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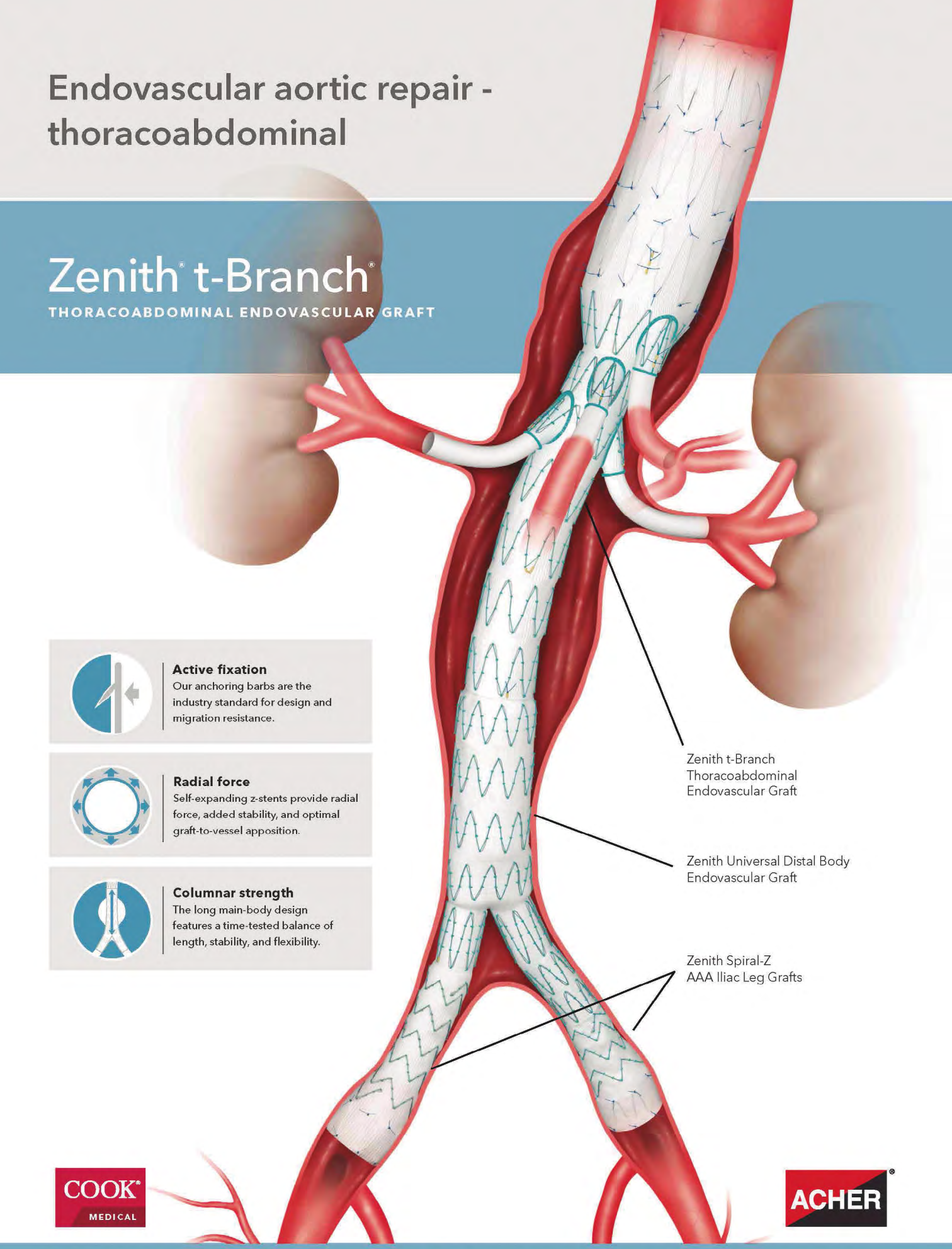

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SUMMARY

ORIGINAL ARTICLE

HEART TRANSPLANTATION IN PATIENTS WITH TYPE II DIABETES MELLITUS

ABSTRACT

Introduction: The risk of infections and failure of other organs can complicate the evolution of the diabetic patient who receives a heart transplant. The experience of a group of diabetic patients who received a heart transplant is presented.

Objective: To analyze the evolution, complications, and survival of diabetic patients receiving heart transplants.

Material and methods: Diabetic patients receiving heart transplants between October 1, 2010, and April 30, 2020, were selected. We analyzed age, sex, time of evolution with diabetes and treatment received, additional risk factors, presence of exacerbation of these complications, rejection reactions, post-transplant diabetes, infections, survival, and mortality and its causes.

Results: During the period analyzed, 181 heart transplants were performed; of these, 29 (16.02%) were performed in patients with diabetes mellitus. The predominant sex was male, and the indication for transplantation was ischemic cardiomyopathy; the time of evolution of diabetes was 10.2 years, and most patients were receiving treatment with oral hypoglycemic agents. As a cause of mortality, infections were predominant in the early and late postoperative stages, graft failure was predominant in the perioperative period, and graft vasculopathy was the cause of death after one year after transplantation. The highest number of deaths occurred in men (83.3%). The mean follow-up was 5.4 ± 2.95 years (range: 1-9 years).

Conclusion: When comparing the results with other series, we conclude that diabetic patients with end-stage heart failure can receive a heart transplant if there is strict metabolic control.

Keywords: *diabetes mellitus, heart transplantation, heart failure, graft vasculopathy, hypoglycemic agents, post-transplant diabetes.*

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INTRODUCTION

Diabetes mellitus is one of the most prevalent chronic metabolic disorders; the International Diabetes Federation predicts that by 2025, there will be 300 million diabetics worldwide[1-4].

In diabetic patients, cardiovascular pathology causes 65% of mortality and can present as ischemic heart disease, heart failure, cerebrovascular disease, and peripheral arterial disease[1,2]. In addition, people with diabetes are more susceptible to infections[5,6].

Most complications and 80% of deaths from atherosclerotic disease result from endothelial and vascular smooth muscle abnormalities, platelet dysfunction, vasoconstriction, and proliferative response at sites of injury.

The role that diabetes plays in heart transplant patients has not yet been uniformly defined[1,2]. There are case series in which no significant differences are observed in the mortality of diabetic and non-diabetic heart transplant patients[6,8]. However, it is known that heart transplant patients who present the three metabolic risk factors (systemic arterial hypertension, diabetes mellitus, and obesity) have a 63% increase in mortality. The results of heart transplantation as a treatment for end-stage heart failure in diabetic patients are shown here.

MATERIAL AND METHODS

The casuistry was taken from the High Specialty Medical Unit of the Mexican Social Security Institute, which, at the time of the review, had the most significant experience in heart transplantation in Mexico. Adult patients with type II diabetes mellitus with end-stage heart failure accepted and treated by heart transplantation between October 1, 2010, and April 30, 2020, were selected.

Age, sex, time of evolution of diabetes and its treatment, additional risk factors (systemic arterial hypertension, obesity, renal failure, retinopathy, peripheral vasculopathy), and, in the perioperative period, the presence or exacerbation of these complications and the presence of rejection reaction, post-transplant diabetes, infections, survival, and mortality and their causes were analyzed.

The preoperative evaluation considered the degree of target organ involvement. It included diabetic patients with retinopathy present (without legal blindness or previous ophthalmologic surgery), creatininemia of 2.0-2.5 mg/dL or proteinuria of 300-1000 mg/d, ankle-brachial index equal to 1 in any of the pelvic extremities, without pelvic limb amputation, and peripheral neuropathy or gastroparesis. Diabetic patients with heart failure and blindness or previous ophthalmologic surgery, creatininemia >2.5 mg/dL, proteinuria 1 g/d, history of toe or pelvic limb amputation, and symptomatic orthostasis were not included in the transplant program.

RESULTS

Between October 1, 2010, and April 30, 2020, 181 heart transplants were performed, of which 29 (16.02%) were performed in patients with diabetes mellitus. In this group, the male sex predominated and, as the etiology of terminal heart failure, ischemic cardiomyopathy. Although the range of time of evolution of the disease is vast, the average was 10.2 years; the great majority of patients were receiving treatment with oral hypoglycemic agents (*Table 1*).

Tables 2 and *3* present the causes of mortality and the period in which they occurred. It is possible to observe the predominance of infections that appeared early and late in the postoperative period. In contrast, graft failure was predominant in the perioperative period, and graft vasculopathy was the cause of death after one year after transplantation. The highest number of deaths occurred in men (83.3%).

The mean time the deaths occurred was 659.2 days ± 941.03, equivalent to 1.8 ± 2.57 years (range: 1-2719 days).

In the group of patients who continue to be followed up at 5.4 ± 2.95 years (range: 1-9 years), no significant difference was observed in the age at which they received heart transplantation, which was 53.6 ± 7.15 years (range: 43-62 years), and 66.6% were male. In addition to the immunosuppression scheme, for diabetes control, one of them only takes diet (11.1%), four receive oral hypoglycemic agents (44.4%), and the remaining four, insulin glargine (44.4%).

DISCUSSION

Although diabetic patients with heart transplants have a shorter survival than non-diabetic patients, with a late mortality that varies between 20 and 40%, and, in addition, this group of patients is more susceptible to renal failure and post-transplant infections, when stratified, the diabetic patient with less severe disease has a survival comparable to the non-diabetic patient with heart transplant[5,6,10,11]. In this same aspect, Megna et al. studied a group of 952 heart transplant patients between 2010 and 2018, of whom 28.78% were diabetic. When they separated them into two groups with and without adequate metabolic control, they did not observe significant differences in chronic vasculopathy, non-fatal major cardiac events, transplant rejection, need for dialysis, or infection at one year of follow-up. However, they did observe higher mortality at

Demographic data	Number and percentage (%) of patients
Males	22 (75.86)
Women	7 (24.13)
Age (years)	52,75 ± 7,05 (rango 40-65)
Ischemic cardiomyopathy	24 (82.75)
Dilated cardiomyopathy	5 (17.25)
Time of evolution of diabetes (years)	10,27 ± 5.92 (rango: 1-21)
Pretransplant treatment of diabetes	
Diet	1 (3.44)
Oral hypoglycemic agents	22 (75.8)
Insulin	6 (20.7)
Blood group	
O+	13 (44.8)
A+	12 (42.4)
B+	3 (10.3)
AB+	1 (3.4)

TABLE 1. Demographic characteristics of the patients, time of evolution, and preoperative treatment received.

Etiology	Number and percentage (%) of patients
Post cardiorespiratory encephalopathy	2 (10.52)
Pulmonary sepsis	7 (38.8)
Abdominal sepsis	2 (10.52)
Primary graft dysfunction	2 (10.52)
Chronic graft vasculopathy	4 (21.04)
COVID-19	1 (5.25)

TABLE 2. Causes of mortality in diabetic patients with heart transplantation.

Time after transplant (days)	Number and percentage (%) of patients
<30	4 (22.2)
30-90	3 (15.78)
90-180	0
180-365	2 (10.52)
>365	9 (47.36)

TABLE 3. Periods in which deaths occurred after heart transplantation in diabetic patients.

one year in the group with poor metabolic control[11]. This evolution in survival may be influenced by the fact that it has been observed that, in healthy hearts transplanted into diabetic patients, there is an early and progressive deposition of lipids in the myocardiocytes. This was shown by Marfella et al. in a study in which they found a significant difference in deposition (p = 0.019) when comparing three groups of patients: i) healthy patients (without lipid deposition), ii) diabetic patients treated with metformin, and iii) untreated diabetic patients who presented significant lipid deposition[12].

Evidence indicates that the diabetic patient with end-stage heart failure who has reasonable metabolic control may have a prognosis of recovery after heart transplantation equivalent to that of the patient with heart failure without diabetes, so the possibility of offering transplantation to diabetic patients should not be ruled out after careful selection[8,13]. On the other hand, perhaps patients with complicated diabetes should be included in the lists of high-risk patients, destination therapy, or other treatment options[9,10,13] since it has been observed that diabetic patients who receive support with ventricular support systems in the pre-transplant stage have a better evolution. Therefore, it would be a good option to leave mechanical circulatory support as destination therapy and not as a bridge to transplantation[14].

When comparing the results of our study, there is a lower percentage of diabetic patients in our series (16.02% versus 28.78%), 30-day mortality similar to that of other series (22.2% versus 28.6%), and mortality due to sepsis higher than in other series (50% versus 17.2%)[9,14]; therefore, these results could not be attributed to diabetes alone given the lower prevalence of the disease in our series. Undoubtedly, other perioperative and mid-term factors (surgery time, hemorrhage, duration of ventilatory support, and home care) have an influence.

On the other hand, the appearance of post-transplant diabetes has been observed in an average of 37% of patients (48% at six months, 36% at one year, 26% at two years, and 17% at three years). This condition implies a higher frequency of infections (80% versus 61%; p = 0.036) and lower survival at two years (16% versus 35%, p = 0.046), with a greater possibility of acute cellular rejection[15].

Based on these findings, we conclude that it is feasible to perform heart transplantation in patients with diabetes mellitus after a comprehensive evaluation and very clear selection criteria, accompanied by strict metabolic control before and after transplantation. It is also necessary to keep in mind other treatment options for heart failure in this type of patient[9].

Declarations

The authors declare no conflict of interest.

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REVIEW ARTICLE

SYSTOLIC ANTERIOR MOTION OF THE MITRAL VALVE AFTER SURGICAL REPAIR: RISK FACTORS, DIAGNOSIS, AND MANAGEMENT ALGORITHM

ABSTRACT

The exponential growth of mitral repair surgery has transformed the prognosis of patients with severe mitral regurgitation. All expert consensus and current clinical guidelines recognize the preponderance of mitral reconstruction. The incidence of complications inherent to more conservative repair techniques, such as systolic anterior motion (SAM), has also increased. The mechanisms of SAM are complex and depend directly on the characteristics of the mitral apparatus after plasty and, of course, on the hemodynamic state of the left ventricle. In this context, echocardiography has an important diagnostic and prognostic role. Approximately 90% of cases of SAM can be resolved conservatively; therefore, an orderly multidisciplinary approach is critical in understanding the substrate of the problem and knowing when and how to intervene. A simple algorithm for the management of perioperative MAS is presented below.

