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Rapid Cavitation of a Regressing Pulmonary Infarction in Takayasu Arteritis: Surgical Salvage of a Secondary Superinfection, a Case Report

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Case Report

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Abstract

Background: Although pulmonary artery involvement is radiologically common in Takayasu Arteritis (TA), symptomatic pulmonary infarction is exceptionally rare. Furthermore, the clinical course of a regressing infarct evolving into a septic cavity under immunosuppression is sparsely described, and the distinction between vasculitis progression and secondary superinfection presents a major diagnostic challenge.

Case presentation: A 59-year-old female with TA complicated by left lower lobe pulmonary infarction was treated with glucocorticoids and tocilizumab. Follow-up imaging demonstrated marked infarct regression. However, the patient subsequently presented with acute dyspnea, and computed tomography revealed explosive cavitation of the previously shrinking infarct. The avascular, necrotic tissue acted as a "pharmacological sanctuary," rendering conservative antimicrobial therapy ineffective against the subsequently identified Escherichia coli and occult Aspergillus flavus superinfections. Following a multidisciplinary consultation, the patient underwent video-assisted thoracoscopic surgery (VATS) left lower lobectomy, achieving definitive source control and complete clinical recovery.

Conclusions: In immunosuppressed TA patients, paradoxical cavitation of a regressing pulmonary infarct strongly indicates secondary superinfection rather than an autoimmune flare. Because devitalized cavitary lesions serve as impenetrable pharmacological sanctuaries, early anatomic lung resection is a crucial, life-saving intervention when pharmacotherapy fails.

1. Background

Takayasu Arteritis (TA) is a rare, chronic granulomatous vasculitis that primarily affects the aorta and its major branches, with a significantly higher incidence in East Asian populations. Although pulmonary artery involvement is radiologically identified in 50% to 80% of patients, symptomatic pulmonary infarction is exceptionally rare owing to the compensatory mechanism of the bronchial collateral circulation. According to previous statistics, approximately 4% to 7% of pulmonary infarction cases subsequently lead to cavity formation.[1] Furthermore, cavitary lung lesions present a remarkably broad differential diagnosis, making the distinction between an atypical autoimmune manifestation and a superimposed opportunistic infection extremely challenging.[2] Current guidelines predominantly address immunosuppression or vascular reconstruction, leaving a significant void in the management of massive pulmonary necrosis complicated by superinfection. The purpose of this report is to present a unique case characterized by a specific phenomenon of "paradoxical cavitation"—initial regression under immunosuppressive therapy followed by explosive liquefaction. We aim to highlight that early video-assisted thoracoscopic surgery (VATS) is the essential strategy for controlling the source of infection when such infarct cavitation occurs.

2. Case presentation

A 59-year-old female was admitted on November 17, 2025, with a 16-day history of worsening dyspnea, cough, and purulent expectoration. She was diagnosed with Takayasu Arteritis (TA) involving the pulmonary arteries in April 2025. Initial treatment consisted of intravenous methylprednisolone (80 mg/day for 7 days) and cyclophosphamide (0.4 g). Maintenance therapy involved oral methylprednisolone (gradually tapered to 8 mg/day), methotrexate (12.5 mg/week), and tocilizumab (560 mg intravenously, every 4 weeks for a total of 5 doses). Other long-term medications included atorvastatin (20 mg qn) and rabeprazole (20 mg qd). Her current illness began on November 1, 2025, with mild exertional dyspnea. Following a cold on November 11, her symptoms progressed to include cough and yellow mucoid sputum. Prior to transfer, she received empirical treatment at a local hospital, including oral amoxicillin/clavulanate, intravenous moxifloxacin (400 mg qd), and cefoperazone-sulbactam (3.0 g q12h). Despite these interventions, her dyspnea continued to deteriorate. Upon admission, physical examination revealed tachycardia (102 bpm) and diminished breath sounds in the left lower lung. Laboratory investigations showed leukocytosis (10.48×10^9/L) and elevated interleukin-6 (179.1 pg/mL). A retrospective review of serial computed tomography (CT) scans showed that the initial consolidation from April 2025 had significantly resolved by October 2025 under immunosuppression. However, the admission CT in November revealed a large thick-walled cavity with an air-fluid level in the residual fibrotic area (Fig. 1). Three-dimensional reconstruction visually confirmed the sequestered, avascular nature of the necrotic lesion (Fig. 2). Next-generation sequencing (NGS) analysis of the bronchoalveolar lavage fluid (BALF) identified Escherichia coli. Despite nine days of targeted antibiotic therapy with intravenous moxifloxacin (400 mg qd) and cefoperazone-sulbactam (3.0 g q12h), the patient’s condition stagnated. Following a multidisciplinary team consultation, the lesion was identified as a devitalized "sequestered lesion" acting as a pharmacological sanctuary, necessitating surgical intervention. The patient underwent a video-assisted thoracoscopic surgery (VATS) left lower lobectomy on November 26, 2025. Postoperatively, repeat BALF NGS identified a co-infection of Aspergillus flavus and E. coli. The treatment was adjusted to intravenous ceftazidime (2.0 g q8h) and oral voriconazole (loading dose: 400 mg q12h on day 1; maintenance dose: 200 mg q12h). The patient achieved complete symptom resolution and was discharged on December 4, 2025, with a planned 4-to-6-week course of voriconazole. Patient perspective: The patient expressed high satisfaction with the surgical outcome, noting that her respiratory distress resolved immediately following the procedure. She stated that the timely surgery significantly restored her quality of life.

