Reversal of Electrical Storm after Intra-Aortic Balloon Pump Counterpulsation

Balloon pump counterpulsation has been used for stabilization of refractory ventricular arrhythmia both in coronary and non-coronary patients; its usefulness for complex circulatory support is still under consideration.

We report a case of electrical storm reversal after intra-aortic balloon pump counterpulsation in a patient without coronary artery disease and under biventricular support with continuous-flow devices.

Our case is a 44-year-old male patient with idiopathic dilated cardiomyopathy and severe impairment ventricular function (22%) determined by echocardiography; he was admitted to the intensive care unit due to progression of dyspnea in functional class II to IV with urine output deterioration. He presented a cardiac index (CI) of 1.2 L/min/m2 and 35 mm Hg of wedge pressure. He received diuretics and dobutamine at an initial intravenous dose of 5 µg/kg/min, increasing the dose to 15 µg/kg/min due to oliguria, acidosis, and persistent low CI.

The patient developed complex ventricular arrhythmia (frequent ventricular extrasystoles, couplets and triplets, and several episodes of self-limiting ventricular tachycardia), which was treated successively with magnesium, lidocaine, and amiodarone.

Due to the potential dobutamine proarrhythmic effect, milrinone therapy (0.5 µg/kg/min) was started in order to reduce the dobutamine dose.

The following day, under dobutamine 2.5 µg/kg/min and milrinone 0.75 µg/kg, the patient developed sustained ventricular tachycardia and required defibrillation, orotracheal intubation and mechanical ventilation, and underwent implantation of balloon pump counterpulsation with amiodarone load and maintenance.

In the next 12 hours, under balloon support, the patient remained without complex forms of ventricular arrhythmia, but due to his progression to anuria with metabolic acidosis and CI of 1.2 L/min/m2 (under milrinone 0.75 µg/kg/min), percutaneous placement of a CentriMag continuous-flow left ventricular assist device (Levitronix LLC, Waltham, Mass) and balloon removal was decided. Subsequently, and given the severe right ventricular dysfunction resistant to pharmacological therapy, a second CentriMag right ventricular assist device was placed.

Two hours after the intervention, the patient developed electrical storm that was refractory to lidocaine, amiodarone loading, pacing attempts –suppression – and multiple defibrillations; he persisted with ventricular tachycardia for 24 hours (Figure 1).

In such circumstances, balloon pump counterpulsation was reimplanted to stop the arrhythmia, which was resolved 2 hours after the intervention.

Four days after the procedure, under balloon and

Fig. 1. Electrical storm (A & B) under circulatory support with 2 CentriMag devices (C).
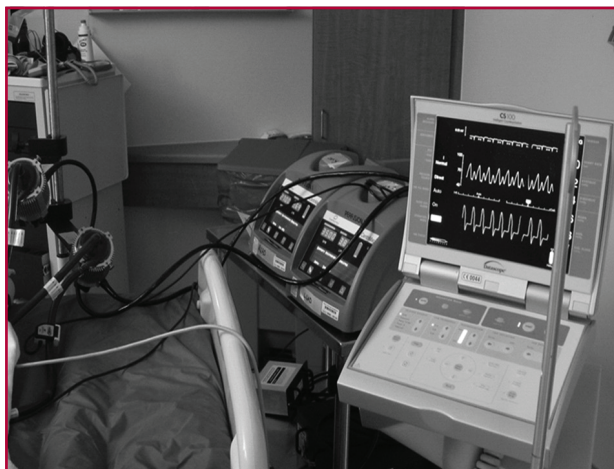

Fig. 2. Resolution of arrhythmia after the reimplantation of balloon pump counterpulsation.

the two continuous-flow device assistance (Figure 2), the patient recovered urine output, CI gradually improved to 1.9 L/min/m2, and the process of weaning from the continuous-flow devices was started, with echocardiographic monitoring to detect the gradual improvement in both ventricles.

On the 6th day, the patient was extubated, and both devices were successfully removed in the following 48 hours. Balloon support was maintained for another 48 hours.

During that period, the patient remained free from complex ventricular arrhythmia.

Complex ventricular arrhythmia is frequently found in patients with impaired left ventricular function, which sometimes does not respond to regular therapies -such as electrolyte correction, antiarrhythmic agents, and electrical defibrillation-, posing a significant management problem.

