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A systematic review: Pathogenesis & management of Takayasu arteritis

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Abstract
Takayasu arteritis is a chronic increasing granulomatous necrotizing large vessel vasculitis that involves the aorta and its branches. Females are mainly affected by this disease. Here the inflammation and intimal proliferation cause the thickening of the wall, stenotic or occlusive lesions, thrombosis, and aneurysms, and dissection is developed by the destruction of elastic and muscular layers. It is also known as the pulseless disease. The general symptoms observed are fever, malaise, weight loss, and anorexia. Due to inflammation, the arteries are narrowed which leads to a decrease in the blood flow. Angiography, Ultrasound, and Doppler techniques are used in the diagnosis of the Takayasu arteries. The disease is majorly observed in Asia, Africa, and Latin America. The future of TA management is precision medicine, which uses biomarkers and molecular profiling to personalize treatment regimens and improve patient results. Additional study is required to understand the underlying mechanisms of TA and create targeted therapeutics. MTX, AZA, MMF, LEF, and corticosteroids are among the traditional immunosuppressive drugs that are most frequently utilized. In individuals who continue to be resistant or intolerant to these treatments, biologic medications such as tocilizumab, rituximab, and TNF inhibitors appear to hold possibilities. In TA, antiplatelet therapy may also reduce the incidence of ischemic events. When there is serious artery stenosis in a short tract, balloon angioplasty or stent graft replacement could be appropriate. However, surgical bypass of the afflicted segment is necessary for long-segment stenosis with severe periarterial fibrosis or blockage. This procedure is certainly associated with better outcomes than endovascular treatments. In general, during the active stage of the illness, endovascular intervention and surgical operations should be avoided.

Keywords: Takayasu arteritis, granulomatous, intimal proliferation, aneurysms, pulseless disease, angiography

Introduction
Takayasu arteritis (TA) is the main uncommon granulomatous large vessel vasculitis of concealed origin chiefly affecting the aorta and its main branches [1]. Although the exact cause is unknown, research has revealed that an immune response can activate a particular portion of T and B lymphocytes and macrophages, which can cause acute inflammation and artery wall necrosis, which can result in stenosis and aneurysms [31]. Takayasu arteritis is named after the Japanese ophthalmology professor Mikito Takayasu [2]. The disease is referred to the different names like pulseless disease, aortic arch syndrome, idiopathic aortitis, stenosing aortitis, aortoarteritis, and thromboarteriopathy [3]. Although in the beginning believed to be a sickness in general affecting younger girls of Asian descent seeing that it originally described in Japan via way of means Takayasu, Kagoshima, and Onishi at the start of the twentieth century [1]. Throughout the world the disease is widespread but it affects most of the Asia population. Takayasu arteritis was identified in greater frequency in Japan and in lower frequency in the United States was 0.9 per million [4]. The disease's prolonged inflammation and damage to the artery wall can result in stenosis, the creation of aneurysms, and arterial occlusion, among other problems. Because of these complications, it may lead to organ ischemia, hypertension, and sometimes life-threatening events like myocardial infarction or stroke. This disease pathogenesis includes autoimmune-mediated inflammation, vascular remodeling, and endothelial dysfunction [5].

Symptoms

In the early stages of the disease, the non-specific constitutional symptoms are fever, malaise, weight loss [6], night sweats, loss of appetite, headache, dizziness, arthralgia, and skin rashes [7].

Further inflammation leads to the arteries progressing, which results in segmental stenosis, occlusion, dilation, aneurysms [6], reduced blood flow, angina, hypertension, secondary to renal artery stenosis, limb claudication, and neurological symptoms due to artery insufficiency and complications are hardening and narrowing of blood vessels, can cause reduced blood flow to organs and tissues [8].

