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CASE REPORT

Tuberculous Aortic Pseudoaneurysm: An Unexpected Encounter with an Old Acquaintance

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Introduction: *Mycobacterium tuberculosis* (MT) is a rare cause of mycotic aneurysms. Diagnosis and management of tuberculous arterial infection is challenging. A case of a patient diagnosed and successfully treated for an aortic pseudoaneurysm caused by MT infection is reported.

Report: An 83 year old man was admitted with recurrent back pain over five months associated with constitutional symptoms. Computed tomography angiography (CTA) revealed a psoas collection associated with terminal aorta and proximal left common iliac artery posterolateral wall ulceration. Percutaneous drainage was performed and both the acid fast bacillus test and the molecular test for MT DNA were positive. The patient started on anti-tuberculous treatment, showing an excellent response. Three month CTA revealed arterial ulceration stability. However, the six month CTA revealed evolution to an asymptomatic 40 mm pseudoaneurysm. He was submitted to open repair with an aorto-bi-iliac interposition silver acetate/triclosan collagen coated polyester graft. The post-operative course was uneventful.

Discussion: Increased awareness and pursuit of an histological and microbiological diagnosis along with close surveillance allow anticipation of complications that can develop without any warning symptoms, as reported in this case. Agent identification and a combination of prolonged anti-tuberculous drug therapy with extensive excision of the infected field along with aortic reconstruction contributed to a good outcome.

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INTRODUCTION

Mycotic aneurysms are a diagnostic and therapeutic challenge. Considering their rarity, strong evidence regarding their management is lacking.[1] Tuberculosis (TB) infection rarely involves the arterial tree, with few reports in the literature, with the aorta (thoracic and abdominal) being the most commonly affected arterial segment.[2,3] Direct invasion or spread from lymph nodes, abscess, or bone TB is the most common mode of arterial involvement and the vessel wall damage typically induces a weakening of it, which degenerates into pseudoaneurysm formation.[2,3] Signs and symptoms of tuberculous arterial involvement are insidious and the diagnosis requires a high index of suspicion. Despite the use of modern anti-tuberculous therapy, disastrous complications like pseudoaneurysm formation or frank rupture still occur, highlighting the need for early diagnosis, close surveillance, and expeditious treatment when such complications occur.[4] The case of an immunocompetent octogenarian patient who presented with miliary tuberculosis and a mycotic aortic pseudoaneurysm is presented.

CASE REPORT

An 83 year old man was admitted with a five month history of recurrent back pain, with no history of trauma. Episodic low grade fever, night sweats, loss of appetite, along with significant weight loss (about 10 kg over the past four months) were also reported. On examination, he appeared cachectic and unwell, presenting left flank tenderness without rebound. Apart from slight elevation of C reactive protein (9.4 mg/dL), no remarkable findings resulted from the blood chemistry. Ultrasound revealed a left retroperitoneal collection, better defined on computed tomography angiography (CTA) as a multiloculated psoas muscle collection with perivascular fistulisation to the terminal aorta and proximal left common iliac artery (LCIA) posterolateral wall. At this arterial level six mm diameter, 6 mm depth wall ulceration was visible (Fig. 1). Chest Xray and computed tomography (CT) scan were performed, revealing bilateral reticulonodular infiltrates in both lung fields. Sputum and blood cultures were negative as were immunological tests (anti-nuclear antibodies, anti-neutrophil cytoplasmic antibodies, rheumatoid factor, C3, C4 and immunoglobulins). Magnetic resonance imaging ruled out spondylodiscitis but

