Endovascular treatment of bilateral spontaneous, symptomatic carotid dissection, refractory to medical therapy

Carotid dissection (CD) may occur spontaneously in apparently healthy individuals or in those with weakened arterial wall by a primary arteriopathy, collagenopathies, connective tissue disorders or more commonly after trauma. (1, 2) Carotid dissection or vertebral dissection is a cause of stroke in young patients.

The natural history of CD is not completely understood, and it is generally believed that, despite the involvement of extracranial arteries, stroke secondary to CD may be asymptomatic in some cases. (3-5)

Anticoagulant therapy (ACT) is used in most institutions. This recommendation is based on the idea that the most common mechanism of stroke secondary to arterial dissection is thromboembolism developed from de intimal flap. Another stroke mechanism is hemodynamic failure by critical stenosis of the true arterial lumen. (6)

Recently, endovascular treatment with stenting has been proposed as an alternative to the traditional use of ACT in patients with CD.

Stenting restores vessel caliber and normal circulation, covering the arterial defect, preventing the formation of emboli.

In symptomatic dissections, once the penumbra area of the brain is evidenced, prognosis is usually poor to anticoagulant therapy. (5-7) Therefore, endovascular treatment may be especially appropriate as a means to change the clinical course during the acute phase of CD, which is the clinically dynamic phase of dissection.

We present the case of an emergent stenting performed in a patient with acute symptomatic bilateral CD refractory to drug treatment and in whom neuroimaging indicated the existence of significant hypoperfusion, or a large penumbra area. Stenting modified the patient's clinical course to a favorable therapeutic outcome.

CLINICAL CASE

A 47-year old, right-handed, sedentary male patient consulted for an 8/10 intensity holocraneal headache, drowsiness, weakness in both lower limbs and gait instability. A brain computed tomography (CT) without contrast showed no signs of ischemia, a magnetic resonance imaging (MRI) without contrast evidenced a right hyperintense lacunar temporal image and brain digital angiography (DA) revealed a postbulbar bilateral carotid dissection (Figure 1). Anticoagulant therapy was indicated. After 72 hours of ACT targeted to an INR value of 2, the patient presented spatiotemporal confusion, headache and weakness that prevented walking. Brain CT showed no added ischemic image. The patient received 300-mg clopidogrel loading dose and 16 hours after a new brain DA revealed a moderate left internal carotid artery stenosis and progression of stenosis in the right internal carotid artery (Figure 2), with proximal irregularity, interpreted as the beginning of the dissection, and severe stenosis in the horizontal segment of the petrous portion which was interpreted as the maximum growth of the mural thrombus. This stenosis generated distal hemo-

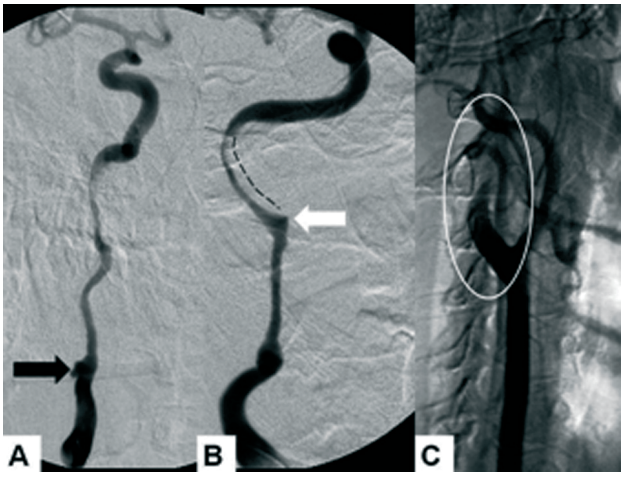

Fig. 1. A. Selective digital angiography of the right internal carotid artery, showing the beginning of the postbulbar flap dissection (*black arrow*). **B.** Distal flap (*white arrow*) and mural thrombus (*dotted line*). **C.** Flow arrest in the carotid bulb during spontaneous hypotension.

