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Acute thrombosis of the superior vena cava associated with atrial tachycardia catheter ablation

A 44-year-old menopausal woman with cardiovascular risk factors treated with hormone replacement therapy was being evaluated due to nodular goiter with normal thyroid function.

She had a 3-year history of atrial tachycardia with palpitations and episodes of collapse associated with hypotension. During follow-up she was treated with beta blockers, group 1C antiarrhythmic drugs and sotalol, but was refractory to treatment. The patient continued presenting episodes of paroxysmal tachycardia requiring frequent hospitalizations. An echocardiogram showed normal left ventricular function, normal left ventricular dimensions, normal right chamber dimensions and function, and normal left atrial dimension. The heart valves had no abnormalities. The lab tests were normal. Functional tests ruled out myocardial ischemia as the cause of the arrhythmia. Radiofrequency catheter ablation of atrial tachycardia with three-dimensional navigation system was indicated due to the lack of response to medical treatment. An EnSite NavX system version 8.0 (St. Jude Medical, SJM) was used.

A duo-decapolar Livewire catheter (SJM) was inserted in the right atrium for mapping the crista terminalis. A quadripolar catheter was positioned in the His bundle and a Blazer 8-mm tip quadripolar ablation catheter with thermistor (Boston Scientific) was inserted for mapping and ablation. The anatomical reconstruction of the right atrium and of both venae cavae was performed. A programmed atrial stimulation protocol was conducted; intranodal reentry circuit and accessory pathway were ruled out, and atrial tachycardia was induced. During mapping of the automatic atrial tachycardia, the maximal precocity was identified in relation with the P wave at the level of the superior vena cava. Upon performing local activation time (LAT) mapping with the navigation system, the origin of the tachycardia was identified in the same place. The tachycardia was reproduced with stimulation protocols and had the same characteristics as the tachycardia presented by the patient. Nine radiofrequency energy applications were delivered to the area described and tachycardia was eliminated and could not be induced after rapid pacing and isoproterenol infusion. The patient was admitted to the coronary care unit for 24 hours without complications and was discharged.

Twenty-four hours later, she was admitted to the emergency department due to headache and chest pain propagating to the anterior cervical region, with vomiting and cyanosis of upper extremities and head. A contrast-enhanced computed tomography scan of the chest was performed to confirm the diagnosis of acute obstruction of superior vena cava. The scan showed a reduction of the superior vena cava lumen at the level of its connection with the right atrium with a transverse diameter of 7 mm and endoluminal filling defect, and reflux of contrast into the azygos system. There was also a filling defect in the posterobasal and anterobasal arterial segmental branches of the right lower lobe artery and in the anterobasal arterial segmental branch of the left lower lobe artery, as well as mild pleural effusion and mild pericardial effusion (Figure 1).

The patient evolved with cardiogenic shock requiring orotracheal intubation, mechanical ventilation and vasoactive agents. The patient persisted with hypotension and poor distal perfusion. An urgent cavography was performed.

A complete obstruction of the proximal superior vena cava involving 100% of the lumen was observed. A pig tail catheter was introduced in the lumen and balloon angioplasty was performed. The success of the procedure was confirmed by intravascular ultrasound. The vessel was patent, without residual endoluminal filling defect. Hyper reflectivity was observed between hour 9 and 12 in the upper third of the structure visualized by endovascular ultrasound. Anticoagulation with heparin was started.

During hospitalization the hemodynamic parameters improved and the inotropic agents were stopped. The patient was extubated and did not present signs of brain injury. Two-dimensional Doppler echocardiography showed normal left and right ventricular function, normal wall motion, ejection fraction of 67%, mild tricuspid regurgitation and absence of pericardial effusion. A stenosis was observed in the superior vena cava with flow velocity of 1.5m/s (normal value < 0.8m/s). The patient started oral anticoagulation with K vitamin antagonists.

The neurological examination by imaging tests

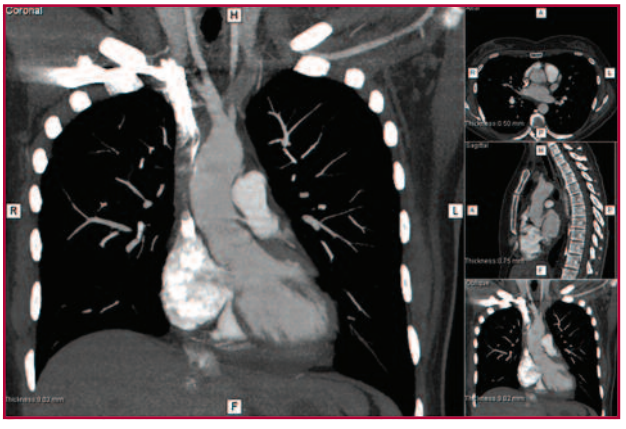

Fig. 1. Contrast-enhanced multi-detector row chest computed tomography. A complete obstruction of the proximal superior vena cava is observed, with a filling defect suggestive of luminal thrombus.

