endovascular revascularization has emerged as a safe and efficient alternative, standing out for being less invasive and risky in comparison with open vascular surgery.[3] However, CLI is often associated with diseases in multiple levels of severe calcification, demanding a combined strategy approaching both inflow (aortoiliac and femoral) and outflow (tibial).[4] Although a large part of evidence on the endovascular treatment of inflow comes from studies focused on claudication, specific results in patients with CLI are limited.[1] Angioplasty, that could be made endoluminally or by percutaneous intentional extraluminal recanalization (PIER), offers a viable option, but results in the long term in this population are uncertain.[5,6] This study attempts to describe the results of angioplasty in the treatment of CTO in the superficial femoral artery.

MATERIALS AND METHODS

Design of the study

Observational, descriptive, cross-sectional and prospective study. Its design adhered to the STrengthening the Reporting of Observational studies in Epidemiology (STROBE) recommendations.[7] The study population was made up by patients diagnosed with chronic total occlusion of the superficial femoral artery, who received care in a reference center in the Autonomous City of Buenos Aires, during the term from January 2022 to December 2023.

Selection criteria

All patients older than 18 years, with diagnosis of CLI, with pre-procedure CTO (type III or IV according to the Leriche – Fontaine scale), intervened by angioplasty.[2] Patients with non-atherosclerotic occlusion (thrombosis, dissection or embolism) were not considered in this study. Also, patients in whose clinical files there was no information necessary for this study were excluded.

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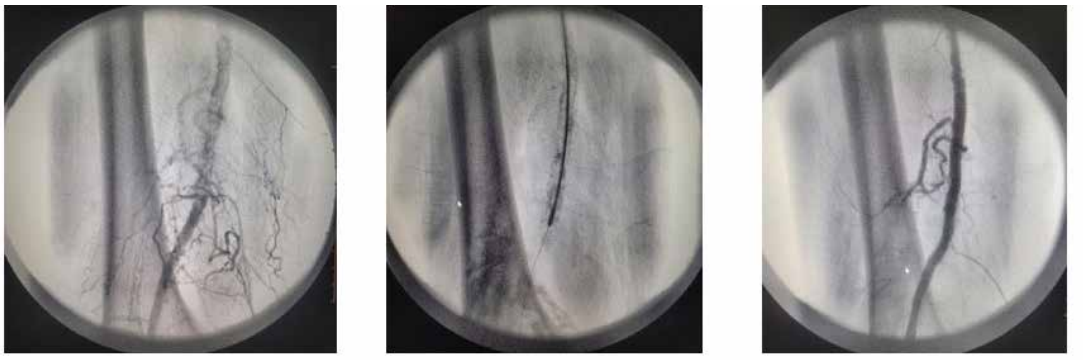

FIGURE 1.
Lower limbs arteriography.
3A. Chronic occlusion of the left superficial femoral artery with abundant calcification. **3B.** Endovascular treatment with peripheral rotational atherectomy devices, with aspiration system and self-expandable blades. **3C.** Final outcome with peripheral prosthesis, with appropriate flow and diameter.

Procedure and technique

Anterograde puncture was made with hydrophilic wire 0.35, supported through vertebral catheter of 4 or 5 Fr. Once the wire could be advanced, the catheter was pushed through. Devices for peripheral rotational atherectomy were used, with aspiration system and self-expandable blades *(Figure 1)*. Confirmation was made by angiography. About catheter placements, a wire was placed, through which the balloon or prosthesis was pulled.[8]

Outcomes in the short and long term

Outcomes in the short term constituted the rate of technical and clinical success, and adverse events related with the procedure. Clinical success was defined as the identification of a femoral artery with adequate flow and improvement in ankle-brachial index (ABI). Outcomes in the long term constituted permeability six months after the procedure, and rate of reintervention.

Statistical analysis

Continuous variables are expressed as mean and standard deviation (SD) or median (interquartile range - IQR), according to normal or asymmetrical distribution of data, which was evaluated by the Kolmogorov-Smirnov test. Categorical variables are presented as numbers and percentages.

ABI was contrasted before and after the procedure by Wilcoxon test, and illustrated by alluvial diagram. Work was carried out with a 95% confidence interval, a critical or rejection (alpha) region of 0.05 was preset, associated to the p or probability value. The statistical program SPSS version 25 was used for data analysis.

Ethical and legal aspects

The investigators participating in this study have followed the applicable ethical and legal regulations, namely the Declaration of Helsinki. Informed consent has been obtained from patients, the Committee of Ethics of the hospital has granted its approval.

RESULTS

From a total of 42 selected patients, the median of age was 68 years (IQR 65-72) and 16 were female (38.1%). In regard to comorbidities, 33 had diabetes mellitus (78.6%), 29 hypertension (69%) and 26 dyslipidemia (61.9%) *(Table 1)*. Grade III from the Leriche-Fontaine scale was estimated in 32 patients (76.2%) and grade IV in 10 (23.8%). The length of occlusion was more than 10 cm in the whole sample, with a median of 13 cm (IQR 10-15). A high content of calcium was identified in 31 patients (73.8%), while pre-procedure ABI was less than 0.9 in all cases *(Table 1)*.

The subintimal technique was applied in 33 patients (78.6%) and the endoluminal one in 9 (21.4%), requiring the placement of an endoluminal prosthesis in 33 (78.6%). After angioplasty, all cases presented ABI of 0.9 to 1.0 ($p<.001$). From the 28/42 patients with ABI of less than 1.0 (66.7%), 4/28 presented pre-procedure ABI of 0.5, 0.6 in 9/28, 0.7 in 14/28, and 0.8 in 1/28. From the 14/42 patients with post-procedure ABI of 0.9 (33.3%), 6/14 presented pre-procedure ABI of 0.5, 0.6 in 6/14, 0.7 in 1/14, and 0.8 in 1/14 *(Figure 2)*.

TABLE 1.
Baseline characteristics of the study sample

	(N=42)
Age (years), median (IQR)	68 (65 – 72)
Sex (female), n (%)	16 (38.1)
Diabetes mellitus, n (%)	33 (78.6)
Hypertension, n (%)	29 (69.0)
Dyslipidemia, n (%)	26 (61.9)
Smoking, n (%)	33 (78.6)
Pre-procedure occlusion (Leriche – Fontaine scale), n (%)	
Grade III	32 (76.2)
Grade IV	10 (23.8)
Length of occlusion (cm), median (IQR)	13 (10 – 15)
High calcium content	31 (73.8)
Pre-procedure ABI, n (%)	
0.5	10 (23.8)
0.6	15 (35.7)
0.7	15 (35.7)
0.8	2 (4.8)

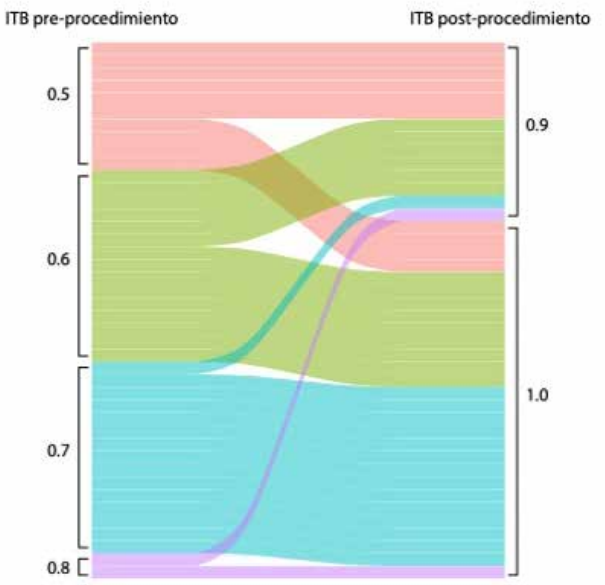

FIGURE 2.
Evolution of the lesions with ischemic origin

TABLE 2.
Percutaneous management, outcomes in the short and long term

	(N=42)
Technique, n (%)	
Endoluminal	9 (21.4)
Subintimal	33 (78.6)
Use of prosthesis, n (%)	26 (61.9)
Post-procedure ABI, n (%)	
0.9	14 (33.3)
1.0	28 (66.7)
Intra-procedure complications, n (%)	
Dissection	8/28
Recoil	20/28
Local hematomas, n (%)	8 (19.0)
Permeability six months after the procedure, n n (%)	40 (95.2)
Vascular reintervention, n (%)	3 (7.1)

Intraprocedure adverse events were documented in 28 patients (66.7%), being dissections in 8/28 patients, and recoils in 20/28. Likewise, local hematomas were documented in eight patients (19%), but no retroperitoneal hematoma. In 40 patients, permeability was shown six months after the procedure (95.2%) *(Figure 3)*. Only in three patients reintervention was necessary, including two in whom there was permeability (7.1%) *(Table 2)*.

DISCUSSION

The first attempt of subintimal angioplasty was in 1987 to treat CTO in the femoral-popliteal segment, with rates of technical success of 76%, and an acceptable rate of complications of 5.6%.[9] In general, the procedure uses the wire and catheter technique to approach the subintimal plane, right above the level of the CTO.[5] A wire forming a loop is used to go through the occlusion in this subintimal space of low resistance, to return to the true lumen of the artery beyond the occlusion. In a systematic review of 23 cohort studies, including 1549 patients with CLI who underwent subintimal angioplasty of peripheral arterial occlusive disease, the reported rates of success and the rates of saving a limb within a year ranged between 80% and 90%. The study concluded that subintimal angioplasty has an important role, particularly in patients with chronic ischemia, to provide wound healing and saving limbs.[10]

A recent Egyptian study compared angioplasty in 260 patients treated at the level of the superficial femoral artery vs 190 treated at the level of the popliteal artery, reporting a global technical success in 93.3% of cases. Lesions at the level of the superficial femoral artery presented a technical success of 91.2%. The prevalence of vessels of null leakage in patients treated at the level of the superficial femoral artery was 11.5%, contributing to a better rate of technical success. Nevertheless, technical success in literature can reach up to 95.1% for the treatment with subintimal angioplasty of CTO at femoral-popliteal level.[11]

In the case of patients with CLI that present severe symptoms and that also present a prohibitive surgical risk, a deficient drainage, and lack of a proper autologous duct, many medical societies consider that angioplasty is the first line therapy, in spite of the anatomical limitation of the disease.[12,13] However, conventional femoral access during angioplasty of complex arterial lesions

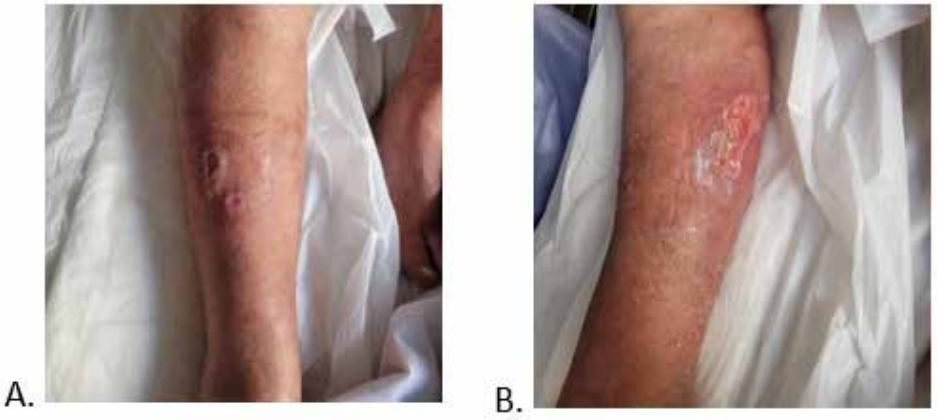

FIGURE 3.
Evolution of the lesions with ischemic origin

could be very complicated, and may fail in up to 20% of cases.[14] Furthermore, there are different preferences in regard to the anterograde vs retrograde approach, with the anterograde being the one most commonly used. However, one of the advantages attributed to using retrograde access is a softer distal occlusion cap in comparison with the hard fibrotic proximal cap, through which the retrograde wire can pass more easily. It also provides a greater capacity for the wire to be pushed due to the closeness of the CTO with the retrograde access, together with a lower possibility for the retrograde wire to shift toward the lateral branches that originate in a craniocaudal direction.

It is important to highlight that a selective placement of the endoluminal prostheses is more frequent in lesions at superficial femoral level vs popliteal level. Unlike the superficial femoral artery approach, the popliteal artery has a different embryology, as it originates at the sciatic system. Furthermore, while the superficial femoral artery and the tibial artery have an intramuscular course, the popliteal artery does not go through any muscle compartment. Even more important, the popliteal artery is exposed to huge biomechanical forces due to the frequent flexion of the knee and the repetitive movement of the ankle. These distinctive properties of the popliteal artery may affect the results of the endovascular treatment options.[15] Consequently, the acceptable concerns about vessel kinking and a possible restenosis and prosthesis fractures lead to consider that this vessel is not appropriate for prosthesis placement.

The ideal result from revascularization in patients with peripheral arterial disease is the complete relief of ischemic symptoms, particularly wound scarring in patients with ischemic wounds. Therefore, the evaluation of these patients should consider wound scarring and maintenance of a state free from wounds.[16] The scarring rates of wounds are significantly better in the superficial femoral artery approach within three months (more than 30%) and in one year (more than 80%).[17] Even with moderate permeability rates in the long term, one of the valuable results of subintimal angioplasty in patients with CLI is helping to wound scarring and recovering the limb, as supported by an extensive systematic review.[12]

This study presents certain limitations. It was a retrospective, single-center study, with one arm, focused on the management of superficial femoral artery. The number of cases prevented making some specific subgroups analyses. A future aim is performing a large prospective multicenter study, thoroughly comparing subintimal angioplasty of CTO using different types of prosthesis, as well as the results from subintimal angioplasty versus intraluminal angioplasty.

CONCLUSIONS

Subintimal and endoluminal angioplasty represent feasible and efficient revascularization techniques in the treatment of patients with CLI of the CTO type at the level of the superficial femoral artery. A high rate of revascularization is achieved with a low rate of reintervention. These techniques should be considered a first line treatment in patients with such a process, saving surgical revascularization for individuals in whom the percutaneous procedure has failed.

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Original Investigation Reports

National Registry of Intraoperative/Intraprocedural transesophageal echocardiography

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ABSTRACT

This national registry represents the first systematic survey on the use of transesophageal echocardiography (TEE) in cardiovascular surgery and interventional cardiology procedures in Argentina. A multicenter survey was conducted among members of the Argentine Federation of Cardiology, with responses obtained from 32 centers across 16 provinces. Results showed that 68.8% of centers use TEE in cardiovascular surgery and 78.1% in structural procedures, although limitations remain related to insufficient equipment, trained personnel, and standardized protocols. Only half of the centers reported access to three-dimensional technology, and a similar proportion had institutional guidelines for TEE implementation. Most procedures did not report major complications, confirming the safety of the technique. These findings highlight the growing adoption of TEE in Argentina, though with regional heterogeneity, and underscore the need to strengthen interdisciplinary training and to develop national consensus recommendations adapted to local healthcare settings.

INTRODUCTION

Using transesophageal echocardiogram (TEE) is increasingly more frequent in the fields of the OR and catheterization laboratories.[1] However, in Argentina, there are scarce data published. Use of TEE in ORs and catheterization laboratories adds additional challenges, proper of the scenario: who is in charge of performing the test, what are the indications, procedure standardization, device management, detection and management of complications.[2,3,4,5] On the other hand, access to more modern technologies, such as 3D images, is very promising but unequal in the different centers using TEE in the OR and/or catheterization laboratory.[6]

Plenty of international medical societies have concerned themselves to describe these data. It is essential to have our own information available in our country.[7,8]

AIMS

To determine the frequency of use of transesophageal echocardiogram both at intraoperative level and intraprocedural level in interventional cardiology, as well as indications of use, time during procedure and possible complications.

MATERIALS AND METHODS

A survey was carried out with multiple choice questions, where echocardiography cardiologists participated, all members of the societies that are part of the Argentine Federation of Cardiology (FAC) *(Annex, Table 1)*. Since its dissemination, carried out by direct phone contact with the professionals, and reinforced by e-mail through FAC, a period was determined to complete it, from September to December 2024. Starting from it, a multicenter, prospective, observational, cross-sectional study was developed.

Registry inclusion criteria

Patients with ages greater or equal to 18 years, in whom transesophageal echocardiogram was carried in a scenario of heart surgery or structural interventional cardiology procedure.

Definitions

- Heart surgery: myocardial revascularization surgery, valve replacement surgery, aortic surgery, valvuloplasty, surgical correction of congenital heart disease, heart transplant.
- Structural interventional cardiology procedure: percutaneous repair of the mitral or tricuspid valve, percutaneous implant of the aortic, pulmonary, mitral or tricuspid valve, percutaneous closure of left atrial appendage.

Statistical analysis

A descriptive analysis was made on the answers to the survey. Categorical variables are expressed as percentages and were compared by the Chi-square test. Continuous variables are presented,

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ANNEX

CENTER DATA

- Name of the center
- Province
- City
- In average, how many heart surgeries in adults are made in your center yearly?
- In average, how many procedures of structural interventional cardiology in adults are made in your center yearly?
- Is intraoperative transesophageal echocardiography conducted in your center?
- Is SHD intraprocedural (TAVI, appendage closure, mitral valve repair, etc.) echocardiography conducted in your center?

IF INTRAOPERATIVE/INTRAPROCEDURAL TEE IS NOT PERFORMED IN YOUR CENTER

- The reason not to do it is mainly:
  o It is not considered necessary.
  o There is no necessary equipment available.
  o There is no necessary staff available.
  o Another reason.
- Are you thinking about starting to use intraoperative/introprocedural TEE in your center soon?

INTRAOPERATIVE TEE

- About the person in charge of carrying out the TEE, what is their specialty? Cardiology, anesthesiology, surgery/hemodynamics, technician.
- About the person in charge of placing the TEE probe, what is their specialty? Cardiology, anesthesiology, surgery/hemodynamics, technician.
- Does TEE have 3D mode?
- In what percentage of mitral valvuloplasties is TEE used?
- In what percentage of valve replacements is TEE used?
- In what percentage of on-pump coronary artery surgeries is TEE used?
- In what percentage of off-pump coronary artery surgeries is TEE used?
- In what percentage of combined surgeries is TEE used?
- In what percentage of heart transplants is TEE used?
- In what percentage of aortic surgeries is TEE used?
- Is there a specific TEE protocol in use in your center?
- In general, how many times is TEE used during surgery? Before, during, at the end, when pumping ends.
- How frequently did TEE modify the surgical strategy during surgery? Never, a few times, most of times, always.
- Are the videos and images obtained during TEE stored?
- Is the TEE report made?
- Did a complication threatening the life of patients ever occur? No, esophageal perforation, bleeding, another.

INTRAPROCEDURAL TEE

- About the person in charge of performing the TEE, what is their specialty? Cardiology, anesthesiology, surgery/hemodynamics, technician.
- About the person in charge of placing the TEE probe, what is their specialty? Cardiology, anesthesiology, surgery/hemodynamics, technician.
- Does TEE have 3D mode?
- In what percentage of percutaneous aortic valve implantations is TEE used?
- In what percentage of percutaneous mitral valve repairs is TEE used?
- In what percentage of LA appendage closures is TEE used?
- In what percentage of percutaneous tricuspid valve repairs is TEE used?
- In what percentage of percutaneous pulmonary valve implantations is TEE used?
- In what percentage of septal ablations is TEE used?
- In what percentage of congenital diseases procedures in adults is TEE used?
- Is there a specific TEE protocol available in your center?
- In general, how often is intraprocedural TEE used? Before, during, at the end.
- With what frequency did TEE modify the surgical strategy during surgery? Never, a few times, most of times, always.
- Are the videos and images obtained during TEE stored?
- Is the TEE report made?
- Did a complication threatening to the life of patients ever occur? No, esophageal perforation, bleeding, another.

USE OF TEE

- How frequently is TEE used for the following?
  (Never, sometimes, frequently, always)
  o Adjustment of pharmacotherapy
  o Help in device placement (IABP, ventricular assist device, etc.)
  o Evaluation of valve disease
  o Post-bypass evaluation

- Is TEE used to evaluate the following parameters?
  Never, sometimes, frequently, always.
  o Left and right ventricular function
  o Contractility anomalies
  o Diameters
  o Valve function
  o Diastolic function
  o Presence of patent foramen ovale
  o Intracardiac thrombi
  o Atheroma and aortic dissection.

TABLE 1
Answers by province.

Province	Frequency	Percentage (%)
Buenos Aires	3	9.4
Chaco	2	6.3
Chubut	1	3.1
Córdoba	4	12.5
Corrientes	1	3.1
Entre Ríos	2	6.3
Formosa	2	6.3
Jujuy	1	3.1
La Pampa	1	3.1
Mendoza	2	6.3
Misiones	2	6.3
Neuquén	1	3.1
Salta	1	3.1
Santa Fe	7	21.9
Santiago del Estero	1	3.1
Tucumán	1	3.1
TOTAL	32	100

according to their distribution, as mean and standard deviation or median and interquartile range, and compared by Student's t-test or Mann-Whitney U test as it may correspond. A p value of <0.05 in all analysis was considered statistically significant. All statistical analyses were made by the IBM SPSS Statistics 25 software.

Data capture and safety

An advanced system of electronic capture of data through the Internet was used, with https:// connection for a safe transfer of information. Data were encoded during the Internet transfer and saved in a database protected against unauthorized access.

RESULTS

Replies were obtained from 32 centers, with Santa Fe being the province that most contributed with 7 centers, followed by Córdoba with 4 centers and Buenos Aires with 3 centers *(Table 1)*. As the two first provinces represented 34% of the surveyed, they were analyzed jointly compared to the rest of the country, with no statistically differences being found in terms of use of TEE in the OR or in catheterization laboratories.