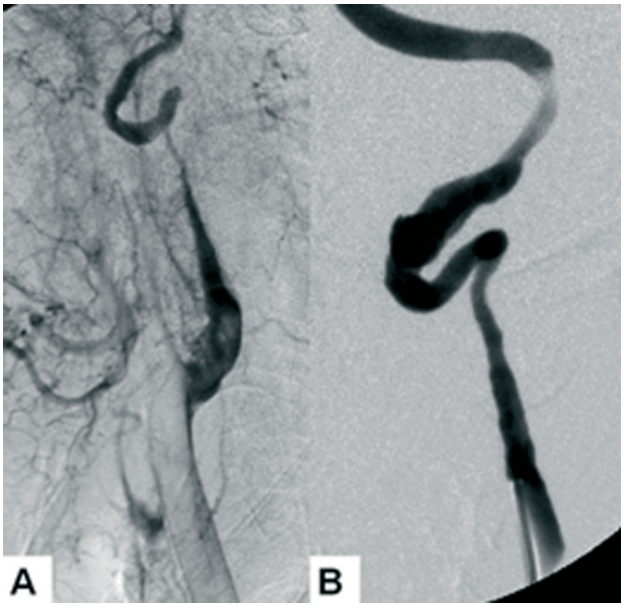

Fig. 2. A. Left carotid, prior to drug treatment, showing severe stenosis secondary to the arterial dissection of the cervical trajectory, with postdissection loop. **B.** Digital angiography of the left carotid, after treatment with anticoagulant drugs showing decreased arterial stenosis and fusiform pseudoaneurysm distal to the carotid loop.

dynamic changes observed in the three times of the parenchymogram, showing contrast passage through the anterior communicating artery, from left to right.

During the angiographic procedure decreased blood pressure (BP) was observed, reaching a systolic blood pressure (SBP) of 80 mm Hg, accompanied by postbulbar right carotid flow arrest, which reversed with increasing BP. Endovascular treatment with stenting was decided.

A 6 Fr ENVOY guiding catheter over a 0.035 inch hydrophilic wire was introduced under general anesthesia and systemic heparinization (sodium heparin bolus infusion10000 I.U, reinforced with 1500 I.U. per hour) and advanced to the right common carotid. A 0.021 Prowler Select Plus microcatheter (Cordis Neurovascular) with a 300 cm long, 0.014 inch Transcend microwire (Boston Scientific Corporation, Fremont, CA) was then placed and the distal segment was catheterized at the point of maximum stenosis. The microcatheter was exchanged and a 4.5 × 37 mm self-expanding Enterprise Neuro-stent (Cordis Neurovascular) was advanceded in apposition, covering the point of maximum stenosis. A self-expanding 5 × 40 mm Precise stent (Cordis) was then telescopically placed covering the extracranial cervical segment of the internal carotid artery. Because of the presence of more than 30% stenosis in the petrous segment, not amenable to balloon angioplasty, a balloon-expandable 4 × 18 mm Liberte stent (Boston Scientific Corporation, Fremont, CA) was impacted at 8 atm, showing no residual stenosis and distal hemodynamic change, with symmetry in the parenchymogram (Figure 3).

The patient was transferred to ICU maintaining drug therapy. Aspirin/clopidogrel 100 mg/75 mg/day and anticoagulation were indicated for 3 months due to persistent left carotid dissection. Controls by Doppler echocardiography at 30 days and a digital brain angiography at 90 days showed patency of the right carotid artery and pseudoaneurysm in the postbulbar region of the left carotid artery, which did not generate stenosis or distal hemodynamic changes. The patient returned to work without neurological deficit.

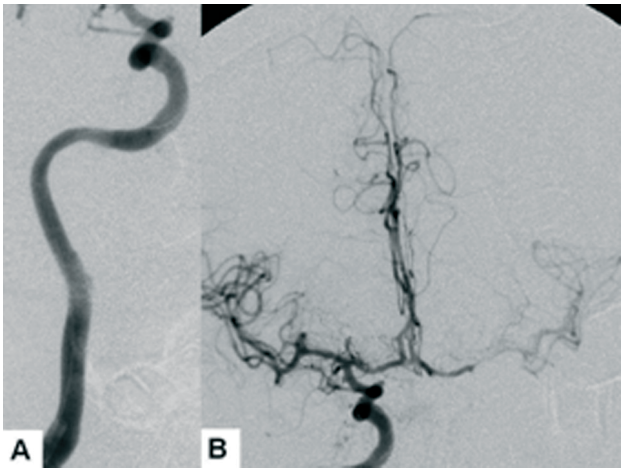

Fig. 3. A. Right carotid digital angiography after treatment with endovascular stenting. **B.** Brain digital angiography after stenting showing brain hemodynamic change in symmetrical arterial time parenchymogram, evidencing spontaneous contrast passage to the anterior contralateral circulation through the anterior communicating artery.

