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Takayasu Arteritis: Multimodal Imaging and Histopathologic Findings—A Case Report and Narrative Review

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Abstract

Background: Takayasu arteritis (TAK) is a chronic large-vessel vasculitis predominantly affecting the aorta and its major branches. Its heterogeneous clinical manifestations and progressive vascular remodeling frequently complicate early recognition and the distinction between active inflammation and established arterial damage. **Objective:** To review the clinical characteristics, diagnostic imaging modalities, histopathologic features, and therapeutic considerations of TAK, illustrated by a case of upper-extremity ischemia with multimodal vascular assessment and histopathologic correlation. **Methods:** A narrative review of the literature was conducted, focusing on the diagnosis, vascular imaging, disease activity assessment, and management of TAK. An illustrative case of a young adult presenting with upper-extremity ischemia was examined using Doppler ultrasonography, computed tomography angiography (CTA), conventional catheter angiography, and vascular histopathology. **Results:** In the presented case, vascular assessment identified involvement of the right subclavian and axillary arterial territories, with proximal large-vessel abnormalities demonstrated by CTA and corresponding luminal abnormalities documented by conventional angiography. Histopathologic examination revealed marked intimal thickening and inflammatory infiltration composed of lymphocytes and plasma cells, supporting an inflammatory arteriopathy in the appropriate clinical and imaging context. The accompanying literature review highlights the complementary roles of ultrasonography, CTA, magnetic reso-

nance imaging, and FDG-PET/CT in characterizing vascular involvement, while emphasizing the limitations of individual modalities in determining inflammatory activity. **Conclusion:** This case illustrates the value of integrating clinical presentation, complementary vascular imaging, and available histopathologic findings when evaluating suspected TAK in a young patient with upper-extremity ischemia. Multimodal assessment provides distinct anatomical, hemodynamic, and tissue-level information that may support diagnostic reasoning and inform individualized management and surveillance.

Keywords

Takayasu Arteritis, Large-Vessel Vasculitis, Doppler Ultrasonography, Computed Tomography Angiography, Histopathology, Multimodal Imaging

1. Introduction

Takayasu arteritis (TAK) is a rare, chronic, immune-mediated large-vessel vasculitis characterized by granulomatous inflammation predominantly affecting the aorta and its major branches, although pulmonary and coronary arteries may also be involved. The inflammatory process may progressively induce arterial wall thickening, fibrosis, stenosis, occlusion, dilatation, and aneurysm formation, ultimately resulting in tissue ischemia and irreversible vascular damage. TAK predominantly affects young individuals, particularly women, although its demographic distribution varies considerably across geographic regions and ethnic populations. A systematic review and meta-analysis estimated an overall incidence of approximately 1.11 cases per million person-years, highlighting the rarity of the disease and the consequent challenges associated with its recognition and study [1].

The pathogenesis of TAK remains incompletely understood and likely results from a complex interaction between genetic susceptibility and dysregulated innate and adaptive immune responses. Inflammation begins within the arterial wall and may evolve from active cellular infiltration to progressive remodeling and fibrosis. Consequently, the clinical phenotype reflects a continuum between inflammatory activity and accumulated vascular damage rather than two completely independent stages. This distinction has important implications for diagnosis and management because persistent symptoms or vascular abnormalities do not necessarily indicate ongoing inflammation, while apparently quiescent disease may coexist with progressive arterial injury [2].

Clinical manifestations are heterogeneous and depend on both the degree and anatomical distribution of vascular involvement. Early disease may present with nonspecific constitutional manifestations, whereas progressive arterial involvement can result in upper- or lower-extremity claudication, diminished or absent pulses, arterial bruits, discrepancies in blood pressure between extremities, renovascular hypertension, cerebrovascular events, and other manifestations of end-organ is-

chemia. The absence of a disease-specific biomarker and the limited correlation between conventional inflammatory markers and vascular inflammation further complicate recognition of active disease. As a result, diagnostic delay remains an important clinical problem, particularly before the development of overt vascular manifestations [3].

The increasing importance of objective vascular assessment is reflected in contemporary classification systems. The 2022 American College of Rheumatology/European Alliance of Associations for Rheumatology (ACR/EULAR) classification criteria incorporate imaging evidence of large-vessel vasculitis as an absolute requirement, together with age ≤ 60 years at diagnosis, before weighted clinical and vascular characteristics are considered. These criteria demonstrated high sensitivity and specificity in their validation cohort, although they were developed for classification in research rather than as standalone diagnostic criteria for individual patients [4].