In regard to the interventions made, 3 centers replied that they do not conduct cardiothoracic and vascular surgeries (CTVS), and 3 centers that they do not conduct structural heart disease procedures (SHD). The median of CTVS performed was 70 (IQR 85) and of SHD procedures 19 (IQR 40).

As to TEE, 25 centers (87.1%) replied that they carry it out during SHD procedures. From those that do not do it, 4 do not consider it necessary, 2 do not have the appropriate equipment, and 1 does not have the required staff. In the field of CTVS, 22 centers perform it (68.8%). From those that do not do it, 4 do not consider it necessary, 3 do not have the appropriate equipment, and 3 do not have the required staff. In both cases, 7 centers are considering using TEE in the short term.

In Argentina, the preparation for the procedure presents a different reality from other parts of the world, as the person in charge of performing the TEE, both in the OR and the catheterization laboratory, is mainly a cardiologist *(Figures 1 and 2)*. A similar distribution is observed at the time of placing the probe, although with more participation of anesthesiologists (15.6% in SHD and 25% in CTVS).

When analyzing technical issues of TEE use, only half of centers have 3D mode; specific protocols are made in 64% of SHD and 72% of SHD. At the time of storing images, this is carried out in 96% of SHD and 95% of CTVS; while reports are made in 80 and 77% of tests for SHD and CTVS, respectively. In regard to complications, only one case of bleeding was recorded in the OR;

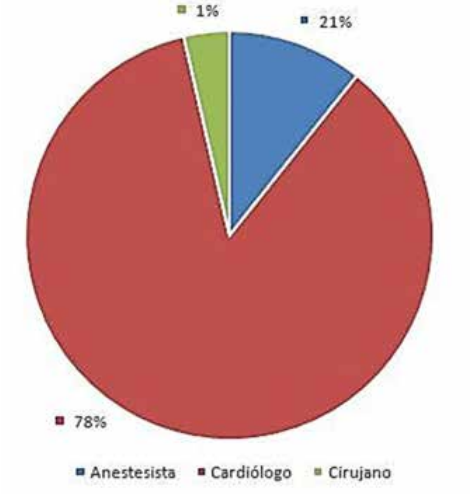

FIGURE 1.
Person in charge of performing TEE in the operation room

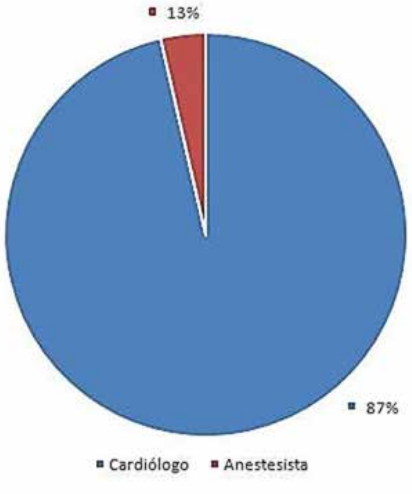

FIGURE 2.
Person in charge of performing TEE in the catheterization laboratory

while in the catheterization laboratory, 1 case of bleeding and 1 case of esophageal perforation.

Uses of TEE

In *Table 2*, details are given on uses of TEE in cardiovascular surgeries, reflecting its predominant use in mitral valvuloplasty and isolated cases of valve replacements, surgical correction of congenital heart diseases and aortic surgeries. As to the time to use it, 59% do it before, 33% during and 36% when the surgery ends, and 36% at the time of ending extracorporeal circulation; thus, only 15% of centers use TEE in all the surgical times (35% use it only at a time and 50% in two times). From all the surveyed, 63.6% reported that TEE modified surgical behavior only a few times (22.7% most of the times, 4.5% never).

In *Table 3*, details are given on the use of TEE in procedures of structural heart disease, where it is manifest that most of its use is in procedures to close the left atrium appendage and closure of periprosthetic leaks. Another relevant piece of information is that, in our country, centers that do not perform percutaneous mitral valve or tricuspid valve repairs, or pulmonary valve implants, are a majority. Remarkably, centers that do perform these practices do not use TEE in all procedures. As to the time of using it, 56% do it before, 20% during and 15% at the end of the procedure, making a total of 44% of centers that use TEE at all times of the procedure (28% at just one time). Only 12% of the surveyed reported that TEE always modified their management (4% never, 40% a few times, and 36% most of times).

During the survey, there were also questions about the use of TEE to estimate parameters other than those needed to guide CTVS or SHD procedures. Results are presented in *Table 4*. The parameters that are predominantly evaluated are left ventricular ejection fraction, valve function and the presence of intracavitary thrombi and/or aortic dissection. In regard to diastolic dysfunction, 71% of cases are evaluated a few times or never.

DISCUSSION

This registry constitutes the first national endeavor to characterize the use of transesophageal echocardiography (TEE) in an

TABLE 2.
Use of TEE in cardiovascular surgery.

	Mitral valvuloplasty (%)	Valve replacement (%)	MRS w/o ECC (%)	MRS w/ECC (%)	Combined surgeries (%)	Congenital heart disease surgeries (%)	Heart transplant (%)	Aortic surgery (%)
0%	4.5	22.7	59.1	59.1	18.2	22.7	13.6	31.8
1-24%	22.7	40.9	9.1	13.6	40.9	27.3	4.5	31.8
25-49%	9.1	9.1	4.5	4.5	4.5	9.1	0	18.2
50-74%	4.5	4.5	0	0	4.5	4.5	0	0
75-99%	4.5	4.5	0	0	4.5	4.5	0	0
100%	27.3	4.5	0	0	4.5	0	0	9.1
Not performed	22.7	13.6	27.2	22.7	22.7	31.8	81.8	9

MRS: myocardial revascularization surgery. **ECC:** extracorporeal circulation.

TABLE 3.
Use of TEE in structural heart disease procedures.

	Percutaneous aortic valve implantation (%)	Percutaneous mitral valve repair (%)	Percutaneous tricuspid valve repair (%)	LA appendage closure (%)	Peripros-thetic leak closure (%)	Percutaneous pulmonary valve implantation (%)	Septal ablation with alcohol	Congenital heart diseases repair
0%	(%)	4	0	0	0	0	12	0
1-24%	36	8	0	12	8	8	16	16
25-49%	4	4	0	0	4	4	0	20
50-74%	4	0	4	0	0	0	0	0
75-99%	8	4	0	8	4	0	0	8
100%	4	16	8	52	44	8	20	24
Not performed	16	64	88	28	40	80	52	32

intraoperative and SHD intraprocedural scenario in Argentina. The results show that, although most centers report using TEE in cardiovascular surgery (68.8%) and in SHD procedures (78.1%), there are still limitations linked to equipment availability, training of human resources and the existence of standardized protocols.

These findings are in agreement with what the American Society of Echocardiography has pointed out, in collaboration with the Society of Cardiovascular Anesthesiologists and the Society of Thoracic Surgeons, where the role of TEE is emphasized as an essential tool to make intraoperative decisions, with an impact on safety and surgical results.[1] Particularly, international evidence has shown that a systematic use of TEE allows optimizing the evaluation of valve repairs, guide the implantation of prosthesis and detect complications in real time; aspects that in this survey were reported as factors that modified behavior in a limited number of cases.

Likewise, the recent publication of the American Heart Association on the intraoperative use of TEE in heart surgery underscores the need to have specific protocols and interdisciplinary training, aspects that in Argentina appear as inadequate.[9] In this registry, only half of the centers reported having a standardized protocol, and a similar percentage of devices with 3D echocardiography, reflecting a gap in regard to international recommendations.

Another relevant finding is the predominance of cardiologists as the person in charge of the procedure, both in the OR and in catheterization laboratories, in contrast with the usual practice in the United States and Europe, where anesthesiologists have a more central role. Another aspect poses a deeper analysis, as it reflects the organization of health care teams in Argentina, while the importance of encouraging joint training and multidisciplinary work stands out.

Finally, the low incidence of complications reported in this registry agrees with the previous literature, in terms of describing TEE as a safe technique, although not free from risks when carried out without the appropriate experience. The heterogeneity observed between centers, both in the frequency of use and in the times of implementing it during procedures, highlights the need to promote training programs, national consensuses and institutional policies oriented toward guaranteeing the universal availability of this tool.

Jointly, the results obtained allow to place Argentina in line with a growing tendency in the adoption of TEE in cardiovascular practice, although they show the need to strengthen organizational, educational and technological aspects to reach international standards. This registry constitutes a valuable starting point for future multicenter studies and for the development of local guidelines adjusted to the national health care reality.

Limitations

The main limitation of this survey is the scant number of participating centers. Although the initial invitation was through direct phone contact with 60 professionals, later invitations were sent by e-mail to all FAC members, so the final percentage of responders in relation to the total number of invitations is unknown. Anyway, responses were obtained from 16 provinces (67%), so the survey is considered wide enough to represent the reality of the whole country in the socioeconomic spectrum.

Second, we do not have the information discriminated by center in regard to the number of each type of surgery or each type of structural heart disease procedure. It was decided not to add questions in this sense to prevent inaccurate information or that may turn the collection and subsequent replying tedious.

As the survey was distributed from a federation of cardiologists, there may be a bias as the replies were predominantly provided by this group of professionals, so the centers where TEE are being made by anesthesiologists may be inaccurately represented.

CONCLUSIONS

In this survey, TEE was used in 69% of CTVS and 78% of SHD procedures, mainly being carried out by echocardiography cardiologists, with indications similar to those described internationally, as well as a similar frequency of complications.

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TABLE 4
Parameters evaluated during use of TEE

	LVEF (%)	Contractility (%)	RVSF (%)	Dimensions (%)	Valve function (%)	Diastolic function (%)	PFO (%)	Thrombi (%)	RA Atheromatosis (%)	AO dissection (%)
Never	-	-	-	8,7	-	21,7	4,3	4,3	9,1	4,3
Sometimes	8,7	13,0	21,7	39,1	-	47,8	17,4	4,3	13,6	21,7
Frequently	26,1	39,1	39,1	17,4	34,8	13,0	30,4	21,7	22,7	8,7
Always	65,2	47,8	39,1	34,8	65,2	17,4	47,8	69,6	54,5	65,2

LVEF: left ventricular ejection fraction; **RVSF:** right ventricular systolic function; **PFO:** patent foramen ovale; **DA:** descending aorta; Ao: aortic.

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Original Investigation Reports

Prevalence of abdominal aortic aneurysm detected by ultrasound screening in individuals over 50 years of age: Argentine Multicenter Registry (SCREANRA)

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ABSTRACT

Introduction: Abdominal aortic aneurysms (AAA) are a vascular condition associated with high morbidity and mortality, particularly when rupture occurs. Their prevalence increases with age and is more common in individuals older than 65 years, although they may occur from the age of 50 in the presence of cardiovascular risk factors. Among the most relevant risk factors are smoking, hypertension, and atherosclerotic disease. Most patients with AAA remain asymptomatic for long periods, and diagnosis is therefore often incidental or occurs after complications develop. Early detection through ultrasound allows identification of aneurysms at earlier stages and facilitates appropriate clinical follow-up.

Objective: To evaluate the detection of abdominal aortic aneurysms through ultrasound screening in patients older than 50 years in Argentina. As a secondary objective, to analyze the feasibility of evaluating the abdominal aorta by cardiologists during echocardiographic studies.

Materials and Methods: During cardiovascular or abdominal ultrasound examinations, the anteroposterior and transverse diameters of the abdominal aorta were measured. Patients older than 50 years with risk factors for AAA were included. Public and private medical centers from different provinces of Argentina participated in the study.

Results: A total of 2148 patients were included: 1063 men and 1085 women. Seventy-seven abdominal aortic aneurysms were detected, corresponding to an overall prevalence of 3.58%. Most cases were observed in patients older than 65 years.

Conclusions: Ultrasound evaluation of the abdominal aorta during cardiovascular or vascular studies allows detection of aneurysms in patients with risk factors.

INTRODUCTION

Abdominal aorta aneurysm (AAA) is a potentially lethal vascular disease, characterized by progressive dilatation of the abdominal aorta, that may evolve into rupture if not detected and treated timely. Its prevalence increases significantly with age, being estimated in between 3% and 8% in men older than 65 years in western populations. Aneurysm rupture is one of the main causes of sudden death of vascular origin in the elderly, and is associated to mortality rates of more than 80%, turning this pathology into a relevant problem of public health.[1,2,3,4,5,6]

From an anatomical point of view, the abdominal aorta extends from the diaphragm to its bifurcation in the common iliac arteries, where it splits into significant visceral branches like the celiac trunk, the superior mesenteric artery, the inferior mesenteric artery and the renal arteries. The diameter of the abdominal aorta varies according to age, sex and body surface, with its proximal segment being larger than the distal one. It is considered normal when its maximum diameter is approximately 20 mm[3].

The abdominal aorta aneurysm is defined as a segmental dilatation that compromises the three layers of the arterial wall with a diameter equal or greater than 30 mm, measured in an anteroposterior or transversal sense, or when it exceeds by 50% the normal caliber or is 1.5 times greater than the adjacent segment. The diameters between 26 and 29 mm are considered subaneurysmal dilatations.[1]

From an epidemiological point of view, AAA constitutes a relevant cause of cardiovascular mortality. In the United States, it is the tenth cause of death in men older than 65 years, and it is estimated that in Argentina it affects more than 150,000 individuals, with more than 1000 yearly deaths associated to its rupture.[2]

Different population studies have shown that the prevalence of AAA increases progressively with age. In men, a prevalence of

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1.3% up to 54 years of age has been estimated, and of 3.9-7.2% in those older than 65 years, and 12.5% among those between 75 and 84 years; while in women older than 65 years, prevalence is between 1% and 1.3%.[4] Due to this epidemiological distribution, most screening programs have focused on populations older than 65 years.

The pathogenesis of AAA is multifactorial and is mainly associated with degenerative processes of the vascular wall and atherosclerotic phenomena. Between the most important risk factors, the following stand out: advanced age, male gender, smoking, hypertension, dyslipidemia, peripheral artery disease and family history of aneurysmal disease.[4]

AAAs usually remain asymptomatic for long periods, and are frequently diagnosed incidentally during imaging studies made due to other clinical reasons. In approximately 85% of cases, no symptoms are present until the time of rupture, a situation that is associated to an estimated mortality between 80% and 90% in patients that reach the hospital.[5] Due to this silent evolution, and the risk of catastrophic complications, AAAs have been frequently called "silent killers".[3]

Aneurysms can develop in any segment of the aorta, although the most frequent location is the infrarenal region. Approximately, 85% of AAAs are located below the renal arteries, while around 5% compromise the suprarenal aorta. From a morphological point of view, they may present as a fusiform, saccular or eccentric aneurysm, with the latter presenting greater risk of rupture. Likewise, the more proximal the location of the aneurysm, the greater the complexity for a surgical or endovascular repair.[6]

Between 25% and 40% of patients with AAA present associated peripheral aneurysms, mainly in the popliteal artery (70%) or in the iliofemoral territory (20%). A popliteal aneurysm can be bilateral in up to 50% of cases, and their main complication is distal embolization, which may cause critical ischemia of lower limbs and risk of amputation.[7] Furthermore, approximately two thirds of patients with AAA present concomitant CAD.

Early diagnosis and surgical or endovascular repair of the larger aneurysms (generally from 55 mm of diameter) allow to significantly improve prognosis. In this context, echocardiographic screening has proven to be an efficient tool for an early detection of AAA. Different international studies, including the Multicentre Aneurysm Screening Study (MASS), the Western Australia Screening Study, the Viborg Trial, the UK Small Aneurysm Trial and the ADAM Trial, have shown that ultrasound tracking significantly reduces the mortality associated to aneurysmal rupture in risk populations.[8,9,10,11,12,13,14,15,16,17,18,19,20,21,22,23,24,25,26]

In spite of the strong evidence backing the screening strategies, epidemiological data on the prevalence of AAA are still limited in many Latin American countries. In Argentina, the information available is scant and mainly comes from international registries. In this context, this study had the goal of evaluating AAA detection by echocardiographic screening in individuals older than 50 years in Argentina. As secondary goals, we sought to estimate the prevalence of this pathology in the studied population, and to analyze the feasibility of the evaluation of the abdominal aorta by cardiologists during echocardiographic studies.

MATERIALS AND METHODS

General characteristics and type of study. Design

A screening campaign on abdominal aorta was carried out during April 2023, in multiple centers of Argentina. It developed in a context of performing other auxiliary ultrasound imaging tests, such as color Doppler ultrasound, Doppler echocardiography of neck vessels, arterial Doppler echocardiography of lower limbs, aortoiliac, renal or abdominal Doppler echocardiography, from which the measurements of the maximum anteroposterior and transverse diameters of the abdominal aorta were obtained.

It was a multicenter, observational, transversal and descriptive registry of a screening program. Data were collected on the prevalence of AAA and on the distribution of associated risk factors, diagnosed by ultrasound according to the following criteria: segmental dilatation of abdominal aorta with a diameter equal or greater than 30 mm, or exceeding 50% of its normal caliber.

Echocardiographic screening was indicated in the whole population older than 50 years, in both genders. The main risk factors for the development of AAA were considered, such as current smoking or history of smoking, hypertension, family history of aneurysm, aneurysms in other locations, subclinical atherosclerosis and clinical atherosclerosis, including acute myocardial infarction, intermittent claudication or stroke.

Public and private medical centers from the different provinces of Argentina participated.

Inclusion criteria

Patients older than 50 years were included, of both sexes, with risk factors for AAA like history of current or past smoking, hypertension, family history of aneurysm, CAD (including history of AMI, coronary angioplasty or myocardial revascularization surgery), peripheral vascular disease, atherosclerosis, aneurysm in other locations, bypass or peripheral angioplasty, symptomatic arterial disease, pain in limbs or intermittent claudication, and cerebrovascular disease, including stroke or carotid disease.

Exclusion criteria

Patient with any clinical situation that would prevent them from going to the center, those who rejected participating in the study, those who presented history of previous AAA surgical or endovascular intervention and those who did not meet the requirements established in the inclusion criteria were excluded.

Enrollment and ending periods

Enrollment of patients was made at the time of their coming to the assigned public or private center, to undergo color Doppler ultrasound, Doppler ultrasound of the neck vessels, arterial or venous Doppler ultrasound of lower limbs, aortoiliac, renal or abdominal Doppler echogram.

The enrollment and ending periods were conducted between April 17, 2023, and April 30, 2023. The seven regions of the Argentine Federation of Cardiology (FAC) participated: Northwest, Northeast, Buenos Aires, Patagonia, Litoral, Cuyo and Center, along with its respective societies and associations.

The study had physicians with credited training and experience in echocardiography, vascular Doppler echocardiography or

abdominal ultrasound, using the equipment available in each center. Sectorial, convex or linear probes were used, from different brands and models according to local availability. The cardiologists acquire skills to explore the abdominal aorta when getting familiar with the subxiphoid views in echocardiography, particularly when defining severity of aortic valve failure by evaluation of reverse flow at that level, or when seeking flow alterations compatible with aortic coarctation.[9]

ECHOCARDIOGRAPHIC EXPLORATION

Significance of ultrasound

Echography or ultrasound (US), in its different modalities (2D, color and Doppler) allows to evaluate abdominal structures and to detect anomalies. This is a highly sensitive and specific imaging test (Se 93.3%, Sp 98.5%) to detect AAA, besides being cost-effective, affordable and applicable in different levels of care. It is a feasible, repeatable and safe tool; free from radiation and harmless, as it does not require nephrotoxic contrast.[10] It is also useful for follow-up and surveillance of aneurysms.

US is the most used method for screening and diagnosis in asymptomatic patients, in ER units with no previous diagnosis and in symptomatic patients. In a scenario of AAA rupture, it is the tool that less delays diagnosis.

Exploration technique

All ultrasound studies were made by physicians trained and with previous experience in cardiovascular ultrasound, vascular Doppler or abdominal echocardiography, which guaranteed a standardized acquisition and accurate measurements of the aorta diameters. The studies were carried out using the ultrasound devices available in each participating center.

Cardiological transducers were used, with average frequencies of 2.5 MHz and second harmonic imaging, and linear transducers with frequencies between 7 and 12-18 MHz.

A transversal sweep was made with the transducer in subcostal position, from the epigastrium in a caudal direction, followed by longitudinal exploration from the diaphragm until the aortoiliac bifurcation, and the first centimeters of the common iliac arteries.

The measurements were made in a transversal view, with the transducer beam perpendicular to the major axis of the abdominal aorta. The greatest diameters were registered, both anteroposterior (AP) and transversal (T), measuring from adventitia to adventitia during systole. The measurement of the anteroposterior diameter is considered more accurate than that of transverse diameter, due to the perpendicularity of the echography beam.

When it was not possible to obtain a circular section of the aorta (due to dilatation or tortuosity), the mean diameter was estimated for the ellipse, or the diameter was measured in an adequate longitudinal view, as long as it was perpendicular to the aortic axis.[11]

When aneurysmal dilatation was identified, both the maximal anteroposterior diameter and the maximal transversal diameter were recorded. Normal aorta was defined as a diameter ≤25 mm, ectasia between 26 and 29 mm, and aneurysm when it was ≥30 mm.

Patients with aneurysms of up to 4 mm were informed about the pathology and they were encouraged to undergo echocardiographic monitoring in their medical centers, given the low yearly probability of rupture. In the cases with aneurysms in a surgical range (55 mm in men, 50 mm in women, family history or saccular aneurysms), referral to the vascular surgery services of reference of each center was indicated.

Statistical analysis

A Coordinating Scientific Committee (CSC) was constituted for the registry, made up by members of the Committee on Peripheral Vascular Disease and Stroke of FAC, in charge of leading its development in all its aspects, with the support of the Data Control Group and the Group of Publications. In the statistical analysis, categorical variables were expressed as percentages and were compared by the Chi-squared test of Pearson. Continuous variables were presented as mean and standard deviation, being compared by Student's t-test.