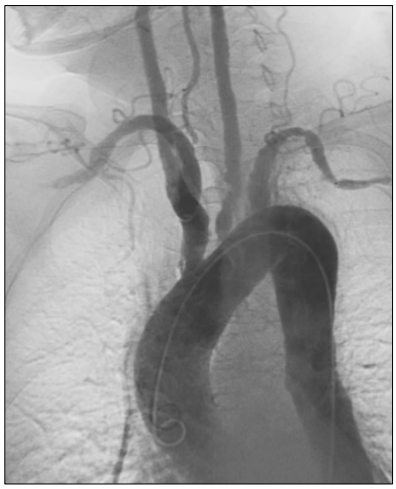

Fig 1: Takayasu arteritis

Clinical features

The scientific capabilities were properly documented with the aid of cohort research of over 570 sufferers from distinct countries. A two-stage mechanism has been proposed: a "pre-pulseless" phase with non-specific inflammatory characteristics, followed by a chronic phase with vascular insufficiency. Some people experience sporadic flares; however, this is not always the case. A degree system has been counseled with a “pre-pulseless” section characterized by the aid of non-particular inflammatory capabilities, accompanied with the aid of using a continual section with the improvement of vascular insufficiency, in a few instances accompanied by the aid of intermittent flares, even though now no longer all sufferers agree to this pattern [9]. 13 to 14% of patients are suffering with non-specific arthralgia and myalgia and it is difficult to differentiate the symptoms of other rheumatic disorders which includes polymyalgia rheumatica, giant cell arteritis and rheumatoid arthritis [10].

Classification

A try has been made to categorize the sickness based on angiographic findings. The early system, revised through Lupi-Herrera *et al* in 1977, has been outdated through the new type of Takayasu arteritis. These systems are beneficial in that they permit a contrast of patient traits in line with the vessels worried and are useful in making plans for surgery, however, they provide little thorough manner of prognosis [11].

Table 1: New Angiographic Classification of Takayasu arteritis, Takayasu Conference 1994 [11, 15]

Type	Vessel involvement
Type 1	Branches from the aortic arch
Type 2a	Ascending aorta, aortic arch, and its branches
Type 2b	Ascending aorta, aortic arch and its branches, and thoracic descending aorta
Type 3	Thoracic descending aorta, abdominal aorta, and renal arteries
Type 4	Abdominal aorta and renal arteries
Type 5	Combined features of type 2b and 4

Table 2: Ishikawa clinical classification of Takayasu arteritis [12, 14]

Group	Clinical features
Group 1	Uncomplicated disease, with or without pulmonary artery involvement
Group 2a	Mild/moderate single complication together with uncomplicated disease
Group2b	Severe single complication together with uncomplicated disease
Group3	Two or more complications together with uncomplicated disease