Vascular imaging has therefore become fundamental to the evaluation of TAK, allowing characterization of disease distribution, arterial wall abnormalities, luminal changes, and vascular complications. Importantly, different imaging modalities interrogate distinct components of the disease process. Doppler ultrasonography can identify arterial wall abnormalities and characterize their hemodynamic consequences, particularly in accessible vascular territories. Computed tomography angiography (CTA) provides high-resolution assessment of the arterial lumen, mural abnormalities, calcification, stenosis, occlusion, aneurysmal changes, and collateral circulation. Magnetic resonance imaging and magnetic resonance angiography (MRI/MRA) permit evaluation of both vessel-wall and luminal abnormalities without exposure to ionizing radiation. A systematic review and meta-analysis demonstrated that ultrasonography, CTA, and MRA all have substantial diagnostic utility in TAK, while also emphasizing the greater uncertainty surrounding the ability of individual imaging findings to accurately determine inflammatory activity [5].

Current recommendations consequently favor a multimodal and clinically integrated approach rather than reliance on a single imaging technique. The 2023 EULAR recommendations identify MRI as the preferred imaging modality for patients with suspected TAK, with FDG-PET, CT, and ultrasonography as alternatives depending on clinical circumstances, vascular territory, availability, and local expertise. During follow-up, imaging may also provide important information when relapse is suspected and conventional laboratory markers are unreliable, while MR angiography, CT angiography, and ultrasonography can contribute to long-term surveillance of structural vascular damage [6].

Metabolic imaging has further expanded the spectrum of vascular assessment. ^{18}F-fluorodeoxyglucose positron emission tomography (^{18}F-FDG PET), frequently combined with CT, evaluates increased glucose metabolism within inflamed arterial walls and may therefore provide information regarding inflammatory activity that is complementary to anatomical imaging. Nevertheless, FDG uptake is not a

perfect surrogate for histopathologic activity, and its interpretation requires consideration of treatment status, vascular remodeling, atherosclerosis, imaging protocols, and the clinical context. Conversely, conventional catheter angiography primarily depicts luminal anatomy and does not directly characterize vessel-wall inflammation; although historically central to the diagnosis of TAK, its contemporary role is more selective and particularly relevant when detailed vascular mapping or endovascular intervention is required [7].

Despite these advances, distinguishing active vascular inflammation from established arterial damage remains one of the principal challenges in TAK. Clinical manifestations, acute-phase reactants, anatomical imaging, and metabolic imaging may provide discordant information, and no single parameter can reliably define disease activity in every patient. This limitation reinforces the need to integrate clinical findings with biochemical markers and serial multimodal vascular imaging. Such integration is particularly relevant when therapeutic decisions depend on differentiating potentially reversible inflammatory disease from fixed stenotic or occlusive lesions resulting from chronic vascular remodeling [8].

In this report, we describe a patient with Takayasu arteritis and clinically significant upper-extremity arterial involvement, illustrating the complementary contribution of Doppler ultrasonography, computed tomography angiography, conventional angiography, and histopathologic examination. By correlating clinical manifestations with structural, hemodynamic, and histopathologic findings, and integrating these observations with a narrative review of the current literature, this report aims to illustrate the diagnostic spectrum of TAK and the value of multimodal assessment in characterizing vascular involvement.

2. Case Presentation

A 28-year-old woman presented with a six-month history of progressive right upper-extremity claudication, initially occurring during strenuous physical activity and subsequently developing during routine daily activities. The symptoms were accompanied by intermittent paresthesia, coldness, and fatigue of the affected limb, without rest pain, digital ulceration, or tissue loss. She had no history of cardiovascular disease, previous vascular interventions, smoking, diabetes mellitus, hypertension, or dyslipidemia.

Physical examination revealed a cooler right upper extremity, with markedly diminished right brachial and radial pulses compared with the contralateral limb. Bilateral blood pressure measurements demonstrated a significant inter-arm discrepancy, with readings of 88/56 mmHg in the right arm and 122/76 mmHg in the left arm, corresponding to a systolic difference of 34 mmHg. A systolic bruit was audible over the right supraclavicular region. No evidence of acute limb-threatening ischemia was identified.

Laboratory investigations demonstrated elevated inflammatory markers, including an erythrocyte sedimentation rate of 52 mm/h and a C-reactive protein

concentration of 24 mg/L. Complete blood count and renal function were within normal limits. The combination of young age, progressive ischemic manifestations, asymmetric peripheral pulses, inter-arm blood pressure discrepancy, and elevated inflammatory markers raised suspicion for an inflammatory large-vessel arteriopathy. Consequently, multimodal vascular imaging was undertaken to characterize the anatomical distribution and hemodynamic consequences of the suspected arterial involvement.

