

Juxtarenal Mycotic Aneurysm as a Complication of Acute Exacerbation of Chronic Cholecystitis Treated by Resection and Replacement by a Fresh Allograft

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Abstract: We present a case of a female patient with infectious (mycotic) juxtarenal abdominal aneurysm with atypical symptoms beginning as acute exacerbation of chronic cholecystitis. Apart from common antibiotic treatment, the patient successfully underwent resection of the diseased segment and replacement by a fresh allograft in order to reduce the risk of infection of the graft, but with the need of subsequent life-long immunosuppressive therapy. Perioperative monitoring of the spinal cord by near infrared spectroscopy was used to identify possible spinal ischemia. The choice of the fresh allograft was based on our experience supported by review of the literature.

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Introduction

In the recent years, we have observed increasing numbers of patients with infectious aneurysms with atypical disease course. We decided to report one particular case of juxtarenal mycotic aneurysm with atypical symptoms beginning as acute exacerbation of chronic cholecystitis. Apart from antibiotic treatment, the patient underwent resection of the aneurysm with replacement of the segment by a fresh aortic allograft in order to reduce the risk of infection of the graft. The procedure was performed in general anaesthesia with perioperative monitoring for spinal ischemia.

Case report

In September 2012, a 68-years-old female patient was admitted to a General University Hospital for abdominal and back pain. The pain was increasing gradually for a week, with maximum in the right upper quadrant and irradiation into the

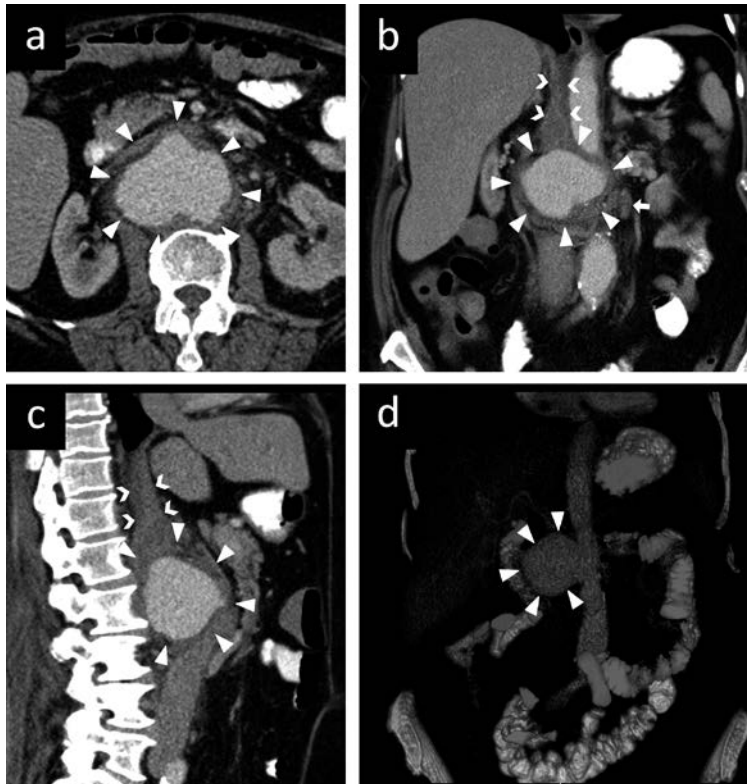


Figure 1a–d – CT of abdomen in portal venous phase shows large juxtarenal sacular mycotic aneurysm (72×51×66 mm, arrowheads) with thick (up to 7.4 mm) indistinct wall, stranding of adjacent retroperitoneal fat, enlarged lymph nodes (arrow) and signs of past leakage (chevrons) in axial (a), coronal (b), sagittal (c), and 3D (d) reconstructions.

right lumbar region. She reported no fever or chills. Initial clinical examination was unremarkable, but blood tests revealed elevated leukocyte count $14.1 \times 10^9/l$ (reference = $4-10.7 \times 10^9/l$, Leu), and high C-reactive protein 220 mg/l (reference = 0–7 mg/l, CRP). Abdominal ultrasound showed solitary bile stone and signs of acute exacerbation of chronic cholecystitis (irregular thickening of the gallbladder wall up to 9.5 mm with increased echogenicity), liver steatosis, and aortic aneurysm 32×30 mm in diameter. Therefore, she was treated for acute exacerbation of chronic cholecystitis by cephalosporin antibiotics (cefoperazone, Cefobid[®], Pfizer, Czech Republic, 2 g every 12 hours i.v.). However, this treatment had no influence on her symptoms and laboratory markers of inflammation were increasing (CRP 315 mg/l, Leu $16.7 \times 10^9/l$). Blood cultures were negative, but serology identified acute *Salmonella enteritis* infection. CT of abdomen was requested to rule out complications. It showed moderate thickening of the gallbladder wall outlined by a thin rim of fluid consistent with the previously stated diagnosis. More importantly, the juxtarenal aneurysm previously identified by ultrasound substantially increased in size (diameter = 72×51 mm, length = 66 mm) and became saccular in shape (Figure 1). Its wall was thick (up to 7.4 mm), indistinct, blurred with stranding of the adjacent retroperitoneal fat and signs of past leakage. One week after admission, the patient was referred to the Department of Cardiovascular Surgery for juxtarenal infectious (mycotic) aneurysm.