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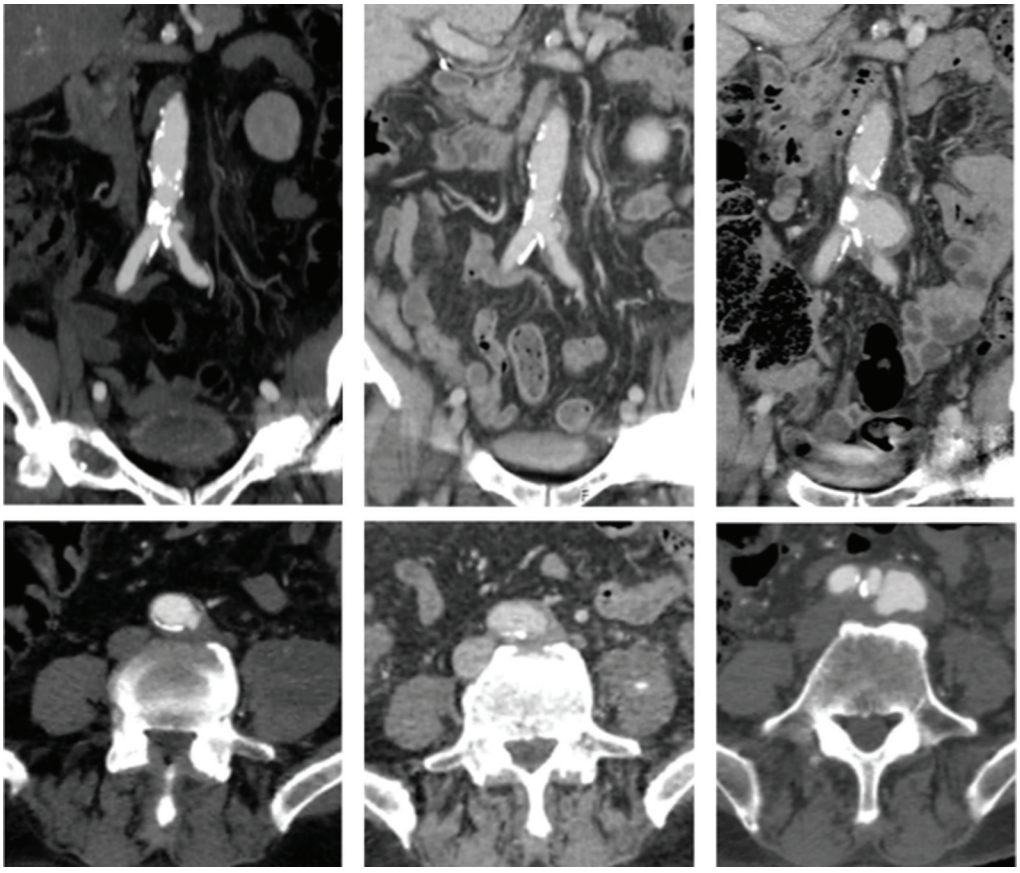

Figure 1. Computed tomography angiography (CTA) evolution (axial and coronal views). Admission CTA: six mm diameter, 6 mm depth terminal aorta and proximal left common iliac artery posterolateral wall ulceration. Three month CTA demonstrated arterial wall defect stability. Asymptomatic evolution to a 40 mm pseudoaneurysm detected on six month CTA. Magnetic resonance imaging documenting apparent arterial wall ulcer (dotted circle) related to psoas collection (*).

documented the arterial wall involvement (Fig. 2). Percutaneous needle biopsy of the psoas collection was performed, under CT guidance. About 30 mL of a cheese like liquid was aspirated and sent to analysis. Both the acid fast bacillus test and the molecular test for *Mycobacterium tuberculosis* DNA were positive. The patient immediately started antituberculous treatment (isoniazid, rifampicin, pyrazinamide, and ethambutol), and was discharged home after clinical improvement. He was compliant with medication and there were no side effects. During treatment he showed an impressive improvement of his clinical condition: fever remitted completely, he regained his body weight, and became functional and fully oriented. CTA follow up at three months showed complete resolution of the psoas collection with no changes in the known arterial ulceration. However, the six month CTA (Fig. 1) revealed ulceration evolution to a 40 mm pseudoaneurysm. The patient did not report any change in symptoms during this period. Despite his age, the patient was considered fit for surgery, and open repair was selected considering the infectious cause. Intra-operatively the pseudoaneurysm was excised revealing complete disruption of the terminal aorta and proximal left common iliac posterolateral wall (Fig. 3A). Surrounding tissues (caseous necrotic tissues and pus) were thoroughly debrided. An aorto-bi-iliac interposition graft was performed (16 × 8 mm silver acetate/triclosan collagen coated polyester graft Intergard Synergy™ Getinge®) (Fig. 3B). The histopathological report of the aneurysm wall was caseating

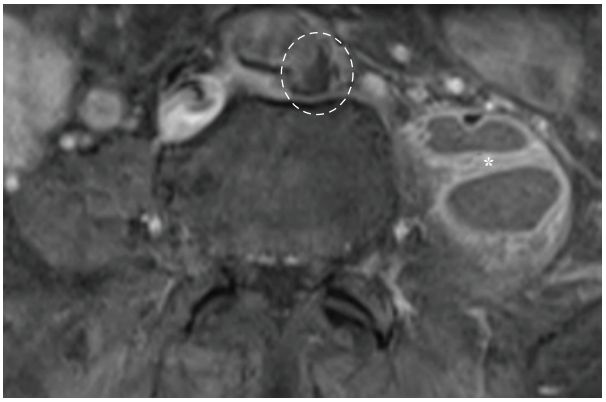

Figure 2. Magnetic resonance imaging documenting apparent arterial wall ulcer (dotted circle) in relation to psoas collection (*).

