## Late Giant Left Ventricular Outflow Tract Pseudoaneurysm After Aortic Valve Replacement

We describe the case of a 52-year-old female patient who underwent aortic valve replacement 9 years ago using a 19 mm mechanical Carbomedics prosthesis, due to infectious endocarditis of the aortic valve. The patient was referred for a coronary angiography after being diagnosed giant calcified left ventricular outflow tract pseudoaneurysm and aortic prosthetic valve dysfunction with an ambulatory echocardiography. Coronary arteries were free from angiographic disease. Her case was presented in a medical-surgical session, where it was decided to complement the study with cardiac magnetic resonance imaging, positron emission computed tomography and cardiac computed tomography (Figure 1 A, B and D) to assess the surgical approach, and positron emission tomography-cardiac computed tomography (Figure 1 C) to rule out any active infectious process. After these complementary studies, the patient underwent cardiac surgery with favorable clinical outcome.

Pseudoaneurysm is defined as a myocardial wall rupture that is contained by pericardial adhesions or wall. It has been demonstrated that the use of hypothermia in surgical interventions can increase the stiffness of the extracorporeal circulation tubing, and tubes in contact with the endocardium could damage the cardiac walls due to friction and small traumas, later causing a pseudoaneurysm in that site. (1)

The most common sites for pseudoaneurysms of the ascending aorta are the aortotomy site, the aortic cannulation site and the proximal anastomosis of venous transplants or grafts, and less common sites are found in aortic sutures after heart transplantation or in prosthetic valve infective endocarditis. The time elapsed between surgery and the onset of this condition varies from the first few days to 10-20 years after the procedure. A pseudoaneurysm may grow to become a huge pulsatile mass that compresses the trachea, bronchi, the superior vena cava, and the main pulmonary artery. (2)

Delayed pseudoaneurysms can also be the result of thoracic injuries. About 2% of patients with thoracic aortic trauma treated with a conservative approach or with undiagnosed aortic injuries will develop a delayed pseudoaneurysm, more than 90% of which are located in the aortic isthmus. (3)

In 1998, Katsumata et al. published a series of nine late pseudoaneurysms, two of which were treated conservatively with positive outcome. (4) Although there are small series of cases like the one described above, we believe this condition should be treated surgically as soon as possible, after being diagnosed with the cardiac imaging tools currently available.

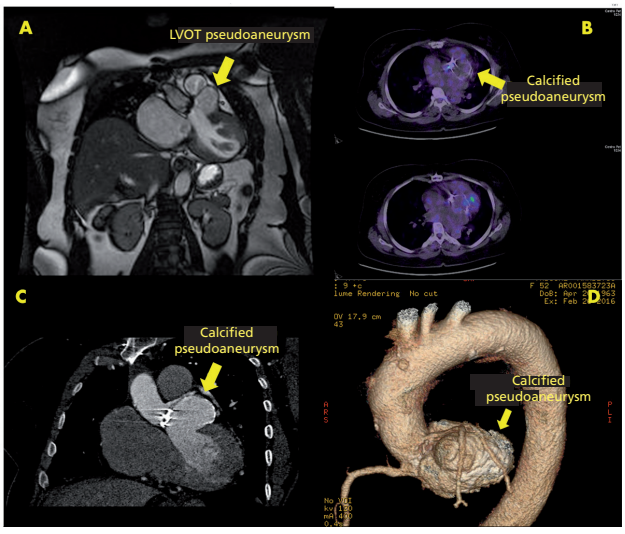


**Fig. 1.** **A.** Cardiac magnetic resonance image showing left ventricular outflow tract (LVOT) pseudoaneurysm. **B.** Positron emission computed tomography scan-cardiac computed tomography scan revealing calcified pseudoaneurysm with no evidence of macroscopic periprosthetic infection. **C.** Cardiac computed tomography scan indicating calcified pseudoaneurysm. **D.** Cardiac computed tomography scan showing volumetric reconstruction of calcified left ventricular outflow tract pseudoaneurysm.

### Conflicts of interest

None declared.

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

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## Transapical Sapien Valve on Previous Mitral Annulus

Mitral valve disease, mainly mitral regurgitation, is the second most common heart valve disease in our population. (1) Mitral valve repair, which includes in most cases annuloplasty, is the preferred treatment option whenever possible. (2) The stability of mitral valve repair results in excellent long-term outcomes. (3) When recurrence of mitral regurgitation occurs after valve repair, the standard procedure is reoperation; however, in some cases it may implicate unacceptable risk. Patients with severe pulmonary hypertension or right ventricular dysfunction are considered inoperable.