Because implantation of a stentgraft into an infectious aneurysm would pose a high risk of infection of the prosthetic material, we decided to perform replacement of the diseased segment by an allograft. The fresh allograft was available five days later.

Anaesthesia

Replacement of a 16 cm long visceral segment of the abdominal aorta was performed in general anaesthesia with selective intubation of the right bronchus and patient in the right lateral decubitus position. Prior to induction of anaesthesia, a thoracic epidural catheter was inserted at the level of T6-T7, as well as intraspinal catheter (Codman Neuro, USA) for drainage of the cerebrospinal fluid and measurement of the pressure inside the subarachnoid space. The anaesthesiologist applied complex hemodynamic monitoring including invasive blood pressure measurement above and below the aortic clamp during the procedure. Monitoring of advanced hemodynamic parameters, such as the stroke volume, cardiac output/index and systemic vascular resistance was achieved with a Vigileo system (Edwards Lifescience, USA) and heart performance was assessed with the transesophageal echocardiography (TEE) throughout the procedure. Regional dermatomal oxygenation at the level of L1 was continuously measured using near-infrared spectroscopic (NIRS) oximetry (INVOS, Covidien, USA) (Etz et al., 2013).

Surgical procedure

The aorta was accessed through a left thoracotomy in the fifth intercostal space (left lung was deflated) and midline laparotomy. There were inflammatory changes in the tissue around the aorta with enlarged periaortic lymph nodes. The aortic wall was of inferior quality with decreased compliance due to multiple atherosclerotic plaques. After introduction of a left heart bypass (from the left upper pulmonary vein to the left common femoral artery) under full heparinization (Heparin Leciva, Leciva, Czech Republic, 0.5 mg/kg), an aortic cross-clamp was placed on the thoracic aorta 5 cm above the diaphragm and on the abdominal aorta above the celiac artery. The proximal end-to-end anastomosis was constructed after transverse aortotomy 2 cm above the diaphragm. Then, a cross-clamp was placed onto the distal end of the allograft and on the aorta above the bifurcation. The diseased visceral aorta was resected while the origins of the celiac artery, upper mesenteric artery, and the left and right renal artery were connected to a biopump (BPX-80 BIO Pump Plus Centrifugal Blood Pump, Medtronic, USA) in order to maintain their perfusion. Then the visceral arteries were connected to the allograft in the following order: right renal artery (end-to-side), celiac artery (end-to-end), upper mesenteric artery (end-to-end), and left renal artery (end-to-end). Finally, the proximal aortic cross-clamp was removed to restore circulation in the visceral arteries and the distal aortic anastomosis was constructed (end-to-end). The biopump was suspended and after cancellation of heparinization by equal dose of protamine sulfate (Protamin ME, MEDA Pharma, Germany), control of bleeding, and placement of drains, the wounds were sutured.

Postoperative care

The double-lumen tube was replaced with a standard endotracheal tube at the end of surgical procedure. The patient was then transferred to a postoperative intensive care unit, ventilated, with circulation on a moderate vasopressor support (noradrenaline, 0.3 µg/kg/min). As hemodynamic parameters gradually improved, she was extubated on the third postoperative day.

On the 7th postoperative day, debridement of the laparotomy was performed because of purulent discharge (*Staphylococcus aureus* was identified by culture) and vacuum-assisted closure (VAC) system (V.A.C.[®] Therapy System, KCI, USA) was introduced and exchanged every four days, four times altogether. Later the wound was resutured.

The antibiotics were administered during the postoperative period for another four weeks. Postoperatively, we observed a marked decrease of CRP (23.6 mg/l) and Leu ($7.15 \times 10^9/l$). The patient was discharged on the 25th postoperative day with the following medication: acetylsalicylic acid 100 mg in the morning (Anopyrin 100 mg, Zentiva, Czech Republic) as antiaggregant and tacrolimus 1 mg, 1 pill in the morning and evening, 2 pills at noon (Prograf 1 mg, Astellas Pharma, Czech

Republic) as immunosuppressant with dose adjustment to maintain plasmatic levels between 4 and 7 ng/l.

Follow up

One month after discharge, the patient did well and the operative wounds were healed. CT angiography showed 80% stenosis of the proximal part of the left renal artery (Figure 2), which was successfully treated by angioplasty and stenting two weeks later. Three months postoperatively, the patient underwent laparoscopic cholecystectomy. Histology showed chronic fibrous cholecystitis with foci of acute exacerbation.

