Section: General Medicine

Case Report

MULTIVESSEL TAKAYASU ARTERITIS WITH BILATERAL CAROTID, LEFT SUBCLAVIAN, AND AORTIC INVOLVEMENT ASSOCIATED WITH AN INFRARENAL ABDOMINAL AORTIC ANEURYSM: A CASE REPORT

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ABSTRACT

Background: Takayasu arteritis (TA) is a rare chronic granulomatous large-vessel vasculitis that predominantly affects the aorta and its major branches, especially in young women. Progressive inflammation results in arterial wall thickening, stenosis, occlusion, and less commonly, aneurysm formation. Abdominal aortic aneurysm is an uncommon manifestation and may increase the risk of serious vascular complications.

Case Presentation: A 37-year-old woman presented with progressively worsening bilateral lower limb claudication for eight months, left upper limb claudication for one week, and exertional dyspnea for one year. Clinical examination revealed bilateral carotid bruits, a right subclavian bruit, absent peripheral pulses, and blood pressure recordable only in the right upper limb (110/80 mmHg). Laboratory investigations demonstrated elevated inflammatory markers with an erythrocyte sedimentation rate of 80 mm/hour and C-reactive protein of 12 mg/L, while antinuclear antibody profile and other autoimmune serological tests were negative. Computed tomography aortography showed diffuse circumferential wall thickening involving the thoracic and abdominal aorta, bilateral common carotid artery stenosis, complete proximal occlusion of the left subclavian artery, left vertebral artery occlusion, and a wide-neck saccular infrarenal abdominal aortic aneurysm. Based on the clinical, laboratory, and imaging findings, a diagnosis of extensive multivessel Takayasu arteritis was established. The patient was treated with oral prednisolone (1 mg/kg/day), azathioprine (3 mg/kg/day), and antiplatelet therapy, resulting in significant symptomatic improvement after six months.

Conclusion: This case emphasizes the importance of early recognition of Takayasu arteritis in young women presenting with limb claudication, pulse deficits, and vascular bruits. Comprehensive vascular imaging is essential for detecting extensive arterial involvement and rare complications such as infrarenal abdominal aortic aneurysm. Prompt immunosuppressive therapy may improve clinical outcomes and prevent irreversible vascular damage.

Keywords: Takayasu arteritis; large-vessel vasculitis; abdominal aortic aneurysm; carotid artery stenosis; subclavian artery occlusion; CT angiography; limb claudication; vasculitis.

INTRODUCTION

Takayasu arteritis (TA) is a rare, chronic, idiopathic granulomatous large-vessel vasculitis that primarily affects the aorta and its major branches, resulting in progressive inflammation of the arterial wall with subsequent stenosis, occlusion, dilatation, or aneurysm formation. It predominantly affects women

of reproductive age, with the highest prevalence reported in Asian countries, particularly India, Japan, China, and Southeast Asia. Although the global annual incidence is estimated to be only 1–3 cases per million population, TA constitutes an important cause of large-vessel vasculitis in young adults and is associated with significant morbidity due to delayed diagnosis and irreversible vascular damage.

The pathogenesis of TA is believed to involve a complex interplay between genetic susceptibility, immune dysregulation, and environmental triggers. Granulomatous inflammation affects all layers of the arterial wall, leading to intimal proliferation, medial destruction, adventitial fibrosis, and elastic lamina disruption. These pathological changes ultimately result in vascular stenosis, complete occlusion, arterial wall thickening, or aneurysmal dilatation. While stenotic lesions are the predominant vascular abnormality, aneurysm formation is less common but represents an important complication because of the increased risk of rupture, thrombosis, and distal embolization.[1]

The clinical manifestations of Takayasu arteritis vary according to the stage and extent of vascular involvement. The initial inflammatory phase is often characterized by nonspecific constitutional symptoms such as fever, malaise, weight loss, arthralgia, and fatigue, making early diagnosis challenging. As the disease progresses into the chronic occlusive phase, symptoms arise from ischemia of affected vascular territories and include limb claudication, diminished or absent peripheral pulses, blood pressure discrepancies between limbs, arterial bruits, hypertension secondary to renal artery involvement, cerebrovascular insufficiency, and visual disturbances. Because these manifestations develop gradually and mimic several other vascular disorders, diagnosis is frequently delayed until significant arterial stenosis has occurred.[2]