2.1. Vascular Imaging Findings

Initial vascular assessment was performed using Doppler ultrasonography, focusing on the right subclavian and axillary arterial territories. Grayscale examination of the right subclavian artery demonstrated an abnormal vascular appearance, while color Doppler interrogation of the right axillary artery documented flow within the affected arterial circulation (**Figure 1(A)**). These findings localized the suspected vascular process to the proximal right upper-extremity circulation. However, the available documentation did not provide reproducible measurements of arterial wall thickness, peak systolic velocity, velocity ratios, or Doppler waveform characteristics. Consequently, the hemodynamic severity of the lesion could not be quantified from the available records.

Computed tomography angiography (CTA) was subsequently performed to evaluate the thoracic great vessels and proximal arterial branches. Axial and coronal reconstructions demonstrated vascular abnormalities involving the aortic arch and proximal branch-vessel territory, with the affected regions highlighted by red arrows (**Figure 1(B)**). This examination provided a broader anatomical assessment of the vascular distribution and its relationship to the symptomatic right upper-extremity circulation. Nevertheless, the available imaging description did not establish the exact proximal and distal boundaries of the lesion, a measurable percentage of luminal narrowing, or the presence of a complete arterial occlusion. Accordingly, the findings were interpreted as evidence of proximal large-vessel involvement without assigning an unsupported stenosis grade.

Conventional catheter angiography was subsequently undertaken to further characterize the arterial lumen. Angiographic images demonstrated abnormal luminal morphology and contrast opacification within the arterial circulation supplying the affected upper extremity (**Figure 1(C)**). These findings were anatomically concordant with the proximal vascular abnormalities identified on CTA and the right subclavian and axillary abnormalities observed on Doppler ultrasonography. Although catheter angiography provided additional luminal characterization, the available documentation did not permit reliable quantification of lesion length, percentage stenosis, collateral circulation, or distal runoff.

Overall, the three imaging modalities demonstrated complementary abnormalities involving the proximal right upper-extremity arterial circulation, particularly the subclavian territory. Doppler ultrasonography contributed localized vascular and flow assessment (**Figure 1(A)**), CTA characterized the relationship of the af-

fected circulation to the aortic arch and proximal branch vessels (**Figure 1(B)**), and conventional angiography documented the corresponding luminal abnormalities (**Figure 1(C)**). Their combined interpretation supported a proximal large-vessel arteriopathy consistent with the clinical presentation, while the absence of quantitative imaging measurements limited precise anatomical and hemodynamic grading.

2.2. Histopathologic Findings

Histopathologic examination of vascular tissue was included in the diagnostic assessment (**Figure 1(D)**). However, the available case documentation did not specify the exact arterial segment from which the specimen was obtained, the procedure used for tissue acquisition, or whether sampling occurred before or after the initiation of immunosuppressive treatment. These limitations precluded a definitive correlation between the sampled tissue and a particular vascular lesion identified on imaging.

On hematoxylin and eosin staining, the lower-magnification section demonstrated marked intimal thickening, characterized by expansion and remodeling of the intimal layer (**Figure 1(D)**, left panel; yellow arrow). Higher-magnification examination revealed chronic inflammatory cellular infiltration, including lymphocytes and plasma cells (**Figure 1(D)**, right panel; blue arrows). Together, these observations demonstrated structural vascular remodeling accompanied by inflammatory changes.

The histopathologic findings were interpreted in conjunction with the clinical presentation and the abnormalities demonstrated by Doppler ultrasonography, computed tomography angiography, and conventional catheter angiography. Although intimal thickening and lymphoplasmacytic infiltration are compatible with an inflammatory arteriopathy, these findings are not specific to Takayasu arteritis and cannot independently establish the diagnosis. Furthermore, the absence of information regarding the sampling site, acquisition technique, and treatment status at the time of tissue collection limits the interpretation of the histopathologic findings and their direct anatomical correlation with the affected right subclavian and axillary arterial territories.

2.3. Differential Diagnosis

The differential diagnosis of Takayasu arteritis in a young patient presenting with upper-extremity ischemia includes other inflammatory large-vessel disorders, infectious arteritis, and noninflammatory causes of arterial narrowing. Although the clinical presentation and multimodal imaging findings suggested a proximal large-vessel arteriopathy, the diagnosis required consideration of alternative conditions capable of producing similar vascular abnormalities.

Among inflammatory disorders, giant cell arteritis represents an important differential diagnosis because it may involve the aorta and its major branches, including the subclavian arteries. However, its usual presentation in individuals

older than 50 years makes it less likely in the simulated young-adult scenario. Other systemic inflammatory conditions, including Behçet disease and IgG4-related vascular disease, may also produce large-vessel abnormalities, although their recognition generally depends on associated systemic manifestations, characteristic vascular patterns, and additional clinical or laboratory evidence.