In such circumstances, the boom of transcatheter techniques offers an alternative for these patients. Valve-in-valve procedures in aortic position offer promising outcomes. (4)

Similarly, valve-in-valve procedures in mitral position have also been performed. (5)

With all these elements, series of transcatheter mitral valve-in-ring implantations are limited. (6)

From 2007 to April 2016, more than 90 transapical aortic valves were implanted in our center. Among them, eight cases were valve-in-valve procedures. We describe our initial experience with transapical mitral valve-in-ring implantation.

We present the case of an 84-year-old female patient, who underwent mitral valve repair in 2002 due to degenerative disease by resection of posterior leaflet prolapse and corrective annuloplasty. The patient made good progress for the first 10 years, until she progressively developed mitral regurgitation that worsened her functional class. When the intervention was proposed, mitral regurgitation was severe, with clear reversion of blood flow in the pulmonary veins. The patient had developed tricuspid regurgitation and ventricular dysfunction (LVEF 42%), and the pulmonary hypertension became severe affecting the right ventricle (TAPSE 13 mm). Due to right ventricular involvement, age, and a certain degree of renal failure, a transcatheter procedure was decided.

The procedure was carried out in the cardiac catheterization laboratory by a team of cardiac surgeons, an interventional cardiologist and an anesthesiologist. Following left anterior thoracotomy in the fifth intercostal space, the pericardium was dissected to access the cardiac apex. Implantation through a double purse-string suture with Teflon-felt pledgets was performed. Under fluoroscopic and echocardiographic guidance, a flexible guidewire was passed through the mitral valve, and subsequently, a right coronary artery catheter allowed for the exchange of the guidewire to obtain high support by means of a guidewire placed in the pulmonary vein (Figure 1). A semi-rigid Carpentier-Edwards Physio I annuloplasty ring size 32 was used in the hope of a circular implantation of the transcatheter prosthesis. The calculated size of the prosthesis was #29 Sapiens 3 (Edwards Lifesciences; Irvine, California, United States of America). The height of the implant is crucial in these cases, and after aligning the mitral ring ortogonally using fluoroscopy, the prosthesis was released leaving 50% on each side of the ring (See Figure 1). Nondisplaced prosthesis was verified by progressive inflation, and its normal functioning was confirmed by fluoroscopy and echocardiography. Postoperative course was uneventful, and the patient was discharged without mitral regurgitation (Figure 2).

Reintervention is the treatment of choice for patients with mitral regurgitation after annuloplasty. However, valve-in-ring implantation seems to be a suitable option for high-risk patients. Easy transapical access turns it into an attractive approach in these cases.

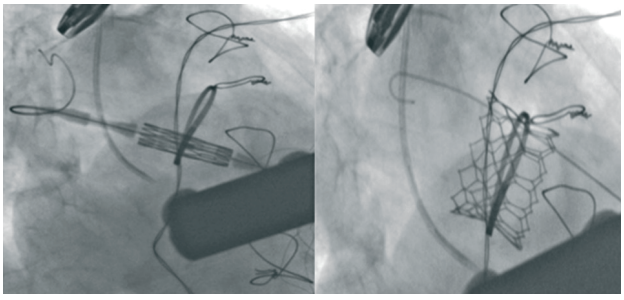

**Fig. 1.** Prosthesis alignment with guidewire placement in the pulmonary vein (*left*). Fluoroscopy following definitive implantation (*right*).

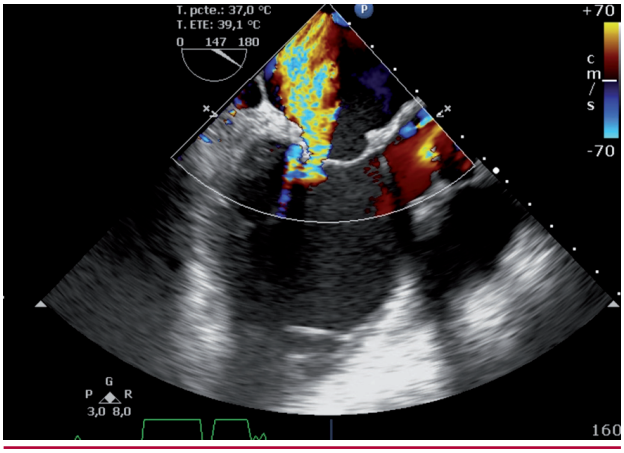


**Fig. 2.** Echocardiographic image following self-expandable valve implantation.

### Conflicts of interest

None declared.

