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**Radiology**

# Mycotic Aneurysm of the Aorta and Necrotizing Pneumonitis Complicating a Shoulder Abscess: An Exceptional Association

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**Abstract** | **Case Report**

Mycotic aneurysm refers to an infection of the arterial wall caused by bacterial, fungal, or viral pathogens. Although uncommon, it represents a severe and often fatal complication of systemic infection or underlying atherosclerosis, posing significant diagnostic and therapeutic challenges. The pathogenesis involves hematogenous seeding during bacteremia, septic embolization, or direct invasion of the vessel wall, typically in patients with pre-existing atherosclerosis or immunosuppression. The aorta, intracranial arteries, and visceral or peripheral arteries are the most frequently affected sites. The natural history is characterized by progressive arterial dilation, pseudoaneurysm formation, and a high risk of rupture. Prompt recognition and management are crucial, combining targeted antimicrobial therapy with surgical or endovascular repair to optimize outcomes. We report an unusual case of a 63-year-old male with a shoulder abscess complicated by a mycotic aneurysm of the aorta and necrotizing pneumonitis, with multimodality imaging illustrating the temporal evolution of the disease.

**Keywords:** Aortic arch, Endovascular treatment, Mycotic aneurysm, Necrotizing pneumonia, Septic emboli, Staphylococcus aureus, Thoracic aorta, Vascular infection.



## INTRODUCTION

Mycotic aneurysm is an infection-driven dilatation of an arterial wall with the formation of saccular outpouching that is contiguous with the arterial wall [1]. The term, introduced by William Osler after observing fungus-like vegetations in endocarditis, is a historical misnomer—most infected aneurysms are bacterial (Staphylococcus and Streptococcus species are the most common causative pathogens) rather than fungal [1,2]. Pathogenesis typically involves hematogenous seeding during bacteremia, septic embolization (e.g., from endocarditis), direct inoculation after vascular injury, or contiguous spread from adjacent infection, culminating in transmural inflammation, necrosis, and pseudoaneurysm formation with a real risk of rupture [1]. The risk of a mycotic aneurysm is higher in immunocompromised individuals, including those with HIV infection, diabetes mellitus as well as those receiving high-dose glucocorticoids or chemotherapy [3]. Several factors are implicated in the pathogenesis of mycotic aneurysms, including bacteremia, vessel injury with bacterial inoculation, local bacterial spread, and septic emboli [1]. Vascular injury predisposes the intima to infection from bacterial seeding. This process is particularly common in older patients with atherosclerosis or preexisting aneurysms who experience sepsis or bacteremia. The most commonly affected blood vessels, in order of frequency, include the aorta due to the high prevalence of atherosclerosis and aneurysm formation in this structure, intracranial vasculature, and femoral and visceral arteries (eg, superior mesenteric and splenic). The natural history of these aneurysms is characterized by expansion, leading to pseudoaneurysm formation, where rupture is contained, followed by eventual rupture, hemorrhage, sepsis, and multiple organ failure [1]. Early recognition is challenging and depends on a synthesis of clinical, laboratory, and imaging findings. Cross-sectional imaging is central to diagnosis and follow-up. Contrast-enhanced CT typically shows a lobulated saccular outpouching contiguous with the arterial lumen, irregular wall enhancement, and perivascular inflammatory change (fat stranding, soft-tissue cuff, possible gas) [4]. Rapid interval enlargement supports an infectious etiology. Mycotic aneurysms are among the most challenging clinical problems for vascular surgeons due to their associated perioperative mortality [5]. Management requires prolonged, culture-directed antibiotics coupled with prompt exclusion of the infected segment. Open repair remains definitive but carries substantial perioperative risk. Endovascular

aortic repair offers a less invasive alternative—often lifesaving in unstable or high-risk patients—yet mandates vigilant surveillance and extended antibiotics given the persistent risk of late infection [6].

