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Multiple Arterial Aneurysms Probably of Tuberculous Origin in HIV–Tuberculosis Coinfection: A Fatal Case Report

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Abstract **Case Report**

Tuberculous arterial aneurysms are a rare but severe complication of tuberculosis. We report the case of a 37-year-old woman with no known medical history who was admitted with deterioration of her general condition. Laboratory investigations revealed severe anaemia with a haemoglobin level of 6.6 g/dL, leukopenia with lymphopenia, markedly elevated lactate dehydrogenase levels, and impaired renal function. Cervicothoracoabdominopelvic computed tomography angiography demonstrated multiple aneurysmal lesions involving the right common carotid artery, the descending thoracic aorta, and the abdominal aorta, associated with multiple necrotic thoracoabdominal lymph nodes. Infectious work-up revealed HIV infection with a high viral load and active tuberculosis, documented by positive acid-fast bacilli testing and GeneXpert MTB/RIF. Antituberculous and antiretroviral therapies were initiated, together with transfusion of three units of packed red blood cells. The initial strategy for the aneurysms was to achieve preliminary control of the infectious process before definitive vascular treatment. The following day, however, the patient developed severe shock, probably haemorrhagic, strongly suggesting aneurysmal rupture. The clinical course was rapidly fatal. This case highlights the severity of multiple infected aneurysms and the difficulty of determining the optimal timing of vascular repair.

Keywords: Tuberculosis; HIV; infected aneurysm; aortic aneurysm; multiple aneurysms; rupture.

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INTRODUCTION

Vascular involvement in tuberculosis is rare. It may present as aortitis, pseudoaneurysm, or infected aneurysm and carries a substantial risk of progression and rupture. Mycobacterium tuberculosis most commonly reaches the arterial wall by direct extension from an adjacent tuberculous focus, particularly lymphadenopathy, although lymphatic or haematogenous dissemination and spread through the vasa vasorum have also been described [1,2].

Multifocal forms are exceptional [3]. In addition, HIV infection may be associated with aneurysmal vascular manifestations and favours active and disseminated forms of tuberculosis [4]. We report the case of a young woman with multiple, anatomically distinct arterial aneurysms in the setting of active HIV–tuberculosis coinfection and a rapidly fatal clinical course.

CASE PRESENTATION

A 37-year-old woman from Côte d'Ivoire, residing in Morocco, was admitted to the emergency department with deterioration of her general condition. According to her history, she had no known medical or surgical comorbidities and was not receiving chronic treatment.

On admission, the patient was conscious. Blood pressure was 140/80 mmHg, heart rate 110 beats/min, and respiratory rate 22 breaths/min. General examination revealed marked mucocutaneous pallor with pale conjunctivae.

Vascular examination showed palpable and symmetrical peripheral pulses, without trophic changes or clinical signs of peripheral ischaemia. Cardiovascular and pulmonary examinations were unremarkable. Abdominal examination revealed no significant abnormality. Peripheral lymph-node stations were clinically free, with no palpable lymphadenopathy.

Initial laboratory investigations revealed severe anaemia with a haemoglobin level of 6.6 g/dL. The white blood cell count was 3,540/mm³ with lymphopenia, while the platelet count was normal. Blood urea was 0.35 g/L and serum creatinine 19.05 mg/L, with an estimated glomerular filtration rate of 34.74 mL/min/1.73 m². Serum electrolytes showed no significant abnormality. Total bilirubin was 3.33 mg/L and direct bilirubin 1.16 mg/L. Lactate dehydrogenase was markedly elevated at 1,399 IU/L. Inflammatory markers showed a C-reactive protein level of 66 mg/L and an erythrocyte sedimentation rate of 22 mm in the first hour.

As part of the aetiological work-up for deterioration of the general condition and severe anaemia, cervicothoracoabdominopelvic computed tomography angiography was performed. It demonstrated a bilobed, partially calcified aneurysm of the proximal right common carotid artery, located 17 mm from its origin, measuring 19 × 15 mm and remaining patent.

At the distal descending thoracic aorta, two partially calcified aneurysmal lesions were identified. The first measured 43 × 30 mm, had a 20-mm neck, and was completely thrombosed. The second measured 26 × 17 mm, had a 10-mm left posterolateral neck, and was partially thrombosed. An abdominal aortic aneurysm located distal to the origin of the superior mesenteric artery measured 55 × 48 mm. Imaging also demonstrated multiple thoracoabdominal lymph nodes, several of which showed necrotic changes.

Serological testing for hepatitis B and C was negative. In contrast, HIV screening using a fourth-generation combined assay detecting p24 antigen and anti-HIV-1/HIV-2 antibodies was positive and subsequently confirmed. Plasma HIV RNA quantification showed a high viral load. The CD4+ T-lymphocyte count was not measured.