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REV ARGENT CARDIOL 2017;85:48-49. http://dx.doi.org/10.7775/rac.v85.i1.9737

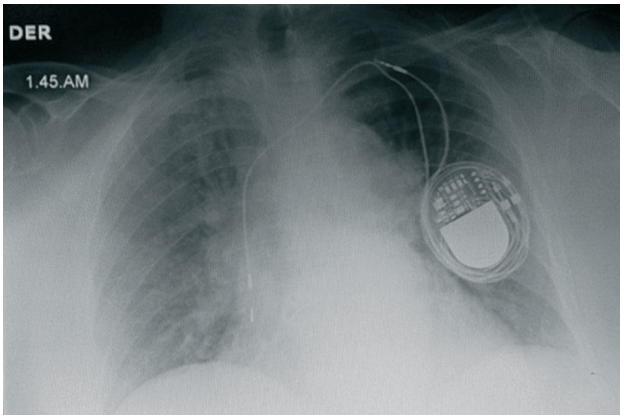


**Fig. 1.** Chest x-ray showing atrial lead displacement and ventricular lead traction. Rotation of the generator in its transverse axis and rolling of leads around it (Reel syndrome) can also be observed.

## Reel Syndrome: A Rare Complication in a Patient with Recent Endocavitary Pacemaker Implantation

We present the case of a 61-year-old male patient with a history of systemic hypertension under treatment with enalapril 10 mg every 12 hours, dyslipidemia, physical inactivity, overweight (BMI: 27), and acute myocardial infarction of the inferior wall in 2003, who was admitted in the emergency room of our hospital due to syncope. The electrocardiogram (ECG) showed third-degree AV block with idioventricular rhythm at 35 beats per minute. A Medtronic Adapta ADD-R01 permanent pacemaker was implanted, with its respective Medtronic PJN4103433 atrial electrode lead and Biotronik Selox ST 60 ventricular electrode lead, both through the left cephalic vein. Once the proximal ends of the electrodes were connected, the generator was placed in a preformed pocket in the left deltopectoral region. Intraoperative measurements revealed proper atrial and ventricular detection and stimulation thresholds. Within 24 hours after implantation, a chest x-ray showed adequate anatomic position of the electrode leads, and no detection or stimulation failures were identified in the ECG in both chambers. At the pacemaker follow-up 15 days later the patient referred minor dizziness. Telemetric and electrocardiographic evidence of atrial and ventricular stimulation and detection failures were targeted. The chest x-ray showed atrial electrode displacement to the subclavian vein, and traction on the ventricular lead to the RA (Figure 1). The ECG showed a third-degree atrioventricular block with a ventricular escape of 55 beats per minute, causing the patient's symptoms (dizziness). Surgical examination was indicated to reposition both leads. Since the patient's heart rate was dependent on ventricular stimulation, and was asymptomatic at times, a temporary pacemaker lead was placed via the right jugular vein as a bridge to repositioning the electrodes. Chest x-ray showed the rotation of the pacemaker generator on its own transverse axis with the atrial and ventricular electrodes rolling around it (Reel syndrome) (See Figure 1).

Surgical examination of the pocket was performed, both leads were repositioned, and the pacemaker generator was fixed to the fascia of the pectoralis muscle. The patient recovered without further complications after 6 months of follow-up.

We consider it important and useful to review the terminology order about macro-dislodgement of endocavitary pacemaker leads, which would be distributed as follows and is outlined in Figure 2:

Twiddler's Syndrome: Retraction and dislodgement of leads due to device generator rotation around the axis defined by the lead. Although patient's external manipulation can facilitate the process, it is not necessarily a condition. Due to the rotational movement of the generator, the lead rolls like a braid, showing a defined and characteristic aspect.

Reel's Syndrome: This is the one described in our case, and it is defined as lead retraction and dislodgement due to a rotating movement of the generator over its sagittal axis, causing the lead to roll as a reel or yo-yo over, below or around the generator. Due to the mechanism in both the Twiddler and the Reel syndrome, all the leads –in case there are several– would be affected to a greater or lesser extent.