To fulfill the enrollment and data loading, an online form was designed, where personal data, risk factors, center data, type of base study and results of measurements were recorded. Google Forms was used as software support to collect data, only accessible for the researchers in charge and the coordinators appointed for this goal.

The Center for Medical Teleinformatics of FAC (CETIFAC) acted as the only data center, managing the database, its maintenance, and control of load from the different centers. Data was encoded during internet transference and stored in a base protected against unauthorized access.

The study was made according to the ethical principles established in the Declaration of Helsinki for research in human beings. All participants were informed about the goals of the study and granted their informed consent before being included in the registry. The data of patients were anonymized and treated confidentially according to the current regulations for data protection.

Each patient obtained the corresponding consent, according to institutional policies and the current privacy regulations. The purpose of the study was explained in detail, namely evaluating the anatomy of abdominal aorta and detecting alterations such as dilatations, aneurysms, thrombosis and other pathologies. Likewise, it was clarified that the procedure was made as a supplement of other requested studies and that it would have an approximate duration of 20 minutes, with no complications or adverse effects expected.

Patients were given the chance to ask all the questions they would deem necessary for them to be at ease and understand the procedure. Participation was voluntary, with a chance of withdrawing their consent at any time, not affecting the medical care they would receive. The patients already incorporated to the registry would remain in it. The regulations also contemplated a voluntary withdrawal or not, from an investigator or a center.

RESULTS

Characteristics of the population

During the period of the study, 2252 patients were evaluated, from whom 14 were excluded as they did not meet the age cri-

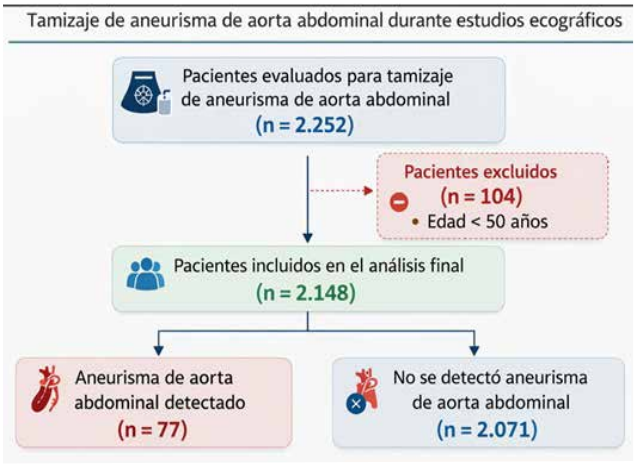

FIGURE 1.
Flowchart of the selection of patients and detection of abdominal aortic aneurysm in the study population

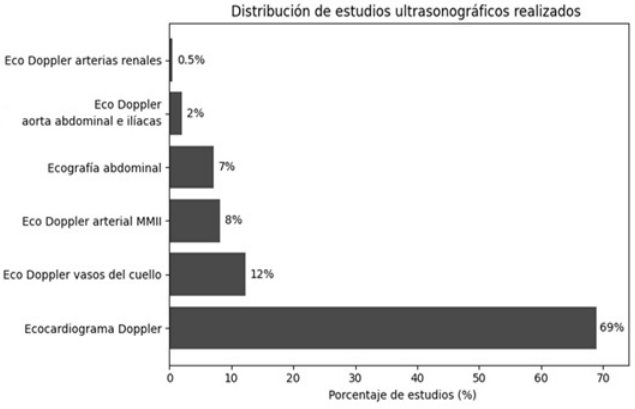

FIGURE 2.
Type of study made that allowed detecting AAA

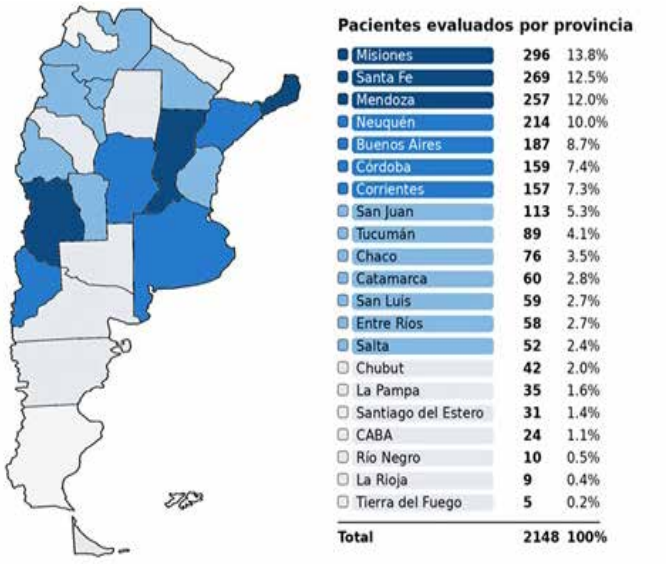

FIGURE 3.
Geographical distribution of the study.
Patients evaluated by abdominal aortic aneurysm (AAA) in Argentina. The political map shows the provincial distribution of patients included in the multicenter study. The intensity of color reflects the number of patients evaluated in each province. Total of evaluated patients: n = 2148.

TABLE 1.
Characteristics of the population

Variable	n	%
Total de pacientes evaluados	2148	100
Hombres	1063	49
Mujeres	1085	51
Aneurisma de aorta abdominal detectado (AAA)	77	3.58

TABLE 2.
Prevalence of abdominal aortic aneurysm according to sex

Grupo	n	Prevalencia (%)
Hombres	51	4,80
Mujeres	26	2,40
Total	77	3,58

teria. The final population analyzed included 2148 patients. The flowchart of the study is shown in *Figure 1*.

The average age of the population was 67±9.12 years in men. From the total of participants, 1063 patients were males (49%) and 1085 females (51%), showing a relatively balanced distribution between both genders *(Table 1)*.

Modality of the study and geographical distribution

The study that most frequently allowed to perform the abdominal aorta screening was color Doppler echocardiogram, made in 1489 patients (69.3%). Other studies that made the evaluation of the abdominal aorta possible included Doppler echography of neck vessels, arterial Doppler echography of lower limbs and abdominal echography *(Figure 2)*.

The geographical distribution of patients included multiple provinces of Argentina. The provinces that contributed the greater number of registries were Misiones (296 patients; 13.78%), Santa Fe (269; 12.52%) and Mendoza (257; 11.96%). Neuquén (214; 9.96%), Buenos Aires (187; 8.71%), Córdoba (159; 7.40%) and Corrientes (157; 7.31%) also had a relevant participation, reflecting a wide territorial representation of the registry *(Figure 3)*.

Cardiovascular risk factors

Hypertension was the most frequent risk factor, followed by dyslipidemia and history of smoking, with predominance of former smokers over active smokers. Other risk factors include diabetes mellitus, obesity and history of cardiovascular disease and peripheral artery disease, configuring a global profile of high atherosclerotic risk in the analyzed population.

Prevalence of abdominal aortic aneurysm

In the total population analyzed, 77 AAA were detected, representing a global prevalence of 3.58% (95% CI: 2.8-4.5) *(Table 2)*.

TABLE 3.
Comparative analysis of abdominal aortic aneurysm according to sex and age

Variable	Casos AAA n (%)	Sin AAA n (%)	Medida	IC95%	p
Sexo					
Hombres	51 (4,8)	1012 (95,2)	OR 2,05	1,25-3,35	0,004
Mujeres	26 (2,4)	1059 (97,6)	Referencia	-	-
Edad					
>65 años	59 (76,6)	-	RR 3,27	1,90-5,63	<0,001
50-64 años	18 (23,4)	-	Referencia	-	-

AAA: abdominal aortic aneurysm . **OR:** odds ratio . **RR:** relative risk. **95% CI:** 95% confidence interval

TABLE 4.
Multivariate analysis by logistic regression

Variable	OR ajustado	IC95%	P
Sexo masculino	2,01	1,22-3,30	0,006
Edad >65 años	3,18	1,84-5,49	<0,001
Tabaquismo	1,72	1,05-2,80	0,031
HTA	1,34	0,82-2,19	0,24
Dislipidemia	1,21	0,74-1,97	0,43

Comparative analysis by gender

Fifty-one cases were identified in men (4.8) and 26 cases in women (2.4%). Male gender was significantly associated with a greater risk of AAA (OR: 2.05; 95% CI: 1.25-3.35; p = 0.004), indicating approximately twice the chance of disease in comparison to women.

Comparative analysis according to age

A significant increase in the prevalence of AAA was observed with age. From the total of cases detected, 59 aneurysms (76.6%) were identified in patients older than 65 years, while 18 cases (23.4%) corresponded to the group from 50 to 64 years.

In the group of patients older than 65 years, 38 aneurysms were identified in men and 21 in women; while in the age group from 50 to 64 years 13 aneurysms were recorded in men and 5 in women *(Figure 4)*.

Patients older than 65 years presented a significantly greater risk of AAA (RR: 3.27; 95% CI: 1.90-5.63; p<0.001) *(Table 3)*.

Analysis of associated factors

The analysis of association showed that the male gender, age of more than 65 years and smoking were significantly associated with the presence of AAA. Together, these findings show a clear association between advanced age, male gender and cardiovascular risk factors with a higher prevalence of AAA in the studied population.

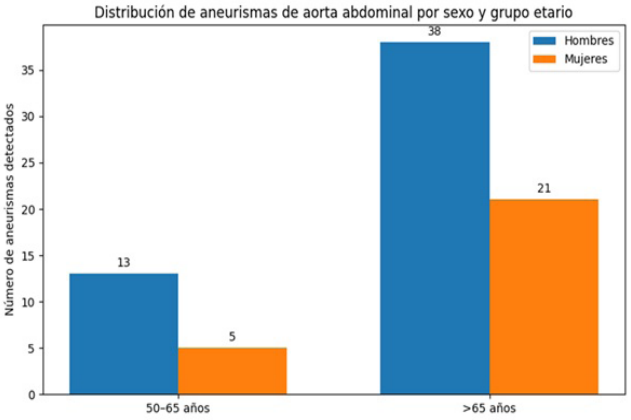

FIGURE 4.
Percentage distribution of abdominal aortic aneurysms according to sex and age group in the studied population
A higher frequency of cases is observed in patients older than 65 years and the male gender was predominant.

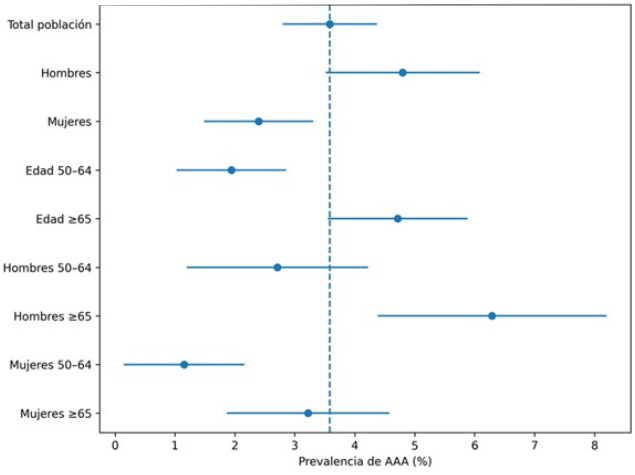

FIGURE 5.
Factors associated to AAA

Multivariate analysis

A multivariate logistic regression analysis was made to identify the factors associated independently with the presence of AAA. The variables included in the model were age, sex and relevant cardiovascular risk factors.

Multivariate analysis showed that the male gender and an age of more than 65 years were independently associated with the presence of AAA. Likewise, smoking remained as a significant risk factor in the adjusted model, while hypertension and dyslipidemia did not show a statistically significant association after adjustment by confusion variables *(Table 4)*.

Forest plot shows the prevalence of AAA in the total population and in different subgroups according to gender and age, with their respective 95% confidence intervals. Prevalence was higher in men than in women, and significantly increased in patients older than 65 years *(Figure 5)*.

DISCUSSION

Abdominal aortic aneurysm screening by Doppler echography has consolidated as an efficient strategy for early detection and reduction in the mortality associated to its rupture, particularly in risk populations like smokers older than 65 years.[12] Use of ultrasound allows to identify aneurysms in asymptomatic phases, facilitating its clinical follow-up and the implementation of timely therapeutic interventions.

Different clinical trials have supported the implementation of population screening programs. Among the most relevant, we find the MASS, Viborg, Western Australia and Chichester studies, which showed that echographic tracking significantly reduces mortality specifically due to AAA, mainly by decreasing the incidence of aneurysmal rupture.[12,15,16,17,18] The MASS study showed a significant reduction of specific mortality and rupture rate; while the Western Australia Screening Study confirmed these benefits in a long-term follow-up.[12,13]

Aneurysmal diameter constitutes the main rupture predictor, with a risk that increases progressively and exceeds 50% in aneurysms of more than 70 mm.[13] The goal of screening is to identify aneurysms with a clinically relevant diameter before the appearance of complications, allowing elective repair and reducing associated mortality.[16] However, there are differences between clinical guidelines in regard to the populations that should undergo screening, recommending in general doing it in men older than 65 years with history of smoking.[17]

Similarly, the UK Small Aneurysm Trial and ADAM studies established that echographic surveillance constitutes a safe strategy in smaller aneurysms, contributing to defining the current criteria of follow-up and intervention.[14,25,26]

In this multicenter registry, a prevalence of AAA of 3.58% was observed in adults older than 50 years, a figure comparable to that reported in international studies. Most cases were detected in patients older than 65 years, confirming the strong association between age and the development of the disease. Likewise, male gender and smoking were identified as independent risk factors, in agreement with the epidemiological evidence available.

In scenarios where there are no national screening programs, as it currently happens in Argentina, timely detection during imaging studies represents a potentially cost-effective strategy. In this regard, the evaluation of the abdominal aorta during transthoracic echocardiography acquires special relevance, as many of these patients present cardiovascular risk profiles similar to those associated with AAA.[19,20]

Although the prevalence of aneurysm is smaller in women, this group presents particular clinical characteristics, like risk of rupture and presentation with smaller diameters.[22,23] In specific populations, like elderly women or patients in high risk, screening could be considered individually.[17,21,24]

The screening programs have proven to significantly reduce specific mortality related to AAA, although this benefit does not always translate to a decrease in overall mortality in the long-term.[18] This situation has generated a debate on the universal implementation of screening programs, particularly in health care systems with no organized programs.

From a clinical point of view, the systematic incorporation of echographic evaluation of the AAA during cardiovascular ultrasound tests could facilitate detection in asymptomatic stages, optimize risk stratification and allow a timely referral for treatment. This strategy can contribute to reduce morbimortality associated to AAA rupture in the usual clinical practice.

In this regard, the risk of rupture of AAA is directly related with its maximal diameter, main rupture and mortality predictor, and a determinant factor for surgical indication. Other associated factors include smoking, the female gender, hypertension, family history, saccular morphology and decrease in FEV1. Rupture presents a mortality of more than 80% in patients who reach hospital care.

Elective repair presents significantly less mortality in comparison with urgency surgery, with rates between 0.73% and 5%, while in broken aneurysms it may reach 50-60% in the hospital environment, and up to 100% with no intervention.[27] In this scenario, early detection and timely treatment constitute the mainstay of clinical management.

Intervention in aneurysms with diameters ≥55 mm in men and ≥45 mm in women, as well as those with growth >0.5 cm/year or associated symptomatology.[28] Echography represents a safe, accurate, affordable and cost-effective diagnostic method for the detection of aneurysms in asymptomatic patients.

Within this framework, echographic screening of the abdominal aorta in a selected population allows an early detection of aneurysms and other aortic pathologies, favoring the implementation of timely therapeutic managements and improving morbimortality.[29]

However, the results of this study should be interpreted considering certain limitations. First, this is an observational registry of cross-sectional character, which prevents establishing causal relationships between the risk factors evaluated and the presence of AAA. Second, enrollment included patients who went to health care centers to undergo ultrasound tests because of different clinical indications, which may introduce a selection bias, given that this population probably presents a greater load of cardiovascular risk factors in comparison with the general population. Similarly, the tests were made in multiple centers with ultrasound devices with different features, which may generate a certain variability in measurements; however, all studies were made by physicians with experience in ultrasound, which contributes to reducing this effect. On the other hand, the enrollment period was limited to two weeks, which prevented having a longitudinal follow-up and evaluating the progression of the aneurysms detected or its clinical results in the long term. Finally, although the results match international evidence, the differences observed between studies may be attributed to demographic, epidemiological and methodological variations, including the age of inclusion, selection criteria and the duration of the screening programs.

CONCLUSIONS

This Argentine multicenter registry showed a prevalence of AAA of 3.58% in a population older than 50 years with cardiovascular risk factors. Most cases were detected in patients older than 65 years (76.6%), with predominance of the male gender in both age groups.

The risk factors most frequently associated were hypertension, dyslipidemia and history of smoking. The echographic evaluation of the abdominal aorta during cardiovascular or vascular ultrasound tests proved to be feasible, useful and applicable for a timely detection of aneurysms, both in asymptomatic and symptomatic patients.

These findings back the incorporation of the AAA evaluation to supplement the usual echographic tests, even in the field of primary care by trained physicians. This strategy could improve the early detection of AAA by a simple, affordable and cost-effective method.

Although these results do not modify the current recommendations that prioritize screening in people older than 65 years, they do suggest that a directed evaluation in patients older than 50 years with risk factors could constitute a valuable supplementary strategy in clinical practice.

Participants in AAA screening

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Original Investigation Reports

Chain Mediation of the Aortic Path in the Relationship between Body Mass Index and Abdominal Aortic Diameter.

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ABSTRACT

Introduction: the aorta, sensitive to excess weight, can undergo changes associated with cardiovascular risk. Studying its segments will allow a better understanding of the relationship between BMI and distal structural alterations.

Objective: to analyze the relationship between BMI and abdominal aortic diameter, considering the thoracoabdominal aortic segments as mediators.

Materials and methods: a cross-sectional study based on secondary data from 801 adults evaluated by thoracoabdominal computed tomography. The independent variable was BMI. The dependent variable was abdominal aortic diameter at the origin of the celiac trunk. The potential mediating effect of aortic diameters was evaluated using a six-segment chain mediation model: aortic sinus of Valsalva, sinotubular junction, mid-ascending aorta, mid-aortic arch, mid-descending aorta, and aorta at the level of the diaphragm. A directed acyclic graph was developed to support the causal structure.

Results: BMI was not directly associated with abdominal aortic diameter but was associated with the aortic sinus of Valsalva (B=14.159; p<0.001) and the sinotubular junction (B=0.4686; p=0.0036). There was no relationship with the middle ascending aorta (B=-0.0715; p=0.757). A positive effect was observed on the middle descending aorta (B=0.4837; p<0.001) and the aorta at the diaphragm (B=0.6110; p<0.001), both of which were directly associated with abdominal aortic diameter (B=0.1570 and B=0.6113, respectively; p<0.001). These findings suggest a structural sequence where distal thoracic diameters progressively influence abdominal aortic caliber. Three significant indirect pathways were identified: BMI → sinus of Valsalva→ mid-descending aorta → diaphragmatic aorta → abdominal aorta (effect = 0.1807; 95% CI [0.0878–0.2778]), BMI → mid-ascending aorta → mid-aortic arch → mid-descending aorta → abdominal aorta (effect = 0.0611), and BMI → mid-descending aorta → abdominal aorta (effect = 0.0759; 95% CI [0.0306–0.1343]).

Conclusions: the findings suggest that BMI may indirectly influence abdominal aortic diameter through interconnected aortic segments. This structural pattern could have clinical implications for the early detection of vascular abnormalities associated with excess weight.

INTRODUCTION

Body mass index (BMI) is an anthropometric measure widely used to estimate nutritional state in population and clinical contexts.[1] Its simplicity has favored its adoption as a standard criterion to classify weight excess, which has allowed to establish associations consistent with multiple outcomes, among them cardiovascular, metabolic diseases and premature death.[2] However, BMI presents significant limitations: it does not differentiate fat mass from lean mass, nor does it allow to assess fat tissue distribution.[3] These limitations become relevant when we seek to understand how body weight influences the structure and function of organs and blood vessels.

The aorta, as the largest artery of the body and the main pathway to transport oxygenated blood from the heart to the rest of the body, is a structure highly sensitive to hemodynamic and metabolic changes induced by body weight excess.[4,5] It is known that aortic dimensions can change in response to factors such as hypertension, low-grade chronic inflammation and mechanical stress.[6] These alterations may lead to progressive dilatations that increase the risk of aneurysms, dissections and other major cardiovascular events.[6] For this reason, the morphological evaluation of the aorta in individuals with a different anthropometric profile has important clinical implications.

In spite of the growing interest to understand the relationship between BMI and aortic structure, a large part of the evidence available has focused on isolated segments, such as the ascending or the abdominal aorta.[7,8] This fragmented approach prevents characterizing comprehensively the possible remodeling patterns along the aortic trajectory. Likewise, although the morphological changes of the aorta can be influenced by multiple

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factors, in this study we propose the hypothesis that BMI, as a clinical marker of the cardiometabolic state, could be associated to the distal aortic morphology through its effect on the proximal segments. Although BMI does not differentiate between fat mass and lean mass, it has been associated consistently with metabolic alterations like resistance to insulin, low grade chronic inflammation, endothelial dysfunction and arterial rigidity, all of them factors that can contribute to vascular remodeling. Particularly, it is proposed to explore whether the alterations in these segments could mediate the relationship between BMI and abdominal aorta diameter, where the clinically relevant dilatations are most frequently located.[9]

In this context, the need to explore the relationship between BMI and the dimensions of the aorta are justified, considering its segments from the sinus of Valsalva. Analyzing the mediating role of the different aortic sections would allow to clarify anatomical and functional mechanisms, by which body weight may influence on distal vascular morphology.

