ON TWO CASES OF PENETRATING ATHEROSCLEROTIC AORTIC ULCER

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INTRODUCTION

In 1920, Krukenberg described the existence of a pathologic condition, which he called “a dissection without flap”.¹ However, the recognition of the intramural hematoma and of the penetrating ulcer of the aorta as a distinct clinical and pathological entities needed the development of modern imaging studies. On this basis, Stanson was the first to describe this pathology, in 1986.² Subsequently, many reports identified this condition in asymptomatic, as well as in symptomatic patients.³⁻⁴ Most of them present with acute conditions and the follow-up is scarce. To date, there is only one study that attempted to identify the natural disease course of these two, closely related, conditions.⁵ There are no specific guidelines available for the treatment of these pathologies.

In what follows, we present two cases of penetrating aortic ulcers, which were treated in our institution and which benefited by different therapeutic strategies.

CASE REPORTS

Case 1

A 63-year old male patient was admitted to the intensive care unit of our department, accusing precordial thoracic pain. His cardiovascular risk factors included untreated hypertension, smoking and obesity. His medical history was uneventful. A contrast-enhanced CT scan was carried out in emergency. It showed an enlarged size of the ascending aorta, which was measured at 50 mm in the largest transversal diameter, with a small focal contrast-filled pouch located at the level of the posterior wall. (Fig. 1)
No dissection membrane was found. The suspicion of penetrating ulcer of the ascending aorta, associated with an intramural hematoma, arose. A transesophageal echocardiography was performed, in order to better assess the lesions. The examination demonstrated the presence of severe atherosclerosis of the aorta, with a complex, 4 mm thick plaque of the ascending segment, an atheroma of more than 4 mm in thickness of the aortic arch, and a smaller atheroma of the descending thoracic aorta. A localized rupture of an atherosclerotic plaque was also noted, located approximately 2 cm above the sinotubular junction, with passage of the blood in the aortic wall. (Fig. 2) No dissection flap could be individualized. There was no aortic insufficiency.

The patient was operated in deferred emergency the next day, as he had stable hemodynamics. Upon opening the chest, we found an enlarged ascending aorta, with a large intramural hematoma on its anterolateral aspect, starting just above the sinotubular junction and extending up to the origin of the innominate artery in its upper part. There was extensive atherosclerosis and calcification of the aortic wall, so the aorta was incised using Mayo scissors. (Fig. 3,4) The aortic valve had a normal appearance, and the aortic root had a normal size. Replacement of the ascending aorta with a prosthetic Dacron graft was performed, with a brief, fifteen-minute long, circulatory arrest period for the distal anastomosis. The postoperative evolution was uneventful and the patient was discharged 10 days later.

The pathologic examination of the aortic specimen revealed an extensive atherosclerosis with calcification of the intima, and, associated to these, cystic medionecrotic lesions of the media, with blood cells infiltrating the medial layer.

Case 2

The second patient was an 86-year old woman, with unremarkable medical history (hysterectomy at the age of 45, venous thromboprophylaxis). She presented a violent back pain at home, which regressed...
spontaneously. However, she remained with a persistent, dull, back pain, which determined her to check-in. A contrast-enhanced CT scan was performed upon admission, which showed an intramural hematoma of the ascending aorta, the aortic arch and the proximal segment of the descending aorta, with a contrast-filled pouch of the left anterolateral aspect of the arch. (Fig. 5) Extensive atherosclerosis of the aorta, with calcified plaques, was noticed, especially at the level of the aortic arch. (Fig. 6) No dissection membrane could be seen, and the maximal diameter at the level of the ascending segment was 38 mm. As she wasn’t symptomatic and taking into account her advanced age, she was treated medically with calcium-blockers. Regular surveillance by CT scan was deemed necessary. The CT scan at discharge showed no evolution of the above-mentioned lesions.

Subsequent tomography scanning, which was performed one month later, showed a decrease in size of the intramural hematoma localized at the level of the ascending aorta. However, an aneurysmal dilation of the arch at the level of the arch ulcer could be found. (Fig. 7) The maximal diameter at this level was measured at 57.5 mm.

However, 5 months after discharge, she died a sudden death while at home. No post-mortem examination was performed.