Ratchet Syndrome: Lead retraction and dislodgement due to progressive lead displacement through its fixation parts or protector sleeves, facilitated by movements of the ipsilateral arm and due to incomplete lead fixation to the protector sleeve, but without generator rotation over any of its axes. In this case, the problem could involve all system leads in a patient or, more commonly, only one of the leads, with all the

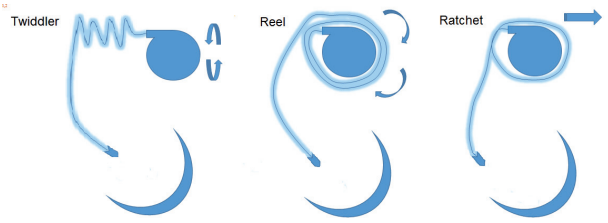


**Fig. 2.** Outline of each of the mechanisms of lead macro-dislodgement.

others in normal position; this is a key finding for identifying the Ratchet syndrome and distinguishing it from the other two lead macro-dislodgement syndromes. (1)

In our case and in the others described above, the final result is the displacement of electrode leads and the loss of atrial and/or ventricular stimulation. The symptoms include dizziness, syncope or presyncope, and may even be life threatening for patients whose heart rate absolutely depends on pacemaker stimulation.

Reel's syndrome is a rare entity. Like Twiddler's syndrome, it is due to similar, but not identical mechanisms, which easily leads to confusion. The difference lies in the rotation axis of the pacemaker: when rotation occurs around the longitudinal axis, it is defined as 'Twiddler syndrome', while in the 'Reel syndrome', it rotates around the transverse axis. The first mechanism causes electrode lead displacement and/or fracture; the second mechanism only causes the displacement of those elements. The Ratchet syndrome is slightly more common. Female gender, a large pacemaker 'pocket', obesity or excessive subcutaneous fat tissue, and voluntary or involuntary manipulation of the pacemaker 'pocket' are contributing factors. (2, 3) It is an entity easily diagnosed with chest x-ray, which shows the displaced leads rolling around the pacemaker generator. (4) In our case, the lack of fixation of the device to the surrounding tissues, which is not frequently performed in our service, together with the patient's voluntary manipulation of the device played a key role in causing this syndrome. It is possible to prevent this phenomenon by fixing the lead and generator to the muscular fascia with a stitch, and by making a small 'pocket'.

### Conflicts of interest

None declared.

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## Management of Coronary Perforation and its Complications during Angioplasty in a Patient with Systemic Lupus Erythematosus

Coronary artery perforation is one of the most severe complications during percutaneous transluminal coronary angioplasty (PTCA). Its incidence is low and varies according to lesion characteristics, type of procedure and clinical history of the patient. It may present with different degrees of severity, the most complicated being coronary perforation (CP) to the pericardium, as it produces fast development of hemopericardium resulting in cardiac tamponade (CT) needing, in some cases, emergency pericardiocentesis. Moreover, systemic lupus erythetamotosus (SLE) is a multisystemic inflammatory disease affecting the coronary arteries, increasing atherosclerotic disease and predisposing to arteritis and coronary thrombosis.

We present the case of a 45-year-old female patient with a history of SLE who, during an emergency angioplasty due to non-ST-segment elevation myocardial infarction (NSTEMI), suffered CP which progressed to CT requiring drainage.

The patient had a history of untreated hypertension (HTN), SLE with 9-year of treatment abandonment and one-month progressive chest pain. She presented at the emergency room for burning chest pain of 8/10 intensity, lasting 30 minutes, that started in functional class (FC) II and persisted in FC IV, 12 hours before presentation. On admission, she was hemodynamically stable, with high blood pressure (160/90), asymptomatic for angina and dyspnea. The electrocardiogram revealed sinus rhythm at 80 bpm and negative T in V1-V6. Troponin dosage showed a positive value of 0.11 ğg/ml. Full medical treatment was initiated. A coronary angiography of the left anterior descending coronary artery (LAD) performed due to recurrent angina symptoms revealed critical, segmental blockage, involving its proximal and medial third (Figure 1 A and B) and moderate blockage in the medial third of the circumflex