## Case Presentation

The patient was a 63-year-old male with multiple cardiovascular risk factors, including type 2 diabetes mellitus under treatment, dyslipidemia, and obliterating arteriopathy. In 2023, he underwent a surgical aorto-femoral bypass graft for management of peripheral arterial disease. He had no known history of chronic infections, tuberculosis, or immunosuppressive therapy. In January 2025, he was admitted to the emergency department following a motorcycle accident. He reported no loss of consciousness or major trauma. Clinical examination was unremarkable, and initial radiographic evaluation did not reveal any fractures or visceral injuries. The patient was discharged after a short observation period, with no acute complications identified. On 13 March 2025, he presented with acute dyspnea and fever; clinical assessment confirmed febrile illness with elevated white blood cell count and C-reactive protein. A contrast-enhanced chest CT revealed a left lower lobe consolidation consistent with pneumonia, two cavitary nodules in the upper and lower lobes consistent with necrotizing pneumonitis secondary to septic emboli, a saccular aneurysmal dilatation of the aortic arch diagnostic of a mycotic aneurysm, and an emphysematous soft-tissue collection in the right shoulder musculature compatible with an abscess **[Figure 1]**. Empiric broad-spectrum antibiotics were initiated for the shoulder abscess, and the patient was scheduled for endovascular treatment of the aortic aneurysm. On 21 March 2025, one week later, he experienced worsening dyspnea with persistent fever and a marked rise in inflammatory biomarkers, including leukocytosis and elevated C-reactive protein. A repeat chest CT demonstrated persistence of the right shoulder abscess despite antibiotic therapy, progression of the pulmonary lesions with extension of consolidation to additional lobes and enlargement of the cavitary nodules, along with a further increase in the size of the mycotic aneurysm **[Figure 2]**. On 23 March 2025, a right shoulder MRI revealed a soft-tissue collection containing air bubbles without evidence of underlying bony involvement **[Figure 3]**. Two subsequent chest CT scans, obtained on 30 March and 8 April 2025, showed further progression of the pulmonary lesions with multilobar necrotizing consolidation and enlarging cavitary nodules, partial regression of the shoulder abscess following surgical drainage and antibiotic therapy, and continued expansion of the mycotic aneurysm **[Figures 4–5]**. Culture of the drained collection, along with blood cultures, grew methicillin-sensitive Staphylococcus aureus, prompting a switch to targeted antibiotic therapy. Despite these measures, the patient's condition deteriorated with worsening sepsis, and he ultimately developed septic shock, leading to his death on 11 April 2025.

