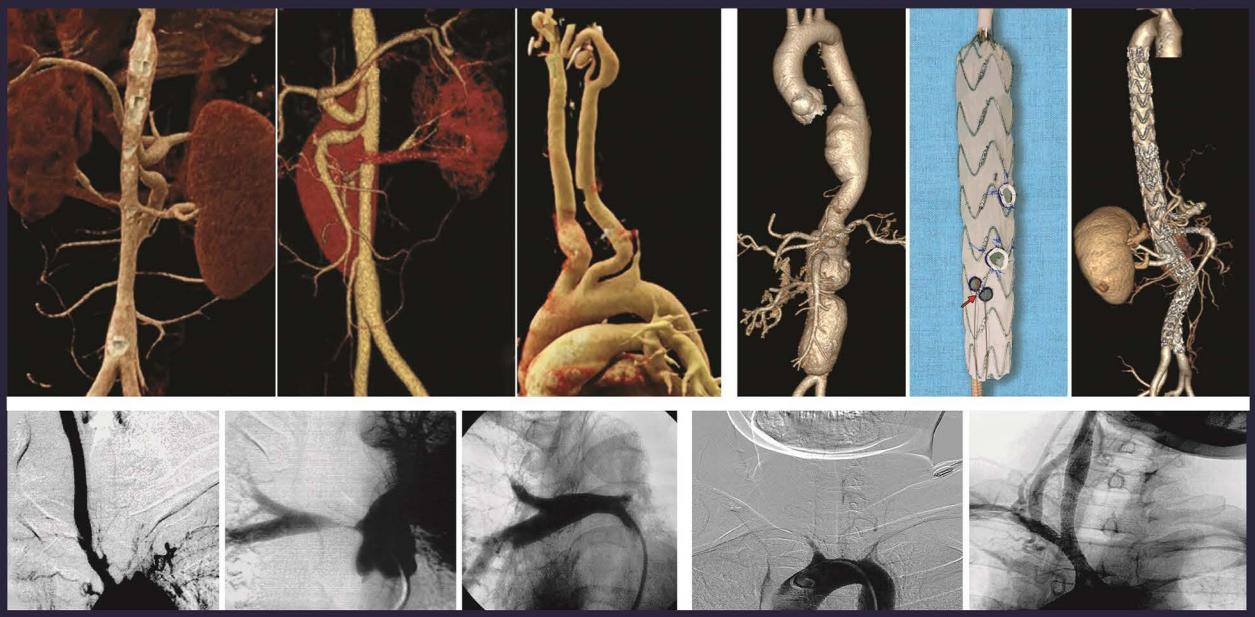
Cardiological Society of India

MONOGRAPH

TAKAYASU ARTERITIS

(Aortoarteritis)



Editor-in-Chief
Sanjay Tyagi

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CHAPTER

Challenges in the Diagnosis and Management of Takayasu Disease (Aortoarteritis)

Durga Prasanna Misra, Vikas Agarwal

INTRODUCTION

Aortoarteritis or Takayasu arteritis (TA) is an uncommon form of large-vessel vasculitis (LVV), relatively more frequently encountered in tropical regions such as India. TA more commonly affects female patients; however, the proportion of female patients varies from different regions of the world. In this chapter, we shall discuss the challenges related to the diagnosis and management of TA.

CHALLENGES IN THE DIAGNOSIS OF TAKAYASU ARTERITIS

Takayasu Arteritis—The Great Masquerader

Although commonly seen in the cardiology or the clinical immunology and rheumatology clinic, patients with TA can present in practically any medical specialty **(Fig. 1)**. The onset of TA can be in the pediatric or the adult age group.[1,2] Therefore, internal medicine specialists or pediatricians are often the first point of healthcare contact. Ocular manifestations such as TA retinopathy or ischemic vision loss can present to the ophthalmologist. Stroke or dizziness due to vertebrobasilar insufficiency can present to the neurologist.[3] Incidental pulse loss or the asymmetry of pulses or blood pressure might be detected during preanesthetic checkups for unrelated surgical procedures or during antenatal checkups. TA during pregnancy can associate with difficult-to-control hypertension, intrauterine growth retardation, or fetal loss.[4] TA can present to the nephrologist due to renal artery stenosis with or without renal impairment. TA can present to the medical or surgical gastroenterologist or the general surgeon due to bowel ischemia or associated ulcerative colitis or Crohn's disease.[5] Endovascular procedures in TA are commonly performed by cardiologists or by interventional radiologists. Rarely, TA can cause neck pain or chronic cough, and therefore, can present to the otorhinolaryngologist or the endocrine surgeon as thyroid pain.[6] Awareness by the medical doctor to carefully check for the symmetry of peripheral pulses and blood pressure in all four limbs might enable the early diagnosis of this great masquerader.