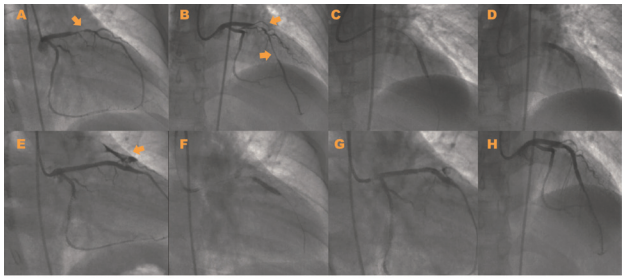


**Fig. 1. A.** Right oblique projection of left coronary artery angiography showing segment lesion involving the proximal segment of the anterior descending coronary artery (*arrow*). **B.** Anteroposterior cranial projection of the left coronary artery showing segment lesion involving the proximal and middle segment of the anterior descending coronary artery (arrows). **C.** Anteroposterior cranial projection showing implantation of the first 3.0 × 22 mm stent in the middle segment of the anterior descending coronary artery. **D.** Anteroposterior cranial projection showing implantation of the second 3.5 × 26 mm stent in superposition with the first one in the middle and proximal third of the anterior descending coronary artery. **E**. Right oblique projection during control angiography showing contrast leakage towards the pericardium. F. Right oblique projection showing balloon dilatation on the leakage site. **G.** Right oblique projection showing MGuard stent placement. **H.** Anteroposterior cranial projection of control angiography evidencing absence of contrast leakage after micromesh stent implantation.

coronary artery (CxA) and right coronary artery (RCA), without significant angiographic obstructions.

Due to these findings, an angioplasty was performed in the LAD with 3.0 × 22 mm and 3.5 × 26 mm drug-eluting stents implanted in superposition (Figure 1 C and D). Control angiography evidenced contrast dynamic leakage to the pericardium (Figure 1 E). As a first approach, it was decided to reverse heparin with protamine and simultaneously perform 90-second 3.5×26 mmm balloon dilatations every two minutes on the perforation site. Next, a 3.5 × 23 mm MGuard mesh-covered stent was implanted, stopping contrast leakage (Figure 1 F, G and H). At the end of the procedure, a transthoracic echocardiogram revealed mild pericardial effusion (PE), and the patient was transferred to the coronary care unit with progressive pain decrease.

Three hours later, the patient presented with hemodynamic instability and progressive inotropic requirement associated with clinical signs of CT. A new echocardiogram showed increased PE compared with the previous study. A new coronary angiography evidenced that both previously implanted stents were permeable with new image compatible with contrast leakage at the distal level of the stent located in the middle third (Figure 2 A). A new MGuard stent was implanted at 12 atm on the perforation site, achieving the interruption of contrast leakage (Figure 2 B and C). Finally, periocardiocentesis was performed draining 170 ml of hematic fluid with normalization of blood pressure until complete closure, administration of inotropic agents and favorable outcome.

Coronary perforation is a rare complication of percutaneous interventions, with a reported incidence ranging between 0.1% and 3%. (1)

The Ellis classification (2) is the one most commonly used. (2)

Type I: Extraluminal crater without extravasation.

Type II: Pericardial or myocardial blush without contrast jet extravasation.

Type Ill: Contrast extravasation through frank (>1 mm) perforation.

Although perforations can develop asymptomatically, they may sometimes progress to CT, myocardial infarction, malignant arrhythmias and occasionally death. Development of early PE is associated with worse prognosis, as a third of these patients require emergency surgery, either because pericardiocentesis is inefficient or because it is necessary to repair the arterial damage.

Coronary perforation predictors may be arbitrarily classified as angiographic or clinical. Angiographic predictors include lesions with abundant calcium content, tortuous arteries and chronic occlusions, whereas higher percentage of women, age, hypertension, diabetes and NSTEMI are among clinical predisposing factors. (3)

