Treatment of a Complicated Chronic Aortic Dissection with Distal False Lumen Embolization

Endovascular repair for complicated type B aortic dissection consists mainly in covering the primary entry site, redirect blood flow to the true lumen, and therefore decompress the false lumen. However, retrograde flow through distal reentries, particularly in chronic patients, makes thrombosis of false lumen difficult. This is the Achilles' heel for chronic patients with aneurysmal dilatation, so that the survival of these patients, related with aortic remodeling, is directly associated with flow persistence in the false lumen.

The original "Candy-plug" technique was described in 2013 by the group of T. Kölbel et al. from the University of Hamburg, who introduced this procedure to facilitate occlusion of aneurysmal dilatation of the false lumen, secondary to chronic type B dissection. The device used was a short stent-graft segment, which was modified on-table, giving it a wrapped candy–like shape. (1) This plug was deployed into the false lumen, thus achieving its thrombosis.

We report a case of complicated chronic type B aortic dissection with aneurysmal dilatation, successfully treated using the plug occlusion technique by means of an aortic extension placed in the false lumen, as the second treatment stage.

An uncomplicated acute type B aortic dissection was diagnosed in 2015 in a 74-year-old man with history of smoking and hypertension, which was controlled with medication. However, aortic dissection progressed with aneurysmal dilation, which at the time of consultation had a maximum transverse diameter of 78 mm. In September 2017, a carotid-subclavian by-pass and aortic endograft placement (Zenith TX2, Bloomington, IN, USA) were performed, covering the origin of the subclavian artery to the origin of the celiac trunk, followed by proximal subclavian artery occlusion with a plug.

Four months after the procedure, the retrograde blood flow persisted through distal reentries in the visceral, abdominal and iliac aortic branches, and the aneurysm expanded to 89 mm at its maximum transverse diameter.

In order to exclude the false lumen without visceral or intercostal flow or lumbar flow involvement, the modified Candy plug technique was used. Under general anesthesia, cerebrospinal fluid (CSF) drainage, strict control of blood pressure and angiographic and intravascular ultrasound guidance (Philips Volcano Intravascular Ultrasound, IVUS, Visions® PV .035 digital IVUS catheter, Volcano Corporation, San Diego, CA), the surgical team proceeded to cannulate the false lumen.

At the same time, a short 36 x 50 mm abdominal aortic segment (Zenith Graft Main Body Extension, Cook Medical, Bloomington, IN, USA) was modified in the operating room. To prepare the cup-shaped device, only the first row of stents was partially deployed. To restrict the opening of this first row, a double 3-0 PTFE suture was placed around it, thus limiting its opening to a maximum diameter of 16 mm, resulting in a shape similar to an inverted cup. Inside the narrowed area, there should be enough space left to remove the distal end of the device carrying the stent (cone) once this was deployed. The stent was then recharged and prepared as usual.

Under IVUS control, the aortic extension was deployed in the false lumen, exactly at the level of the distal end of the previous stent graft placed in the true lumen (Figure 1). Finally, a 22 mm Vascular Plug II Amplatzer (AGA Medical Corp., North Plymouth, MN, USA) was placed in the center of the plug to complete the occlusion. Final angiography showed no residual retrograde flow in the aneurysmal lumen.

The patient was discharged on the second postoperative day without complications.

Computed tomography angiography on discharge showed no endoleak, with complete thrombosis of the false lumen above the occluding device (Figure 2).

The treatment of aneurysmal dilatation of the descending aorta secondary to chronic dissection is, undoubtedly, one of the most challenging problems of vascular surgery. The goal is the complete exclusion of