Discussion

Infectious aneurysms represent 1% of all thoracic and 3% of abdominal aneurysms (Laohapensang et al., 2012). Patients with diabetes, immunodeficiency, drug users,

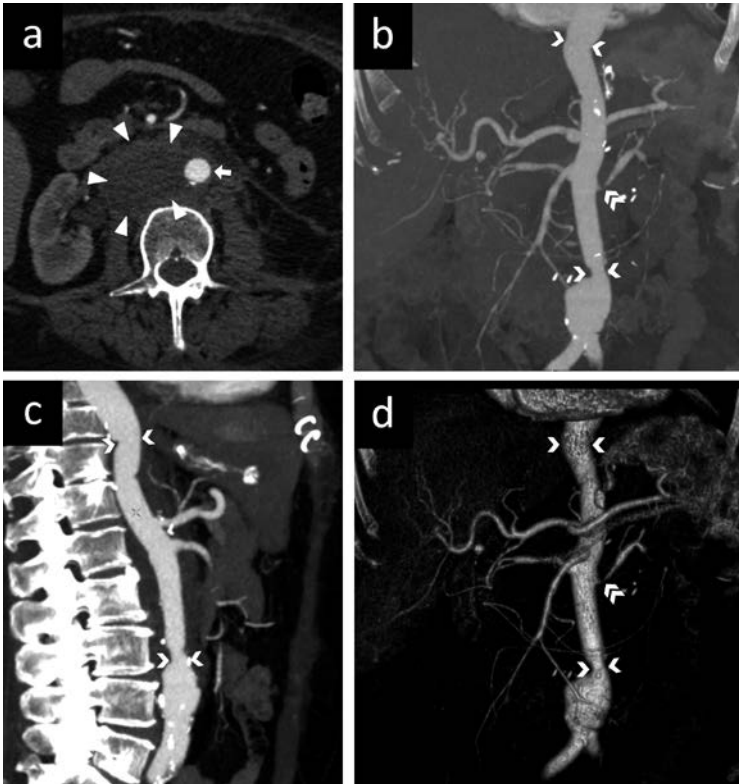


Figure 2a–d – CT angiography of abdominal aorta after resection of mycotic aneurysm and replacement by a fresh allograft shows good postoperative result apart from stenosis of the left renal artery (double chevron) and retroperitoneal hematoma (arrowheads) in axial (a), coronal (b), sagittal (c), and 3D (d) reconstructions. Chevrons indicate the proximal and distal anastomosis.

and patients after surgical procedures in the thorax (coronary bypass, replacement of the ascending aorta) are at increased risk (Karkos et al., 2014). The infection usually invades the aortic wall through a diseased intima (most commonly by atherosclerosis), or through vasa vasorum (Sharma et al., 2011). Apart from sepsis, patients with infectious aneurysms are endangered by imminent rupture of the aneurysm due to accelerated destruction of the aortic wall. Blood cultures are positive only in half of the patients. Even though all patients have elevated markers of inflammation (Leu, CRP, procalcitonin), the mainstay of diagnosis consists in cross-sectional imaging, CT, MRI, or PET-CT in particular (Steverlynck and Van de Walle, 2013).

The treatment of infectious aneurysms is complex. Its primary objective is to manage the infection by means of antibiotic therapy for at least six weeks. The definite treatment, however, is surgical resection of the aneurysm and bridging of the aortic segment by a prosthetic graft with the risk of its infection or by an extra-anatomic bypass, which has a limited durability. Pure endovascular treatment, first published by Semba et al. in 1998, that is reserved for high risk patients, who cannot undergo operation, has a high infection rate of the stentgraft (9–20%) with undiminished risk of subsequent rupture (Semba et al., 1998; Strahm et al., 2012). Therefore, replacement of the diseased segment by an allograft is a viable option. However, even this solution has its drawbacks: the allograft is usually not readily available and, above all, the patient has to be on lifelong immunosuppressive treatment. In the abdominal aorta, the reported mortality is lower, about 10%, but in the thoracic aorta prohibitively high, up to 45% (Scali et al., 2013).

Replacement of the abdominal aorta with reconstruction of the visceral arteries is a complex procedure and there are several technical aspects that need to be considered. Firstly, to maintain perfusion of the abdominal viscera, a biopump is the preferred option, because lower dose of heparin is required compared to other alternatives. Secondly, although the abdominal aorta can be accessed via the retroperitoneal approach, we chose the intraperitoneal access via the left paracolic gutter because it is less demanding, faster, offers better visualization of the operating field and direct assessment of perfusion changes of the abdominal viscera. Thirdly, hypothermia which may be desirable for protection of the spinal cord and the abdominal organs increases the risk of bleeding from the wound area, which is rather large in this procedure. Fourthly, although we prefer to perfuse the disconnected vessels with blood, which contains buffers and transports gases in physiological amounts, protective solutions such as Ringer's lactate solution with mannitol and solumedrol may be used alternatively. Fifthly, perioperative and postoperative monitoring of the spinal pressure is mandatory for early detection and management of hypertension that may be caused by edema of the spinal cord. Sixthly, antibiotic treatment was guided by the clinical and laboratory examinations and in the postoperative period it was terminated

after the fourth postoperative week based on mutual agreement with the Clinical Microbiology and Antibiotic Center, although the general recommendations suggest a minimum of 6 weeks.

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