In SLE patients (multisystemic inflammatory disease characterized by antibody production with immune complex deposition and multiple laboratory abnormalities and clinical manifestations), cardiac injury is frequent causing important morbidity and mortality. The causes postulated for this association are the development of early atherosclerosis (probably accelerated by corticosteroid administration), coagulopathy, specially related with antiphospholipid antibodies, coronary aneurysms and vasculitis. Coronary arteritis, which causes cardiac ischemia, is very infrequently produced in SLE patients and is difficult to diagnose accurately, except with anatomopathological examination. The histological study of the coronary arteries in SLE patients reveals the presence of cellular proliferation in the intima and focal or diffuse fibrous blockage in the media. It is not clear whether these findings correspond to arteritis or thrombosis sequels, either primary (as a consequence of the presence of lupus anticoagulant or antiphospholipid antibodies), or secondary (due to platelet and neutrophil aggregation in the lumen of these vessels. (4, 5) An experimental study revealed that increased fibrous content in the atherosclerotic plaque subjected to balloon compression produces a higher amount of horizontal fissures in the plaque wall and greater susceptibility to vessel rupture. (6) In this sense, it could be assumed that an increase in the fibrous component of the atherosclerotic plaque in SLE patients is associated more directly with an increased rate of CP during PTCA.

The in-hospital outcome of the perforation is related with the degree of CP, the development of CT and the need to perform an emergency surgery. The type

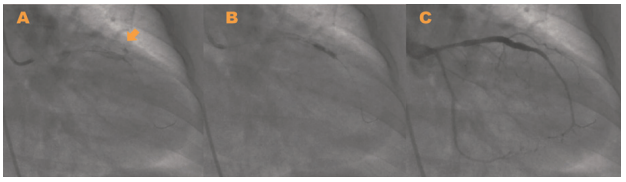


Fig. 2. **A.** Right oblique projection showing new contrast leakage. **B.** MGuard stent implantation. **C.** Right oblique projection of control angiography evidencing absence of contrast leakage

III perforation is associated with a greater incidence of complications and high mortality, ranging between 25% and 40% according to different series. (1)

The first treatment consists in sealing the perforation using an inflated balloon in the rupture site, previous reversion of anticoagulation with protamine. If the artery occlusion is well tolerated, more prolonged balloon dilatations may be done, which might control the perforation. However, it is usually necessary to continue with other measures, such as embolization using coils, uncovered or covered stents with an impermeable coat of polytetrafluoroethylene and use of covered stents coated with a porous polymeric mesh (MGuard), available in multiple diameters and lengths and which enable better navigation than stent grafts. However, it is important to mention that their use in perforations is associated to the development of intrastent restenosis at 9-12 months, so a strict follow-up should be established in this regard.

Concerning CT, both early diagnosis as CP resolution are the fundamental elements to reestablish hemodynamic stability.

In our patient, pericardiocentesis was used to solve CT without need of resorting to revascularization surgery after sealing the perforation.

Coronary perforation with blood flow towards the pericardium may lead to hemodynamic collapse due to CT, an infrequent but potentially lethal complication of PTCA with stent implantation. We assume that given the patient's history, the complication was influenced by the implantation of a stent on a lesion with high fibrous content. It was successfully resolved by implanting micro-mesh stents, sealing the perforation, and performing percutaneous pericardial drainage. We emphasize the importance of strict echocardiographic control after this complication.

### Conflicts of interest

None declared.

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## Incidental Finding of Thrombus-in-Transit in the Right Ventricle

The detection of free-floating thrombi in the right ventricle (RV) is a very rare event, especially if no thromboembolic events have been documented. This finding is a medical emergency requiring immediate treatment due to its high mortality.

We present the case of a free-floating thrombus detected in a 27-year-old female patient undergoing preoperative risk assessment for plastic surgery.

The patient referred no symptoms; however, in the directed interrogation, she mentioned fatigue and intolerance to moderate exertion for the past 3 months, after undergoing sclerotheraphy of lower extremity varices. The patient was a current smoker of 10 cigarettes per day, and her usual medication was oral contraceptives (ethinyl estradiol associated with drospirenone). At the physical examination her heart rate was 80 beats per minute, blood pressure was 120/80 mmHg and oxygen saturation 98%, with no relevant findings per system except for mild bilateral ankle edema. The electrocardiogram evidenced sinus rhythm with negative T waves from V3 to V6. She was referred to the Doppler echocardiography laboratory, where a transthoracic study (Figure 1) revealed a 8 × 1.5 cm, mobile, meandering, thrombus-like mass, anchored at the tricuspid subvalvular apparatus. The RV showed dilation and impaired systolic function, and the Doppler analysis revealed severe tricuspid regurgitation, with an estimated systolic pulmonary pressure of 34 mmHg. Venous Doppler echography of the inferior extremities showed signs of subacute venous thrombosis of the right popliteal and tibio-fibular trunk territories. A subsequent computed tomography of the thorax with intravenous contrast (Figure 2) demonstrated central emboli in both pulmonary arteries. Laboratory tests on

