Acute Myocardial Infarction Due to Coronary Embolism in a Young Woman with Mechanical Aortic Valve Prosthesis and Anomalous Origin of Two Coronary Vessels: A Case Report

Heart valve disease affects approximately 2.5% of adults in developed countries. Since 1960, valve replacement with mechanical prostheses is one of the therapeutic alternatives for the management of valve disease. Its main complication is the development of thrombosis or embolic phenomena, with an estimated annual incidence of 0.3-1.3% and 0.7-6%, respectively. (1) The risk is increased in the first months of implantation, depending on its anatomical position and its association with other thromboembolic risk factors (e.g., atrial fibrillation).

We present a case of ST-segment elevation acute myocardial infarction in a young woman with a prosthetic aortic valve who had voluntarily discontinued anticoagulation.

A 23-year-old woman, from the Colombian Pacific region, with a history of mitral regurgitation and mechanical valve prosthesis implantation at 8 years of age, was anticoagulated with warfarin until 2 years ago when she discontinued medical treatment. She consulted the emergency department due to 8 hours of high-intensity oppressive chest pain radiating to the right upper limb, with no other associated symptoms. On physical examination, she was afebrile, with blood pressure of 121/76 mmHg, heart rate 82 bpm, and respiratory rate 19 rpm. Auscultation revealed a grade III/VI holosystolic murmur in the mitral focus, and a grade III/VI diastolic murmur in the aortic focus, with no signs of acute heart failure and no other relevant findings. The electrocardiogram showed sinus rhythm with ST-segment elevation from V1 to V3 and inferior ST-segment depression, with the presence of pathological Q waves in leads I and aVL, and signs of left ventricular enlargement. Emergency coronary angiography was performed 12 hours after admission, which documented a total occlusion of chronic appearance in the mid-proximal segment of the left anterior descending artery (Figure 1) and a thrombotic lesion in the first obtuse marginal of the circumflex artery, with 90% stenosis (Figure 2), without other angiographically significant lesions. Loss of mobility of one of the hemidiscs of the double-disc mechanical valve prosthesis was evidenced, due to in situ thrombus.

An anomalous origin of the right coronary artery and a second marginal obtuse artery, independently from the left coronary sinus were demonstrated as incidental findings. Due to the high risk of prosthetic thrombosis, it was initially decided to anticoagulate the patient with low molecular weight heparin and warfarin until international normalized ratio (INR) goals were reached. Laboratory tests showed positive

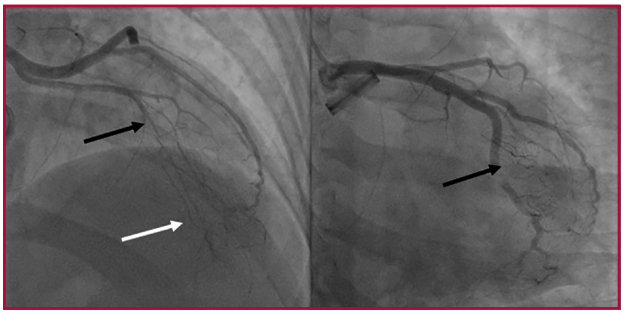

Fig. 1. **Left**: Chronic occlusion of the left anterior descending artery at the junction of the proximal to middle segment (black arrow) with heterocoronary and homocoronary collateral circulation (white arrow). **Right**: Obtuse marginal artery with filling defect compatible with thrombus generating subocclusion and TIMI 2 flow (black arrow).

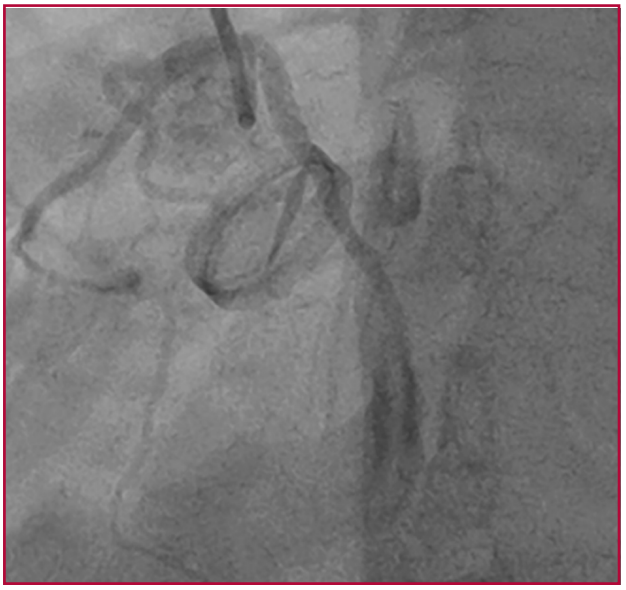

Fig. 2. Loss of mobility of one of the hemidiscs of a mechanical double disc prosthesis due to in situ thrombus

