Research Article ISSN 2771-5906

Trends in Internal Medicine

Large-Vessel Inflammatory Disease: A Clinical Approach to Diagnosis and Vascular Involvement

María Camila Patiño-Pérez[1], Joyce Guerra-Cabarcas[2], Jesús Anaya-Amador[1], Natalia Nicole Meza-Prada[1], Valentina Canabal-Anchique[1], Maria Isabel Nuñez-Donado[1], Valentina Llerena-Montoya[1], Jesus Bermejo-Gomez[3], Mathius Uscategui-Muñoz[4], Daniela Gómez-Pacheco[5], and Martin Ramos-Perez[6]

[1]*General Medicine, Universidad del Sinú, Cartagena, Colombia.*

[2]*General Medicine, Universidad del Cartagena, Cartagena, Colombia.*

[3]*General Medicine, Corporación Universitaria Rafael Núñez, Cartagena, Colombia.*

[4]*General Medicine, Universidad del Magdalena, Santa Marta, Colombia.*

[5]*General Medicine, Universidad Metropolitana, Barranquilla, Colombia.*

[6]*General Medicine, Universidad Cooperativa de Colombia, Bogotá, Colombia.*

***Correspondence:**

Joyce Guerra Cabarcas, General Medicine, Universidad del Cartagena, Cartagena, Colombia.
ORCID ID: https://orcid.org/0009-0007-2178-7542.

María Camila Patiño Pérez, General Medicine, Universidad del Sinú, Cartagena, Colombia.
ORCID ID: https://orcid.org/0009-0008-3407-0556.

Received: 03 May 2026; **Accepted:** 01 Jun 2026; **Published:** 12 Jun 2026

Citation: María Camila Patiño-Pérez, Joyce Guerra-Cabarcas, Jesús Anaya-Amador, et al. Large-Vessel Inflammatory Disease: A Clinical Approach to Diagnosis and Vascular Involvement. Trends Int Med. 2026; 6(1): 1-5.

ABSTRACT

Background: *Large-vessel inflammatory diseases comprise a group of chronic vasculitides characterized by immune-mediated inflammation of the aorta and its primary branches. These conditions, including Takayasu arteritis, are associated with significant morbidity due to progressive vascular remodeling leading to stenosis, occlusion, or aneurysm formation. Early clinical manifestations are frequently nonspecific and dominated by constitutional symptoms, which often results in delayed recognition and diagnosis at more advanced stages of vascular involvement.*

Objective: *To describe a representative case of large-vessel vasculitis and to provide a clinically oriented discussion of the diagnostic approach, patterns of vascular involvement, and underlying pathophysiological mechanisms relevant to internal medicine practice.*

Methods: *We present a case of a young adult with clinical features suggestive of arterial insufficiency. A focused narrative review of the literature was conducted to contextualize the clinical findings, emphasizing current concepts in the diagnosis and evaluation of large-vessel inflammatory diseases, particularly Takayasu arteritis.*

Results: *The case demonstrates the heterogeneity of clinical presentation, ranging from systemic inflammatory symptoms to localized vascular compromise. Diagnostic evaluation revealed involvement of major arterial territories, supporting the diagnosis of large-vessel vasculitis. The integration of clinical assessment, laboratory markers of inflammation, and complementary diagnostic studies allowed for accurate characterization of disease extent and activity.*

Conclusion: *Large-vessel inflammatory diseases remain a diagnostic challenge due to their variable and often subtle presentation. A systematic, clinically driven approach is essential to facilitate early recognition and prevent irreversible vascular damage. Takayasu arteritis serves as a prototypical entity within this spectrum, illustrating the importance of integrating pathophysiological understanding with bedside clinical evaluation.*

Keywords
Takayasu arteritis, Large-vessel vasculitis, Vasculitis, Aortic inflammation, Arterial stenosis, Vascular remodeling, Diagnostic evaluation.

Introduction

Large-vessel inflammatory diseases comprise a group of chronic vasculitides characterized by immune-mediated inflammation of the aorta and its major branches, leading to progressive structural damage and clinically significant vascular complications [1]. Within this spectrum, Takayasu arteritis represents a prototypical entity, predominantly affecting young individuals and associated with substantial morbidity due to delayed diagnosis and irreversible vascular remodeling [2].

One of the major challenges in clinical practice is the early recognition of these conditions. Initial manifestations are frequently nonspecific and include constitutional symptoms such as fever, malaise, and weight loss, which may precede overt vascular involvement by months or even years [3]. As a result, diagnosis is often established only after the development of advanced vascular lesions, including stenosis, occlusion, or aneurysm formation, which are responsible for ischemic complications and long-term disability [4].

