Takayasu Arteritis: Current Perspective and Brief Narrative Review

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Abstract

Takayasu Arteritis (TA) is a rare chronic inflammatory vascular disorder that mainly causes stenosis and occlusion of large vessels of the body. TA is prevalent in the Asian population with a higher evidence seen in young females. Even though TA has an unclear etiopathogenesis, there are numerous genetic links found associated with TA. Various interventional and radiological investigations prove to be the key for confirming the diagnosis of TA, following which appropriate medical and surgical line of treatment are done, however there is no universal protocol for managing TA. TA has a tendency to progress to serious life threatening complications, hence early diagnosis and treatment should be the goal, also aimed at preventing future re-stenosis, a well-studied complication of TA. It is also crucial to diagnose TA from other overlapping diseases, owing to its peculiar clinical presentation and characteristic radiological findings, which is often confused with other vascular disorders. TA as a disease greatly impacts the quality of life in affected patients with a worse prognosis seen in those who are predisposed to certain well documented risk factors. Furthermore, an increasing number of cases reporting an interesting link between arteritis and the COVID-19 virus have been reported in recent times, which might prove to be crucial in the forthcoming years for establishing this association. Our article aims to sensitize clinicians with regard to an increased awareness of TA in various clinical settings for better patient management and improved prognosis.

Keywords: COVID-19, diagnosis, endovascular, giant cell arteritis, interventions, takayasu arteritis, treatment

Introduction

Takayasu arteritis (TA) is a rare type of a chronic inflammatory and granulomatous vascular disorder, which typically is seen to affect large vessels of the body, mainly affecting aorta and its main branches.[1] This disease has also been referred to as pulseless disease, idiopathic arteritis, aortoarteritis, and occlusive thromboarthropathy in keeping with its etiology and typical clinical presentation.[2] First described by Mikito Takayasu *et al* in 1908 as a case of retinal vasculitis in Japan,[2] TA is now a well-established clinical entity in medical literature. However, it is crucial for clinicians to differentially diagnose TA from giant cell arteritis (GCA), which has been studied to have an overlapping stigmata of clinical presentation and findings,[3] once thought to be a different clinical phenotype of the same parent disease.[4] TA can be a challenging disease owing to its unclear etiopathogenesis, peculiar radiological findings and varied clinical presentations, however, it is crucial to correctly and timely diagnose this disease in a clinical setting because of the long-term complications that may follow the course of TA.

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Etiology and Pathogenesis

The etiopathogenesis of TA remains unclear and debatable because of the wide variety of factors which play a key role in its etiopathogenesis ranging from genetic to immune mediated mechanisms.[5] There is still paucity of data confirming the ethnic and genetic link to TA but the pathogenesis of TA has been largely studied to be associated with the HLA complex and its various sub-types found in different geographical populations.[5] Moreover, the autoimmune cell-mediated immunity plays a key role in this process[6] leading to somewhat of a granulomatous type of inflammation affecting all the layers of the vessel wall (panarteritis),[1] thereby causing tissue damage and complement and cell mediated cytotoxicity.[7,8] However, the exact sequence of events leading to vessel wall inflammation and damage has not been established yet. The most common arteries that are involved include the aorta, arch of aorta and its branches, and abdominal aorta, while subclavian, renal, and coronary arteries are often at insult too.[1,9] This vascular involvement mostly leads to stenosis and/or occlusion of the arteries, which in-turn results indifferent clinical presentations in a patient of TA, which though sometimes can be diagnosed on clinical examination, should always be confirmed with help of radiological and interventional techniques as is discussed ahead.

