Cureus
Part of SPRINGER NATURE

Open Access Case Report

Disseminated Tuberculosis Complicated by Infectious Aortitis and Pseudoaneurysm in Suspected Takayasu Arteritis

Review began 09/30/2026
Review ended 11/01/2026
Published 11/04/2026

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DOI: 10.7759/cureus.119127

Ana Margarida Morgado [1], António Padilha [2], João Marques Cardoso [1], Joana Lopo [1], José Ignacio Moreno [1]

1. Internal Medicine, Unidade Local de Saúde do Algarve, Faro, PRT 2. Intensive Care, Unidade Local de Saúde do Algarve, Faro, PRT

Corresponding author: Ana Margarida Morgado, amargarida.marcelo@gmail.com

Abstract

Disseminated tuberculosis with aortic involvement is an uncommon but potentially fatal complication of *Mycobacterium tuberculosis* infection. We report the case of a 25-year-old man from Nepal with a history of renovascular hypertension secondary to renal artery stenosis diagnosed during adolescence, who presented with persistent fever, cough, anorexia, asthenia, and abdominal pain. Microbiologic evaluation confirmed smear-positive pulmonary tuberculosis without rifampicin resistance, and standard first-line antituberculous therapy was initiated. Thoracoabdominopelvic computed tomography demonstrated diffuse reticulonodular pulmonary infiltrates with cavitary lesions, calcified mediastinal lymphadenopathy, hepatosplenomegaly, and extensive aortic calcification. A large periaortic collection immediately inferior to the celiac trunk origin was also identified. Further vascular imaging demonstrated disruption of the vascular wall, critical celiac trunk stenosis, superior mesenteric artery occlusion, and bilateral renal artery occlusion. The combination of early-onset renovascular hypertension and extensive multivessel arterial disease raised suspicion of underlying Takayasu arteritis, while the periaortic lesion was considered consistent with infectious aortitis and pseudoaneurysm formation in the setting of active tuberculosis. The patient declined vascular surgical intervention. He subsequently developed sudden abdominal pain, massive gastrointestinal hemorrhage, severe metabolic acidosis, and refractory shock, followed by cardiac arrest. Autopsy demonstrated disseminated necrotizing granulomatous disease involving the lungs, kidneys, and aortic wall. Histopathologic examination of the aorta revealed transmural arteritis with neutrophilic infiltration, microabscesses, and tissue necrosis, consistent with infectious involvement of the vascular wall and pseudoaneurysm formation. This case highlights the potentially fatal vascular complications of disseminated tuberculosis and the diagnostic complexity of concomitant infectious aortic disease and suspected large-vessel vasculitis. Early recognition of aortic involvement and multidisciplinary assessment are essential in patients presenting with tuberculosis and extensive vascular abnormalities.

Categories: Internal Medicine, Cardiac/Thoracic/Vascular Surgery, Infectious Disease
Keywords: aortic pseudoaneurysm, infectious aortitis, large-vessel vasculitis, takayasu arteritis, tuberculosis

Introduction

Takayasu arteritis is a chronic granulomatous large-vessel vasculitis predominantly affecting the aorta and its major branches. Vascular inflammation can result in stenosis, occlusion, aneurysmal disease, and end-organ ischemia [1,2]. The disease primarily affects younger individuals and may present with renovascular hypertension, vascular bruits, limb claudication, or other manifestations of arterial insufficiency [1,2].

An association between tuberculosis and Takayasu arteritis has been described, although a causal relationship has not been established [3]. A systematic review identified reports of active or latent tuberculosis infection among patients with Takayasu arteritis but found insufficient evidence to determine whether tuberculosis contributes directly to its development [3]. Immunologic mechanisms involving host responses to mycobacterial antigens have also been proposed as potential contributors to the pathogenesis of Takayasu arteritis [4].

Tuberculous involvement of the aortic wall is rare but may result in destructive aortitis, mycotic aneurysm, or pseudoaneurysm formation, with potentially fatal complications [5]. The diagnosis may be particularly challenging when aortic infection occurs in the setting of pre-existing large-vessel disease, as inflammatory and infectious vascular processes may produce overlapping clinical and radiologic findings.