This approach is particularly pertinent within the framework of cardiovascular prevention, given that early detection of structural alterations may contribute to a better risk stratification.

For this reason, the goal of this study was to analyze the relationship between BMI and the diameter of the abdominal aorta, considering as possible mediator variables the dimensions of the different segments of the aorta along its thoracoabdominal trajectory.

MATERIALS AND METHODS

Design and population

A cross-sectional and analytical study was carried out, using a secondary database obtained from the Harvard Dataverse repository. The database included adults with and without comorbidities, evaluated between March 2018 and October 2019, by chest CT without contrast. This technique, although it does not allow to make a thorough functional or tissue characterization, offers a reliable measurement of the aortic diameters, particularly in standardized axial views, being useful for the morphological analysis of the aorta. The sample consisted of 801 adults older than 18 years, all undergoing chest CT. According to the authors of the original study, individuals with significant arterial anomalies or diseases severely affecting the structure of the arteries were not considered in the database, so individuals with history of revascularization or aortic valve replacement, stent placement, genetic syndromes related to aortic diseases (like Marfan or Turner), relevant congenital malformations, inflammatory diseases affecting the aorta, acute aortic syndromes, aneurysms, hypovolemia or severe hemodynamic instability, advanced heart failure with reduced ejection fraction and patients in hemodialysis were all ruled out. All the available data in the database were used for the analysis, not applying sampling or randomization methods.

Variables and measurements

Different variables were analyzed with the aim of understanding the relationship between BMI and the diameter of the abdominal aorta, mediated by different anatomical sections of the aorta. BMI was used as an independent variable, categorized in two groups: 0 (with no overweight or obesity) and 1 (with overweight or obesity), with the measure unit kg/m^2.

BMI was classified in two levels; without overweight or obesity (BMI $<$25 kg/m2) and with overweight or obesity (BMI ≥25 kg/m2). The cutoff point of BMI ≥25 kg/m2 was used following the classification established by the World Health Organization, which defines overweight as a BMI between 25 and 29.9 kg/m2 and obesity as BMI ≥30 kg/m2. This categorization allows to identify individuals with increased risk of cardiometabolic alterations and cardiovascular diseases. Although this threshold spans a wide range of conditions, it is justified in this context due to its clinical and epidemiological applicability, besides facilitating a comparative analysis with previous studies using the same classification.

The dependent variable was the diameter of the abdominal aorta, measured in millimeters (mm) by chest CT with no contrast. Besides, several anatomical sections of the aorta were considered as mediators in the relationship between BMI and the abdominal aorta diameter. These regions were sequentially modeled according to their anatomical arrangement along the aorta. The sections considered as mediators were aortic sinus of Valsalva, sinotubular junction, mid-ascending aorta, middle aortic arch, mid-descending aorta and the aorta in the diaphragm. All these sections were measured in millimeters (mm) and their role in mediation was evaluated through regression models. Likewise, as the chain mediation model of the PROCESS procedure of Hayes only admits a maximum of six mediators, the proximal aortic arch and the proximal descending thoracic aorta were excluded. This decision was based in its lower level of statistical significance in preliminary analyses. Although the exclusion of these segments could limit a full characterization of the aortic trajectory, the inclusion of those with greater contribution to the model were prioritized, so as to optimize the explicative capacity of the analysis and to fulfill the methodological requirements of the procedure.

According to the authors of the database, the aortic diameters were obtained by an automated system based on artificial intelligence (AI) for the segmentation of organs in computerized tomography (CT) imaging. The CT images were acquired using high resolution scanners (Siemens Somatom Force, Toshiba Aquilion ONE and GE CT750 HD), with matrices of 512x512 pixels and slice thickness between 0.625 and 1.5 mm. Later, the images were automatically processed by the AI-Rad Companion (Chest CT) prototype of Siemens-Healthineers, that uses deep algorithm algorithms to detect the central line of the aorta and to make a partition into nine specific anatomical regions, according to the 2010 AHA clinical guidelines. Mean diameters were estimated in a standardized manner as the root square of the product between the maximum and minimum diameters in each transverse plane. This methodology guarantees a high reproducibility in measurements, minimizes human error and allows to work with error margins within the acceptable standards in images of chest CT.[10] These measurements were treated as continuous variables for the serial or sequential mediation model.

Statistical analysis

To evaluate the indirect effect of BMI, categorized in two levels (0 = no overweight or obesity, 1 = with overweight or

obesity), on the diameter of the abdominal aorta through multiple anatomical aortic mediators, a chain mediation analysis was carried out using the model 6 of the PROCESS Macro for SPSS. The mediator variables included the aortic sinus of Valsalva, the sinotubular junction, the mid-ascending aorta, the middle aortic arch, the mid-descending aorta and the aorta in the diaphragm, sequentially modeled according to their anatomical arrangement.

The model 6 of mediation of Hayes is an approach used to examine how an independent variable (in this case BMI) can influence a dependent variable (abdominal aorta diameter) through a sequence of interrelated mediators. This model is known because it allows to evaluate the indirect effects through a chain of mediator variables.[11] In this study, model 6 was applied to analyze the chain relationship between BMI and the abdominal aorta, and considering how the different anatomical regions of the aorta mediate this relation, the model assumes that BMI influences the proximal sections of the aorta, which in turn, progressively affect the distal segments, ending in the abdominal aorta. This assumption is based on the structural and hemodynamic continuity of the aorta as the ground for the vascular alterations induced by weight excess being able to spread along the aortic axis.

The mediation analysis was made by linear regression models for each mediator and the dependent variable. The indirect effects were estimated by bootstrap resampling with 5000 samples and the generation of 95% confidence intervals. The effects were considered statistically significant if the confidence intervals did not include the null value. The significance threshold was set in $p < 0.05$. The IBM SPSS Statistics 25 supplemented with the PROCESS Macro (version 5.0 of Andrew F. Hayes) were used for data processing.

Finally, a directed acyclic graph (DAG) was built using the Dagitty library (www.dagitty.net) to represent the causal relationships between the variables of the study. This diagram was useful to identify the causal paths and verify the relationships between BMI, the anatomical aortic mediators and the abdominal aorta. From the DAG, it was possible to clearly visualize the possible confounding variables and the structure of the mediation model was theoretically supported.

Ethical considerations

The information used in this study comes from the open access repository Harvard Dataverse, and is found under the Creative Commons Zero (CC0) license, which allows using it without restrictions. As this is a set of completely anonymous secondary data, it was not necessary to obtain additional ethical approvals or specific permissions. The confidentiality of the data was ensured according to the ethical principles established in the Declaration of Helsinki.

The set of data is available at:
https://dataverse.harvard.edu/dataset.xhtml?persistentId=doi:10.7910/DVN/C0YY9I

For specific details, it is possible to check the following link:
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RESULTS

In the descriptive analysis of clinical variables, a wide variability was observed in the diameters of the different sections of the aorta, from the aortic sinus of Valsalva up to the abdominal aorta, reflecting the morphological diversity of the analyzed sample. The average measures of the different aortic regions showed a progressive decrease of the diameter from the ascending aorta until the descending and abdominal aorta, matching the normal physiology of the aortic tree. In regard to BMI, a little more than half of the population presented normal ranges, while close to 44% corresponded to people with overweight or obesity *(Table 1)*.

The table summarizes the direct and indirect effects of BMI on the diameter of the abdominal aorta in a model of chain mediation, considering six sequential anatomical mediators, corresponding to the thoracoabdominal aortic segments. It is observed that the direct effect of BMI on the abdominal aorta is not statistically significant ($p = 0.7060$), suggesting the absence of a direct relationship between both variables once mediators are considered. Nonetheless, the total indirect effect is statistically significant (bootstrapped 95% confidence interval: 0.4790-1.0725), indicating a significant mediation through the anatomical structures of the aorta. These findings highlight the relevance of the anatomical mediators of the aortic segments in the association between BMI and the abdominal aorta diameter, supporting the hypothesis of an indirect influence of BMI mediated by structural changes along the aortic axis *(Table 2)*.

Regression models were analyzed, relating BMI with the abdominal aorta diameter, through the different anatomical regions of the aorta as mediator variables. The results indicate that

TABLE 1.
Descriptive statistics and frequency of aortic segments and BMI

Variable (unit)	Minimum	Maximum	Mean	SD
Variable (unit)	21.4	51.1	35.23	42.53
Aortic sinus of Valsalva (mm)	23	51.8	32.98	40.45
Sinotubular juncture (mm)	23.5	53.6	37.12	47.31
Mid-ascending aorta (mm)	22	44.7	33.15	37.08
Proximal aortic arch (mm)	19.3	39.4	30.31	30.83
Middle aortic arch (mm)	19.5	40.6	28.6	32.77
Descending thoracic aorta (mm)	15.9	35.5	25.64	31.94
Mid-descending aorta (mm)	15.3	33.5	25.25	25.95
Aorta in the diaphragm (mm)	13	31.7	23.85	25.63
Abdominal aorta (mm)				
Variable	Category	Frequency	Percentage	
BMI	Normal	449	56.1	
	Elevated	352	43.9	

TABLE 2.
Mediation analysis of BMI on the abdominal aorta: direct and indirect effects

Type of effect	Coefficient	p-value	Bootstrapped confidence interval (CI)
Direct effect (BMI → abdominal aorta)	-0,0381	0,706	—
Total indirect effect (via mediators)	0,7765	—	[0,4790, 1,0725]

overweight or obesity are significantly associated to a greater size in the aortic sinus of Valsalva and in the sinotubular junction, suggesting a first impact of BMI on the initial sections of the aorta. Although the mid-ascending aorta did not show a direct association with BMI, it was indeed influenced by the previous sections, highlighting their role as mediators in transmitting the effect. Instead, in the mid-descending aorta, a significant direct relationship was observed with BMI, besides the accumulated effect of the ascending and arch sections, showing a structural continuity in the expansion of the aortic diameter. In the region of the diaphragm, BMI did not have a direct effect, but it did have an effect on the mid-descending aorta, consolidating as a key link in the chain mediation. Finally, the abdominal aorta diameter was not directly associated to BMI, but was strongly influenced by the dimensions of the mid-descending aorta and the region of the diaphragm, reinforcing the role of these two zones as essential mediators in the relationship between nutritional state and aortic morphology. These findings reveal a structured sequence, where the effects of overweight or obesity are progressively transmitted along the aorta, particularly toward the inferior sections, which may have clinical implications in the identification of anatomical modifications associated to cardiovascular risk *(Table 3)*.

Chain mediation of BMI on the abdominal aorta diameter by different anatomical aortic structures was evaluated, revealing 63 trajectories, of which 4 were selected, that were more significant in the transmission of the effect. The trajectory, that includes the sinus of Valsalva, the mid-descending aorta and the diaphragm region showed the greater indirect impact, reflecting a central

TABLE 3
Regression models for mediator variables and the result (abdominal aorta)

Dependent variable	Predictors	B	SE	t	p	95% CI
Aortic sinus of Valsalva	**BMI**	14.159	0.2988	4.739	<0.001	[0.8294. 2.0024]
Sinotubular junction	**BMI**	0.4686	0.1605	2.921	0.0036	[0.1537. 0.7836]
	Aortic sinus of Valsalva	0.7841	0.0187	41.847	<0.001	[0.7473. 0.8209]
Mid-descending aorta	**BMI**	-0.0715	0.2310	-0.310	0.757	[-0.5249. 0.3819]
	Aortic sinus of Valsalva	0.1703	0.0480	3.551	<0.001	[0.0762. 0.2644]
	Sinotubular junction	0.7132	0.0507	14.071	<0.001	[0.6137. 0.8127]
Middle aortic arch	**BMI**	0.0982	0.1307	0.752	0.452	[-0.1582. 0.3547]
	Aortic sinus of Valsalva	0.0376	0.0273	1.376	0.169	[-0.0160. 0.0913]
	Sinotubular junction	0.0968	0.0320	3.021	0.0026	[0.0339. 0.1596]
	Mid-ascending aorta	0.4392	0.0200	21.925	<0.001	[0.3999. 0.4786]
Mid-descending aorta	**BMI**	0.4837	0.1262	3.832	<0.001	[0.2359. 0.7315]
	Aortic sinus of Valsalva	0.0426	0.0264	1.611	0.108	[-0.0093. 0.0945]
	Sinotubular junction	-0.0344	0.0311	-1.105	0.270	[-0.0954. 0.0267]
	Mid-descending aorta	0.2065	0.0245	8.425	<0.001	[0.1584. 0.2546]
	Middle aortic arch	0.5732	0.0342	16.743	<0.001	[0.5060. 0.6404]
Aorta in the diaphragm	**BMI**	-0.0200	0.1046	-0.191	0.848	[-0.2253. 0.1853]
	Aortic sinus of Valsalva	0.0336	0.0217	1.548	0.122	[-0.0090. 0.0763]
	Sinotubular junction	0.0172	0.0256	0.674	0.501	[-0.0330. 0.0674]
	Mid-ascending aorta	-0.0214	0.0210	-1.022	0.307	[-0.0627. 0.0198]
	Middle aortic arch	0.0660	0.0327	2.020	0.044	[0.0019. 0.1302]
	Mid-descending aorta	0.6110	0.0291	20.991	<0.001	[0.5539. 0.6682]
Abdominal aorta (final section)	**BMI**	-0.0381	0.1010	-0.377	0.706	[-0.2363. 0.1601]
	Mid-descending aorta	0.1570	0.0350	4.480	<0.001	[0.0882. 0.2258]
	Aorta in the diaphragm	0.6113	0.0343	17.839	<0.001	[0.5440. 0.6786]

SS: standard error.

TABLE 4
Indirect effects of BMI on the abdominal aorta through aortic anatomical mediators (model 6 of PROCESS of Hayes)

Indirect effect	Effect (coefficient B)	Standard error	95% CI
BMI → aortic sinus of Valsalva → mid-descending aorta → aorta in the diaphragm → abdominal aorta	0,1807	0,0484	[0,0878, 0,2778]
BMI → mid-ascending aorta → middle aortic arch → mid-descending aorta → abdominal aorta	0,0611	0,0169	[0,0319, 0,0981]
BMI → mid-descending aorta → abdominal aorta	0,0759	0,0268	[0,0306, 0,1343]
BMI → mid-ascending aorta → middle aortic arch → abdominal aorta	0,0108	0,0049	[0,0029, 0,0218]

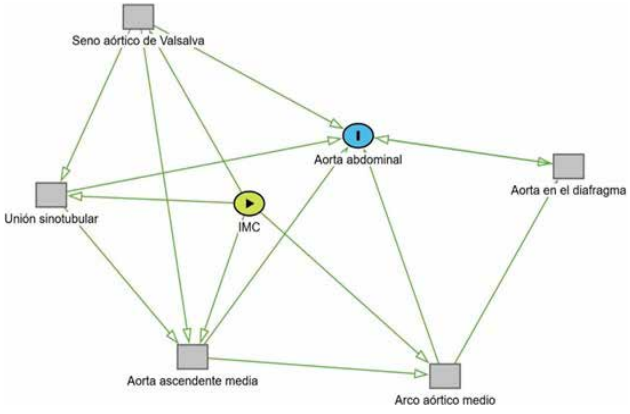

FIGURE 1.
Relationship between BMI and abdominal aorta diameter mediated by anatomical segments of aorta: directed acyclic graph model

role of these sections in the propagation of the effect of the nutritional state toward the abdominal aorta. Likewise, the sequence that involves the mid-ascending aorta, the middle aortic arch and the mid-descending aorta also showed a statistically robust mediation. Other shorter paths, although with effects of less magnitude, were significant and contributed to a comprehensive understanding of the mechanism. These findings highlight the structural relevance of the aortic anatomy as a transmission path for BMI effects, reinforcing the importance of considering complex physiological paths in the evaluation of cardiovascular risks associated to overweight and obesity *(Table 4)*.

In the DAG, the relationship between BMI and the abdominal aorta diameter was evaluated, considering different anatomical segments of the aorta as possible mediators. BMI was defined as the variable of exposition, with the aim of evaluating its indirect impact on the abdominal aorta diameter, through intermediate anatomical structures. The mediators were the aortic sinus of Valsalva, the sinotubular junction, the mid-ascending aorta, the proximal aortic arch, the middle aortic arch, the proximal descending thoracic aorta, the mid-descending aorta and the aorta in the diaphragm. These segments played an essential role in the configuration of the causal trajectories that explain how body fat may induce structural modifications in the distal aorta. Likewise, the existence of possible unobserved variables is acknowledged, such as diet habits, physical activity and blood pressure, which may influence both body composition and vascular architecture, without being explicitly included in the model *(Figure 1)*.

DISCUSSION

The findings of the study suggest the existence of a complex network of associations between BMI and the abdominal aorta diameter, mediated by different anatomical regions of the aorta. Through an approach of chain mediation (model 6 of PROCESS of Hayes), it was shown that BMI, particularly when within ranges of overweight and obesity, is significantly associated with an increase in the dimensions of certain proximal sections of the aorta, especially the aortic sinus of Valsalva and the sinotubular junction. This finding is coherent with the previous literature linking weight excess with vascular structural remodeling, even in absence of manifest cardiovascular disease.[12,13]

Although BMI did not show a significant direct association with the abdominal aortic diameter, the indirect effects through aortic mediators were statistically relevant. In particular, the trajectory that included the sequence aortic sinus of Valsalva → mid-descending aorta → aorta in the diaphragm → abdominal aorta presented the highest indirect coefficient (0.1807; 95% IC: 0.0878-0.2778), which emphasizes the role of the descending axis of the thoracic and diaphragmatic aorta as predominant path of transmission of the BMI effect on the abdominal aorta. This pattern suggests that the remodeling induced by BMI starts in proximal regions and advances toward distal segments, a phenomenon possibly mediated by cumulative hemodynamic and structural adaptations.

Furthermore, other significant paths like BMI → mid-ascending aorta → middle aortic arch → mid-descending aorta → abdominal aorta (coefficient = 0.0611; 95% CI: 0.0319-0.0981) and BMI → mid-descending aorta → abdominal aorta (coefficient = 0.0759; 95% CI: 0.0306-0.1343) reinforce the significance of the mid-descending aorta like a key node in mediation. This is consistent with studies that have reported that the diameters in the descending regions of the aorta are more closely related to metabolic factors and central adiposity.[13] The mid-descending aorta, when receiving the influence both from ascending regions and the aortic arch, acts as an anatomical and functional bridge in the propagation of the BMI effect toward the abdominal aorta.[14,15]

We should highlight that, although some sections like the mid-ascending aorta and the middle aortic arch did not show significant direct association with BMI, its participation in relevant mediator trajectories was identified from the analysis of indirect effects within the chain mediation model. This suggests that they could play a role in the transmission of the BMI effect toward the abdominal aorta, in spite of not having a significant direct

link. This finding highlights that even anatomical segments with marginal contributions in the direct analysis may play relevant roles within chain models, where their effect strengthens when interacting with other structures.

Although the relationship between central obesity and aortic dilatation has been described previously, these results reinforce its clinical relevance. For example, in individuals with central obesity, the identification of dilatation in thoracic segments like the aortic sinus of Valsalva or the mid-descending aorta could be considered an anatomical indicator of progression toward dilatation in the abdominal aorta, even before becoming clinically significant. This suggests that the monitoring of these segments by imaging techniques (transthoracic echocardiography or CT) could improve the early detection of vascular structural changes. Similarly, these mediator paths could be useful in the risk stratification of abdominal aorta aneurysm, allowing earlier interventions in populations in high metabolic risk. Although an intensive control of the cardiovascular risk factors is a widely established recommendation in the presence of arterial disease, the findings in this study suggest that in patients with obesity and mid-descending aorta dilatation, even with abdominal aorta within normal parameters, could justify a closer surveillance and an anticipated therapeutic approach to prevent progression of the distal vascular damage.

This study has significant limitations. As its design is cross-sectional, it does not allow to establish causality between BMI and aortic changes. Furthermore, no possible confounding factors were considered, like blood pressure, physical activity or metabolic parameters. Measurements were static anatomical, not evaluating functions like aortic stiffness. Finally, the results cannot be extrapolated to other populations with different characteristics.

To conclude, the findings in this study suggest, in an exploratory manner, that BMI impact on the abdominal aorta diameter would not occur in isolation or linearly, but that could be mediated by a sequence of interconnected anatomical structures; however, these interpretations should be considered cautiously, given the limitations of the study, among them its cross-sectional design, the omission of relevant clinical variables (like blood pressure or physical activity), and the lack of functional evaluations of the aorta. In spite of these limitations, the proposed approach could work as a basis for the development of future research, and potentially, contribute to identifying clinical tools oriented toward an early detection of distal aortic dilatations in people with weight excess, from the analysis of proximal sections. This line of investigation may favor, in the long term, anatomical risk stratification and the orientation of more personalized therapeutic interventions in preventive contexts or in vascular surveillance.

Future longitudinal studies would allow to clarify whether these associations reflect reversible adaptive processes or if they represent early stages of progressive structural alterations in a context of weight excess. Furthermore, the incorporation of hemodynamic, inflammatory and genetic markers can contribute to clarify the specific mechanisms involved in the aortic remodeling induced by BMI.

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Original Investigation Reports

Assessment of use of technological resources in cardiac rehabilitation in Argentina. Continuation of Census of the Cardiology of Exercise Committee 2022. Reality of cardiac rehabilitation in Argentina in the last 10 years.

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Technological resources.

ABSTRACT

Introduction: cardiac rehabilitation (CR) is an essential therapeutic strategy to reduce cardiovascular morbidity and mortality and to improve quality of life. In Argentina, its implementation is limited by geographical disparities, infrastructure constraints, and shortages of human resources. Digital health technologies have emerged as a potential tool to expand access and optimize patient follow-up; however, their use has not been systematically assessed at national level.

Objective: to describe the use of technological resources in cardiac rehabilitation programs (CRPs) in Argentina and to characterize centers implementing these tools.

