

CASE REPORT

A FATAL CASE OF TAKAYASU ARTERITIS PRESENTED WITH AUTOIMMUNE HEMOLYTIC ANEMIA

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Abstract

Takayasu arteritis (TA), a rare granulomatous vasculitis primarily affecting large vessels, presents with a myriad of complex clinical features. This case report discusses a 24-year-old female presenting with fever, breathlessness, and heart failure, ultimately diagnosed with Takayasu arteritis (TA) complicated by autoimmune hemolytic anemia (AIHA). Despite treatment with antibiotics, steroids, and methotrexate, alongside interventions for pneumonia and tuberculosis, the patient's condition deteriorated, resulting in mortality. The case underscores TA's diverse and challenging clinical manifestations, including cardiac involvement and AIHA, emphasizing the need for multidisciplinary management and heightened clinical awareness. Despite efforts to address complications, the patient's outcome highlights the complexity and severity of TA, warranting further research and awareness in clinical practice.

Keywords: Takayasu arteritis (TA), Autoimmune hemolytic anemia (AIHA), Arterial Thrombosis, POU

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Introduction

Takayasu arteritis (TA), also referred to as pulseless disease, was first identified by Japanese ophthalmologist Dr. Mikito Takayasu in 1908. It is a chronic inflammatory condition that primarily affects the medium and large arteries and their branches. The disease often targets the aorta and its major branches, including the renal, carotid, and subclavian arteries, leading to narrowing, blockages, or aneurysmal changes in these vessels.^{1,2} TA is recognized globally, with its incidence estimated at 2 cases per 1,000,000 people per year.^{3,4} Although the disease is found worldwide, it is believed to be significantly more

prevalent in the Asian population. In Japan, the highest recorded prevalence of Takayasu arteritis is estimated at 40 cases per million, whereas in the United States, the lowest reported frequency is 0.9 cases per million.^{5,6} It is the most common cause of granulomatous inflammation of large arteries and the third most common cause of vasculitis in the pediatric age group.⁷ The active stage of TA arises most commonly in young females of reproductive age.⁸ The hallmark feature of Takayasu arteritis is inflammation that leads to stenosis, occlusion, or aneurysmal dilation of the affected vessels.⁹ The cardiovascular system is the main focus of the wide range of clinical symptoms

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that follow from this. The disease's unpredictable character frequently results in a range of clinical situations, despite the fact that the traditional presentation is decreased or missing pulses caused by artery constriction or occlusion.¹⁰ The complex interaction between systemic inflammation and the cardiovascular system is demonstrated by the cardiac involvement in TA. Widespread vasculitis in major arteries, such as the aorta and its branches, can impair blood supply to essential organs, such as the heart, leading to ischemia and consequent heart failure.¹¹ The rarity of Takayasu arteritis, coupled with its diverse clinical presentations, often poses diagnostic challenges. The insidious onset and the absence of specific biomarkers contribute to delayed recognition, allowing the disease to progress and involve multiple organ systems.⁵ This case report aims to highlight the distinctive presentation of Takayasu arteritis, emphasizing how it can impersonate other common conditions, which may result in a delayed diagnosis. The inclusion of heart failure as a significant manifestation emphasizes the importance for clinicians to consider TA when evaluating patients with unexplained fever, weight loss, and cardiovascular symptoms.

Case report:

A 24-year-old non-diabetic, non-hypertensive female, student presented with a one-year history of persistent high-grade fever and three months of breathlessness. The fever initially responded to paracetamol but recurred after a brief afebrile periods. Despite signs of urinary tract infection (UTI) with pus cells in urine RME and positive *E. coli* in urine culture, multiple antibiotic courses failed to alleviate the fever. For the last 3 months, she experienced progressive breathlessness, orthopnea, and paroxysmal nocturnal dyspnea. She received multiple blood transfusions due to persistent anemia, losing 10 kg of weight in the process. She was admitted in a specialized cardiac hospital for three weeks and subsequently referred to our hospital for further management.

Upon examination, she appeared cachexic and moderately anemic but non-icteric. She exhibited a temperature of 100°F, with no cyanosis but bilateral pitting edema. Cardiovascular assessment revealed a regular pulse of 110 bpm on right side, but absence of radial and brachial pulses on the left side. Carotid bruit was present bilaterally, more prominent on the left. Blood pressure was nonrecordable on the left and 110/60 mmHg on the right. Cardiac auscultation revealed normal heart sounds with no signs of pulmonary hypertension.