DISCUSSION

Carotid dissection is a dynamic process, and hence neuroimaging and clinical findings may change dramatically in a matter of days or hours. (1) Most dissections are finally resolved. About two-thirds of occlusions are recanalized and a third evolves as pseudoaneurysm. (1-3) The healing process may take 2 to 3 months after dissection, and occasionally longer. Imaging studies suggest that the highest ratio strokes due to CD are secondary to thromboembolic events. Transcranial Doppler ultrasound studies demonstrate a high rate of microemboli. (2) These observations led to the common practice of anticoagulation targeted to an INR of 2.0 to 3.0 for 3 to 6 months.

Intravascular stenting involves less risk than surgical treatment and, in many cases, has replaced surgery as the treatment of choice once drug treatment fails. Stenting is an attractive alternative to other forms of therapy, as it allows immediate artery recanalization and the resolution of the dissection with immediate reperfusion of the penumbra area. (5-7)

Stent eliminates the need for surgical repair procedures, avoids ACT, leaving the patient under the effects of antiplatelet agents.

The main limitation of endovascular treatment with stenting is associated with the specific technical difficulties: selective microcateterization of the true arterial lumen is often very difficult; the arterial segment to be treated with stents is usually long and often requires real stent overlapping (telescoping), with recommendation of stent placement from healthy-to-healthy segments.

Despite the relative advantages of CD endovascular treatment, there are no guidelines as to which patients may benefit from this procedure. In our case, the decision to use stents was based primarily on the clinical impression of impending stroke in the right cerebral hemisphere.

Neuroimaging is helpful in selecting patients for endovascular treatment, as it clearly and precisely evidences penumbra area.

The therapeutic value of this procedure in selected patients with neuroradiological evidence of imminent ischemia may be deduced from the rapid resolution of ischemic symptoms after endovascular treatment and positive results in angiographic and echo-Doppler neck vessels control studies. In conclusion, our case report showed that CD endovascular treatment with stenting may be considered in patients with neurological deficit (caused by embolism or hemodynamic failure) despite the best drug therapy, and that angioplasty with stent placement shows that it is a feasible and effective solution for the symptomatic patient with CD severe stenosis in the presence of neuroradiological signs of cerebral penumbra.

The use of MRI (to detect perfusion-diffusion) and the angiographic data (parenchymograph) are necessary to select patients with penumbra signs and exclude those with irreversible changes. (5)

Pablo Antonio Angelani, Jorge Miano, Jorge Mrad
Hemodynamic and Interventional Neuroradiology Service, Clínica Olivos, Vicente López, Pcia. de Buenos Aires, Argentina
Dr. Pablo Angelani
angelanipablo@yahoo.com.ar; pabloangelani@intramed.net
Radiologist and Interventional Neuroradiologist
Hemodynamic Service, Clínica Olivos

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Therapeutic hypothermia in patients presenting out-of-hospital cardiac arrest secondary to ventricular fibrillation

We present two case reports describing feasibility of hypothermia in patients with out-of-hospital cardiac arrest secondary to ventricular fibrillation (VF).

CASE REPORT 1

This is the case of a 56-year old male patient with no history of cardiovascular disease and smoking, hypertension, dyslipidemia and obesity coronary risk factors (CRF), who presented with out-of-hospital cardiopulmonary arrest (CPA).

Basic cardiopulmonary resuscitation maneuvers were performed at 2 minutes of event presentation. He was transferred to our hospital within 20 minutes, where ventricular fibrillation (VF) rhythm was verified. Basic and advanced cardiopulmonary resuscitation maneuvers (CPR) were performed [following the ABC according to ACLS guidelines (Advanced Cardiac Life Support)] reverting the heart to sinus rhythm after electrical defibrillation, with pulse recovery at 30 minutes of CPR initiation.