**DISCUSSION**

Penetrating aortic ulcer is a pathology recently defined as an atherosclerotic ulcer that penetrates the internal elastic lamina and extends into the media.\(^3\,^4\,^5\) It is a pathology of advanced age (mean age 72 years) and of extensive atherosclerosis. The radiographic image on contrast-enhanced angiograms, CT scans or MRI studies is a well-limited, contrast-filled defect at the level of the aortic wall. (Fig. 8)

It must be differentiated from the simple aortic ulcer of the intima, which doesn’t disrupt the internal lamina, and doesn’t extend into the other aortic layers. At the level of the media it can have different effects: it can extend transversally or vertically.
The transversal extension, along the medial layer of the aortic wall, can produce either an intramural hematoma, or an aortic dissection. The first situation is the most common, as generally these patients have extensively atherosclerotic aortas, which are thought to prevent the creation of a reentry site.6,7 However, there are reports that present the progression of a penetrating ulcer, accompanied by an intramural hematoma, to frank aortic dissection, with the classical radiological appearance of the "double barreled" aorta on contrast-enhanced CT scan or MRI.8,9

The vertical extension results in the creation of aneurysm at the level of the aortic ulcer,10,11 as was the case with patient 2. However, frank rupture can also happen, either at the initial presentation or in the case with patient 2. Nevertheless, they showed that penetrating ulcer is a pathology that bears a very high morbidity and mortality, with 38% of the patients having rupture at the initial presentation and 73% of the patients surviving discharge and showing worsening or progression to frank dissection over time. On the basis of this dismal outlook, the authors of the study recommend aggressive surgical therapy for all symptomatic patients with this condition, if their associated comorbidities do not contraindicate such a therapy.

However, other groups advocate a more prudent approach.10 They operate on ascending aortic hematomas in emergency only if there is a pericardial tamponade, a frank rupture, a superimposed dissection, an aortic diameter greater than 60 mm or persistent thoracic pain present. Patients are treated medically with β-blockers, ACE inhibitors or calcium blocker in order to maintain the systolic arterial pressure between 100 and 130 mm Hg. Close clinical surveillance is maintained for approximately a month. Patients are converted to surgical treatment for the first three months thereafter and at least yearly afterwards. Patients are converted to surgical treatment if there is persistent pain, an aneurysmal evolution, or the development of a new aortic dissection.

Moizumi Y. et al.10 had 27% of their patients operated on in emergency, with an additional 24% who had their treatment converted to surgery. The remaining 49% of patients presented a regression of the maximum aortic diameter at 1 year, on the control CT-scan images. This strategy implies a very close surveillance of the patient, with serial radiographic examinations. However, as case number two shows, rupture can happen at any time after the acute syndrome, especially in the first 6 months.

More recently, endovascular stent-graft treatment of acute aortic syndromes gained increasing acceptance, especially in the high-risk subgroup of patients and with lesions located in the descending thoracic aorta.13,14 The advantages of this procedure are: mini-invasive approach, by surgical cutdown of the femoral artery, low heparinization, avoiding cardio-pulmonary bypass and aortic crossclamping. As penetrating ulcer is a pathology of advanced age and widespread atherosclerosis, these patients often have important comorbidities (coronary artery disease, renal failure, neurological deficits, "porcelain thoracic aorta") that might contraindicate a surgical approach. Acute (<30 days) complications associated with this procedure are essentially ischemic: left arm ischemia, if the ostium of the subclavian artery was overstented, and paraparesis/paraplegia in the case of lesions located in the distal third of the descending aorta, when stenting might occlude the origin of the Adamkiewicz artery. Chronic complications after endovascular stenting are pseudoaneurysm formation and rupture of the aorta, usually at the proximal stent insertion site.

In our opinion, to operate a patient with intramural hematoma of the ascending aorta, with or without penetrating ulcer, is a sensible thing to do, when we take into account the natural history of this condition. Most of the studies published agree on this point, in spite of the lack of definitive guidelines for the treatment of this pathology. On the other hand, lesions in the descending aorta may be managed by either medical treatment alone, with close clinical and radiological surveillance, when the initial presentation is subacute, or by stent-graft placement, when occurring in an acute setting, or on an elective basis.

REFERENCES