Diagnosis is based on a combination of clinical findings, elevated inflammatory markers, and vascular imaging. Laboratory investigations typically demonstrate elevated erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP), although these markers are neither sensitive nor specific for disease activity. Autoimmune serological markers are usually negative, helping exclude other connective tissue diseases. Cross-sectional vascular imaging, particularly computed tomography angiography (CTA) and magnetic resonance angiography (MRA), has become indispensable for establishing the diagnosis by demonstrating characteristic circumferential arterial wall thickening, long-segment stenosis, occlusions, collateral vessel formation, and aneurysmal changes. These imaging modalities also facilitate assessment of disease extent, therapeutic planning, and long-term surveillance.[3]

Current management of TA focuses on suppression of vascular inflammation and prevention of irreversible arterial injury. High-dose glucocorticoids remain the cornerstone of initial therapy, while steroid-sparing immunosuppressive agents such as methotrexate, azathioprine, mycophenolate mofetil, cyclophosphamide, or biologic agents are increasingly employed to achieve sustained remission and reduce relapse rates. Antiplatelet therapy is often recommended in patients with significant vascular stenosis to decrease ischemic complications. Surgical or endovascular interventions are reserved for critical stenotic lesions, refractory ischemia, uncontrolled hypertension, or aneurysmal disease, preferably when the inflammatory activity is adequately controlled.[5,6]

Although involvement of the aorta and its major branches is well recognized in Takayasu arteritis, the coexistence of extensive multivessel disease involving bilateral carotid arteries, complete proximal left subclavian artery occlusion, diffuse thoracoabdominal aortic involvement, and an infrarenal abdominal aortic aneurysm is relatively uncommon. Such cases present significant diagnostic and therapeutic challenges because they combine extensive occlusive disease with aneurysmal degeneration, requiring careful clinical assessment and long-term follow-up.

Here, we report the case of a 37-year-old woman presenting with progressive bilateral lower limb claudication, left upper limb claudication, and exertional dyspnea, who was found to have markedly elevated inflammatory markers, absent peripheral pulses, and extensive multivessel Takayasu arteritis on computed tomography aortography, including bilateral carotid artery involvement, complete proximal left subclavian artery occlusion, diffuse thoracoabdominal aortic disease, and a wide-neck infrarenal abdominal aortic aneurysm. This case highlights the importance of early clinical suspicion, comprehensive vascular imaging, and prompt immunosuppressive therapy in preventing irreversible vascular complications in patients with Takayasu arteritis.

Case Presentation

A 37-year-old woman presented to the Department of General Medicine with progressively worsening pain in both lower limbs while walking (intermittent claudication) for the past eight months, associated with left upper limb claudication for one week and exertional dyspnea for one year. She denied any history of fever, weight loss, night sweats, chest pain, abdominal pain, headache, visual disturbances, syncope, skin rash, oral ulcers, Raynaud's phenomenon, or joint swelling. There was no history of diabetes mellitus, hypertension, dyslipidemia, smoking, tuberculosis, or previous vascular interventions. Family history was unremarkable for autoimmune or cardiovascular diseases.

On physical examination, the patient appeared conscious, alert, and afebrile. Bilateral carotid bruits and a right subclavian bruit were audible on

auscultation. Peripheral vascular examination revealed absent or markedly diminished pulses in the left upper limb and both lower limbs. Blood pressure was 110/80 mmHg in the right upper limb, whereas blood pressure could not be recorded in the left upper limb or either lower limb because of absent peripheral pulses. Clinical examination was otherwise unremarkable, with no evidence of heart failure or focal neurological deficits.

Laboratory investigations revealed microcytic hypochromic anemia with hemoglobin of 11.4 g/dL, mean corpuscular volume (MCV) of 69.3 fL, mean corpuscular hemoglobin (MCH) of 20.4 pg, and elevated red cell distribution width (RDW) of 16.9%. The total leukocyte count was 9,500/µL, and platelet count was 4.15×10^5 /µL. Markers of systemic inflammation were elevated, with an erythrocyte sedimentation rate (ESR) of 80 mm/hour and C-reactive protein (CRP) of 12 mg/L. Renal and liver function tests were within normal limits, including serum creatinine 0.72 mg/dL, blood urea nitrogen 11 mg/dL, and normal transaminases. Random plasma glucose was 99 mg/dL. Lipid profile demonstrated total cholesterol 113 mg/dL, LDL cholesterol 76 mg/dL, triglycerides 64 mg/dL, and low HDL cholesterol 27 mg/dL. Urinalysis was unremarkable.