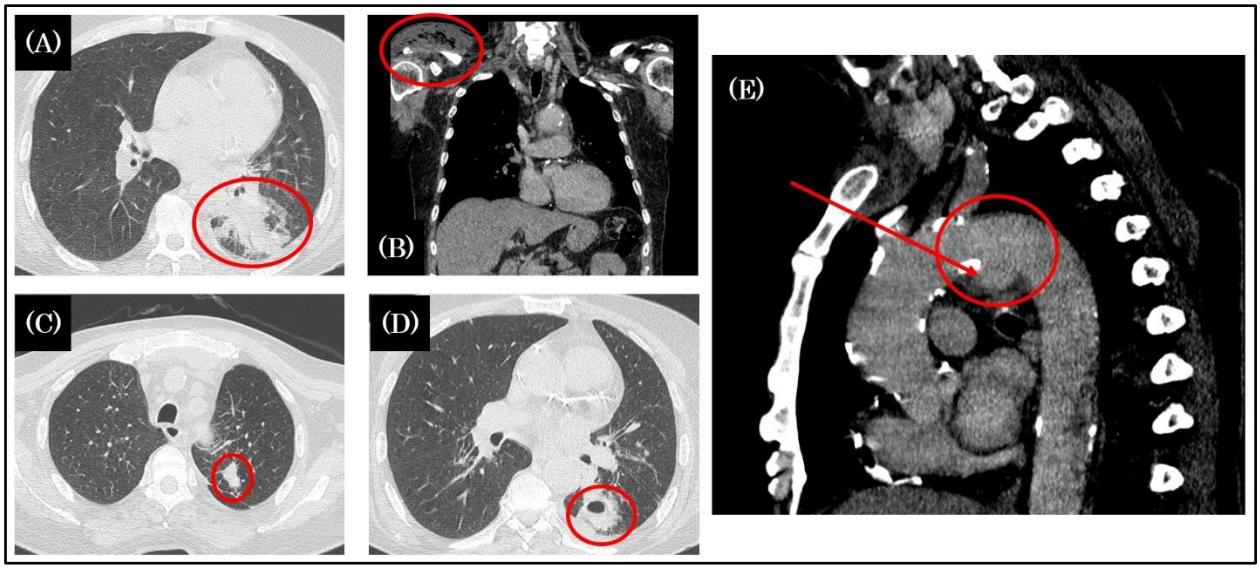


**Figure 1: Contrast-enhanced chest CT demonstrating multiple infectious complications. (A) Axial image showing left lower lobe consolidation with cavitation consistent with necrotizing pneumonitis (red circle). (B) Coronal image depicting an emphysematous soft-tissue collection in the right shoulder compatible with an abscess (red circle). (C–D) Axial lung window images revealing cavitary nodules in the upper and lower lobes, suggestive of septic emboli (red circles). (E) Sagittal reconstruction highlighting a saccular aneurysmal dilatation of the aortic arch, diagnostic of a mycotic aneurysm (arrow, red circle)**

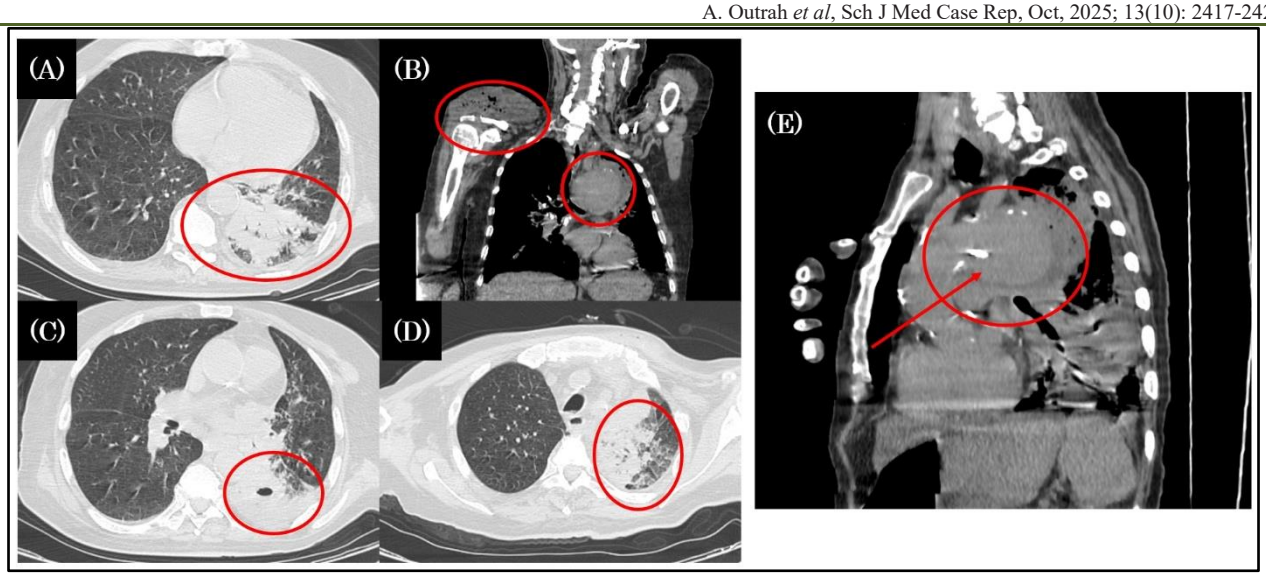


**Figure 2: Follow-up chest CT showing progression of infectious and vascular complications (21 March 2025). (A, C, D) Axial images demonstrate worsening necrotizing pneumonitis with multilobar consolidation and enlarging cavitary nodules (red circles). (B) Coronal reconstruction highlights persistence of the right shoulder abscess with gas formation and interval increase in the size of the aortic arch aneurysm (red circles)**

(E) Sagittal reconstruction confirms further expansion of the mycotic aneurysm of the aortic arch (arrow, red circle).

