Case Report *Open Access*

Journal of Innovative Clinical Trials and Case Reports

ISSN: 3068 - 1936

DOI: doi.org/10.63721/26JCTC0140

Takayasu Arteritis with Multivessel Involvement: A Case Report

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Citation: *Geianne Renci Atienza, Giormaru Cuntapay, Rica Keiza King (2026) Takayasu Arteritis with Multivessel Involvement: A Case Report. J. of Inn Clin Trail Case Reports 2(3), 1-5. WMJ/JCTC-140*

Abstract

Background: *Takayasu arteritis (TAK) is a rare, chronic large-vessel vasculitis affecting the aorta and its major branches, leading to stenosis, occlusion, or aneurysmal changes. The global incidence is approximately 1.11 cases per million person-years, with a strong female predominance (80%–90%), typically manifesting between 40 and 50 years of age. Although TAK occurs worldwide, its prevalence is highest in Asia with 61 reported cases in the Philippines. Clinical manifestations are heterogeneous, ranging from nonspecific systemic symptoms to severe vascular complications, often delaying diagnosis, particularly in atypical cases.*

Case Presentation: *We report a 43-year-old woman with hypertension, diabetes and chronic ischemic stroke without residuals who presented with a two-month history of easy fatigability, generalized weakness, arthralgia, intermittent claudication with progressive dark discoloration of both lower extremities, undocumented low-grade fever and alopecia. She developed acute respiratory distress with abrupt loss of consciousness requiring emergent intubation. Examination revealed a marked discrepancy in blood pressure between the upper extremities, diminished peripheral pulses and an audible abdominal bruit. Laboratory studies showed elevated Troponin I, ESR and CRP, with borderline ANA and normal complement levels. Neuroimaging demonstrated multiple acute and chronic cortical and cerebellar infarcts. Initially managed as a cerebrovascular accident, she required tracheostomy for ventilatory dependence. Antiplatelet and anticoagulant therapy was started after evidence of myocardial infarction. Progressive ischemia of the left lower extremity necessitated below-knee amputation. CT aortography later revealed diffuse thoracoabdominal aortic and iliac involvement with mural thickening, multiple aneurysms, dissection and unilateral renal artery stenosis. Further immunologic workup showed her to be negative for lupus anticoagulant. Immunosuppressive therapy with corticosteroids and methotrexate was initiated to control vascular inflammation and limit further progression.*

Conclusion: *This case highlights the diagnostic complexity of TAK with atypical, multi-organ ischemic presentations, emphasizing the importance of clinical vigilance, comprehensive vascular imaging and timely immunosuppressive therapy to mitigate complications and improve outcomes.*

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Submitted: 02.06.2026 **Accepted:** 08.06.2026 **Published:** 16.06.2026

Introduction

Takayasu arteritis (TAK) is a rare, chronic, granulomatous vasculitis that involves medium- and large-sized arteries, primarily affecting the aorta and its major branches. Although it has a worldwide distribution, it is reported more frequently in Asian populations [1,2] The global incidence is approximately 1.11 cases per million person-years, with a strong female predominance (80%–90%), typically manifesting between 40 and 50 years of age [2,3]. Its epidemiology remains poorly defined, largely due to its rarity and variable clinical presentation [4]. Early symptoms are often nonspecific and the disease tends to follow an indolent course contributing to diagnostic delays. Without timely recognition and treatment, progressive vascular inflammation may lead to obstructive or aneurysmal lesions, resulting in severe complications such as stroke, refractory hypertension, heart failure, blindness and even death [5].

The updated 2022 ACR/EULAR classification criteria integrates both clinical and imaging features, including vascular stenosis identified on angiography or Doppler ultrasound, with a total score of ≥5 required for a confirmed diagnosis [1].

Case Presentation

A 43-year-old woman with a one-year history of hypertension, diabetes and ischemic stroke without residual deficits was evaluated at our institution. She had been poorly compliant with her prescribed antihypertensive and antiplatelet regimen. She denied smoking, alcohol use, oral contraceptive intake, or history of miscarriage. Her family history was significant only for hypertension.

Two months prior to presentation, she developed intermittent claudication with progressive dark discoloration of both lower extremities, most marked in the left great toe (Figure 1). One month later, she experienced constitutional symptoms of fever, nonproductive cough and generalized weakness, followed by worsening dyspnea, cyanosis and chest pain. This culminated in sudden loss of consciousness and emergency admission, during which she required intubation and mechanical ventilation. She was managed as a case of acute coronary syndrome, acute respiratory failure secondary to high-risk community-acquired pneumonia, acute decompensated heart failure and cerebrovascular accident. Despite multiple extubation attempts, she remained ventilator-dependent and underwent tracheostomy. She was discharged against medical advice due to financial constraints, on medical therapy consisting of dual antiplatelet agents, anticoagulation, beta-blocker, angiotensin-receptor blocker, statin and guideline-directed heart failure medications.