Infectious arteritis must also be considered, particularly in the presence of constitutional symptoms, bacteremia, or imaging findings suggestive of vascular infection. Relevant etiologies include bacterial infections, tuberculosis, and syphilis, depending on the clinical and epidemiological context. Because the available documentation does not include a complete infectious evaluation, these causes cannot be considered definitively excluded.

Noninflammatory arteriopathies, particularly premature atherosclerotic disease, fibromuscular dysplasia, congenital arterial abnormalities, and vascular compression syndromes, may similarly present with upper-extremity ischemia. Their differentiation depends on the distribution and morphology of the arterial lesions, associated risk factors, and clinical context. The proximal large-vessel involvement and accompanying inflammatory features favored an inflammatory arteriopathy, although these findings alone were insufficient to establish a specific etiology.

Histopathologic examination demonstrated marked intimal thickening and lymphoplasmacytic inflammatory infiltration (**Figure 1(D)**). These findings supported vascular remodeling associated with chronic inflammation but were not pathognomonic for Takayasu arteritis. In particular, the absence of documented sampling-site information, detailed arterial-wall layer assessment, and ancillary studies limited the ability to distinguish Takayasu arteritis from other inflammatory or infectious vascular disorders on histopathologic grounds alone.

Overall, Takayasu arteritis was considered the leading diagnostic possibility based on the combined clinical presentation, proximal arterial involvement demonstrated by multimodal imaging (**Figures 1(A)-(C)**), and supportive histopathologic findings (**Figure 1(D)**). Nevertheless, the absence of documented exclusion testing and complete pathological characterization should be acknowledged as a limitation of the diagnostic assessment.

2.4. Final Diagnosis

The overall diagnostic assessment supported Takayasu arteritis presenting with clinically significant right upper-extremity ischemia. The combination of young age, progressive vascular symptoms, asymmetric peripheral pulses, substantial inter-arm blood pressure discrepancy, elevated inflammatory markers, and proximal large-vessel abnormalities provided a coherent clinical and vascular pattern compatible with the disease.

Multimodal imaging demonstrated complementary aspects of the arterial involvement, including abnormalities of the right subclavian and axillary circulation on Doppler ultrasonography (**Figure 1(A)**), proximal great-vessel involvement on

CTA (Figure 1(B)), and corresponding luminal abnormalities on conventional angiography (Figure 1(C)). Histopathologic examination additionally demonstrated intimal remodeling and lymphoplasmacytic inflammatory infiltration (Figure 1(D)). Together, these findings supported the proposed diagnosis within the simulated clinical scenario.

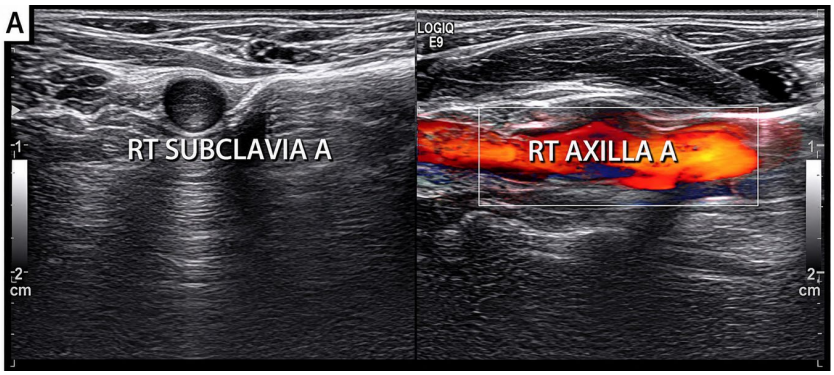
3. Treatment and Follow-Up

The diagnostic evaluation supported Takayasu arteritis with clinically significant right upper-extremity arterial involvement. However, the available case documentation did not include a sufficiently detailed record of the patient's subsequent treatment or clinical course. Specifically, the initiation, dosage, and duration of glucocorticoid therapy; the use of conventional or biological immunosuppressive agents; and the administration of antiplatelet or other vascular medications could not be verified.

Similarly, although multimodal vascular imaging demonstrated abnormalities involving the right subclavian and axillary arterial circulation (Figures 1(A)-(C)), the available records did not establish whether endovascular revascularization, open surgical intervention, or conservative vascular management was undertaken. No documented information was available regarding procedural outcomes or vascular complications.

Longitudinal clinical outcomes were also unavailable, including changes in upper-extremity claudication, pulse examination, inter-arm blood pressure discrepancy, inflammatory markers, and follow-up vascular imaging. Consequently, treatment response, disease remission, progression of arterial lesions, and recurrence of ischemic symptoms could not be assessed.