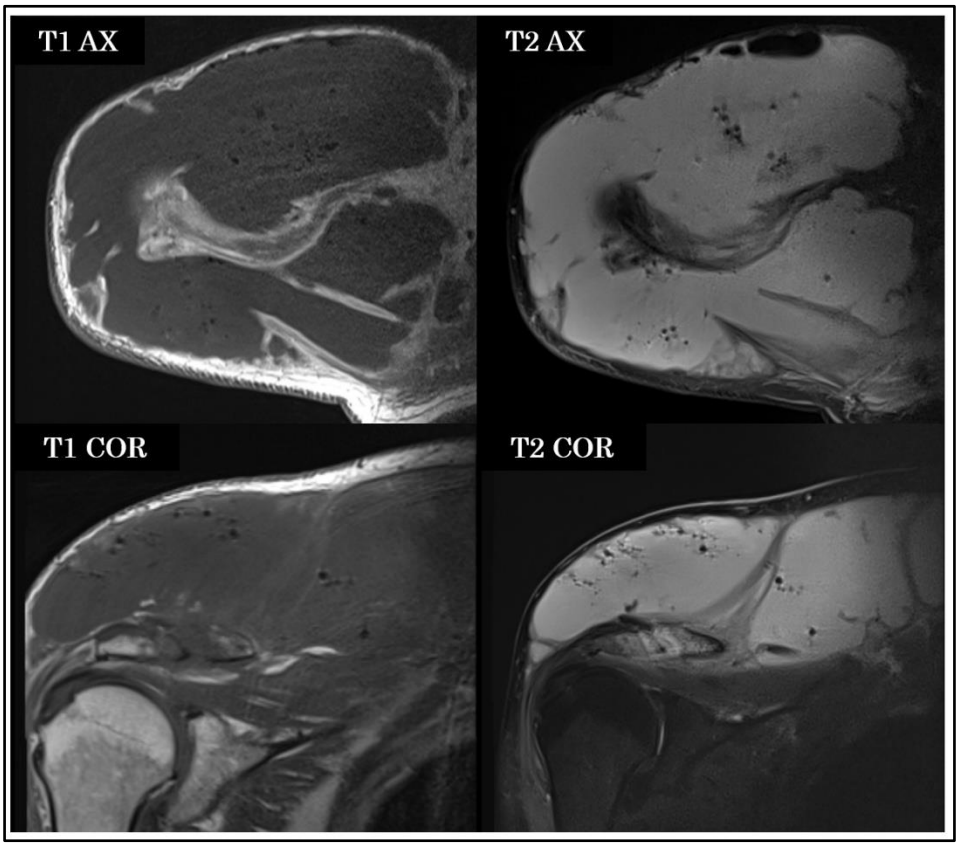


**Figure 3: Shoulder MRI confirming soft-tissue abscess (23 March 2025). Axial (T1-weighted and T2-weighted) and coronal (T1-weighted and T2-weighted) sequences show a large heterogeneous soft-tissue collection in the right shoulder containing multiple internal air bubbles, without evidence of underlying osseous involvement, consistent with an emphysematous abscess**