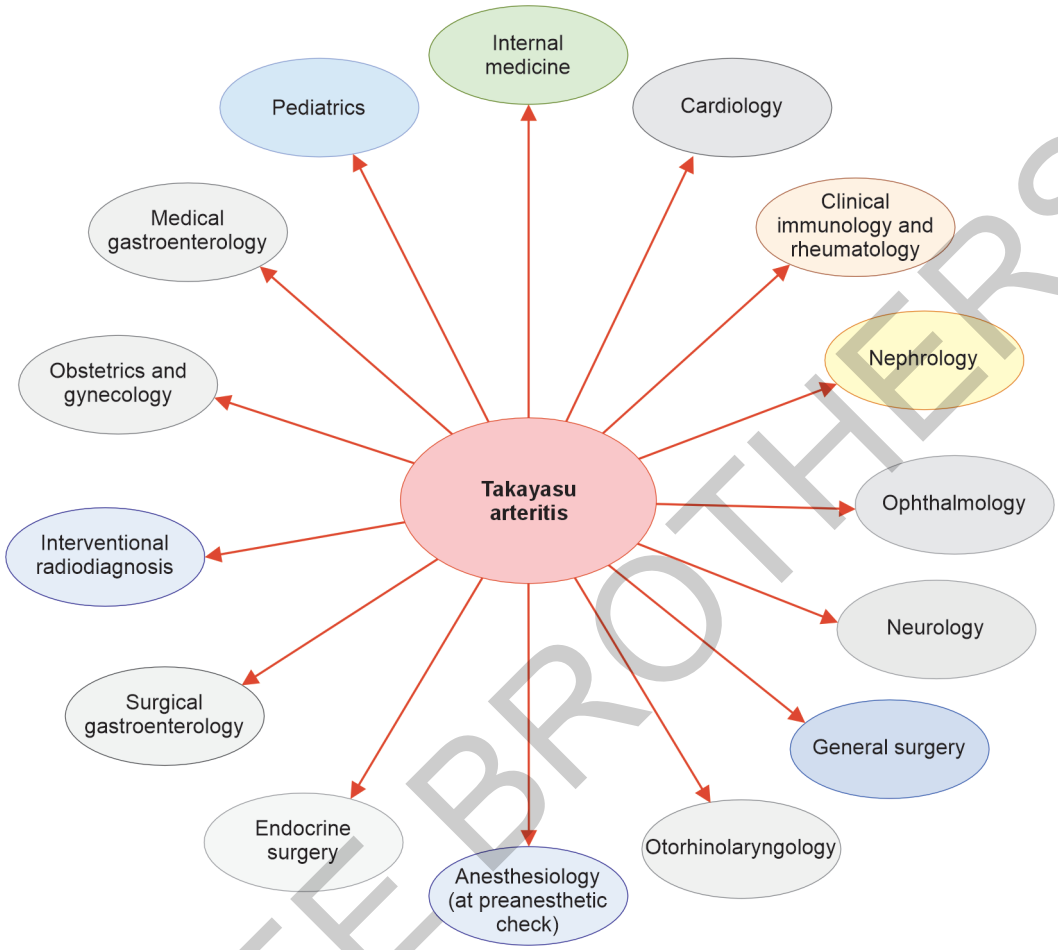


FIG. 1: Different specialties that Takayasu arteritis can present to.