Prevalence and Epidemiology

In a global scenario, the occurrence of TA is seen to be more common in Eastern and mainly Asian countries in comparison to European and American counterparts.[10,11] TA continues to be a rare occurrence in Asia, however, it still has a prevalence many folds higher than North America, which was found to have a prevalence of about 2.69 cases per million per year.[12,13] That said, there is no recent large multicentric study which correctly assesses the prevalence of TA in India. The significant geographical variation in the prevalence of TA can be attributed to the genetic link of the HLA association,[14-16] which also possibly results in the different clinical presentations and arterial network involvement as is seen in different populations with TA.[16-18] It is also well-studied that TA has a strong female predominance, accounting to a female to male ratio of 1.58:1 as is reported in the Indian population and that TA commonly affects women before the 4th decade of life.[10,16,19,20] TA is also observed to be the most common cause of renovascular hypertension in India among other South East Asian countries.[21-23] Moreover, it is seen that among Indian patients, hypertension is seen to be one of the most common findings at the time of presentation contributed by the involvement of the renal arteries in most cases.[21,22,24,25] Furthermore, it has been found that gender differences might also play a role in determining the distribution and type of involvement in TA among Indian patients, with males having a higher frequency of hypertension and involvement of abdominal aorta while females more commonly having involvement of the arch of aorta and its branches.[26] Thus even though TA is a rare entity, it is of a considerable clinical focus in the Indian setting, even though the exact epidemiological determinants and incidence of TA remain to be found and estimated in the Asian-Indian population.

Clinical Presentation and Disease Course

Broadly speaking TA encompasses 3 clinical stages- an acute stage of early systemic illness with non-specific constitutional symptoms, an inflammatory stage, and a chronic 'pulseless' stage characterised by ischemia and occlusive crisis/peripheral claudication.[27] Some of the clinical symptoms with which the patient presents include severe headache, pain in the neck/back/limbs, fatigue, weight loss, fever, and severe ones like cerebrovascular accidents (CVAs), congestive cardiac failure (CCF), aortic regurgitation, among others[27-29] which might prove to be fatal. Overall the clinical features may include early constitutional symptoms like fever, weight loss, malaise, arthralgias, myalgias, etc. which are seen in roughly 40-60% of the patients; vascular problems like extremity claudication, neurological involvement resulting in transient ischemic attack, haemorrhagic/ischemic stroke, headaches, seizures etc seen in up to 60% of the affected population; and other cardiac, pulmonary, renal and even dermatological features.[23] However, these three stages of TA are not universal and differ according to the severity and pattern of vascular involvement. Ishikawa *et al*,[30] in a study done in Japanese patients (n=108, 96 patients with TA, 12 patients with other diseases of the aorta) showed that TA has an insidious onset in 76% cases and a sudden onset in 24% cases because of the non-uniform disease course resulting in variation in time and pattern of patient presentation. In another study[31] by Ishikawa *et al*, it was found that 40% of cases showed a plateau-crescendo course of symptoms, 36% of cases showed a plateau, 19% of cases showed a decrescendo, and about 5% showed decrescendo-plateau-crescendo pattern, thereby confirming the variation in patient presentation and disease course. Therefore it is important for physicians to be aware of such varied clinical presentations when diagnosing TA, which in-turn will reflect positively in early

patient stabilization and better management.

Diagnostic and Imaging Criteria

Earlier in 1988 Ishikawa *et al*[30] had proposed a criteria to diagnose TA which included one obligatory criterion (age <40 years at diagnosis or onset of disease), two major criteria (left and right mid subclavian artery lesions) and nine minor criteria (high ESR, common carotid artery tenderness, hypertension, aortic regurgitation or annulo-aortic ecatisa, lesions of pulmonary artery, left mid common carotid artery, distal branchiocephalic trunk, thoracic aorta, and abdominal aorta). In addition to the obligatory criterion, the presence of two major criteria, or one major plus two or more minor criteria; or four or more minor criteria suggests a high probability of the presence of TA.[30] However, these criteria showed a greater sensitivity for patients with an active disease (96% and 80% in active young and old groups respectively) than for those with inactive disease (67% and 64% in active young and old groups respectively),[30] and thus there was a need felt for a more uniform criteria which could overcome such discrepancies observed during different phases of disease activity. Nevertheless, clinical diagnosis of TA can now be established by using the widely recognized criteria laid down by the American College of Rheumatology for TA in 1990.[32] According to these criteria,[32] diagnosis of TA can be established if at least 3 of the 6 criteria are fulfilled, which include:

1. Age at disease onset ≤ 40 years,
2. Claudication of extremities,
3. Decreased brachial artery pulse,
4. Systolic BP difference >10mm Hg between arms,
5. Bruit over subclavian arteries/aorta, and
6. Arteriogram abnormality.