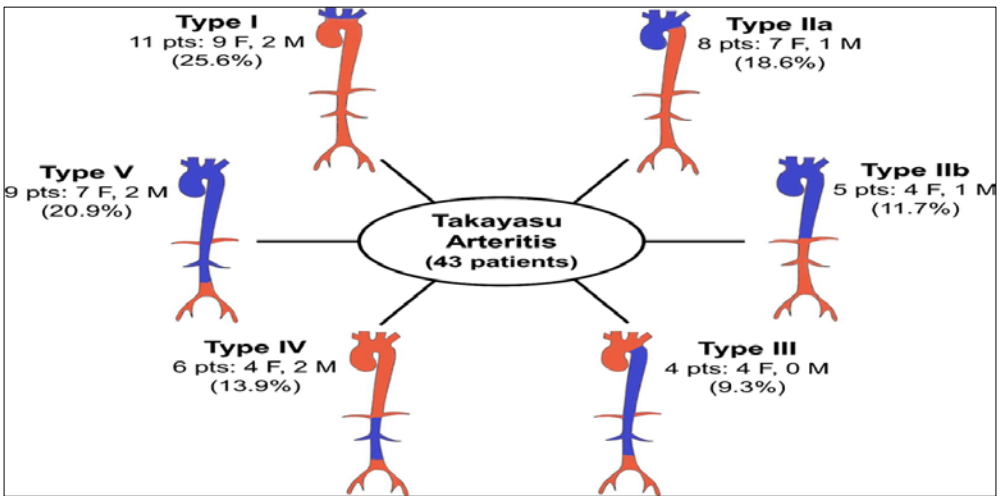

Fig 2: Angiographic classification of Takayasu arteritis

Epidemiology

In Japan, the female-to-male ratio for TA is 8-9:1, indicating that the disease mostly affects young Asian women. Western estimates have indicated occurrences ranging from 0.4 to 2.6 every one million people. The finding is that the majority of TA patients are from Japan-, and that the prevalence of TA is 100 times higher in Eastern Asia than in Europe and the US, with a peak age of onset between 15 and 30 years old. Provides evidence for a genetic foundation [29]. TAK is widely distributed but has a varied distribution; it is typically more prevalent in Asian populations. According to a recent comprehensive study, the global prevalence ranges from 3.2 to 40 instances per million people, with an annual incidence of 0.4 to 2.6.TAK is characterized by a majority of women, while female-to-male ratios differ by region, ranging from 12:1 in Turkey to 3:1 in China and India. TAK patients often report the disease's beginning between the ages of 20 and 30, while it is not unusual for it to occur later in life between 9% and 32% of cases occur after 40 years [30].

Pathophysiology

It is a panarteritis. These include a complex interplay of immune-mediated processes, vascular remodeling, and genetic factors. In the early phase, there is a presence of active inflammation and necrosis; and also followed by mononuclear cell infiltration, lymphocytes, histiocytes, plasma cells, with edema. This indicates the presence of intense inflammatory response and tissue damage in the affected arteries. These lead to chronic inflammation-development of stenosis and occlusion and weakening of arterial wall, leading to aneurysm formation. Cell-mediated autoimmunity plays a role in TA. The following cells play a role in TA macrophages, gamma-delta T-cells, cytotoxic cells, and T-helper cells. In the active state, a study of peripheral blood cells showed increased levels of $CD4^+/CD8^+$ lymphocytes, and basal activity of protein kinase C and intercellular calcium state [5]. Active lesions typically have edema and inflammatory infiltrates containing dendritic cells, macrophages, and NK cells, and T cells (αβ, γδ).

Granulomatous response with giant cells, necrosis in the media and adventitia, intimal fibro cellular hyperplasia, and thrombus development, with subsequent degenerative alterations resulting in the weakening of the muscle layer and the establishment of aneurysmal formation [3].