These limitations restrict the interpretation of the patient's clinical course and prevent conclusions regarding therapeutic effectiveness or prognosis in this individual case. Accordingly, the present report emphasizes the diagnostic contribution of multimodal vascular imaging and histopathologic assessment rather than case-specific treatment outcomes. General therapeutic strategies and principles of longitudinal vascular surveillance are addressed separately in the accompanying narrative review.



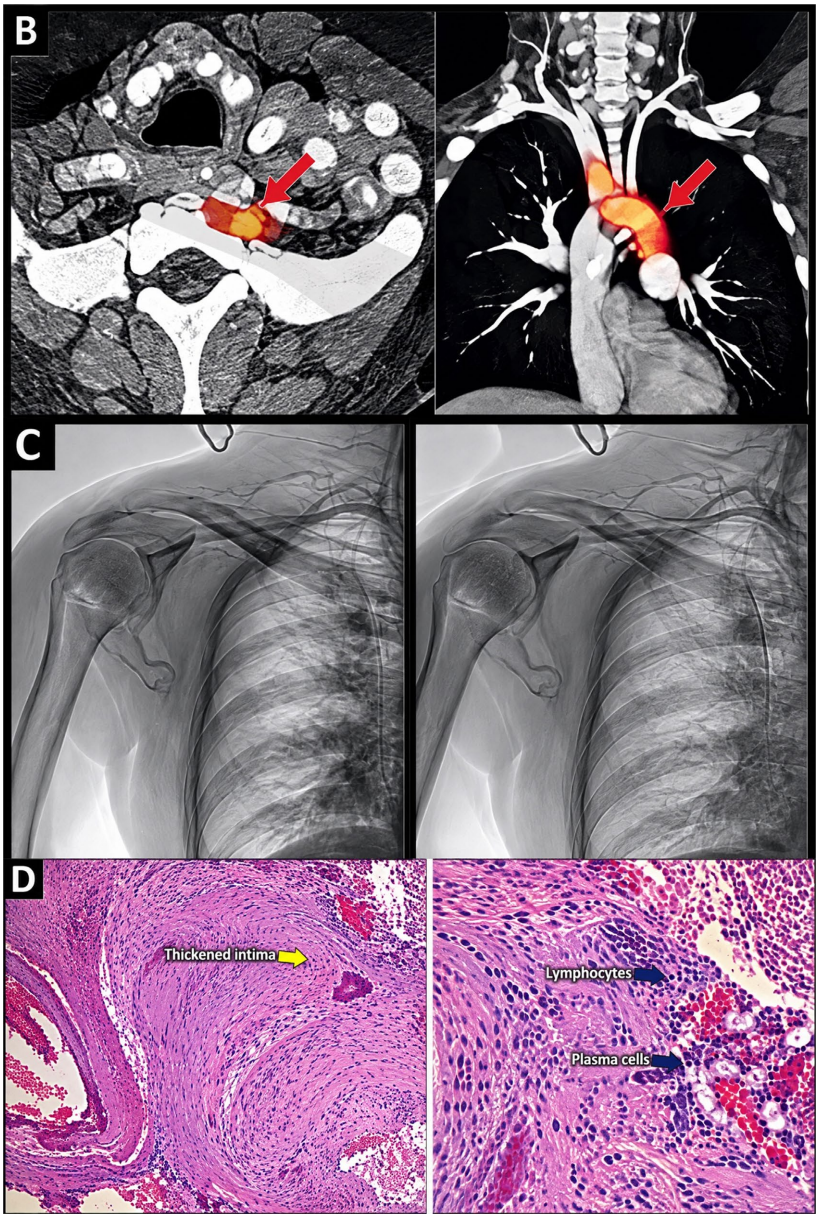


Figure 1. Multimodal imaging and histopathologic findings in Takayasu arteritis. (A) Grayscale and color Doppler ultrasonography of the right subclavian and axillary arterial territories demonstrating abnormalities within the affected upper-extremity circulation. (B) Computed tomography angiography with axial and coronal reconstructions demonstrating vascular abnormalities involving the aortic arch and proximal branch-vessel territory (red arrows). (C) Conventional catheter angiography demonstrating abnormal luminal morphology involving the arterial circulation of the affected upper extremity. (D) Hematoxylin and eosin-stained vascular tissue demonstrating marked intimal thickening (yellow arrow) and inflammatory cellular infiltration composed of lymphocytes and plasma cells (blue arrows).

4. Discussion

4.1. Clinical Spectrum and Vascular Involvement

Takayasu arteritis (TAK) is characterized by a heterogeneous clinical course in

which inflammatory injury of the aorta and its major branches progressively translates into structural vascular damage. The resulting stenosis, occlusion, aneurysmal change, and collateralization explain much of the clinical burden of the disease. Importantly, the vascular phenotype may dominate the presentation, particularly when diagnosis occurs after significant arterial remodeling has already developed. Long-term cohorts demonstrate that vascular complications and relapse remain frequent despite treatment, emphasizing that TAK represents both an inflammatory disorder and a chronic vascular disease with cumulative structural consequences [9] [10].