Keywords: *mitral repair surgery, severe mitral regurgitation, systolic anterior motion.*

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INTRODUCTION

At the height of the boom in mitral reconstruction and more conservative techniques based on the exclusive use of neo-strings, perioperative SAM has been reported in up to 15% of patients[1]. Moreover, according to the latest publications, the medium and long-term incidence may be as high as 7%[2]. SAM rarely occurs in patients with degenerative disease, whatever the size of the leaflets or the degree of myxomatous degeneration. For this reason, the occurrence of MAR after reconstructive plasty is considered an iatrogenic phenomenon[3].

The degree of SAM after reconstructive surgery depends directly on the characteristics of the mitral valve apparatus, which includes the annulus, leaflets, chordae tendineae, papillary muscles, and left ventricle. The hemodynamic status of the latter is critical to the onset, assessment, evolution, and treatment of SAM. The onset of SAM is typically associated with mitral insufficiency and LVOT obstruction, which, without treatment, can lead to acute pulmonary edema, heart failure, and sudden death. Despite multiple techniques and surgical maneuvers to avoid it, its incidence is still high[4]. Consequently, specialists involved in the post-surgical care of patients after mitral plasty must have the capacity to manage this complication.

Although most cases of SAM resolve with conservative management (medical and hemodynamic treatment), about 10% of cases may require early reintervention[5]. Furthermore, the cardiovascular specialist should not only be comfortable with SAM's diagnosis and essential medical management but also with risk analysis and coordination of an orderly multidisciplinary approach to understand the substrate of the problem and know when and how to intervene. A simple algorithm based on echocardiographic calculations and hemodynamic findings for the perioperative management of SAM may be especially valuable for managing any patient presenting with this complication.

DEFINITION

SAM is defined as the displacement of the distal portion of the anterior leaflet of the mitral valve into the left ventricular outflow tract (LVOT) in systole. The main trigger for SAM after mitral reconstruction is the mismatch between the annulus dimension (annuloplasty-induced alteration) and the resulting amount of leaflet tissue (directly proportional to the repair techniques employed). Although several potential mechanisms have been studied and described, the Venturi effect (pull) and the drag phenomenon (push) are among the predominant ones. In this context, both mechanisms describe the displacement of the anterior leaflet into the LVOT with consequent interruption of outflow[6].

As the left ventricle contracts and expels blood into the aorta through the outflow tract, it pulls and pushes the anterior leaflet tissue into the LVOT, interrupting the laminar flow. The greater the presence of tissue below the coaptation line (subannular tissue or coaptation surface), the greater the push or pull effect. Likewise, turbulence in the LVOT causes the Venturi effect of the anterior leaflet and mitral insufficiency. Undoubtedly, the drag phenomenon plays a much more critical role than the Venturi effect, significantly the closer the coaptation surface is to the LVOT[7].

There are several risk factors for the development of SAM. These can be divided into clinical or anatomical factors specific to the patient, hemodynamic factors, and technical factors related to the repair (*Table 1*). Early recognition of MAR as a significant cause of hypotension during the perioperative period is critical, as many routine measures to increase blood pressure significantly aggravate MAR.

ECHOCARDIOGRAPHIC CHARACTERIZATION

Echocardiography is still relevant during perioperative anamnesis of the functional anatomy of a dynamic structure such as the mitral valve. In addition, handheld echocardiography has revolutionized the care of unstable patients and, of course, the management of patients with perioperative MAS. Accurate knowledge of the anatomical characteristics of valve lesions and their hemodynamic impact is essential to proceed with an individualized therapeutic plan for each patient[8].

More than four decades ago, based on the echocardiographic motion of the leaflets, Carpentier established the pathophysiological triad of mitral pathology for an orderly and systematic approach to the disease[9]. The triad highlights the importance of differentiating between the disease that leads to insufficiency or stenosis (etiology), the resulting lesions, and how these lesions affect leaflet mobility, i.e., the type of dysfunction they cause. Over time, cardiovascular specialists have adopted this triad or classification, and today. However, its use still needs to be more generalized and widespread; it is conducive to a perfect understanding among cardiologists, cardiac imaging specialists, and surgeons (*Figure 1*).

Clinical	• Age (<60 years) • Male sex • Advanced degenerative disease (severe myxomatosis) • Prolapse of both leaflets • Elongated anterior leaflet • Anterior coaptation line: anterior:posterior ratio <1.3 mm • Limited end-systolic diameter (small ventricles) • Hypertrophic obstructive cardiomyopathy (HOCM) • Reduced mitroaortic angulation (<20°) • Abnormalities of the subvalvular apparatus (anomalous chordal insertion)
Hemodynamic	• Hypercontractility (hyperdynamic ventricle or tachycardia) • Limited preload (short ventricular filling time or hypovolemia) • Limited afterload (hypotension)
Technical	• Excess residual posterior leaflet (anterior coaptation line) • Excess coaptation surface area (>10 mm) • Exclusive use of neo-strings in unusually redundant leaflets • Wrong prosthetic ring size (too small) • Untreated underlying HOCM during the procedure

TABLE 1. Risk factors for systolic anterior motion.

The classification of the different mitral dysfunctions is based on the position of the leaflet margins in relation to the plane of the mitral annulus:

- Type I dysfunction: normal mobility of the leaflets with severe annular dilatation resulting in a central regurgitant jet, perforation, or clefting of one of the leaflets.
- Type II dysfunction: excessive mobility of the leaflets, usually secondary to pathological elongation or rupture of the chordae tendineae. In this case, the regurgitant jet is directed to the opposite side of the affected leaflet.
- Type III dysfunction: restricted mobility due to retraction of the subvalvular apparatus (frequent in rheumatic disease or inflammatory processes) (IIIA) or displacement of the papillary muscle (ischemic remodeling or dilated cardiomyopathy) causing apical displacement (ventriculization or tethering) of the leaflet (IIIB). The jet is directed to the same side of the affected leaflet.

The original classification of the pathophysiological triad included only preoperative dysfunctions. However, if we include dysfunctions secondary to other clinical identities (e.g., hypertrophic cardiomyopathy) or postoperative (e.g., SAM or dysfunction opposing postoperative ventricular failure), there are two more dysfunctions:

- Type IV dysfunction: anterior systolic motion due to hypertrophic cardiomyopathy, induced by hemodynamic disturbances or after mitral repair. A closing jet or leaflet snapping jet of erratic direction is produced in this case.
- Type V dysfunction: hybrid conditions such as posterior prolapse due to anterior valve thrust (a particular form of SAM), opposite dysfunction, anterior pseudo prolapse, or advanced pathologies due to endocarditis[10].

In expert hands, systematic examination of the mitral valve by echocardiography should provide generalized (anterior, posterior, or bivalve pathology) and segmented information on both leaflets (analysis of each segment and commissure), identify excess or scarcity of tissue in the leaflets, evaluate annular dimensions, detail the state of the subvalvular apparatus and estimate ventricular resistance. As for the specific evaluation of the SAM, the parasternal view is of choice. The parasternal plane (long axis), if transthoracic echocardiography is used or the sagittal view if the transesophageal probe is rotated to 120°, cuts perpendicularly across the coaptation line, crossing P2 (on the left of the image) and A2 (on the right of the image). This view is relevant, especially in evaluating the SAM, as the coaptation surface and extent are clearly visible[10]. Additionally, this view allows us to evaluate the annular surface, extrapolating the annular diameter with the anterior leaflet surface.

TYPE I	Normal leaflet mobility	Annular dilatation (without ventriculization) Perforation of leaflets Cleavage of leaflets	Atrial fibrillation Ischemic cardiomyopathy/dilatation of atrial fibrillation Endocarditis ("windsock" deformity) Congenital pathologies
TYPE II	Excessive velocity mobility	"Billowing" findings Localized prolapse Prolapse of both leaflets	Degenerative disease* Endocarditis Rheumatic disease Trauma Ischemic cardiomyopathy Marfan/Ehler-Danlos diseases
TYPE IIIA	Restricted leaflet mobility	Systolic and diastolic restriction	Rheumatic disease Carcinoid syndrome Radiotherapy Systemic lupus erythematosus Mucopolysaccharidosis Hypereosinophilia
TYPE IIIB	Restricted leaflet mobility	Symmetric systolic restriction Asymmetric systolic restriction Annular dilatation (with ventriculization)	Ischemic/dilated cardiomyopathy
TYPE IV	Anterior systolic motion	After mitral repair Hemodynamic induction Hypertrophic cardiomyopathy	Hypertrophic obstructive cardiomyopathy Hypovolemia Inotropic stimulation Tachycardia (arrhythmias)
TYPE V	Opposite dysfunction	Anterior pseudoprolapse** Postoperative ventricular failure Posterior prolapse due to SAM Untreated intrinsic pathology Severe endocarditis	Barlow's disease Ischemic/dilated cardiomyopathy Hypovolemia Inotropic stimulation Tachycardia (arrhythmias) Infectious process

FIGURE 1. Echocardiographic classification of mitral pathology.
SAM: Systolic anterior motion.
*Degenerative disease includes fibroelastic deficiency, advanced fibroelastic deficiency, forme frustre forms, and Barlow's disease.
**Pseudoprolapse: defines the echocardiographic image of anterior leaflet prolapse due to posterior leaflet lesions (posterior leaflet repair is sufficient for resolution).
Adapted from: Castillo & Adams, Valvular Heart Disease: A Companion to Braunwald's Heart Disease, Chapter 19:370, 5th Edition, 2020.

Echocardiographic evaluation of SAM should be performed in the presence of a full ventricle (maximized end-systolic diameter after optimization of preload and afterload) and normodynamics (adequacy of heart rate and ventricular contractility). Although the surgeon assesses the position of the coaptation line during the saline test (as well as the height of the coaptation surface using ink), the line can also be assessed by three-dimensional echocardiography (glass view). The remaining structural and geometric variables to be collated include: a) the ratio between the anterior and posterior leaflet height; b) changes in annular diameter; c) the distance between the coaptation line and the interventricular septum; d) the end-systolic and end-diastolic diameters; and e) the thickness of the interventricular septum (if addressed during surgery).

Any SAM phenomena should be characterized and documented, including LVOT gradient (ideally ≤ 30 mmHg) and the degree of turbulent flow and mitral insufficiency[11].

PERIOPERATIVE MANAGEMENT OF SYSTOLIC ANTERIOR MOTION

With any threat or suspicion of SAM after mitral repair, the checklist of preventive maneuvers should be reviewed, which includes: (a) posterior leaflet height ≤ 1.5 cm, precisely what an open surgical clamp measures, (b) appropriate ring or band size, and (c) dye saline test showing a posterior coaptation line (reflecting the distance between the anterior leaflet and interventricular septum) and a coaptation surface ≤10 mm (some experts are permissive up to 12 mm, the larger the coaptation surface, the greater the entrainment effect in systole) (*Figure 2*). Optimal management of SAM depends on two factors: identification and localization of the anatomical lesion responsible and echocardiographic calculation of its severity[12]. The first step in any algorithm should be conservative, consisting of medical therapy focused on ventricular filling. This will involve volume, immediate and short-acting beta-blockers (e.g., esmolol) and, in some cases, agents with selective alpha-adrenergic effect (e.g., phenylephrine)[13]. Arterial vasoconstriction is also accompanied by venous vasoconstriction. This increases blood pressure and reflex bradycardia, which will be needed in the presence of SAM. The potent arterial vasoconstriction increases systemic vascular resistance (increase in afterload). The overall result is a reduction in cardiac output.

Furthermore, the approach to SAM, whatever the patient's condition, should be a logical and orderly stepwise process. First, cessation of any inotropic medication is indicated to avoid ventricular hyper dynamism and to control the heart rate (the slower the rate, the longer the ventricular filling time, and, consequently, the larger the end-systolic diameter). We aim to bring the heart rate to 60 bpm. In an overlapping manner, we will administer volume to increase preload, maximize ventricular filling, and try to raise afterload as well. The aim is to achieve a mean arterial pressure ≥80 mmHg so that the increase in afterload hinders ventricular emptying to some extent and thus maintains the end-systolic diameter (agents with selective alpha-adrenergic effect can be introduced here, and some surgeons compress the aorta manually for seconds to recreate the scenario). If this fails, beta-blockers are also indicated to try to minimize the hyperdynamic state of the ventricle and the number of beats per minute (as tolerated or allowed by the mean arterial pressure)[14].

