In this atypical case resulting in death, the first sign of the hyperadrenergic state was STEACS. Pheochromocytoma crises resembling AMI have been reported.<sup>5,6</sup> The abnormalities were explained in terms of severe coronary vasospasm, direct myocardial damage by catecholamines, and increased oxygen uptake as a result of tachycardia and increased afterload. Characteristically, the abnormalities were transient, with normalization after treatment of the tumor. Darzé et al<sup>5</sup> reported a similar case, but with no abnormal cardiospecific markers and with total recovery after treatment with  $\alpha$ -blockers. Our case had the particular feature of clinical, laboratory, and pathological confirmation of established AMI in a patient with no obstructive lesions in her coronary arteries. The involvement of the inferior and posterior walls in the electrocardiogram, ventriculography findings, and the autopsy point to an unusually prolonged coronary vasospasm in the right coronary artery as the most probable trigger of the events experienced by the patient.

Although the patient was transferred for treatment of ACS, the fatal outcome was related to massive brain and pulmonary hemorrhage, probably as a result of hypertensive crises or direct vascular damage caused by the hyperadrenergic state. In any case, administration of antiplatelet agents and anticoagulants as treatment for STEACS may have exacerbated the patient's condition.

In conclusion, the present case highlights one of the characteristics of adrenergic crises. It also points to the wide range of possible presentations that can make an accurate diagnosis so complicated in emergency situations. Santiago F. Coroleu,<sup>a,\*</sup> Juan F. Muñoz-Camacho,<sup>a</sup> Carlos Fernández-Gómez,<sup>a</sup> and Xavier Tarroch-Sarasa<sup>b</sup>

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#### Acute Aortic Syndrome and Rheumatoid Arthritis

# Síndrome aórtico agudo y artritis reumatoide

#### To the Editor,

Rheumatoid arthritis (RA) is an inflammatory immune system disorder that is associated with a higher level of morbidity and mortality than found in the general population, mainly because of cardiovascular disease. In Switzerland, mortality due to RA increased by >50% and, in England, it was 1.5–1.6 times higher, principally due to myocardial infarction but also to valvular dysfunction.<sup>1,2</sup> We report the case of a patient with RA who was admitted for acute aortic syndrome (i.e. an aortic intramural hematoma developing into a dissection). A literature search carried out using PubMed and the keywords "rheumatoid arthritis" AND "aortic dissection" revealed no evidence of an association between the two, which we believe is a possibility.

The patient was a 47-year-old man who smoked 70 cigarettes/ day and had high blood pressure of recent onset, which was being treated with enalapril. He had been diagnosed with RA 7 years earlier and was currently receiving methotrexate, leflunomide, indometacin, folic acid, and deflazacort. He presented with severe retrosternal pain that radiated to the neck and back and which was associated with autonomic symptoms. On admission, his blood pressure was 110/60 mmHg and his heart rate was 80 beats/min. A hemogram and biochemical study gave normal results: the erythrocyte sedimentation rate was 79 mm/h. Thoracic multidetector computed tomography CT (Fig. 1) showed an aortic hematoma associated with a Stanford type-B dissection (i.e. distal to the left subclavian artery). The patient was admitted to intensive care unit where his blood pressure was maintained at <120/80 mmHg. The chest pain disappeared and subsequent examinations revealed partial resolution of the aortic hematoma. An aortic endoprosthesis was used to stabilize the patient's condition.

The association between RA and loss of elasticity in the aorta is principally due to inflammatory changes and structural alterations, such as smooth muscle hypertrophy and changes in the extracellular matrix characterized by an increase in collagen and a reduction in elastin, all of which predispose to increased rigidity of the arterial wall. Moreover, smooth muscle cells can produce bioapatite (they express osteoblastic markers under inflammatory conditions) which causes calcification of the aortic tunica media. Perivascular inflammation and cellular infiltration around the vasa vasorum have been observed to result in ischemia.<sup>3</sup> It is widely recognized that aortic intramural hematoma is caused by rupture and bleeding from the vasa vasorum. We believe that these mechanisms (principally ischemic phenomena in the vasa vasorum themselves) may have triggered the condition in our patient. Moreover, in RA, a type of peripheral neuropathy caused by necrotizing vasculitis of the vasa vasorum has been reported.<sup>4</sup>

Hollan et al.<sup>5</sup> studied changes in the aortic wall (i.e. inflammatory changes) in specimens obtained following coronary artery revascularization surgery, and compared findings in patients with and without RA. They excluded patients with infiltration close to the intima, which can occur in normal individuals, and focused on observations in the tunica media and tunica adventitia. They concluded that the observed changes were greater in patients with RA and that they could represent an



**Figure 1.** Thoracic multidetector computed tomography. A: aortic intramural hematoma (short arrows) associated with dissection (long arrow); hemothorax (asterisk). B: (lower level than A) hematoma in the wall of the ascending and descending aorta (short arrows) associated with dissection (long arrow); hemothorax (asterisk).

inflammatory state that promotes the development of arteriosclerosis and the formation of aneurysms by decreasing the resistance of the arterial wall.

Although initially the vasculitis was attributed to occlusive vascular disease associated with giant cell arteritis (which corresponds to the current understanding of RA, principally with regard to the coronary arteries), it was subsequently clear that inflammatory changes in the aorta associated with high blood pressure can also lead to the formation of an aneurysm and dissection.<sup>6,7</sup>

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# Rotational Atherectomy Through Radial Access With a 7.5 Fr Sheathless Guiding Catheter

## Aterectomía de rotación por vía radial con catéter guía 7,5 Fr sin introductor

## To the Editor,

Rotational atherectomy (RA) is a useful technique in calcified and diffuse lesions. Drug-eluting stents make it possible to treat long lesions and, thus, the use of RA has increased.<sup>1</sup> However, the gauge of the catheters employed in radial access limits the utilization of RA. We describe the use of a 7.5-Fr catheter with a 0.081-inch lumen, designed for radial access, that enables the use of burrs measuring 2 mm or less and allows revascularization immediately after coronary angiography when carried out using a radial approach and when a 1.75-mm or 2-mm burr is required.

Four patients with stable angina underwent coronary angiography via right radial artery with a 6-Fr catheter and 5000 IU of sodium heparin. In all four patients, we found a highly calcified lesion in a vessel larger than 3 mm, and revascularization during the same procedure was indicated. Three of the lesions treated were located in mid left anterior descending coronary artery, at the origin of the first diagonal branch and at the origin of the circumflex artery; the fourth was a long lesion in proximal and mid right coronary artery. Due to the marked calcification and the diameter of the vessel, RA with a burr diameter greater than or equal to 1.75 mm was indicated and the 7.5-Fr Sheathless Eaucath® catheter (Asahi Intecc, Japan) was chosen. Once the heparin dose had been administered, the radial introducer sheath was withdrawn over the 280-cm-long 0.035-inch guiding catheter and the catheter was advanced with its guidewire in the interior. Previously, the catheter had been prepared by introducing the guidewire to the extreme end and maintaining it in that position for 3 to 4 min, in accordance with the recommendation of the manufacturer. At the level of ascending aorta, we began to withdraw the guidewire and the 0.035-inch guiding catheter, and the catheter was introduced into the coronary artery. In two of the