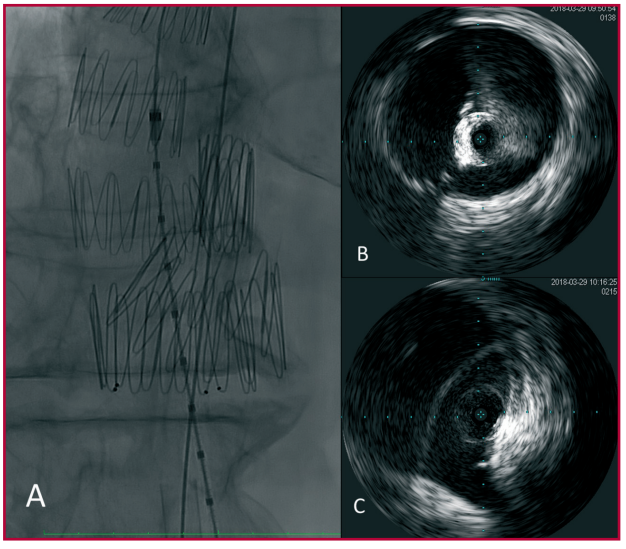

Fig. 1. A. Intraoperative angiographic image showing thoracic endograft in the true lumen, and plug (inverted-cup shaped endograft) in the false lumen. **B and C.** Intravascular ultrasound images both from the true and the false lumen, showing the presence of flow, plug deployment, and celiac trunk origin.

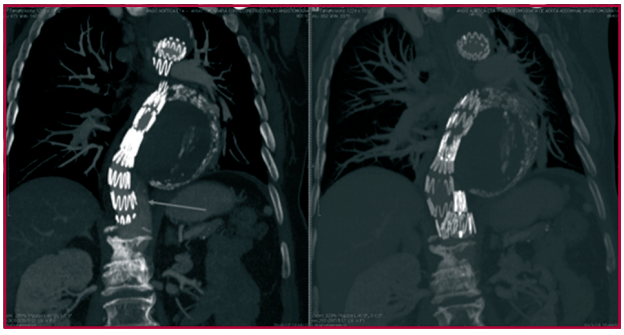

Fig. 2. Preoperative (left) and postoperative (right) computed tomography scan image with evidence of flow in the distal false lumen (red arrow), which disappears after placing the plug.

the false lumen, to achieve its depressurization and, therefore, minimize the risk of rupture.

In these cases, open surgery implies aneurysmal resection through thoraco-phreno-laparotomy, which is performed with sequential aortic cross-clamping, usually with some circulatory complement (partial bypass of the left heart, perfusion technique using branched catheters or axillofemoral bypass) and reimplantation of the intercostal arteries, as well as of the visceral branches. Even in experienced hands, this surgery presents a high morbidity and mortality rate.

On the other hand, endovascular treatment through the placement of fenestrated or branched grafts in the true lumen, even in sequential procedures, implies not only high investment in training, technology and devices but also a high risk of paraplegia close to 12% and mortality rate above 10%. (2)

This is the reason for the development of this approach, combining procedures in the true lumen (endograft placement for occlusion of the entrance orifice, depressurization, and redirection of the flow towards the true lumen) with procedures in the false lumen. Occlusion of the false lumen avoids retrograde flow into the aneurysm without covering distal aortic segments, which would imply an increased risk of paraplegia.

The "Candy-plug" technique is a useful treatment option for false lumen occlusion in chronic type B aortic dissection. Kölbel et al. originally described this technique using a Zenith TX2 ProForm endovascular graft (Cook Medical, Bloomington, IN, USA). (1) Years later, Rholffs et al. reported the first results with this technique in 18 patients. (3) In that publication, the authors demonstrated the feasibility of the method associated with low morbidity and mortality due to its minimal invasiveness. (3)

Our results were similar. We successfully thrombosed the aneurysm with a 36 mm aortic graft for the false lumen, and a second 32 mm graft in the descending aortic artery, both immediately above the celiac trunk. It did not imply increased spinal risk, despite the distal location of both endografts.

A relevant technical detail was the use of intravascular ultrasound. This technique is beneficial, particularly for patients with chronic dissections, with multiple entry and re-entry orifices. In addition, intravascular ultrasound allowed us to verify the exact level at which the stents should be opened to avoid occluding visceral branches. Especially in this case, we also selected the adequate re-entry orifice to introduce the occluder device.

A sequential endovascular repair was successfully performed using the modified 'Candy-plug' technique for a false lumen aneurysm in the descending thoracic aorta. This technique has the potential to effectively occlude the dilated false lumen with low risk of neurological complications.

Conflicts of interest

None declared.

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Unilateral Chronic Thromboembolic Pulmonary Disease: A Different Entity?

Chronic thromboembolic pulmonary hypertension is the only variant of the disease that can be potentially cured with surgery. Recurrence after surgery is rare, but it has been described (1) and would be more frequent in a particular anatomical subgroup of patients. The therapeutic approach in case of recurrence deserves further analysis, as patients currently have not only the surgical alternative but also other options (angioplasty, medical treatment or lung transplantation).