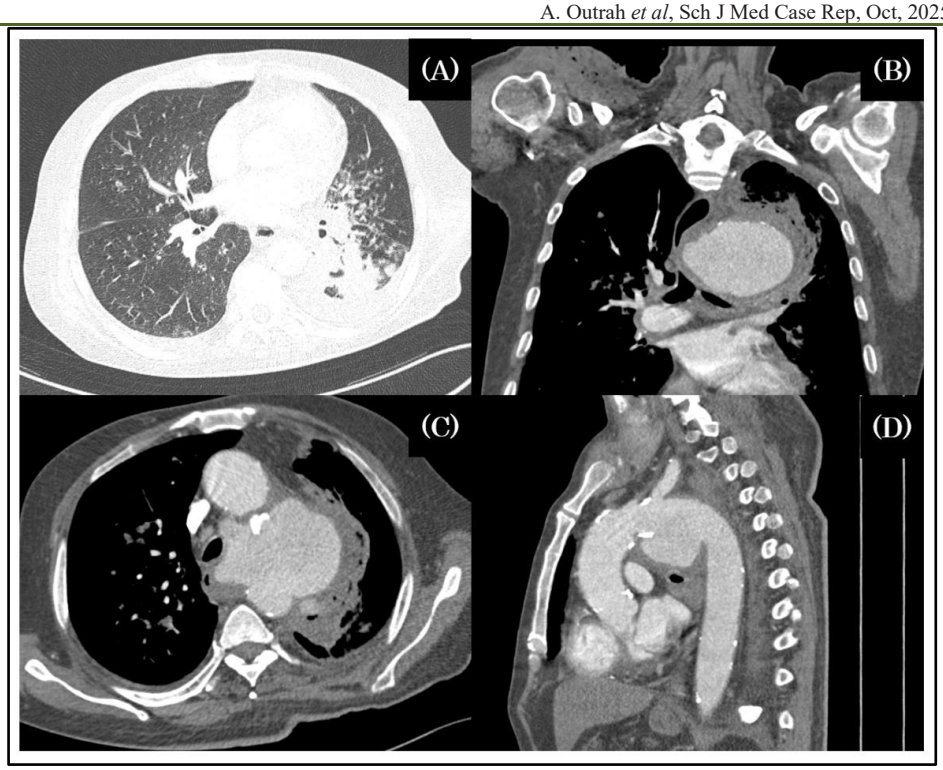


**Figure 4: Follow-up chest CT showing persistent infection and vascular progression (30 March 2025). (A) Axial lung window demonstrates worsening necrotizing pneumonitis with multilobar consolidation and cavitary changes. (B) Coronal reconstruction shows partial regression of the right shoulder abscess after surgical drainage. (C–D) Contrast-enhanced axial and sagittal reconstructions reveal interval enlargement of the mycotic aneurysm of the aortic arch**

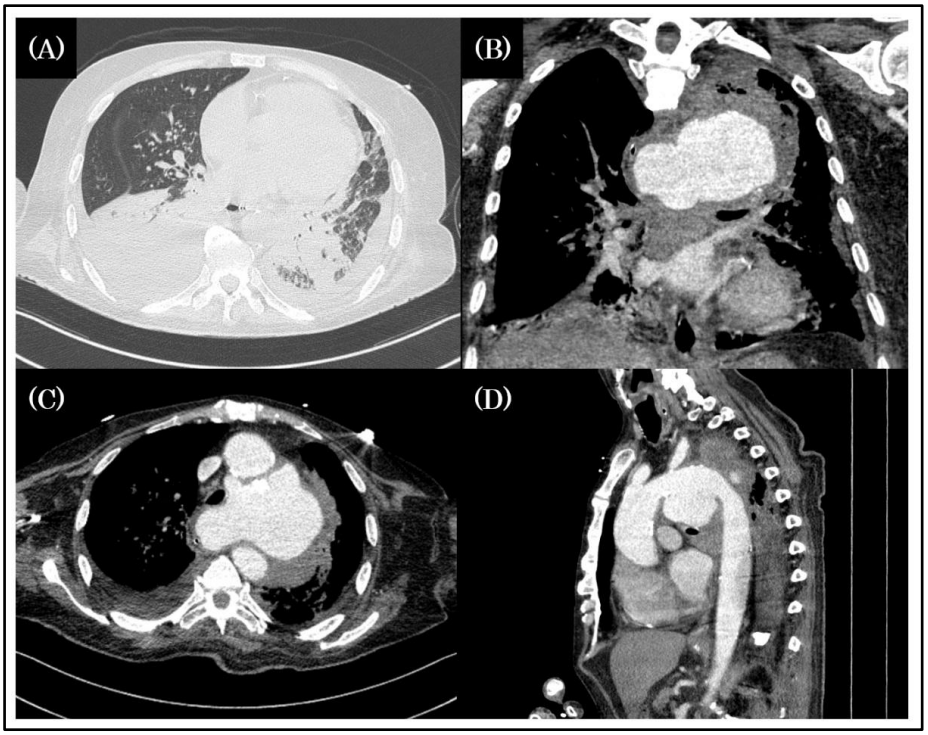


**Figure 5: Follow-up chest CT demonstrating further disease progression (8 April 2025). (A) Axial lung window shows persistent multilobar necrotizing consolidation with cavitary changes. (B) Coronal reconstruction demonstrates near-complete resolution of the right shoulder abscess after surgical drainage. (C–D) Contrast-enhanced axial and sagittal reconstructions reveal continued enlargement of the mycotic aneurysm of the aortic arch**