3. Discussion and Conclusions

This case elucidates a critical clinical paradigm rarely reported in East Asian populations: the biphasic evolution of a pulmonary infarct in a patient with TA undergoing long-term immunosuppression, characterized by initial significant regression followed by explosive cavitation. It provides a vital clinical reference for this life-threatening condition, highlighting the limitations of medical therapy and the decisive role of early surgical source control.

TA is a large-vessel vasculitis with a significantly higher incidence in East Asian countries, where up to 80% of patients may exhibit pulmonary artery involvement. However, symptomatic pulmonary infarction

and secondary severe cavitary infections are rarely reported. Typical pulmonary lesions in TA progress alongside vasculitis activity via chronic ischemia or in situ thrombosis, which can occasionally manifest as hemorrhagic infarction with intricate pathological correlations.[3] In contrast, the paradoxical rapid cavitation of a previously healing fibrotic scar observed here is pathognomonic for secondary liquefactive necrosis rather than a vasculitis flare. Notably, given the high endemic rate of tuberculosis in Asia, pulmonary tuberculosis is the primary differential diagnosis for cavitary lesions in immunocompromised hosts and must be strictly excluded.[4]

The patient’s long-term use of glucocorticoids combined with tocilizumab created a critical diagnostic blind spot. Recent comprehensive reviews highlight that cavitary lung lesions in immunocompromised hosts are exquisitely susceptible to opportunistic superinfections, often presenting with muted clinical signs until severe necrosis occurs.[5] By suppressing the acute-phase response, biologic agents mask fever and the elevation of inflammatory markers. Simultaneously, the ischemic infarcted lung tissue transforms into a "pharmacological sanctuary" impenetrable to systemic antimicrobial agents. This mechanism aligns with a similar TA case reported by Narita et al., but our case is unique in that cavitation occurred after the infarct had significantly regressed, rather than progressing from an acute infarct.[6]

VATS anatomic lobectomy was the only viable salvage option. By physically removing the avascular reservoir of infection, surgery achieved definitive source control and laid the foundation for accurate microbiological diagnosis. The preoperative BALF NGS only detected E. coli; however, the postoperative bronchoscopic BALF NGS successfully revealed an occult A. flavus infection. Butler et al. demonstrated that infected cavitary pulmonary infarctions are notoriously refractory to conservative medical management; therefore, pulmonary resection is a definitive treatment modality required to eradicate the septic focus.[7] Recent surgical literature strongly reinforces this approach: cavitary infarctions complicated by necrotic debris, such as those secondary to pulmonary emboli, are notoriously refractory to conservative management and typically warrant surgical intervention.[8, 9]

Regarding surgical timing, we recommend early intervention for patients whose condition continues to deteriorate after 72 hours of standard antibiotic therapy to prevent fatal complications such as cavity rupture. Although surgery during the active phase of vasculitis carries risks of delayed wound healing and bronchopleural fistula, the mortality risk of uncontrolled sepsis is significantly higher; thus, emergency surgical source control takes absolute precedence.

Ultimately, paradoxical cavitation of a regressing pulmonary infarct in immunosuppressed TA patients strongly indicates secondary superinfection rather than vasculitis progression. Because devitalized cavitary lesions act as impenetrable pharmacological sanctuaries, early anatomic lung resection is not merely an option, but the definitive life-saving intervention that ensures a favorable prognosis. Preventing fatal septic complications dictates strict imaging surveillance and a decisive shift toward surgical source control before cavity rupture occurs. While limited by the single-case design, our findings warrant future multicenter studies to validate this aggressive surgical paradigm.

Abbreviations

TA
Takayasu Arteritis
CT
Computed tomography
BALF
Bronchoalveolar lavage fluid
NGS
Next-generation sequencing
VATS
Video-assisted thoracoscopic surgery

Declarations

Ethics approval and consent to participate

Institutional review board approval was obtained from the Ethics Committee of Hebei General Hospital.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor of this journal.