We present a fatal case of disseminated tuberculosis with destructive aortitis and aortic pseudoaneurysm in a young man with longstanding renovascular hypertension and extensive multivessel arterial disease suggestive of underlying Takayasu arteritis. The case highlights the diagnostic challenge of distinguishing chronic large-vessel vasculopathy from superimposed infectious aortic involvement and underscores the importance of recognizing this potentially complex vascular presentation.

How to cite this article
Morgado A, Padilha A, Marques Cardoso J, et al. (November 04, 2026) Disseminated Tuberculosis Complicated by Infectious Aortitis and Pseudoaneurysm in Suspected Takayasu Arteritis. Cureus 18(11): e119127. DOI 10.7759/cureus.119127

Case Presentation

A 25-year-old man, originally from Nepal and living in Portugal for approximately four years, where he worked in agriculture, presented to the emergency department with a subacute history of respiratory and constitutional symptoms. His relevant medical history included arterial hypertension secondary to renal artery stenosis, diagnosed at the age of 17, for which bilateral renal artery stents had been placed.

The patient reported a progressively worsening cough that was initially dry and subsequently became productive. This was associated with persistent fever, anorexia, asthenia, and mid-back pain that later became localized to the right hypochondrium.

A first thoracoabdominopelvic computed tomography angiography (CTA) demonstrated diffuse reticulonodular pulmonary infiltrates with apical cavitary lesions, calcified mediastinal lymphadenopathy, and bilateral pleural effusions, findings suggestive of pulmonary tuberculosis. Hepatosplenomegaly and cardiomegaly were also present. The aorta showed extensive calcification, giving the radiologic appearance of a "porcelain aorta." A large periaortic collection was also identified immediately inferior to the origin of the celiac trunk, raising suspicion of a pseudoaneurysm or mycotic aneurysm.

Microbiologic evaluation confirmed smear-positive pulmonary tuberculosis, with strongly positive sputum smears and no evidence of rifampicin resistance. Standard first-line antituberculous therapy with isoniazid, rifampicin, pyrazinamide, and ethambutol was initiated.

During the prolonged hospitalization, abdominal Doppler ultrasonography demonstrated critical stenosis of the celiac trunk, occlusion of the superior mesenteric artery, and bilateral renal artery occlusion. Transthoracic echocardiography revealed severe dilated cardiomyopathy, with an estimated left ventricular ejection fraction of approximately 23%. For re-evaluation, a second CTA further characterized the periaortic collection, measuring 80 × 35 mm in its largest axial dimensions, with peripheral enhancement and disruption of the intimal calcifications at this level. It also showed severe stenosis at the origins of the celiac trunk and superior mesenteric artery, stents at the origins of the renal arteries, and bilateral loculated pleural effusions (Figure *1*).



FIGURE 1: Computed tomography angiography (CTA) showing a periaortic lesion

Contrast-enhanced CTA demonstrating a large periaortic lesion immediately inferior to the origin of the celiac trunk, with focal disruption of the adjacent aortic wall, concerning for an infectious aortic pseudoaneurysm. CTA, computed tomography angiography.

The combination of renovascular hypertension diagnosed at a young age, extensive multivessel arterial stenoses, renal artery involvement, mesenteric arterial disease, celiac artery stenosis, and extensive aortic calcification raised the clinical suspicion of Takayasu arteritis. In parallel, the periaortic collection and disruption of the aortic wall in the setting of active tuberculosis were considered highly suggestive of infectious aortitis with pseudoaneurysm formation.

A month after admission, the patient was transferred to a tertiary center for vascular surgical assessment; however, he declined further diagnostic investigations and surgical intervention. During the subsequent hospitalization, he remained febrile and, nine days after the vascular surgical assessment, developed sudden-onset abdominal pain followed by massive gastrointestinal hemorrhage, severe metabolic acidosis,

and refractory shock. His condition deteriorated rapidly, culminating in cardiac arrest and death.