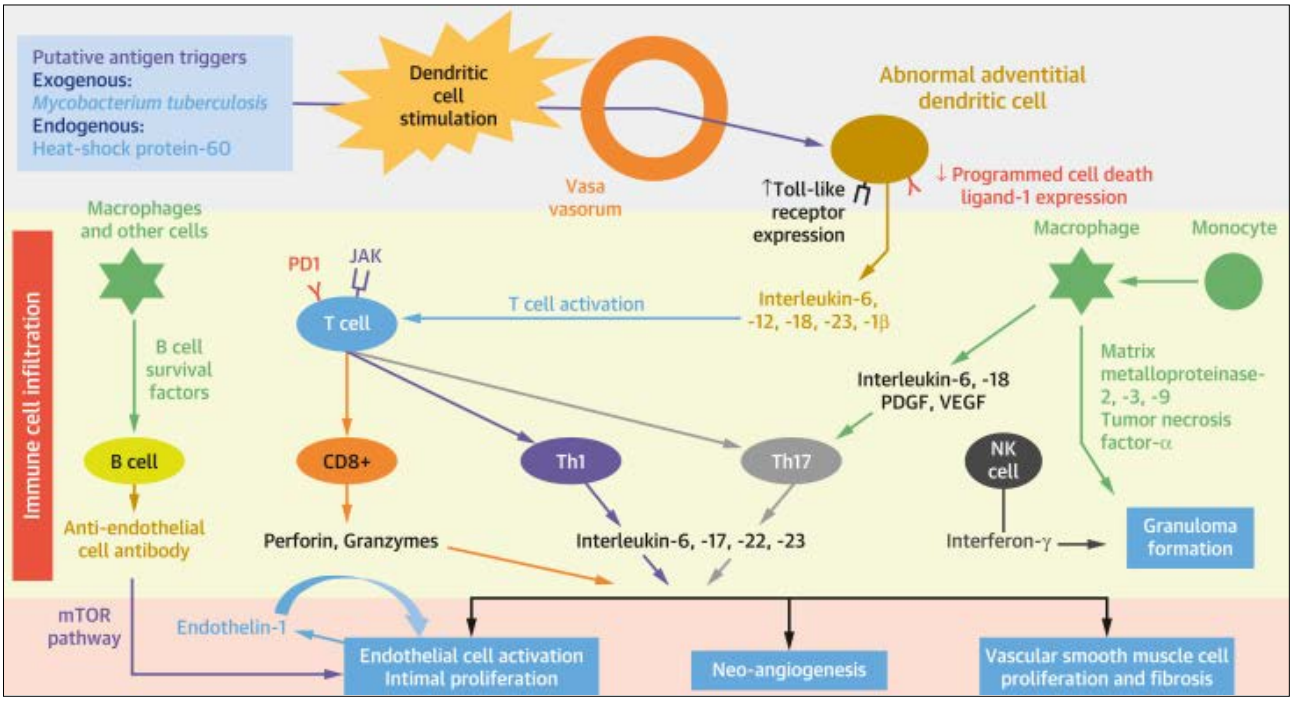

Fig 3: Proposed immunopathogenesis of Takayasu arteritis

Pathogenesis

Takayasu arteritis etiology is unclear, however, its link with the HLA complex suggests a genetic component to its pathophysiology. In Indian patient research, the G allele at TNF-α-308 was discovered to be more common in TA patients than in controls, but the A allele was found to be comparatively less common in study participants than in patients with the condition from Western individuals. Several researchers have proposed that infection plays a pathogenic role, however there is currently inconclusive data to support this. Reports of TA in HIV patients, following influenza vaccination, a patient experienced arteritis of both carotid arteries, as described by Watanabe *et al.* There has been a case report of post-hepatitis B vaccination. Likewise, there is ongoing debate on the involvement of tuberculosis (TB) in TA. Numerous case studies that have been published have demonstrated that a different percentage of TA patients have evidence of a prior or concurrent Mycobacterium TB infection. In a Brazilian study of 71 children with TA, 32% (23 patients) got anti-TB medications for suspected or diagnosed TB. In a brief sample of nine children with Chinese TA, four out of nine had tuberculosis before the development of symptoms. The IS6110 and HupBgene sequences linked to M. tuberculosis were found in the aorta tissue of TA patients, according to a Mexican case-control study. The authors speculated on the pathogenic involvement of tuberculosis in the development of arteritis. It has been proposed that the human 65-kDa HSP and the mycobacterial 65-kDa HSP are molecularly similar. This might trigger a cross-reaction mediated by the

immune system and trigger an autoimmune reaction. Numerous writers have documented the existence of T cells that are reactive to both mycobacterial and human homologous 65-kDa HSP, together with serum IgG antibodies targeting both mycobacterial and human 65-kDa HSP, in TA patients. Moreover, in aorta biopsies from TA patients, the 65-kDa HSP has been isolated from the intermediate layer and vasa vasorum. According to Chauhan *et al*., individuals have circulating anti-aortic endothelial cell antibodies (AAECAs) against 60-65 kDa HSP. TA development may include many immunological pathways. Inflammation and tissue damage are caused by humoral immune responses as well as cell-mediated ones. Humoral immunity is implicated in both autoantibody-producing B cell infiltrates in inflamed arteries and circulating anti-endothelial cell antibodies (AECA). In patients with active disease, complement and cell-mediated cytotoxicity by AECA have been observed. Patients with TA had elevated serum levels of TNF-α, IFN-α, interleukin-6 (IL-6), IL-8, IL-17A, and IL-18. Specifically, there is a connection between disease activity and blood levels of IL-6, IL-12, and IL-18, and high expression of IL-6 in reports of aortic tissue from TA patients. According to Misra *et al.*, TA patients had higher serum levels of IL-17 and IL-23 and an observed increase of Th17 cells when compared to healthy controls [3].

