

REFERENCES

1. Basson CT, Cowley GA, Traill TA, Soloman S, Seidman JG, Sedman CE. The clinical and genetic spectrum of the Holt-Oram syndrome (heart-hand syndrome). *N Engl J Med.* 1994;330:885-91.
2. Brassington AM, Sung S, Toydemir R, Le T, Roeder A, Rutherford A. Expressivity of Holt-Oram syndrome is not predicted by *TBX5* genotype. *Am J Hum Genet.* 2003;73:74-85.
3. Borozdin W, Bravo Ferrer Acosta AM, Bamshad MJ, Botzenhart EM, Froster UG, Lemke J, et al. Expanding the spectrum of *TBX5* mutations in Holt-Oram Syndrome: detection of two intragenic deletions by quantitative real time PCR, and report of eight novel point mutations. *Hum Mutat.* 2006;27:975-6.
4. Kohlhasse J, Chitayat D, Kotzot D, Ceylaner S, Froster UG, Fuchs S. *SALL4* mutations in Okhiro syndrome (Duane-radial ray syndrome), acro-renal-ocular syndrome, and related disorders. *Hum Mutat.* 2005;26:176-83.

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Recurrent Ventricular Fibrillation and ST Segment Elevation in the Right Precordial Leads Due to Acute Occlusion of the Conus Branch

Fibrilación ventricular recurrente y elevación del segmento ST en precordiales derechas por oclusión aguda de la arteria conal

To the Editor,

We present the case of a 79 year-old man diagnosed with high blood pressure and chronic liver disease due to hepatitis C virus. The patient was referred for surgery for severe mitral valve regurgitation associated with the prolapse of P2 and P3 through the spontaneous breakage of the chordae tendineae. The left ventricle (LV) and coronary arteries were normal.

Mitral valve reconstruction was undertaken using a Carpentier-Edwards Physion no. 28 annuloplasty ring and the implantation of two GoreTex cords attached to P2 and P3. No intraoperative complications were encountered. On arrival at the postoperative care unit the patient suffered several episodes of ventricular fibrillation. Advanced cardiopulmonary resuscitation was begun with multiple electrical defibrillations; the situation stabilized following the administration of amiodarone. Emergency bloodwork revealed no ion abnormalities, although an electrocardiogram showed a previously undescribed convex elevation of the ST segment in V1-V3 (Fig. 1). Emergency coronary angiography was performed, which revealed the complete occlusion of the conus branch of the right coronary artery (RCA); no such findings were

seen in pre-surgery coronary angiography (Figs. 2A and B). The left coronary artery tree was normal. After discussing the images with the surgical team it was concluded that the cause of this acute occlusion was a suture used to fix the cable of the epicardial pacemaker in place. Balloon angioplasty was performed and blood flow restored. Good coronary angiographic results were obtained (Fig. 2C).

The first artery arising from the RCA is the conus branch, although in 30%-50% of cases it may originate from the aorta.¹ Its function is to irrigate the pulmonary trunk (the outlet tract of the right ventricle [RV]). When very developed it can reach as far as the lateral wall of the RV. Its acute occlusion, either because of vasospasm (the most common cause), atherosclerotic disease or accidental injury during surgery in the region of the pulmonary infundibulum, etc., can give rise to myocardial ischemia in the latter area and in the RV lateral wall. This is manifested by two major events. The first is ST segment elevation in the right precordial leads, which has also been described in isolated ischemia of the RV. Geft et al.² concluded that, after inducing ischemia in experimental animals, the isolated involvement of the RV (i.e., with no LV involvement) gives rise to the displacement of the electrical forces towards the right, and thus ST segment elevation for these leads. However, when the LV is involved (generally the inferoposterior segments), left forces predominate and ST segment elevation is seen in the leg leads accompanied by ST segment depression in the right precordial leads. The second is a greater predisposition towards suffering polymorphic ventricular tachycardia and/or ventricular fibrillation. In animal models it has been shown that ischemia of the RV outlet tract leads to abnormal

