

Haemopericardium in a Fibrinolysis in Acute Myocardial Infarction Secondary to a Spontaneous Coronary Artery Dissection

Hemopericardio en infarto agudo de miocardio fibrinolizado secundario a disección coronaria espontánea

To the Editor,

Spontaneous coronary artery dissection is an uncommon cause of myocardial ischaemia. It has conventionally been described in young women without cardiovascular risk factors (CVRF).¹ The clinical manifestation of this condition varies between sudden death and acute coronary syndrome (ACS). The appearance of a haemopericardium is rare.²⁻⁴

The patient is a 66-year-old female with hypertension that sought treatment for typical tightness in the chest at rest and vegetative reactions over a period of 2 h. Upon arrival, arterial blood pressure was 165/88 mmHg; examination revealed truncal obesity and grade II/VI systolic murmur in basal foci. An ECG

showed elevations in the V2-6 and the ST segment of the inferior face. We performed fibrinolysis using 9000 U of tenecteplase, since the door-to-balloon time was estimated >90 min and was found within the first 2 h of evolution. Enoxaparin, acetylsalicylic acid, nitroglycerine, and morphine were administered simultaneously. An emergency echocardiogram demonstrated akinesis of the apex and anterior face, and the ejection fraction was estimated at 40%. We also observed a small posterior pericardial effusion. The persistence of the symptoms and consistently abnormal ST-segment led to salvage angioplasty. Before arriving at the operating room, the patient went into pulseless electrical activity, and died when reanimation failed.

A clinical biopsy revealed haemopericardium with a pressure of 650 ml. We observed a partially organised haematoma 1.5 cm from the apex (Fig. 1A) in relation to the anterior descending artery (Fig. 1B). The ventricular wall and intraventricular septum were intact. The middle portion of the anterior face and the apex of the left ventricle had the appearance of an acute infarction. Serial histological sections demonstrated extensive dissection of the anterior descending artery (Figs. 2A and B), which extended outside of the external elastic lamina (Figs. 2C and D) and broke

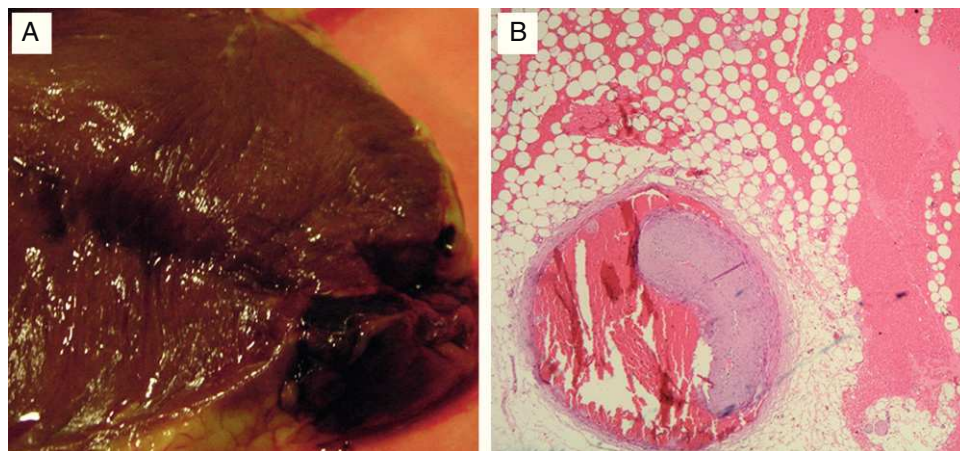


Figure 1. A: Macroscopic view of the apical subepicardial haematoma. B: Dissection of the anterior descending artery, with an intramural haematoma which compresses the lumen (H-E, $\times 10$).

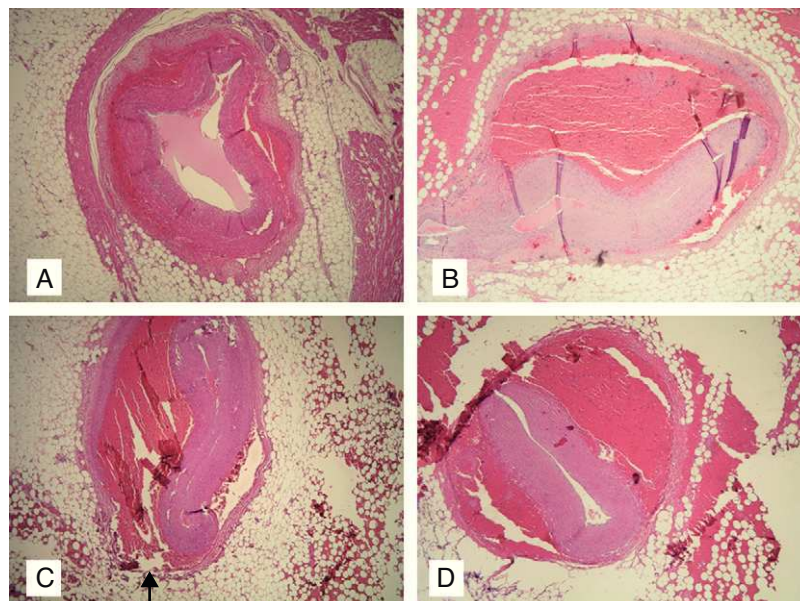


Figure 2. A-D: Dissection of the anterior descending artery outside of the external elastic lamina. Arrow: ruptured adventitia (H-E, $\times 10$).

the vessel wall (Fig. 2C, arrow). Atheromatous plaques were not observed.