Classical Presentation of Takayasu Arteritis

A diagnosis of TA is clear when it presents with constitutional features such as hectic fever, weight loss, malaise, and joint pains along with loss of peripheral pulses and vascular bruits with elevated acute phase reactants [erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP)]. Classically, it has been believed that TA presents initially with constitutional features, then with features of vascular involvement such as tender arteries (typified by carotidynia), pulse inequality, pulse loss, or vascular bruits. This is followed by occlusive symptoms such as stroke or myocardial ischemia. However, such a triphasic presentation is uncommon and only occurs in about one-fifth of patients with TA.[7]

Differential Diagnosis of Takayasu Arteritis

Giant cell arteritis (GCA) is the counterpart LVV of TA, more often seen in older individuals (older than 50 years). While well-described among Indians, GCA is much more common in those of Caucasian ancestry.[8] Temporal headache and temporal arteritis evident on ultrasonography or temporal artery biopsy are important diagnostic clues. GCA can also affect the large arteries (large-vessel GCA). While TA classically has onset before 50 years of age and GCA usually starts

after 60 years, either TA or GCA can have onset between the ages of 50 and 60 years. Usually, TA involves the abdominal aorta, including the renal arteries and the mesenteric arteries, whereas large-vessel GCA commonly affects the subclavian arteries. Symmetrical involvement of arteries is also classical of TA rather than large-vessel GCA.[9]

Atherosclerosis is another differential diagnosis of TA. Atherosclerosis might coexist with TA (particularly in those undergoing imaging assessment at an older age). Atherosclerosis might also be secondary to long-standing arterial wall inflammation. Atherosclerotic plaques in a young individual on computed tomographic angiography (CTA) without traditional risk factors for cardiovascular disease such as dyslipidemia, diabetes mellitus, tobacco use, or family history of premature coronary artery disease or cerebrovascular disease should raise the suspicion of TA. Atherosclerotic plaques typically occur at the bifurcation of large arteries, although other sites can also be affected.[10]

Immunoglobulin G4-related disease (IgG4-RD) is another inflammatory disease that can present with periaortitis and vascular narrowing. This commonly involves the abdominal aorta. Histopathology of the periaortic soft tissue demonstrates a lymphoplasmacytic infiltrate with obliterative phlebitis and fibrosis. Immunohistochemistry of the periaortic soft tissue demonstrates a predominance of IgG4-positive plasma cells.[10] Lymphoma is an important differential of IgG4-RD. Pleural effusion can occur in IgG4-RD due to blockage of lymphatic drainage. However, pleural effusion is extremely unusual in TA unless there is associated tuberculosis or severe heart failure.[11]

Fibromuscular dysplasia is a noninflammatory arterial disease typically associated with a beaded appearance of involved arteries along with normal inflammatory markers and lack of vascular wall metabolic activity on 18-fluorodeoxyglucose (18-FDG) positron emission tomography computed tomography (PET-CT). Fibromuscular dysplasia often involves the renal, carotid, or vertebral arteries. Less commonly, TA can present with vascular aneurysms. In such instances, inherited disorders of connective tissue such as Ehlers–Danlos syndrome type IV or Loeys–Dietz syndrome should be considered in the differential diagnosis.[10] Chronic periaortitis is another less frequently encountered differential diagnosis of TA, particularly when the age of onset is older. Nonspecific aortitis or midaortic syndrome, where isolated involvement of the abdominal aorta or the renal arteries is evident on imaging without other features of TA, is another differential diagnosis. In this context, a clinical pearl to note is that isolated renal artery stenosis can occur in adults but is rare in children. Renal artery stenosis in children most often indicates underlying TA.[2] **Box 1** summarizes the differential diagnosis of TA commonly encountered in clinical practice.

Diagnostic or Classification Criteria for Takayasu Arteritis

The first diagnostic criteria for TA were proposed by Ishikawa in 1988.[12] These criteria mandated the age of onset at or before 40 years, major criteria (two) as the presence of right or left mid-subclavian artery lesions, and minor criteria (nine) as ESR elevation, carotidynia, hypertension, aortic insufficiency, pulse loss, vascular bruits,

BOX 1 Differential diagnosis of Takayasu arteritis.

