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.

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

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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 g/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

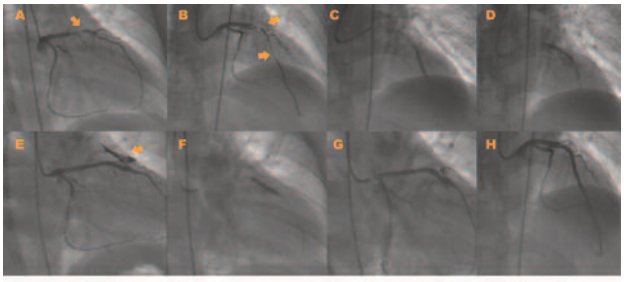

Fig. 1. A. Right oblique projection of left coronary artery angiography showing segment lesion involving the proximal segment of the anterior descending coronary artery (*arrow*). **B.** Anteroposterior cranial projection of the left coronary artery showing segment lesion involving the proximal and middle 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

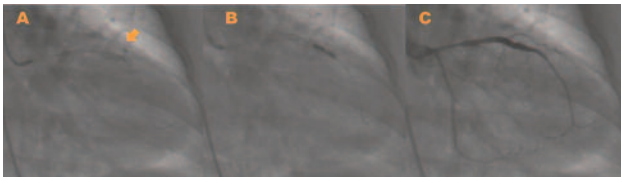

Fig. 2. **A.** Right oblique projection showing new contrast leakage. **B.** MGuard stent implantation. **C.** Right oblique projection of control angiography evidencing absence of contrast leakage

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