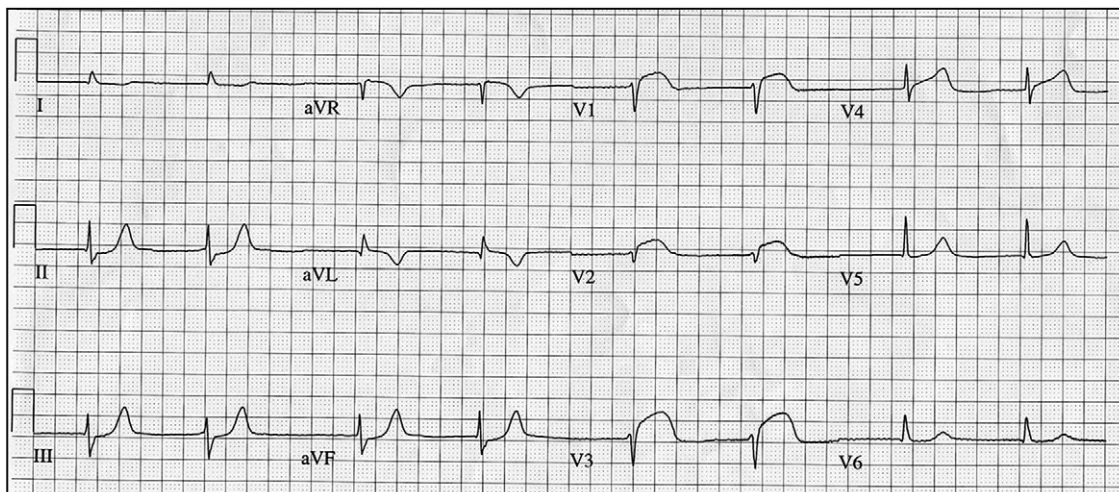


Figure 1. Convex elevation of the ST segment in the right precordial leads.

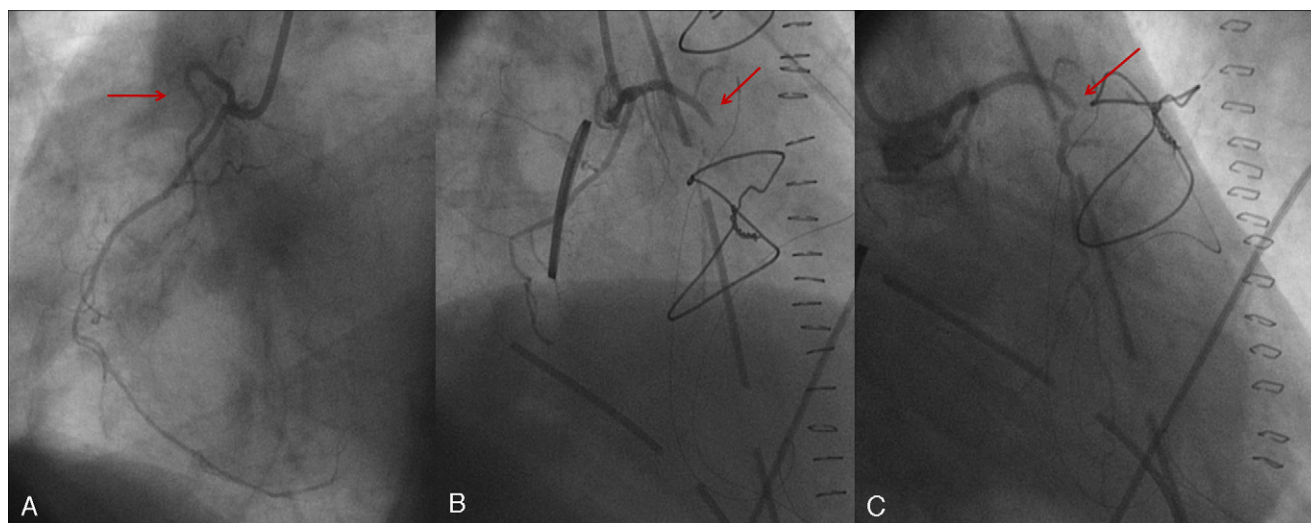


Figure 2. A: right coronary tree before surgery; non-dominant vessel with no sign of obstruction. B: complete occlusion of the conus branch of the right coronary artery. C: recovery of blood flow in the conus branch following balloon angioplasty.

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.

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REFERENCES

- Levin DC, Beckmann CF, Garnic JD, Carey P, Bettmann MA. Frequency and clinical significance of failure to visualize the conus artery during coronary arteriography. *Circulation*. 1981;63:833-7.
- Celik T, Yuksel UC, Kursaklioglu H, Iyisoy A, Kose S, Isik E. Precordial ST segment elevation in acute occlusion of the proximal right coronary artery. *J Electrocardiol*. 2006;39:301-4.
- Yamaki M, Nobuyuki S, Myojo T, Nishiura T, Nishimura M, Nakamura H, et al. Possible contribution of ischemia of the conus branch to induction or augmentation of Brugada type electrocardiographic changes in patients with coronary artery disease. *Int Heart J*. 2010;51:68-71.
- Ogano M, Iwasaki Y, Morita N, Tanabe J, Shiba K, Miyauchi Y, et al. Proarrhythmic ECG deterioration caused by myocardial ischemia of the conus branch artery in patients with a Brugada ECG pattern. *Pacing Clin Electrophysiol*. 2011;34:e26-9.

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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