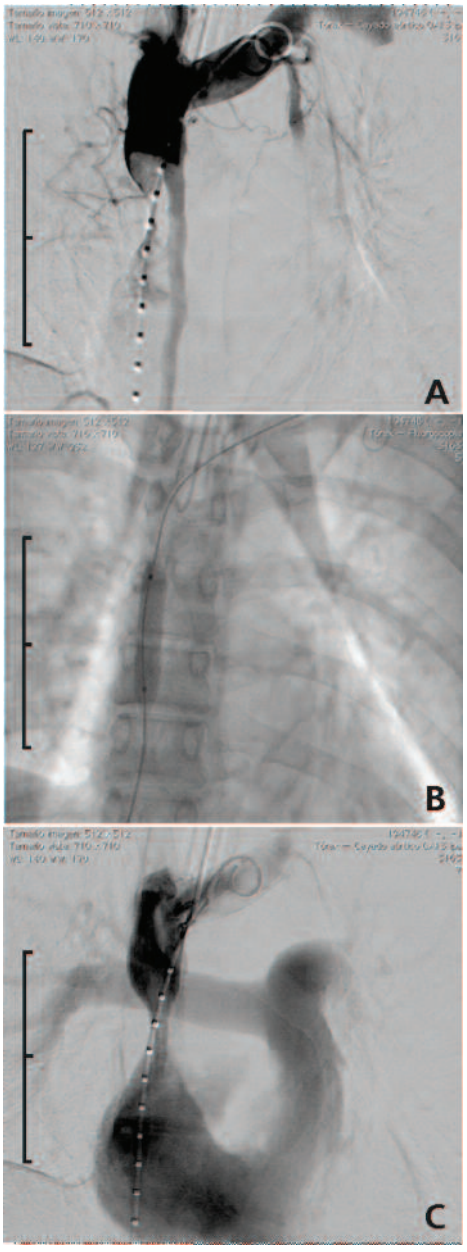

Fig. 2. Images obtained during cavography. **A.** The image shows a complete obstruction of the superior vena cava, the pig catheter inside the vein used for injection of contrast agent, and blood flow from the azygos system. **B.** Balloon angioplasty in the superior vena cava. **C.** Patent vena cava with passage of contrast agent to the right atrium and right ventricle, the pulmonary artery and its branches.

(magnetic resonance imaging and magnetic resonance angiography of the brain), transcranial Doppler ultrasound and funduscopy were normal.

The patient was discharged and is currently followed-up in the outpatient clinic.

Our patient was admitted after undergoing radiofrequency catheter ablation of atrial tachycardia using a three-dimensional navigation system. Twenty-four hours later, the patient presented acute thrombosis of the superior vena cava with superior vena cava syndrome, requiring immediate balloon angioplasty, with favorable outcome.

Non-thrombotic etiology of superior vena cava syndrome is related with lung neoplasms. (1)

With the advent of intravascular devices, thrombosis has become an important cause of this syndrome (30%) particularly with dialysis catheters, portacaths, pacemakers and defibrillator devices. (2) However, there are no reports of thrombosis associated with invasive procedures as radiofrequency catheter ablation.

Undoubtedly, in this patient the early diagnosis and the adequate treatment were possible due to the history of the procedure on the superior vena cava, and the presence of clinical signs and symptoms. Congestion and swelling in the upper part of the body associated with signs of venous congestion and increased central venous pressures were consistent with this syndrome. The most common signs are collar of Stokes edema, distended veins of the nape and chest and facial swelling. The symptoms associated with high venous pressure are commonly present. Dypnea, laryngeal stridor, dysphagia and coughing are related with swelling of the larynx and pharynx. (3) Our patient rapidly evolved with hemodynamic impairment and consciousness deterioration. Cardiac tamponade was an alternative diagnosis that was ruled out.

The diagnosis was confirmed by computed tomography angiography with intravenous contrast injection, although magnetic resonance angiography is another option depending on its availability. (4)

The initial approach during acute thrombosis depends on the cause and on the severity of symptoms. The first step is intravenous anticoagulation. (5) Thrombolysis associated with anticoagulant therapy is recommended in most cases. Endovascular management, as balloon angioplasty, is also recommended, as we did with our patient. This procedure rapidly restores blood flow and vessel patency. The combination of both treatments increases the risk of bleeding without improving the long-term outcome. (6)

This is the first case report of superior vena cava thrombosis after radiofrequency catheter ablation of atrial tachycardia. This entity is infrequent but may develop due to the greater use of radiofrequency catheter ablation as treatment of cardiac arrhythmias.