Serological investigations for infectious diseases, including hepatitis B surface antigen (HBsAg), hepatitis C virus (HCV), and human immunodeficiency virus (HIV), were non-reactive. Autoimmune evaluation showed a negative antinuclear antibody (ANA) profile, including antibodies against RNP/Sm, Sm, SS-A (Ro), SS-B (La), Ro-52, Scl-70, PM-Scl, Jo-1, centromere, PCNA, double-stranded DNA, nucleosomes, histones, ribosomal P-protein, and AMA-M2. Antistreptolysin-O (ASO) titre was negative.

Ultrasonography of the abdomen demonstrated a borderline enlarged spleen measuring 13 cm, with otherwise normal liver, gallbladder, pancreas, kidneys, urinary bladder, and uterus. A small simple left ovarian cyst measuring 16 × 14 mm was incidentally noted.

Computed tomography (CT) aortography demonstrated extensive large-vessel involvement characterized by diffuse circumferential wall thickening and calcification involving the aortic arch, thoracic aorta, descending thoracic aorta, suprarenal and infrarenal abdominal aorta, producing varying degrees of luminal narrowing. The left subclavian artery showed complete proximal occlusion over an approximately 18-mm segment with distal collateral reformation. The left vertebral artery was occluded at its proximal segment and was hypoplastic distally, while the right vertebral artery was normal. Both common carotid arteries exhibited diffuse circumferential wall thickening and luminal narrowing. A focal occlusion was noted in the distal left common carotid artery with distal collateral formation. The proximal left internal carotid artery demonstrated a short-segment occlusion, whereas the right internal carotid artery showed moderate luminal narrowing. Mild narrowing of the brachiocephalic artery and celiac trunk was also present.

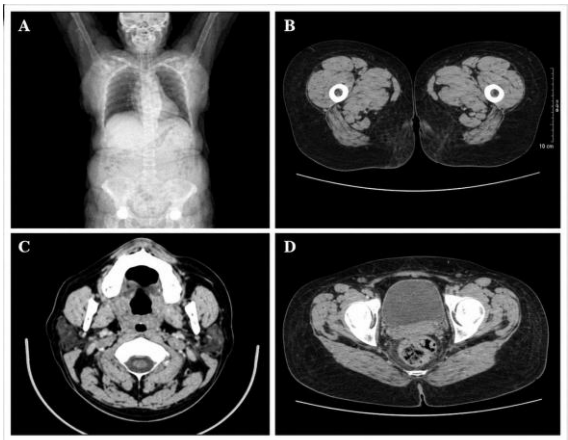

Figure 1. ^{18}F-FDG PET/CT scan of the patient. **(A)** Maximum intensity projection image showing physiologic tracer uptake in the brain, myocardium, liver, kidneys, and urinary bladder. **(B)** Axial CT image of the proximal thighs showing symmetrical muscles with no focal lesion. **(C)** Axial CT image of the neck showing no abnormal mass or lymphadenopathy. **(D)** Axial CT image of the pelvis showing no abnormal lesion.

The descending thoracic and abdominal aorta demonstrated diffuse inflammatory wall thickening with moderate luminal narrowing. A wide-neck saccular infrarenal abdominal aortic aneurysm measuring approximately 9 × 5.8 mm with a neck measuring 9.7 mm was identified arising from the left anterolateral aspect of the infrarenal abdominal aorta at the L4 vertebral level. Circumferential wall thickening with moderate luminal narrowing was also noted in the left common femoral artery. In addition, a 24 × 21 mm enhancing lesion arising from the inferior pole of the right thyroid lobe, suggestive of a thyroid neoplasm requiring further evaluation, and a 7 × 7 mm left renal angiomyolipoma were incidentally detected.