If any patient fulfills at least 3 of the 6 mentioned criteria, it confirms the diagnosis of TA with a sensitivity of 90.5% and specificity of 97.8% as has been stated.[32]

Biochemically speaking, the diagnosis of TA is not straight forward due to absence of any specific laboratory investigation(s) or biomarker(s), which might be indicative of a direct association with TA,[1] thus, leading to a considerable delay in timely diagnosis. Various multi-modality imaging techniques and radiological interventions continue to be the 'modality of choice' worldwide for confirming the diagnosis of TA.[33,34] Even though magnetic resonance angiography (MRA), computerized tomography angiography (CTA), and digital subtraction angiography (DSA) have replaced conventional angiography as the gold standard, various other modalities like colour doppler ultrasonography (CDU), positron emission tomography (PET) and 18F-fluorodeoxyglucose positron emission tomography/computerized tomography (18F-FDG PET/CT) have also become preferred choices of clinicians for confirming the diagnosis of TA in state-of-the-art clinical setups wherein these modalities may be available.[11,34-36] The differential diagnosis of TA primarily includes GCA, aortic coarctation, atherosclerosis, Behcet's disease, Kawasaki disease, among other systemic, infectious, auto-immune and non-inflammatory conditions.[6] Keeping all these differentials in mind, it becomes crucial for a clinician to correctly diagnose TA initially on clinical examination, which though difficult at times because of certain technical and anatomical difficulties should always be confirmed with appropriate radiological and interventional studies.

Treatment Modalities and Management

There is no specific treatment for TA because of its largely unknown etiology, however, the primary goal of treatment in TA is majorly aimed at decreasing clinical symptoms and reducing the underling pathology causing inflammation and other immune system mediated effects. First line management of TA includes drug therapy with the aim of immunosuppression and reduction in inflammation with drugs like methotrexate (MTX), azathioprine (AZA), mycophenolate mofetil (MMF), and leflunomide (LEF), corticosteroids including prednisone and certain biological agents including monoclonal antibodies like tocilizumab (TCZ), T-cell co-stimulation modulators like abatacept (ABA), and TNF-alpha inhibitors like infliximab (IFX)and etanercept (ETA), which have been tried in different regimens, both alone and in combination.[37,38] Other supportive drugs like anti-platelets, oral anti-coagulants and vasodilators can be advised in cases wherein there is a high predictability or past history of a pro-thrombotic or thromboembolic event(s), however, the efficacy of these solely for treating TA is still uncertain. In chronic conditions wherein reversal of the suppressed immune system might take place or in cases with severe tissue ischemia or limb claudication, endovascular interventions and revascularization procedures including balloon angioplasty, stent implantation and graft placements are the mainstay.[39,40] Even though latest advances in endovascular techniques continue to have excellent results and prognosis on long-term follow up, there is always a high risk and suspicion for restenosis and recurrence when treating TA.[41,42] Future advancements in interventional and endovascular pro-

cedures might prove to be the cornerstone for a less invasive, risk-free modality in treating TA, also by allaying the need for other supplementary aids of management.