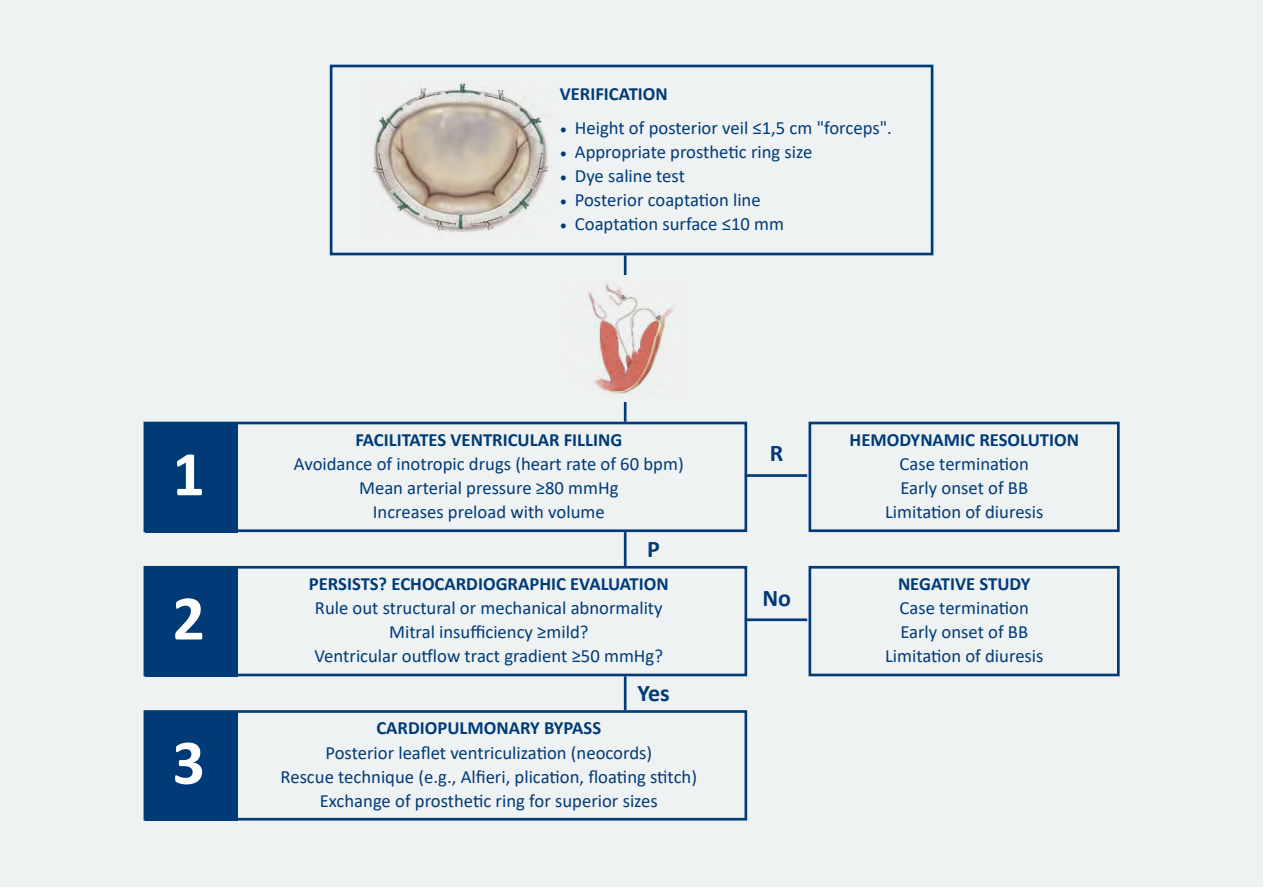

FIGURE 2. Intraoperative management algorithm.
BB: beta-blocker, P: persists, R: resolution.

Resolution of SAM after conservative management entails termination of the case, early initiation of beta-blocker, and, of course, limitation of diuresis (or controlled administration adjusted to preoperative body weight). The persistence of SAM makes it necessary to perform a new echocardiographic study in search of underlying structural or mechanical abnormalities and to calculate the degree of mitral insufficiency and the gradient in the LVOT. If the regurgitation is mild or greater, or the gradient is ≥50 mmHg, we will proceed with a second bypass time for SAM correction. Furthermore, suppose there is a clear excess of tissue in either leaflet, whatever the response to hemodynamic interventions. In that case, the threshold will be too low to reestablish cardiopulmonary bypass and proceed with surgical correction[15]. This eliminates the possibility of SAM in the future. In young patients, especially those with asymptomatic mitral regurgitation, the threshold for surgical correction of SAM is equally low, regardless of the response to conservative treatment. There is a crucial axiom about MAR: if MAR was successfully eliminated with conservative management in the operating room, it could also be managed in the inpatient ward regardless of its severity.

The management of MAS generated in the inpatient ward during the postoperative stay can be much more complex and risky. However, even so, the conservative approach is always the initial one (*Figure 3*). If regurgitation is less than mild or the gradient is <50 mmHg, proceed to increase the dose of oral beta-blocker (as tolerated) and avoid the use of diuretics and antihypertensives if the systolic blood pressure is <135 mmHg. If there is improvement or normalization in the next 48 hours, the patient is discharged, and a follow-up echocardiogram will be performed in four weeks[16]. If there is no improvement, we should assemble a team of cardiovascular specialists to plan individualized management and reoperation (*Table 2*).

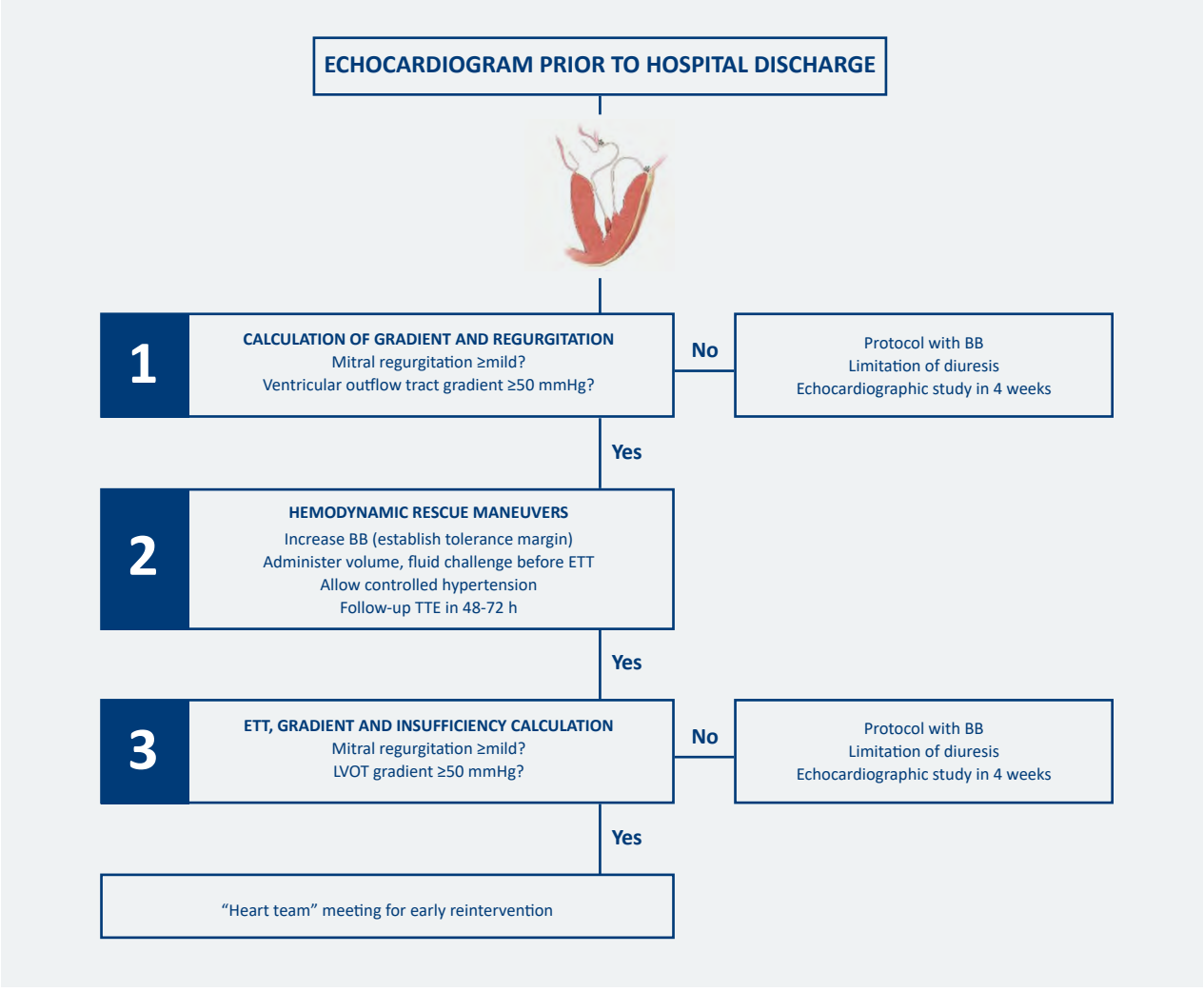

FIGURE 3. Postoperative management algorithm.
BB: beta-blocker, TTE: transthoracic echocardiogram, LVOT: left ventricular outflow tract.

Salvage techniques	Features
Posterior leaflet displacement (ventriculization) with neo-strings	First option due to its speed and simplicity. Ventriculization with neo-strings (Gore-Tex™ CV-5) of the posterior leaflet leads to more significant deployment of the anterior leaflet and displacement towards the posterior annulus of the coaptation line (greater distance between the anterior leaflet and the interventricular septum).
Alfieri's point (symmetric or asymmetric)	• Displacement of the coaptation line and anchoring of the anterior leaflet. • May not work in very redundant leaflets, distorts the native morphology of the mitral valve (dehiscence), and generates higher postoperative gradients.
Floating stitch	• Gore-Tex™ stitch between the free edge of A2 and the posterior prosthetic annulus (P2) to deploy the anterior leaflet and facilitate its anchoring without altering the movement of the posterior leaflet. Variants with two lateral points have been described. • A priori, the possibility of dehiscence is lower.
Posterior leaflet longitudinal plication (shortening)	• Posterior displacement of the coaptation line after reducing the height of the posterior leaflet by about 5 mm. 4-0 polypropylene suture is used, and four mattress stitches are made with pledget support, U-stitches, or cut and sew technique. • It takes an additional 10 to 15 minutes.
Anterior leaflet longitudinal plication (shortening)	• As described above for the posterior leaflet. • It requires the certainty that the problem is not the height of the posterior leaflet since, with this technique, the anterior leaflet is shortened, and the coaptation line can be displaced towards the LVOT.
Replacement of the prosthetic ring with a larger size or a band	• Remodeling annuloplasty with a larger annulus size (either because of a technical measurement error or to accommodate changes in the native annulus) leads to increased deployment of the anterior leaflet. This also happens if bands are used instead of rings (greater anteroposterior distance). • It is much more time-consuming.

TABLE 2. Surgical techniques to correct systolic anterior motion after mitral repair.

CONCLUSION

The occurrence of SAM after reconstructive mitral surgery is considered a mainly iatrogenic phenomenon. It is a complex mechanism involving the entrainment of the anterior leaflet into the LVOT in the systolic phase. Outflow tract obstruction causes turbulent flow that can lead to mitral regurgitation, acute pulmonary edema, and even sudden death. Perioperative diagnosis is eminently echocardiographic. The management of MAS requires a logical, stepwise approach ranging from conservative medical management to reoperation. Decision-making should be based on the severity of the MAS and the experience of the multidisciplinary team in charge of the patient.

Declarations
The authors declare no conflict of interest.

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SCIENTIFIC LETTER

LERICHE SYNDROME: A RARE PRESENTATION OF ARTERIAL OCCLUSIVE DISEASE IN LATIN AMERICA

ABSTRACT

Leriche syndrome is a chronic arterial occlusive disease that primarily affects the lower abdominal aorta and iliac artery bifurcation. We present the case of a 53-year-old male with a classical Leriche triad of gluteal claudication, erectile dysfunction, and the absence of a femoral pulse. The patient had a history of hypertension and heavy smoking. Physical exam showed no ulcers. Distal capillary fill was greater than 10 seconds. Complementary exams reported normal findings. Leriche syndrome was suspected, and thoracoabdominal computed tomography angiography and lower limb computed tomography angiography were performed, revealing complete occlusion of the lower aortic bifurcation and iliac artery involvement. A retroperitoneal approach for bifemoral aortic bypass was performed. The patient had an optimal postoperative period and recovery.

Keywords: *Leriche syndrome, aortoiliac occlusive disease, vascular surgery.*

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INTRODUCTION

A chronic arterial occlusive disease affecting the infrarenal aorta, iliac arteries, and femoropopliteal vessels is called Leriche syndrome (LS). Classical symptoms known as the Leriche triad include the absence of femoral pulses, intermittent claudication, and impotence[1].

The exact incidence and prevalence of LS remain unknown. However, incidence increases in the presence of various risk factors, including male sex, advanced age (≥50 years), atherosclerosis, dyslipidemia, smoking, and diabetes[1,2]. Only 10% of LS patients experience symptoms, indicating advanced disease and resulting in limitations for treatment selection[3]. Therefore, early diagnosis is crucial to enhance the patient's quality of life and reduce the risk of unfavorable outcomes, such as acute myocardial infarction, stroke, and even death[1,2].

This case report series aims to showcase the significance of an accurate diagnosis and optimal treatment approach in improving outcomes on LS within the Latin-American population.

CASE REPORT

A male patient, 53 years old, presented to the emergency department of a local hospital with a 7-day history of gluteal claudication, the absence of femoral pulses, and erectile dysfunction. Over the past week, the claudication had progressed to a point where it was persistent even during rest periods, prompting his visit to the emergency department. His medical history revealed a background of hypertension and heavy smoking, with a calculated pack-year index of 20. Initial vital signs indicated grade 2 hypertension (140/82 mmHg). On physical examination, femoral pulses were notably absent, and distal capillary refill time was measured at 10 seconds. The patient exhibited gluteal and lower limb claudication, manifesting even after covering a distance of 50 meters (164 ft), and this was concomitant with erectile dysfunction. Notably, there were no ulcers on his extremities or signs of necrosis at the level of his fingers.

Laboratory tests were conducted, revealing the following results: hemoglobin level of 14.8 g/dL, platelet count of 253,000/mm^3, serum creatinine concentration of 1.04 mg/dL, leukocyte count of 11,500/mm^3, and an international normalized ratio (INR) of 1.01. An electrocardiogram and chest X-ray were also performed, which yielded normal findings.