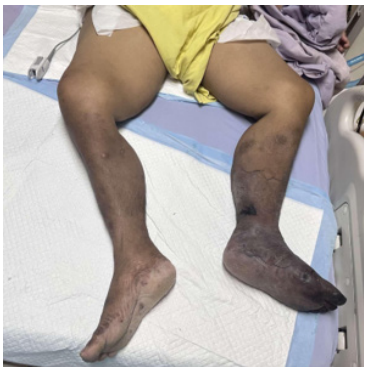

Figure 1: Lower Extremities Demonstrating Bilateral Discoloration with Advanced Ischemic Changes in the Left Limb.

On transit home, she was readmitted for acute dyspnea and cyanosis. On evaluation, her blood pressure remained elevated (160–180/90–100 mmHg) with a notable interarm discrepancy. Physical examination revealed marked discoloration of the left lower extremity with faint to absent distal pulses. Popliteal and posterior tibial pulses were diminished bilaterally, dorsalis pedis pulses were absent and an abdominal bruit was appreciated. She subsequently underwent left below-knee amputation for critical limb ischemia.

Laboratory testing showed elevated Troponin I. Electrocardiogram demonstrated sinus tachycardia. Chest radiography revealed bilateral pulmonary infiltrates and tuberculosis screening was shown to be negative. Cranial CT scan showed multiple infarcts in the cerebral and cerebellar regions. Transthoracic echocardiography revealed concentric left ventricular hypertrophy with preserved ejection fraction (61%) and mild aortic regurgitation. Contrast-enhanced chest CT demonstrated a fusiform aneurysm of the descending

thoracic aorta. CT aortography revealed Stanford type B aortic dissection, aneurysms involving the left renal and right internal iliac arteries, significant right renal artery stenosis and occlusion of the right external iliac artery with collateral flow from the right internal mammary artery (Figure 2). Carotid Doppler ultrasonography showed >70% stenosis of the left internal carotid artery. Arteriovenous imaging confirmed bilateral peripheral arterial disease, probably thromboembolic in origin. These findings fulfilled the 2022 ACR/EULAR classification criteria for TAK (score ≥5).

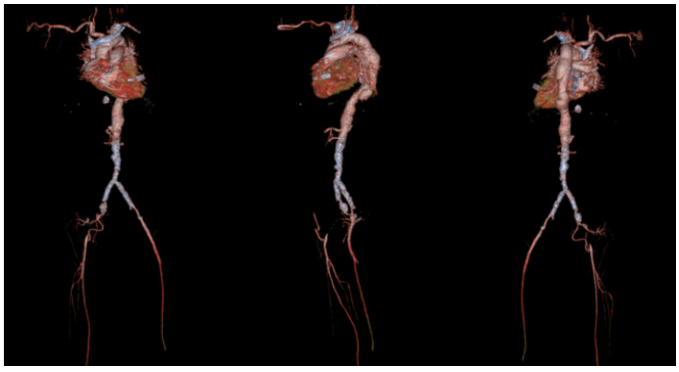

Figure 2: CT aortogram showing fusiform aneurysm of the descending thoracic aorta with Stanford type B dissection. Additional findings include aneurysms of the left renal and right internal iliac arteries, significant right renal artery stenosis and complete occlusion of the right external iliac artery with collateral supply from the right internal mammary artery.

She was initially managed with IV hydrocortisone 100 mg every 8 hours, which was later transitioned to oral prednisone 80 mg daily. Weekly methotrexate at a dose of 17.5 mg, along with folic acid supplementation, was subsequently initiated. During the course of treatment, the patient developed poor wound healing at the left stump site, necessitating redebridement; methotrexate was temporarily withheld and IV hydrocortisone was reinstated. She also developed persistent leukopenia but remained afebrile; thus, antibiotic prophylaxis and filgrastim were administered to which she responded well. Her antihypertensive regimen was optimized and she demonstrated marked clinical improvement. The patient was eventually discharged in stable condition.

Discussion

TAK, first described by Mikito Takayasu in 1908, is a rare, chronic, granulomatous vasculitis that primarily involves large- and medium-sized arteries, particularly the aorta and its major branches. Although the disease has a global distribution, it is more commonly reported in Asian populations [2,3]. The estimated global incidence is approximately 1.11 cases per million person-years, with a marked female predominance of 80%–90%, typically presenting between the fourth and fifth decades of life [2,3]. Reported prevalence varies geographically, ranging from 40 cases per million in Japan to approximately 9 cases per million in the United States [5]. Meanwhile, in the Philippines, there are 61 reported cases of TAK [6]. Several studies have demonstrated an association with HLA-B52 and HLA-B39, supporting a possible immunogenetic predisposition [7].

Histopathologically, TAK is characterized as panarteritis, with the earliest inflammatory changes localized around the vasa vasorum and the medio-adventitial junction [8]. This inflammatory process leads to endothelial injury and activation, reflected by elevated levels of biomarkers such as circulating endothelial cells, circulating endothelial progenitor cells and vascular endothelial growth factor [9].