Outcomes, Prognosis and Quality of Life

TA can complicate to cause certain life threatening complications, some of which are acute myocardial infarction (AMI), CVAs, CCF and chronic kidney disease (CKD) leading to renal failure.[43,44] The overall outcome of TA largely depends on the distribution and pattern of the vascular network involved, wherein the duration, extent and severity of the inflammation and immune response are studied to be important factors for assessing the prognosis of a patient.[45] Naturally, in cases with a delayed diagnosis and disseminated multi-systemic illness, prognosis is certainly poor. Differences in the medical line of management and surgical interventions greatly influence the patient prognosis, and hence may also affect mortality rates.[11] Prognosis is worsened in presence of unfavourable factors like TA complications, progressive disease course and older age of patients.[31,38,46,47] Subramanyan *et al*[24] documented the mortality rates to be 9% after 5 years and up to 16% after 10 years of follow-up in patients (n=88, M=34, F=54, average age= 24.0 +/- 8.8 years at onset of symptoms and 28.3 +/- 9.9 years at diagnosis, period of follow up=83.6 +/- 74.4 months from onset and 33.2 +/- 37.0 months from diagnosis) who either succumbed to fatal complications or suffered major non-fatal outcomes, thereby implicating the gravity of TA in causing life threatening outcomes. It is seen that the overall survival rates decrease when TA progresses severe enough to cause life threatening cardiovascular complications like CCF, AMI, CVAs and even certain post operative complications, to which the patient eventually succumbs.[24,39,46-48] TA is associated with functional impairment, particularly in younger age groups, which is contributed by its peculiar 'waxing-weaning' progressive nature.[28,49] Owing to the well-known complication of restenosis/recurrence in patients, TA has a major impact on the quality of life in such patients, especially those who are affected at a younger age. Thus, early diagnosis is a key to success in patients who can be effectively managed with different treatment modalities in an effort to enhance their quality of life. That said, in elderly patients who have a progressive TA disease course and high chance of recurrence along with other complications, the overall quality of life is grossly hampered at both physiological and functional levels reaching up to a point of no-return, for which there is no permanent cure as of today.

Arteritis and COVID-19 Virus

A recent link has been found between arteritis and the COVID-19 virus, which includes not only documentation of cases reporting the occurrence of arteritis, particularly large vessel vasculitis (LVV) and GCA after either COVID-19 contagion or vaccination, but also arteritis, itself being a potential risk factor for numerous COVID-19 related complications.[50-54] LVV has been associated with an increased risk for COVID-19 infection, in addition to also playing a role in causing grievous outcomes in the same.[55,56] In majority of the patients having LVV, GCA is studied to be associated with a higher COVID-19 prevalence and an increasing number of cases in recent times, wherein the SARS-CoV-2 viral infection itself is seen to trigger the pathogenesis of GCA in patients, which stems from the fact that GCA and COVID-19 share somewhat of a similar clinical sigmata.[50,57-59] Even though there is limited evidence that points towards the relationship between TA and the COVID-19 virus, it is still wise to say that there is an underlying pathogenetic mechanism mediated by inflammatory, immunological and genetic factors which maylink these two entities, however due to paucity of literature this fact also remains uncertain and needs to be further elaborated. Future studies evaluating the link between TA and COVID-19 may positively result in confirming and later establishing this relationship, however as of today, these facts remain inconclusive.

Conclusion and Limitations

Takayasu Arteritis (TA) continues to be an uncommon yet significant vascular disease, however, there is lack of awareness and understanding amongst clinicians with regard to the etiopathogenesis and critical attributes pertaining to important clinical and diagnostic findings of the same. Moreover, the current medical literature available on TA remains to be further elaborated and studied in respect to its outcomes and long term complications, while also confirming the existing available data. The prevalence and severity of TA with other comorbidities in the Indian population still requires further medical research. Also, there is no universally accepted criteria for defining TA in different populations of varied ethnicities.

Furthermore, it becomes crucial to correctly diagnose TA in a clinical setting which in-turn will reflect positively on better patient management and prognosis. Latest advancements in endovascular and minimally invasive interventional technologies, both radiological and surgical will certainly prove to be successful in the forthcoming years for diagnosing as well

as treating TA optimally. Lastly, an interesting overlapping link between arteritis and COVID-19 virus has been found which needs to be further studied and elaborated to establish a concrete relationship between these two diseases. Once certain, this might guide future treatment options for formulation of primary preventive strategies and also help improve patient care.

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