- Atherosclerosis
- Large-vessel giant cell arteritis
- Nonspecific aortitis or midaortic syndrome
- Immunoglobulin G4 (IgG4)-related disease
- Fibromuscular dysplasia
- Ehlers–Danlos syndrome type IV
- Loeys–Dietz syndrome
- Chronic periaortitis

involvement of the pulmonary arteries, the middle part of left common carotid artery, the distal portion of the brachiocephalic trunk, and descending thoracic aorta or abdominal aorta. Involvement of the aortoiliac trunk 2 cm proximal to the iliac bifurcation excluded TA as per Ishikawa et al. possibly to differentiate versus atherosclerosis. The presence of two major, one major with two minor, or four minor criteria was required to diagnose TA, with a sensitivity of 84% and a specificity of 100%.[12,13] These criteria were modified by Sharma et al. to remove the mandatory age criterion, include the presence of clinical features consistent with TA for at least a month as a third major criterion, include coronary arteritis as a tenth minor criterion, and remove the involvement of aortoiliac trunk as an exclusion criterion. A similar number of major or minor criteria to the Ishikawa criteria were required to be fulfilled under Sharma's criteria, which were more sensitive (92.5%) but less specific (95%) to diagnose TA.[12,14] The American College of Rheumatology (ACR) in 1990 proposed classification criteria for TA, which mandated the presence of three of the following six features: (i) onset at or before 40 years, (ii) limb claudication, (iii) asymmetry of blood pressure between the upper limbs, (iv) diminished upper limb pulse, (v) subclavian or aortic bruit, or (vi) angiographic findings consistent with TA. These criteria were highly specific (97.8%) while also being relatively sensitive (90.5%) for TA.[12,15] The Chapel Hill Consensus Conference definitions for the different types of vasculitis proposed in 2012 define TA as granulomatous aortitis and arteritis with onset by 50 years of age.[12,16] Separate classification criteria have been proposed by the European Alliance of Associations for Rheumatology/ Pediatric Rheumatology European Society/ Pediatric Rheumatology International Trials Organization in 2008. These criteria require the presence of angiography consistent with TA along with one of the following features: pulse loss, limb claudication, asymmetry of blood pressure, hypertension, vascular bruits, or elevated acute phase reactants. These criteria had a sensitivity and specificity of nearly 100%.[12,17]

The major bone of contention with all the above diagnostic or classification criteria for TA is that these were arbitrary rather than being data-driven. The multicentric DCVAS (Diagnostic and Classification Criteria for Vasculitis) study has collected data on thousands of patients with vasculitis over the past decade to develop new classification criteria for vasculitis including for TA using a data-driven approach. These criteria, which were recently published, define preconditions before applying the criteria as a diagnosis of LVV having been made and mimics of LVV excluded. Thereafter, mandatory entry criteria require an age of diagnosis ≤60 years and imaging evidence of vasculitis. Subsequently, points are provided for clinical indicators of TA, viz., female gender, cardiac ischemia, limb claudication, vascular bruit, decreased brachial or radial pulse, diminished or absent carotid pulse or carotidynia, and a difference in systolic blood pressure of at least 20 mm Hg between the upper limbs. Angiographic criteria define the number of arterial territories involved (up to three) and further define whether any set of paired arteries is involved. Additional points are provided for the involvement of the abdominal aorta with either the renal or mesenteric arteries. A score of at least five points is required for the classification of TA. The sensitivity and specificity of these criteria were reported as 93.8% and 99.2%, respectively, with an area under the curve of 0.97.[12,18] A point to note is that most of these criteria are classification criteria, meant to be used to homogenize the recruitment of patients in studies (including clinical trials) involving TA so that they are comparable across the world. Classification criteria should be applied only after a clinical diagnosis of TA has been made, rather than being used for diagnosis (for which purpose they are commonly misused).[19]

CHALLENGES IN THE DISEASE ASSESSMENT OF TAKAYASU ARTERITIS

Suboptimal Concordance of Acute Phase Reactants and Circulating Biomarkers with Disease Activity

Erythrocyte sedimentation rate and CRP poorly associate with disease activity in TA. Open surgical bypass grafts or endovascular procedures in TA are generally undertaken during periods of inactive disease with normal inflammatory markers. However, even during such periods of clinical vascular inactivity, histopathological evidence of active vasculitis is evident in about 40% of patients.[20] Circulating pentraxin 3, and matrix metalloproteinases (MMPs) 2, 3, and 9 have shown an association with active TA in some but not in other studies.[20] Recent studies have shown the promising association of circulating T helper 17 (Th17) and Th17.1 lymphocytes with TA disease activity.[20-22] The identification of suitable biomarkers to assess disease activity in TA is still a work in progress.