Respiratory examination indicated bilateral lower zone pleural effusion. Neurological examination revealed muscle wasting, particularly in the upper limbs. Laboratory investigations showed consistently low hemoglobin, elevated ESR, and high CRP. Reticulocyte count was elevated at 5.33%, and the direct Coombs test was positive. LDH levels were high at 400 U/L. Liver enzymes SGPT and SGOT were elevated (105 and 167, respectively) along with Ferritin (1139 ng/ml) and transferrin saturation (19%). Serum troponin I and NT-proBNP were significantly elevated (0.178 ng/ml and > 35000 pg/ml respectively). Sputum showed growth of pseudomonas. Chest x-ray revealed bilateral pleural effusion with right mid zone consolidation (Figure 1).



Figure 1: Chest X-ray PA view showing bilateral pleural effusion with consolidation of right middle zone

Autoimmune markers, including ANA, anti dsDNA, RA, and ANCA, were negative. Chest X-ray revealed bilateral pleural effusion and consolidation of right middle zone, while echocardiography indicated global hypokinesia and severe left ventricular systolic dysfunction (EF: 29%) with grade 2 diastolic dysfunction. Abdominal ultrasound revealed mild free fluid in the peritoneal cavity. CT angiography of carotid and vertebral arteries demonstrated smooth narrowing of the left common carotid and left subclavian arteries (Figure 2).

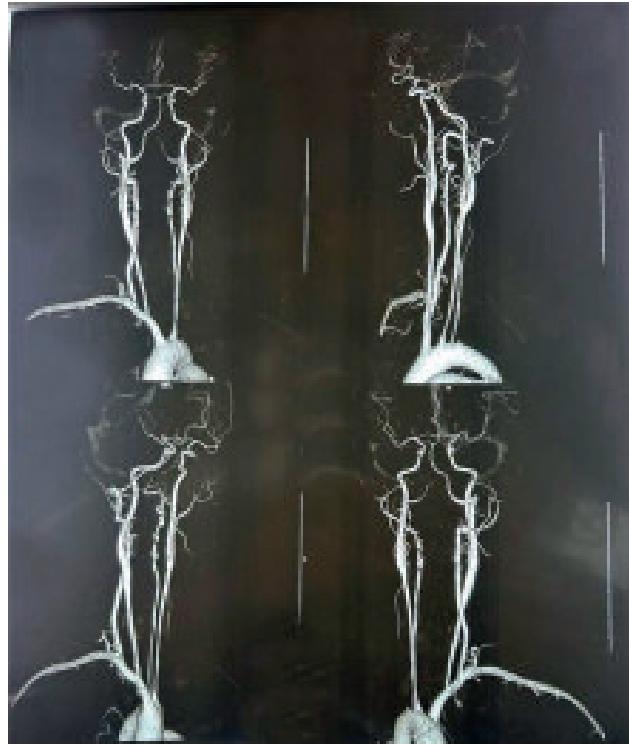


Figure 2: CT angiography of carotid and vertebral arteries showing smooth narrowing of the left common carotid and left subclavian arteries

The patient initially received a combination of amoxicillin and clavulanic acid for pneumonia, alongside a steroid at a dosage of 1mg/kg for Takayasu Arteritis. Unfortunately, there was no positive response to this treatment. Subsequently, the medical team initiated a regimen involving meropenem, levofloxacin, and fluconazole for pneumonia. After a month without improvement, methotrexate was introduced in conjunction with steroids for the management of Takayasu Arteritis. Methotrexate was initially administered orally at a dosage of 15 mg per week. After one month, the administration route was changed to subcutaneous, with an increased dosage of 20 mg per week, subsequently raised to 25 mg per week after two weeks. However, after two weeks of receiving 25 mg methotrexate subcutaneously, the patient developed methotrexate-induced mucositis, prompting discontinuation of the medication. Inj folinic acid and other supportive treatment were ensured immediately. However, after three months the patient passed away from the circumstances, not showing any signs of improvement.

Our diagnosis was Takayasu arteritis with autoimmune haemolytic anaemia and hospital-acquired pneumonia. Detailed investigations are in Table 1.

Table - I
Detailed information of the investigations

	15/05/2022	17/07/2023	13/09/2023
CBC			
Hb (gm/dl)	8.7	6.3	9.9
ESR (mm 1st hour)	94	123	78
WBC ($10^9/L$)	4.5	8.6	9.9
MCV	88	72	80.7
CRP (mg/L)	89.4		
Reticulocyte			5.33%
S.creatinine (mg/dl)	0.8	0.9	0.9
SGPT (U/L)		100	105
SGOT (U/L)		98	167
ALP (U/L)			115
S.albumin (gm/dl)	3.9		3.74
LDH (U/L)			400
S.Iron			124
S. Ferritin			13747
TIBC			188
Tsat			66
D-dimer			5.83
Hs Troponin			0.178
NT pro BNP			> 35000 pg/ml

Discussion:

