

Figure 2. A: right coronary tree before surgery; non-dominant vessel with no sign of obstruction. B: complete occlusion of the conus branch of the right coronary artery. C: recovery of blood flow in the conus branch following balloon angioplasty.

action potentials in the epicardial cells. This induces a greater dispersal of the repolarization wave, which precipitates ventricular arrhythmias through re-entry mechanisms during phase 2 of the action potential.³ This mechanism is similar to that seen in patients with Brugada syndrome, and cases have been reported in which ischemia in this territory, generally due to vasospasm, has caused ventricular arrhythmias.^{3,4}

In conclusion, the occlusion of the conus branch of the RCA by vasospasm or other mechanisms can cause myocardial ischemia in the region of the pulmonary infundibulum and the lateral wall of the RV, and ST segment elevation in the right precordial leads. The risk of arrhythmia generated by a mechanism similar to that seen in Brugada syndrome is high. It is essential that the surgeon know the origin and trajectory of the conus branch of the RCA before operating.

Felipe Hernández Hernández,^a Elvira Barrios Garrido-Lestache,^{a,*} Primitivo Arribas,^b and Julian Gutierrez^b

^aServicio de Cardiología, Hospital 12 de Octubre, Madrid, Spain

^bUnidad de Cuidados Postoperatorios, Hospital 12 de Octubre, Madrid, Spain

*Corresponding author:

E-mail address: Elvira_barrios@hotmail.com

(E. Barrios Garrido-Lestache).

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Aortic Intramural Hematoma and Pulmonary Embolism. Diagnostic Challenge and Therapeutic Dilemma

Hematoma intramural aórtico y embolia pulmonar. Un reto diagnóstico y un dilema terapéutico

To the Editor,

We present the case of a 77-year-old woman with morbid obesity and hypertension. She was admitted to the cardiology service after being diagnosed as having a type B aortic intramural hematoma (AIH). Three days later, she had a new episode of chest pain. The electrocardiogram revealed the presence of sinus tachycardia and the most important finding upon laboratory

analysis was a new increase in D-dimer levels (which had been decreasing). Computed tomography was repeated and showed that the hematoma had not changed. However, arterial filling defects compatible with bilateral pulmonary embolism were observed (Fig. 1A).

The decision was made to begin anticoagulation with sodium heparin. Eighteen days later, the patient had another episode of chest pain, this time with a decreasing D-dimer levels. Computed tomography demonstrated an increase in thickness of the AIH and dissection (double barrel) of the wall of descending aorta from its proximal portion to the diaphragmatic hiatus (Fig. 1B). The decision was made to continue treatment with heparin, under strict clinical control.

After two weeks with no complications, the patient was discharged with oral anticoagulation therapy. Three months after

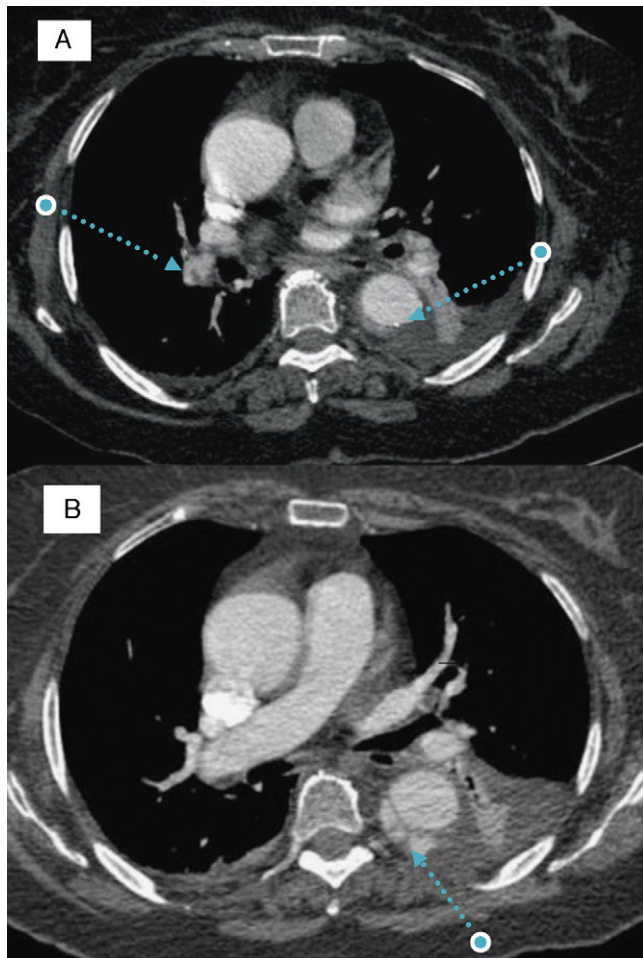


Figure 1. A: filling defects in right pulmonary arterial tree and type B aortic intramural hematoma. B: progression of the aortic intramural hematoma toward double barrel aortic dissection. Note the *cobweb sign* in the false lumen.