Given the presence of the classic clinical triad of LS in a patient with a history of smoking, we proceeded to perform thoracoabdominal computed tomography angiography (TA-CTA) and lower limb computed tomography angiography (LL-CTA). The TA-CTA results revealed chronic complete occlusion (CCO) of the aortic bifurcation with the involvement of the common iliac arteries (*Image 1*). Furthermore, the LL-CTA indicated the absence of macroscopic vascular lesions (*Image 2*). Based on these findings, we established a diagnosis of aortoiliac occlusive disease classified as Trans-Atlantic Inter-Society Consensus II (TASC II) type D, total occlusion with bilateral iliac artery involvement.

Considering the patient's age and the lower perioperative mortality risk associated with the procedure, we opted for a retroperitoneal bifemoral aortic bypass (BFAB) as the optimal treatment choice in this case rather than covered endovascular reconstruction of the aortic bifurcation (CERAB) (*Image 3*). No reoperation was necessary, and no surgical site infection (SSI) signs were detected. The patient remained in the Intensive Care Unit for one day for postoperative monitoring before being hospitalized for three days. The patient continued to abstain from smoking entirely and maintained the COMPASS-VOYAGER pharmacologic regimen, which included daily administration of Aspirin 100 mg, Atorvastatin 40 mg, and Rivaroxaban 2.5 mg every 12 hours indefinitely. Following the surgery, the patient experienced a 100% improvement in claudication symptoms, with femoral and pedal pulses reappearance.

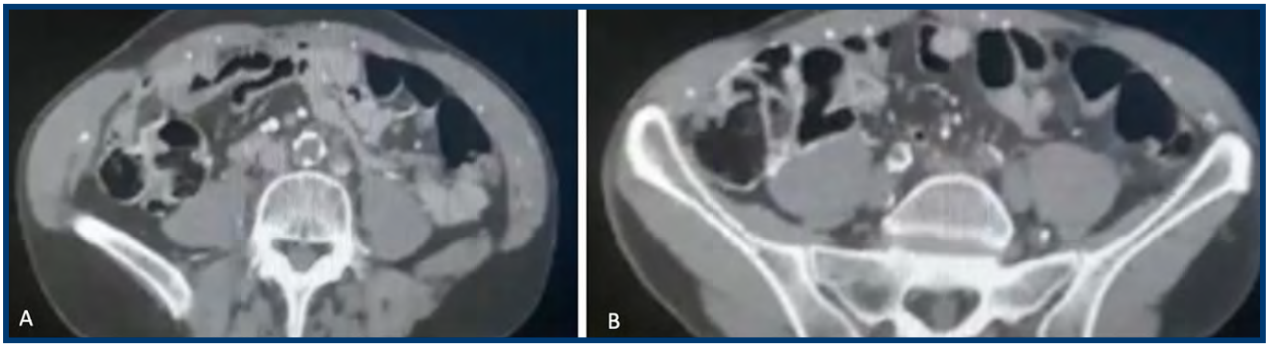

IMAGE 1. Thoracoabdominal-computed tomography angiography. Evidence of chronic complete occlusion of (A) the infrarenal aorta and aortic bifurcation and (B) involvement of the common iliac arteries.

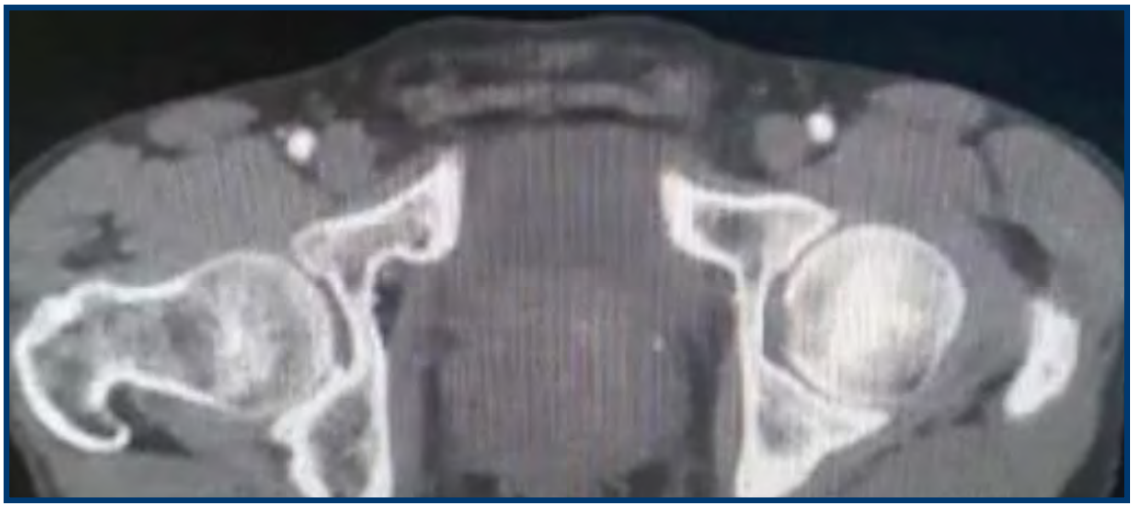

IMAGE 2. Lower limb-computed tomography angiography. No macroscopic vascular lesions were detected.

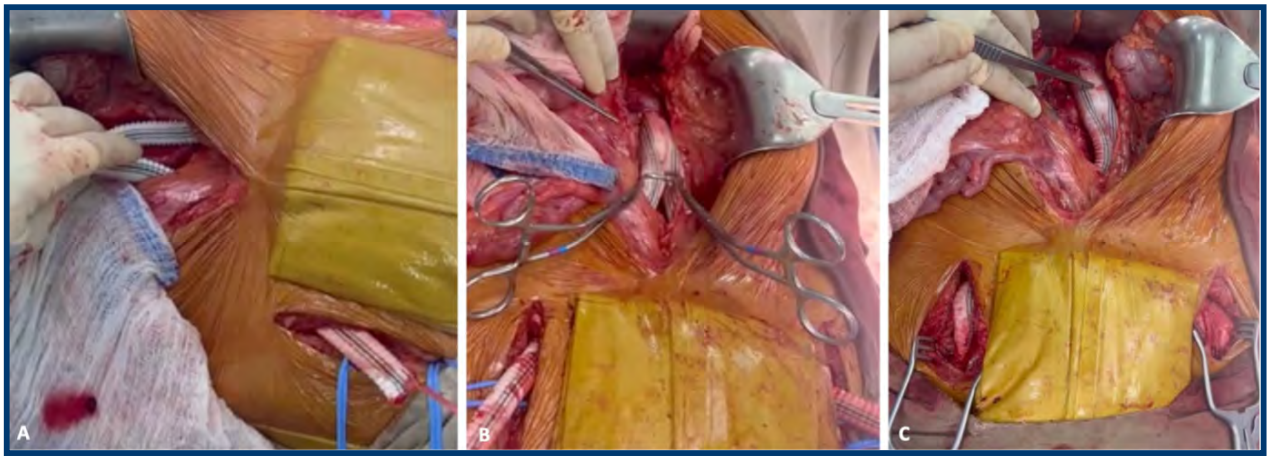

IMAGE 3. Retroperitoneal bifemoral aortic bypass procedure. **A.** Creation of retroperitoneal iliac tunnel and passage of the aortobifemoral graft. **B.** Proximal aortic anastomosis. **C.** Final aortobifemoral bypass with bifurcated Dacron graft.

DISCUSSION

LS predominantly manifests in male patients between their third and sixth decades. The classical triad typifies it. Nevertheless, there exist cases in which erectile dysfunction and claudication may be attenuated due to the presence of collateral circulation. Hence, the absence of femoral pulses is the primary symptom to investigate in patients suspected of having LS[4].

A retrospective study revealed that 82.8% of patients with LS had a recent history of tobacco use, 63.2% presented with dyslipidemia, and 55.2% had arterial hypertension5. These variables are significant in assessing our patients, as poorly controlled hypertension increases the risk for peripheral artery disease (OR: 1.75; 95%CI: 0.97-3.13), smoking (OR: 4.46; 95%CI: 2.25-8.84) and hypercholesterolemia (OR: 1.68; 95%CI: 1.09-2.57)[6]. Chronic endothelial damage and inflammation of the aortoiliac segments can result in significant stenosis, with more than 50% narrowing of the total lumen in these segments[2].

Smoking holds particular importance within the Latin-American context, as an estimated smoking initiation age of 11.9 years is evident, with a prevalence of consumption ranging from 7.4% to 34.1% among 663,703 school students[7]. Therefore, it is imperative to strengthen public health policies aimed at reducing morbidity and mortality associated with tobacco consumption, particularly when it begins at a young age.

A comprehensive clinical history and physical examination are essential to suspect LS diagnosis. However, radiological findings obtained through computed tomography angiography (CTA) are instrumental in achieving a more precise characterization of the luminal defect, facilitating the diagnosis and classification of LS. CTA demonstrates sensitivity and specificity exceeding 95% in detecting stenosis or occlusion within the aortoiliac and femoropopliteal segments, although its performance is comparatively lower in the infrapatellar segments. The inclusion of an electrocardiogram in patients

suspected of having LS is crucial since 10-71% of such patients may concurrently present with coronary artery disease[3,4].

Considering the management of LS, a comparative analysis was undertaken to assess the efficacy of open surgical repair versus endovascular repair using self-expanding covered stents for treating complex TASC type C and D aortoiliac lesions in a cohort of 114 patients. Among these patients, 58 underwent CERAB, exhibiting shorter hospital stays (2.6 days vs. 8.6 days) and reduced ICU stays (0.1 days vs. 0.9 days) compared to the 56 patients who underwent BFAB[8]. However, both groups demonstrated similar primary patency rates (87.3% and 81.4%, respectively) and comparable limb salvage rates (98.9% and 98.4%, respectively). This trend persisted even among the subset of younger patients, those under 60 years old[8].

In a retrospective study conducted by Fujimura et al., a comparison of clinical outcomes between BFAB and CERAB was performed in 436 patients with chronic total occlusion of the infrarenal abdominal aorta. The study revealed that CERAB also substantially reduced hospital length of stay by approximately 10 days. However, procedural success rates were consistently high and comparable, with CERAB demonstrating a success rate of 98.9% compared to 96.6% for BFAB. Similarly, the incidence of complications was 9.1% for CERAB and 12.3% for BFAB. In comparison, the respective mortality rates were 2.3% for CERAB and 3.8% for BFAB, with no statistically significant differences observed between the two treatment groups[9].

A single-center retrospective study by Smith et al. analyzed the treatment outcomes of patients with TASC II D lesions who underwent either BFAB or CERAB. The primary surgical indications were claudication in 55.6% of patients, rest pain in 28.3%, and tissue loss in 16.2%. The study demonstrated that patients treated with CERAB experienced shorter hospital stays and lower surgical site infection rates compared to those treated with BFAB (8.0% vs 37.5%, respectively). However, patients treated with BFAB showed significantly higher five-year primary patency rates compared to CERAB (88.1% vs. 50.8%, respectively) and superior five-year survival rates (100% vs. 76.5%, respectively)[10].

It is important to emphasize that patients undergoing surgery exhibit rest-induced disabling claudication that remains unresponsive to medical interventions or present with limb-threatening ischemia. Preoperative smoking cessation is mandatory to mitigate recurrence rates and the need for re-interventions. The CERAB approach was designed for patients TASC II C and D. This technique is advantageous in minimizing perioperative mortality for patients over 65. However, its 10-year patency is not well established. Thus, younger patients suitable for surgery are better candidates for BFAP intervention for LS treatment.

Our study has some limitations, as it is a monocentric, observational, and descriptive study of a unique case presented. However, reporting cases of interest is essential to take a first step towards creating a multicentric database for future studies in Colombia and Latin America, leading to a better approach to the prevention, diagnosis, and treatment of this condition within our population.

CONCLUSION

As an unusual presentation of arterial occlusive disease, LS is a critical clinical condition where the clinical approach is vital for optimal events. For patients with LS with non-advanced age and favorable conditions for surgery, open bifemoral aortic bypass is a better approach for definitive cure as it shows better long-term patency compared to endovascular treatment.

Declarations

The authors declare no conflict of interest.

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SCIENTIFIC LETTER

ACUTE ATRAUMATIC BILATERAL PERONEAL COMPARTMENT SYNDROME AFTER DIALYSIS ACCESS SURGERY

ABSTRACT

Acute atraumatic compartment syndrome is a rare entity; of the peroneal compartment, it is even rarer and bilateral even more so. We present the case of a patient who underwent surgery to rescue a brachiocephalic mega-fistula with three aneurysms associated with a stenosis of the cephalic arch. The technique consisted of aneurysmectomy, removal of the excess vein, transposition, and a new anastomosis distal to the axillary vein. The procedure lasted 220 minutes, and general anesthesia was used. The following day, the patient reported left leg paraesthesia, pain, and inability to evert the left foot or walk. Arterial pulses were normal, as was pulse wave Doppler. MRI confirmed peroneal compartment syndrome, and an emergency fasciotomy was performed. While recovering with full sensation and motility for the next 24 hours on the left side, she presented with pain and cramps in the right peroneal compartment without more severe symptoms, so fasciotomy was not performed; the condition resolved spontaneously within 24 hours. This is the first reported case of atraumatic bilateral peroneal compartment syndrome after dialysis access surgery, with full recovery after prompt treatment.