Takayasu arteritis is classified as a systemic granulomatous large-vessel vasculitis according to the 2012 Chapel Hill Consensus Conference.¹² Various contemporary imaging techniques, such as high-resolution Doppler ultrasound, cardiovascular magnetic resonance, and 18F-fluorodeoxyglucose positron emission tomography, can assist in the early detection of the disease.^{13,14} It is generally recommended to start medical treatments early, particularly using a combination of corticosteroids and immunosuppressive agents, to reduce vascular injuries.¹⁵ The case presents a unique and challenging clinical scenario, highlighting the intricate interplay between Takayasu arteritis (TA) and its atypical manifestation with heart failure, compounded by the concurrent presence of autoimmune hemolytic anemia (AIHA) and pneumonia. The varied clinical symptoms of the disease can create diagnostic challenges, as illustrated by this case where the initial signs resembled a urinary tract infection, delaying the diagnosis of vasculitis. Cardiovascular involvement complicates the clinical picture, potentially leading to heart failure. Although Takayasu arteritis usually causes arterial stenosis or occlusion, resulting in reduced pulses and systemic hypertension, its effects on the myocardium are less commonly reported.¹⁶ In this case, the cardiac involvement manifested as global hypokinesia and severe left ventricular systolic dysfunction, with an ejection fraction (EF) of 29%. The development of heart failure can be attributed to compromised blood flow to the myocardium due to the extensive vasculitis affecting the coronary arteries. Such cardiovascular complications in TA are rare but have been reported in the literature. A case report by Quan et al documented a patient with TA presenting with heart failure and severe coronary artery involvement, emphasizing the diverse cardiovascular manifestations of this vasculitic condition.¹⁷ CT angiography of the carotid and vertebral arteries revealed smooth narrowing of the left common carotid and left subclavian arteries in the patient's report. Similarly, Kim et al found a large mural thrombus in the left ventricle of a 14-year-old girl with Takayasu arteritis.¹⁸ Akazawa et al also argued that Takayasu arteritis might lead to a hypercoagulable state.¹⁹ Regarding treatment, anti-platelet therapy is typically recommended to prevent additional ischemic events.²⁰ Furthermore, her concomitant autoimmune hemolytic anemia (AIHA) adds another layer of complexity to the clinical picture. Though around 35% patients of Takayasu disease have anemia, autoimmune haemolytic anemia is uncommon.²¹ The positive Direct Coombs test, elevated LDH, and markedly elevated ferritin and transferrin

saturation are consistent with AIHA secondary to TA. The autoimmune component of the disease contributes to the hemolytic process, leading to anemia and necessitating multiple blood transfusions. The co-occurrence of TA and AIHA constitutes an uncommon yet well-documented phenomenon within the scientific literature. A similar case reported by Parvaneh et al. described a 9-year-old girl with TA presenting with Coombs-positive autoimmune hemolytic anemia.² The authors highlighted the importance of recognizing the autoimmune component in TA, as it significantly influences the management and prognosis of the disease. The elevation of inflammatory markers, including ESR and CRP, further supports the systemic inflammatory nature of TA.⁹ The involvement of the liver, as evidenced by elevated SGPT and SGOT, is another facet of the multisystemic impact of this vasculitis. While hepatic involvement in TA is less common, it has been reported in the literature. Vasculitis and granulomatous inflammation may impair liver function.²² The presence of bilateral pleural effusion and the mild free fluid in the peritoneal cavity observed in imaging studies underline the systemic nature of TA. Pleural effusion is a recognized but infrequent complication of TA, and its pathogenesis involves inflammation of the adjacent arteries leading to serositis. Similarly, the involvement of the peritoneal cavity may result from the vasculitic process affecting the mesenteric arteries. The clinical situation presents significant challenges for timely diagnosis and necessitates the prompt application of a customized treatment plan due to the presence of contraindications.

In conclusion, this case provides valuable insights into the complex and varied manifestations of TA. The coexistence of heart failure, pneumonia and AIHA adds a layer of complexity to the clinical presentation, underscoring the importance of a multidisciplinary approach for accurate diagnosis and effective management. The lack of specific symptoms and laboratory biomarkers, along with challenges in evaluating disease activity and progression, often results in the disease being unrecognized at onset and its activity underestimated. The disease typically remains persistently active, leading to unnoticed damage and considerable short- and long-term morbidity and mortality. However, early diagnosis and comprehensive management approaches may improve survival rates.

Conflict of Interest:

The authors stated that there is no conflict of interest in this study.

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Consent for publication:

Informed written consent was taken from the parents of the patient to publish details relevant to the disease and management.

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Competing interests:

None.

Author contributions

All authors were involved in the management of the patient and all authors contributed to the conception, writing, and editing of the case report.

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