Given the newly diagnosed HIV infection and the presence of multiple necrotic thoracoabdominal lymph nodes, active tuberculosis was suspected. Sputum testing for acid-fast bacilli was positive. QuantiFERON-TB was also positive. GeneXpert MTB/RIF testing of a respiratory specimen detected Mycobacterium tuberculosis, confirming active tuberculosis.

Lymph-node biopsy was not performed because the lymphadenopathy was predominantly deep and thoracoabdominal, making access invasive and technically challenging. Correlation of the clinical, microbiological, and radiological findings strongly suggested an infectious, probably tuberculous, origin of the aneurysmal lesions, although no direct histological evidence of arterial-wall involvement was available.

Therapeutically, antituberculous treatment and antiretroviral therapy were initiated. Because of the severe anaemia, the patient also received three units of packed red blood cells.

Regarding the aneurysmal lesions, the initial decision was to defer definitive vascular treatment in order to achieve better control of the infectious process before considering open surgical or endovascular intervention.

However, the following day the patient suddenly developed severe shock, probably haemorrhagic in nature. Given the presence of multiple aortic aneurysms, aneurysmal rupture was strongly suspected. The extremely rapid haemodynamic deterioration precluded radiological confirmation. Despite resuscitative measures, the clinical course was rapidly fatal, and the patient died shortly after the onset of shock.

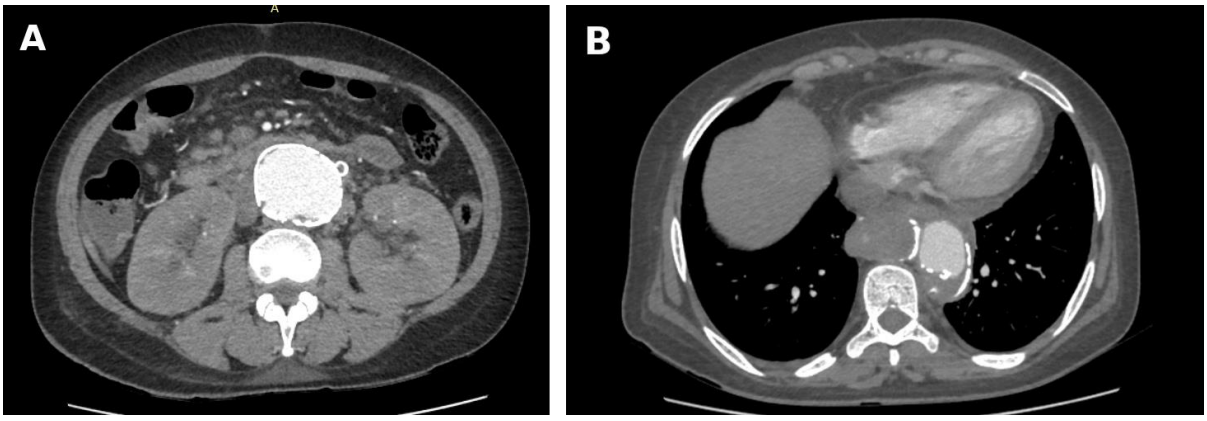


Figure 1: Axial CT angiography images. A: Abdominal aortic aneurysm with mural calcifications. B: Distal descending thoracic aortic aneurysmal lesion with mural calcifications and a thrombotic component