Ischemia is commonly the substrate for persistent

arrhythmia, which can be resolved with coronary artery bypass procedures, for example. When ischemia is not detected or resolved, other methods such as pacing suppression or balloon pump counterpulsation are considered.

(1) In a retrospective series of 21 patients, Fotopoulos et al. reported the stabilization of refractory ventricular arrhythmia using intra-aortic balloon counterpulsation, and included 3 patients without coronary artery disease, while Goyal et al. described the effectiveness of intra-aortic balloon pump for the treatment of complex refractory ventricular arrhythmia in a patient with dilated cardiomyopathy and normal coronary arteries. (1, 2)

The effectiveness of balloon pump for refractory arrhythmias in coronary patients is evident when correcting imbalance between oxygen supply and demand, due to increased coronary perfusion and reduced cardiac work. The effectiveness of the device is not so evident in non-coronary patients.

Fotopoulos, mentioned above, proposed an indirect mechanism mediated by reduction of adrenergic tone, reducing myocardial vulnerability to arrhythmias. The balloon also reduces ventricular systolic pressure, lowering wall and myocardial oxygen tension. (1) Another proposed mechanism is the direct effect of the balloon based on the concept of mechano-electric feedback or contraction-excitation feedback.

This concept is supported by studies on animals showing which post-load increases result in increased-ventricular ectopy and tachycardia.

From a physiological viewpoint, increasing myocardial stretch results in shortening of action potential duration, abnormal refractoriness and increased diastolic depolarization, while the effect of the balloon -reducing stretch and distension- benefits myocardial responsiveness.

A similar phenomenon was described in humans by Taggart et al, when detecting the proarrhythmic effect of increased loading conditions.

(1, 3) On the other hand, the use of a balloon together with complex circulatory support devices is an uncommon combination, with limited references in the literature.

Swinney et al. analyzed the use of balloon pump counterpulsation in combination with the HeartMate II continuous-flow device in 51 patients, and observed that the first one augmented systolic flow and decreased diastolic flow of the second one, by increasing pulsatility (targeted by an increased pulsatility index in the continuous-flow device console).

(4) Ma et al. analyzed the combination of extracorporeal membrane oxygenation (ECMO) with intra-aortic balloon pump in 34 patients, 15 of whom underwent ECMO first. This action involved the opening of the aortic valve, which improved with reduction of afterload due to balloon pump counterpulsation. While the devices in both references differ from the one we have described, the basic functional physiological

mechanisms (continuous-flow devices) are equivalent, the opposite effect to increased afterload and its potential antiarrhythmic action resulting attractive.

(5) Hu et al. described the case of two patients with cardiogenic shock and electrical storm.

Those patients required circulatory support with ECMO, developing refractory ventricular arrhythmia which was resolved with balloon implant, as in our case. The authors posed the efficacy of the device in reducing left ventricular congestion by modifying ECMO non-pulsatile flow through the balloon.

(6) While evidences are somewhat speculative, it is evident that the use of the balloon in our patient resolved the ventricular arrhythmia in two instances, which was refractory to conventional treatment approaches. The usefulness of the balloon as antiarrhythmic treatment was evident in this complex scenario.

Ricardo Levin MTSAC**, Marcela Degrange** MTSAC**,**
John Byrne, Rafael Porcile MTSAC**, James Mykytenko**
Department of Cardiology, Hospital Universitario,
Universidad Abierta Interamericana
Buenos Aires, Argentina
Vanderbilt University Medical Center - Nashville, USA
Ricardo Levin
Portela 2975 - CABA
rllevin@gmail.com

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Urgent Percutaneous Closure of Patent Foramen Ovale in a Patient with Platypnea - Orthodeoxia Syndrome after Right Upper Lobectomy

The platypnea-orthodeoxia syndrome (POS) is characterized by dyspnea and hypoxemia in upright position, relieved by recumbence. (1) Pre-existing intracardiac shunting such as patent foramen ovale (PFO) is one of its most common causes.

Platypnea-orthodeoxia syndrome is caused by a

right-to-left shunt. (2) This shunt can occur without increased pulmonary pressures, and is due to anatomic conditions that redirect flow from the right atrium (RA) to the left atrium (LA), more evident in upright posture, although atrial pressures are higher. (1)

We present a case of POS secondary to PFO in a patient who recently underwent right lobectomy due to lung neoplasm.

His clinical presentation included rapidly progressive respiratory failure refractory to oxygen therapy, so there was an urgent need for PFO closure. The procedure was successfully performed with immediate improvement of dyspnea and hypoxemia.