cardiac troponin I (6.53 ng/mL for a normal upper limit of 0.12 ng/mL). Transesophageal echocardiogram revealed akinesia without thinning of the anterolateral and inferolateral walls, with a left ventricular ejection fraction of 47% by Simpson's method. It also showed mechanical aortic prosthesis in adequate position with restriction of the posterior leaflet movement, and presence of pannus and marked turbulence in the antegrade flow, with maximum velocity of 3.2 m/s and a mean gradient of 22.5 mm Hg, as well as severe mitral regurgitation secondary to perforation of the anterior leaflet.

With these findings, she underwent aortic valve prosthesis replacement using a n° 23 Medtronic mechanical prosthesis. Enlargement of the aortic an-

nulus with a heterologous pericardial patch using Manougian´s technique, and repair of the mitral valve with closure of the anterior leaflet orifice were performed. Fresh thrombi in the aortic mechanical prosthesis at the hinge level of both discs and severe subvalvular pannus were found. It was not possible to perform revascularization of the anterior descending artery as its course could not be visualized due to the presence of epicardial-pericardial adhesions from the previous surgery, so coronary angioplasty was indicated. The second coronary angiography performed 25 days after admission revealed complete resolution of the thrombotic lesion in the obtuse marginal artery. The total occlusion of the left anterior descending artery persisted, but it was not possible to perform percutaneous revascularization as it was a vessel with a small caliber. It was decided to continue medical treatment, accompaniment by the Psychology and Education service, and she was discharged after 41 days of hospitalization, without complications, with an INR of 3.2 and indications for strict medical control.

Ischemic heart disease is the leading cause of death worldwide, mainly associated with atherosclerosis. Significant atherosclerotic lesions are not found in up to 7% of cases. Coronary embolism is a cause of non-atherosclerotic infarction, and it is estimated that it represents 3% of all myocardial infarctions. It generally affects the left coronary circulation, (1) as in the case of our patient.

The main associated causes are atrial fibrillation, cardiomyopathies, presence of prosthetic valves, endocarditis, tumors, and prothrombotic conditions. Coronary thrombosis associated with acute infection by SARS-CoV-2 during the pandemic has been reported for this entity. (2) Before the use of prosthetic valves, endocarditis was the main cause of death; now atrial fibrillation is mainly considered. (1) Currently, prosthetic valve replacement is the gold standard for the management of severe valve disease in patients with low or intermediate surgical risk. Mechanical valves have a longer life, but are prothrombotic, which requires indefinite anticoagulation to prevent valve thrombosis and embolic events.

There are three types of coronary embolism: direct, paradoxical, and iatrogenic. Direct coronary embolism occurs when an embolus enters the coronary circulation from the left ventricle, left atrial appendage, pulmonary veins, and the aortic or mitral valve. (3)

The clinical, electrocardiographic, and echocardiographic manifestations of myocardial infarction due to coronary embolism are indistinguishable from infarction of atherosclerotic origin, and it should be suspected in patients with prothrombotic risk factors who present with sudden chest pain. (4)

There are currently no guidelines for the management of coronary embolism. Intracoronary thrombus aspiration vs. angioplasty alone has been tested in patients with ST-segment-elevation myocardial infarction, without demonstrating an additional benefit in mortality. However, patients with a high thrombotic burden, such as patients with coronary embolism, could benefit more from this measure. (5) In cases of coronary embolism, systemic thrombolysis with tissue plasminogen activator (t-PA) has been reported to be successful in restoring coronary flow. Karakoyun et al (5) effectively and safely treated three patients with coronary embolism associated with prosthetic valves with low-dose intravenous t-PA. Similarly, intravenous infusion of bivalirudin for 48 hours in coronary embolism of the distal right coronary artery has been described, with complete resolution of the thrombus without major bleeding. (6) Other therapies include balloon angioplasty, which has been shown to be successful in restoring blood flow, both as isolated treatment and as adjunctive therapy to thrombotic aspiration. (5)

In conclusion, we describe the case of a young woman with mechanical aortic valve prosthesis who voluntarily discontinued anticoagulation and who presented an acute myocardial infarction due to coronary embolism. This condition is potentially fatal, so adherence to pharmacological treatment and education about the disease is essential in a patient at high risk of thrombosis. Permanent anticoagulation, strict clinical monitoring and education are the most important measures to prevent new events.

Conflicts of interest

None declared.

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Ethical considerations

Not applicable.