Upper-extremity ischemia is particularly relevant in this context because the subclavian arteries are among the vessels frequently affected by TAK. In a longitudinal North American cohort, subclavian involvement was documented in 65% of patients, while pulse asymmetry, inter-arm blood-pressure differences, and arterial bruits were among the most common clinical manifestations. These findings illustrate how proximal arterial involvement may ultimately manifest as limb claudication, pulse abnormalities, or clinically significant ischemia [11]. In the present case, upper-extremity ischemia associated with subclavian involvement represented the principal clinical manifestation, and the vascular abnormality could be demonstrated sequentially by Doppler ultrasonography, CTA, and conventional angiography (**Figures 1(A)-(C)**).

4.2. Multimodal Vascular Imaging

The imaging assessment of TAK has evolved substantially from an approach centered predominantly on conventional angiography toward noninvasive techniques capable of evaluating both the arterial lumen and the vessel wall. This distinction is important because luminal abnormalities represent only one component of the disease. Ultrasonography, CTA, MRA, and metabolic imaging provide different and potentially complementary information regarding vascular morphology, distribution, hemodynamic consequences, and inflammatory activity.

Evidence from systematic reviews supports the complementary role of noninvasive imaging in the assessment of Takayasu arteritis. Dua *et al.* evaluated the diagnostic performance of imaging modalities and reported pooled sensitivity and specificity estimates of 72% and 69%, respectively, for the selected noninvasive imaging studies included in their analysis. However, the certainty of evidence was very low, highlighting methodological limitations and the need to interpret imaging findings within the broader clinical context [12].

These characteristics are reflected in the present case. Doppler ultrasonography demonstrated abnormalities involving the right subclavian and axillary circulation (**Figure 1(A)**), whereas CTA provided a broader anatomical assessment of the aortic arch and proximal branch-vessel territory (**Figure 1(B)**). Conventional angiography subsequently demonstrated the corresponding luminal abnormalities (**Figure 1(C)**). Rather than being redundant, these examinations represent different perspectives on the same vascular process. Their principal findings, clin-

ical applications, and limitations in TAK are summarized in **Table 1**.

Table 1 therefore highlights an important practical consideration: selection of imaging modality should depend on the clinical question. Ultrasonography offers accessible evaluation of superficial vessels and their hemodynamic consequences but is operator-dependent and limited for deeper vascular territories. CTA provides high-resolution anatomical characterization of stenosis, occlusion, aneurysmal changes, wall abnormalities, and collateral circulation, at the expense of radiation exposure and iodinated contrast. MRI/MRA permits combined evaluation of the vessel wall and lumen without ionizing radiation, whereas conventional angiography provides detailed luminal mapping and remains particularly relevant when an endovascular procedure is contemplated.

4.3. Metabolic Imaging and Assessment of Disease Activity

A persistent challenge in TAK is differentiating ongoing arterial inflammation from established vascular damage. Structural stenosis or occlusion may remain after inflammatory activity has subsided, while clinical symptoms and conventional inflammatory markers do not invariably reflect the inflammatory state of the arterial wall. Consequently, anatomical imaging and assessment of disease activity should not be considered interchangeable.

^{18}F-FDG PET/CT has emerged as an additional modality for this purpose because increased glucose metabolism can identify metabolically active arterial segments. However, its performance is not absolute. A meta-analysis evaluating FDG-PET for TAK activity reported pooled sensitivity of approximately 70% and specificity of 77%, supporting an adjunctive rather than standalone role for metabolic imaging [13]. Subsequent longitudinal data have similarly demonstrated only moderate discrimination between clinically active disease and remission, and PET-derived vascular activity scores have not consistently predicted subsequent relapse [14].

Accordingly, PET/CT should be interpreted alongside symptoms, inflammatory markers, previous treatment, and structural vascular imaging. This principle also explains why the modalities summarized in **Table 1** should be regarded as complementary rather than competing diagnostic tools. Although PET/CT was not part of the multimodal assessment illustrated in **Figure 1**, its inclusion in the diagnostic framework is relevant because it adds metabolic information that cannot be obtained directly from Doppler, CTA, MRA, or catheter angiography.

4.3.1. Histopathologic Correlation

Histopathologic examination provides a distinct perspective on TAK by demonstrating the arterial-wall consequences of the inflammatory process. The disease is characterized by chronic inflammation and progressive vascular remodeling, with subsequent intimal proliferation and fibrosis contributing to luminal narrowing and eventually to ischemic manifestations. The relative prominence of active inflammation and chronic remodeling varies according to disease duration, vascular territory, and the stage at which tissue is obtained.