the initial event, the dissection had progressed, with increases in the aortic diameter (from 36 mm to 40 mm) and in the false lumen (from 20 mm to 24 mm). Anticoagulation therapy was definitively terminated.

The originality of this case lies in the concomitance of an acute aortic syndrome and pulmonary embolism, since we have found

no similar reports in the literature. These two conditions share similarities in their clinical signs and analytical results (D-dimer) at presentation and, thus, it is extremely important to make a correct differential diagnosis at the time of the acute phase. This difficulty becomes a real diagnostic challenge when the two conditions occur concomitantly. Although the role of the D-dimer levels in AIH has not been clearly established, as it has been in classic aortic dissection, there are series that confirm an increase in the incidence of AIH, although it is lower than that of dissection.

D-dimer titration as a predictor of the course of acute aortic syndrome has yet to be defined. In our patient, there was an increase in the concentration secondary to the embolism and, yet, it remained stable despite the progression of the AIH toward dissection (Fig. 2).

The dilemma arose at the time of therapeutic decision-making. Anticoagulation therapy has traditionally been avoided in these patients, although the subject is controversial and the documentation is limited. Ruggiero et al.¹ reported the case of a man with chronic atrial fibrillation and type B AIH in whom an increase in the thickness of the hematoma was detected following reinstitution of anticoagulation therapy, and they suspended the treatment, to which they attributed a role in promoting rebleeding. On the other hand, Cañadas et al.² described the cases of three patients with AIH and atrial fibrillation who received anticoagulation therapy during the acute phase; the clinical and morphological outcomes were satisfactory in all three.

In our case, given the limited knowledge concerning the role of anticoagulation therapy in the course of AIH, we decided to initiate treatment with heparin once pulmonary embolism had been documented, afterwards, the unfavourable course was confirmed. We do not know what role anticoagulation therapy plays in this complication; it may facilitate its development, although we also know that up to 36% of the cases of AIH evolve spontaneously to dissection.³

Once the presence of dissection had been established, another dilemma arose. Although the literature indicates that anticoagulation therapy could be associated with an increase in morbidity and mortality in these patients, there are no clear guidelines. On this occasion, treatment was maintained for 3 months after the embolism.

This case demonstrates the complexity involved in decision-making in patients with AIH who require anticoagulation, which will depend on a thorough evaluation of the risk-benefit balance. Even so, a comprehensive knowledge of the prognostic implications of anticoagulation therapy in acute aortic disease would be of great aid in decision-making. Studies involving larger samples of

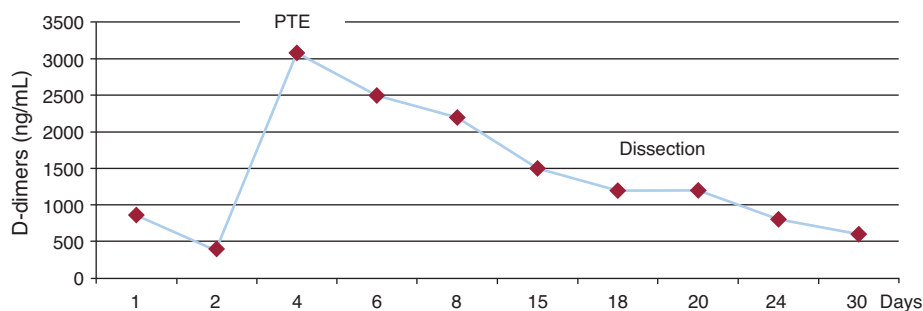


Figure 2. Changes in D-dimer concentration over the course of the study of this case. PTE, pulmonary thromboembolism.

patients and long-term clinical and radiological follow-up would be necessary.