Keywords: *acute compartment syndrome, atraumatic fasciotomy, peroneal compartment, lateral compartment, mega-fistula, dialysis access complication, bilateral compartment syndrome.*

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INTRODUCTION

Patients with end-stage renal disease are often complex cases for the vascular team. Solving dialysis access failure requires a thorough understanding of the hemodynamic problems and the corresponding techniques and procedures. There is no consensus definition of a mega-fistula, although it can be defined as a generalized aneurysmal dilatation of the entire outflow tract of an arteriovenous fistula (AVF). There is consensus that an aneurysmal segment has twice the diameter of the adjacent normal vein, together with a high intra-access pressure and an access flow greater than 2000 ml/min[1]. Outflow stenosis is often present, and previous renal transplantation may also be a risk factor[2]. In addition to dilatation of the entire vein, true aneurysms may also occur, which worsen the prognosis of the AVF and require additional procedures for treatment. Treatment options often include segmental graft replacement, angioplasties, covered stents, and, most commonly, ligation of the AFV.

Acute compartment syndrome (ACS) is a severe limb-threatening condition. It is caused by abnormally high tissue pressure in a compartment enclosed by the fascia in the limbs, which contains muscle tissues, nerves, and blood vessels. If the pressure within the compartment exceeds the ischaemic threshold of the tissue, irreversible damage can occur[3]. Traumatic ACS is much more common, while spontaneous compartment syndrome presents in the most unexpected ways. Bilateral lateral or peroneal compartment syndrome is rare. It has been described in cases such as methanol intoxication, alcohol, sinvastatin-induced, prolonged positioning, spontaneous bleeding due to anticoagulation, and saphenous vein stripping.

CLINICAL CASE

We present the case of a 28-year-old male patient with a brachiocephalic fistula of 9 years of evolution who presented with elevated venous pressure during dialysis. He was on chronic treatment with statins, valsartan, nevibolol, and amlodipine and had a secondary hyperparathyroidism under study. The AVF consisted of a mega-fistula with three aneurysms and a cephalic arch stenosis. Initially, an 8 mm balloon angioplasty was performed with instant recurrence. Due to the problems encountered, surgical treatment was chosen. A salvage technique described by Nezakatgoo et al.[4] was used. Under general anesthesia, the vein was wholly dissected 2 cm from the arterial anastomosis and sectioned as distally as possible; a 24 Fr thoracostomy tube was used to calibrate the AVF and excess vein, and aneurysms were removed (*Figure 1*). A continuous Prolene™ 6-0 suture line was used over the tube, tunneled with a 90-degree rotation, and reanastomosed to the axillary vein (*Figure 1*). The procedure lasted 220 minutes with the patient in the supine position, with low-dose noradrenaline requirements, and no complications were observed. The patient woke up with moderate pain in the left calf, full mobility, and sensation; all pulses were palpable. The following day, he evolved with intense pain, lack of sensibility, and no eversion, with a precise diagnosis of peroneal palsy.

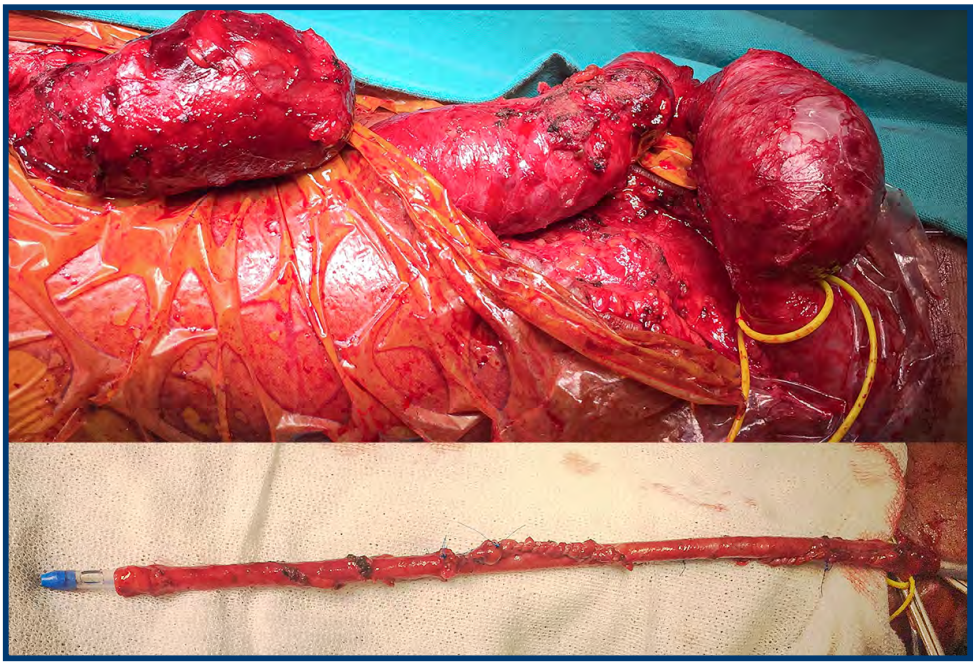

FIGURE 1. Mega-fistula with three aneurysms (top) and fistula calibration with thoracostomy tube (bottom).

The creatine phosphokinase (CPK) value was 13,624 IU/L. Arterial Doppler reported normal pulse waves with arterial calcification. MRI showed significant edema only in the left lateral compartment (*Figure 2*). An emergency fasciotomy was performed, where the muscles (pale at the time) showed rapid recovery of perfusion within a couple of minutes. The skin was sutured and closed without tension. The patient regained total motility and sensation within 24 hours. He started with pain and cramping in the contralateral peroneal compartment, with no nerve palsy or other symptoms. Another MRI was performed and again showed edema in the lateral compartment (*Figure 3*). No sensation and motility decreased in this limb, so no decompression was performed. CPK decreased to 4118 IU/L, 2588 IU/L, and 1295 IU/L in the following days. On the seventh day, already asymptomatic, he was discharged from the hospital. The patient has total motility and sensibility three years later, with a functional native AVF.

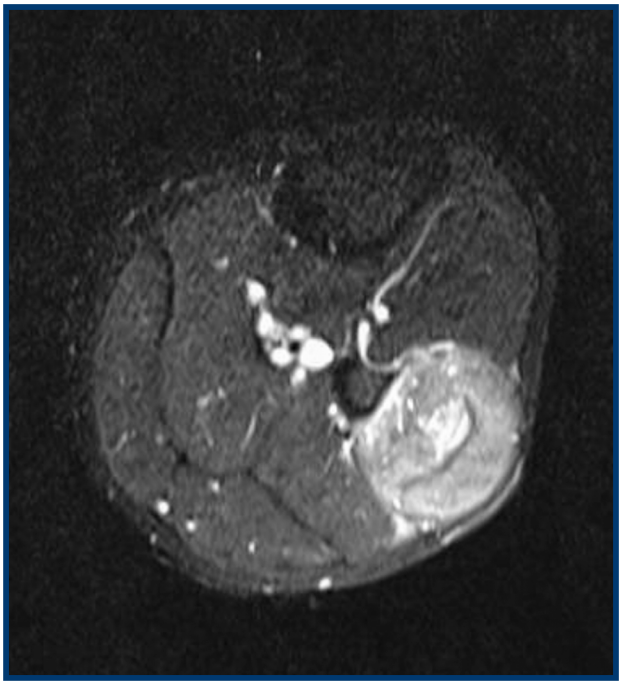

FIGURE 2. MRI of the left leg. Edema is seen in the peroneal compartment.

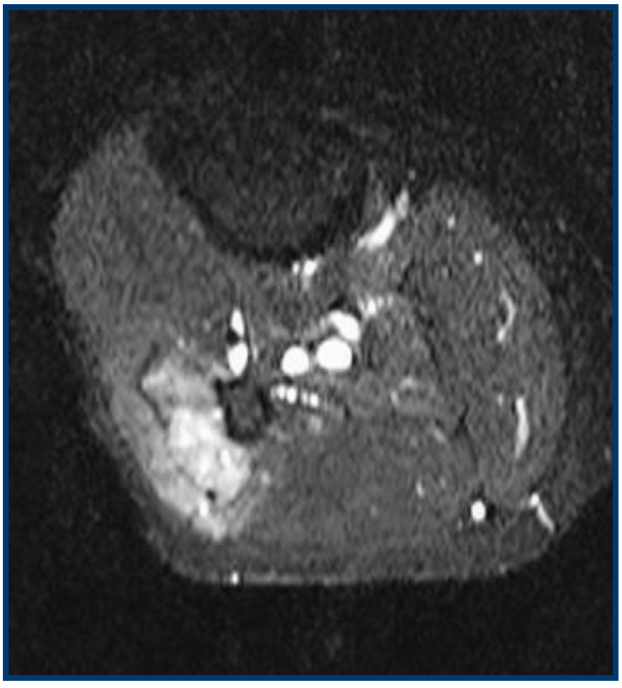

FIGURE 3. MRI of the right leg. Non-tension edema is seen in the peroneal compartment.

DISCUSSION

Compartment syndrome is expected in the context of acute trauma and vascular pathology, mainly in situations of acute ischaemia[5]. Younger male patients are at increased risk of developing compartment syndrome due to the relative hypertonicity of the muscles, which does not leave adequate space for localized inflammation[5]. Acute atraumatic compartment syndrome (ACS) is a rare situation, demanding suspect and requiring immediate treatment. Several comorbidities have been considered for the development, such as hypertension, dyslipidemia, obesity, diabetes, renal disease, cancer, and cardiac disease; multiple medications such as antihypertensives, statins, anticoagulants, antidiabetics, opioids, paracetamol, vasoactive drugs, and diuretics; and several situations such as trauma, acute ischemia, exercise, postural and some surgeries (cardiac and orthopedic). Both anticoagulants and statins have been independently associated with an increased risk of developing ACS[6]. In 2020, the first case of spontaneous gene-related ACS in a young male with sequential four-limb ACS was published. The association to a genetic mutation in exon 3 of the GYG1 gene, related to muscle metabolism, is likely[7].

Long-term surgeries in lithotomy position are recognized as a risk factor for ACS[8]. ACS has been reported in patients with cardiovascular pathology after cardiac surgery, usually on the same limb from which the saphenous vein was removed. Diagnosis is not accessible after this type of surgery, as the patient is usually on assisted ventilation, and no information on functional status can be obtained[9]. ACS has also been described after prolonged aortic endovascular procedures, most likely due to prolonged ischaemia[10]. In our case, position, hypotension due to anesthesia, and the need for vasoactive drugs were risk factors influencing the outcome.

Spontaneous or atraumatic bilateral compartment syndrome is a rare condition. It has been reported most frequently after exercise, in both upper and lower limbs, and after prolonged standing[11,12]. Bilateral peroneal ACS has been described after horseback riding, usually with a very late diagnosis[13].

Fasciotomies should be performed promptly once ACS is established. Fasciotomies performed within 6 hours achieved complete functional limb recovery in a study by Rorabeck and Macnab[14].

When performed within 12 hours of acute onset, 68% achieved normal limb function, but only 8% did so when fasciotomy was performed after that period[15]. Hyperbaric oxygen therapy has been reported as another treatment for ACS, but fasciotomy is still the standard treatment[16]. In our case, muscle perfusion recovered a few minutes after fasciotomy and peroneal nerve palsy recovered within 24 hours despite having a 32-hour interval from symptom onset and treatment. The pain was the only symptom in the right leg, and the MRI showed mild edema, so it was decided to maintain expectant management. As the patient was already on dialysis, elevated CPK levels and renal failure were not a concern.

CONCLUSIONS

We present the first case of acute compartment syndrome after dialysis access surgery; the fact that it is of the peroneal compartment and bilateral makes it even rarer. A high index of suspicion, early diagnosis, and a multidisciplinary approach are the best ways to deal with this type of situation, as they maximize the chances of recovery and avoid permanent disability.

Declarations

The authors declare no conflict of interest.

Consent

Written informed consent was obtained from the patient to publish this case report.

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SCIENTIFIC LETTER

SUCCESSFUL SURGICAL MANAGEMENT OF THREE MECHANICAL COMPLICATIONS OF ACUTE MYOCARDIAL INFARCTION: VENTRICULAR SEPTAL DEFECT, SEVERE MITRAL REGURGITATION AND VENTRICULAR ANEURYSM

ABSTRACT

Mechanical complications after acute myocardial infarction have a very high mortality rate on their own (40-50%), and the sum of these increases the risk of mortality after surgical repair. In this case report, we present a patient with a postinfarction ventricular septal defect of posterior location and early presentation with hemodynamic stability. Subsequently, the patient presented with severe acute mitral regurgitation plus posterior ventricular aneurysm. A surgical procedure with successful resolution is performed. Early diagnosis, patient selection, and adequate multidisciplinary management optimize successful outcomes. This communication aims to expose a clinical case with these characteristics since, in the literature, there are very few cases of living patients with these three complications who survived the acute event and even more after surgical repair.

Keywords: *post-infarction complications, ventricular septal defect, mitral regurgitation, ventricular aneurysm.*

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INTRODUCTION

Although the incidence of mechanical complications in patients after acute myocardial infarction (AMI) is low (0.21%), the associated mortality rate separately is very high (40-50%)[1]. However, given the sum of these and their presentation with subsequent localization and, in addition, the time to surgical resolution, especially in elderly patients, the therapeutic options are usually complex and require the experience of a multidisciplinary team.

Over the past 30 years, improvements in timely reperfusion techniques within regionalized care systems and advances in optimal medical therapies have reduced AMI mortality rates. However, nowadays, patients present with more comorbidities. The clinical characteristics of these mechanical complications differ and depend on the rupture site (the free wall of either the ventricle, the interventricular septum, or the papillary muscles). Ventricular septal rupture (75%) is the most frequent type of mechanical complication after AMI; the remaining patients develop mitral regurgitation (19%) due to papillary muscle rupture or free wall rupture (6%)[2]. The optimal timing of corrective surgery for mechanical complications is still controversial in the medical community. The main factor determining survival after the surgical procedure is the timing of the procedure, so most centers defer corrective surgery whenever possible[3].