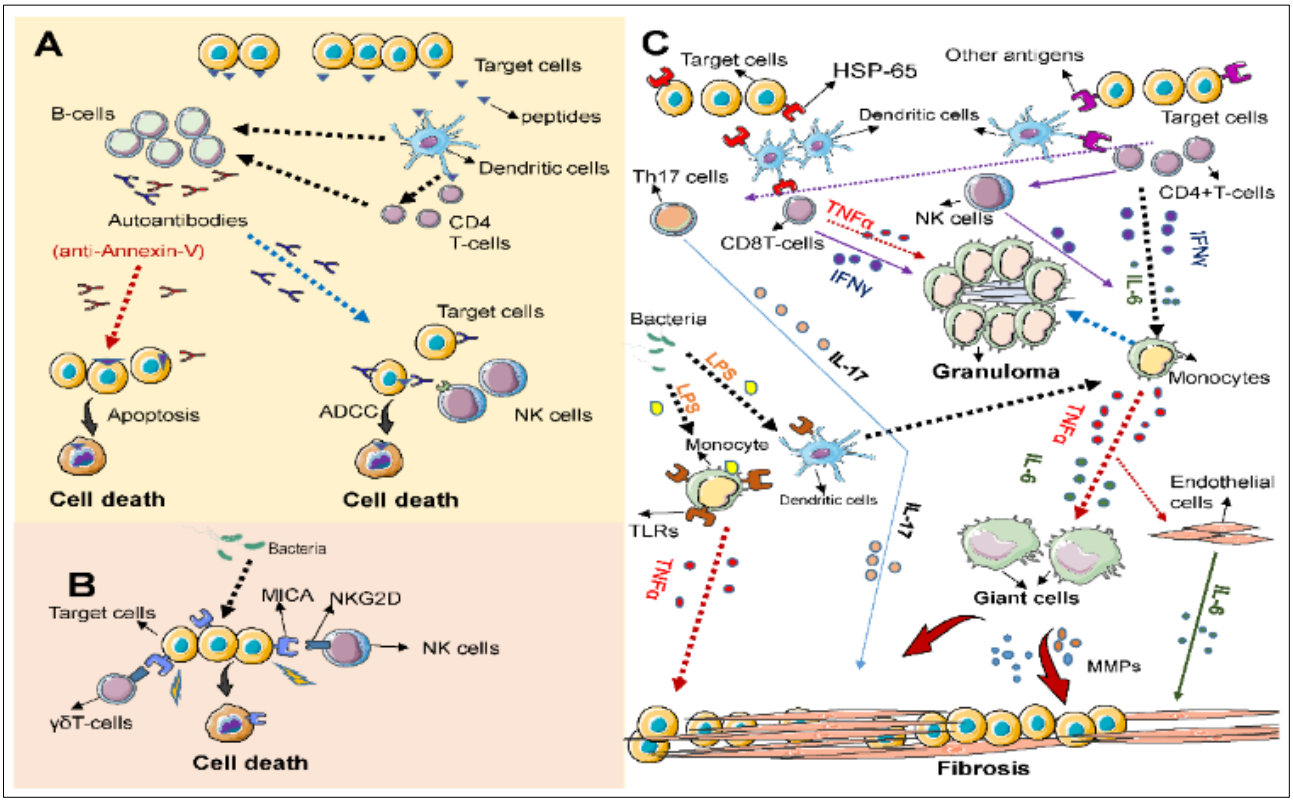

Fig 4: Current understanding of the pathogenesis of Takayasu arteritis

Diagnostic Criteria

The crucial element in diagnosing Takayasu arteritis involves maintaining a clinical suspicion, necessitating a thorough history and comprehensive physical examination. Imaging plays a pivotal role, in the following diagnostic Parameters.

Computed Tomography Angiography (CTA)

Computed Tomography Angiography (CTA) is now established as the primary method for the initial assessment of disease distribution, replacing conventional angiography as per the 1994 Tokyo International Conference Classification of Takayasu arteritis. CTA enables the visualization of vessel wall thickening and luminal narrowing [26].

Magnetic Resonance Imaging (MRI)

Magnetic Resonance Imaging (MRI) and Magnetic Resonance Angiography (MRA) also prove valuable in identification, offering excellent multiplanar imaging without ionizing radiation. However, it's important to note that MRA, while effective, tends to overestimate the degree of stenotic disease, and access to MRI or MRA can be more challenging [13].