Challenges in Vascular Imaging

Conventional angiography and digital subtraction angiography are the gold standards for assessing the anatomy of the arterial tree. These modalities help to detect vascular stenosis, occlusion, or dilatation, all of which can occur in TA. However, the characteristics of the vascular wall cannot be assessed using these modalities. CTA requires the administration of intravascular contrast to assess both the arterial wall anatomy as well as wall thickening. Wall thickening assessed using CTA may reflect active disease (resulting in vascular progression in these segments) or inactive disease. Magnetic resonance angiography (MRA) can be done without intravenous contrast administration. MRA with intravenous contrast helps to detect wall characteristics through vascular wall contrast uptake. However, intravenous contrast for either CTA or MRA is contraindicated in a patient with renal failure. In such instances, diffusion-weighted MRA without contrast may still provide information about the inflamed arterial wall.[20]

Over the past decade, PET-CT has been increasingly used in LVV. PET-CT uses intravenously administered 18-FDG or other ligands to detect metabolic activity in different areas of the body, including in the arterial wall. Metabolic activity in the arterial wall, particularly if confluent, indicates active vascular wall inflammation (vascular wall uptake in atherosclerosis is generally patchy and linear). Such vascular wall inflammation may be dampened if a patient is on corticosteroids. Therefore, 18-FDG PET-CT is preferably done in the corticosteroid-naïve, active TA. Emerging literature indicates that PET-CT may indicate active TA in a patient with normal ESR or CRP.[20,23]

Color Doppler ultrasound enables the noninvasive assessment of vascular wall characteristics and vascular stenosis for accessible vessels such as the carotid arteries and femoral arteries. The inflamed arterial wall in TA undergoes neovascularization. Recent literature suggests the promise of air bubble contrast-enhanced ultrasonography (CEUS) to assess vascular wall disease activity (since this contrast enters into the inflamed arterial wall through the vasa vasorum). However, CEUS requires considerable technical expertise and validation before it can be more widely used.[20]

Challenges in the Clinical Assessment of Disease Activity

The National Institutes of Health (NIH) disease activity criteria, proposed in 1994, denote active TA when at least two of the following four points are worse: constitutional symptoms, elevated ESR

(>20 mm/h), vascular symptoms, or angiographic evidence of worsening disease. The limitation of these criteria is that a prior visit is required for comparison. Hence, they cannot be applied at the first visit.[20,24] The Disease Extent Index in TA (DEI.TA) and the Indian Takayasu Clinical Activity Score 2010 (ITAS2010) have been devised by researchers from India over the past decade to enable a points-based objective assessment of disease activity. However, they are limited by the lack of established cutoffs for active disease and the absence of inclusion of information from angiography while calculating these scores.[20,25,26] Also, scores on the first and second visit have poor concordance with physician assessment of disease activity.[20,27] Till date, there is no gold standard tool to distinguish active from inactive TA.

Patient-reported Outcome Measures in Takayasu Arteritis

Few studies have assessed patient-reported outcomes in TA. The limited data in this area suggests that patients with TA have poorer quality of life than control subjects. However, this requires further exploration, particularly in an Indian population.[28]

Composite Scores for Assessment of Takayasu Arteritis Disease Activity

Given the limitations of presently available disease activity indices for TA, combining information from different modalities might enable a better distinction between active and inactive TA. A recent study reported the better performance of a disease activity model derived from mean standardized uptake value (SUV_{mean}) from 18-FDG PET-CT, ESR, and serum interleukin 2 receptor levels when compared with ESR or CRP alone. This score is promising but remains to be validated across different populations.[20,29,30] This approach might be suitable for developing a valid disease activity score for TA in the future.