Materials and Methods: a collaborative, prospective, observational, multicenter, nationwide registry was conducted. Cardiac rehabilitation centers registered with the Argentine Federation of Cardiology were included. A structured online survey was administered to medical directors, collecting data on institutional characteristics, use of digital technologies, follow-up modalities, clinical outcomes, and patient acceptance. Data was analyzed using descriptive statistics.

Results: Twenty-five centers participated, predominantly private institutions (72%), distributed across several provinces. Technological tools were used by 56% of centers, mainly smartwatches (50%) and center-developed applications (29%). Most centers had been using these resources for more than three years, weekly or more frequently. Patients were predominantly classified as in moderate cardiovascular risk (64%). Clinical improvements were reported by 69% of centers, with high or moderate adherence and good patient acceptance. Remote follow-up was primarily conducted through instant messaging platforms and telephone calls.

Conclusions: the incorporation of digital technologies into cardiac rehabilitation in Argentina is increasing but remains heterogeneous. These tools are perceived as beneficial for improving access, adherence, and follow-up, representing an opportunity to promote more accessible and equitable models of care. Further studies are needed to standardize their use and ensure safety and effectiveness.

INTRODUCTION

Cardiac rehabilitation (CR) constitutes a fundamental therapeutic intervention, recommended by the international clinical guidelines due to its impact on reduction of cardiovascular morbimortality, improvement of quality of life and functional reintegration of patients.[1,2,3] In spite of the proven efficacy, its implementation is still limited, particularly in contexts with limited resources and wide geographical dispersion, as it occurs in Argentina.

According to the 2022 Survey of the Committee of Cardiology of Exercise of the Argentine Federation of Cardiology, in the last decade, an increase has been observed in the number of centers of CR in the country, although there are inequalities persisting in its geographical distribution, as well as limitations in infrastructure and access.[4] This scenario poses the need to explore innovative strategies, that would allow to enhance coverage and optimize the available resources.

In this scenario, the incorporation of digital technologies includes telemonitoring, mobile apps and portable devices, emerging as an alternative to supplement the traditional models of rehabilitation. Different studies have shown that these tools can improve functional capacity, quality of life and adherence to CR programs.[5,6] Similarly, recent evidence suggests that its implementation could be associated with a reduction of hospitalizations in patients with heart failure.[7]

In spite of this evidence, the adoption of technologies in cardiac rehabilitation programs in Argentina has not been evaluated systematically.

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AIMS

1. Describing the use of technologies in CR centers in Argentina.
2. Characterizing implementation modalities.
3. Exploring the variables reported by health care teams.

MATERIALS AND METHODS

A collaborative, prospective, observational, multicenter and national study, based on a survey.

Data collection was made between March and June, 2025.

The study population was constituted by cardiac rehabilitation centers registered in the Committee of Cardiac Rehabilitation and Cardiology of Exercise of the Argentine Federation of Cardiology (FAC), active at the time of the survey and that agreed to participate voluntarily. Centers with incomplete data were excluded.

A structured online questionnaire was used (Google Forms), addressed at the people in charge of every center. The instrument included closed and multiple choice questions about the characteristics of the center, human resources, modality of care, use of technologies, tools used and remote follow-up strategies.

The questionnaire was developed by the research team and previously tested through a pilot test.

The studied variables, degree of adherence, acceptance and clinical improvements were defined based on the perception of the treating teams. Acceptance was characterized as very good, good or regular. The degree of adherence on the use of technologies by the patients was categorized as high (>75%) or moderate (50-75%). Clinical improvements were defined according to the subjective global evaluation of clinical evolution.

A descriptive statistical analysis was made through InfoStat, version 2020.

RESULTS

There were 25 centers of cardiac rehabilitation participating, distributed in different provinces of the country *(Figure 1)*; 72% corresponded to the private field, 20% to the public sector and 8% to a mixed modality.

The average of patients that participated in the CR sessions per month was 25 (range 4-300). Sixty percent of patients were older than 60 years.

All centers had cardiologists; 84% had physical education professors, 60% had kinesiologists, 60% had nutritionists and 44% had psychologists.

Taking into account the modality of operation, it was observed that 84% was in person and 16% hybrid or virtual.

Fifty-six percent of the centers used technologies to monitor patients. The most used tools were: smartwatches 50%, their own apps 29%, telemetry 14% and web platforms 7% *(Figure 2)*.

Sixty-four percent of patients that were using these tools were of moderate risk, 29% of high risk and 7% of low risk.

Using these technologies had been done for more than 3 years in 57% of centers.

As to the reported variables, the degree of adherence was considered high in 54% and moderate in 46%. Acceptance was very good in 43%, good in 50% and regular in 7%. Sixty-nine percent of the centers reported clinical improvements perceived according to the report by the health care team.

The remote follow-up was made mainly by messaging apps in 64%, phone calls in 21%, apps with automatic feedback in 14% and videoconferences in 7%.

DISCUSSION

Results show that more than half of CR centers in Argentina have incorporated technologies, although with heterogeneity in their implementation. This finding is consistent with international trends, showing a transition toward hybrid health care models.[8,9]

The recommendations of the American Association of Cardiovascular and Pulmonary rehabilitation emphasize how these in-person and remote modalities supplement each other, which is reflected in these results.[8]

The predominant use of devices such as smartwatches and mobile apps suggests an adoption based on the availability rather than in standardization. The variability observed highlights the need to develop uniform criteria for quality and safety.[9,10]

Although the centers report improvements in adherence and clinical outcomes, these observations should be interpreted cautiously, and be based on objective measurements.[11,12]

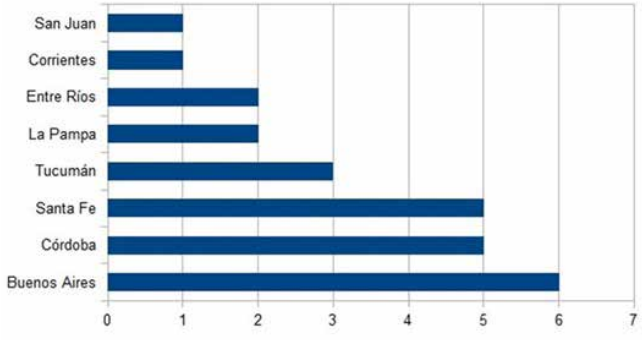

FIGURE 1.
Distribución de los centros de rehabilitación cardíaca

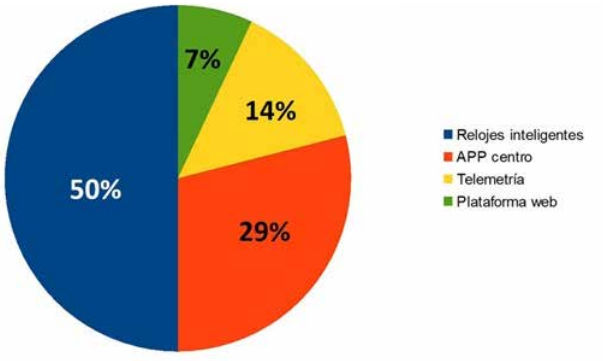

FIGURE 2.
Recursos tecnológicos utilizados

The implementation of these tools also poses challenges, including professional training, regulation and equity in access.[13,14,15] Nonetheless, it represents a chance to extend the coverage of CR, especially in scenarios with geographical limitations.

Limitations

The study presents inherent limitations due to its cross-sectional design, based on a survey, including selection and information biases. The variables reported were not measured by validated instruments.

Ethical considerations

The study was made in agreement with the Declaration of Helsinki and National Law N° 25.326. Participation was voluntary and anonymous.

CONCLUSIONS

In Argentina, approximately half of cardiac rehabilitation centers use technologies in their programs. The use of technological resources is a practice adopted by more than half of the CR centers surveyed in Argentina (56%), being characterized by a predominant use of smartwatches and apps, with a mainly weekly follow-up through instant messaging and calls. The implementation of these technologies was associated to a favorable perception by health care teams, reporting high levels of acceptance, adherence and clinical improvements in the evolution of patients. Although they consolidate an expanding alternative to optimize follow-up, standardization strategies are required for their regulation at national level. Their implementation is heterogeneous and supplementary to the model in person.

By the way, future studies are required to evaluate their clinical impact, by standardized methodologies.

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Position Statement

Position statement of the Argentine Federation of Cardiology regarding psychological stress and its impact on cardiovascular health.

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ABSTRACT

Psychological stress is a clinically relevant and modifiable determinant of cardiovascular risk. This Position Statement of the Young Cardiologist Committee of the Argentine Federation of Cardiology (FAC) synthesizes the evidence and establishes operational recommendations for practice in the Argentine health context.

This statement maintains that systematic stress screening should be incorporated at key moments of the cardiological process (onset of disease, hospitalization due to events and follow-up controls), using validated and brief instruments; that non-pharmacological interventions with empirical support (psychoeducation, cognitive-behavioral therapy, stress management, mindfulness, physical activity) constitute the therapeutic pillar; and that early referral to mental health is essential when the burden of symptoms compromises adherence, safety or quality of life.

Services and managers are urged to organize screening and referral circuits, train teams and monitor process and result indicators. This statement guides professionals, institutions and authorities to integrate Mental Health and Cardiology, promote equity in access and stimulate local research on implementation and outcomes.

INTRODUCTION

In recent decades, understanding the link between mind and heart is no longer just a theoretical field, but has turned into a field with increasing clinical relevance. Nowadays we know that psychological stress (both acute and chronic) not only accompanies cardiovascular diseases, but it may also act as a causal and modulator factor, and also prognostic for its course.

Scientific evidence is conclusive: the impact of mental health on cardiovascular health is circular and bidirectional. Different scientific societies in the world acknowledge this link, highlighting the recent publication of the Guideline of the European Society of Cardiology on Mental Health and Cardiovascular Disease, being one of the most significant landmarks in the integration of mental health into the field of modern cardiology.[1]

As cardiologists, we consider that understanding and approaching psychological stress is an essential aspect of providing care for patients, both in the medical office and in more complex scenarios (hospital admission or rehabilitation). In a society characterized by high levels of demand and the growing prevalence of chronic stress in the population, cardiology is responsible for considering this perspective in prevention, diagnosis and treatment.

This position statement attempts to reaffirm the importance of integrating the knowledge of stress and its impact on cardiovascular disease as part of the professional competency of contemporary cardiologists. Providing care to the heart also entails providing care to the emotional context influencing it and conditioning it. This document seeks to reinforce this point of view and to promote a more humane and comprehensive practice, and based on evidence.

Scientific or ethical basis

Cardiovascular disease is the main cause of morbidity and mortality in the world. Psychological stress, both acute and chronic, is acknowledged as an independent risk factor, modifiable in its genesis and progression. An early detection of stress in the clinical practice can reduce its impact on cardiovascular health, with positive implications on the health of the population and on health care costs.

This position statement seeks to reinforce the need for a systematic identification and an initial adequate approach by the health care team.[2,3]

CHAPTER 1: STRESS AND ACUTE ANXIETY

Acute stress is a short-duration response to a sudden or threatening stimulus. Unlike chronic stress, characterized by a prolonged exposition to adverse factors, this happens suddenly and triggers the classical "fight or flight" response by an immediate neurohor-

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monal activation. Intense emotions (anger, extreme fear) or unexpected traumatic events (natural disasters) can trigger it.

Its clinical relevance lies in its capacity to trigger major cardiovascular events. Different studies have shown a transient increase in the risk of acute myocardial infarction and sudden cardiac death immediately after intense stress situations. Paradigmatic examples include large-scale natural disasters like earthquakes and tsunamis. Similarly, anger episodes have been associated to an increase in the risk of presenting an acute coronary syndrome within hours after it. Even emotional situations like important sports competitions can increase the rate of infarctions and arrhythmias suddenly and significantly in the population. These findings prove that acute stress is not a benign phenomenon for the cardiovascular system, and that it may trigger acute myocardial infarction, malignant arrhythmias and even sudden cardiac death, even in people with no traditional risk factors.[4,5,6,7,8]

As to pathophysiological mechanisms, acute stress massively activates the sympathetic nervous system and the hypothalamic-pituitary-adrenal axis. Catecholamine release generates tachycardia, increases blood pressure abruptly, and increases the myocardial demand for oxygen while reducing coronary perfusion. This, added to vagal tone decrease, favors the appearance of myocardial ischemia and ventricular arrhythmias. Furthermore, it has been observed that mental stress can induce transient myocardial ischemia even in the absence of significant coronary lesions, a phenomenon known as mental stress-induced myocardial ischemia, attributable to paradoxical coronary vasoconstriction and endothelial dysfunction. This ischemia occurs with less strain than that induced by exercise, and it is usually silent, making its detection difficult. On the other hand, neurohormonal activation generates a transient prothrombotic state, with greater platelet aggregation and procoagulant activity, which may precipitate atheroma plaques rupture and the subsequent formation of coronary thrombi.[6,7]

From a clinical point of view, it is crucial to ask about the presence of recent acute stressors. Incorporating psychosocial risk factors in the stratification of cardiovascular risk is advisable, as its impact on morbimortality has already been proven. A directed anamnesis can reveal triggers such as strong arguments, sudden losses and significant scares.[9,10,11]

The initial management of acute stress in the medical office is mainly non-pharmacological. It is important to educate patients in basic stress control techniques (such as deep breathing, relaxation and mindfulness) and to encourage healthy lifestyle habits (regular physical activity, good rest, an adequate emotional support network). It has been proven that strengthening social support and active strategies of coping can attenuate the impact of stress on cardiovascular health. These interventions contribute to reducing an adverse physiological reactivity to stress and are a first-line strategy in patients with a high load of acute stress.[8,9,10,12,13,14]

When distress exceeds the capacity of coping or interferes with the cardiological treatment, an early referral to a mental health team should be considered (for instance, psychocardiology programs). An interdisciplinary approach enables a comprehensive treatment and it may improve the prognosis.

To conclude, a proper identification and management of acute stress as a cardiovascular risk factor should be part of the contemporary clinical practice. Approaching these psychosocial factors may not just improve the quality of life of patients, but also contribute to the prevention of major cardiovascular events.

CHAPTER 2: CHRONIC STRESS

Chronic stress emerges from a prolonged exposition to adverse life conditions, such as hostile work environments, financial insecurity, interpersonal conflicts or time limitations. Its definition is more complex than what is commonly expressed, and is not exclusively limited to the duration of the stressor stimulus. It has been demonstrated that this entity is associated, both in cross-sectional and prospective studies, with a greater risk of developing multiple diseases. This link is explained by direct pathophysiological mechanisms, as well as the adoption of little healthy coping strategies, which may negatively impact on general health and reduce life expectancy.[15,16,17]

In recent decades, evidence has been collected linking psychosocial factors, including stress, with cardiovascular events. Variables such as chronic stress, anxiety and depression are difficult to quantify objectively, which has represented a methodological challenge in clinical investigation. Nonetheless, the INTERHEART study, which evaluated risk factors for acute myocardial infarction (AMI) in 52 countries throughout the world, showed a significant association between perceived psychosocial stress and the risk of AMI. In particular, chronic stress in the home and work showed a strong relationship with cardiovascular events, with an odds ratio (OR) of 2.67.[8] In regard to depression, a meta-analysis made by Howren et al. found that it is associated to elevated levels of inflammatory markers such C-reactive protein, interleukin-1 and interleukin-6, all of them linked to a greater risk of CAD and cardiovascular mortality.[18,19]

The pathophysiological mechanisms that link chronic stress with cardiovascular disease include a sustained activation of the sympathetic autonomic nervous system, systemic inflammatory response and vascular dysfunction. Besides these biological processes, chronic stress could be accompanied by risky behaviors, such as smoking, alcohol consumption and sedentary life; risk factors properly established for the development of CAD. Likewise, a strong association has been documented between stress and comorbidities like obesity, diabetes mellitus and hypertension, conditions that increase cardiovascular risk significantly. In this sense, a meta-analysis by Wu et al. showed that a proper control of these comorbidities can partially attenuate the relationship between depression and myocardial infarction or death by coronary cause.[20]

In recent years, the role of non-traditional risk factors has become relevant in the development of cardiovascular risk factors, particularly those of the psychological and socioenvironmental types. In a systematic review from 2017, the link between chronic stress and CAD, hypercholesterolemia and type 2 diabetes mellitus has been proven. Numerous studies have shown that a low socioeconomic level, poor working conditions and demands related to health care can generate chronic stress, which is associated to a greater risk of cardiovascular events.[21,22]

TABLE 1.
GAD-2 screening tool

Within the last two weeks, how often have you experienced these feelings?	Not at all	Some days	More than half of days	Almost every day
Feeling nervous, anxious or on edge	0	1	2	3
Not being able to stop or control concerns	0	1	2	3

A score of more than 3 has a positive clinical significance for symptoms of anxiety.
GAD-2: Test of 2 items for generalized anxiety disorder

Financial stress, originated by financial hardships, has been consistently associated to negative effects on health and a higher cardiovascular risk, both in the short and the long term. During the financial crisis in Argentina from 1997 to 1999, the decrease of GDP coincided with an 11.36% increase in mortality by acute myocardial infarction, showing the impact of the socioeconomic context on cardiovascular health.[23,24]

In terms of occupational conditions, it has been proposed that cardiovascular risk does not depend on a single factor, but on the interaction between high psychological demands, low control on tasks and physiological consequences like the development of metabolic syndrome. This point of view emphasizes the need to approach work-related stress from a multidimensional perspective, transcending the individual characteristics of workers.[25]

To conclude, chronic stress and the associated emotional factors represent significant determinants in the genesis and progression of CAD. The integration of specific therapeutic approaches for stress management could not only improve the quality of life of patients, but also constitute an efficient strategy in the prevention of major cardiovascular events.

CHAPTER 3: DIAGNOSIS

In any visit by patients, professionals should be open to identifying previously unknown or new symptoms about the state of mental health. It has been verified that health care services for patients with cardiovascular disease sometimes lack the necessary education to identify mental health issues.[25]

a. Psychometric tests

To facilitate the stress screening of patients, questionnaires can be used, as they are practical and not too time-consuming. Among them, there is the General Anxiety Disorder (GAD) questionnaire, having a high sensitivity and low specificity for the diagnosis of anxiety. It starts with 2 questions, which if positive, continue with the GAD-7 with 7 questions, with greater specificity. In the consensus of Mental Health of the European Society of Cardiology from 2025, it is suggested to perform this questionnaire during the diagnosis of new cardiovascular disease or new cardiovascular event, including the period of admission, with a follow-up that could be yearly or according to the clinical judgment of the intervening health care professionals *(Table 1)*.[1,26,27,28]

The Perceived Stress Scale (PSS) is another widely used tool, with an appropriate population validation. This scale evaluates the stress perceived in the last month, and on the other hand, the capacity to cope (resilience). It could be self-reported by the patient. There are 14 questions with a scale of points, from 0 or never, to 4 very often. PSS-10 is a shortened version with 10 questions.[29,30]

b. Biomarkers

For a biomarker to be used for diagnosis, it should be quantifiable, sensitive and specific. Although some studies have shown a link between stress and the alteration of biomarkers, among them cortisol and adrenaline, the low evidence makes it not to be suggested as a diagnostic method.[31,32]

CHAPTER 4: THERAPEUTIC MEASURES

Stress management should be considered part of a comprehensive treatment of cardiovascular disease. During the interview, professionals should seek a good communication between patients and physicians, both active and empathetic, to facilitate the detection of psychosocial and emotional factors. Thus, the causes for the problems of patients can be better unmasked, and tools for stress management could be approached. Comprehensive therapeutic adherence and quality of life will improve in turn. An interdisciplinary approach is recommended, to promote healthy habits, relaxation techniques and accompaniment by mental health professionals.

a. Psychotherapy

Psychological therapy guided by a professional has been studied in follow-up programs after cardiovascular events, with good results in terms of disease-health process acceptance, decrease of stressing symptoms and improvement of adherence to treatment of base pathologies. In patients with arrhythmias like atrial fibrillation and patients with implantable cardioverter defibrillators, it showed improvement in quality of life questionnaires.[33,34,35,36,37,38]

Cognitive behavioral therapy (CBT) has the most evidence. Its approach is on changing negative thought and behavior patterns. Two systematic reviews in patients with CAD showed that CBT can reduce new CV events. There are studies that show that CBT after a CV event decreases the rate of readmission or new CV events. In the studies that did not show a decrease in major cardiovascular events, an improvement in quality of life was observed, both for AF and myocardial infarction. This therapy has indications like treatment in guidelines and consensuses of hypertension, chronic CAD and hypertension. A variable is CBT for groups, where strategies are aimed at the prevention of anxiety, with several protocols with proven efficacy. A small stu-

dy in university students showed faster improvements in issues like depression and anxiety when undergoing group therapy vs individual therapy. Group therapies based on mindfulness also displayed improvement in terms of anxiety and management of negative emotions.[39,40,41,42,43,44,45,46,47,48,49,50,51,52]

b. Mindfulness and meditation

Mindfulness entails concentration in the present time, by helping cognitive resilience when facing stressor factors, helping emotional regulation. It seeks full attention in the present time, with a kind attitude and no judgement. It is paying attention to the present time, without criticisms. This practice attempts to foster a mental state of openness and acceptance, favoring emotional regulation, stress reduction and a greater feeling of general wellbeing.[53,54,55]

Therapies based on mindfulness have proven evidence of improvement of quality of life in patients who had a cardiovascular event like myocardial infarction with coronary angioplasty. In these patients, it has been shown that mindfulness helps to control stress and symptoms of anxiety. In cardiac rehabilitation, it was shown that rehabilitation with exercise and mindfulness was better than the former in isolation in terms of personal wellbeing. A reduction in values of blood pressure has been demonstrated, and its use was included in consensuses. Perfectionist personalities, with decrease in HR variability, can benefit from mindfulness.[46,56,57,58,59,60,61,62]

Meditating focuses on breathing, sounds or feelings to reduce levels of stress and its negative effects. Meditating entails having your mind focused and reaching or looking for inner peace. Its efficacy has been proven both in primary prevention (decrease in CV events) and in secondary prevention, both with mindfulness and with meditation. Therapies like yoga and listening to classical music can also be used as treatment, due to its proven cardioprotective effect.[62,63,64,65,66]

To conclude, all of the evidence available supports mindfulness and meditation like an efficient, safe and potentially cardioprotective strategy. Its implementation in rehabilitation and secondary prevention programs allows to optimize the comprehensive management of patients with cardiovascular disease, approaching psycho-emotional factors influencing on clinical evolution.

c. Prescription of social activities

Prescription of social activities means providing people with personalized treatments extending beyond the traditional pharmacological treatment, aimed at resources and activities in the community. They improve adherence to the usual cardiovascular treatment, reduce symptoms of stress and anxiety, and improve quality of life questionnaires. Between the prescribed activities that have been studied, we find dancing, climbing, cooking classes, gardening. Social interventions that include professionals are more effective to improve blood pressure values and adherence to physical activity. Activities such as gardening, exercising in green areas and walking in nature proved to be efficient to reduce blood pressure values and to moderate anxiety scores.[67,68,69,70,71]

d. Modifications in lifestyle

A holistic treatment should include lifestyle modifications to reduce cardiovascular risk, improve mental health state and increase adherence to treatment. Physical activity generates release of endorphins, which among other neuroactive substances, improve mood and reduce stress, and is particularly useful in patients with CV disease.[72,73]

Interventions in diet have twice the result in improving mental health and CV prevention goals. Diets with a high consumption of sugar, refined carbohydrates and saturated fats are linked to an increase in anxiety. In this aspect, there should be more studies linking diets generating low arterial inflammation and a significant reduction in stress.[74,75]

Quitting smoking has proved that CBT associated to medication or nicotine was better than medication alone to achieve smoke abstinence.[76]

e. When to refer. Pharmacological treatment

In the 2025 ESC position statement on Mental Health and Cardiovascular Disease, it is advised to create an interdisciplinary cardiology-mental health team for a comprehensive approach of patients. When faced with cases of severe mental disease (anxiety, depression) or suspicion of severity, diagnosed by the abovementioned questionnaires (Chapter 3: diagnosis), a referral to mental health teams is suggested for an initial care by a specialist.[1]

Use of benzodiazepines presents levels of overindication and an excessive use all over the world, particularly in the elderly. The indication of benzodiazepines relates to an increase in all-cause mortality. This position statement defines indication of pharmacological treatment as out of reach, and mainly advises against using it as first line, being saved only for moderate-severe scenarios of anxiety and depression.[77,78,79]

CHAPTER 5: OFFICIAL POSITION

As an emerging cardiovascular risk factor, and with growing scientific evidence, it is considered that in medical practice, primary care professionals should have, among their tools, the capacity to detect psychological stress in consulting patients.