A 70-year-old male patient came to the emergency room with an episode of sudden dyspnea in the upright posture. Physical examination revealed oxygen desaturation increasing with upright position to 80%, and hypoventilation in the right lung, with no signs of cardiac failure. He referred several episodes of dyspnea in upright position for a month. He had a history of hypertension, stroke of unclear etiology in 2009 (echo Doppler of supra-aortic trunks, NAD), colon adenocarcinoma treated with surgery and chemotherapy in 2009, and squamous cell carcinoma of the lung treated with right upper lobectomy 5 months ago.

On admission, a CT scan of the lungs showed volume loss of the right upper lobe, residual middle lobe collapse, and right hemidiaphragmatic elevation related to a past surgery.

No signs of pulmonary thromboembolism were observed.

Blood tests revealed polycythemia (hematocrit 41.5%), which was not present in previous tests.

In the absence of a pulmonary condition to justify the clinical manifestations and the refractoriness of hypoxemia to oxygen therapy, a transthoracic echocardiography (TTE) was performed to rule out intracardiac shunting.

Baseline TTE showed no signs of pulmonary hypertension. An agitated saline bubble test showed a massive passage of bubbles at baseline in the first and second beats, suggestive of right-to-left shunt. With these findings suggestive of POS due to PFO, a transesophageal echocardiography (TEE) was planned for PFO closure.

Due to rapidly progressive clinical deterioration in 24 hours, with severe refractory hypoxemia (65% O2 Sat) both in upright and recumbent positions, the patient was transferred to the intensive care unit under high-flow noninvasive mechanical ventilation, and an urgent TEE was performed.

Transesophageal echocardiography showed atrial septal aneurysm (ASA) with significant detachment of the oval fossa membrane and massive passage of bubbles with agitated saline from RA to LA. Given the patient's abrupt worsening with sustained hypoxemia that could not be relieved with posture or oxygen therapy, urgent percutaneous closure of the PFO was decided. The procedure was performed under intra-
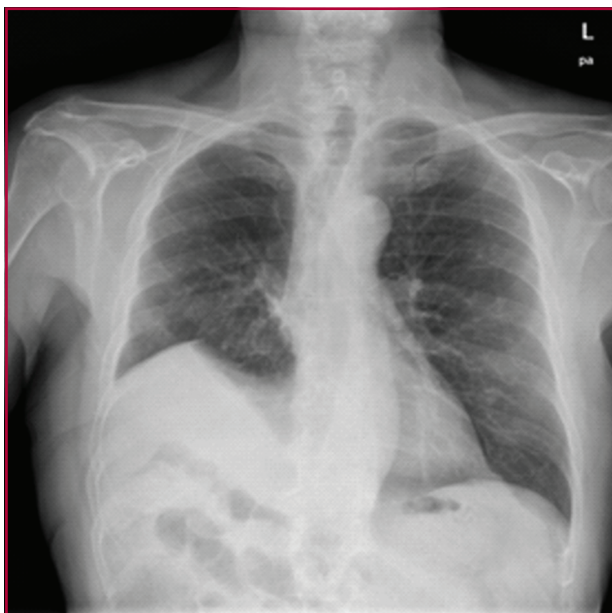
Fig. 1. Chest X-ray showing volume loss of the right upper lobe, residual middle lobe collapse, and right hemidiaphragmatic elevation.
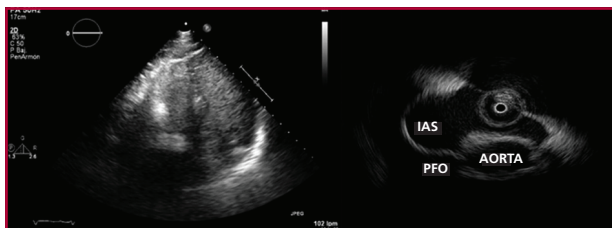
Fig. 2. A. Transthoracic echocardiography after bubble infusion showing massive passage of bubbles in the first beat.
B. ICUS prior to PFO closure, showing patent foramen ovale (PFO) and aneurysmal interatrial septum.

cardiac ultrasound (ICUS) and fluoroscopy.

A Figulla® Flex II 31/35 device was placed without incidence.

After PFO closure, immediate increase of O2 Sat from 66-70% to 95-97% was evidenced, with disappearance of dyspnea. Two months later, the patient remained asymptomatic, without evidence of residual shunt in TTE and with 97% O2 Sat both in upright and recumbent positions.