Clinical trial number: not applicable

Availability of data and materials

The datasets analysed during the current study are not publicly available due to third-party commercial laboratory policies and institutional patient privacy restrictions, but the relevant clinical diagnostic data are fully described within the article.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

WH analyzed and interpreted the patient data, and wrote the main manuscript text. MW contributed to the conception and design of the study and the acquisition of data. DY was involved in the acquisition of

data and data analysis. YL contributed to data analysis interpretation and the drafting of the manuscript. LP participated in the conception and design of the study and the drafting of the manuscript. JY was involved in the acquisition of data and data analysis. DZ contributed to the conception and design of the study and the acquisition of data. XZ supervised the clinical management, was a major contributor in revising the manuscript, and served as the corresponding author. All authors read and approved the final version of the manuscript.

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Authors' information

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Figures

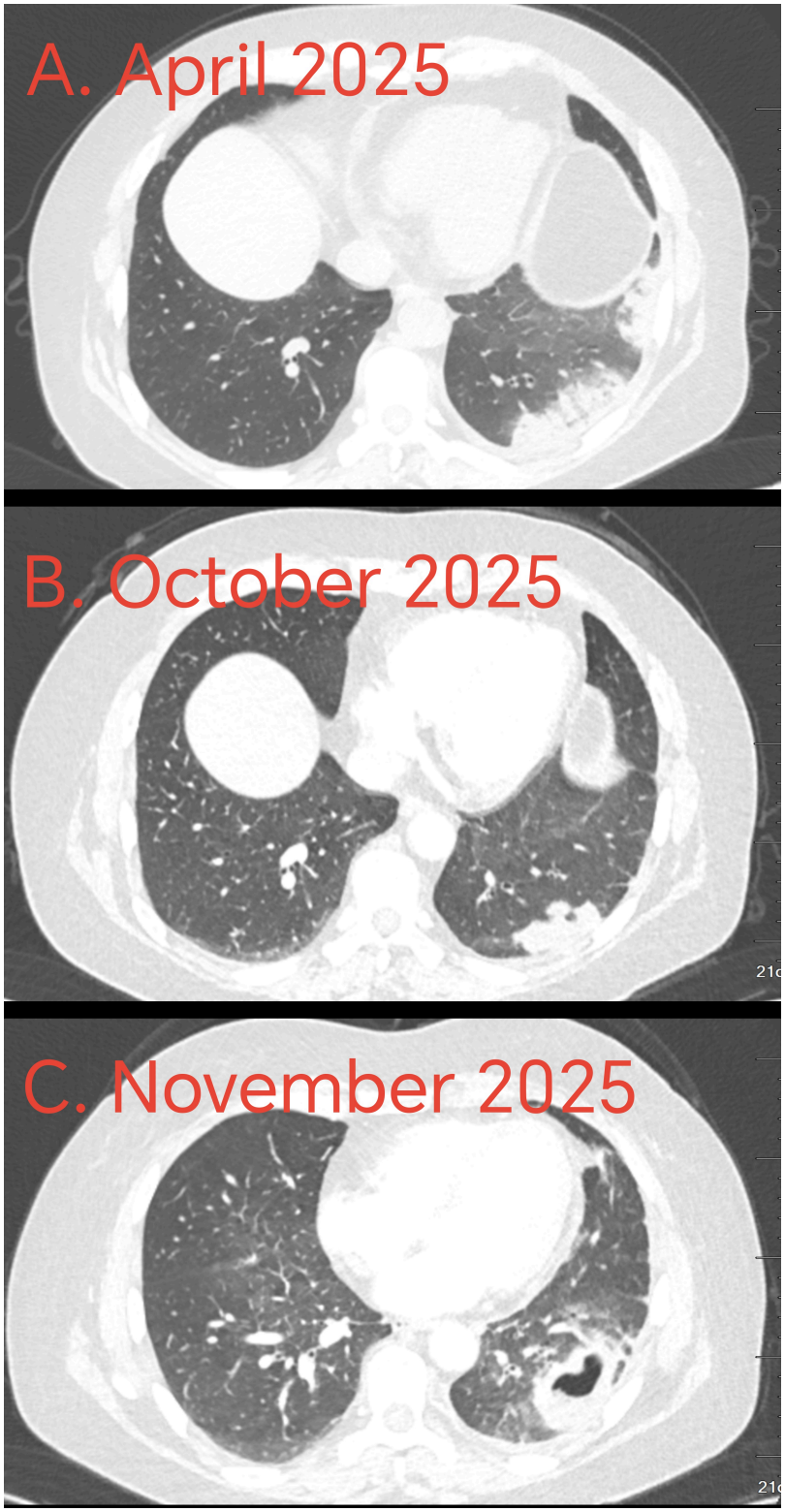


Figure 1

Serial computed tomography (CT) scans revealing a distinct evolutionary pattern of the left lower lobe lesion. (A) April 2025: A large area of consolidation in the left lower lobe consistent with acute pulmonary infarction. (B) October 2025: Marked resolution and reduction of the lesion, confirming the efficacy of immunosuppressive therapy. (C) November 2025: The residual fibrotic area is replaced by a large thick-walled cavity with an air-fluid level, indicating rapid liquefactive necrosis.