Simultaneously, taking into consideration the high prescription of benzodiazepines, we advise against their use as first line systematically.

CHAPTER 6: ETHICAL CONSIDERATIONS AND CONFLICTS OF INTEREST

We state there were no conflicts of interest to generate this medical position statement.

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Position Statement

Position statement of the FAC Committee on Peripheral Vascular Disease and Stroke on prevention of deep vein thrombosis in prolonged trips.

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ABSTRACT

Deep vein thrombosis (DVT) and pulmonary embolism (PE) are the most common manifestations of venous thromboembolism (VTE). According to published data, VTE affects nearly 10 million people worldwide each year, contributing significantly to the global burden of disease. The pathophysiology of thrombosis is explained through Virchow's triad: presence of blood stasis, damage or dysfunction of the vascular wall, and hypercoagulability. Risk factors for developing DVT include prolonged travel, defined as trips lasting more than 4 hours, which is why VTE is often referred to as "economy class syndrome" or "traveler's thrombosis". However, it is worth noting that travel-related DVT is even more closely linked to people who have other risk factors for developing venous thrombosis. Thus, these individuals are grouped into low, medium/moderate and high risk, with this categorization being very useful when deciding on the best therapeutic option. Given the high morbidity and mortality associated with DVT cases and the resulting PE, related to prolonged immobilization, which is very common in contemporary times during long journeys, a series of measures and recommendations have been proposed to prevent and reduce the risk of developing VTE.

INTRODUCTION

Most DVT affect the veins in the inferior limbs or the pelvis (70-80%). As the symptoms of DVT are unspecific, it is essential to make a fast and standard diagnosis to minimize the risk of PE in the acute phase, and prevent the progression of thrombosis, post-thrombotic syndrome and recurrence of VTE in the long term.[1]

VTE, affecting almost 10 million people of all ethnicities throughout the world and every year, significantly contributes to the world load of morbidity. The yearly incidence of acute venous thromboembolism is 1 to 2 cases per 1000 inhabitants; a figure that increases exponentially with age both for men and women, and is 4 times greater in countries with high income than in those with low income. The risk of VTE over life does not differ according to sex, but women have a higher risk between 20 and 40 years of age, reflecting their exposition to risk factors during reproductive age, while men have a higher risk in other age groups.[2]

Known risk factors for VTE include immobilization, recent surgery, pregnancy, use of oral contraception, obesity, trauma, malignant neoplasia and hereditary thrombophilia. It has been proven that there is an association between long-duration trips (more than 4 hours) and the development of deep venous thrombosis, because of prolonged immobilization.[3]

Taking into account the circumstances and triggering factors that contribute to the development of DVT, in this position statement we present management strategies to prevent the development of DVT in people who take long trips.

PATHOPHYSIOLOGY OF THROMBOSIS

Deep vein thrombosis (DVT) is a disease in which a blood clot develops in the veins; blood vessels that carry blood back to the heart. Venous thromboembolism is a multicausal disease. It is believed to be triggered by interactions between multiple causal factors that could add to each other or be synergic. Some clinical risk factors are stronger and may cause venous thromboembolism in the absence of other risk factors. These causal factors can be transient or persistent. Transient risk factors, such as major surgery, extended immobilization and major trauma, represent approximately 20% of all episodes of venous thromboembolism.[2]

The pathophysiological mechanisms that lead to thrombosis are traditionally explained by Virchow's triad: blood stasis, vas-

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cular wall damage or dysfunction, and hypercoagulability. It is believed that venous thrombi start in the valve pockets of large veins, which are susceptible to blood stasis, particularly during extended immobilization. Stasis and vascular wall damage or dysfunction may lead to hypoxia or inflammation, processes that regulate negatively the natural anticoagulant properties of the endothelium and, therefore, induce a state of hypercoagulability. The activation of procoagulant venous endothelial cells leads to a greater expression of molecules of superficial adhesion, such as P-selectin and von Willebrand factor, which facilitate the subsequent binding of leukocytes, microparticles and circulating platelets. The activated leukocytes express the procoagulant tissue factor, which is believed to initiate the extrinsic pathway of coagulation cascade (the intrinsic pathway, contact pathway, activated through factors XII and XI), leading to factor X activation, production of thrombin, and ending in the formation of thrombi that include fibrin, red blood cells and platelets.[2]

Recent evidence suggests a complex interaction between coagulation and inflammation, by which the coagulation cascade activation triggers the immunology system and, in turn, innate immune cells contribute to the formation of thrombi, in a process called immunothrombosis. In fact, drugs with anti-inflammatory properties, such as statins, may reduce incident and recurrent venous thromboembolism. Activated leukocytes are the main source of positive procoagulant microparticles for tissue factor, which may stimulate the formation and growth of thrombi. Neutrophils, the most abundant leukocytes, produce neutrophil extracellular traps (NET), that are composed by DNA, histones and antimicrobial proteins. NETs provide a structure for red blood cells, platelets and procoagulant molecules to promote thrombi formation. The concentrations of microparticles positive for circulating tissue factor and NET markers (e.g.: extracellular DNA) increase in patients with venous thromboembolism, but their use as biomarkers for VTE has not been established yet.[2]

Platelets help to induce the formation of NET and increase procoagulant activity in innate immune cells. This could be the mechanism by which aspirin reduces the risk of incident and recurrent venous thromboembolism. The genetic contribution to the risk of venous thromboembolism is not completely characterized. Traditional hereditary thrombophilia, included protein C, protein S and antithrombin deficiency, the gene mutation of prothrombin and factor V Leiden, is associated to genes of the coagulation system. However, a meta-analysis of studies of association of the whole genome identified several genes associated with inflammation, red blood cells and platelets, that were also associated with the risk of venous thromboembolism.2

RELATIONSHIP WITH PROLONGED TRIPS

Venous thromboembolism (VTE) is an ailment that has also been known as the "economy class syndrome" or "traveler's thrombosis" due to its incidence in individuals who have underwent a long period of traveling (more than 4 hours), whether by plane, train, car, truck or bus, within the 8 weeks before diagnosis.[4]

In year 1944, professor John Homans described the sign that carries its name, which is characterized by presenting pain in the calf muscles when performing foot dorsiflexion. The appearance of DVT after traveling by plane was first reported by John Homans himself, who published the relationship between the development of venous thrombosis and immobilization due to long trips in 1954. Initially, it was called the "economy class syndrome", as it was considered that the risk of presenting DVT increased by the narrow or compact configuration of the economy class seats. Later, PE occurred in business class seats, which include additional space for the legs. This suggested that a reduced movement of passengers in long distance flights is the most important determinant factor. However, a significant point is that DVT associated to trips is even more related with people with other risk factors to develop venous thrombosis.[5]

It has been proposed to group these individuals into people with low, medium and high risk of suffering traveler's thrombosis, in such a way that people in high risk are those with personal history of VTE, neoplasia or recent major surgery; people with low to medium risk of VTE have a risk of 0-2% of developing DVT associated with long distance trips. This increases to 5% for people with high risk, as those with history of DVT or states of hypercoagulability, including factor V Leiden mutation and obesity.[6] Another specific factor that may increase the risk of trips is pregnancy. Pregnancy and puerperium are periods known to be of high risk of VTE. An estimated risk of 0.03%-0.1% has been suggested for this population, and is included within the group of medium risk travelers.[6]

Several definitions of long-distance or prolonged trips are used. According to evidence, these vary from trips of more than 4 hours to more than 8 hours. A dose-response or exposure-response relationship has been proven, in the sense that for every 2 hours of additional flight trip, it is estimated that there is a 26% increase in the risk of VTE. Although the risk of VTE is greater during the first week after the trip, the risk can remain elevated for up to 8 weeks after a single long-distance flight. The risk of VTE related to traveling is based, in part, to a passanger's profile.[7] A meta-analysis found an 18% increase in risk for every 2 hours of increase in the duration of trips by any means (car, truck, bus, train), with a higher increase, 26%, associated to flights (due to the hypobaric environment that may induce hypercoagulability).[8] Furthermore, the more flights made, the greater the risk, which generates a greater risk for frequent travelers or business travelers. The risk tripled after five or more long-distance trips in a study, and each additional flight increase the risk by 1.4 times.[9]

DVT PREVENTION STRATEGIES

Because of the known risk of developing DVT and PE as a consequence of immobilization during prolonged periods of time related to trips, several strategies have been proposed to prevent and decrease risk. The recommendation of such strategies will depend on the individual risk of presenting VTE (low, medium or high risk), considering mechanical methods and other that include performing physical movements (non-pharmacological methods), besides pharmacological methods.

As non-pharmacological measures, using compression socks during long distance flights, which may help to reduce the risk of developing DVT, as well as moving the lower limbs, standing up or walking occasionally during the flights, and appropriate hydration. Pharmacological methods encompass from a preventive use of aspirin to low doses of low-molecular-weight heparin.[10]

Several scientific trials have researched these methods to reduce the risk of VTE. The LONFLIT-2 and 5 trials explored the use of compression socks, showing a statistically significant difference between the control group and those using socks, 4.5% and 5.8% of controls developed DVT in the LONFLIT-2 and 5, respectively, in comparison with 0.24% and 0.97% of those using compression socks.[11,12] Current reviews coincide in that there is evidence that compression socks reduce the risk of VTE related to trips, and moreover, help reduce lower limb edema in low risk people.[6]

The pharmacological prophylaxis of DVT has been tested, including using aspirin and low-molecular-weight heparin (LMWH). So much so, that the LONFLIT-3 study showed that aspirin reduced DVT rates significantly; however, the risk was abolished in the group treated with LMWH.[13] Another study evaluated using pirokinase in 150 mg doses (a fibrinolytic agent of oral administration) in individuals in high risk, showing that no thrombotic events occurred in the treated group in comparison with the control group, who presented DVT in 5.4% and a lower number of cases with superficial venous thrombosis (SVT).[14]

It is necessary to consider the potential use of new oral anticoagulants, such as apixaban. Their short half-life, its fast onset of action and its oral administration turns them into an attractive option for prophylaxis of DVT in high risk travelers.

However, trials are necessary to examine their safety and efficacy in the prevention of travelers' thrombosis.[7]

The function of thromboprophylaxis with LMWH during pregnancy varies and will depend on the threshold of risk of VTE used during pregnancy exceeds 1 or 3%, the more recent guidelines recommend a risk threshold of 2% during pregnancy and a risk threshold of 1% in the postpartum period to guide the use of thromboprophylaxis.[8]

Performing exercises with the lower limbs during the trip decreases the risk of developing VTE. These exercises include: feet movements with active contraction of feet and calves muscles (activating the muscle pump), feet exercises against moderate resistance (plantar flexion and active dorsiflexion made against a flat, non-rotating board), feet movements against a higher resistance using a rotating pedal connected to a resistance band. All these exercises proved to provide a maximum contraction of the feet and calves muscles, thus activating the muscle pump while in a sitting position; so much so, that in a series of cases it was proven that the flow of blood volume in the popliteal vein was reduced in almost 40% with individuals sitting and immobilized, and nearly twice that when sitting, immobilized and with their feet not touching the ground. Feet exercise against a greater resistance positively improved the flow of venous blood volume.[15]

Therefore, and in brief, this committee recommends:

- Doing, whenever possible, short trips, of less than 4 hours.
- If long and uninterrupted trips are made in transportation means (bus, car, plane, and so on), increase the seat tilt as much as possible, during a long journey.
- Moving before the fourth consecutive hour of an uninterrupted trip, whether within the transportation means (bus, train, plane) or if traveling by car, stopping it (in a safe place) and move outside of it.
- Ingest abundant water to ensure proper hydration.
- Use loose clothes during and after the trip.
- During the immobilization period, perform regular exercises, at least every hour, extending both legs and moving both feet forward and backward in a circular motion; besides placing the feet on the ground and pointing the fingers upward (activating the calves muscle pump).
- For people with inherited or acquired prothrombotic disease, or with risk factors, reinforce or enhance the previous recommendations (that is to say, do not remain sitting in a car for more than 2 uninterrupted hours), use compression socks during and immediately after the trip, do not forget to take anticoagulation medication (in patients already in treatment).
- Consider using LMWH according to the thrombotic risk of each individual.
- In any case, consult a physician if signs or symptoms of VTE appear, within 4 to 8 weeks after a long trip and prolonged immobilization.

CONCLUSIONS

Given the proven relationship between the incidence of venous thromboembolism and prolonged trips, there is a quite reasonable weight to support effective measures to prevent thrombosis related to long-distance trips, also taking into account the fact that most travelers, even flight passengers, have a deficient knowledge of the thrombotic risk associated to long-distance trips/flights.

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Position Statement

Position Statement of the Argentine Federation of Cardiology's Exercise Cardiology Committee on sports activity in hypertrophic cardiomyopathy.

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ABSTRACT

Historically, individuals with hypertrophic cardiomyopathy (HCM) have been excluded from vigorous exercise and competitive sports due to the risk of sudden cardiac death (SCD), based on retrospective studies. Recent evidence has challenged this paradigm, demonstrating improvements in functional capacity, quality of life, cardiovascular and psychological health when competitive sports are included in low-risk individuals. For those who do not meet these criteria, evidence is more limited. However, participation in competitive sports can still be considered on an individual basis.

A contemporary approach to HCM should be based on individualized, goal-oriented exercise prescription, multidisciplinary intervention, and shared decision-making.

In this review, we critically appraise the evidence on vigorous exercise and competitive sports and explore how new data are reshaping clinical practice and international guidelines, laying the groundwork for a new paradigm in the field.

INTRODUCTION

Hypertrophic cardiomyopathy (HCM) is the most frequent genetic heart disease in young athletes, acknowledged as one of the main causes of sudden cardiac death (SCD) associated to exercise in this age group, although most of patients remain asymptomatic and with low risk of severe events.[1,2,3] It has traditionally been considered a contraindication for the practice of sports, particularly of high intensity and competitive, due to the risk of SCD. However, current evidence and recommendations have shown that most individuals with HCM present a stable clinical course and a low absolute yearly risk of arrhythmic events when evaluated, stratified and followed properly.[1,2]

The most recent guidelines of the American College of Cardiology (ACC), American Heart Association (AHA), Heart Rhythm Society (HRS) and other societies recommend that patients with HCM can benefit from recreational exercise of mild to moderate intensity, improving functional class and quality of life, not increasing the risk of arrhythmic events.[1,2] In competitive sports, in selected cases and after an expert evaluation, with a structured process of shared decision making (SDM), it may be reasonable to authorize participation at competitive level.[4,5] The classical restrictive approach has been replaced by an individualized model supported by a better understanding of phenotypical heterogeneity. The development of advanced tools of cardiovascular imaging, and the incorporation of the SDM concept, have made a significant contribution in the final advice for sports practice.[2,3]

INITIAL EVALUATION

A detailed and multidimensional evaluation of arrhythmic risk represents the cornerstone of clinical management in people with HCM, and is key to determine the ideal level of activity.

The evaluation of patients with HCM that wish to carry out physical activity or participate in sports should be comprehensive, systematic and made by physicians with experience in sports cardiology.

This initial evaluation should include: clinical history with particular emphasis on family and personal history of SCD, unexplained syncope, physical examination, 12-lead ECG, Holter monitoring, graded exercise testing (GXT) and/or cardiopulmonary exercise test (CPET), and cardiovascular imaging studies such as echocardiogram and cardiac MRI.

ECG

The extent of left ventricular hypertrophy (LVH) by imaging is not specific of HCM, and could be secondary to different pathologies. In this context, ECG is extremely useful to guide the differential diagnosis between sarcomere HCM and its phenocopies. Although Q waves with positive T waves in the same leads (Q/T mismatch) and giant T wave (0.10 mm) in the anterolateral leads support the diagnosis of sarcomere hypertrophic cardiomyopathy, short PR interval and/or ventricular preexcitation are frequent findings in deposit diseases, such as glycogen storage disease, Danon disease, PRLAG2 gene mutations and Anderson-Fabry

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disease, and can also be found in mitochondrial diseases.[6]

In HCM, numerous electrocardiographic anomalies are observed and these could be the only manifestation of disease in an early stage. These alterations are determined by extension, degree and distribution of LVH, as well as by the presence of fibrosis and/or necrosis of the cardiac muscle, and by the appearance of intraventricular conduction disorder.[7,8] Around 95% of patients with diagnosis of HCM present ECG alterations, with no pathognomonic signs. Likewise, normal ECG is present in 5% to 10% of cases, and is associated with less severe phenotypical forms and a favorable course; however, an abnormal ECG pattern does not predict future SCD events or appropriate ICD therapy.[9]

Abnormal Q waves are due to localized loss of electrical vectors associated to transmural fibrosis, and the result of the initial QRS vector due to abnormalities due to disproportionate hypertrophy of the interventricular septum and/or free baseline wall. Recent studies that investigate the correlation between the Q waves in ECG and increase in myocardial late gadolinium enhancement in MRI have obtained mismatching results.

Although increased QRS voltages are usually identified in young healthy individuals, this characteristic is usually associated with other anomalies in HCM, and just 2% can show isolated criteria of QRS voltage (Sokolow-Lyon index and Cornell voltage criteria). Complete bundle branch block is extremely rare to see, and in general is due to surgical history.

ST-T segment anomalies are usual in HCM, associated to T-wave inversion.

Inverted T-waves in inferior wall in absence of hypertrophy are identified in 6% of black athletes and in 2% of white ones. Inverted T waves in lateral wall should always be considered abnormal, and may precede the phenotypical manifestations of cardiomyopathy; therefore, clinical surveillance is fundamental.

QTc prolongation probably reflects the interaction of hypertrophy, fibrosis and left ventricular outflow tract obstruction and electrophysiological remodeling of cardiomyocytes.[7]

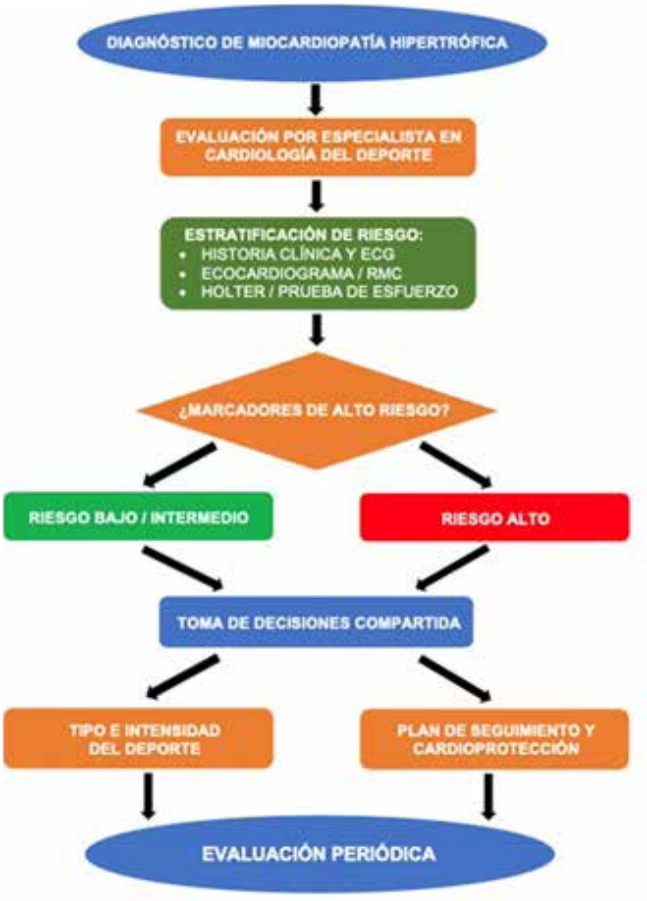

FIGURE 1.
FAC algorithm for evaluation and sports eligibility in hypertrophic cardiomyopathy
Description: confirmed diagnosis → comprehensive evaluation → risk stratification → identification of high risk markers → SDM → definition of sports and cardioprotected environment → longitudinal monitoring.
Based on 2024-2025 AHA/ACC, 2023 ESC Cardiomyopathy and 2020 ESC Sports Cardiology guidelines.