Platypnea-orthodeoxia syndrome can be explained by three main mechanisms: intracardiac shunting, pulmonary vascular shunting, and ventilation-perfusion mismatch.

Patent foramen ovale is the most common cause of intracardiac shunting due to its high prevalence in the general population (10-24%). (2) Hypoxemia results from significant right to left shunting.

There are certain situations that can course with flow inversion in the context of normal pulmonary pressures, due to a transient increase in right atrial

pressure (hemodynamic cause) or to a redirection of flow (anatomic distortion). (3) The extrinsic compression in the RA either by a hydrothorax or a pneumonectomy could increase intracavitary pressure, facilitating right to left shunting.

(1) On the other hand, anatomic conditions that modify the relation between the inferior vena cava (IVC) and ASA in upright position can redirect blood flow to the PFO.

(4) Several mechanisms can lead to this anatomic distortion, such as a prominent Eustachian valve, aortic dilation, or a pneumonectomy. (5) In the case of severe hypoxemia secondary to PFO associated with history of right upper lobectomy presented here, the hemithorax with volume loss and the right hemidiaphragmatic elevation might have caused RA compression with increased intracavitary pressure, and redirected blood flow from IVC to PFO. Both mechanisms would cause a right to left shunt first in the upright position and then also in recumbence, due to the displacement and compression of the RA as a result of the right hemidiaphragmatic elevation, the shunt being responsible for dyspnea and hypoxemia. The presence of polycythemia revealed a certain degree of hypoxemia that could be explained with the occurrence of POS a few months before, by the time of the pulmonary surgery.

Platypnea-orthodeoxia syndrome secondary to PFO is a rare cause of dyspnea but should be considered for differential diagnosis due to its high morbidity.

It should be particularly suspected in patients with hypoxemia in the upright position, refractory to oxygen therapy, improving with recumbency, and with possible precipitating factors such as a history of lobectomy. In situations with significant anatomic distortion, right to left shunting could occur in all the positions and cause sustained severe hypoxemia, as was our case, the percutaneous closure of PFO being the only effective treatment.

Elena Rivero Fernández, Marta López Ramón, María Lasala Alastuey, Pilar Lapuente González, Ana Marcén Miravete, Isabel Calvo Cebollero
Department of Cardiology.
Hospital Universitario Miguel Servet.
Zaragoza, Spain
Elena Rivero Fernández
Phone: 0110034655434251
e-mail: elenariverofernández@gmail.com

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Early Detection of Left Ventricular Noncompaction in a Newborn

Left ventricular noncompaction is a cardiomyopathy characterized by excessive trabeculation of the left ventricle and deep intertrabecular recesses that communicate with the ventricular cavity. It is rarely diagnoses early in life. We report the case of a newborn, where color echo-Doppler in the first day of life showed: Left ventricular noncompaction (LVNC) due to evidence of excessive and prominent trabeculation and deep intertrabecular recesses on the left ventricular lateral wall and apex (Figure 1).

Intertrabecular recesses were filled by direct blood flow from the ventricular chamber.

Relation between LV noncompaction and LV compaction: 2.3.

Good left ventricular function.

Heart failure occurred at 14 days of life, color Doppler echocardiography showing dilatation of the left chambers, reduced systolic function, and a shortening fraction of 27%.

ECG: Right atrial overload, biventricular damage with right ventricular predominance, repolarization disturbances. Furosemide and beta-blockers were indicated, and inotropes were added at 26 days of life due to severe heart failure.

The patient died at 28 days of life due to multiple organ failure. Pathology confirmed the diagnosis: noncompaction cardiomyopathy (spongiform cardiomyopathy).

Gross examination: cardiomegaly; horseshoe kidney: small, polycystic left kidney; agenesis of the corpus callosum. Microscopical examination: myocardium with endocardial invaginations penetrating the ventricular wall thickness; endocardial fibroelastosis (Figure 2).

Left multicystic dysplastic kidney.

In 1990, Chin et al (1) described, for the first time, the isolated noncompaction of left ventricular myocardium. In 1995, the World Health Organization included it as a non-classified cardiomyopathy, and since 2006, it has been reclassified as genetic primary cardiomyopathy.

(2) According to the most widespread and accepted pathogenic theory, LVNC represents a detention in endomyocardial morphogenesis, causing excessive and prominent trabeculation and deep intertrabecular recesses.