The underlying pathophysiology involves a complex interplay between innate and adaptive immune responses, resulting in granulomatous inflammation of the arterial wall. This inflammatory process leads to intimal hyperplasia, medial destruction, and adventitial fibrosis, ultimately causing luminal narrowing and altered vascular compliance [5]. These mechanisms explain the heterogeneous clinical presentation, which may range from systemic inflammatory symptoms to localized manifestations such as limb claudication, pulse deficits, blood pressure discrepancies, and organ ischemia [6].

From a diagnostic standpoint, large-vessel vasculitis requires a structured and clinically driven approach. Laboratory markers of inflammation, including erythrocyte sedimentation rate and C-reactive protein, may support the presence of systemic inflammation but lack specificity and may not accurately reflect disease activity [7]. Therefore, diagnosis relies on the integration of clinical findings, careful vascular examination, and appropriate use of complementary studies, alongside the exclusion of alternative etiologies such as atherosclerosis, infectious arteritis, and other systemic autoimmune diseases [8].

Recent advances in classification criteria and clinical guidelines have improved the recognition and standardization of large-vessel vasculitis, emphasizing the importance of early diagnosis and timely initiation of therapy to prevent irreversible vascular damage [9]. In this context, case-based analyses remain highly valuable, as they illustrate real-world diagnostic challenges and highlight the importance of clinical reasoning in the evaluation of complex inflammatory vascular diseases.

The present report describes a representative case of large-vessel inflammatory disease, using Takayasu arteritis as a model to illustrate the clinical approach to diagnosis, patterns of vascular involvement, and the integration of pathophysiological concepts into internal medicine practice.

Case Presentation and Clinical Correlation

Initial Clinical Presentation and Diagnostic Suspicion

A young adult patient presented with a subacute history of upper extremity fatigue and exertional discomfort, progressively limiting functional capacity. The clinical picture was preceded by nonspecific constitutional symptoms, including asthenia and malaise, without an identifiable etiology during early evaluation. This pattern reflects the initial inflammatory phase described in large-vessel vasculitis, in which systemic symptoms often precede overt vascular manifestations and contribute to diagnostic delay [10].

On physical examination, asymmetry of peripheral pulses and a significant inter-arm blood pressure discrepancy were documented. These findings are highly suggestive of large-vessel involvement and remain among the most valuable bedside clues for early recognition. In the appropriate clinical context, such findings should prompt immediate evaluation for inflammatory vascular disease, particularly when alternative causes are not evident [11].

Patterns of Vascular Involvement in Large-Vessel Disease

Large-vessel inflammatory diseases exhibit characteristic patterns of arterial involvement, most frequently affecting the aortic arch and its major branches. In Takayasu arteritis, the subclavian arteries are among the most commonly involved territories, often leading to upper limb ischemia and claudication [12]. The distribution of vascular lesions is not random but reflects underlying immunopathological mechanisms that preferentially target elastic arteries.

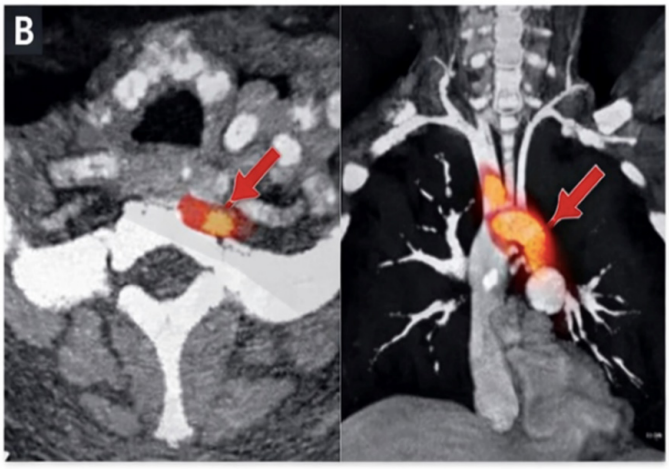

Figure 1: Contrast-enhanced computed tomography angiography demonstrating focal inflammatory involvement of the left subclavian artery. Axial (left) and coronal (right) views reveal circumferential wall thickening with associated luminal narrowing (arrows), consistent with

large-vessel vasculitis. These findings reflect active inflammatory changes and early structural remodeling characteristic of Takayasu arteritis

In this case, imaging studies confirmed involvement of the subclavian artery and its branches, consistent with the classical distribution described in the literature. The presence of focal stenosis and compensatory collateral circulation further suggests a chronic inflammatory process with progressive vascular remodeling [13].