## DISCUSSION

Mycotic aneurysms represent a rare but highly lethal vascular complication, accounting for less than 2% of all aortic aneurysms, with thoracic involvement being particularly uncommon yet associated with higher morbidity and mortality [1]. Their pathogenesis involves seeding of the arterial wall by circulating pathogens, most often Staphylococcus aureus or Salmonella spp., through mechanisms such as bacteremia, septic emboli, direct inoculation during trauma or procedures, or contiguous spread from adjacent infection [5]. The risk is markedly increased in patients with underlying vascular disease such as atherosclerosis, diabetes mellitus, prior vascular surgery, or immunosuppression, where endothelial integrity is already compromised and bacterial adhesion is facilitated. In our patient, several predisposing factors converged: long-standing diabetes, severe peripheral arterial disease treated surgically with an aorto-femoral bypass, and recent bacteremia with methicillin-sensitive Staphylococcus aureus originating from a musculoskeletal abscess, creating the perfect substrate for infection and subsequent aneurysm formation [2]. Clinically, infected aneurysms are notoriously difficult to recognize early, as fever, malaise, and inflammatory marker elevation are common but nonspecific, and many remain silent until rupture or systemic sepsis occurs [7]. In this case, serial chest CT scans were pivotal, demonstrating the characteristic hallmarks of a mycotic aneurysm: a lobulated saccular dilatation of the aortic arch, perivascular inflammatory stranding, and rapid enlargement over short intervals, along with necrotizing pneumonitis and cavitary nodules reflecting septic embolization [4]. The presence of an emphysematous shoulder abscess further underlined the disseminated septic state. CT remains the cornerstone of diagnosis, but MRI is valuable for soft-tissue characterization, while FDG-PET/CT has been described in literature for differentiating infected from non-infected aneurysms, although it was not applied here [4,5]. Microbiological confirmation of S. aureus supported the imaging diagnosis, in line with reports that identify this organism as a leading cause of thoracic mycotic aneurysms, especially in association with musculoskeletal infections and prior vascular interventions [3]. Management requires a combined approach of prolonged targeted antimicrobial therapy and exclusion of the infected arterial segment. Open repair with radical debridement and graft replacement is considered the gold standard but carries high perioperative mortality, particularly in fragile or septic patients, and adjuncts such as omental flaps, homografts, or antibiotic-soaked prostheses have been advocated to reduce reinfection [3]. Endovascular repair has emerged as an important alternative, offering immediate hemodynamic stabilization with lower short-term mortality, yet it does not eradicate infection and requires lifelong antibiotic therapy and close imaging surveillance due to persistent risks of reinfection and rupture [6]. In our patient, despite empiric then targeted antibiotics and surgical drainage of the abscess, the aneurysm continued to enlarge, reflecting uncontrolled infection; although endovascular treatment was considered, rapid clinical deterioration culminating in septic shock precluded intervention. This unfavorable outcome mirrors the poor prognosis widely reported in the literature, where mortality exceeds 50% in untreated or inadequately controlled cases [5]. Ultimately, this case underscores the importance of maintaining a high index of suspicion for mycotic aneurysms in septic patients with vascular risk factors, the critical role of early multimodal imaging and microbiological correlation, the need for urgent but carefully tailored surgical or endovascular management, and the indispensable contribution of a multidisciplinary team in optimizing outcomes in these complex and often fatal infections.

## CONCLUSION

Mycotic aneurysms of the thoracic aorta are rare but highly lethal infections that require prompt recognition and aggressive management. This case highlights how predisposing vascular risk factors, disseminated Staphylococcus aureus infection, and delayed control of the infectious source can converge to produce rapid aneurysm progression with fatal outcome. Early multimodal imaging, timely microbiological correlation, and coordinated multidisciplinary care are essential to establish the diagnosis, guide treatment, and improve prognosis. Despite advances in antibiotics and endovascular techniques, mortality remains high, underscoring the need for vigilance in at-risk patients presenting with sepsis and unexplained arterial dilatation.

**Conflict of Interest:** The authors declare no conflicts of interest.

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