We describe the case of a 41-year-old male patient, ex-smoker, who suffered pulmonary embolism 6 years before hospital admission. He was started on oral an-

ticoagulation and was diagnosed antiphospholipidic syndrome and heterozygous prothrombin gene mutation G20210A. The patient was then referred to surgery due to persistent advanced functional class dyspnea and severe pulmonary hypertension. Pulmonary angiography showed occluded origin of the left pulmonary artery branch, without contralateral involvement. A pulmonary endarterectomy was performed. Exploration of the right branch showed no thrombus, and the left endarterectomy was considered adequate (Figure 1). The patient remained in good functional class during 4 years, and was monitored in his province of residence.

Seven months before readmission, the patient presented with functional class III dyspnea. No new embolic event could be defined by interrogation, but apparently, the administration of anticoagulation might have suffered eventual interruptions. A ventilation/perfusion scintigraphy was performed, showing no perfusion in the left lung, with preserved ventilation. The echocardiography reported dilated right chambers and mild deterioration of the right ventricular function; estimated pulmonary systolic pressure was 93 mmHg. Pulmonary angiotomography showed pulmonary thromboembolism that completely occluded the left pulmonary artery branch and the lower lobe of the right lung. Right catheterization reported pulmonary wedge pressure: 15 mmHg; pulmonary artery pressure: 70/23 (40) mmHg; right atrial pressure: (10) mmHg; pulmonary vascular resistance: 6.2 U and Wood: 499.5 dines. Pulmonary arteriogram showed proximal occlusion of the left pulmonary artery branch and perfusion defects in the right middle and lower lobes.

Due to symptoms, recurrence of thromboembolic disease, and an anatomical condition that could not be resolved with an alternative treatment (angioplasty), it was decided to perform a new surgical treatment.

A bilateral pulmonary endarterectomy revealed a large, relatively recent thrombotic component occluding the origin of the left branch, with a chronic homo- and contralateral distal component. The development of the right plane to perform the endarterectomy was more complex than usual (Figure 2).

During the postoperative course, lung pressure decreased adequately; anticoagulation with heparin sodium was initiated within the first 12 hours after surgery, and extubation was achieved on the first postoperative day.

Invasive pulmonary pressure monitoring was withdrawn on the second postoperative day; the last assessed values were 48/10/27 mmHg for systolic, diastolic and mean pressure, respectively.

Due to progressive thrombopenia, the presence of antibodies against heparin was analyzed on the third postoperative day, with positive result. Given the critical platelet count and the impossibility to use bivalirudin, anticoagulation was discontinued until platelets in the blood smear were >50,000, upon which the patient restarted treatment with acenocumarol. The patient was discharged 15 days after surgery.

Some publications have considered chronic thromboembolic pulmonary hypertension to be a special subgroup in which the anatomical pattern is the complete occlusion of a pulmonary artery branch alone or associated with low involvement of the contralateral lung. (1)

This pattern would be more common in young women, with lower lung pressure values than in patients with bilateral disease. It has been associated with a higher rate of rethrombosis both in the immediate postoperative course as during follow-up. (2)

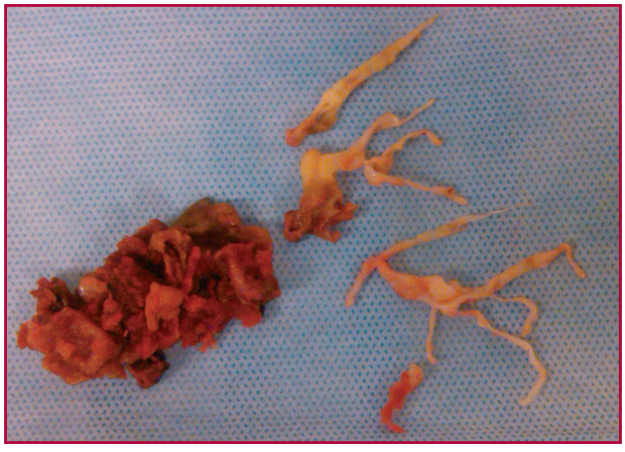

Fig. 1. Surgical specimen of pulmonary endarterectomy in the first intervention.