It has been determined that conventional angiography is required for the diagnosis of TA and its consequences. However, it has recently been demonstrated that CT and MRI (Particularly CT and MR angiography) can identify aortic mural alterations that are challenging to identify with traditional angiography. As such, these methods serve as substitutes for traditional angiography. MRI is superior to CT in several ways:

It is more sensitive in detecting mural edema,

It is non-nephrotoxic and rarely causes anaphylactic reactions,

It does not involve ionizing radiation, soft-tissue differentiation is improved, mural edema can be seen with greater sensitivity using MRI, and cine MRI can show aortic regurgitation. Nevertheless, there are drawbacks to MRI as well, such as poor visualization of arterial calcification and trouble seeing tiny branch arteries. Further tests might be necessary if vascular calcification or the visibility of tiny branch arteries is required. Furthermore, MR angiography may exaggerate the severity of vascular stenosis [22].

Angiography

Angiography is the most accurate way to diagnose TA. It permits clear pictures of the vessel lumen even though it is unable to recognize alterations to its walls. Based on their arterial distribution, six different forms of vascular involvement have been defined. However, given the procedure's intrinsic invasiveness and the fact that it lacks the properties of the artery wall, this treatment has limits. Its application is now restricted to certain indications, such as arterial revascularization preparation or central artery pressure monitoring when peripheral performance is not feasible [24].

Positron emission tomography

Positron emission tomography (PET/CT) is a popular and sensitive imaging technique, but it is costly and involves radiation exposure. The usage of 2-(F)-fluoro-2-deoxy-D-glucose is necessary. (F-FDG), which measures the degree and type of inflammation in the artery wall in TA, and is taken up by metabolically active cells, primarily monocytes and macrophages. It's unclear how F-FDG-PET/CT will be used to monitor TA patients at this time. According to a meta-analysis of many TA studies, the absorption of F-FDG had a low specificity of 73% and sensitivity of 87% [23].

High-Resolution Ultrasound

The most often utilized approach for managing TA patients is high-resolution ultrasonography (HRUS). This is its low cost, good toleration, and ability to differentiate the artery wall of the lumen, measure intima-media thickness (IMT), and identify the extent of stenosis or aneurysms are its main advantages. Its drawbacks include the fact that it is operator-dependent and can only assess the proximal and axillary subclavian arteries, as well as the vertebral and carotid circulation. Nonetheless, it can be applied to assess the abdominal aorta in TA patients. When TA patients undergo HRUS, concentric thickening of the artery wall may be seen; this thickening is frequently bright because of edema and active inflammation. The correlation between disease activity and wall examination findings is not yet fully understood. However, IMT has demonstrated to High-Resolution Ultrasound decrease in response to successful therapy. Currently, the diagnosis, treatment, and follow-up of TA patients have significantly improved with the use of imaging techniques; however, further research is required to determine the disease's activity, changes in the course of the illness before and after immunosuppressive therapy, and its relationship to plasma biomarkers and disease activity scores [25].

Table 3: Diagnostic criteria

Modality	Accessibility	Ease of use	Purpose/use	Radiation	Principal limitation
DSA	Low	Low	Diagnosis/treatment	yes	Invasive, lack of information on the vessel wall
CTA	High	High	Diagnosis/monitoring	yes	Cannot be used in patients with renal failure or allergies to contrast media
MRI	Low	High	Diagnosis	No	Cannot be performed when some types of metals are present in the body or when patients with claustrophobia
DUS	High	High	Diagnosis/monitoring	No	Examiners technical proficiency strongly affects the result, subjective, acoustic shadow
PET	Low	High	Diagnosis/monitoring	yes	Lack of criteria for positivity (FDG uptake) low resolution for small vessels.

Management of Takayasu arteritis

Treatment

Corticosteroids and Methotrexate (MTX) are the best evidence treatment and are the first-line treatment for TA. Cooperation between the patient and the doctor and educating the patient is important for managing Takayasu arteritis. The main aim of the medical treatment is to suppress systemic and vascular inflammation by using immunosuppressive agents and corticosteroids. The cornerstone for the induction of remission in TA is glucocorticoids when methotrexate and azathioprine fail, biological immunosuppressive medications such as infliximab, adalimumab, and tocilizumab are frequently used. For certain patients, the reduction of symptoms resulting from persistent damage necessitates procedures such as surgery, angioplasty, and stent implantation. The role of intravenous immunoglobulin, recombinant IL-1 receptor antagonists, IL-4, and transforming growth factor is unclear [13]. The most effective way of treating Takayasu arteritis is steroids with steroids alone, the treated patients were shown remission of about 60% and had associated side effects which led to the search for effective treatment [9].