Based on the patient's age, characteristic clinical findings of limb claudication, absent peripheral pulses, vascular bruits, elevated inflammatory markers, and CT angiographic evidence of diffuse large-vessel arteritis involving the aorta and its major branches with associated infrarenal abdominal aortic aneurysm, a diagnosis of Takayasu arteritis with extensive multivessel involvement was established. The disease fulfilled the 1990 American College of Rheumatology (ACR) classification criteria for Takayasu arteritis.

The patient was initiated on oral prednisolone at 1 mg/kg/day, azathioprine at 3 mg/kg/day as a steroid-sparing immunosuppressive agent, and antiplatelet therapy. She was advised regular clinical and radiological follow-up to monitor disease activity and progression of the abdominal aortic aneurysm. At six months of follow-up, she reported significant improvement in lower limb claudication and exercise tolerance, with reduction in inflammatory markers and no evidence of new vascular complications. Continued long-term surveillance was planned owing

to the extensive arterial involvement and presence of the infrarenal abdominal aortic aneurysm.

DISCUSSION

Takayasu arteritis (TA) is an uncommon chronic granulomatous vasculitis affecting the aorta and its major branches, predominantly involving women younger than 40 years. The disease was first described by Mikito Takayasu has since been recognized as the prototype of large-vessel vasculitis in young females. Despite advances in vascular imaging, delayed diagnosis remains common because early manifestations are nonspecific and constitutional symptoms often precede vascular complications by several months or years.

Our patient fulfilled the 1990 American College of Rheumatology (ACR) classification criteria for Takayasu arteritis, satisfying four of the six criteria, including age below 40 years, limb claudication, diminished peripheral pulses, and angiographic abnormalities. Similar findings have been emphasized by Arend et al[1] who demonstrated that these criteria possess high sensitivity (90.5%) and specificity (97.8%) for the classification of TA.

The demographic profile of our patient closely resembles previously reported epidemiological studies. Numano et al[4] observed that Takayasu arteritis predominantly affects women in the second and third decades of life, with female-to-male ratios ranging from 8:1 to 9:1 in Asian populations. Our patient, a 37-year-old woman, falls within the typical age group for disease onset.

One of the most striking clinical manifestations in our patient was progressive bilateral lower limb claudication associated with left upper limb claudication and absent pulses in multiple extremities. These findings correspond to the classic "pulseless disease" originally described in Takayasu arteritis. Johnston et al[3] reported that diminished or absent peripheral pulses occur in nearly 84% of patients, while limb claudication is present in approximately 50–70% of cases. Similarly, Kerr et al[2] identified vascular insufficiency symptoms as one of the commonest reasons for hospital presentation.

Inflammatory markers were significantly elevated in our patient, with an ESR of 80 mm/hour and CRP of 12 mg/L, whereas ANA profile and other autoimmune antibodies were negative. Elevated ESR and CRP are frequently reported during the active inflammatory phase of Takayasu arteritis but correlate imperfectly with disease activity. Keser et al[7] highlighted that although inflammatory markers are useful adjuncts, imaging findings remain more reliable for assessing disease progression because vascular inflammation may persist despite normal laboratory parameters.

The angiographic findings in our patient demonstrated diffuse circumferential wall thickening involving the thoracic and abdominal aorta with bilateral carotid artery disease, complete proximal left subclavian artery occlusion, left vertebral artery occlusion, and moderate narrowing of the descending thoracic and abdominal aorta. These imaging findings are consistent with the characteristic vascular distribution described by Hata et al[5], whose angiographic classification identifies combined aortic arch and abdominal aortic involvement as one of the common patterns observed in Asian patients. Based on this classification, our patient demonstrates extensive disease involving the aortic arch and descending thoracoabdominal aorta with major branch vessel involvement.

An unusual feature of the present case is the coexistence of an infrarenal abdominal aortic aneurysm. Although arterial stenosis and occlusion represent the predominant vascular lesions in Takayasu arteritis, aneurysmal degeneration is considerably less frequent, occurring in approximately 10–25% of patients depending on ethnicity and disease duration. Maksimowicz-McKinnon et al[8] reported that aneurysm formation reflects advanced transmural inflammation leading to destruction of elastic fibers and weakening of the arterial wall. In our patient, the aneurysm was identified incidentally during CT angiography before the development of rupture or thromboembolic complications, emphasizing the importance of comprehensive vascular imaging.