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Extracorporeal Ventricular Assistance in In-hospital Cardiac Arrest: A Feasible Reality in Our Setting?

Extracorporeal cardiopulmonary resuscitation (ECPR) is the use of extracorporeal membrane oxygenation (ECMO) in patients in whom standard cardiopulmonary resuscitation (SCPR) measures do not achieve a sustained return of spontaneous circulation after cardiac arrest (CA). (1) Patients undergoing ECMO implantation during or immediately after CA have a particularly unfavorable prognosis. (2)

Despite there are no current systematic recommendations on the indication of ECMO in CA, it could be considered an emerging therapy in selected cases when SCPR fails. (3) At present, no randomized controlled trials have been reported comparing the results of ECPR versus SCPR in in-hospital CA (IHCA). (1) Though numerous cohort studies have shown that this therapy is associated with a higher survival rate until discharge, and with favorable neurological results, (4) to our knowledge, limited information has been published in our setting.

The aim of this study was to analyze and report the characteristics and clinical results of a retrospective and consecutive cohort of adult patients treated with ECPR after IHCA in a high complexity center of Argentina.

Patients over 18 years of age treated with venoarterial (VA) ECMO for IHCA between 2014 and 2022 were analyzed. The study included patients with witnessed IHCA, possibly of cardiac origin (mainly ventricular tachycardia or ventricular fibrillation as initial rhythm, extending for more than 20 minutes), (1) even with adequate CPR since its onset. Patients with CA during cardiac surgery were excluded from the study. Table 1 summarizes the inclusion criteria for ECPR at our center.

An analysis of the ventricular assistance database, which is prospectively completed, including among its main variables, demographic characteristics, information on the type of ventricular assistance, complications, relevant clinical events and clinical evolution, biochemical and echocardiographic predictors was performed. Regarding relevant clinical events, two types of survival were evaluated:

- Survival in ECMO: It assesses survival in ECMO and up to 24 hours from ventricular assistance weaning. In this case, the reasons for weaning from ECMO are cardiac function recovery or heart transplantation.

Table 1. Inclusion criteria for ECPR

Age <70 years
In-hospital CA
Time of first CA onset <5 minutes
Initial cardiac rhythm: ventricular fibrillation, ventricular tachycardia, or pulseless electrical activity
Estimated time from CA to ECMO flow <60 minutes
Recovery from intermittent spontaneous circulation or from recurrent ventricular fibrillation
Absence of previously known life-limiting comorbidities

CA: cardiac arrest; ECMO: extracorporeal membrane oxygenation

- Survival at discharge. It evaluates survival at hospital discharge, either by release from hospital or referral to another healthcare center (e.g., third level of rehabilitation).

In addition, neurological complications, brain death (irreversible loss of consciousness and neurovegetative functions, including breathing capacity) and stroke (acute neurological focus and new ischemic or hemorrhagic changes in brain computed tomography) were analyzed.

The analysis included 8 patients, representing 11.9% of VA ECMO implanted during this period in the center. Median (interquartile range, IQR) age was 46 years (IQR 30-58) and 66% were women. Three patients had history of hypertension and dyslipidemia and one of diabetes. No patient presented with previous history of obstructive pulmonary disease, chronic kidney disease, stroke, peripheral vascular disease, atrial fibrillation, or anemia.

Three patients presented with acute coronary syndrome, two with electrical storm and the remaining causes were peripartum cardiomyopathy, myocarditis, and unidentified restrictive cardiomyopathy.

Cannulation was peripheral in 87.5% of cases (7 patients). The same number of patients required use of intra-aortic balloon pump, and 2 cases needed surgical left ventricular decompression, through pulmonary vein venting. In all the cases, ECMO was implanted as bridge to recovery.

Median circulatory assistance duration was 5 days (IQR 2-8). Successful VA ECMO weaning was achieved in 5 patients.

The rate of survival in VA ECMO was 62.5% (n=5) and at discharge 37.5% (n=3). The cause of death was non-cardiovascular in 4 of the 5 deaths.

Complications included major hemorrhage (66%), non-dialytic acute kidney failure (66%), infection (33%), seizures (11%) and thromboembolic complications (33%). No brain death was reported, and one patient suffered an ischemic stroke.

Median follow-up after discharge was 14 months (IQR 7-30). One of the 3 surviving patients is on the waiting list for elective heart transplantation, and 2 are followed-up with preserved biventricular function.