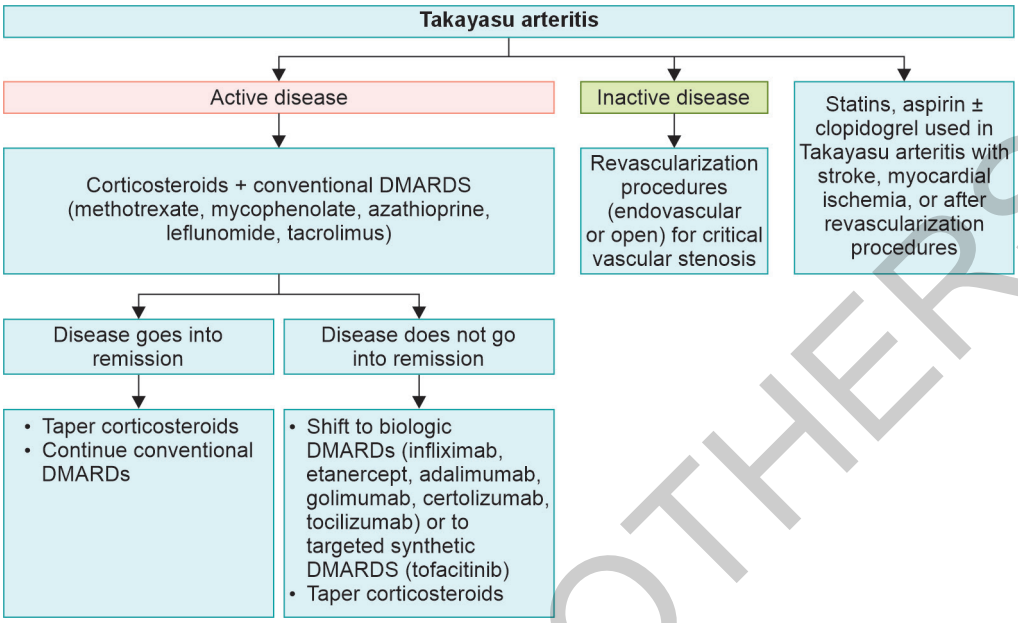
CHALLENGES IN THE TREATMENT OF TAKAYASU ARTERITIS

Corticosteroids are Effective for TA but the Response is not Sustained

Since the pathology of TA is immune-mediated, corticosteroids are the mainstay for the initial treatment of active TA. They are effective in inducing remission of active disease in 60% of patients. Stabilization of angiographic changes was also observed in 28% of TA treated with corticosteroids alone. However, relapses occur in about two-thirds of TA once corticosteroids are tapered off.[31] This necessitates the use of maintenance immunosuppressive or disease-modifying antirheumatic drugs (DMARDs) along with corticosteroids for active disease.

Disease-modifying Antirheumatic Drugs Appear Effective but are Unproven in Clinical Trials

To date, no DMARD has been proven to be effective in a clinical trial of patients with TA.[32,33] Both tocilizumab and abatacept failed to meet their primary end points in clinical trials of TA (tocilizumab met the secondary end point of lesser time to relapse in a per-protocol analysis).[34,35] Conventional DMARDs such as methotrexate, mycophenolate mofetil, azathioprine, leflunomide, and tacrolimus; biologic DMARDs such as tocilizumab and antitumor necrosis factor alpha (anti-TNF-α) agents (infliximab, etanercept, adalimumab, golimumab, and certolizumab); and the targeted synthetic DMARD tofacitinib are all used in TA based on favorable results from observational studies. Similar clinical responses as well as retardation of angiographic progression have been observed with anti-TNF agents or tocilizumab.[32,33] As a rule of thumb, conventional DMARDs are first used, followed by



FLOWCHART 1: Proposed management plan for Takayasu arteritis.
(DMARDs: disease-modifying antirheumatic drugs)

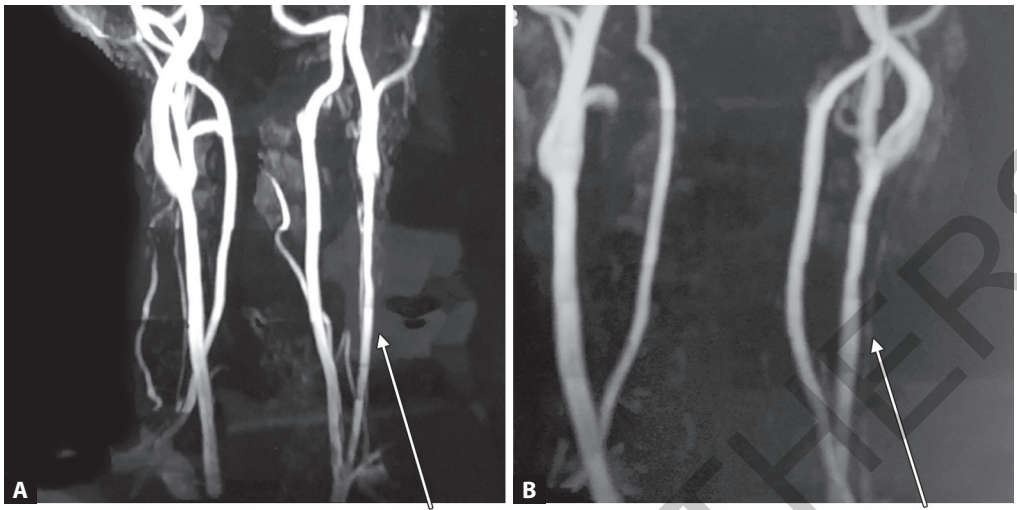
biologic or targeted synthetic DMARDs in refractory disease **(Flowchart 1)**.[32,33]