CASE PRESENTATION

A 71-year-old male patient, hypertensive, with a history of chronic kidney disease KDIGO III and positive smoking, presented to the emergency department with precordial pain 9/10 on the visual analog scale (VAS); an electrocardiogram was performed showing positive ST-segment elevation in DII, DIII, and AVF with initial Q wave, as well as third-degree atrioventricular block. A temporary transvenous pacemaker was placed. Ten days later, the patient presented hemodynamic instability and bacteremia from a urinary tract infection (with the presence of pyuria). Antibiotic treatment was started. Once hemodynamically stable and no longer receiving vasopressor drugs, the patient presented a pericardial effusion of 18 mm without cavity collapse detected by transthoracic echocardiography (TTE). It was decided to admit him to the post-surgical therapy unit for close monitoring. A positive blood culture for Acinetobacter baumannii was received three days later, so the antibiotic was rotated. The patient evolved with clinical improvement, and two weeks after the initial event, he presented hemodynamic instability and cardiorespiratory arrest that required orotracheal intubation and amine management. A Levine grade III-IV mitral murmur radiating to the axilla was detected and blood cultures were requested, which showed Pseudomonas aeruginosa. Conservative treatment was decided, and the patient progressed to anuria. With hemodialysis support, the administration of vasopressor drugs was suspended, diuresis improved, and cultures were negative. Forty days after the initial event, a transesophageal echocardiogram (TEE) was performed, which showed an apical and posterior ventricular septal defect, 5 mm long, with left to right shunt with maximum trans ventricular septal defect gradient of 64 mmHg, Vmax of 4 m/s, ratio between pulmonary output and systemic output (Qp/Qs) of 3.3; basal and mid-inferior inferolateral akinesia, presence of pseudoaneurysm involving the basal and mid-segment, with a neck of 19 mm and a depth of 21 mm, and a left ventricular ejection fraction (LVEF) at rest of 48%. The mitral valve showed moderate insufficiency secondary to symmetric tenting of its leaflets, with a 5 mm jet vena contracta and a pulmonary artery systolic pressure of 40 mmHg.

The patient was referred to the hemodynamics service with evidence of a single lesion in the circumflex artery and posterobasal VSD (*Figure 1*), and it was decided to perform surgical correction of the three post-infarction complications.

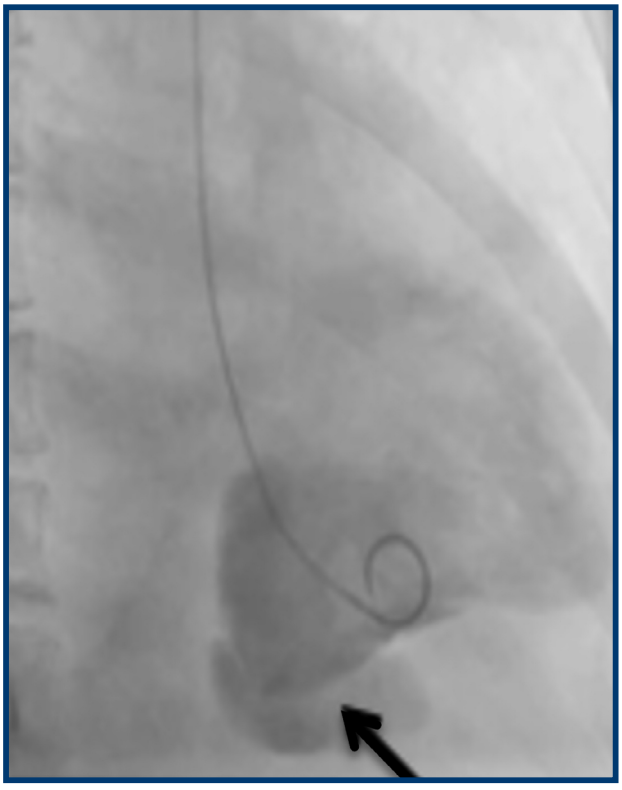

FIGURE 1. Catheterization with ventriculogram. There is evidence of a high posterior ventricular septal defect.

Due to the history of hemodynamic instability and the presence of an echocardiographic image compatible with contained ventricular rupture, dissection and peripheral cannulation of the femoral artery and vein were performed before sternotomy. Once the sternotomy was performed, lax adhesions were observed, with an area of hematoma on the posterolateral and inferior aspect of the left ventricle (*Figure 2*, yellow arrow).

Once free of adhesions, the superior vena cava was cannulated, aortic clamping was performed, and the cardioplegic solution was administered, in addition to maintaining the patient in moderate hypothermia (32-34 °C) for myocardial protection. Left ventriculotomy was performed through the area of the pseudoaneurysm, which extended very close to the posterior atrioventricular groove. Once the ventricular cavity was exposed, the ventricular septal defect was visualized approximately 1.5 cm x 1.5 cm, 5 mm from the posteromedial commissure (*Figure 2*, green arrow).

A patch was placed from Dacron™ with a 2-0 Ethibond suture with pledget and reinforced with a 3-0 Prolene continuous surge (*Figure 3*).

An endoaneurysmorrhaphy was performed with a 3-0 Prolene pericardial patch (*Figure 4*), and the closure was reinforced with the sandwich technique.

An incision was made in Waterston's groove for the left atrial approach. Visual examination of the mitral valve confirmed the presence of an area of mitral regurgitation between P3 and A3 (*Figure 5*).

According to the pre-surgical TTE, the U-stitch plasty was performed with 5-0 Prolene, and a hydraulic test was performed, which showed a competent mitral valve. It was decided not to revascularize due to the pathological process and repair zone in the circumflex artery territory. The patient was weaned from extracorporeal circulation, and decannulation was performed with minimal doses of inotropic drugs.

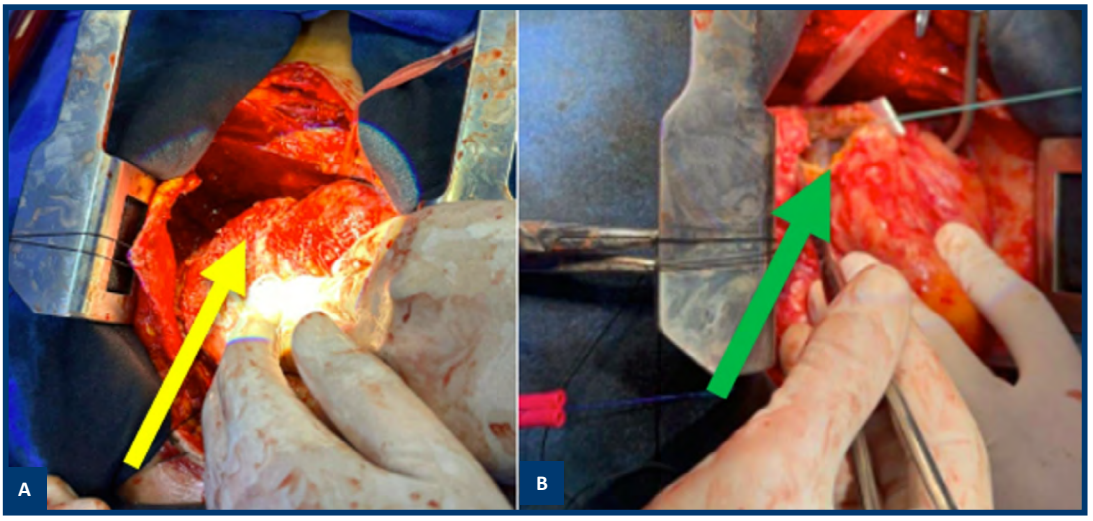

FIGURE 2. A. Collapsed aneurismatic zone (left ventricle is empty) (yellow arrow). **B.** Approach of the ventricular septal defect through the aneurysm (green arrow).

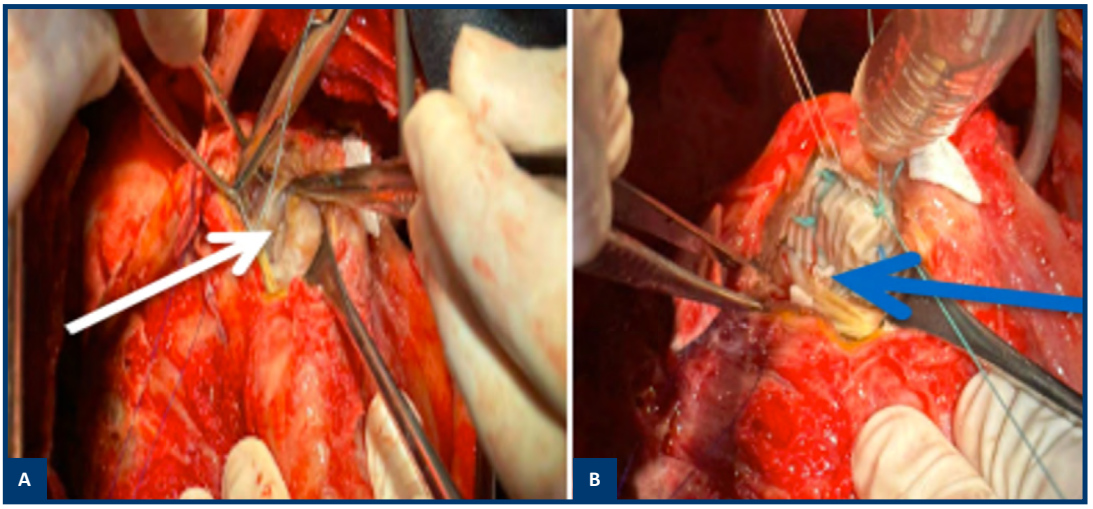

FIGURE 3. A. Presence of a 1.5 cm x 1.5 cm ventricular septal defect (VSD) (white arrow). **B.** Closure of the VSD with a patch of Dacron™ (blue arrow).

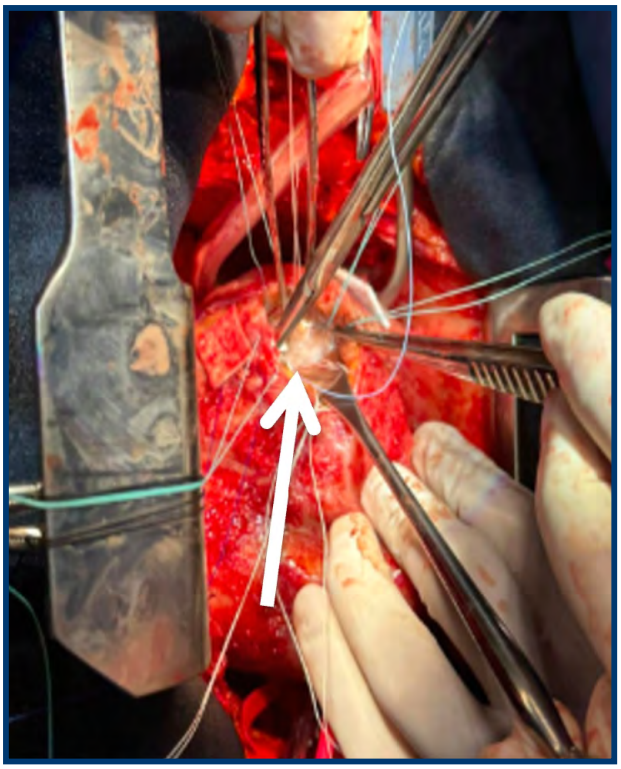

FIGURE 4. Pericardial patch placement for endoaneurysmography.

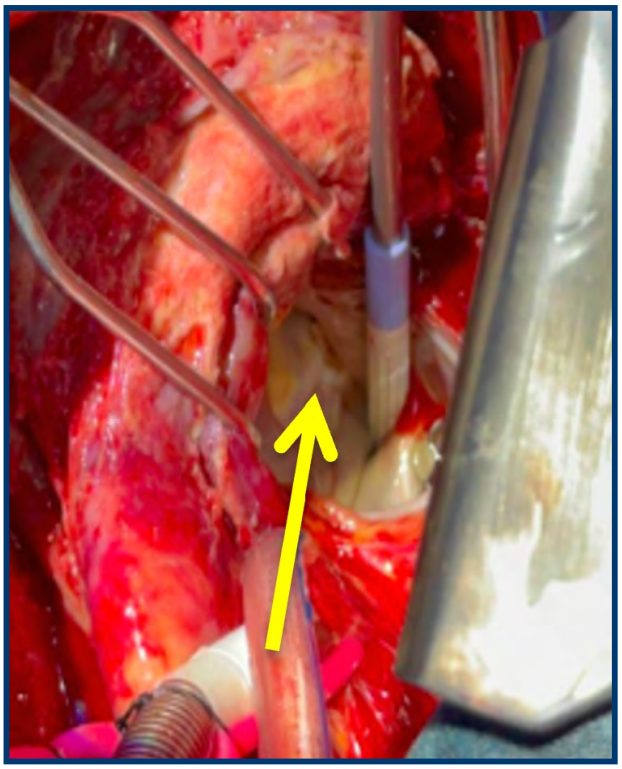

FIGURE 5. Alfieri mitral valve plasty at A3 and P3.

In the postoperative evolution, minimal doses of inotropic drugs and vasopressors were used, and the patient presented adequate diuresis and normal acid-base balance during the first hours of the postoperative period (POP). He was weaned from mechanical ventilation at 48 hours without complications.

Eight days postoperatively, the patient was transferred to the general ward, already extubated and with hemodynamic status. An echocardiogram showed an LVEF of 43%, no residual interventricular shunt, and no evidence of residual aneurysm and mild mitral insufficiency (*Figure 6*).