Role of Imaging in Structural and Functional Assessment

Although not the central focus of this report, imaging plays a complementary role in confirming clinical suspicion and defining the extent of vascular involvement. Doppler ultrasonography (Figure 1) demonstrated concentric arterial wall thickening and luminal narrowing, findings consistent with inflammatory involvement of large vessels. These early changes reflect active inflammation and are particularly useful in accessible vascular territories [14].

Computed tomography angiography (Figure 2) provided detailed anatomical characterization, revealing focal stenotic segments and vascular remodeling. This modality allows precise evaluation of luminal changes and is essential for assessing disease extent and planning management strategies [15].

These findings illustrate the dynamic nature of vascular remodeling in response to prolonged inflammation and ischemia [16].

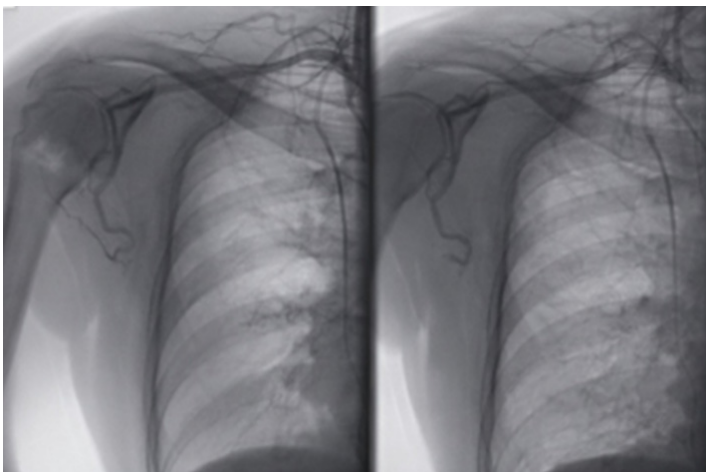

Figure 2: Conventional angiography of the aortic arch and its branches demonstrating significant luminal narrowing of the left subclavian artery with irregular vessel contour and reduced distal flow. Collateral circulation is evident, reflecting chronic vascular remodeling secondary to inflammatory large-vessel disease. These findings are characteristic of advanced-stage involvement in Takayasu arteritis.

Pathophysiological Correlation

The vascular findings observed in this case are the result of a complex inflammatory process involving granulomatous infiltration of the arterial wall. Activation of dendritic cells and T lymphocytes within the vessel wall leads to cytokine release, macrophage recruitment, and subsequent tissue damage. This cascade results in disruption of the elastic lamina, smooth muscle cell proliferation, and progressive intimal hyperplasia [17].

These mechanisms ultimately lead to luminal narrowing and impaired blood flow, explaining the clinical manifestations of ischemia. In advanced stages, fibrosis and vascular remodeling contribute to irreversible structural damage, emphasizing the importance of early recognition and intervention [18].

Clinical Reasoning and Differential Diagnosis

From an internal medicine perspective, the diagnosis of large-vessel inflammatory disease requires careful exclusion of alternative etiologies. Atherosclerotic disease, infectious arteritis, and other systemic vasculitides may present with overlapping features and must be considered in the differential diagnosis [19].

In this patient, the combination of young age, systemic inflammatory symptoms, characteristic vascular findings, and absence of traditional cardiovascular risk factors strongly supported an inflammatory etiology. The pattern of arterial involvement further reinforced the suspicion of Takayasu arteritis as the most likely diagnosis within the spectrum of large-vessel vasculitis [20].

Clinical Implications and Diagnostic Approach

This case highlights the importance of a structured and clinically driven diagnostic approach. Early recognition depends on maintaining a high index of suspicion in patients with unexplained systemic symptoms and subtle vascular findings. Physical examination remains a cornerstone of diagnosis, particularly in identifying pulse deficits and blood pressure discrepancies.

The integration of clinical, laboratory, and imaging data is essential to establish the diagnosis and assess disease extent. Delayed diagnosis may result in irreversible vascular damage and increased morbidity, underscoring the need for heightened clinical awareness in internal medicine practice [21].

Discussion

Large-vessel inflammatory diseases represent a clinically complex group of conditions in which delayed recognition remains a major determinant of morbidity. This case illustrates the typical diagnostic challenge, where early nonspecific systemic symptoms precede overt vascular involvement, often leading to under-recognition during initial stages. Such delay is well documented and contributes significantly to the development of irreversible arterial damage [22].