There is an increasing worldwide use of ECPR as a rescue technique in patients with refractory CA. Although controlled randomized trials are still missing demonstrating its efficacy in this setting, observational studies have reported 20% to 40% survival. (5) Currently, there is no sufficient data available to identify patients who could benefit from ECPR. It is internationally recommended to establish agreed inclusion criteria in each center to guide physicians on how to balance the intelligent use of resources among patients who are believed to have a better probability of survival after CA. (2) In our center, inclusion criteria were standardized since the creation of the multidisciplinary "ECMO team" (Table 1), considering that decision making for ECPR is often time critical and influenced by external factors such as hours and day of the week. It is therefore essential to present with adequate logistics, 24/7 trained staff for cannulation (as it is recommended that ECMO is functioning within 60 minutes after CA) and for the fast assembly and purge of the device in the emergency, and healthcare professionals who can detect within 10 minutes of CA the possible ECPR candidates.

Protocols and algorithms endeavor to quickly identify the cases with higher probability of survival with a favorable neurological outcome, as well as patients with witnessed CRA in whom high-quality CPR was quickly administered, and also cardiac arrests with a presumably reversible disorder, such as acute coronary obstructions. (2) Other factors which may influence ECPR indication are age, cause of CA, time, comorbidities and cardiac rhythm at CA onset. (3) Recently, the RESCUE-IHCA survival predictive score derived from 1075 patients was published, showing 28% survival at discharge, and identifying 6 variables associated with in-hospital mortality: age, time of day, initial rhythm, history of kidney failure, type of patient (cardiac vs. non-cardiac and clinical vs. surgical) and duration of cardiac arrest. (5) The greatest probability of success occurs in a young patient (in some working teams 50 years of age is considered the limit for ECPR), with few comorbidities, with a witnessed CA, preferably during daytime (when logistics is easier and there is more access to trained staff), with adequate CPR maneuvers performed immediately (preferably in intensive care units), and of cardiac origin, with a shockable initial rhythm.

Our results are comparable to those reported by the ELSO (Extracorporeal Life Support Organization) international multicenter registry, in which ECMO survival was 41%, and at hospital discharge 30% at an international level, (6) and to the results of the RESCUE-IHCA. (5)

In our center, VA ECMO as treatment for IHCA presented an acceptable survival at hospital discharge, and it can be considered an effective treatment in highly selected patients when conventional therapies fail, being useful and applicable in a country with low and medium income and limited access to circulatory assist devices. Probably these results cannot be extrapolated to other centers of the region, as our institution is a referral VA ECMO high complexity monovalent cardiovascular center, with a developed care program, more than 7-year experience and currently, with more than 15 implants per year. Although the number of patients included in this series was low, it is still a novelty, as it would be the first experience published analyzing the results of VA ECMO in refractory IHCA in our country.

Conflicts of interest

None declared.

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Ethical considerations

The study was conducted according to research principles (Declaration of Helsinki) and was approved by the institutional Ethics Committee.

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Posterior Embolic Stroke Secondary to Subclavian Artery Thrombosis

Posterior stroke (PS) together with upper limb ischemia is an infrequently associated clinical presentation caused by embolism, aortic dissection, vascular trauma, thoracic outlet syndrome, coagulation disorders and, less commonly, subclavian artery thrombosis.

A 59-year-old male patient, former smoker (40 pack/year), hypertensive and dyslipidemic, presented with a 2-week gait disorder and referred a left upper limb hypotension record. Upon consultation with his occupational physician, expressive aphasia, right temporal hemianopsia and gait instability determined the decision to hospitalize him. The electrocardiogram confirmed sinus rhythm, and the echocardiogram evidenced preserved left ventricular systolic function, without shunt or intraluminal thrombi. Neck vessels Doppler ultrasound showed subintimal carotid plaques, without significant hemodynamic findings, and very low flow velocity in the left vertebral artery. Brain computed tomography (CT) revealed hypodense frontoparietal white matter areas in both hemispheres. Brain magnetic resonance angiography showed acute left temporo-occipital ischemic lesion in the left posterior cerebral artery territory, with absence of flow in the intracranial segment of the vertebral artery (Figure 1).

On the second day the patient referred left upper limb paresthesia, and lower temperature was detected with absence of humeral, radial, and ulnar pulses.

Left upper limb arterial echo-Doppler revealed very low velocity monophasic flow, and high resistance in the humeral, radial and ulnar arteries, with subclavian artery thrombosis. Neck vessels and aortic arch computed tomography angiography ruled out aortic dissection and thoracic outlet syndrome and showed complete left subclavian artery thrombosis from its origin, and part extending as intraaortic thrombus, and altered homolateral vertebral artery staining in the intraosseous and intracranial segments (Figure 2A). Thorax, abdomen, and pelvis CT scan revealed bilateral pulmonary emphysema. Laboratory results were: platelets 373 000 ml/mm3, normal D-dimer and IgG and IgM antiphospholipid antibodies, negative lupus inhibitor, normal homocysteine, negative anti-beta 2 antibodies and IgG and IgM glycoproteins, protein C 108% and free protein S 66%. Electrocardiographic 24-hour Holter monitoring indicated predominant sinus rhythm, without ventricular or

supraventricular extrasystoles. Control brain magnetic resonance imaging (MRI) performed on the 4th day did not evidence hemorrhagic temporo-occipital lesion transformation.

