CASE REPORTS

RUPTURED MYCOTIC ANEURYSM OF THE ILLIAC ARTERY COMPLICATED BY EMPHYSEMATOUS PSOAS MUSCLE ABSCESS: REPORT OF TWO CASES

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Abstract: Emphysematous psoas muscle abscess has rarely been described and has not been reported to be associated with ruptured mycotic aneurysm. We report two cases of ruptured mycotic iliac arterial aneurysm complicated by emphysematous abscess of the left psoas muscle. Case 1 occurred in a 70-year-old man and Case 2 in a 63-year-old woman. Both patients presented with fever for several weeks. Clinical clues leading to the diagnosis included a palpable abdominal mass with (Case 2) or without (Case 1) pulsation, blurring of the psoas muscle shadow with abnormal gas distribution on the plain abdominal film (Case 1), and peripheral vascular insufficiency and Salmonella bacteremia (Case 2). Ruptured mycotic aneurysm of the left iliac artery complicated with left psoas muscle abscess was clearly demonstrated by abdominal computerized tomography scan and intravenous digital subtraction angiography in both cases. Causative agents, multidrug resistant Acinetobacter baumannii and Klebsiella pneumoniae, unusual pathogens for mycotic arterial aneurysm, were cultured from debrided tissue in Case 1, and this finding led to the speculation that the infection was hospital-acquired. The favorable outcome in Case 2 resulted from early vascular surgery and a prolonged course of effective antimicrobial therapy.

Mycotic aneurysm is uncommon in the era of modern antimicrobial therapy. Although the major reported pathogen has been Salmonella species, the incidence of Staphylococcus aureus infection has been increasing [1]. The diagnosis of mycotic aneurysm is usually based on the classical features of fever, abdominal or chest pain, positive blood cultures, and a pulsatile mass [2, 3]. However, the clinical manifestations of mycotic aneurysm are variable. A patient may present with Salmonella sepsis of unknown origin with rapid deterioration into a fatal outcome due to aneurysmal rupture [2]. We report two cases of ruptured mycotic aneurysm of the left common and internal iliac artery, respectively, both of which were complicated by surrounding emphysematous psoas muscle abscess.

Case Reports

Case 1
This 70-year-old man with diabetic nephropathy who had received regular hemodialysis therapy for 7 years visited a local hospital due to nonproductive cough and fever, where urine and blood cultures yielded no pathogen and no abnormal infiltration was noted on chest roentgenogram. Piperacillin, amikacin, and vancomycin were prescribed without response. Due to progressive consciousness disturbance and prolonged fever for 40 days, he was transferred to National Cheng Kung University Hospital (NCKUH) on April 2, 1996. At admission, he had hypotension (93/54 mmHg) and tachycardia (pulse rate 161/min). Physical examination

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revealed a non-pulsatile mass of 8 cm in diameter over the left peri-umbilical area. The serum C-reactive protein (CRP) concentration was 212 mg/L. Computerized tomography (CT) scan showed only brain atrophy and cerebrospinal fluid study excluded meningitis. Abdominal ultrasonography showed bilateral renal parenchymal disease and left renal stone with hydronephrosis and hydroureter. Blurring of the left psoas muscle shadow with abnormal gas distribution was noted on abdominal plain film. Abdominal CT scan disclosed left psoas muscle abscess with gas formation (Fig. 1A) and an aneurysm with pseudoaneurysm formation in the left common iliac artery, as demonstrated by intravenous digital subtraction angiography (DSA) (Fig. 1B). No valvular vegetation could be detected by echocardiography. Profound shock and coma occurred on the second day after admission. Surgery was deferred due to high surgical risk and broad-spectrum antibiotic therapy with vancomycin, clindamycin, and ciprofloxacin was prescribed empirically. His hemodynamic status and consciousness level improved gradually. Multiple sets of blood cultures were found to be sterile. On the 10th day of hospitalization, he underwent surgical excision of the fragile abdominal aorta and bilateral common iliac arteries and debridement of the psoas muscle abscess. An aortobifemoral bypass was created. *Acinetobacter baumannii* and *Klebsiella pneumoniae* grew in cultures of debrided tissue. However, he died of profound shock on the day after surgery.