Carotid artery involvement was another significant finding in our patient. Bilateral common carotid artery stenosis with internal carotid artery involvement increases the risk of transient ischemic attacks and stroke. Tso and Flamm demonstrated that carotid artery disease is among the most frequent extracranial vascular manifestations of Takayasu arteritis and may initially present only with carotid bruits, as observed in our patient before neurological deficits developed.

Unlike several published cases that describe constitutional symptoms such as prolonged fever, malaise, weight loss, or arthralgia during the pre-pulseless phase, our patient had no constitutional manifestations. Similar atypical presentations have been described by Mason et al[9], who emphasized that many patients first present during the chronic occlusive phase, thereby delaying diagnosis until irreversible vascular damage has occurred.

Current treatment recommendations advocate early initiation of glucocorticoids combined with steroid-sparing immunosuppressive therapy. Our patient received oral prednisolone (1 mg/kg/day), azathioprine (3 mg/kg/day), and antiplatelet therapy, resulting in marked symptomatic improvement after six months. This therapeutic strategy is consistent with the 2021 American College of Rheumatology/Vasculitis Foundation Guidelines, which recommend glucocorticoids together with conventional immunosuppressive agents such as

methotrexate or azathioprine for active Takayasu arteritis. Hellmich et al.[10] as EULAR recommendations, similarly advocate early immunosuppressive therapy to reduce relapses and prevent progression of vascular injury.

The present case is noteworthy because of the coexistence of extensive multivessel involvement affecting the bilateral carotid arteries, left subclavian artery, thoracoabdominal aorta, and infrarenal abdominal aortic aneurysm in a young woman without constitutional symptoms and with a negative autoimmune profile. Early CT angiography enabled prompt diagnosis before irreversible ischemic complications occurred. The favorable response to combined corticosteroid and azathioprine therapy further supports the effectiveness of early immunosuppressive treatment in controlling disease activity.[11]

Overall, this case emphasizes that Takayasu arteritis should be considered in young women presenting with limb claudication, pulse deficits, blood pressure discrepancies between limbs, or vascular bruits, even in the absence of systemic symptoms. Comprehensive vascular imaging is essential for defining disease extent, detecting uncommon complications such as abdominal aortic aneurysm, guiding therapeutic decisions, and improving long-term outcomes.

CONCLUSION

This case highlights the diagnostic and therapeutic challenges of Takayasu arteritis (TA) presenting with extensive multivessel involvement in a young woman. The patient presented with progressive bilateral lower limb claudication, left upper limb claudication, absent peripheral pulses, and elevated inflammatory markers without constitutional symptoms or positive autoimmune serology, illustrating the variable clinical presentation of this disease. CT aortography was pivotal in establishing the diagnosis by demonstrating diffuse involvement of the thoracic and abdominal aorta, bilateral common carotid arteries, complete proximal occlusion of the left subclavian artery, left vertebral artery involvement, and a rare wide-neck infrarenal abdominal aortic aneurysm.

The coexistence of extensive stenotic disease with aneurysmal degeneration represents an uncommon but clinically significant manifestation of Takayasu arteritis, emphasizing the importance of comprehensive vascular imaging in defining disease extent and identifying potentially life-threatening complications. Prompt initiation of immunosuppressive therapy with oral prednisolone and azathioprine, together with antiplatelet therapy, resulted in significant clinical improvement during six months of follow-up, underscoring the benefits of early diagnosis and timely treatment.

This case reinforces the need to maintain a high index of suspicion for Takayasu arteritis in young women presenting with limb claudication, pulse deficits, blood pressure discrepancies between limbs, or vascular bruits, even in the absence of constitutional symptoms. Early recognition, detailed angiographic evaluation, and multidisciplinary management are essential to control vascular inflammation, prevent irreversible ischemic complications, and improve long-term outcomes. Furthermore, this report contributes to the limited literature describing Takayasu arteritis associated with an infrarenal abdominal aortic aneurysm, highlighting the importance of long-term clinical and imaging surveillance in patients with extensive large-vessel involvement.

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