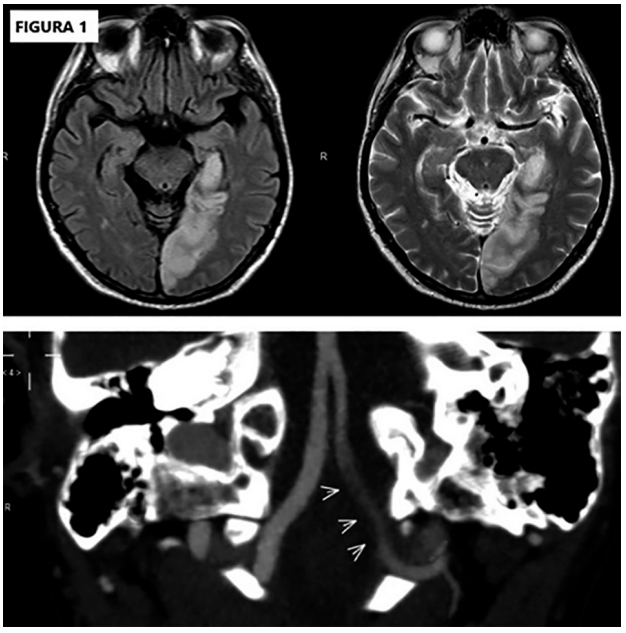

Fig. 1. Brain magnetic resonance angiography: acute left temporo-occipital ischemic lesion with absence of flow in the intracranial segment of the vertebral artery

Endovascular or surgical treatment for complete left subclavian artery thrombosis with intrathoracic extension associated with posterior ischemic stroke was discarded due to high risk of systemic embolic complications, and anticoagulation by continuous pump sodium-heparin infusion was decided, under strict neurological control and subsequent rotation to oral acenocoumarol. The patient was discharged on the 10th day with no neurological or upper limb ischemic complications. At 1-year oral anticoagulation was suspended due to recurrent episodes of hematuria and hematemesis, and the patient continued with oral clopidogrel and cilostazol. The last control computed tomography angiography at 3 years evidenced partial recanalization of the subclavian thrombosis, with complete disappearance of the intraaortic thrombus in the subclavian ostium. (Figure 2B)

The patient is currently asymptomatic, with humeral pulse recovery. Studies for thrombophilia were repeated with negative results.

Almost 20-25% of strokes occur in the posterior circulatory system (posterior cerebral, basilar and vertebral arteries) and can compromise the brainstem, cerebellum, thalamus and/or temporo-occipital region. Caplan et al. described embolism as the most frequent mechanism of posterior stroke (40-54%), mainly of cardiac origin (24% of cases), while arterio-arterial embolism was reported only in 14% of cases. (1) Other mechanisms are atherosclerotic lesions of the great arteries, small vessel occlusion, and rare causes such as coagulation disorders or carotid atheroembolism associated with the fetal origin of the posterior cerebral artery. Due to the wide cerebral area irrigated by

the vertebrobasilar arterial system, ischemic strokes in this territory exhibit with various signs and symptoms. The most frequent signs are gait ataxia, unilateral limb weakness, dysarthria, nystagmus, and visual field defects, while reported symptoms are usually vertigo, dizziness, nausea and vomiting, headache, and consciousness disorders.

Subclavian artery thrombosis occurs due to vascular wall intimal injury. Atherosclerosis is its most frequent etiology, and is more commonly located in the right carotid-subclavian and left subclavian-vertebral areas, so these regions are usually involved in occlusive thrombosis. Subclavian atherosclerosis risk factors are hypertension, smoking, diabetes, obesity, and dyslipidemia. (2) Subclavian artery thrombosis presents in less than 1% of the population and is generally asymptomatic, resulting in an underdiagnosed disease. Left subclavian thrombosis is four time more common that its right counterpart. (2)