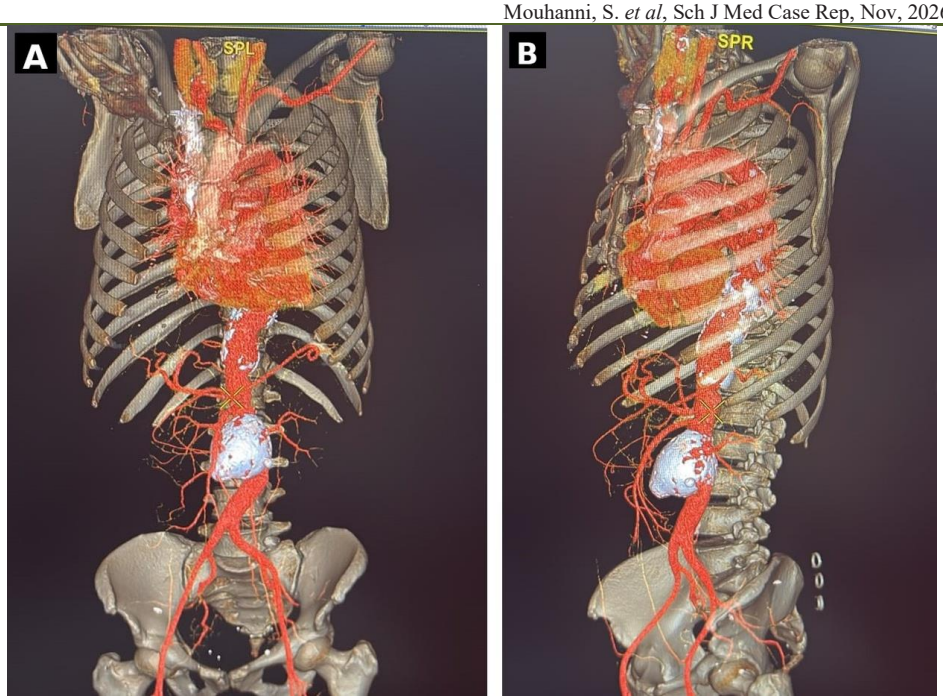


Figure 2: Three-dimensional volume-rendered CT angiography reconstructions. A: Anterior view showing the thoracic and abdominal aneurysmal lesions. B: Lateral view demonstrating their anatomical relationships with the aorta and its major branches

DISCUSSION

Tuberculous arterial aneurysms are an exceptionally rare but particularly severe complication of tuberculosis, mainly because of their potential for rapid enlargement and rupture. Vascular involvement may result from direct extension of an adjacent tuberculous focus, particularly lymph-node, vertebral, or pleuropulmonary disease, but may also occur through lymphatic or haematogenous dissemination or involvement of the vasa vasorum. Inflammatory destruction of the media and adventitia weakens the arterial wall and may lead to a true aneurysm or, more commonly, a pseudoaneurysm [1,2]. In our patient, the association of multiple necrotic thoracoabdominal lymph nodes with several aneurysmal lesions made contiguous infectious involvement of the arterial wall plausible.

In Morocco, tuberculosis remains an important public health problem. World Health Organization data estimated an incidence of approximately 97 cases per 100,000 population in 2022 [6]. The Moroccan National Strategic Plan for Tuberculosis Prevention and Control 2024–2030 further emphasises the heterogeneous distribution of disease, with a particularly high burden in densely populated urban areas, and sets targets of a 35% reduction in incidence and a 60% reduction in tuberculosis-related mortality by 2030 [7]. In this epidemiological context, tuberculous involvement should remain in the differential diagnosis of atypical, saccular, rapidly progressive, or multiple arterial lesions in young patients, particularly when associated with necrotic lymphadenopathy or documented tuberculosis.

For drug-susceptible tuberculosis in adults, Moroccan national recommendations for pulmonary tuberculosis and most extrapulmonary forms are based on a 2-month intensive phase with rifampicin, isoniazid, pyrazinamide, and ethambutol, followed by 4 months of rifampicin and isoniazid (2RHZE/4RH) [8]. Vascular tuberculosis, however, represents a specific situation for which no standardised treatment regimen has been established by comparative trials. Published series generally favour a combination of antituberculous therapy with exclusion or resection of the vascular lesion, with the duration of antimycobacterial treatment often prolonged and individualised according to the extent of infection, clinical response, presence of prosthetic material, and microbiological findings [1,10]. Medical therapy alone therefore appears insufficient once an infected aneurysm or pseudoaneurysm has formed and is exposed to the risk of rupture.

The multifocal distribution of the vascular lesions is one of the main distinctive features of this case. The patient simultaneously had an aneurysm of the right common carotid artery, two aneurysms of the descending thoracic aorta, and an abdominal aortic aneurysm. Multiple tuberculous aneurysms are exceptionally reported in the literature [3]. The young age of our patient, 37 years, is also unusual for conventional degenerative aneurysmal disease and warrants consideration of alternative aetiologies, including vasculitis, connective-tissue disorders, infection, and HIV-associated vasculopathy.

HIV infection itself has a distinct vascular spectrum. HIV-associated large-vessel vasculopathy

may present with aneurysms or pseudoaneurysms, occlusive disease, thrombosis, dissection, or accelerated atherosclerosis. Aneurysmal lesions have been described in relatively young patients, frequently in multifocal and unusual territories, including the carotid, aortic, femoral, and popliteal arteries [4,9]. Several pathophysiological mechanisms have been proposed, including chronic inflammation and immune activation, endothelial dysfunction, intimal proliferation, leukocytoclastic involvement centred on the vasa vasorum, and opportunistic infections. Tuberculosis may therefore act as an additional factor of arterial-wall injury in patients with HIV, sometimes making it difficult to distinguish primary HIV-associated vasculopathy from secondary infectious arteritis [4,9].

In our patient, the HIV viral load was high, whereas the CD4+ T-lymphocyte count was unavailable. This limits assessment of the degree of immunosuppression and its potential contribution to the vascular lesions. Management of HIV–tuberculosis coinfection should combine control of tuberculosis with initiation of antiretroviral therapy, while accounting for drug interactions, particularly those involving rifampicin, and ensuring close clinical monitoring [12]. When arterial lesions are present, virological control and treatment of opportunistic infections are essential adjuncts but do not replace mechanical treatment of an aneurysm at risk of rupture.