The ECG revealed complete AV block with narrow QRS. Atropine was administered, with evolution to sinus rhythm with inferoposterolateral ST-segment elevation. Therefore, an emergency coronary angiography (CA) and placement of intra-aortic balloon counterpulsation was decided. The CA showed severe injury in the middle third segment of the right coronary artery (RCA), 100% occlusion of the circumflex artery (CX) and moderate damage of the first diagonal artery. Conventional stenting angioplasty (CSA) was performed in the RCA and in the CX (door-to-balloon time: 30 minutes). Swan-Ganz: mean arterial pressure 68 mmHg, central venous pressure 13 mmHg, wedge pressure 31 mm Hg, cardiac output 3.9 L/min, cardiac index 2.3 L/min/m2, systemic vascular resistance 1750 dyn/seg/cm-5 and pulmonary vascular resistance 200 dyn/seg/cm-5.

Since the patient met requirements for hypothermia therapy, its initiation was decided with the Cincinnati Sub-Zero cooling system (Figure 1) in order to take the patient to 33 ° C. The equipment consists of a central control panel and two blankets that are placed on the back and front of the patient (Figure 2).

The echocardiogram showed inferoposterior akinesia of the posterior septum and posterior wall (apex to base) with hypokinesia in the rest of the segments and an ejection fraction of 30 %.

Hypothermia was discontinued at 16 hours of onset due to hemodynamic instability with hemodynamic profile of low cardiac output requiring maximum doses of inotropic support. Passive rewarming reaching 36.5 ° C was achieved 8 hours after suspension of induced hypothermia, with partial improvement of the decreased cardiac output allowing lower doses of inotropics and intra-aortic balloon counterpulsation removal.

Subsequently, the patient improved multiorgan failure and analgesia and sedation were interrupted. At 72 hours he showed reactive pupils and a Glasgow score of 15/15, leading to extubation. He remained in the Cardiovascular Intensive Care Unit for 14 days.

He was discharged 28 days after admission.

CASE REPORT 2

A 63-year old male patient with as a history of hypertension, smoking and chronic obstructive pulmonary disease as CRF, presented with necrotic ischemic dilated cardiomyopathy and severe impairment of the left ventricular (LV) function.

He was admitted to the emergency room of our hospital brought by family physicians with CPA. Basic out-of-hospital CPR time was 10 minutes.

On admission VF rhythm was found. Basic and advanced

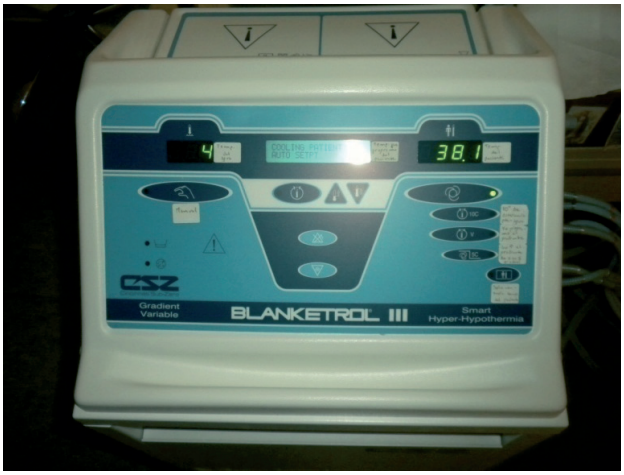

Fig. 1. Equipment for induction of therapeutic hypothermia.

