SHORT REPORT

Spontaneous Rupture of a Non-aneurysmal Non-inflammatory Atherosclerotic Aorta

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Introduction

Aneurysm formation and arterial wall dissection are usually responsible for spontaneous aortic rupture. In this report we present a case of a 78-year-old woman with spontaneous rupture of a non-aneurysmal, non-inflammatory atherosclerotic infrarenal aorta. An emergency aortic repair with an aortobi-iliac graft was successfully performed. We also present the relevant literature because of the rareness of this case.

Case Report

A 78-year old woman was admitted to the General Hospital of Athens, with a two day history of hypogastric pain. She had a 30-year history of hypertensive disease that was controlled with anti-hypertensive medication. She had recently developed atrial fibrillation treated with anticoagulant therapy. The patient had no history of recent infection.

Abdominal examination revealed a painful expansile mass lying to the left of the umbilicus. The blood pressure was found to be 120/78 mmHg. Distal pulses were palpable on both legs equally. Laboratory examination revealed a white blood cell count of $11,800 \times 10^9/l$ and a hematocrit level of 28%.

During her hospitalization in the medical ward, a full work-up was obtained including blood cultures, a transthoracic echocardiography scan for the detection of endocarditis and serological tests. Among them, there were tests for any evidence of chronic infection (such as Syphilis and Brucellosis), and also for evidence suggestive of rheumatic or connective tissue disease. All were negative or within normal range.

An abdominal CT scan was obtained (Fig. 1). At the level of the aortic bifurcation a large para-aortic contrast-containing mass was revealed. The aorta was of normal size but extensively atherosclerotic. Urgent digital subtraction aortography was performed. A leak of contrast-containing material from the aortic bifurcation was noted (Fig. 2).

At laparotomy, a large pulsatile para-aortic mass extending to the left posteriorly and superiorly in the retroperitoneal space was found. There was no evidence of aneurysmal dilatation, dissection, infection or inflammation of the vessel. The abdominal aorta was found to be extensively calcified. There was a hole in the aortic wall, lying posteriorly and to the left, just above the aortic bifurcation, through which a sharp atheromatous plaque was protruding, with bleeding into the left retroperitoneal space. Aortic repair was performed using a $14 \times 7$ mm Dacron prosthesis and the retroperitoneal haematoma was drained. Bacterial cultures obtained from the aortic wall were sterile.

The patient’s postoperative course was uneventful. During her hospitalization, she received a second-generation cephalosporin for five days. She made a quick recovery and was discharged on the eight postoperative day. After a 36-month follow-up she remains in good health. Again, there was no deviation regarding her serological tests for evidence of infection or any chronic inflammatory disease.

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Discussion

Perforation of a normal-sized aorta has rarely been described. In the few reported cases, the site of spontaneous rupture of a non-aneurysmal vessel through an atheromatous plaque is most often seen at the level of the distal descending thoracic aorta. In 1953, Copping, in an autopsy study of 151 patients with a ruptured abdominal aorta, found only two cases of non-aneurysmal rupture. No clinical details were included in that report. In 1974 Lagaay reported a 12-year experience (1958–1970) with 100 ruptured abdominal aortic aneurysms. In this series, he described four patients with spontaneous aortic perforations in the absence of aortic dilatation or inflammation. In two cases the diagnosis was confirmed angiographically, and in two during an emergency laparotomy because of profound hypotension and abdominal pain. In all patients the extravasation of blood was found in the retroperitoneal space to the left of the aorta. The site of rupture was noted to be close to the origin of the inferior mesenteric artery in each case.

The histological appearance of the aorta in our patient was that of severe atherosclerosis. In the recent literature there are five more cases with spontaneous rupture of a non-aneurysmal infrarenal aorta, with a history either of diabetes mellitus, hypertensive disease and alcohol and tobacco abuse.

The radiological and intra-operative findings in our patient revealed the presence of atheromatous thinning and extensive calcified atherosclerotic disease of the infrarenal aorta. The absence of aneurysmal dilatation or any inflammatory process, suggests that aortic rupture may have resulted from transmural plaque fracture through an area of atheromatous thinning or from a penetrating ulcer.

The precise incidence of penetrating aortic ulceration is unknown and is characterized by ulceration of an atheromatous plaque disrupting the internal elastic lamina. The ulceration in the atheroma may extend into the media, causing an intramural hematoma. Penetration through the media or perforation through the adventitia may result in pseudoaneurysm formation or transmural aortic rupture. In all cases, even in penetrating aortic ulceration, surgical intervention should be undertaken promptly, usually by the use of an interposition graft at the site of the ulcer.

Although there are many reports regarding the use of endovascular repair in the treatment of ruptured abdominal aortic aneurysms, we have not yet introduced this therapeutic option in our department. Apart from the fact that endovascular repair requires teamwork with highly specialized, well-coordinated personnel, no endovascular graft is available in our unit on an emergency basis.

However, it seems that our patient could be a candidate for endovascular repair, although aorto-iliac anatomic parameters were suboptimal for such an intervention. Our experience in the endovascular management of acute aortic lesions is poor, and, since the possibility of aortic infection could not be initially excluded, we chose the conventional method of treatment.

Fig. 1. CT scan of the atherosclerotic abdominal aorta, showing a large para-aortic contrast—containing mass at the level of the aortic bifurcation.

Fig. 2. Digital subtraction aortography showing extravasation of contrast material coming out of the non-aneurysmal distal aorta.
References


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