Autopsy demonstrated disseminated necrotizing granulomatous disease involving the lungs, kidneys, and aortic wall. Histopathologic examination of lung tissue showed epithelioid granulomas with central necrosis and multinucleated giant cells, and the aortic wall demonstrated transmural arteritis characterized by prominent neutrophilic infiltration, microabscess formation and tissue necrosis, findings compatible with infectious involvement of the aortic wall and pseudoaneurysm formation in the setting of disseminated tuberculosis and a pre-existing large-vessel vasculopathy clinically suggestive of Takayasu arteritis.

Discussion

This case illustrates the diagnostic and therapeutic complexity of disseminated tuberculosis in a young patient with extensive pre-existing large-vessel disease and a destructive aortic lesion. The coexistence of chronic arterial disease and active tuberculosis raised the possibility of two overlapping vascular processes: an underlying large-vessel vasculopathy and superimposed infectious aortitis. The subsequent development of a destructive periaortic lesion and the autopsy findings provided strong evidence of aortic involvement by the infectious process.

Takayasu arteritis is a chronic large-vessel vasculitis predominantly affecting the aorta and its major branches [1,2]. Vascular inflammation may lead to stenosis, occlusion, and aneurysmal disease, while renal artery involvement can result in renovascular hypertension [1,2]. In the present patient, renovascular hypertension had been diagnosed at 17 years of age and had required bilateral renal artery stenting. At presentation, critical stenosis of the celiac trunk, occlusion of the superior mesenteric artery, and bilateral renal artery occlusion were identified. The early onset, chronicity, and distribution of these abnormalities supported the presence of a pre-existing large-vessel vasculopathy and raised suspicion for Takayasu arteritis [1,2]. The 2022 American College of Rheumatology/European Alliance of Associations for Rheumatology classification criteria provide a framework for classification; however, they were not developed as diagnostic criteria and should not be used independently to establish the diagnosis of Takayasu arteritis [6].

The relationship between tuberculosis and Takayasu arteritis remains uncertain [3,4]. Tuberculosis has been reported in patients with Takayasu arteritis [3], and several immunologic mechanisms have been proposed, including abnormal immune responses to mycobacterial antigens and potential cross-reactivity involving the 65-kDa heat shock protein of *Mycobacterium tuberculosis* [3,4]. However, these observations do not establish a causal relationship between tuberculosis and Takayasu arteritis [3]. In the present case, the early-onset and extensive arterial disease favored a pre-existing large-vessel vasculopathy, whereas the destructive aortic lesion was temporally and pathologically associated with disseminated tuberculosis. Whether tuberculosis contributed to progression of the underlying vascular disease or directly involved a previously diseased aortic wall cannot be determined from the available evidence.

The aortic lesion was the most clinically consequential finding. Tuberculous involvement of the aorta is rare but can cause progressive destruction of the arterial wall, pseudoaneurysm formation, and rupture [5,7]. CTA demonstrated a large periaortic lesion immediately inferior to the origin of the celiac trunk, measuring 80 × 35 mm in its largest axial dimensions, with peripheral enhancement and focal disruption of the adjacent aortic wall. These findings were concerning for an infectious pseudoaneurysm [5,7]. At autopsy, transmural aortitis with neutrophilic infiltration, microabscess formation, and extensive tissue necrosis provided strong pathologic correlation with the radiologic findings. Histochemical staining for microorganisms (Ziehl-Neelsen, Grocott, and Gram), performed on lung, kidney, and aortic tissue at autopsy, yielded inconclusive results due to autolytic artefacts, precluding the direct microbiological demonstration of *M. tuberculosis* within the aortic wall. Nevertheless, the combination of microbiologically confirmed pulmonary tuberculosis, disseminated necrotizing granulomatous disease, and destructive aortic inflammation strongly supported the diagnosis of infectious aortitis in the context of disseminated tuberculosis.

The pathogenesis of aortic involvement in tuberculosis may involve direct extension from adjacent infected tissues or hematogenous dissemination to the arterial wall [5,7]. In this patient, involvement of multiple organs, including the lungs and kidneys, was consistent with disseminated disease and provided a plausible mechanism for vascular involvement. The coexistence of chronic large-vessel disease and active infection may have increased the vulnerability of the affected aortic segment to structural destruction, although this mechanism remains speculative in the present case. Infection may accelerate local necrosis and compromise vascular integrity, while infectious aortitis can produce imaging abnormalities that overlap with those of inflammatory large-vessel vasculitis, making differentiation particularly challenging [5,7].