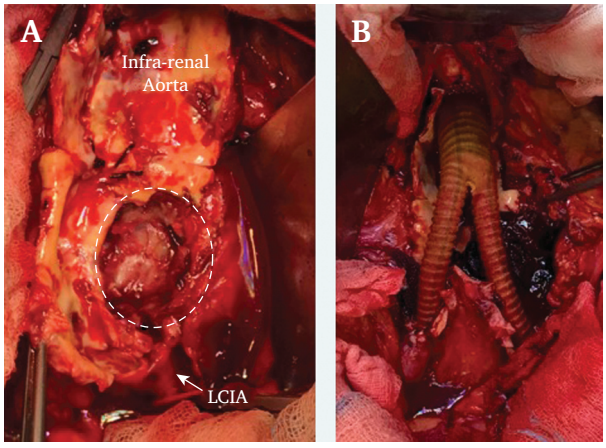

Figure 3. Intra-operative findings and arterial reconstruction. After pseudoaneurysm excision, complete disruption of the terminal aorta and proximal left common iliac artery (LCIA) posterolateral wall was visible (A, dotted line circle). Aorto-bi-iliac interposition graft, after extensive infected tissues debridement (B).

granulomatous inflammation consistent with tuberculosis infection. The *M. tuberculosis* DNA test was also positive in the tissues (aortic wall and adjacent soft tissues) that were collected intra-operatively. The post-operative course was uneventful. After consulting with the Infectious Diseases Department, it was decided to maintain oral anti-TB treatment for one year after intervention. After nine months the patient remained asymptomatic and positron emission tomography CT showed no pseudoaneurysm recurrence or signs suggestive of graft re-infection.

DISCUSSION

Mycotic aneurysms are associated with high morbidity and mortality.[1] Among the most common organisms causing mycotic aneurysms are the *Staphylococcus*, *Salmonella*, and *Streptococcus* species, with *M. tuberculosis* being a rare aetiology.[1,3] Despite the small number of published cases to date, it is possible that the incidence of TB pseudoaneurysms may increase, due to the greater number of immunodeficient patients and the growing number of drug resistant atypical *Mycobacterium* species. The pathogenesis of tuberculous arterial involvement may include two main mechanisms: haematogenous spread during bacteraemia or direct extension to the vessel from a contiguous tuberculous focus (lymph nodes or abscess). The most common is the latter which explains tuberculosis predilection for the thoracic aorta and innominate artery, as a result of the direct spread from involved adjacent pulmonary segments.[4] In this case, a direct extension was also suspected: primary focus within the left psoas muscle with abscess formation and subsequent involvement of the adjacent arterial wall. Tuberculous infection of the vascular wall results in arterial thinning and fragility, which in turn can lead to pseudoaneurysm development or free rupture.[5] At presentation, the patient showed minor ulceration of the terminal aorta and proximal LCIA posterolateral wall with no formal indication for intervention. However, despite the prompt initiation of medical treatment with a spectacular improvement in the patient's condition, a life threatening asymptomatic pseudoaneurysm was detected on the CTA six months later. As reported, the signs and symptoms of TB pseudoaneurysm are insidious, poor, or minimal, requiring close vigilance to detect ominous changes. Regarding treatment, open repair combined with targeted medical therapy remains the first line treatment.[1] However, the active phase of the disease often makes it impossible to perform open intervention due to high anaesthetic risk.[6] In these cases, endovascular therapy may serve as a bridge to a more definitive treatment or can be the definitive treatment by itself with some studies reporting comparable outcomes to open surgery in these frail patients.[6,7] Open repair was the selected option in this case considering the patient was fit for it and because it would allow not only repair of the vessel but also extensive debridement of the "contaminated" surrounding tissue. Type of repair (*in situ* or extra-anatomical) and conduit choice (autologous vein, cryopreserved arteries, bovine pericardium or prosthetic graft) is also debated.[1] *In situ* reconstruction with an antibiotic impregnated graft was selected, considering the longer and more complex intervention that a reconstruction with autologous conduit or an extra-anatomical bypass would require. Pre- and post-operative anti-tuberculous chemotherapy must be maintained to minimise the probability of infection in the implanted graft.[4] Despite the patient's advanced age and the presence of miliary tuberculosis, his excellent response to treatment confirms that even elderly patients may benefit from "aggressive" management and treatment in the setting of a mycotic aneurysm.

Conclusion

Increased awareness and chasing a histological and microbiological diagnosis, and targeted medical treatment combined with close surveillance allowed timely surgical management and led to this excellent outcome.

FUNDING

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CONFLICT OF INTEREST

None.

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