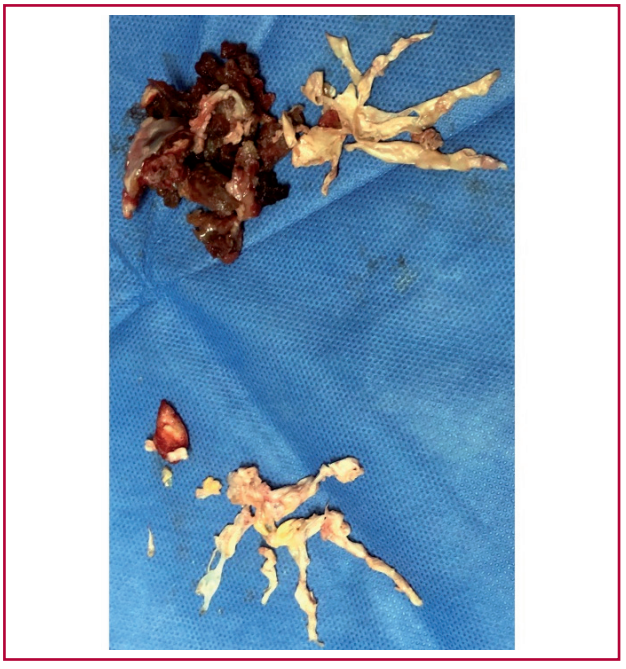

Fig. 2. Surgical specimen of pulmonary endarterectomy in the second intervention.

While the reason for this behavior is not clearly known, it has been described that the bed distal to the complete obstruction could have serious post-obstructive changes, in addition to great development of bronchial circulation.

This development of the systemic circulation -perhaps necessary to maintain the parenchyma vitality- could condition a competitive flow and expose these patients to poor reperfusion (3) and a higher rate of rethrombosis. (4)

All of the above led the San Diego group to change the surgical classification proposed for the disease, based on the branch sizes of the pulmonary artery involved, and to include patients with occlusion of a major pulmonary artery branch in a separate subgroup. (4)

In the case we describe, the patient first underwent pulmonary endarterectomy with bilateral exploration and no targeted right lung disease, either by imaging methods or surgery. Pulmonary hemodynamics was restored, but we did not document the reperfusion quality with scintigraphy.

The patient was asymptomatic for 4 years until recurrence of symptoms with no clinical event leading to suspicion of new embolism. Possible irregularities in the administration of anticoagulants -which the patient referred to as occasional- were analyzed. It is unclear whether the rethrombosis was secondary to new, clinically undetected embolisms or to anticoagulation irregularities in a patient with a major prothrombotic condition. The decision for reoperation was based on age, clinical condition and hemodynamic data, associated with an anatomical pattern that was inadequate for alternative treatments.

Hemodynamic response to surgery was positive, since pulmonary pressure values decreased significantly after the intervention. The ventilation/perfusion scintigraphy showed improved perfusion of the involved lung, and although some discordant defects of peripheral perfusion/ventilation remained in the left lung, the right defects completely disappeared.

Despite publications on reoperation of pulmonary endarterectomy are scarce, (1, 5, 6) all of them agree that increased perioperative risks are to be expected, and perhaps lower functional outcomes than those of the initial endarterectomy due to longer-term disease. Still, the results obtained in patients undergoing surgery are positive, both from the hemodynamic and clinical point of view.

While we do not intend to draw definitive conclusions, we believe that reporting this case helps to highlight characteristics of the disease for which there is no conclusive evidence. We believe that more clinical research is necessary.

Conflicts of interest

None declared.

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Spontaneous Dissection of the Left Anterior Descending Coronary Artery Presenting As Thromboembolic Stroke

A 43-year old woman was admitted because of right hemiparesis and aphasia that had begun suddenly one hour earlier, while she was practicing sports. Her previous history was unremarkable, apart from active smoking. On admission, she presented right-sided hemiparesis and sensitive aphasia, with a NIHSS score of 23. Her physical examination was otherwise normal. ECG showed sinus rhythm at 70 beats per minute, narrow QRS complex and T wave inversion in precordial leads, without evidence of myocardial necrosis. Troponin I assessment was normal, as well as serum biochemistry analysis, coagulation parameters and blood cell count. A multimodal cranial CT scan revealed complete occlusion of the left middle cerebral artery (segment M1) (Figure 1) and the corresponding area of hypoperfusion with preserved cerebral blood volume (Figure 2). The patient underwent intravenous thrombolysis with alteplase followed by mechanical thrombectomy with stent-retriever, achieving complete recanalization 3 hours after symptom onset. The follow-up CT scan performed 24 hours later

showed an established infarct affecting the left basal ganglia, with no haemorrhage. The patient´s clinical condition improved in the following days until complete recovery.

