CASE REPORT

Non-aneurysmal Suppurative Aortic Rupture

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Introduction

Aortic rupture is usually the ultimate consequence of expanding atherosclerotic aortic aneurysms. However, other conditions such as blunt traumatic injury or aortic dissection due to a variety of causes (hypertension, medial degeneration or Marfan's syndrome, and Behcet's disease) may also lead to aortic rupture. In contrast, so-called spontaneous aortic infection usually occurs following secondary infection of the normal or atherosclerotic aortic wall. The main mechanism of aortic infection is the presence of recurrent septic emboli, which may lodge at sites such as the aortic bifurcation, side-branch origins, vasa vasorum or atherosclerotic plaques. A special pathogenetic subgroup are primary or secondary contaminated post-traumatic pseudoaneurysms. These may follow penetrating vascular injury or intraaortic manipulations, such as arteriography or intra-aortic balloon-pumps. During a 5 year period, 54 patients (49 ruptured atherosclerotic aneurysms, one ruptured post-traumatic aneurysm, four infected ruptures) underwent emergency surgery for aortic rupture. The clinical course of the four patients with an infectious aetiology is reported.

Case reports

Patient 1

A 64-year-old woman with fever, leukocytosis and back pain was admitted and a diagnosis of aortic rupture was made after clinical and sonographic examination. Angiography (Fig. 1) showed a rupture without an aortic aneurysm. The location of the retroperitoneal rupture was below the left renal artery. A dacron vascular prosthesis was interposed in the anatomical position and covered with antibiotic chains in combination with omentoplasty. Blood cultures taken before operation grew Staphylococcus aureus, and a diagnosis of bacterial endocarditis was made on echocardiography. The patient was discharged 4 weeks later and was well 5 years later.

Patient 2

A 75-year-old man developed sudden circulatory collapse and signs of septic shock. An aortic aneurysm was not detectable on ultrasound examination but a CT-scan (Fig. 2(a)) showed an infrarenal aortic rupture (Fig. 2(b)) and a tube resection performed. The vascular wall contained Gram-negative bacteria and histologically it was replaced by granulation tissue with suppurative necrosis. A source of the infection could not be found and the patient died 8 weeks later due to septic multiorgan failure.
Patient 3

A 68-year-old man presented with aortic rupture due to direct aortic infiltration of a large retroperitoneal tuberculous abscess. CT-scans showed a hypodense, non-homogeneous abscess formation in the region of the right psoas muscle with ureteral obstruction (Fig. 3). During operation, the distal aorta and the common iliac arteries had to be ligated to control bleeding. The retroperitoneal space and the abdominal cavity were lavaged, and the abdomen closed. An extra-anatomical bypass from the ascending aorta to both femoral arteries was performed. The patient was transferred to an infectious unit 4 weeks later, with primary wound healing and palpable pulses.

Patient 4

A 76-year-old man developed chest pain and signs of haemorrhagic shock. A chest roentgenogram showed bilateral pleural effusions, and a rupture of the thoracic aorta was suspected. A left-side thoracotomy was performed without any further diagnostic procedures. After clamping the descending aorta and clot removal a localised necrosis was found below the left subclavian artery. The necrotic arc was excised and an interposition graft performed, but the patient died postoperatively due to progressive acute respiratory distress syndrome (ARDS). Bacterial endocarditis was diagnosed at autopsy.
Discussion

Spontaneous aortic infection with subsequent necrosis of the vascular wall was first described by Virchow in 1847° and Sir William Osler was the first to recognise a link between infection and aneurysmal disease. In this classical Gulstonian lectures to the Royal College of Physicians in London 1885, he described the association with subacute bacterial endocarditis and coined the term “mycotic” aneurysm. The embolic origin of vascular infection was clarified by Eppinger in 1987 with the term “embolo-mycotic”, since the same bacteria were found in the aortic wall and in vegetative lesions in a patient with endocarditis. Aortic infection can also develop in patients with pneumonia or osteitis, a condition first published by Stengel and Wolferth in 1923. Parkhurst in 1955 created the term “bacterial aortitis”. Systemic infection with Salmonella enteritidis may also lead to arterial rupture. A classification of arterial infections based on the preexisting vascular status (normal; atherosclerotic; aneurysmal), or on the source of infection (intravascular; extravascular) was proposed by Patel. However, the variety of terms, including mycotic aneurysm, embolo-mycotic, primary and secondary aneurysm, cryptic or bacterial aortitis, suppurative aortitis, and non-aneurysmal suppurative aortitis, reflects the lack of common terminology throughout the literature.

Peripheral embolisation of infected debris from cardiac valves in patients with proven endocarditis is a known mechanism, sometimes leading to the development of secondary aneurysms, with a wide spectrum of infectious agents including haemolytic streptococci, staphylococci, pneumococci, Haemophilus influenzae, salmonellae, serratiae, enterococci. In our opinion, the term “spontaneous non-aneurysmal suppurative aortic rupture” best reflects the situation of inflammatory necrotic destruction of the aortic wall without coexisting aneurysm. Its incidence is difficult to assess, since it is a rather rare event. In our series, the incidence was 8% (four of 54 patients with aortic rupture). The clinical signs are often difficult to interpret with intermittent fever and chills, leukocytosis, bacteraemia, back pain and shock. The commonest cause is endocarditis, but rheumatic fever, retroperitoneal abscess formation, salmonellosis, multiple arterial catheterisations, surgical manipulations, penetrating arterial trauma or drug abuse may also lead to suppurative aortitis. Angiography or dynamic CT are usually diagnostic when aortic rupture is suspected but ultrasound shows no aneurysm.

The surgical treatment of suppurative aortic rupture remains controversial and depends on the primary source of infection. If infection is diagnosed intraoperatively by Gram stain or by visible paraaortic abscess formation, extra-anatomical procedures may be safer to avoid postoperative graft infection, but may result in prolonged lower extremity ischaemia. Axillo-bifemoral or bilateral axillo-femoral bypass grafting as recommended by some authors is also associated with lower long-term patency than aorto-bifemoral grafting. In a literature review of 34 survivors with spontaneous abdominal infections, only 12 patients underwent extra-anatomical bypass procedures, but long-term follow-up is not available. There is also debate about whether extra-anatomical procedures should be performed first to avoid graft infection after excision of the infected aorta although this procedure would not seem possible in patients with hypovolaemic shock. In the case of endocarditis or other remote sources of infection with secondary septic embolism to the aortic wall, the decision to perform an in-situ repair is dependent of an intraoperative Gram stain; if it is negative, primary graft interposition following wide debridement may be possible, with or without omental flap. Long-term antibiotic therapy must be guided by knowledge of the final result of the bacteriology and should be continued for at least 3 months.

References

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