The initial phase of arterial inflammation in TA resolves with vascular fibrosis, which ultimately leads to vascular stenosis or occlusion in TA. Therefore, it is prudent to hypothesize that targeting vascular fibrosis with antifibrotic agents along with the immunosuppressive DMARDs might enable better long-term vascular outcomes in TA. While this approach has not been explored in clinical trials, in vitro results and our anecdotal experience suggest a potential benefit with this approach in TA **(Figs. 2A and B)**.[21]

Interventions for Takayasu Arteritis

The vascular stenosis in TA generally occurs insidiously, allowing for the development of extensive collateral circulation. However, whenever such vascular stenosis is critical or organ-threatening, revascularization (endovascular or open surgical) is indicated. Endovascular interventions are more easily performed since expertise in vascular surgery related to TA is scarce.[36] Open surgical revascularization in TA is associated with lesser rates of restenosis but a greater risk of stroke in the perioperative period.[37] Revascularization procedures should be performed during periods when the disease activity of TA is quiescent. If performed during active disease, there is a greater risk of complications (restenosis or vascular rupture) or of mortality.[36,38] Immunosuppressive therapy (including corticosteroids) should be continued during the periprocedural period for the same reason of maintaining the remission of the disease.[36]

Endovascular procedures in TA could involve angioplasty or stenting. The evidence suggests that angioplasty in TA is associated with better outcomes in the renal arteries, stenting is superior for the coronary arteries, whereas both procedures are essentially equivalent for the remaining vascular territories.[36]



Irregular, narrowed left common carotid artery before tacrolimus, methotrexate, and tadalafil

Recanalized left common carotid artery 3 years after treatment

FIGS. 2A AND B: Serial angiographic images of the carotid arteries of a patient with refractory Takayasu arteritis before (A) and after treatment with tacrolimus, methotrexate, and tadalafil (B).

CONCLUSION

While the diagnosis of TA is relatively straightforward, mimickers such as atherosclerosis and fibromuscular dysplasia should be ruled out. The assessment of disease activity in TA is challenging. The distinction between active disease and vascular damage is nebulous. While TA responds well to immunosuppressive therapy, the evidence base for the use of DMARDs in TA is weak, based on observational studies alone. There is an unmet need for high-quality randomized controlled trials to identify effective DMARDs in TA. Endovascular revascularization procedures are more accessible than open surgical revascularization, particularly in an Indian context. However, all revascularization procedures should be undertaken during periods of inactive disease to minimize the risk of complications and periprocedural death.

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MONOGRAPH

TAKAYASU ARTERITIS

(Aortoarteritis)

Salient Features

- This monograph has in-depth discussion on vasculitis in India, large-vessel vasculitis, state-of-the-art reviews on pathology, pathogenesis, advanced diagnostic modalities, clinical features, imaging advances in medical management, endovascular nonsurgical interventions, surgical management, and natural history of Takayasu arteritis.
- It is truly comprehensive, covering every possible aspect of disease in separate chapters along with high-quality illustrations. Authors of different chapters are eminent experts with in-depth knowledge of the subject of Takayasu disease (aortoarteritis). Their original research work in this area has been published in reputed international journals having a high impact factor. Invaluable experience of authors makes this monograph truly indispensable.
- Useful book for suspecting the disease early, carrying out appropriate investigations, making a diagnosis, providing the evidence-based best form of treatment, and conducting future research in this disease.
- It is a reference book for physicians, cardiologists, and cardiac/vascular surgeons world over.

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