A key aspect highlighted by this case is the importance of bedside clinical evaluation. Despite advances in diagnostic modalities, physical examination findings such as pulse asymmetry, inter-arm blood pressure differences, and vascular bruits remain fundamental in raising early suspicion. In many cases, these signs precede imaging confirmation and should prompt further targeted evaluation [23].

Table 1: Clinical approach to suspected large-vessel inflammatory disease in internal medicine.

Step	Clinical Domain	Key Findings	Clinical Implication
1	Systemic symptoms	Fever, fatigue, weight loss, elevated inflammatory markers	Consider inflammatory etiology; early phase of disease
2	Vascular symptoms	Limb claudication, dizziness, visual symptoms, chest pain	Suggests evolving arterial involvement
3	Physical examination	Pulse asymmetry, bruits, inter-arm blood pressure difference	High suspicion for large-vessel disease
4	Initial laboratory evaluation	Elevated ESR/CRP, nonspecific inflammatory profile	Supports systemic inflammation but not diagnostic
5	Vascular assessment	Evidence of arterial insufficiency or asymmetry	Indicates need for targeted imaging
6	Complementary studies	Structural vascular abnormalities and disease extent	Confirms diagnosis and defines severity
7	Differential diagnosis	Atherosclerosis, infectious arteritis, other vasculitides	Essential to avoid misclassification
8	Clinical integration	Correlation of all findings	Establishes diagnosis and guides management

Table 1 Stepwise clinical approach to the evaluation of suspected large-vessel inflammatory disease. The diagnostic process integrates systemic symptoms, vascular findings, laboratory data, and complementary studies, emphasizing the importance of clinical reasoning in early recognition and accurate diagnosis.

From a pathophysiological standpoint, large-vessel vasculitis is driven by immune-mediated inflammation targeting elastic arteries. The resulting granulomatous process leads to progressive vascular remodeling, characterized by intimal proliferation and luminal narrowing. These mechanisms explain the transition from a systemic inflammatory phase to a structurally defined vascular disease, as demonstrated in this patient [24].

The pattern of vascular involvement observed, particularly affecting the subclavian artery, aligns with the classical distribution described in Takayasu arteritis. This predilection for the aortic arch and its branches reflects both anatomical and immunological factors that influence disease localization. The presence of collateral circulation further indicates chronicity and adaptive vascular responses to sustained ischemia [25].

Although imaging studies are essential for confirming diagnosis and assessing disease extent, they should be interpreted within a clinical framework. The integration of imaging findings with clinical presentation and laboratory data allows for a more accurate characterization of disease activity and severity. Importantly, reliance on imaging alone without clinical correlation may lead to misinterpretation, particularly in distinguishing inflammatory disease from atherosclerotic processes [26].

This case also underscores the importance of considering large-vessel inflammatory disease in younger patients without traditional cardiovascular risk factors (see Table 1). Early identification is critical. Early identification is critical, as timely initiation of immunosuppressive therapy has been associated with improved outcomes and reduced progression of vascular damage [27].

Takayasu arteritis serves as a paradigmatic example within this spectrum, illustrating the interplay between systemic inflammation and localized vascular injury. However, the principles derived from its evaluation are broadly applicable to other forms of large-vessel vasculitis, reinforcing the need for a structured and clinically driven diagnostic approach in internal medicine [28].

Conclusion

Large-vessel inflammatory diseases remain a diagnostic challenge due to their heterogeneous and often subtle clinical presentation. This case highlights the importance of early clinical suspicion, careful vascular examination, and integration of complementary studies in establishing the diagnosis. A structured, clinically oriented approach is essential to prevent delayed recognition and irreversible vascular damage. Takayasu arteritis exemplifies the pathophysiological and clinical complexity of this group of diseases, emphasizing the need for heightened awareness in internal medicine practice.