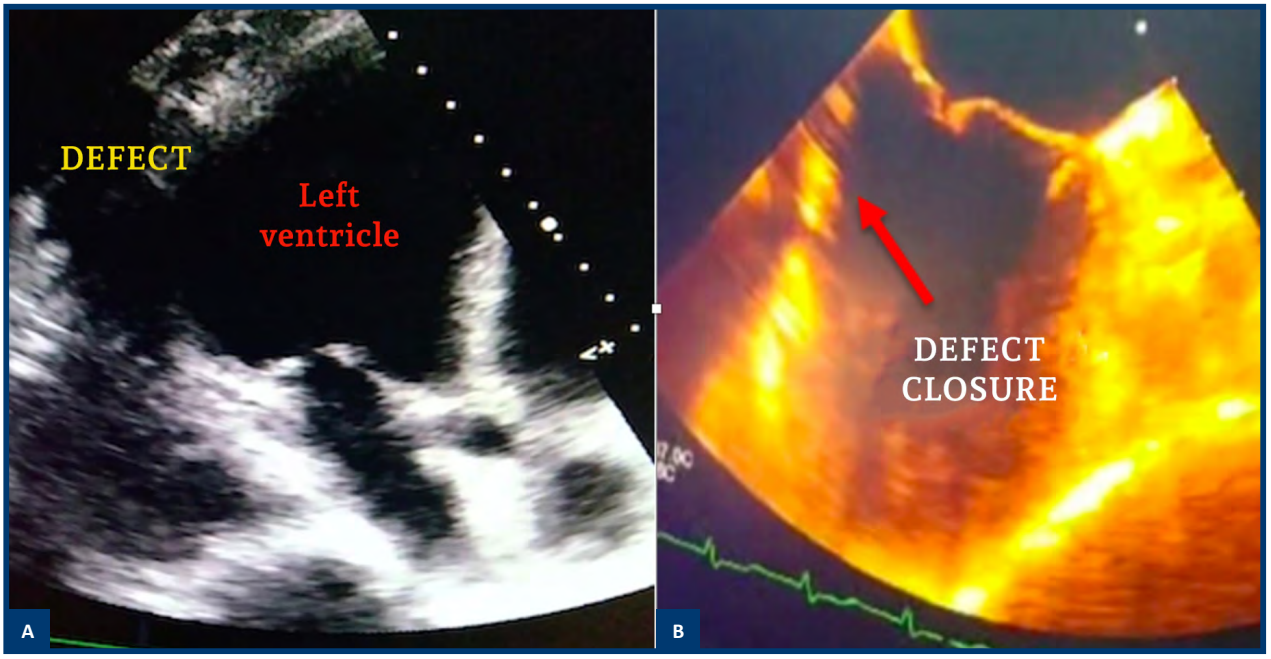

FIGURE 6. Transesophageal echocardiogram before **(A)** and after surgical repair **(B)**.

DISCUSSION

The optimal timing of definitive surgical repair is still difficult to establish. Although the 2013 guidelines of the American College of Cardiology and the American Heart Association[4] recommended emergency surgical repair of postinfarction ventricular septal defect (PIVSD), whatever the hemodynamic status, the timing of surgery in the setting of ventricular septal rupture is still a matter of controversy and should be considered in each patient on an individual basis.

Early corrective surgery should be considered in hemodynamically stable patients with favorable cardiac function and anatomy because sudden and unpredictable hemodynamic compromise is often observed.

Deferred surgery may be considered in stable patients when the surgical anatomy is complex, and there is concern about tissue fragility and the ability to perform a definitive repair.

The perceived benefit of delayed surgery has a mechanistic basis: after infarction, metalloproteinase activity and tissue degradation peak at day 7, whereas new collagen deposition begins between days 2 and 4, and collagen completely replaces necrotic myocytes by 28 days; therefore, a delay may facilitate successful repair by allowing friable tissue to organize, strengthen, and differentiate well from surrounding healthy tissue.

In this scenario, close follow-up in the intensive care unit may allow tissue healing and promote the chances of definitive repair[5] or use some form of ventricular support to manage hemodynamic instability if appropriate.

The 2017 European Society of Cardiology guidelines promote late elective repair in patients initially responding to aggressive conservative management6. Another of the most challenging problems for cardiac surgeons to solve is determining how best to approach a patient with ischemic mitral insufficiency (IMI).

The problem in IMI is not the valve but a ventricle disease due to the dilatation. For this reason, the evolution of IMI is very dynamic (mild to severe insufficiency); this is mainly related to preload and afterload conditions but often also to ongoing ischemia[7]. The surgeon's expertise is establishing the surgical treatment, which may be valve replacement or plasty. In the case presented here, it was decided to perform only mitral valve repair because it was complex (defined by the presence of two other mechanical complications).

In their publication, Kron et al.[7], a team composed mainly of surgeons who analyzed and synthesized the data and established consistent recommendations for surgeons, including how and when to repair or replace the valve, have accepted that surgical treatment is daunting. In addition, they discussed in detail the role of percutaneous therapy.

Myocardial revascularization in this type of patient is still controversial, mainly because of morbidity and mortality, which increase the longer the surgical time and cardiopulmonary bypass. On the other hand, in patients with recent infarction, inflammatory tissue in the infarct zone and, as in the case presented here, adhesions limit the exposure of the coronary vessel to be revascularized. If the hybrid approach is achieved, the result will be better.

Finally, aneurysms, most commonly affecting the LV, occur after myocardial infarction secondary to transmural myocardial necrosis; this tends to be a late complication where the affected segments of myocardial tissue are gradually replaced by akinetic fibrotic tissue, which is scar tissue that can dilate[8]. In our case, we performed endoaneurysmorrhaphy with a pericardial patch, and, as seen on TTE, there was no macroscopic difference in ventricular volume despite infarction and repair.

Declarations

The authors declare no conflict of interest.

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SCIENTIFIC LETTER

CHRONIC PSEUDOANEURYSM OF THE POPLITEAL ARTERY

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ABSTRACT

A 28-year-old male patient was admitted to the Orthopedic Service for a joint biopsy due to significant swelling in the left knee. He had a history of laparotomy for multiple gunshot wounds one year earlier, a mass in the left knee with severe pain, joint retraction, and gait limitation. Physical examination revealed a giant and reluctant mass in the left suprapatellar region. The tomographic study with vascular contrast showed a decrease in the caliber of the left superficial femoral artery in its distal portion prior to the emergence of the adductor canal, secondary to a large mass in the posterior compartment of the ipsilateral thigh. Arteriography showed a pseudoaneurysm of the left popliteal artery with a significant decrease in the caliber of the left superficial femoral artery. A firearm projectile was identified in the right knee. It was treated with open surgery and cell salvager Medtronic™, the pseudoaneurysm was resected, and raffia of the popliteal vein and arterial reconstruction with polytetrafluoroethylene prosthetic graft was performed.

Keywords: *pseudoaneurysm, popliteal artery, arterial reconstruction, open surgery, graft, polytetrafluoroethylene.*

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INTRODUCTION

The origin of arterial pseudoaneurysm is usually traumatic due to penetrating wounds, including firearms or bladed weapons[1]. The diagnosis of this entity is, in general, clinical. Physical examination and, sometimes, auxiliary diagnostic methods are essential to confirm it. We present the case of a male patient in whom the striking feature is the prolonged period of chronicity and in whom, with a complete anamnesis, the differential diagnosis can be established, given that the patient was initially referred for a scheduled joint biopsy.

MATERIALS AND METHODS

A 28-year-old male patient was referred to our center for a giant mass in the left knee of more than nine months of evolution to perform a scheduled osteoarticular biopsy. He had a history of exploratory laparotomy approximately one year before, which required, in another institution, enterorraphy of the small intestine and transverse colon, right kidney and a hepatic segment due to multiple gunshot wounds at close range and a gunshot wound in the external face of the left knee (*Figure 1*). The patient was admitted with a giant mass in the left knee with a reluctant, non-pulsatile consistency, difficulty walking and using crutches due to excellent joint retraction, and high-intensity pain. An angiotomography was performed, which showed a large mass in the left suprapatellar region of approximately 8 cm x 10 cm in diameter with mass effect, so an evaluation by the Vascular Surgery Service was requested (*Figures 2 to 5*).

An arteriography was requested, which revealed a pseudoaneurysm of the left popliteal artery with slowing of the flow of the superficial femoral artery and recanalization in infrapatellar vessels (*Figure 6*). Open surgical exploration under general anesthesia with a right jugular central line and left radial arterial line was decided. The left suprapatellar approach was performed through the inner aspect of the lower third of the thigh, and the cell retriever Medtronic™ was prepared for autotransfusion if necessary. Carefully, the soft tissues were dissected down to the exposure of the left popliteal artery pseudoaneurysm, which involved popliteal vessels (*Figure 7*). The pseudoaneurysm was resected, and a linear lesion was observed on the anterior-external aspect of the popliteal vein, approximately 3 cm, which was corrected with raffia of Prolene™ 5-0 (*Figure 8*). The popliteal artery was reconstructed with a 6 mm diameter ringed Goretex™ prosthetic graft, as the mass effect of the pseudoaneurysm thrombosed the left saphenous vein. Systemic heparinization was indicated with 7500 IU of heparin sodium, and proximal and distal end-to-end anastomosis was performed with 5-0 Prolene. Good recovery of distal flow and pulse was observed. No fasciotomy was performed, and no drains were used (*Figures 9 to 11*). The patient had no intraoperative complications and was hospitalized for 24 hours in the intermediate care unit, where he underwent antibiotic and antithrombotic prophylaxis. He was then transferred to the general ward, where he remained for five days, with good evolution of the wound and a notable reduction of pain that allowed reducing the dose of opioid analgesics. He was discharged for outpatient rehabilitation and physical therapy, and peripheral vasodilators and post-surgical control by outpatient clinic were indicated.

FIGURE 1. The medial approach of the popliteal artery and left popliteal pseudoaneurysm.

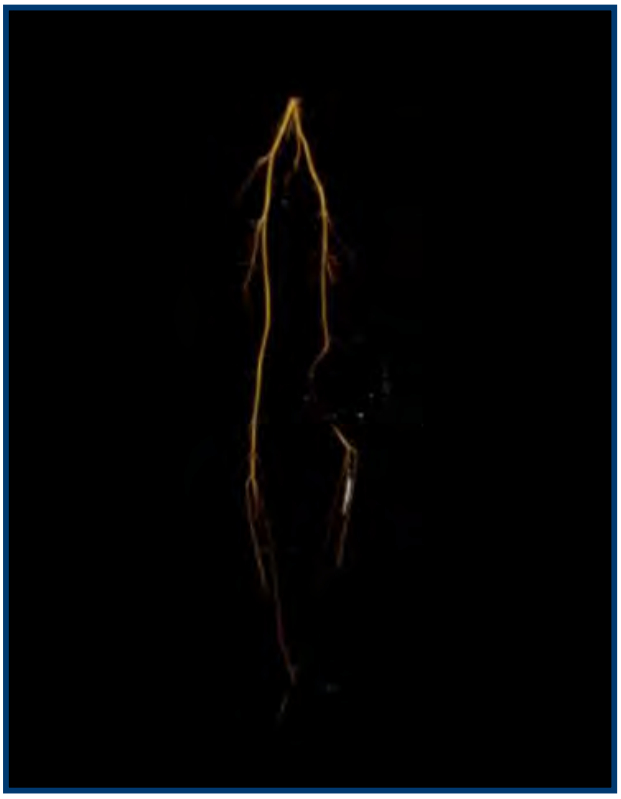

FIGURE 2. Angiotomography of the lower limbs with 3D reconstruction; a popliteal pseudoaneurysm with flow slowing in the superficial femoral artery is identified.

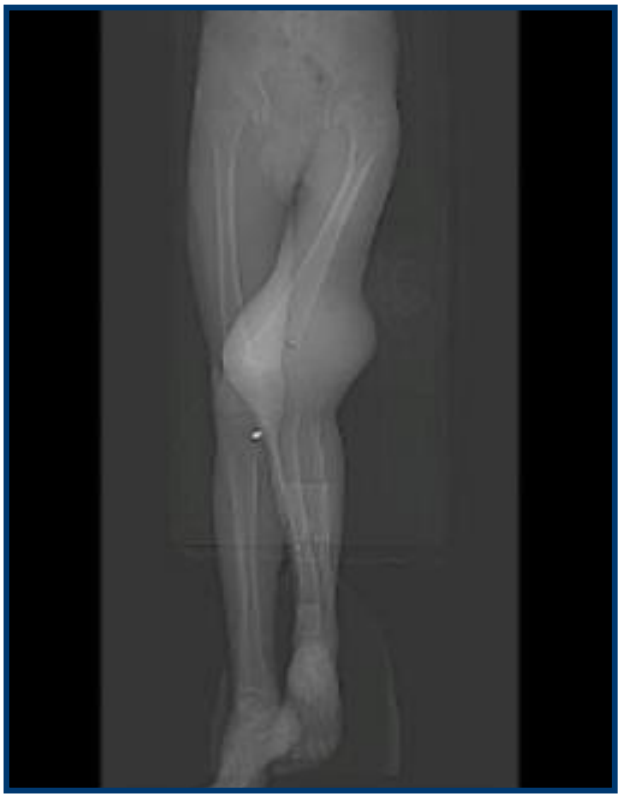

FIGURE 3. Angiotomography in simple plane of radiographic reconstruction showing a left suprapatellar mass.