The histopathologic findings in the present case are therefore particularly valuable when considered together with the vascular imaging. **Figure 1(D)** demonstrates marked intimal thickening accompanied by inflammatory infiltration containing lymphocytes and plasma cells. These findings provide a tissue correlate for the abnormalities observed radiologically and support the presence of an inflammatory arteriopathy rather than an exclusively mechanical or degenerative stenotic process.

The relationship among the panels in **Figure 1** is consequently central to this case. **Figure 1(A)** depicts the peripheral vascular and flow abnormality; **Figure 1(B)** demonstrates proximal large-vessel involvement on cross-sectional angiographic imaging; **Figure 1(C)** depicts the resulting luminal abnormality; and **Figure 1(D)** demonstrates the underlying arterial-wall remodeling and inflammatory infiltrate. This imaging-pathology correlation is one of the principal strengths of the report because it illustrates different manifestations of the same disease process at hemodynamic, anatomical, luminal, and microscopic levels.

4.3.2. Therapeutic Implications

Treatment of TAK has two related but distinct objectives: suppression of vascular inflammation and management of established vascular complications. Contemporary therapeutic recommendations support glucocorticoids for induction of remission together with a glucocorticoid-sparing immunosuppressive agent in TAK. The evidence base remains substantially weaker than that available for many other rheumatic diseases, with much of the therapeutic literature consisting of observational cohorts and uncontrolled studies. Conventional immunosuppressive agents and biologic therapies, particularly targeting TNF or IL-6 pathways, have therefore been incorporated into treatment strategies for relapsing or refractory disease [15] [16].

This distinction between inflammatory activity and irreversible vascular damage becomes particularly important in patients presenting with clinically significant ischemia, as in the present case. Immunosuppressive therapy can control the inflammatory component of TAK but cannot necessarily reverse an established fibrotic stenosis or chronic occlusion. Patients with severe symptomatic ischemia, critical arterial stenosis, renovascular hypertension, cerebral ischemia, coronary involvement, or aneurysmal disease may therefore require vascular intervention in addition to medical therapy.

Both endovascular and open surgical approaches have been used, although their selection depends on lesion location, morphology, extent, clinical urgency, and disease status. A systematic review and meta-analysis including 770 patients and 1363 vascular lesions found restenosis to be more frequent after endovascular treatment than after open surgical revascularization, although outcomes varied according to the vascular territory and procedural complication profiles differed between strategies [17]. More recent multicenter evidence continues to demonstrate that restenosis remains clinically relevant after vascular intervention, reinforcing the need for continued imaging surveillance rather than considering revascularization curative of the underlying disease [18].

Follow-up and vascular damage

Long-term follow-up in TAK must account for both recurrent inflammatory activity and progression of pre-existing vascular damage. This distinction is particularly important because new stenoses, arterial occlusion, aneurysmal transformation, or worsening ischemic manifestations may develop over time even in patients who have previously achieved clinical improvement.

Longitudinal studies have demonstrated that patients with Takayasu arteritis may accumulate irreversible vascular damage despite clinical management. Kermani *et al.* evaluated disease-related damage in patients with Takayasu arteritis, highlighting the importance of assessing cumulative vascular complications in addition to inflammatory activity [19]. Similarly, a prospective cohort of 549 patients demonstrated relapse in approximately half of patients during a median follow-up of 44 months, with previous relapse, extensive arterial involvement, aortic arch or ascending aortic involvement, aneurysms, and elevated inflammatory parameters associated with a greater risk of recurrence [20].

These observations reinforce the need for longitudinal clinical and vascular assessment. Imaging during follow-up should be interpreted in comparison with previous examinations, with attention not only to new lesions but also to progression of known stenoses, development of collateral circulation, aneurysmal transformation, and changes in vessel-wall characteristics. The multimodal framework summarized in **Table 1** therefore extends beyond initial diagnosis and may also guide selection of the most appropriate modality according to the clinical question encountered during surveillance.

Relevance of the present case

The principal value of this case lies in the direct correlation between a clinically significant ischemic presentation and abnormalities demonstrated through several levels of vascular assessment. Upper-extremity ischemia led to identification of subclavian arterial involvement, while Doppler ultrasonography, CTA, and conventional angiography demonstrated different components of the vascular abnormality (**Figures 1(A)-(C)**). Histopathologic examination subsequently demonstrated intimal thickening and lymphoplasmacytic inflammatory infiltration (**Figure 1(D)**), providing a tissue-level correlate for the imaging findings.