STRESS TEST

Stress test, with its different associated methodologies, is an essential component of noninvasive evaluation of HCM. It is involved in all diagnostic and prognostic algorithms of carriers of this disease.[10,11] It provides information on the severity and possible mechanism of functional limitation in case of left ventricular outflow tract obstruction, abnormal arterial response, chronotropic incompetence, arrhythmias, ischemia and/or reduction in heart rate reserve.[1]

The possibility of complications is extremely low, $<0.4\%$ in the series and its diagnostic contribution compensates the risk by far.[12] The stress test with echocardiography has been added to the standard clinical evaluation and the diagnostic armory in tests limited by symptoms. In symptomatic patients, without outflow gradient in rest, stress test with ultrasound is the most used method to prompt obstruction, which may predict the future development of heart failure symptoms and differentiate them from those without obstruction, with the logical therapeutic implications. On the other hand, there is no justification to perform it if patients have a resting gradient >50 mmHg.

We should be cautious when performing exercise tests in patients with cardioverter implantable defibrillators (CDI) to prevent unexpected shocks, whether appropriate or inappropriate.

Insufficient chronotropic response occurs in up to 25% of patients and it may indicate a low peak oxygen consumption.

Hypotensive or insufficient response to stress is defined as the inability to elevate pressure at least 20 mmHg during exercise peak over baseline or a hypotensive response. It could be a determinant factor in risk stratification, and some authors consider it as an indicator of poor outcome. It may appear in 20-40% of patients.[10] It lacks positive predictive value, but its negative predictive value is very useful to identify patients in low risk, who may restart sports practice in the presence of other low risk elements.[13]

As to myocardial ischemia assessment, stress test in HCM is associated to a considerable rate of false positives, due to the high prevalence of preexisting electrocardiographic anomalies. Although alterations have been proven by microcirculation disease.

Stress test associated to perfusion with radioisotopes is as conflictive, displaying low diagnostic specificity, but also with an elevated negative predictive value.[10]

Cardiopulmonary exercise test (CPET) allows to stratify risk in patients with non-conclusive symptoms or with etiological relationship, constituting a central tool for a comprehensive functional characterization of HCM, by allowing a dynamic evaluation of the interaction between heart rate, "systolic volume" and ventilatory efficiency, beyond resting findings.[11] Recent data show a significant correlation between oxygen pulse (O2P) and systolic volume in exercise, allowing to assess cardiac reserve in these patients. The combined identification of chronotropic incompetence (peak HR <80% of the expected value) and O2P reduction (<10% of the expected value), integrated in a new proposed classification, called RoMa, allowed to discriminate hemodynamic profiles with progressive deterioration of peak VO2, increase in the VE/VCO2 slope and less increase in systolic volume and cardiac output, regardless of ejection fraction or the obstructive gradient in rest.

From a practical view, CPET quantifies functional capacity, provides information on the limitation to exercise by differentiating predominantly chronotropic compromise, of systolic reserve or combined, which is particularly relevant for risk stratification, decision making on sports eligibility and therapeutic monitoring in HCM.[14]

Only 1.5% of people with hypertrophic cardiomyopathy (HCM) present a max VO2 exceeding the expected values, and no people have been detected with HCM whose max VO2 is 20% more than the expected maximum or 45 ml/kg/min (absolute value) in bicycle ergometer.[11]

In contrast, elite athletes with borderline left ventricular hypertrophy compared to athletes with borderline cardiomyopathy present max VO2 values in a 55 to 70 ml/kg/min range, exceeding the values expected in up to 50%. A VO2 of 50 ml/kg/min, or 20% of the maximum expected value, allows to completely differentiate both groups.[15]

Doppler echocardiography, including advanced techniques and stress echocardiography

Echocardiogram plays an essential role in diagnosis, prognostic assessment and decision making in patients with HCM. A clear sample of the value of transthoracic value is the fact that prognostic factors can be identified:

- Severe ventricular hypertrophy (maximum septal thickness ≥30 mm).[1,5]
- Left ventricular apical aneurysm.[1,5]
- Left ventricular systolic dysfunction (ejection fraction ≤50%).[1,5]
- Untreated significant left ventricular outflow tract obstruction.[5]
- Extensive myocardial fibrosis in cardiac MRI, defined as late gadolinium enhancement ≥15% of the left ventricular mass, associated to greater arrhythmic risk.[5]

The images obtained in rest and during stress provide complete and supplementary information, which allows to clarify the mechanisms responsible for the obstruction and the symptoms.

Transthoracic echocardiogram (TTE) constitutes the primary imaging modality in most patients, while transesphageal echocardiogram (TEE), MRI and cardiac CT provide supplementary information, particularly in selected patients when TTE is inconclusive. In comparison with cardiac MRI, echocardiogram does not diagnose 6% of HCM cases, underestimates maximum thickness in 20% and presents limitations in visualization of segmental apical, anterolateral or inferoseptal hypertrophy.

TTE should be performed as part of the initial evaluation of all patients with suspicion of HCM to establish a definitive diagnosis and, once confirmed, repeat every 1 to 2 years in the absence of clinical changes. It is, along with rest ECG, the main tool for screening first-degree relatives, both in the initial evaluation and in the periodical follow-up of genotype positive-phenotype negative individuals.

Speckle tracking techniques provide supplementary functional information, allowing to detect subclinical dysfunction and being associated to a greater load of fibrosis and arrhythmic risk.[16]

Myocardial longitudinal velocities and strain parameters (strain and strain rate), derived from tissue Doppler or speckle tracking, are usually reduced in the site of hypertrophy, as evidence of early systolic dysfunction, even before LVEF deterioration.

A decreased septal longitudinal and regional deformation (greater than -10%) is associated with greater susceptibility to ventricular arrhythmias, and may be altered before the development of septal thickness increase in relatives carriers of genetic variants.[17] A greater deterioration of longitudinal strain correlates with a greater degree of fibrosis and with the presence of ventricular arrhythmias.[18]

Stress echocardiography allows to identify dynamic gradients, inappropriate contractile response and elevation of pulmonary pressures induced by exercise, being key for clinical decisions in a context of sports.[19,20] These techniques should be interpreted in an integrated manner with conventional echocardiography and MRI.

GENETICS

Genetics plays an important role in the evaluation of sportspeople with HCM, as a supplementary diagnostic tool and as part of risk stratification.[5] The identification of pathogenic variants in sarcomere genes, mainly MYH7 and MYBPC, confirms the primary nature of hypertrophy and allows to differentiate HCM from the physiological remodeling of "athlete's heart" with more accuracy; particularly in borderline scenarios of mild septal hypertrophy.[21,22]

In the sports field, this differentiation has direct implications on eligibility and the type of activity allowed. Likewise, in genotype positive-phenotype negative individuals, a molecular finding demands a structured longitudinal monitoring, as high intensity exercise may influence penetrance and phenotypical expression in predisposed individuals. From a practical point of view, integrating genetic information with family history of sudden cardiac death, arrhythmic load, presence of fibrosis in MRI and hemodynamic response to exercise allows to make decisions individually and shared in regard to training and competition. Furthermore, the genetic study enables cascade familial screening, a particularly relevant aspect in families with physically active young people.[5]

RISK STRATIFICATION OF SUDDEN CARDIAC DEATH

Risk stratification should integrate clinical, electrophysiological (NSVT) cardiac imaging variables.

High risk markers include:

- Familial history of sudden cardiac death;
- Previous cardiac arrest;
- Sustained VT;
- Arrhythmic syncope;
- Severe hypertrophy (≥30 mm);
- Apical aneurysm;
- LVEF ≤50%;
- Untreated significant obstruction;
- Extensive fibrosis (≥15%);
- Repetitive non-sustained VT and high load of premature ventricular contractions.

In the presence of these markers, decisions should be cautious and guided by specialized teams.[1,2,3,4,5,23]

PARTICIPATION IN SPORTS

Mild to moderate recreational exercise should be recommended to all patients with HCM.

Participation in competitive sports should be evaluated by SDM, considering risk profile, type of sports, preferences of sportspeople and availability of cardioprotected environments.

For vigorous activities or competitive sports, participation should be considered reasonable in selected patients after an evaluation and prognostic stratification, under a model of SDM with Sports Cardiology experts; particularly in those with no high risk factors as those mentioned previously.

Evaluation should be comprehensive, including thorough clinical history, cardiac imaging tests (echocardiogram, MRI), stress test, and when indicated, genetic studies that may clarify specific situations, not just in sportspeople, but also in those who are not.[1,2,3,4,23,24,25]

In young sportspeople, it is essential to carry out yearly reevaluations, and a longitudinal monitoring, given the possible phenotypical progression and variable penetrance of the disease.

The specific recommendations for the practice of sports in young sportspeople with HCM can be summarized in the following:

- Recreational exercise of mild to moderate intensity recommended for all patients with HCM, as it improves functional class and quality of life, without increasing the risk of arrhythmic events.[1,3,24]
- Participation in competitive sports is reasonable in genotype positive by phenotype negative individuals; that is to say, carriers of pathogenic mutations with no evidence of ventricular hypertrophy or symptoms, after expert evaluation and regular follow-up.[1,4,23,26]
- In phenotype positive patients (with ventricular hypertrophy), the participation in competitive sports can be considered after an expert evaluation, SDM and risk management. This includes optimization of treatment, access to automated external defibrillators (AED) and establishment of an emergency plan in the sports environment. In cases of significant obstruction, it is recommended to treat the obstruction before returning to sports activity.[1,2,4,23]
- Universal restriction of vigorous physical activity is not recommended, and neither is the placement of an ICD just to allow the practice of sports. ICDs should be implanted only if patients meet standard criteria of high risk, regardless of the intention to play sports.[1,23,26]

There are limitations and uncertainty areas, particularly in children and teenagers, where prospective data is scarce and phenotypical penetrance may vary with age. Evidence about safety in competitive sports for patients in high risk is limited, so an individualized evaluation is required, as well as more studies in this group.[3,23]

In brief, the practice of sports in young people with hypertrophic cardiomyopathy should be individualized, based on risk stratification, periodical reevaluation and SDM, allowing participation in most cases, particularly in the absence of high risk factors and after an appropriate management of the disease.

CONSIDERATIONS IN REGARD TO THE TYPE OF SPORTS

There are differences according to the type of sports. The guidelines from the American College of Cardiology and the American Heart Association acknowledge that not all sports entail the same risk of young people with HCM.[1,4] Recommendations should adjust to the type of sports and the conditions in which they are carried out, contact, hydration state, altitude, etc., very common in adventure and ultra-endurance sports.

It is important to provide advice in terms of the sports supplements allowed and a safe use of them in the sports field, avoiding use of anabolic steroids, stimulants with caffeine, and all types of substances that stimulate the sympathetic system, due to the risk of arrhythmias and increase in obstructive gradients.

For this reason, in these aspects, specialists should know about types of competitions and the conditions in which the activity will be developed by the patient/sportsperson.

In summary, there are indeed differences in the recommendations for young sportspeople with HCM according to the type of sports they practice. High intensity sports, contact sports, or the risk of dehydration require more caution and an individualized assessment, while low intensity sports are usually safer. Participation should be based on risk stratification, the type of sports and what are the goals of the practice (competitive or recreational with low intensity). SDM with longitudinal follow-up, multidisciplinary suggestions and optimization of the treatment are key in the success of advising patients/sportspeople.[1,4,23,24]

CONCLUSIONS

Based on what was presented here, we can state that sports practice in patients with HCM should be individualized, based on risk stratification, periodical reevaluation and SDM, allowing participation in most cases, particularly in the absence of high risk factors and after an adequate management of the disease, including the indication for ICD in selected cases, having as priority a safe practice in cardioprotected environments, with staff trained in basic CPR and early access to defibrillators.

Physical activity and sports in HCM should be approached from a modern, individualized and non-restrictive view, based on risk stratification, periodical evaluation and SDM with the patient. Participation in sports is possible in most cases when evaluations and follow-up are made by specialists in Sports Cardiology.

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Clinical Case

Myocardial infarction in a patient with anomalous origin of the right coronary artery.

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ABSTRACT

Congenital coronary artery malformations affect between 1% and 5.6% of the general population and include several conditions characterized by the abnormal origin or course of any of the three main epicardial coronary arteries.

We describe the case of a 62-year-old female patient with a history of hypertension, diabetes mellitus, and obesity, who presented to the emergency department with chest pain. An inferior myocardial infarction was found.

Coronary angiography revealed an intermediate lesion in the left anterior descending artery, as well as an anomalous origin of the right coronary artery (RCA) from the left main coronary artery, with a total occlusive lesion in the mid-segment of the RCA. Percutaneous transluminal coronary angioplasty with placement of a drug-eluting stent (3.0 x 30 mm) in the RCA was performed, resulting in successful restoration of coronary flow.

Anomalous coronary artery origins complicate emergency angioplasty and require tailored strategies to achieve revascularization. Using multimodal imaging could guide angioplasty and minimize complications and mortality.

INTRODUCTION

Congenital malformations of coronary arteries affect between 1 and 5.6% of the general population, and include several diseases characterized by an abnormal origin or trajectory of any of the 3 main epicardial coronary arteries.[1] The anomalous origin of the opposite sinus is the most relevant clinically, being more frequent in the anterior descending artery and the circumflex artery; while the origin of the right coronary artery (RCA) from the left Valsalva sinus represents just 0.1%.[2]

Specifically, the anomalous origin of the RCA has been associated with ischemia, acute myocardial infarction (AMI) and sudden cardiac death of cardiac origin in young patients.[3,4] The prevalence of this anomaly ranges from 0.026% to 0.92% in the series analyzed from the registries of invasive and noninvasive coronary angiography.[5] The physiological hypotheses that explain these phenomena include a mechanical compression of the artery by the great vessels, due to an anomalous trajectory between the aorta and the pulmonary artery, or a collapse during exercise at the point where the anomalous RCA meets with the left coronary sinus.[6] It currently represents a true therapeutic challenge, with worse outcome due to its anatomical characteristics, such as ostium diameter and an acute start angle.

The anatomical conditions proper of these coronary anomalies and the effect of cardiovascular risk factors may increase the occurrence of acute coronary events, when compared to the general population.[7] In spite of its relationship with sudden cardiac death; the presence of anomalous coronary origin has not been associated with a greater risk of major adverse cardiac events.[8] It is imperative to carry out research to approach the evolution and prognosis of these anomalies in populations with an elevated cardiovascular risk. The goal of this work is presenting a clinical case that related the occurrence of acute myocardial infarction with the presence of an anomaly in the origin of the RCA.

CLINICAL CASE

Female, 62-year-old patient, with personal history of hypertension, diabetes mellitus and obesity. She came to the ER reporting very intense precordial pain radiating to the left arm, accompanied by nausea and feeling of imminent death, so she was admitted in a rural hospital. During her hospital stay, elevated values of troponin I, were observed, as well as electrocardiographic alterations compatible with inferior wall AMI (myocardial necrosis and negative T waves in leads DII, DIII and aVF) *(Figure 1)*. Before this situation, the patient was moved to a secondary level hospital, and percutaneous coronary interventionism was performed.

The results from coronary angiography show intermediate lesion of anterior descending artery, as well as anomalous origin of RCA from the left main coronary artery, with total occlusive lesion in the middle segment of RCA *(Figure 2)*. Percutaneous transluminal angioplasty was carried out, with placement of drug-

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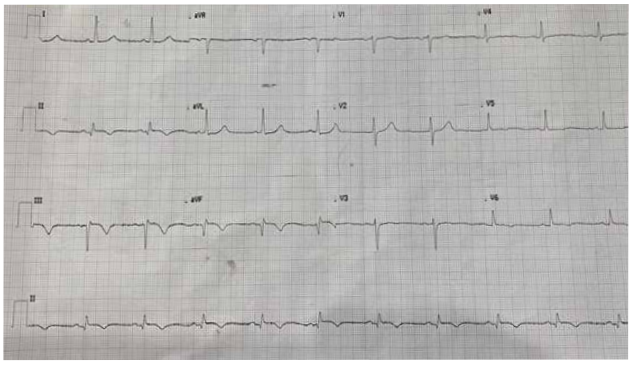

FIGURE 1.
Twelve-lead electrocardiogram
ST segment elevation of <1 mm in DII, DII and aVF with pathological Q waves and symmetrical negative T waves.

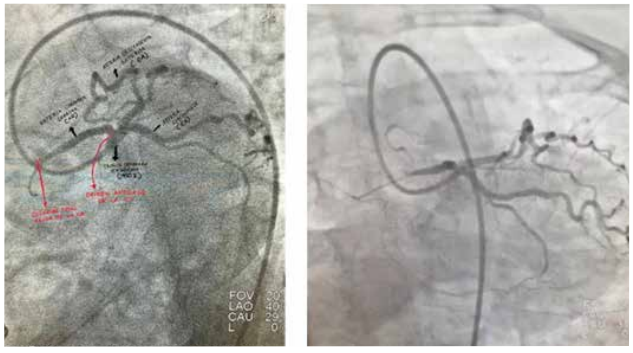

FIGURE 2.
Anomalous origin of RCA with terminal branch of the left coronary artery, with total occlusion in its middle segment.
a) 40° LAO view and 30° caudal view; b) 30° caudal view.

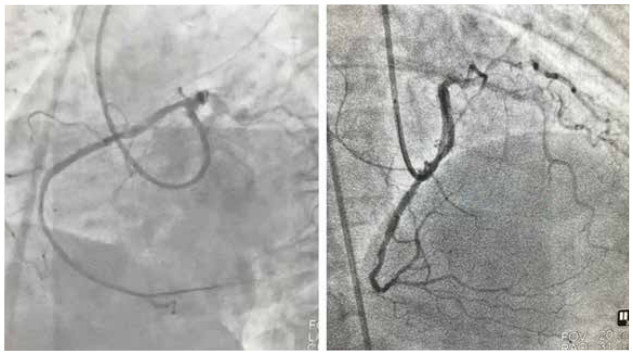

FIGURE 3.
Successful revascularization of the RCA with coronary flow restoration.
a) 45° LAO view; b) 30° cranial view and 30° RAO.

eluting stent (3.0 x 30 mm) in the RCA, obtaining a successful result in coronary flow restoration *(Figure 3)*. Subsequently, the patient was moved to the Intensive Care Unit to continue with the pharmacological treatment and to guarantee an appropriate hemodynamic monitoring.

During the second day of hospitalization, she evolved favorably, with elevated values of troponins (0.48 ng/ml), creatinine (0.73 mg/dl), and the rest of auxiliary tests within normal ranges. In echocardiogram, regional motion alterations were verified in the territory of the inferior and inferobasal wall. After 7 days of hospitalization, she presented a favorable course and she was discharged.

DISCUSSION

The anomalous origin of the RCA is a rare congenital malformation, first described in 1948 by White and Edwards.[9] The prevalence in the white population, according to autopsy studies, is 0.026%; however, in other populations it may be significantly higher.[10,11] In a recent investigation of 10,928 patients referred to CT, 28 cases with anomalous origin of RCA were identified, presenting interatrial trajectory; the average age was 55±17 years, while 64% were men, and 11 (39.3%) presented stable cardiac symptoms.[12]

These anomalies course asymptomatically in general, and their diagnosis in most cases is incidental. Nevertheless, a wide spectrum of cardiac symptoms has been associated with these congenital anomalies, including myocardial ischemia and sudden cardiac death.[13] The patients presenting ischemia, usually do it before the fifth decade of life, and the degree of elevation of troponin is very variable.[14] As it occurred with our patient, diagnosis was made by coronary angiography made after an acute coronary event, and at an age similar to that reflected in the consulted bibliography. A recent investigation has documented myocardial ischemia in patients with anomalous aortic origin of the RCA, even asymptomatic or with minimal symptoms.[15]

A single coronary artery is a rare anomaly that could be associated to acute coronary events like AMI. Antunes et al. documented the occurrence of type 2 AMI in a 22-year-old patient, secondary to cocaine consumption.[16] Likewise, more recent investigations have shown the presence of occlusive plaques in patients with AMI and anomalous origin of the coronary arteries.[17,18] In acute coronary syndromes, these anomalies complicate their management as they present challenges during percutaneous coronary intervention (PCI), such as an acute angle and intramural and intraarterial trajectory between the great vessels; hence, fibrinolytic treatment constitutes a feasible alternative. Currently, a few reports of cases of successful PCIs for these anomalies have been reported in patients presenting AMI; this is why this presentation is significant. However, in some given patients with intraarterial trajectory of the anomalous coronary artery, surgical unroofing of the right coronary ostium could be a reasonable alternative for these patients.[14,19]

CONCLUSIONS

The anomalous origin of the coronary arteries complicates emergency angioplasty and requires adjusted strategies to achieve revascularization. Using multimodal images helps with risk evaluation and to differentiate between malignant and benign trajectories. An early identification of this anomaly may guide PCI and minimize complications and mortality.