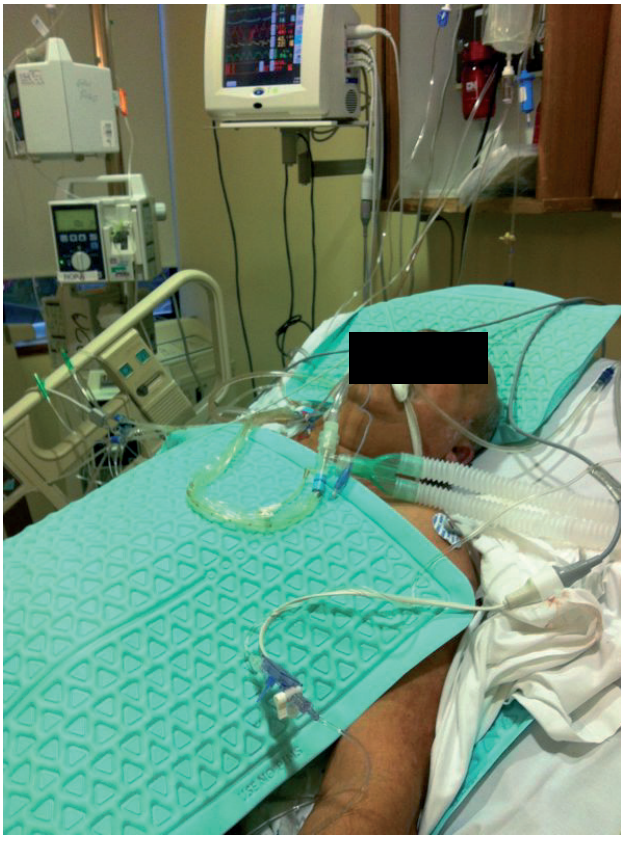

Fig. 2. Equipment for induction of therapeutic hypothermia connected to the patient.

CPR maneuvers (following the ABC ACLS guidelines) with electrical defibrillation were performed and pulse recovery was achieved after 30 minutes.

The total estimated arrest time was 40 minutes.

Emergency CA revealed severe RCA and CX obstruction with no significant damage in the rest of the vessels. Conventional RCA and CX stenting angioplasty plus placement of intra-aortic balloon counterpulsation were carried out. After initial resuscitation, the patient was mechanically ventilated with adequate clinical and hemodynamic parameters, administering moderate doses of noradrenaline and milrinone plus intra-aortic balloon counterpulsation. Swan- Ganz: mean arterial pressure 80 mmHg, central venous pressure 15 mmHg, wedge pressure 29 mmHg, cardiac output 4.2 L/min, cardiac index 2.2 L/min/m2, systemic vascular resistance 1600 dyn/seg/cm-5, and pulmonary vascular resistance 380 dyn/seg/cm-5.

Since the patient met requirements for therapeutic hypothermia, this was decided with the target of bringing the patient to 33 ° C. The echocardiogram showed severe impairment of LV function with an ejection fraction of 27%.

The patient evolved with high doses of inotropic and vasopressor requirements, with multiple episodes of sustained VT requiring electrical cardioversion, leading to suspension of induced hypothermia at 15 hours of initiation.

From a neurological point of view, after suspension of sedation and analgesia for 48 hours, the patient evolved with status epilepticus, refractory to four anticonvulsant drugs. With respect to the respiratory condition, he presented distress criteria with superimposed pneumonia. After 8 days of hospitalization the patient died in asystole.

DISCUSSION

Numerous studies have shown that lowering body temperature, even for a few degrees, decreases ischemic damage. (1) In the normal brain, hypothermia reduces metabolic oxygen consumption slowing the reaction of the temperature-dependent enzymes by 6% for each 1 °C reduction in brain temperature. (1)

There are multiple mechanisms by which mild hypothermia (32-34 °C) may improve neurological outcome when used after reperfusion. It suppresses chemical reactions associated with reperfusion injury, as production of oxygen free radicals, excitotoxic amino acid release, intracellular calcium flow which may lead to mitochondrial damage and apoptosis (programmed cell death). (1) Furthermore, it reduces macrophage and neutrophil phagocytic function and attenuates secondary reperfusion injury after ischemia in other organs.

The prognosis of a patient resuscitated after an out-of-hospital CPR depends largely on neurological involvement.

Multiple studies in patients resuscitated after an "out-of-hospital" CPR secondary to VF or VT have confirmed its benefit.

In 2002, two prospective randomized multicenter clinical trials that compared mild hypothermia (32-34 °C) for 12-24 hours versus normothermia were published. These tests were used as the basis for recommendations and guidelines developed in the last decade. (4, 5) A meta-analysis published in 2005, along with the two above mentioned studies, demonstrated the benefit of hypothermia in patients who survive in a state of comma after out-of-hospital CPR secondary to VF or VT. (1)

The 2002 ILCOR recommendations (International Liaison Advisory Committee on Resuscitation) suggest performing 32-34 °C hypothermia for 12-24 hours in adults with out-of-hospital cardiac arrest secondary to VF or VT (IIa), and they also indicate that hypothermia could be useful in other heart rhythms or in-hospital heart arrest (IIb). These recommendations have also been incorporated in the most recently published guidelines. (6)

The ERC HACA (European Resuscitation Council Hypothermia After Cardiac Arrest) evaluated 650 comatose patients with cardiac arrest at any rhythm and confirmed that hypothermia led to a higher survival rate and neurological improvement. (7)

Candidates for therapeutic hypothermia are: (8)

a) Those with an out-of-hospital CA secondary to VF or VT.
b) Those that subsequently to resuscitation present with no neurological response.