As part of the diagnostic protocol a transthoracic echocardiography was performed, showing left ventricular apical akinesis with an intraventricular 2.2 x 1.2 cm thrombus (Figure 3) in the apical region. Anticoagulation with low molecular weight heparin and low dose aspirin was initiated.

During a new cardiologic interview after recovering from the aphasia, the patient denied any heart-related symptom previous to stroke onset. Seven days after, a coronary artery angiography showed normal main left, circumflex and right coronary arteries. The large recurrent left anterior descending (LAD) artery, without evidence of atherosclerotic disease, had a non-occlusive dissection in its apical segment (Figure 4). Given that the dissection affected a distal segment and there was no evidence of flow-limiting obstruction, conservative medical therapy was adopted, without coronary intervention.

After 12 days of hospitalization, the patient was discharged on low-dose beta-blocker therapy, 100 mg aspirin and warfarin (INR 2-3). In this patient, spontaneous LAD dissection led to silent myocardial stunning and subsequent apical akinesis, with secondary thrombus formation and cerebrovascular embolization. To our knowledge, this is the first case described of spontaneous coronary artery dissection (SCAD) presenting with an acute ischemic stroke as the unique clinical manifestation of the disease.

Spontaneous coronary artery dissection is a rare, life-threating condition usually affecting young healthy women. (1) Its incidence is about 0.2% of patients with acute coronary syndrome (2) and most cases involve the left anterior descending artery.

The pathophysiologic underlying mechanism is a disruption of the intima-media layer, leading to an intimal flap and subsequent intramural hematoma formation. Depending on the degree of flow-limiting severity, the clinical condition may vary, and possible presentations include acute coronary syndromes, ventricular arrhythmias and sudden death.

Spontaneous coronary artery dissection is usually diagnosed by coronary angiography, the most common findings being a radiolucent intimal flap or a delayed clearance of contrast from the false lumen (as shown in our case). However, angiographic images can mimic atherosclerotic stenosis or coronary spasm, representing a diagnostic challenge. (3) In the last years,

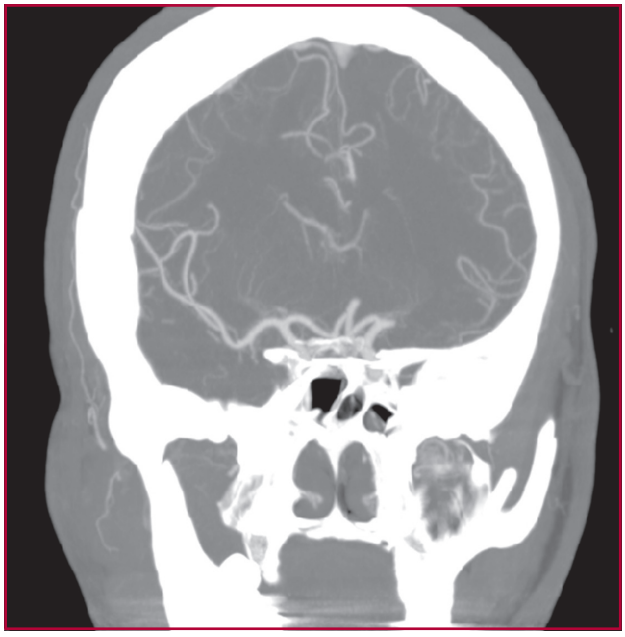

Fig. 1. Baseline CT-angiography (coronal view) showing complete occlusion of the M1 segment of the left medial cerebral artery.