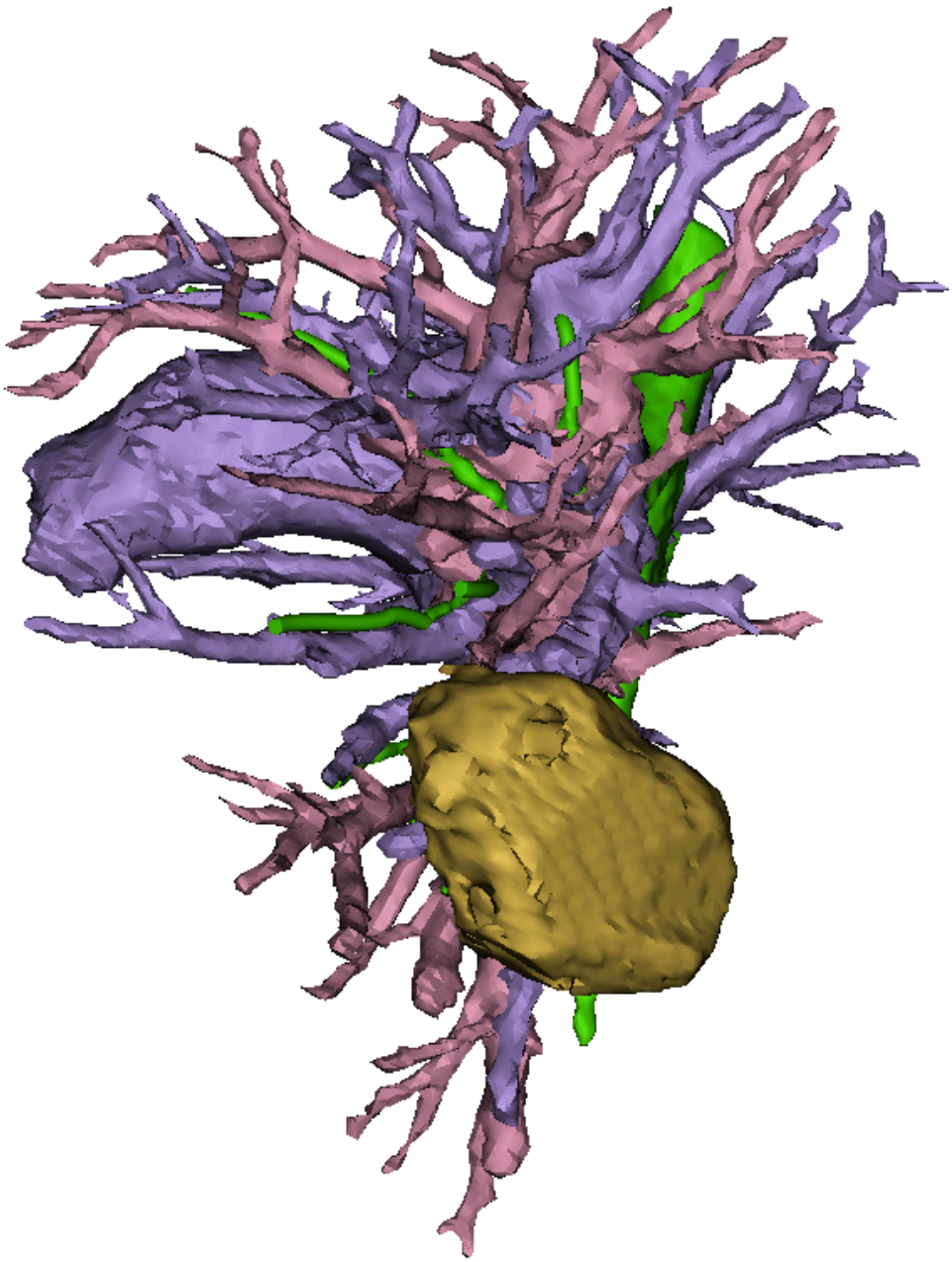


Figure 2

Three-dimensional computed tomography (CT) reconstruction of the left lung (November 2025). The image visually conceptualizes the sequestered, necrotic nature of the left lower lobe lesion. The massive cavitary lesion (yellowish-brown mass) is clearly demarcated from the surrounding pulmonary vasculature (purple) and bronchial tree (green). The evident lack of penetrating vascular supply into the mass visually represents the "pharmacological sanctuary" impenetrable to systemic antibiotics.

Supplementary Files

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