Exclusion criteria for therapeutic hypothermia are: (8)

a) Recovery to a score of 15/15 in the Glasgow scale after initial resuscitation.
b) Age under 18 years.
c) Pregnancy.

d) Hemorrhagic diathesis.
e) Suspicion or documented brain bleeding.
f) Temperature under 30 °C on admission.
g) Patients with terminal illness or limitations to therapeutic effort.

Regarding the clinical cases presented, several issues could be considered. In the first case, total CPR time was shorter (30 vs. 40 minutes).

Furthermore, in the second case, ventricular function was severely impaired prior to CA and the patient had a history of chronic obstructive pulmonary disease, which could lead to a worse outcome from the neurological viewpoint.

In both cases we observed that hemodynamic performance predominantly presented low cardiac output rather than peripheral vasodilation. This could be due to hypothermic attenuation of the systemic inflammatory response.

Induced hypothermia had a transient biological "cost" in pursuit of brain protection: bleeding disorders, possible enhancement of cardiac output and cardiac arrhythmias.

This therapy involves a multidisciplinary approach that begins in the emergency service and includes neurology, hemodynamics, cardiology, therapists, hematology, skilled nursing and kinesiology.

CONCLUSIONS

Therapeutic hypothermia has proved to reduce mortality and improve neurologic outcome. The purpose of presenting these two cases was to demonstrate the feasibility of hypothermia in our center in patients with out-of-hospital cardiac arrest secondary to VF or VT. In both cases, the patients arrived to our hospital with VF, though they could have been defibrillated in situ. This setback could be explained by two reasons: first, there is insufficient availability of AED (Automatic External Defibrillation) in public places and second, health system ambulances arrive at the scene too late.

Federico Lipszyc[1], Victoria Marquevich[2], Pablo Pratesi, José Bonorino[1], Horacio Fernández[1], Jorge Bilbao[1], Alejandro Hita[MTSAC, 1]

[1.] Intensive Care Unit, Hospital Universitario Austral. Buenos Aires, Argentina

[2.] Intensive Care Unit, Hospital Universitario Austral. Buenos Aires, Argentina

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Prosthetic valve obstructive thrombosis in a pregnant patient

Pregnant patients carrying mechanical prosthesis constitute a high risk group for the development of thromboembolic events. Anticoagulation and thrombolytics in case of prosthetic valve thrombosis (PVT) is controversial during this period. We present the case of a patient in her twentieth week of pregnancy, carrying a mechanical mitral valve.

CASE REPORT

This is the case of a 43-year old patient in her 20th week of pregnancy, with history of rheumatic fever and carrying a mechanical mitral valve. In the eighth week of gestation she presented with retroplacental hematoma, so that treatment with acenocoumarol was rotated to suboptimal doses of low molecular weight heparin (LMWH 1 mg/kg/day).

She was admitted with cardiogenic shock parameters (bilateral rales, hypotension -80/40 mm Hg- and oligoanuria), requiring mechanical ventilation and high-doses of inotropic support. Physical examination showed a mitral systolic murmur of 2/6 intensity and absence of prosthetic click. An urgent transesophageal echocardiography (TEE) revealed the mechanical mitral prosthesis, with a large obstructive thrombus (23 × 9 mm) on the atrial surface. The valve area was 0.4 cm2 (PHT), the Doppler mean gradient 30 mmHg and PASP 69 mmHg (Figure 1). Thrombolytic therapy was initiated with administration of a 250.000 U streptokinase bolus followed by continuous infusion of 100000 U/h for 12 hours, and subsequent unfractionated heparin (UFH) by continuous intravenous infusion pump.

She evolved with hemodynamic stability, with no thrombus by transthoracic echocardiography (TTE): the valve area was 4.3 cm2 and the mean gradient 2.4 mmHg (Figure 2). The patient was discharged with LMWH (1 mg/kg every 12 h) treatment, with positive fetal vitality tests, ending pregnancy without presenting new complications.