**Case 2**

This 63-year-old woman was admitted to NCKUH on August 4, 1996, complaining of progressive edema and pain over the left lower leg for 1 month and intermittent high fever for 2 weeks. She had hypertension and received regular medication. At admission, her blood pressure was 150/78 mmHg, and she had fever (axillary body temperature 39°C), tachycardia (pulse rate 123/min), and tachypnea (respiratory rate 25/min). Leukocytosis (leukocyte count 1.35 x 10⁹/L) with left shift and anemia (hemoglobin 9.9 mg/dL) were noted. The serum CRP was 201 mg/L. Physical examination revealed a pulsatile mass over the left lower abdomen. Duplex sonography disclosed deep vein thrombosis of the left femoral vein and arterial insufficiency below the left popliteal artery. Abdominal and pelvic CT scan revealed a ruptured aneurysm of the left internal iliac artery complicated by emphysematous psoas muscle abscess (Fig. 2A). Intravenous DSA revealed an aneurysm of the left internal iliac artery (Fig. 2B). No valvular vegetation was found on echocardiography. Ligation of the aneurysm and femoral-femoral bypass were performed. The postoperative course was complicated by pelvic abscess, which was managed with surgical drainage. Initially, *Salmonella choleraesuis* and *Escherichia coli* bacteremia were found. *S. choleraesuis* grew in cultures of the debrided tissue. The patient was cured with a prolonged course of oral cefixime and was well during 4 years of follow-up.

**Discussion**

Osler first described the term mycotic aneurysm in 1885 as a fungus-like vegetation at the orifice of the aortic arch, but the causative microorganisms were usually bacteria, not fungi [4]. Aortic aneurysm is a pathologic dilatation of the normal aortic lumen and mycotic aneurysms result from inflammatory weakening of the wall of an artery caused by septic microemboli to the vasa vasora or by impaction of an infected embolus in the lumen of the artery. Mycotic aneurysms constitute 2.6% of all aortic aneurysms [5]. Prior to 1965, endocarditis was associated with 37% of mycotic aneurysms, and thereafter with 10% [6]. Other etiologies include arterial trauma due to accident or surgical manipulation, radiologic invasive diagnostic procedures, and drug abuse [6, 7]. Patients with diabetes mellitus, hypertension, a smoking habit, autoimmune disease, immunosuppression due to steroid or chemotherapy, irradiation, and malignancy are all predisposed to the development of mycotic aneurysm [7].

**Fig. 1.** Case 1. A) Computerized tomography scan reveals aortic aneurysm of the left common iliac artery with pseudoaneurysm formation (arrow) and emphysematous psoas muscle abscess. B) Intravenous digital subtraction angiography reveals pseudoaneurysm in the left common iliac artery.

**Fig. 2.** Case 2. A) Abdominal computerized tomography scan reveals left emphysematous psoas muscle abscess. B) Intravenous digital subtraction angiography reveals aneurysm of the left internal iliac artery.

The most prevalent offending microorganisms are Salmonella species, which appear to have a unique predilection for seeding the atherosclerotic abdominal aorta [2, 8]. However, the incidence of S. aureus infection has risen from 19% to 30% [1]. Other pathogens such as E. coli, Streptococcus, Clostridium perfringens, Proteus, Brucella, Pseudomonas, Campylobacter fetus, Yersinia enterocolitica, and Burkholderia pseudomallei have been reported occasionally [9–11]. Infection with mixed flora is rare [12].