References

1. Jennette JC, Falk RJ, Bacon PA, et al. 2012 Revised International Chapel Hill Consensus Conference Nomenclature of Vasculitides. Arthritis Rheum. 2013; 65: 1-11.
2. Keser G, Aksu K, Direskeneli H. Takayasu arteritis: an update. Turk J Med Sci. 2018; 48: 681-697.
3. Kerr GS, Hallahan CW, Giordano J, et al. Takayasu Arteritis. Ann Intern Med. 1994; 120: 919-929.
4. Numano F, Okawara M, Inomata H, et al. Takayasu's Arteritis. Lancet. 2000; 356: 1023-1025.
5. Weyand CM, Goronzy JJ. Medium- and Large-Vessel Vasculitis. N Engl J Med. 2003; 349: 160-169.
6. Arend WP, Michel BA, Bloch DA, et al. The American College of Rheumatology 1990 criteria for the classification of Takayasu arteritis. Arthritis Rheum. 1990; 33: 1129-1134.
7. Direskeneli H, Aydin SZ, Merkel PA. Assessment of disease activity and progression in Takayasu arteritis. Clin Exp Rheumatol. 2011; 29: S86-S91.
8. Hellmich B, Agueda A, Monti S, et al. 2018 Update of the EULAR recommendations for the management of large vessel vasculitides. Ann Rheum Dis. 2020; 79: 19-30.
9. Grayson PC, Ponte C, Suppiah R, et al. 2022 American College of Rheumatology/EULAR classification criteria for Takayasu arteritis. Ann Rheum Dis. 2022; 81: 1654-1660.
10. Hoffman GS, Ahmed AE. Surrogate markers of disease activity in Takayasu arteritis. A preliminary report from The International Network for the Study of the Systemic Vasculitides (INSSYS). Int J Cardiol.1998; 66: S191-S194.
11. Clark TM, Maksimowicz-McKinnon K, Hoffman GS. Takayasu arteritis: clinical features and diagnosis. UpToDate (referenciado en revisiones primarias; equivalente en literatura clínica: Hoffman GS et al.)

12. Hata A, Noda M, Moriwaki R, et al. Angiographic findings of Takayasu arteritis: new classification. Int J Cardiol. 1996; 54: S155-S163.
13. Park MC, Lee SW, Park YB, et al. Clinical characteristics and outcomes of Takayasu arteritis: analysis of 108 patients using standardized criteria for diagnosis, activity assessment, and angiographic classification. Scand J Rheumatol. 2005; 34: 284-292.
14. Schmidt WA. Ultrasound in the diagnosis and management of giant cell arteritis. Rheumatology. 2018; 57: ii22-ii31.
15. Papa M, De Cobelli F, Baldissera E, et al. Takayasu Arteritis: Intravascular contrast medium for MR angiography in the evaluation of disease activity. AJR Am J Roentgenol. 2012; 198: 279-284.
16. Maksimowicz-McKinnon K, Hoffman GS. Imaging in Takayasu Arteritis. Curr Opin Rheumatol. 2009; 21: 41-47.
17. Weyand CM, Goronzy JJ. Immune mechanisms in medium and large-vessel vasculitis. Nat Rev Rheumatol. 2013; 9: 731-740.
18. Samson M, Corbera-Bellalta M, Audia S, et al. Recent advances in pathophysiology of large vessel vasculitis. Nat Rev Rheumatol. 2017; 13: 578-592.
19. Salvarani C, Cantini F, Hunder GG. Polymyalgia rheumatica and giant-cell arteritis. Lancet. 2008; 372: 234-245.
20. Tso E, Flamm SD, White RD, et al. Takayasu arteritis: utility of imaging. Circulation. 2002; 105: 2927-2932.
21. Comarmond C, Biard L, Lambert M, et al. Long-term Outcomes and Prognostic Factors of Complications in Takayasu Arteritis A Multicenter Study of 318 Patients. Circulation. 2017; 136: 1114-1122.
22. Kermani TA, Warrington KJ. Advances and challenges in large vessel vasculitis. Ther Adv Musculoskelet Dis. 2011; 3: 145-151.
23. Saadoun D, Lambert M, Mirault T, et al. Retrospective analysis of Takayasu arteritis. Circulation. 2012; 125: 813-819.
24. Weyand CM, Goronzy JJ. Pathogenesis of large vessel vasculitis. Nat Rev Rheumatol. 2013; 9: 731-740.
25. Cong XL, Dai SM, Feng X, et al. Takayasu Arteritis: clinical features and prognosis. Clin Rheumatol. 2010; 29: 1011-1018.
26. Grayson PC, Maksimowicz-McKinnon K, Clark TM, et al. Imaging in large-vessel vasculitis. Rheum Dis Clin North Am. 2016; 42: 33-46.
27. Comarmond C, Biard L, Lambert M, et al. Long-term outcomes in Takayasu arteritis. Circulation. 2017; 136: 1114-1122.
28. Hellmich B, Agueda A, Monti S, et al. EULAR recommendations for large vessel vasculitis. Ann Rheum Dis. 2020; 79: 19-30.