DISCUSSION

The procoagulant state of pregnancy increases the risk of thrombus formation, and is higher in mitral prosthesis and/or with suboptimal anticoagulation. (1-3) Anticoagulation presents a high risk of bleeding associated to the placenta or cesarean delivery. (3)

Warfarin crosses the placenta and is associated with abortions, malformations and fetal deaths. (2, 3) Unfractionated heparin and LMWH do not cross the placenta, but require two daily administrations and have higher rates of thrombosis. (2) As therapeutic options, LMWH or UFH or oral anticoagulants may be used throughout pregnancy but should be discontinued between weeks 6 and 12 of gestation and from week 36 onwards. (1-3)

Thrombolytic therapy has been developed as an alternative to surgery. In one study, 110 patients with 127 documented episodes of obstructive PVT were treated with different thrombolytic agents. Complete hemodynamic response was observed in 71 % of cases, partial resolution in 17% and no response in 12%. Surgery was performed in 23% of cases due to lack of complete resolution. (4)

A study published in 2011 compared emergency surgery with thrombolytic treatment (rt -PA). Eighteen patients underwent emergency surgery, with two deaths in the perioperative phase and two recurrences.

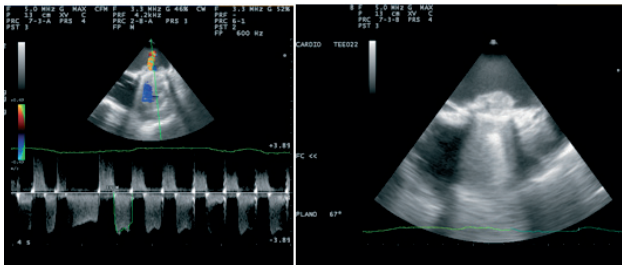

Fig. 1. Transesophageal echo showing thrombus protruding into the left atrium (LA). Mild mitral regurgitation

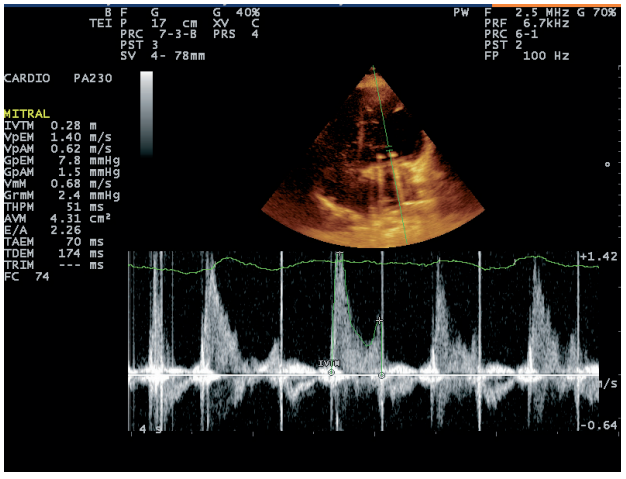

Fig. 2. Transthoracic echo showing the prosthetic valve and the corresponding acoustic shadow, without thrombus evidence. Spectrum of normal filling.

Of the 13 patients treated with thrombolysis, there was immediate clinical improvement in 92%, of which 61 % showed normalization of the echocardiographic findings. Recurrence was observed in 4 patients, with one death. Patients in NYHA FC IV were mainly benefited. (5)

The 2006 guidelines of the American College of Cardiology/American Heart Association (ACCF/AHA) recommend: in left-sided PVT, in functional class III-IV, with a small or large clot, thrombolytic therapy may be considered in case of high risk or unavailable surgery. (1). New guidelines from the American College of Chest Physicians (ACCP) discriminate thrombus size. In patients with left-sided PVT and large thrombus area (> 0.8 cm2) surgery over thrombolytic therapy is advised (Grade 2C). If there are contraindications to surgery, the use of thrombolytic therapy is suggested (Grade 2C). (6)

The optimization of anticoagulant treatment requires proper risk-benefit assessment between hemorrhagic and thrombotic events. Prosthetic valve thrombosis treatment will depend on careful evaluation of surgical risk and the risks associated with thrombolysis. This has the advantage of being accessible in low complexity hospitals without available cardiac surgery, which could be relevant for patients in cardiogenic shock or pulmonary edema needing urgent hemodynamic stabilization.

María del Carmen Solís, Federico Zabala, Luis Medesani, Carlos Zappa, Julia Graieb, Darío Cavalié
Calle 61 n° 585/587 -Piso 1. Dpto. B. La Plata, Bs. As.
e-mail: maria_s_953@hotmail.com.

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