This correlation is clinically relevant because TAK cannot be completely characterized by a single marker or imaging modality. Structural vascular abnormalities may represent a combination of active inflammation and accumulated damage, while biochemical inflammatory markers and clinical manifestations may not always parallel arterial-wall activity. The present case therefore illustrates how integration of clinical findings with complementary imaging techniques and, when available, histopathologic information can provide a more comprehensive characterization of disease.

Taken together, **Figure 1** and **Table 1** emphasize the central concept of this report: multimodal evaluation permits assessment of TAK across different dimensions of the disease process, from altered blood flow and large-vessel anatomy

Table 1. Multimodal imaging modalities in Takayasu arteritis.

Imaging modality	*Main findings in TAK*	*Main clinical role*	*Key limitation*
Doppler ultrasonography	Concentric wall thickening, stenosis, altered flow velocities and waveforms	Assessment of accessible arteries and hemodynamic consequences; follow-up	Operator-dependent; limited evaluation of deep vessels
CTA	Mural thickening, enhancement, stenosis, occlusion, aneurysm and collateral circulation	Detailed anatomical assessment of the aorta and its major branches	Ionizing radiation and iodinated contrast
MRI/MRA	Wall thickening, mural edema/enhancement and luminal abnormalities	Evaluation of vessel wall and lumen; useful for serial assessment	Availability, acquisition time and technical variability
18F-FDG PET/CT	Increased metabolic activity along the arterial wall	Assessment of suspected inflammatory activity and disease extent	Limited anatomical detail; FDG uptake is not entirely specific for active vasculitis
Conventional angiography	Stenosis, occlusion, dilatation and collateral vessels	Detailed luminal mapping and guidance of endovascular procedures	Invasive; limited direct assessment of vessel-wall inflammation

Summary of the main vascular findings, clinical applications, and key limitations of Doppler ultrasonography, computed tomography angiography (CTA), magnetic resonance imaging/angiography (MRI/MRA), ^{18}F-fluorodeoxyglucose positron emission tomography/computed tomography (^{18}F-FDG PET/CT), and conventional angiography in the evaluation of Takayasu arteritis.

to luminal damage and arterial-wall pathology. Early recognition of this spectrum is particularly important in young patients presenting with otherwise unexplained limb ischemia, in whom identification of a large-vessel inflammatory process may permit timely treatment and surveillance before additional irreversible vascular complications develop.

5. Conclusions

Takayasu arteritis remains a challenging large-vessel vasculitis because clinical manifestations may become evident only after substantial vascular involvement has developed. This case demonstrates the complementary role of multimodal assessment in characterizing the disease, with Doppler ultrasonography, computed tomography angiography, and conventional angiography documenting different aspects of the vascular abnormalities responsible for upper-extremity ischemia, while histopathologic examination demonstrated associated intimal remodeling and inflammatory infiltration.

Integration of clinical findings with structural, hemodynamic, and histopathologic information can provide a more comprehensive assessment than any single diagnostic modality alone. Early recognition of Takayasu arteritis should therefore be considered in young patients presenting with otherwise unexplained large-vessel stenosis or limb ischemia. Accurate characterization of vascular involvement is essential not only for establishing the diagnosis but also for distinguishing inflammatory activity from established vascular damage, guiding therapeutic decisions, and planning long-term surveillance.

Declaration of Generative AI and AI-Assisted Technologies

During the preparation of this manuscript, the authors used generative artificial intelligence tools to assist with language editing, manuscript organization, literature searching, and verification of bibliographic and formatting elements. These tools were not used to generate, modify, or interpret clinical data, diagnostic findings, or study results. All AI-assisted content, references, and statements were critically reviewed and verified by the authors, who take full responsibility for the accuracy, integrity, and final content of the manuscript.

Ethics Statement

This case report was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. Written informed consent was obtained from the patient for the publication of the clinical information and accompanying images. All patient-identifying information was removed or anonymized to protect confidentiality and privacy. Institutional ethics committee approval was not required for this single-patient case report in accordance with local institutional policies.

Author Contributions

Conceptualization, G.R.-G. and E.C.-B.; methodology, K.P.-P. and W.M.-S.; validation, J.A.-O. and I.A.-O.; formal analysis, J.J.S.-R. and F.R.-A.; investigation, G.R.-G., E.C.-B. and S.G.-R.; resources, A.S.-B. and A.Á.-A.; data curation, K.P.-P. and J.J.S.-R.; writing—original draft preparation, G.R.-G., W.M.-S. and F.R.-A.; writing—review and editing, E.C.-B., I.A.-O., A.S.-B. and S.G.-R.; visualization, J.A.-O. and A.Á.-A.; supervision, S.G.-R. and A.Á.-A.; project administration, G.R.-G. and E.C.-B.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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