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Clinical Case

Reverse Takotsubo cardiomyopathy in a pediatric patient: atypical presentation of reversible ventricular dysfunction.

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cardiomyopathy.

ABSTRACT

Takotsubo syndrome (TTS) is a transient cardiomyopathy that mimics acute myocardial infarction in the absence of obstructive coronary artery disease. Its "reverse" form, characterized by basal systolic dysfunction with apical preservation, is rare in pediatric patients but may result in significant morbidity and even mortality. We report the case of a 9-year-old boy with reverse TTS who was admitted due to severe hypertension, bradycardia, and acute neurological deterioration with decreased alertness. Echocardiographic and magnetic resonance imaging tests confirmed the basal pattern of myocardial dysfunction, with complete recovery within 20 days. This case underscores the need to consider reverse TTS in the differential diagnosis of acute pediatric heart failure, particularly in the setting of possible adrenergic crises, and highlights the importance of early diagnosis and multidisciplinary follow-up.

INTRODUCTION

Takotsubo syndrome (TTS), also called stress cardiomyopathy, is a transient myocardial dysfunction resembling acute coronary syndrome, but with no evidence of obstructive CAD, and is characterized by transient systolic dysfunction of the left ventricle with anomaly of parietal motion.[1] Initially described in 1990 in Japan, its pathophysiology is still not fully understood, although the participation of a sudden increase in catecholamines is acknowledged as a central factor in its development, and can be associated to multiple emotional and physical triggers.[2]

Although TTS is more frequent in postmenopausal women, in the last two decades it has been reported in men, teenagers and children.[1,3] In the pediatric population, it is a rare entity but with a potential severe hemodynamic compromise and risk of associated mortality. In comparison to adults, pediatric patients present atypical pattern of ventricular involvement more frequently, as well as a higher proportion of affected males.[1,3]

The classical form represents 80% of cases and presents apical region hypokinesis.[2] The “inverse” or basal subtype of TTS represents a rare morphological pattern, characterized by hypokinesis of the basal and midventricular segments with apical preservation, and has been mainly linked to emotional, physical, neurological or endocrine stressors.[4,5] Adrenergic crises, as those induced by external stress or pheochromocytoma, are one of the main triggers of this subtype.[6,7]

Recent evidence suggests that the inverse subtype could be related to a differential distribution of β-adrenergic receptors in the myocardium, with the basal segments being more sensitive to sympathetic stimulation in young people.[4,8] Moreover, it has been proposed that transient myocardial inflammation would also contribute to the ventricular dysfunction observed in the acute phase of TTS.[9]

This report describes a case of TTS in a pediatric patient, emphasizing his clinical presentation, diagnostic evaluation and favorable evolution, with the aim of contributing to the scarce existing literature about this population.

CASE PRESENTATION

Male, 9-year-old patient, with history of erosive lichen planus, in treatment with weekly methotrexate. As relevant family history, myofibrillar myopathy stands out, secondary to amines deposits in grandfather and aunt, and presence of spontaneous bilateral carotid dissection in his mother, with good course.

The patient presented clinical symptoms characterized by nausea, sweating and fever episode. Within 24 hours, in a scenario of a family argument and a fight during an online videogame play, he evolved with intense headache and two episodes of syncope. Testing in the ER yielded the following upon admission: Glasgow score 10/15, blood pressure 185/145 mmHg, 40 bpm bradycardia, glycemia of 150 mg/dl and ECG with sinus bradycardia and negative T waves in V1, V2, V3. Two doses of atropine were administered with no clinical response, and due to acute neurological deterioration (Glasgow <8), orotracheal intubation was carried out; he was admitted in the pediatric ICU, and invasive monitoring of blood pressure was started. Sedoanalgesia was applied with fentanyl, as well as inotropic treatment.

The initial laboratory tests showed leukocytosis, with no other alterations. Blood, urine and cerebrospinal fluid cultures were negative, just as toxicological tests. He received empirical treatment with 3 doses of ceftriaxone. Infectious causes were

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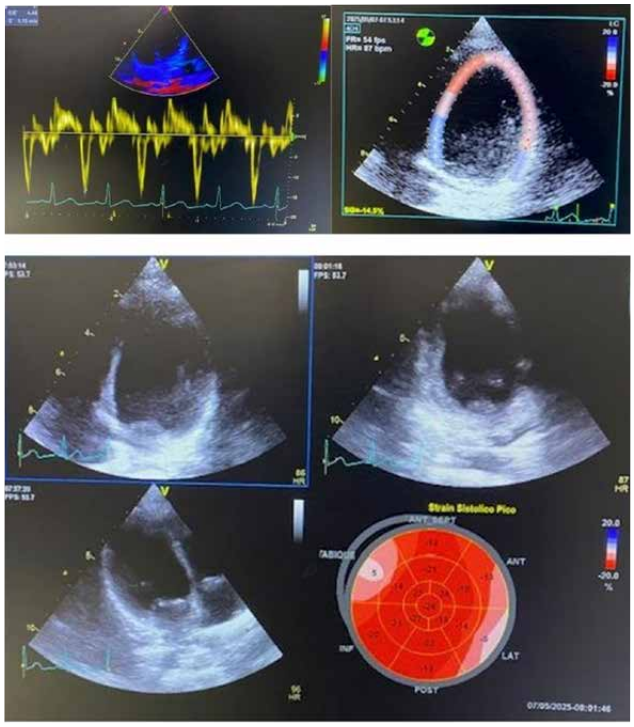

FIGURE 1.
Ecocardiograma control a las 24 hs

ruled out (negative viral serology), as well as endocrinological, neurological and nephrologic causes. CT and brain MRI were normal.

The initial echocardiogram showed a shortening fraction of 13% and ejection fraction of 28% in a context of global myocardial dysfunction, with troponin <2 ng/l and CPK MB of 22 U/l.

In control echocardiogram within 24 hours, he showed hypocontractility of basal sectors with preserved apical contractility (basal shortening fraction 18% and apical shortening fraction 29%), and normal origin of coronary arteries, with no heart valve diseases *(Figure 1)*. Cardiac MRI confirmed the pattern of basal hypocontractility with apical preservation, with no late enhancement or signs of myocarditis, findings compatible with inverse Takotsubo syndrome *(Figure 2)*.

The troponin curve showed a peak of 889 ng/l within 24 h after admission; ProBNP reached 2418 pg/ml within 36 h after admission; and normal electrocardiogram within 8 h after admission.

Twenty-four hours after admission, with clinical and hemodynamic improvement, scheduled extubation was achieved and sedoanalgesia was suspended. Inotropic support was suspended 48 h after admission, coinciding with progressive troponin decrease (31 ng/l) and ProBNP (465 pg/m). Echocardiogram before discharge showed recovery of ventricular function (baseline shortening fraction 21%, apical shortening fraction 26%).

The patient was discharged 6 days after admission, under treatment with enalapril to reduce postload, and outpatient follow-up through the Pediatric Cardiology Service. The patient presented normal catecholamines. Control 15 days after discharge presented echocardiogram with normalization of cardiac function (basal shortening fraction 36%, apical shortening fraction 43%) *(Figure 3)*.

DISCUSSION

Inverse Takotsubo cardiomyopathy represents an atypical form of presentation, with predominant basal compromise of the left ventricle and preserved apical contractility.[4] This pattern is more commonly observed in young patients, including teenagers and children, and has been linked to adrenergic crisis, acute neurostimulation or pheochromocytoma that could be triggered by emotional stress.[5,6,7]

In pediatrics, TTS is associated to more severity upon admission, with higher rates of cardiogenic shock, and lower ejection fractions in comparison to adults. A multicenter study showed that more than 50% of pediatric patients were males, contrasting with the predominantly female pattern in adults.[1] Furthermore, in pediatrics, physical triggers (neurological diseases, severe infections) were the main triggers in more than 90% of cases.[1,3] In this case, no clear stressor factor was identified, but it was associated to a context of family argument and virtual argument in a videogame.

As to pathophysiology, it is proposed that a massive discharge of catecholamines generates transient myocardial dysfunction through direct toxicity on the myocytes, coronary vasospasm,

FIGURE 2.
Imágenes de resonancia cardíaca

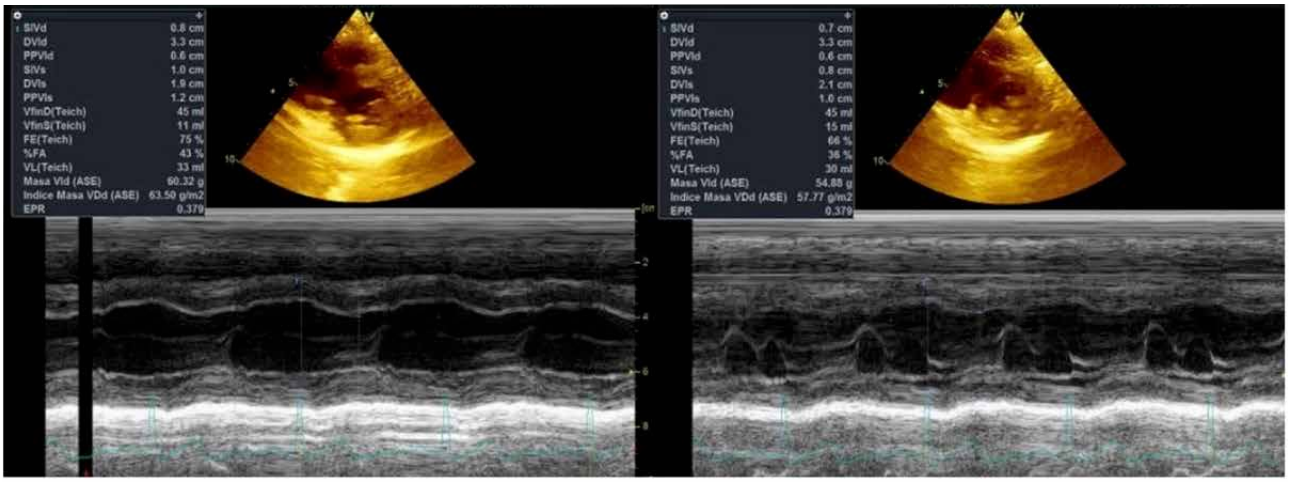

FIGURE 3.
Ecocardiograma a los 15 días.

microvascular dysfunction and adrenergic desensitization.[2,4,7] The adrenergic trafficking model explains how β2 overstimulation may induce a regional negative inotropic response, particularly in the segments with greater density of receptors, like the ventricular base in young individuals.[8]

Inverse TTS has been described in association with pheochromocytomas and catecholamine-secreting tumors, in which sympathetic hyperstimulation generates a characteristic distribution of ventricular dysfunction.[6,7,10] Yuan et al., reported a case of pheochromocytoma rupture with presentation like basal TTS, emphasizing the need to consider this pathology in differential diagnosis.[10]

On the other hand, the more recent studies have explored the role of systemic and myocardial inflammation. Scally et al showed that, by MRI and inflammatory markers, in the acute phase of TTS there is a significant inflammatory response, both local and systemic, that may contribute to ventricular dysfunction.[9]

The clinical presentation of inverse TTS may resemble other causes of acute heart failure, such as myocarditis, congenital heart diseases, or intoxication. The absence of coronary lesions, moderate elevation of biomarkers, echocardiography with regional patterns, and a rapid recovery of ventricular function guide the diagnosis of TTS.[2,4,11,12]

Management is essentially support treatment: therapy for heart failure and therapy with long-term beta blockers are recommended in selected scenarios.[4,11,13] Full recovery usually occurs within days or weeks, although in some cases recurrence has been documented, including the inverse subtype.[8,9] In this patient, ventricular function recovery took twenty days, and he remained medicated with enalapril for a month.

This case contributes to the growing literature on TTS in pediatrics, highlighting the significance of an early identification and multidisciplinary approach. In spite of its rarity, it should be included in the differential diagnosis of acute ventricular dysfunction in children, particularly in a scenario of adrenergic crises.

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Clinical Case

Ultrarapid recovery in Takotsubo Syndrome: a case report

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ABSTRACT

Takotsubo syndrome (TTS) is a transient cardiomyopathy characterized by acute ventricular dysfunction that mimics acute coronary syndrome, usually recovering within days to weeks. We present the case of a 45-year-old woman with cardiovascular risk factors who, after an emotional stress event, developed chest pain and ST-segment elevation, initially treated as acute myocardial infarction with thrombolysis without reperfusion. Coronary angiography revealed unobstructed epicardial arteries, and ventriculography showed the classic "apical ballooning" morphology. Remarkably, the patient achieved complete clinical, electrocardiographic, and echocardiographic recovery in less than 24 hours under conservative management. This outcome represents an unusually rapid course compared to the literature. Our report expands the clinical spectrum of TTS and highlights the importance of recognizing ultrarapid presentations, as well as the need for multicenter studies to identify predictors of accelerated recovery.

INTRODUCTION

Takotsubo syndrome (TTS), also called stress-induced cardiomyopathy or "broken heart syndrome", is a transient cardiomyopathy characterized by acute ventricular dysfunction, usually with apical hypokinesis and preserved basal contraction, which resembles an acute coronary syndrome. It usually presents as chest pain, electrocardiographic changes and elevated cardiac biomarkers, but without significant obstructive lesions in coronary arteries. Pathophysiology is mainly associated with intense catecholaminergic discharges, coronary vasospasm and microvascular dysfunction. It mainly affects postmenopausal women, and ventricular function recovery typically occurs within days or weeks. We present a clinical case of TTS of ultrafast resolution, enhancing the spectrum of classical presentation of this entity.

CLINICAL CASE PRESENTATION

Female patient, aged 45, with cardiovascular risk factors: sedentary life, menopause, smoking and overweight. She presented to a first-level hospital with clinical symptoms characterized by precordial pain of cardiac characteristics after a verbal discussion. Initially, she received treatment with nonsteroidal anti-inflammatory drugs (NSAIDS) with no improvement, so when symptoms persisted, she went to a second-level hospital center six hours after the onset of the pain. Upon admission, the electrocardiogram (ECG) showed ST segment elevation in leads DI, AVL, V5 and V6 *(Figure 1)*. Due to suspicion of ST-segment elevation acute myocardial infarction (STEMI), and the impossibility of performing primary coronary angiography within 120 minutes, thrombolysis was carried out with tenecteplase in a single 40 mg dose. Before the absence of indirect criteria for coronary reperfusion, the patient was referred to this hospital for rescue percutaneous coronary interventionism (PCI). In the center, besides corroborating the electrocardiographic findings, Troponin I elevation of 16 ng/ml was documented (TriageTrue, Quidel; cutoff point <1 ng/ml). Coronary angiography showed epicardial arteries without significant obstructive lesions. In the ventriculography, basal hypercontractility and apical hypokinesis became evident, with classical morphology of apical ballooning *(Figure 2)*. Thus, diagnosis of TTS was established, and the patient was admitted for clinical surveillance. During her stay she did not present new episodes of precordial pain, troponins decreased progressively, and ECG showed ST-segment resolution within just 16 hours *(Figure 3)*. The echocardiogram carried out 24 hours after coronary angiography showed left ventricle with no alterations in segmental motion, with preserved systolic function (ejection fraction 54.1% by Simpson's biplane method) and ventricular geometry with concentric remodeling, so the patient was discharged the following day with hygienic and diet measures and no other pharmacological treatment. Currently, she is in NYHA functional class I *(Figure 4)*.

DISCUSSION

TTS, also called stress-induced cardiomyopathy or "broken heart syndrome", is an increasingly identified entity in clinical practice. Although initially it was described in the Japanese population, currently there have been reports in different regions of the world, including Latin America. Its incidence is estimated to be 1-2% of patients admitted with suspicion of acute coronary syndrome (ACS), reflecting the significance of considering it in differential diagnosis.[1,2]

Clinically, TTS usually presents with chest pain, dyspnea or syncope, accompanied by electrocardiographic changes and mo-

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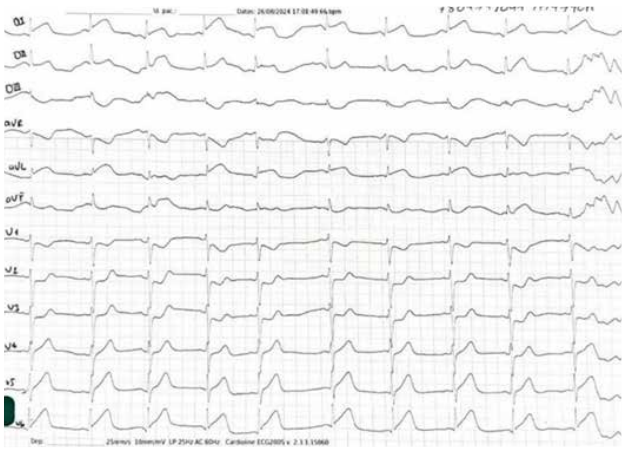

FIGURE 1.
Electrocardiography upon admission

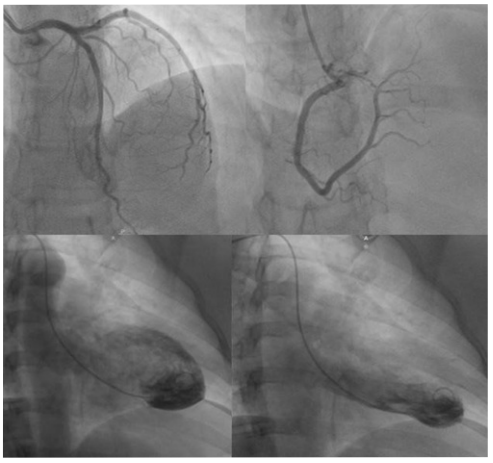

FIGURE 2.
Images obtained in ventriculography

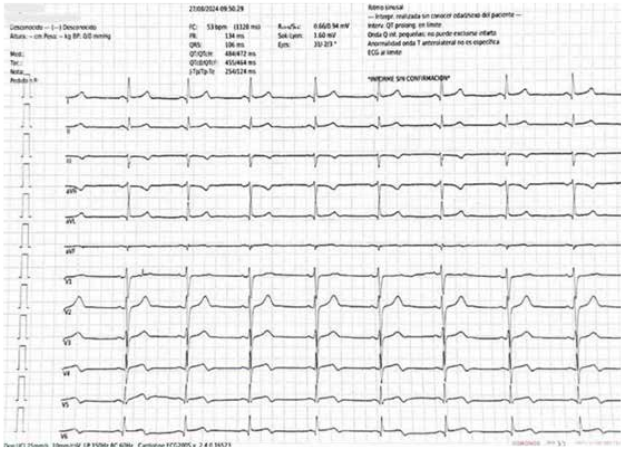

FIGURE 3.
Electrocardiogram 16 hours after admission

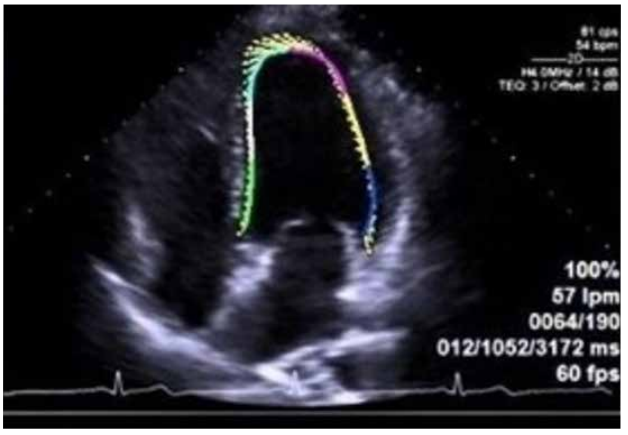

FIGURE 4.
Images of echocardiography 24 hours after admission

derate elevation in cardiac biomarkers, mimicking STEMI. In this regard, the absence of significant obstructive coronary lesions in angiography, together with transient ventricular alterations in ventriculography or echocardiography (generally the classical apical dyskinesia with basal hypercontractility), guide the diagnosis.[3,4]

Pathogenesis is still being debated. The most widely accepted hypothesis links excessive catecholaminergic discharge with the trigger of transient myocardial dysfunction, although mechanisms related to coronary vasospasm, ventricular dysfunction and genetic susceptibility have also been described.[5,6] Most cases occur in postmenopausal women, probably due to the loss of the protective effect of estrogens on the endothelium and adrenergic response.[7] This clinical case coincides with this epidemiological profile, but presents a remarkably atypical outcome.

In most reports, recovery occurs in a period from days to weeks, defining an early resolution when ventricular function improves within the first 10 days after the initial event, reporting cases of improvement in up to the first three days; however, in this patient, full resolution was documented within the first 24 hours under a conservative management, and only with clinical surveillance, representing a more rapid course than what is classically described.[8] This finding suggests that there may be subgroups of patients with transient myocardial dysfunction of ultrashort course, probably influenced by factors such as adrenergic stimulation magnitude, previous myocardial reserve, or mechanisms yet to be clarified.[9]

The global prognosis of TTS is usually favorable, although not exempt from severe complications like heart failure, cardiogenic shock, ventricular arrhythmias or free wall rupture, with reported in-hospital mortality of up to 5%.[10] In this regard, this case reinforces the significance of close monitoring in the cardiovascular care unit, even in patients with good clinical evolution, since variability in the natural course could be significant.

CONCLUSIONS

Takotsubo syndrome represents a diagnostic challenge due to its similarity with acute coronary syndrome. Although most ca-

ses show ventricular recovery within days or weeks, this patient presented a complete resolution in less than 24 hours under conservative management, constituting an unusually fast course. Reports like this one provide valuable information about the clinical range of TTS, particularly in the Latin American population, and highlight the need for multicenter studies to identify ultrafast recovery predictors in comparison with the usual course.

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