Management of tuberculous aortic pseudoaneurysms generally involves antituberculous therapy together with definitive vascular treatment when feasible [7,8]. Both open surgical repair and endovascular approaches have been described, although the available evidence is largely derived from case reports, case series, and retrospective analyses [7,8]. A systematic review comparing endovascular and open surgical treatment reported the use of both approaches in selected patients and emphasized the need for careful follow-up because recurrent aneurysmal disease and late rupture have been reported [8]. In the present case,

standard first-line antituberculous therapy was initiated following microbiologic confirmation of pulmonary tuberculosis. However, the patient declined further vascular investigation and surgical intervention, leaving the structural aortic lesion untreated.

The patient's subsequent abrupt deterioration, marked by abdominal pain, massive gastrointestinal hemorrhage, severe metabolic acidosis, and refractory shock, occurred in the setting of a large aortic pseudoaneurysm and extensive mesenteric arterial disease. This clinical evolution was highly concerning for an acute vascular complication, however, the available findings did not establish the precise mechanism of the terminal gastrointestinal hemorrhage. Regardless of the precise terminal event, the rapid progression highlights the potential severity of aortic involvement in disseminated tuberculosis, particularly when superimposed on extensive pre-existing vascular disease [5,7].

This case has several limitations. Although *M. tuberculosis* was not directly demonstrated within the aortic wall, the diagnosis of tuberculosis was microbiologically confirmed, and autopsy demonstrated disseminated necrotizing granulomatous disease together with destructive transmural aortitis. Likewise, although histopathologic findings specific for Takayasu arteritis were not independently established, the patient's history of early-onset renovascular hypertension, prior bilateral renal artery interventions, and extensive multivessel arterial disease provided substantial clinical and radiologic evidence of a pre-existing large-vessel vasculopathy [1,2]. Finally, the patient's decision to decline vascular intervention limited definitive characterization and treatment of the aortic lesion. These limitations should be considered when interpreting the case, but do not diminish the clinical and pathologic evidence supporting the coexistence of chronic large-vessel disease and superimposed infectious aortic involvement in the setting of disseminated tuberculosis.

This case highlights the importance of considering infectious aortitis and pseudoaneurysm in young patients with active tuberculosis and new or unexplained periaortic abnormalities, particularly when imaging demonstrates aortic wall disruption [5,7]. At the same time, early-onset renovascular hypertension and widespread arterial stenoses should prompt consideration of underlying Takayasu arteritis [1,2,6]. Recognizing the coexistence of these conditions is particularly important because immunosuppressive therapy for active vasculitis may have significant implications in the presence of uncontrolled infection [3]. Multidisciplinary assessment involving infectious disease, vascular surgery, radiology, cardiology, and rheumatology may be appropriate when these conditions coexist, particularly when a destructive aortic lesion is identified [7,8].

Conclusions

Disseminated tuberculosis may rarely involve the aortic wall and lead to destructive aortitis and pseudoaneurysm formation. In patients with pre-existing or suspected large-vessel vasculopathy, this complication may result in rapidly fatal vascular instability. Early recognition of aortic involvement, prompt vascular assessment, appropriate antituberculous therapy, and multidisciplinary management are essential. This case also emphasizes the diagnostic complexity of distinguishing chronic inflammatory large-vessel disease from superimposed infectious aortitis and the importance of integrating clinical, imaging, microbiologic, and histopathologic findings when evaluating these patients.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Ana Margarida Morgado, Joana Lopo, José Ignacio Moreno

Acquisition, analysis, or interpretation of data: Ana Margarida Morgado, João Marques Cardoso, António Padilha

Drafting of the manuscript: Ana Margarida Morgado, João Marques Cardoso

Critical review of the manuscript for important intellectual content: Ana Margarida Morgado, Joana Lopo, José Ignacio Moreno, António Padilha

Supervision: José Ignacio Moreno

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have

declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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