Most patients with mycotic aneurysm have longstanding fever (3 wk to 1 yr), leukocytosis, abdominal or back pain, and a pulsatile abdominal mass if the aneurysm is located in the abdomen [3]. Complications associated with mycotic aneurism include distal embolization of infected thrombi, sepsis, and rupture. Abdominal mycotic aneurysms may rupture into the retroperitoneum (50–70%) with retroperitoneal bleeding, into the gastrointestinal tract (32%) with gastrointestinal bleeding, or into the iliofemoral system resulting in an arteriovenous fistula (8%), as reported by Fichelle et al in 25 cases of infected infrarenal aortic aneurysm [12]. Other reported complications include ureteral colic, sciatica, foot drop, nerve root compression syndrome, intractable back pain, systemic arterial insufficiency with myocardial ischemia, cerebral ischemia with syncope, or renal insufficiency [13, 14]. Our patients with abdominal mycotic aneurysms had the unusual complication of emphysematous psoas muscle abscess. Besides the leading symptom of prolonged fever, a palpable abdominal mass was found in both cases. However, only one of these masses was pulsatile. Progression of the inflammatory process beyond the arterial wall and invasion of the adjacent psoas muscle may result in localized abscess with gas collection. As suggested by the radiologic findings, the scenario of the infectious process may involve progression from the arterial wall with a point of leakage in the adventitia to the surrounding psoas muscle. The speculation that the infection extended from the psoas muscle to the adjacent artery is less likely, because such an invasive process would have had to penetrate the arterial adventitia, which would have led to retroperitoneal hemorrhage with hypovolemic shock and immediate death.

Rapid diagnosis and appropriate antimicrobial therapy followed by prompt surgical intervention are necessary for a favorable outcome. Magnetic resonance imaging is the most accurate technique for diagnosis. Other potential diagnostic techniques include CT scan [15], DSA, and radiogallium study [16, 17]. Angiography may be contraindicated because of the risk of aneurysmal rupture. In Case 1, plain abdominal roentgenogram disclosed blurring of the left psoas muscle shadow with abnormal gas shadow.

This radiologic clue led to further abdominal and pelvic CT scan with intravenous DSA examinations. Both disclosed pseudoaneurysm of the left common iliac artery. No pulsation was noted over the abdominal mass because the main mass was a pseudoaneurysm. Pseudoaneurysm is less pulsatile due to the small inlet with less arterial jet into the space. The pathogens cultured from the debrided tissue were A. baumannii and K. pneumoniae, which are rarely reported in mycotic aneurysm. No bacterium was found in blood either before or after admission to NCKUH. This was probably due to previous treatment at the local hospital. The isolation of multi-drug resistant gram-negative bacilli in debrided tissue suggests that these arterial infections may have resulted from the seeding of nosocomial bactereemic pathogens in the atherosclerotic vessel walls rather than from community-acquired infection. In Case 2, accurate diagnosis was made promptly due to the findings of deep vein thrombosis, peripheral arterial insufficiency, and a pulsatile abdominal mass. Deep vein thrombosis in the lower extremity was caused by external compression of the venous system by the enlarged mycotic aneurysm. Emphysematous psoas muscle abscess also complicated the condition. Cultures of debrided tissue and blood both disclosed S. choleraesuis, a common causative pathogen of mycotic arterial aneurysm.

The prognosis is poor if the infected vessel and surrounding tissue are not surgically removed. Death may result from aneurysmal rupture or overwhelming sepsis [2]. Most surgeons consider the excision of an infected vessel with extra-anatomic vascular reconstruction to be the treatment of choice for abdominal mycotic aneurysm [18]. In addition to surgery, a prolonged course of antibiotics for 6 weeks, as in Case 2, or even lifelong is indicated [10]. The fatal outcome in Case 1 might have been related to the delayed diagnosis and surgical interventions.

In conclusion, a high index of clinical alertness leading to early diagnosis, aggressive surgical intervention, and prolonged medical treatment facilitate a favorable outcome in patients with ruptured mycotic aneurysm. Although Salmonella species have been the most prevalent pathogens of mycotic aneurysm, the list of causative agents is expanding. Emphysematous psoas muscle abscess or deep vein thrombosis may be a clinical clue for ruptured mycotic aneurysm.

References