In its early phase, TAK often presents with nonspecific constitutional symptoms, including low-grade fever, malaise, arthralgias, myalgias, weight loss, or night sweats, which may precede vascular involvement by several months [10,11]. There are no pathognomonic laboratory tests for TAK; although elevated erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) levels may reflect disease activity, they are neither sensitive nor specific. Owing to its nonspecific presentation, diagnosis is frequently delayed until significant vascular compromise has occurred.[7] The disease course is typically subacute, evolving over months to years, often with the development of collateral circulation.[12] However, in advanced stages, progressive vascular inflammation may lead to critical vascular lesions, including stenosis, occlusion, aneurysm formation, or dissection, predisposing to severe complications such as stroke, refractory hypertension, heart failure, visual loss and even death [12,13].

Imaging modalities such as magnetic resonance angiography (MRA) and computed tomography angiography (CTA) play a crucial role in establishing the diagnosis and assessing disease extent [14].

Neurological manifestations occur in approximately

20% of patients with TAK. Cerebrovascular events such as stroke or transient ischemic attacks are typically localized in the carotid artery territory where stenosis is the most common lesion. The disease predominantly affects the internal and common carotid arteries, especially on the left internal carotid artery (39%). The underlying vascular inflammation leads to intimal hyperplasia and adventitial thickening, resulting in luminal narrowing and ischemia from impaired tissue perfusion [9,15].

Cardiac involvement is also recognized in TAK. Acute myocardial infarction (AMI) has been reported in approximately 12.1% of patients, with coronary artery involvement estimated at 9%–11% [9,16]. The coronary ostia are most commonly affected leading to symptoms such as exertional dyspnea, angina, palpitations, or, in severe cases, myocardial infarction and sudden death [16]. In the absence of ischemic wall changes, the diagnosis and management of AMI in TAK relies primarily on clinical manifestations and supportive cardiologic findings confirming ischemia—similar to the present case [16].

Isolated aortic aneurysms have been reported frequently in literature; however, multiple aneurysmal presentations remain uncommon [17]. Studies indicate that while saccular or fusiform aneurysms occur in approximately 31% of TAK patients, multiple aneurysms are identified in only 12%–13%, underscoring the rarity of such a presentation [17]. A comparable case was reported in a 20-year-old patient exhibiting multiple saccular aortic aneurysms involving both the descending thoracic and abdominal aorta [18].

Peripheral arterial manifestations primarily result from progressive arterial stenosis. The earliest and most common clinical findings include diminished or absent peripheral pulses and the gradual development of vascular claudication [19]. In the present case, the patient developed critical ischemia of both lower limbs—a rare but severe manifestation of TAK [20].

Visceral arterial involvement occurs in approximately 11% to 68% of TAK cases, most frequently affecting the renal arteries [15,21]. This typically presents as secondary hypertension due to bilateral ostial or proximal stenotic lesions, in contrast to the distal pattern observed in fibromuscular dysplasia (FMD) [21].

All these vascular findings were likewise observed in our index patient, consistent with previously reported patterns in TAK.

According to the 2022 American College of Rheumatology/European Alliance of Associations for Rheumatology (ACR/EULAR) classification criteria for TAK, a minimum score of five points is required to establish the diagnosis, after excluding other conditions that may mimic vasculitis. Our patient met these criteria. The classification system demonstrates a sensitivity of 93.8% and a specificity of 99.2% [1].

Glucocorticoids remain the cornerstone of treatment, particularly during the acute phase, achieving remission rates of up to 60% [5]. Prednisone is typically initiated at 1 mg/kg/day (maximum 60–80 mg), maintained for 2–4 weeks and gradually tapered over three months based on clinical response. Given the risk of relapse and steroid-related adverse effects, concurrent immunosuppressive therapy is often recommended. Commonly used agents include methotrexate (20–25 mg weekly) or azathioprine (2 mg/kg/day) to facilitate steroid tapering and maintain remission [22].

In refractory or severe cases, biologic disease-modifying antirheumatic drugs (DMARDs) such as tocilizumab or tumor necrosis factor-α inhibitors (infliximab, adalimumab, etanercept, rituximab) may be considered. Endovascular or surgical intervention is reserved for irreversible arterial stenosis causing critical ischemia or for large aneurysm formation [22].

Conclusion

TAK is a rare but potentially fatal vasculitis that can present with widespread arterial involvement. This case demonstrates extensive vascular involvement, including cerebrovascular, coronary, aortic, renal and peripheral arterial disease. It highlights the need for early recognition, comprehensive clinical assessment and strategic use of multimodal imaging to delineate disease extent. Timely diagnosis and tailored medical therapy are essential to prevent irreversible ischemic complications, particularly in settings where advanced interventions may be limited.\

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