This trabeculation often develops in the free wall and in the inferior portion of the interventricular septum, separating the ventricular inflow tract from the outflow tract. Usually between the 5th and the 8th week of gestation the myocardium matures from the

epicardium to the endocardium and from the base to the apex thanks to myocardial compaction of the myocardial trabeculae, with increasing thickening of the compact myocardial layer and reduction of the trabecular layer, especially in the LV. The epicardial coronary arteries are usually normal in those patients and do not have contact with intertrabecular recesses.

At the same time, a detention in the normal development of the annulus fibrosus could explain the possible association of LVNC with Wolff Parkinson White syndrome.

Left ventricular noncompaction is genetically heterogeneous and can be inherited as an autosomal dominant or associated with cardiac or noncardiac disorders, or as part of genetic syndromes.

Over 50% of the rare cases described would be familial in the form of dominant autosomal transmission or associated with the X chromosome. The exact prevalence of this disease –probably underdiagnosed– is unknown.

In pediatric patients, most diagnoses are made during childhood, rarely in newborns.

In adults, the range is wide, slightly increased between 20 and 40 years of age. In children, LVNC represents between 7% and 9.5% of the total cardiomyopathies, after dilated and hypertrophic cardiomyopathy (3, 4); in adults, the prevalence is 0.014%. The man/woman ratio is 1.8:1. Left ventricular noncompaction can develop due to severe, early heart failure, ventricular arrhythmias, and systemic thromboembolic events.

Diagnosis is made with color Doppler echocardiography and the criteria defined by Jenni et al are the most widely used (5):

1. Absence of any other cardiac abnormalities.
2. Presence of excessive and prominent trabeculation and deep intertrabecular recesses, especially in the segments of the mid lateral, mid inferior wall and apex.
3. Color Doppler evidence of perfused intertrabecular recesses from the ventricular chamber.
4. The relation between the compacted and noncompacted ventricular portion measured at the end of systole for better visualization of the two layers
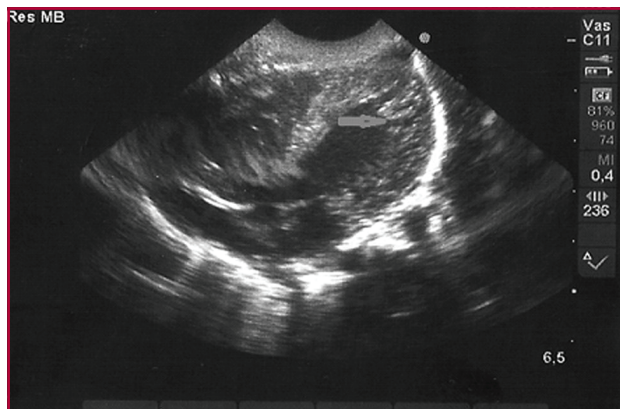

Fig. 1. Trabeculation with deep intertrabecular recesses.
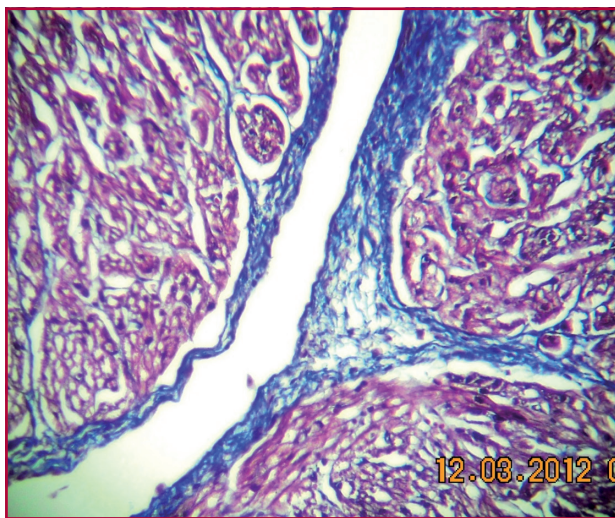

Fig. 2. Masson's trichrome technique. 400×.
Endocardial processes penetrating the myocardial thickness.

equal to or greater than 2.

In turn, the criteria proposed by Stollberger et al. include the presence of at least three trabeculations in the LV wall, apically from the insertion of the papillary muscles, visualized within one echocardiographic imaging plane.