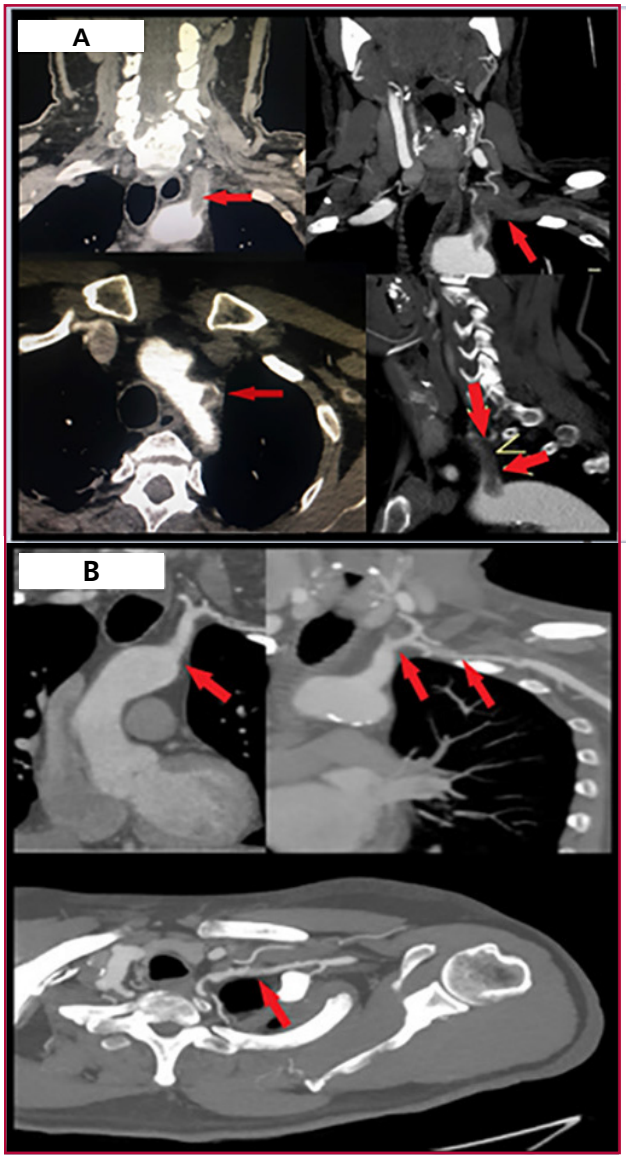

Fig. 2. A. Neck vessels and aortic arch computed tomography angiography: complete left subclavian artery thrombosis from its origin, with part of intraaortic thrombus. **B.** Control computed tomography angiography at 3 years: partial recanalization of subclavian thrombosis, with complete disappearance of the intraaortic thrombus in the subclavian ostium

The emergence of ischemic symptoms as a result of decreased arterial flow due to subclavian artery thrombosis is conditioned to the presence or not of collateral circulation, and the most usual clinical manifestations include upper limb intermittent claudication and paresthesia. Subclavian artery thrombosis complications are upper limb, mainly digital, ischemic gangrene, acute ischemia of the limb artery and rarely, posterior ischemic stroke. The pathogenesis of these infarctions in the vertebrobasilar territory is due to arterio-arterial embolism or the "retrograde" propagation to the vertebral artery from a homolateral subclavian artery thrombosis. (3,4)

Other causes of subclavian artery thrombosis associated with posterior stroke are hypercoagulability states (S-protein deficit, essential thrombocytopenia, etc.), aortic dissection, arterial trauma, cardiac embolism, and thoracic outlet syndrome with arterial involvement (arterial TOS). The latter is characterized by a subclavian artery disease due to compression by osseous anomalies as a cervical rib, with intimal lesion with or without post-stenotic dilation and thrombus formation prone to distal embolization, generating severe complications such as upper limb arterial ischemia and less frequently a posterior ischemic stroke. (3-6)

Castillo Costa et al. reported a case of posterior stroke with upper limb ischemia due to thrombosis of a structurally healthy aorta with systemic embolism. (7)

Computed tomography angiography of the aortic arch and the compromised upper limb allows confirming the diagnosis of subclavian artery thrombosis as possible embolic source in patients with posterior stroke, as well as identifying some of its causes (atherosclerosis, arterial TOS, dissection, trauma, etc.).

Treatment of subclavian artery thrombosis complicated with a posterior stroke will depend on the degree of upper limb ischemia and the vascular disease that originated it. In general, endovascular or surgical therapeutic interventions (embolectomy or decompression treatment with arterial TOS revascularization) are only indicated in patients presenting threatened upper limb, due to the risk of systemic embolization during the intervention. (3,4) Anticoagulant treatment through initial intravenous infusion of sodium heparin and subsequent oral anticoagulation is an effective therapy described for patients with subclavian artery thrombosis and posterior stroke coursing with compensated upper limb ischemia, to avoid arterial thrombosis and its progression. (2,5,6)

In conclusion, posterior ischemic stroke is an infrequent complication of subclavian artery thrombosis,

that should be suspected in patients with vertebrobasilar infarctions and absence of homolateral upper limb arterial pulse. Computed tomography angiography can confirm its diagnosis.