Nevertheless, the tuberculous origin of the aneurysms cannot be considered definitively proven in our case. No arterial-wall specimen or lymph-node biopsy could be obtained. The most rigorous diagnosis is therefore that of multiple aneurysms probably of tuberculous origin in the setting of HIV–tuberculosis coinfection, with a possible contribution from HIV-associated vasculopathy. When feasible, intraoperative sampling is particularly useful for histological examination, mycobacterial culture, and molecular testing; however, it should not delay urgent treatment in the setting of rupture or haemodynamic instability.

From a therapeutic standpoint, management should be multidisciplinary and involve infectious-disease specialists, vascular surgeons, interventional radiologists, and critical-care physicians. In infected aneurysms, open surgery allows resection of infected arterial tissue, extensive debridement, and vascular reconstruction, either in situ or extra-anatomically according to lesion location and the degree of contamination. It also provides an opportunity to obtain microbiological and histological specimens. Its main disadvantage is its invasiveness, particularly for thoracic or thoracoabdominal lesions and in frail, malnourished, or immunocompromised patients [1,11].

Endovascular treatment, by contrast, allows rapid exclusion of the lesion without laparotomy or thoracotomy and may be particularly useful in the setting of rupture or impending rupture, in patients at high operative risk, or when multifocal disease makes extensive open surgery difficult. Its principal limitation in an infectious setting is the absence of debridement and the implantation of prosthetic material within or adjacent to a potentially contaminated focus, with a risk of persistent or recurrent infection. A systematic review of 122 cases of tuberculous aortic aneurysm reported perioperative mortality rates of 10.0% after open repair and 4.8% after endovascular repair, whereas 2-year mortality rates were 11.3% and 16.7%, respectively. These data, derived predominantly from case reports and subject to major selection bias, do not establish superiority of one strategy over the other but emphasise the importance of early antituberculous therapy when an endovascular approach is selected [10]. A recent international consensus on infective native aortic aneurysms similarly supports an individualised strategy combining antimicrobial treatment, anatomical repair when indicated, and prolonged surveillance [11].

In HIV-associated aneurysmal vasculopathy without evidence of active arterial-wall infection, no universally accepted disease-specific surgical recommendations exist; conventional vascular-surgical principles remain applicable, with the choice between open and endovascular treatment based on anatomy, lesion location, symptoms, rupture risk, and the patient's overall condition [4]. Conversely, when an opportunistic infection such as tuberculosis is concomitantly present, it is prudent to consider the lesion potentially infectious until proven otherwise. In multifocal disease, a staged or hybrid strategy may be discussed, prioritising the lesion considered most threatening and adapting subsequent interventions according to the response to anti-infective therapy.

The optimal timing of repair remains one of the most challenging issues. In a stable patient, preliminary antituberculous therapy may reduce the infectious burden before planned reconstruction. Conversely, a symptomatic, rapidly progressive, fissured, or ruptured lesion requires urgent repair, and the objective of infection control should not lead to delay of vascular exclusion when the immediate risk of rupture is considered high [1,10,11]. In our patient, the initial decision was to achieve better control of the infectious process before definitive vascular treatment. The occurrence, on the following day, of probable haemorrhagic shock with suspected aneurysmal rupture illustrates the narrow therapeutic window in these aggressive forms and the difficulty of balancing infectious risk, operative risk, and rupture risk.

Finally, our report has several limitations: the absence of direct histological or microbiological evidence of tuberculous involvement of the arterial wall, the absence of a CD4 count, the inability to distinguish the respective contributions of tuberculosis and HIV-associated vasculopathy, and the lack of radiological or

post-mortem confirmation of the terminal rupture. Despite these limitations, the association of documented active tuberculosis, necrotic lymphadenopathy, HIV infection, and multiple arterial aneurysms in a young patient represents an exceptionally unusual presentation highly suggestive of complex infectious vascular involvement.

CONCLUSION

Multiple arterial aneurysms probably of tuberculous origin represent an exceptional and severe complication of tuberculosis. In a young patient with active tuberculosis, particularly in the setting of HIV infection, the presence of atypical or multiple aneurysmal lesions should raise suspicion of an infectious origin and/or HIV-associated vasculopathy. Management should combine antituberculous therapy, control of HIV infection, and early vascular assessment. The choice between open surgical and endovascular treatment depends on lesion location, the infectious context, the patient's overall condition, and, above all, the risk of rupture. Our case further highlights the difficulty of determining the optimal timing of intervention and the potentially fulminant course of this condition.

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