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Torsades de pointes in amiodarone-associated acquired long-QT syndrome

Amiodarone was developed in Belgium in 1961 and became popular in Europe for the treatment of angina. Based on Dr. Bramah Singh's investigation, (1) the Argentine physician Dr. Mauricio Rosenbaum started using amiodarone for the treatment of ventricular and supraventricular arrhythmias with good outcomes. (2, 3) This drug is a class III agent in the Vaughan Williams classification, with class I. II and IV antiarrhytmic effects. Amiodarone produces bradycardia, prolongs myocardial action potential and delays ventricular repolarization. Due to these three pharmacological properties amiodarone prolongs the QT-interval, predisposing to torsade de pointes (TDP), a polymorphic ventricular tachycardia (VT), in about 1% of patients under this treatment. (4)

We report the case of a 69-year-old woman with history of hypertension, coronary artery disease with previous myocardial infarction requiring stent implantation in the proximal left anterior descending coronary artery. Figure 1 shows the electrocardiogram (ECG) after the angiography. A carotid endarterectomy was performed 5 months before due to an atheroembolic stroke. During the postoperative period, she presented atrial fibrillation with rapid ventricular response and amiodarone was added to her normal treatment. Her current treatment is ASA 325 mg/day, atenolol 50 mg bid, enalapril 20 mg bid and amiodarone 200 mg bid. One month before the event she attended the outpatient clinic and an echocardiogram was performed showing: normal left ventricular dimensions, mildly increased wall thickness, normal left atrium and aorta, mild left ventricular dysfunction with an estimated ejection fraction of 50%, hypokinetic basal inferior and mid inferior segments and mitral inflow filling pattern of delayed relaxation (according to her age). Right chamber dimensions and right ventricular function were normal (TAPSE of 20 mmHg). A fibrocalcific trileaflet aortic valve with normal leaflet excursion was observed, with normal gradients and no regurgitation. The mitral valve was normal, without regurgitation, and the tricuspid and pulmonary valves were also normal. There was absence of pericadial effusion and both septae were intact.

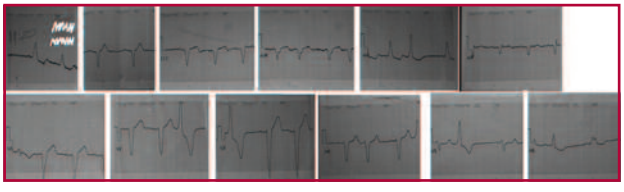

Fig. 1. Electrocardiogram before atrial fibrillation.

She was attended at home by an emergency care system due to several episodes of syncope at rest. When the emergency system arrived, the patient was in cardiac arrest (CA) due to ventricular fibrillation (VF); advanced cardiopulmonary resuscitation was initiated (ACPR) and was defibrillated with 300 J with return of spontaneous circulation (ROSC). Another episode of CA with VF occurred while she was being transferred, requiring two 300 J shocks and 300 mg of intravenous amiodarone, with ROSC.

She was admitted to the emergency department, with a Glasgow score of 9 and breathing room air. Her blood pressure was 90/70, presenting slow capillary refill time and regular heart rhythm at 50 bpm. Another episode of CA in VF occurred at the emergency department. ACPR was initiated and required two 300 J shocks, with ROSC in sinus bradycardia at a heart rate of 40 bpm. Inotropic support was started with dobutamine and a loading dose of 300 mg of amiodarone was administered intravenously. The patient was sedated, intubated, received mechanical ventilation assistance and was transferred to the intensive care unit. The laboratory tests showed: BUN 1.37 g/L, creatinine levels 3.13 mg/dl, K+ 4.8 mEq/L, Na+ 138 mEq/L, Ca++ 10.8 mg/dl, Mg++ 1.9 mEq/L.

Coronary angiography was performed due to a diagnosis of electrical storm secondary to myocardial ischemia.

The ECG showed sinus bradycardia at a heart rate of 35 bpm, normal P-wave and PR interval, old inferior myocardial infarction and poor R wave progression from V1-V6; the QTc was 509 ms. In Figure 2, lead II shows the episode of self-limiting ventricular tachycardia (VT) of 16 beats with change in QRS polarity at 250 bpm and one ventricular triplet in V3.

A diagnosis of polymorphic VT in a patient with long QT-interval (TDP) was made. Amiodarone and inotropic agents were stopped and 2 g magnesium sulphate was administered intravenously. A Furman temporary pacemaker (Figure 3) was implanted and a unipolar lead stimulated the atria at 80 bpm with adequate capture and blood pressure of 120/80 mm Hg.

During the next 48 hours she did not present further episodes of TDP and pacing was stopped. The QT