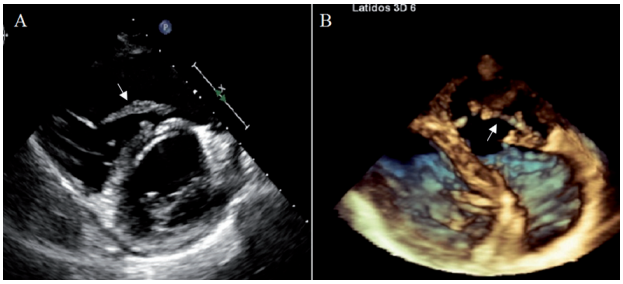


**Fig. 2. A.** Short-axis paresternal view of transthoracic echocardiogram, showing a floating thrombus in a dilated right ventricle. **B.** In vivo 3D image of the right ventricular outflow tract, showing the thrombus attached to the tricuspid subvalvular apparatus. (*arrow*).

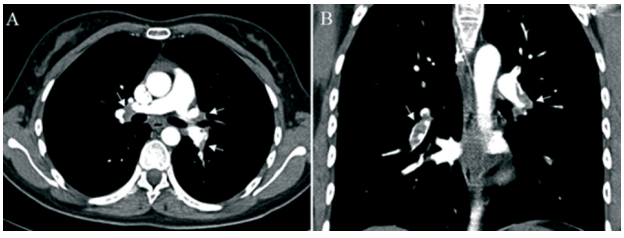


**Fig. 2.** Axial (**A**) and coronal (**B**) sections showing thrombi in both pulmonary arteries (*arrows*).

admission revealed normal ultrasensitive troponin T and elevated N-terminal brain natriuretic propeptide (NT-proBNP: 793 pg/mL). A hemophilia study was negative in all the tests performed. The patient was hospitalized, initially treated with intravenous unfractionated heparin at a dose of 18 IU/kg and then with oral acenocoumarol to achieve an international normalized ratio between 2 and 3. Seven days after, she was discharged with good clinical outcome. Anticoagulation was maintained for 6 months and interrupted since, with no new episodes up to the present

The incidence of free-floating thrombi in the right cavities reported in the literatures varies from 4% to 18%. (1) They are often associated with pulmonary thromboembolsm (PTE) and are mainly located in the right atrium (80%). In addition, in the absence of catheters, atrial fibrillation or structural heart disease, they invariably represent clots that travel from the lower extremities, with an elevated risk of death due to their possible fragmentation and massive pulmonary embolism. (2)

Thrombus morphology described in this case corresponds to thrombi-in-transit from the lower extremities, since rounded or laminar ones, attached to the heart wall with a broad implantation basis and stationary, are usually due to dilatation of the cavities, blood stasis or endothelial damage, and have the best prognosis with anticoagulation therapy. (3)

Treatment recommendations include anticoagulation, administration of thrombolytic agents or thrombus mechanical removal by percutaneous or surgical embolectomy. (4) However, despite the presence of thrombus in the right cavities with hemodynamic stability is considered a medical emergency due to the risk of shock, there is no consensus on the adequate treatment of these patients.

In a 2002 analysis, Rose et al. found that in patients with thromboembolism of the right cavities who received no treatment mortality rate was 100%, in those subjected to anticoagulation with heparin it was 28.6%, in those treated surgically 23.8% and in the ones receiving thrombolytic therapy 11.3%, a significant difference compared with the other conducts. (5) Similarly, Athappan et al. reported that in 328 patients with thrombi in the right cavities and PTE, mortality rate was 23.2%. Nevertheless, patients receiving no treatment had a mortality rate of 90.9%, while in those receiving anticoagulation, surgical embolectomy or thrombolytics these rates were 37.1%, 18.3% and 13.9%, respectively. (6)

Our case involves a young woman, in whom the presence of a floating thrombus in the right cavities associated with PTE was an incidental finding, with no knowledge of thrombus evolution time and degree of organization. In addition, the patient was hemodynamically stable, without hypercoagulability or underlying comorbidities. It was therefore decided to administer anticoagulation therapy with unfractionated heparin and intensive monitoring, achieving, as described, a satisfactory outcome.

## Conflicts of interest

None declared.

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

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