(6) The LV systolic function is diminished both in noncompacted and normal segments, probably due to subendocardial hypoperfusion and reduced microcirculation, which could also be the cause of arrhythmias. Diastolic dysfunction can be associated with restrictive filling and abnormal relaxation caused by the excessive ventricular trabeculations.

Treatment is aimed at alleviating the clinical manifestations, since there is no specific therapy for this disease. Inotropes, beta-blockers, angiotensin-converting enzyme inhibitors, and diuretics can be administered for congestive heart failure.

The presence of malignant ventricular arrhythmias is indication for an implantable cardioverter defibrillator. Prolonged prophylactic anticoagulation has been recommended to prevent embolic complications with or without thrombus. The only definitive treatment is heart transplant.

Long-term survival is usually poor (70-75% at 5 years), and death generally occurs suddenly or due to heart failure.

Only a few publications refer to this disease diagnosed during the neonatal period.

Three cases of neonates with congestive heart failure and severe fetal hydrops were published by Halbertsma et al. in 2001, Kitao et al. in 2004, and Autumm et al. in 2009. Another two patients were diagnosed at days 2 and 10 of life; one was treated with milrinone prior to heart transplant, and the other also had complete AV block and long-QT syndrome.

A patient with fetal bradycardia, sinus node disease, and postnatal long-QT syndrome was diagnosed

at day 1 of life. Shane published a retrospective series of 46 infants with LVNC between December 1999 and February 2005, 9 of which were diagnosed on the first week of life.

Fetal echocardiographic diagnosis of LVNC is difficult to make.

The importance of this presentation lies in that LVNC, confirmed by pathology, is a cardiomyopathy that can be diagnosed in a cardiologically asymptomatic newborn on his first day of life with color Doppler echocardiography.

Graciela M. Chernovetzky[1], M.D. Oppizzi[2], Mónica Rittler[3], Cintia Hernandorena
Hospital Materno Infantil Ramón Sardá.
[1]Coordinator of Perinatal Cardiology
[2]Pediatric Cardiology – Department of Cardiology
[3] Department of Genetics
[4] Department of Pathology
Graciela M. Chernovetzky, M.D.
Hospital Materno Infantil Ramón Sardá.
Esteban de Luca 2151 - (1246) CABA, Argentina
e-mail: gchernovetzky@yahoo.com.ar - Cel.
15-4400-0876

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Endovascular Treatment in Patients with Critical Limb Ischemia

Critical ischemia is the most advanced form of peripheral vascular disease.

These patients have a bad prognosis, with a mortality rate between 19-54% at 1 year (1) and an amputation rate >25% at 6 months in non-revascularized patients or when revascularization fails.

(1) Endovascular treatment represents a new therapeutic approach.

(2) The purpose of this report is to analyze the technical success and the mid-term outcomes of endovascular treatment in patients with critical ischemia treated in the Department of Interventional Cardiology and Endovascular Therapy of the Hospital Italiano de Buenos Aires.

A retrospective analysis of 70 patients with critical ischemia, consecutively treated between October 2011 and June 2014, was carried out. Patients underwent endovascular therapy as first choice treatment.

Inclusion criteria were pain at rest and/or trophic ulcers or gangrene (Rutherford IV-V-VI).

The goals were to evaluate the technical success, periprocedural mortality, and major amputation rate.

We define "technical success" as the patency of aortoiliac, femoropopliteal, and infrapatellar regions with at least one patent vessel to the foot, with residual luminal stenosis less than 30% and absence of dissection involving flow in follow-up post revascularization angiography.

Perioperative mortality has been defined as death that occurs within 30 days of surgery.

Finally, major amputation has been defined as any amputation of the lower limb above the ankle.

Of the 70 patients with critical ischemia under treatment, 59% were men, and mean age was 71.2±14.3 years (range 36 - 92 years).

The reason for admission was pain at rest in 26% of patients, while 74% presented with tissue loss. Risk factors are defined in Table 1.

Contralateral femoral and antegrade femoral approaches were followed. Infrapatellar retrograde access was used in 9 patients in whom femoral access had failed. Percutaneous transluminal angioplasty (PTA) was performed in 29 of 70 patients, and the remaining 41 underwent PTA and stent placement.

Revascularization of one arterial territory was performed in 50% of patients, two territories were revascularized in 32 patients, and 3 patients underwent revascularization in three vascular territories.

Table 2 summarizes the distribution of lesions by arterial territory according to TASC II classification. (3)

Sixty-one percent of the treated arteries were occluded, while the remaining 39% had stenosis >70%.