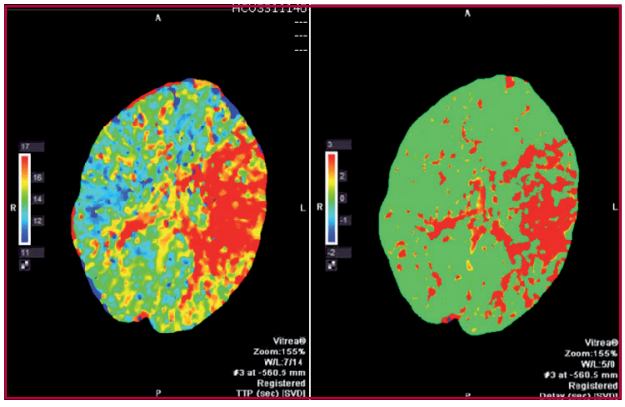

Fig. 2. CT-perfusion images showing a large ischemic area in the territory of the left medial cerebral artery occlusion with preserved cerebral blood volume indicating the persistence of recoverable tissue.

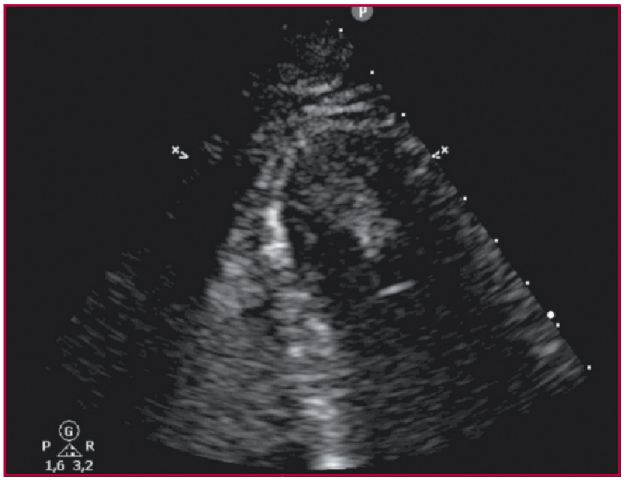

Fig. 3. Transthoracic echocardiography (zoom image from the apical 4-chamber view) showing left ventricular thrombus in the apical region

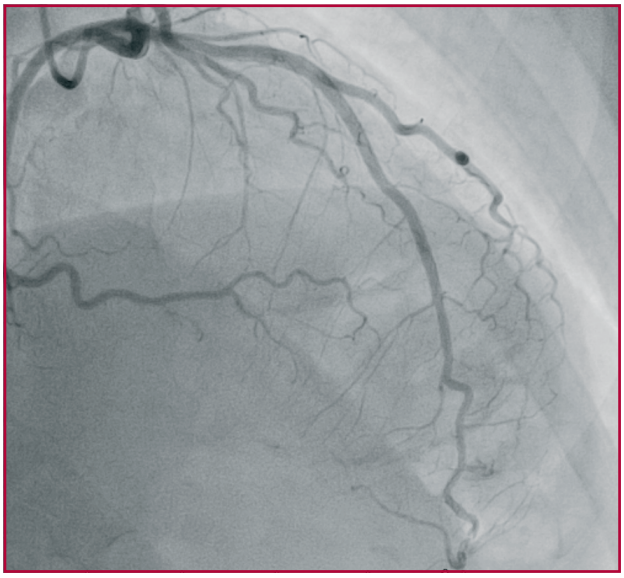

Fig. 4. Coronary angiography (right anterior oblique view) showing left anterior coronary artery apical segment dissection.

intravascular imaging techniques –intravascular ultrasound (4) and optical coherence tomography– have allowed for a better understanding and characterization of SCAD and a more accurate diagnosis.

The therapeutic management options depend on the degree of flow limitation and the localization and extent of the dissection (5). In proximal and flow-limiting cases, coronary intervention and stenting may represent the most appropriate treatment (6), while in distal, non-obstructive dissections medical treatment and close follow up are the most common strategies.

Although there is lack of evidence regarding the most appropriate medical treatment for SCAD, there is a tendency to use double antiplatelet and beta-blocking therapy.

The natural history of the disease, once the acute phase is resolved, is usually spontaneous healing and complete resolution. However, recurrent rates of up to 20% have been described in the first 10 years, emphasizing the importance of close follow up.

In our case, due to the features of the dissection, we chose a conservative management with long-term dual antithrombotic therapy with warfarin and ASA for secondary prevention of myocardial ischemia and cardioembolism.

This case represented a diagnostic and therapeutic challenge for both the neurology and cardiology teams and illustrates the importance of a team-based approach for the treatment of cerebrovascular disease.

Conflicts of interest

None declared.

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