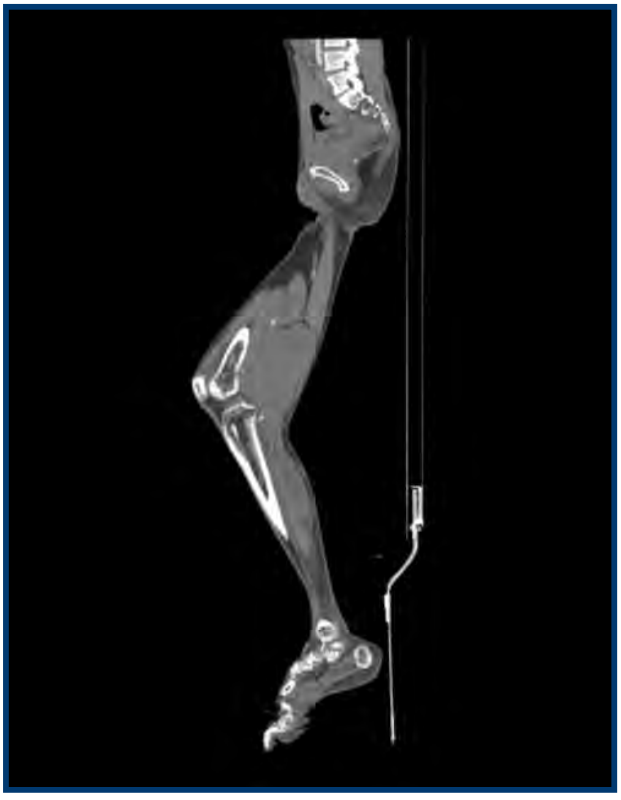

FIGURE 4. Angiographic sagittal plane showing a mass in the left popliteal fossa.

FIGURE 5. Angiographic sagittal plane of the popliteal fossa showing a giant pseudoaneurysm of the popliteal artery.

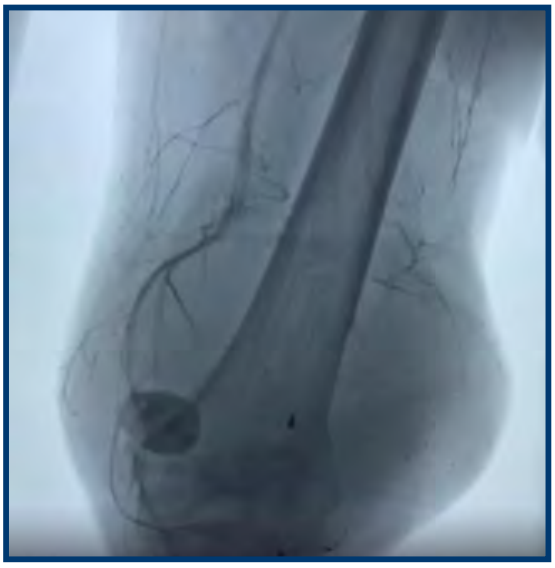

FIGURE 6. Arteriography of the left femoropopliteal axis showing a pseudoaneurysm in the left popliteal artery.

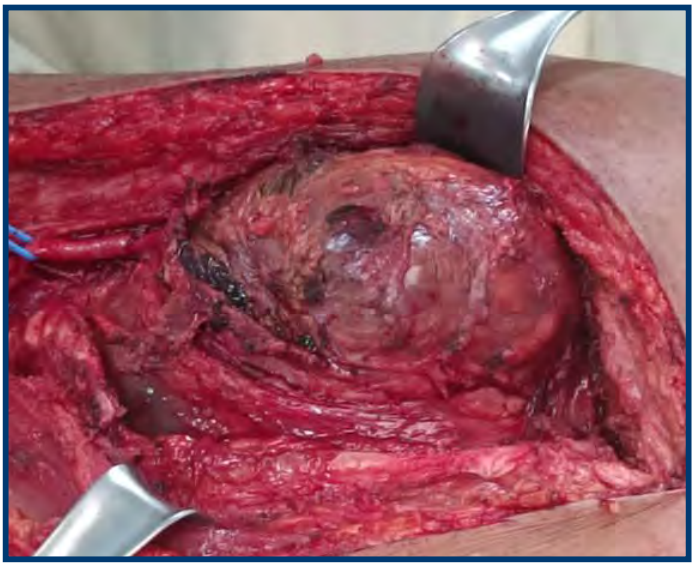

FIGURE 7. Popliteal artery and left popliteal pseudoaneurysm.

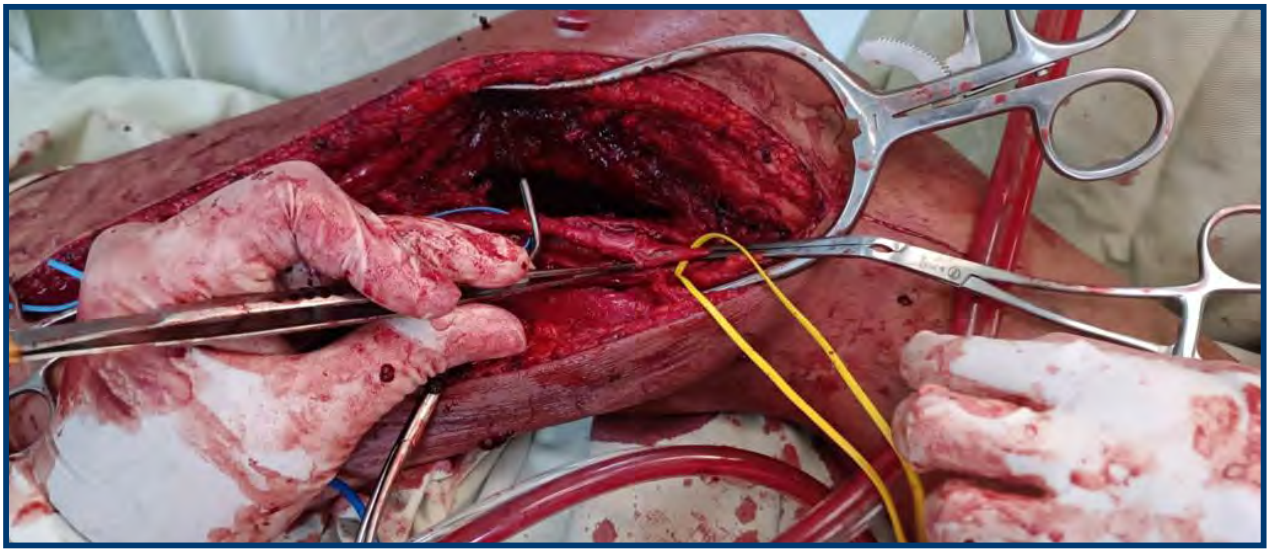

FIGURE 8. Resection of the pseudoaneurysm with an irregular linear lesion of the popliteal vein.

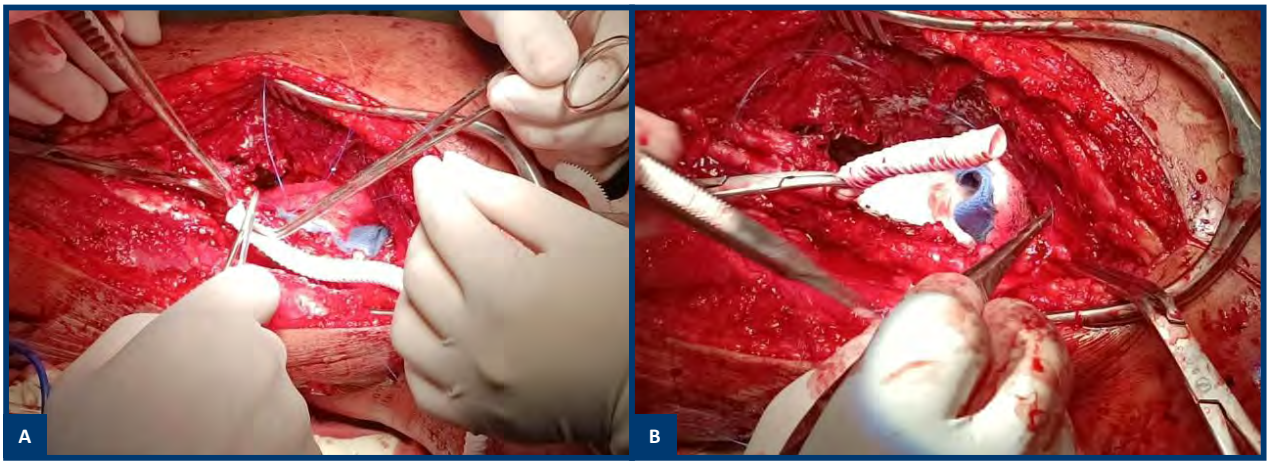

FIGURE 9. **A** and **B**: confection of proximal anastomosis with prosthesis.

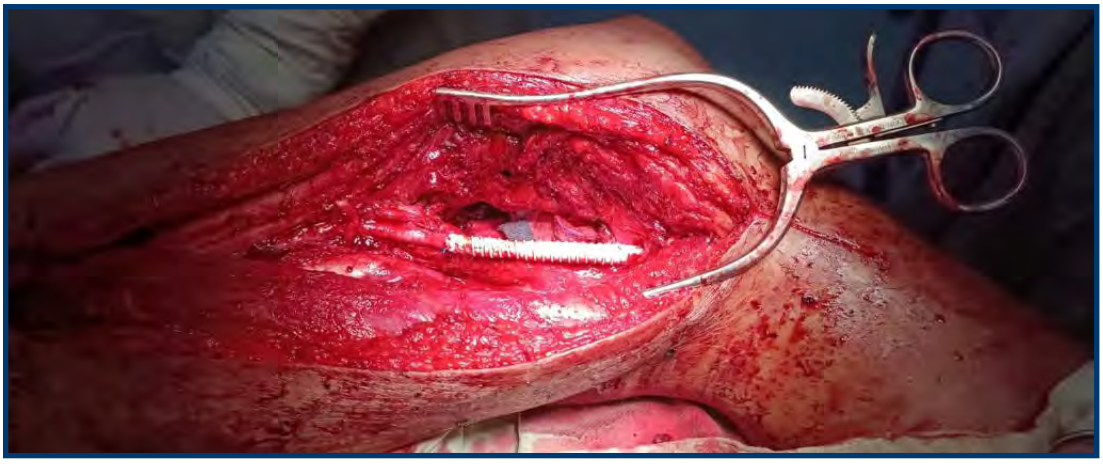

FIGURE 10. Primary reconstruction of the popliteal artery with terminal-terminal anastomosis with prosthesis.

FIGURE 11. Careful soft tissue synthesis in the popliteal region to protect the prosthetic graft.

DISCUSSION

The diagnosis of arterial pseudoaneurysms of traumatic origin in their late or chronic evolution is infrequent since the patient is usually in a window period of 6 to 8 hours to resolve the ischemia; otherwise, the consequences are generally catastrophic due to active bleeding that can be life-threatening[1].

These pseudoaneurysms have a significant risk of complications and limb loss. Sixty-two percent are associated with penetrating traumatic injuries, and 37% with iatrogenic injuries[2,3]. Their presentation is usually late, and the most common clinical findings are painful edema and pulsatile mass on physical examination[4]. Auxiliary diagnostic methods are of great help in establishing the diagnosis of certainty and planning the definitive course of action; among them, arterial duplex and angiotomography with three-dimensional reconstruction stand out[5]. In the case of the patient presented here, it was decided to perform conventional surgery since the popliteal artery aneurysm involved a significant extension of the vessel and also involved the popliteal vein on its external side. The approach was medial with resection of the affected arterial segment with interposition of a 6 mm diameter ringed polytetrafluoroethylene (PTFE) prosthesis with terminal-terminal anastomosis and venous raffia of the affected segment[6,7]. Given that the popliteal fossa is an anatomical area challenging to approach, endovascular repair was considered[8,9]. The risk of migration with placement of an endovascular device was considered to increase the risks of thrombosis, with exacerbation of the pre-existing lesion or the difficulties inherent to the movement of the popliteal artery and the device when walking; that is, flexion of the knee joint could accentuate the tortuosity between two fixed points, one proximal (the adductor muscle conduit) and the other distal to the origin of the anterior tibial artery, which can lead to stent fracture or thrombosis[10-12].

Ligation of the popliteal vein has a high correlation with compartment syndrome. In this case, the venous lesion was at the expense of its external aspect with a linear lesion that required simple raffia[13,14]. Other studies also document that amputation rates do not depend on the venous lesion treatment technique[15,16]. In this procedure, fasciotomy was not

performed due to the careful management of the dissection of the different planes of the region and because a prosthetic graft was used for the primary reconstruction of the popliteal artery. To protect the graft against possible infections, it was covered with the vastus externus muscle and the long portion of the adductor magnus muscle; there was no need to place a drain.

RESULTS

The patient underwent open surgery under general anesthesia with cell salvage to reduce the risk of hypovolemia. A giant pseudoaneurysm of the left popliteal artery was resected, and primary arterial reconstruction was performed with a prosthetic graft. No fasciotomy or drains were used. Bacterial and antithrombotic prophylaxis was indicated, and systemic anticoagulation was not used to reduce bleeding risks[17]. Surgical time was approximately 4.5 hours, which reduced the risk of ischemia and amputation[18,19]. Autologous saphenous vein grafting was not used, given that the giant pseudoaneurysm thrombosed a large part of the homolateral internal saphenous vein; the venous vessels of the contralateral lower limb were preserved for eventual reoperation[20]. The patient presented no complications and was discharged on his fifth day of hospital stay with negative blood cultures at 72 hours. On discharge, peripheral vasodilators (cilostazol and acetylsalicylic acid) were prescribed, and physical therapy sessions were indicated to achieve a favorable evolution with early rehabilitation.

In the first instance, the patient had been referred for an etiological diagnosis of a mass of probably osteoarticular origin. Physical examination and ancillary diagnostic methods allowed for establishing the definitive, timely diagnosis in a patient with joint retraction, gait difficulty, and a giant mass in the knee.

CONCLUSION

Chronic popliteal artery pseudoaneurysm as a nosological entity is rare due to its high morbimortality, either with reversible ischemia with loss of function of the affected limb or loss of the limb and, sometimes, even death of the patient due to incoercible bleeding. In this case, it was mistaken for a pathology of osteoarticular origin, and the medical anamnesis and physical examination played an essential role in making the diagnosis certainty.

Declarations
The authors declare no conflict of interest.

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