Conflicts of interest

None declared.

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Ethical considerations

Not applicable.

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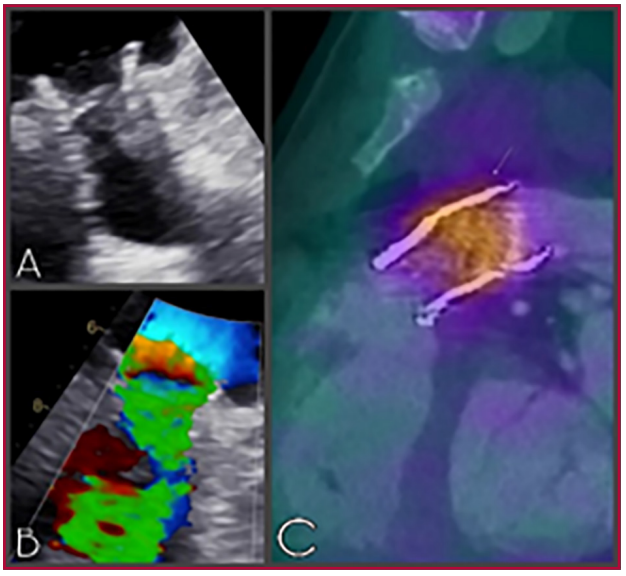

Fig. 1. Lreoperative studies. A, B: Pulmonary valve echocardiogram. C: PET/CT scan with prosthetic uptake.

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Pulmonary Valve Implant Infective Endocarditis. Surgical Resolution with Homograft

We present the case of a female 20-year-old patient who consults for a febrile syndrome of 7 days evolution, with asthenia and adynamia. As a relevant history, the patient presented pulmonary valve stenosis and aortic valve dysplasia at birth, as part of the clinical suspicion of Noonan syndrome. Throughout her growth, the patient had to undergo several surgeries: at one year of age, she was submitted to enlargement of the pulmonary artery outflow tract and plastic repair of the aortic valve; at 13 years, she required aortic valve replacement with a number 18 ATS type bi-disc mechanical prosthesis, with annulus enlargement and pulmonary valve replacement with a Nº 19 Freestyle type biological prosthesis; and then, at 17 years, given the marked increase in gradients through the lung graft, a number 20 Melody type valve was percutaneously implanted. It should be noted that despite the multiple interventions the patient had a normal physical, social, and intellectual development.

Given the relevant cardiological history, it was decided to hospitalize the patient to clarify and identify the focus that caused the fever. As positive data, the gram negative Cardiobacterium bacillus of the HACEK group was identified in serial blood cultures. The transesophageal color Doppler echo showed a marked increase in the Melody-type pulmonary valve gradients, with mobile structures compatible with vegetations (Figure 1. A and B) and the positron emission tomography (PET/CT) scan revealed a clear increase in the uptake of the pulmonary valve region (Figure 1. C).

Considering the clinical diagnosis and complementary studies, the febrile condition was interpreted as endocarditis of the pulmonary endoprosthesis, so a new surgery was performed to replace the clearly infected prosthesis and the pulmonary artery outflow tract with a number 21 homograft. The prosthetic aortic valve was undamaged, so its replacement was not required (Figure 2. A, B and C).

The postoperative evolution was satisfactory and the antibiotic therapy included ceftriaxone and gentamicin according the sensitivity of the Cardiobacterium. At one- year follow-up, the patient leads a normal, hemodynamically stable life, free of cardiac infection, with normal functioning of the pulmonary homograft.

The risk of infective endocarditis (IE) after percutaneous Melody pulmonary valve implantation (MPVI) is significant, at least during the first 3 years after implantation. However, the reported incidence varies considerably between different studies.

In a meta-analysis that included 851 patients, the cumulative incidence of IE on MPVI ranged from 3.2% to 25%, with an annualized incidence rate ranging from 1.3% to 9.1% patient-years. The median (interquartile range) time from MPVI to IE onset was 18 months (9-30.4), with a range between 1 and 72 months. The incidence of IE occurred in 32% of cases in the first year, 27% in the second year, 18% in the third year, and 23% beyond 3 years of MPVI. (1)

In a study conducted by McElhinney et al. in 309 patients with a follow-up of nearly 5 years, multivariate analysis found age under 12 years at the time of MPVI (OR 2.8; 95% CI 1.3–5.7; p=0.006) and a maximum gradient immediately after implantation greater than 15 mmHg (OR 2.6; 95% CI 1.3–5.2; p=0.008) as IE predictors. (2)