In our series, recanalization was technically successful in 68 (97.1%) of the 70 patients, antegrade revascularization (femoral approach) was used in 61 patients, and the retrograde approach (infrapatellar approach) in 7 patients. The two failures were due to the impossibility of identifying a major patent vessel in the foot via angiography -only collateral circulation was identified. Two patients presented residual stenosis >30% following PTA, resulting in a technical success rate of 94%.

Twenty-two patients had iliac lesions, 19 of which were TASC C-D lesions. Of the 42 patients with femoropopliteal involvement, 24 had TASC C-D lesions. Technical success was 100% in both groups. Patients

Table 1. Risk factors

Factor	n (%)
Diabetes	40 (57.14)
Hypertension	64 (91.42)
Dyslipidemia	50 (71)
Active smoker	9 (12.5)
Ex-smoker	28 (40)
Chronic renal failure	7 (10)
Coronary heart disease	22 (31.42)

Table 2. Classification of aortoiliac, femoropopliteal, and infrapatellar lesions

Aortoiliac lesions (n=22)		Aortoiliac lesions (n=22)		Aortoiliac lesions (n=22)	
TASC II	n (%)	TASC II	n (%)		n (%)
A	2 (9.09)	A	6 (14.28)	Anterior tibial	24 (38.70)
B	1 (4.54)	B	12 (28.57)	Posterior tibial	13 (20.96)
C	5 (22.72)	C	7 (16.6)	Peroneal	25 (40.32)
D	14 (63.63)	D	17 (40.47)		

were clinically followed up for a mean period of 13 months (range 1-25 months).

During that period, 44 patients (62.85%) were asymptomatic, 20 patients (29%) showed subjective clinical improvement and favorable course of their ulcers according to the Texas classification.

(4) Seven reinterventions were performed: 4 due to tissue loss and 3 due to pain at rest. Six major (supracondylar) amputations and 10 minor (digital) amputations were done.

Overall mortality during the follow-up period was 7 patients (10%), and none of them was associated with the procedure. A patient died on the 10th day of the periprocedural period, secondary to pneumonia. In the literature, the overall failure rate ranges between 20% and 40%.

Even though this rate falls up to 20% in experienced hands, it will never be <10%.

(2) That level of failure is associated with long, heavily calcified chronic occlusions. The infrapatellar retrograde approach, with technical success rates >90%, can favorably modify that failure rate.

(2) In a study of 343 patients, Montero-Baker et al. reported an overall technical success rate of 95% using retrograde approaches in 51 patients due to antegrade failure. (2) The percentage of major complications for this type of access is low; Montero-Baker et al. reported 1.9% for major complications and 7.9% for minor sequelae at the access site.

(2) In our series, we had no associated complications with any of the approaches followed.

Multisegmental involvement is common in these patients. Revascularization rate in long femoropopliteal and iliac lesions surpasses 95% of cases. (5) Amputation rate with endovascular treatment showed limb salvage figures similar to those with bypass surgery. However, successful revascularization as the only parameter for limb salvage is not enough, since it depends on the clinical presentation and follow-up of each patient.

As for the limitations of our work, it should be noted that it is a descriptive, retrospective study in a small cohort of patients, carried out in a single center.

While a mean follow-up of 13 months is reported, the interval is wide and the results need to be interpreted in a prudent manner. Finally, endovascular treatment in patients with critical limb ischemia is, at least in our experience, feasible, safe and effective. It offers a high rate of recanalization and a low rate of complications. At present, retrograde approaches represent an essential tool to achieve those rates of recanalization.

While this study is only descriptive, its findings are promising and consistent with the literature.

It is very important to continue with records and follow-up.

Martin Rabellino[1], Adolfo Figueroa Casoliba[1], Ignacio Bluro[MTSAC, 2], Sergio Shinzato[1], Vadim Kotowicz[3], Ricardo García-Mónaco[1]
Hospital Italiano de Buenos Aires Buenos Aires, Argentina
[1] Department of Interventional Cardiology and Endovascular Therapy
[2] Department of Cardiology.
[3] Department of Cardiac Surgery
J. D. Perón 4190, Ciudad de Buenos Aires (C1181ACH), Argentina
Phone: +54-11-4959-0453
Fax: +54 (-11) -4959-0471
E-mail: jose.rabellino@hospitalitaliano.org.ar

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