Optimal treatment for spontaneous coronary artery dissection is not well defined. Conservative medical treatment has been efficient in asymptomatic cases with haemodynamic stability and in the absence of residual ischaemia. Heart procedures with stents have been used in localised dissections,² whereas surgery has been considered for cases involving the coronary artery or multiple vessels.^{1,3} The role of fibrinolysis in this condition is controversial: a favourable effect could be expected by dissolving the intramural haematoma.⁵ However, the same treatment could extend the dissection,⁶ with the consequent increase in the risk of coronary rupture into the pericardium. Early reperfusion is the treatment of choice for ACS with ST-segment elevation (ACS-STE). The fibrinolysis value is supported by clinical practice guidelines, above all when performed early or when a primary angioplasty is delayed, and is still the most commonly used type of reperfusion. In any case, an ACS-STE in a young woman without CVRF should make us consider performing a coronarography on an individual basis, even when this implies slightly delaying the time to reperfusion, in order to avoid complications in the fibrinolytic treatment.

We have presented this case primarily because of its rare occurrence, both due to it having appeared in a middle-aged woman with CVRF (arterial hypertension and obesity) as well the appearance of a haemopericardium secondary to a ruptured coronary artery that constitutes the fourth case of its kind,^{3,4} since the first case was described by Pretty in 1931.² We believe that the fibrinolytic treatment was key in the appearance of the haemopericardium. We also consider that necropsies, greatly underused in our field, should be performed more frequently in order to

complement the physician's efforts at confirming the aetiology, as in our case.

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REFERENCES

- Berenguer A, Mainar V, Bordes P, Valencia J, Arrarte V. Disección espontánea de arterias coronarias como causa infrecuente de síndromes coronarios agudos. *Rev Esp Cardiol.* 2003;56:1017-21.
- Pretty HC. Dissection aneurysm of a coronary artery in a woman aged 42: Rupture. *Br Med J.* 1931;1:667.
- Badmanaban B, McCarty D, Mole DJ, McKeown P, Sarsam MAI. Spontaneous coronary artery dissection presenting as cardiac tamponade. *Ann Thorac Surg.* 2002;73:1324-6.
- Hayes CR, Lewis D. Spontaneous coronary artery dissection of the left circumflex artery causing cardiac tamponade and presenting with atrial fibrillation: A case report and Review of the literature. *Angiology.* 2007;58:630-5.
- McCann AB, Whitbourn RJ. Spontaneous coronary artery dissection: a review of the etiology and available treatment options. *Heart Vessels.* 2009;24:463-5.
- Buys EM, Suttorp MJ, Morshuis WJ, Plokker HW. Extension of spontaneous coronary dissection due to thrombolytic therapy. *Cathet Cardiovasc Diagn.* 1994;33:157-60.

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Device Thrombosis During the Percutaneous Closure of a Patent Foramen Ovale

Trombosis de dispositivo durante el cierre percutáneo de foramen oval permeable

To the Editor,

A 58-year-old woman with history of cerebral aneurysm that was embolized 1 year earlier, with no cardiovascular risk factors, was admitted to the hospital with aphasia and hemiparesis due to a cerebral infarction in the left middle cerebral artery. In a transthoracic echocardiography with saline, a moderate tendency of microcavitations in the left atrial was observed, with interatrial septum intact and no structural anomalies. A transesophageal echocardiogram (TEE) demonstrated a hypermobile, >11mm-bulging membrane at the oval fossa that was becoming detached in the anterior segments, and a prominent Eustachian valve directing flow towards it. The patient was referred for percutaneous closure of the patent foramen ovale (PFO) and was treated with 300 mg acetylsalicylic acid. Non-fractionated heparin was administered preoperatively at 6000 UI. A 35mm-Amplatzer device was implanted under general anaesthesia using a TEE-guided right femoral approach (before and during the apposition and release of the occluder). No initial complications were produced during this brief procedure. After releasing the device, we observed a hypermobile filiform image in the left atrial face (Fig. 1) that would correspond to a thrombus, but which did not cause incorrect disc apposition. We administered an additional 7500 UI non-

fractionated heparin and treatment was started with 100 mg acetylsalicylic acid, 75 mg clopidogrel, and therapeutic doses of enoxaparin. The patient stayed in the hospital for 1 week, with no new neurological symptoms or hemorrhagic events associated with the treatment; TEE imaging showed no thrombi and that the occluder was functioning correctly (Fig. 2). Given the patient's history of cerebral aneurysm and the close association between thrombi and this procedure, the patient was discharged with only



Figure 1. Filiform image in the atrial face of the left disc that corresponds to a thrombus.