The diagnosis of this type of IE is challenging, especially in terms of documentation of the valve prosthesis infectious process. The modified classic Duke criteria, based on echocardiographic signs, confirm that the IE diagnosis after MPVI is not so simple. It is well known that echocardiography, especially transthoracic echocardiography (TTE), offers only modest sensitivity (30%) for the detection of pulmonary valve vegetations, probably due to the anterior position of the right ventricular outflow tract and prosthetic valve artifacts (stent, valve degeneration, calcification of the conduit, etc.). Moreover, transesophageal echocardiography (TEE), unlike the high sensitivity of IE detection in the aortic and mitral valves, does not always offer added value with respect to the TTE in pulmonary prosthetic valve IE. In the case reported, we believe that the PET/CT scan was able to identify the infectious process in the pulmonary prosthesis and ruled out aortic valve involvement, an extremely important data when planning a surgical strategy.

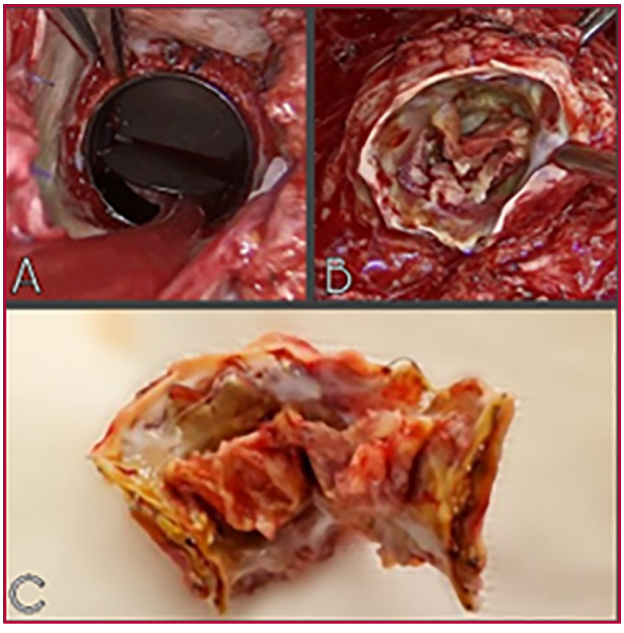

Fig. 1. Intraoperative images. **A**: Intact aortic valve. **B/C**: prosthesis in pulmonary position with clear signs of endocarditis

18F-FDG PET/CT combines a technique with high sensitivity to detect inflammatory-infectious activity and high anatomical resolution to assess structural lesions associated with endocarditis. With 91-97% diagnostic sensitivity, PET/CT has become a useful diagnostic tool in suspected IE of patients with prosthetic valves and/or devices, becoming a major criterion in the diagnostic algorithm for current guidelines. (3)

The most common clinical findings found in MPVI IE were positive blood cultures (93%), fever (89%), and progressive increase in the pulmonary transvalvular gradient (79%). Vegetations by TTE were detected only in 34% of cases. These data reported in the literature were also presented by our patient, with TTE being too weak to define the origin of the infective condition. In the cited meta-analysis, among 69 patients who developed IE after MPVI, 6 (8.7%) died and 35 (52%) underwent surgical and/or transcatheter reoperation. (1)

The most common germs that have been detected in the blood cultures of patients with IE post MPVI are: Staphylococcus 42%, Streptococcus 30.4%, Corynebacterium 5.8%, HACEK group 4.3% and Haemophilus 2.9%; and negative blood cultures have been detected in just over 7% of patients. (1, 4) Cardiobacterium hominis (germ responsible in our case) is a member of the HACEK group, which produces subacute IE; its natural habitat is the oropharynx .

The microbial entry route is related in most cases to oral processes. However, there are reports in patients with a history of gastroenteritis, cystitis, pneumonia, skin and nail processes, as well as skin tattoos. (5)

The incidence of IE after implantation of a percutaneous pulmonary valve is highly variable as reported in the studies, and it occurs mainly during the first 3 years after the procedure and mostly in the presence of increased transpulmonary gradients. The cardiac history should suggest IE in the presence of a febrile condition of unknown origin. Multi-imaging studies, including PET/CT scan, have been extremely useful in identifying the focus of infection secondary to Cardiobacterium IE in our patient. We believe that the satisfactory resolution of the case was due to the interaction of the members of the cardiology and sur-

gery service in adult congenital pathologies, given the complexity of decision-making and previous surgical interventions. The homograft has allowed us to resolve a complex surgical situation.

Conflicts of interest

None declared.

(See authors' conflicts of interest forms on the website/ Supplementary material).

Ethical considerations

Not applicable.

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