Aida Suárez-Barrientos* and Isidre Vilacosta

Servicio de Cardiología, Hospital Clínico San Carlos, Madrid, Spain

* Corresponding author:

E-mail address: aidasuarez@gmail.com (A. Suárez-Barrientos).

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Use of an Explanted Pacemaker Connected to a Regular Screw-in Lead for Temporary Pacing

Utilización de un marcapasos explantado conectado a un electrodo clásico de fijación activa para estimulación ventricular temporal

To the Editor,

In transcatheter aortic valve implantations (TAVI), temporary transvenous pacing is essential for rapid pacing as well as for backup pacing. The use of a temporary ventricular pacing wire may be associated with lead displacement and right ventricular perforation, and limits patient mobilization in case of femoral access. We hereby report our experience using a temporary pacing strategy in TAVI that allows immediate patient mobilization and potentially safer prolonged temporary pacing.

A 58-cm bipolar screw-in lead (Tendril 1888 TC, St Jude Medical) is inserted under aseptic conditions via the internal jugular vein through a peel-away introducer (FlowGuard 7F-sheath, Enpath Medical Inc) and subsequently advanced to the right ventricle under fluoroscopic guidance using a preshaped stylet. The helical screw is extended using the fixation tool provided with the lead, with confirmation of helix extension by fluoroscopy. Satisfactory electrical parameters are capture threshold $<1.5\text{ V}/0.5\text{ ms}$ and R-wave sensing amplitude $>4\text{ mV}$. The lead is then fixated to the skin using the suture sleeve, and sterile dressing is placed over the insertion site. High-rate pacing is achieved using crocodile clips connected between the lead and a pacemaker analyzer. For prolonged backup pacing, the pacemaker lead is connected (Fig. 1) to an explanted conventional permanent pacemaker (<3 years old), retrieved from deceased patients or from those requiring device upgrade. Prior to use, explanted pacemakers are thoroughly cleaned but not sterilized, as they remain outside the sterile field. The pacemaker is programmed for backup single chamber ventricular pacing

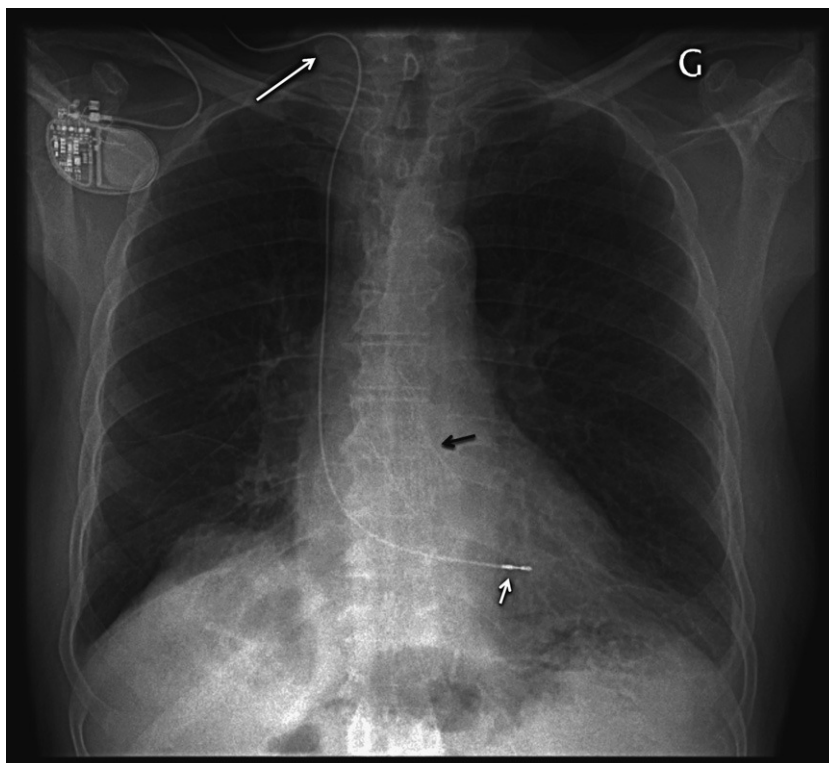


Figure 1. Chest X-ray after CoreValve implantation (black arrow). A screw-in lead was inserted via the right internal jugular vein (long white arrow), fixated to the right ventricle apex (short white arrow), and connected to an explanted permanent pacemaker.