This treatment has been compared to other systemic vasculitis, including Wegener's granulomatosis. Thus, immunosuppressive medications such as methotrexate, azathioprine, and cyclophosphamide have all been tested. However, it's challenging to draw comparisons between Wegener's granulomatosis and Takayasu arteritis because of the distinct morbidity and mortality rates linked to each condition, in addition to the differences in the vascular diameter impacted by the disease process. The typical survival time for untreated systemic Wegener's granulomatosis is five months from the time of disease onset, and the one-year mortality rate is 82% [9]. The combination of glucocorticoids with antihypertensive and antiplatelet drugs has shown good efficacy in medical treatment. In primary treatment, surgical revascularization is rapidly evolving [19]. A recent study revealed that 50% of patients with Takayasu arteritis will relapse and experience a vascular complication less than 10 years from diagnosis. Those most likely to relapse include males and those with elevated C-reactive protein.[19] Corticosteroids respond to 50% of patients, and methotrexate responds to an additional 50%. In individuals who do not respond to traditional immunosuppressor medications, mycophenolate mofetil, an inhibitor of guanine nucleotide synthesis, and biological therapies that block tumor necrosis factor alpha have been suggested more recently. Treatment of vasculitis-related side effects, including thrombosis and hypertension, is another factor. Due to their fluid-retaining adverse effects, corticosteroids can make treating hypertension more difficult. In addition, renal artery stenosis prevents the use of angiotensin-converting enzyme inhibitors. When a patient's stenosis, occlusion, or, less frequently, aneurysm, develops complications similar to those of other diseases, surgery is required. Surgery should be done when the patient

is in remission to prevent complications from inflammation, such as restenosis, anastomotic failure, thrombosis, bleeding, and infection [20].

Surgery

Arterial reconstruction is a surgical option for TA. It depends on where lesions lie and the patient's surgical anatomy. The indications to perform surgery have a high risk which benefits the following

1. Uncontrolled hypertension
2. Cerebrovascular disease
3. Severe aortic regurgitation
4. Occlusive lesions [11].

Revascularization of affected organs both via surgical procedure or endovascular procedures, consisting of an angioplasty balloon, stent, or graft alternative stents, constitutes the principal procedure for the continual section of TA [10]. In addition, using an antiplatelet cure needs to be taken into account, and although its use no longer lowers the frequency of ischemic occasions in TA, it can lessen the danger of restenosis. Therefore, 6 months of antiplatelet treatment is recommended, as properly as post-surgical treatment with immunosuppressants to increase the fee of success results. The surgical complication rate was 37.5% in contrast with the endovascular repair rate was 62%. The method of percutaneous transluminal angioplasty is evolving. More effective and safe substances adapted to small patients make it a valid choice to deal with those lesions. Nevertheless, in Takayasu arteritis, the vessels are firmer, scarred, and fibrotic and bring the threat of excessive headaches mainly while interventions are completed on the cerebral arch vessels. Comparing the results after endovascular therapy and restenosis charge in atherosclerotic disorder and Takayasu arteritis showed greater variability and restenosis within side the latter [18]. The rate of surgical intervention varies between centers, ranging from 12% to 70%, but it appears that less than 20% of patients with TA require revascularization. Uncontrolled hypertension due to renal artery stenosis, severe symptomatic cerebrovascular disease or coronary artery disease, severe aortic regurgitation or coarctation, stenotic or occlusive lesions causing critical limb ischemia, and aneurysms at risk of rupture are among the conditions that warrant consideration of intervention. Surgery has an excellent risk-benefit ratio in these instances. According to Ishikawa and Mahtani's prognostic classification, Miyata *et al*. showed that surgery improves long-term life for patients with stage 3 TA (Severe complication and advancing disease), but decreases survival for stage 1 patients (No significant side effects and no indication of a developing illness) as a result of surgical complications. For patients with stages 1 and 2, conservative medical therapy is therefore advised [21].

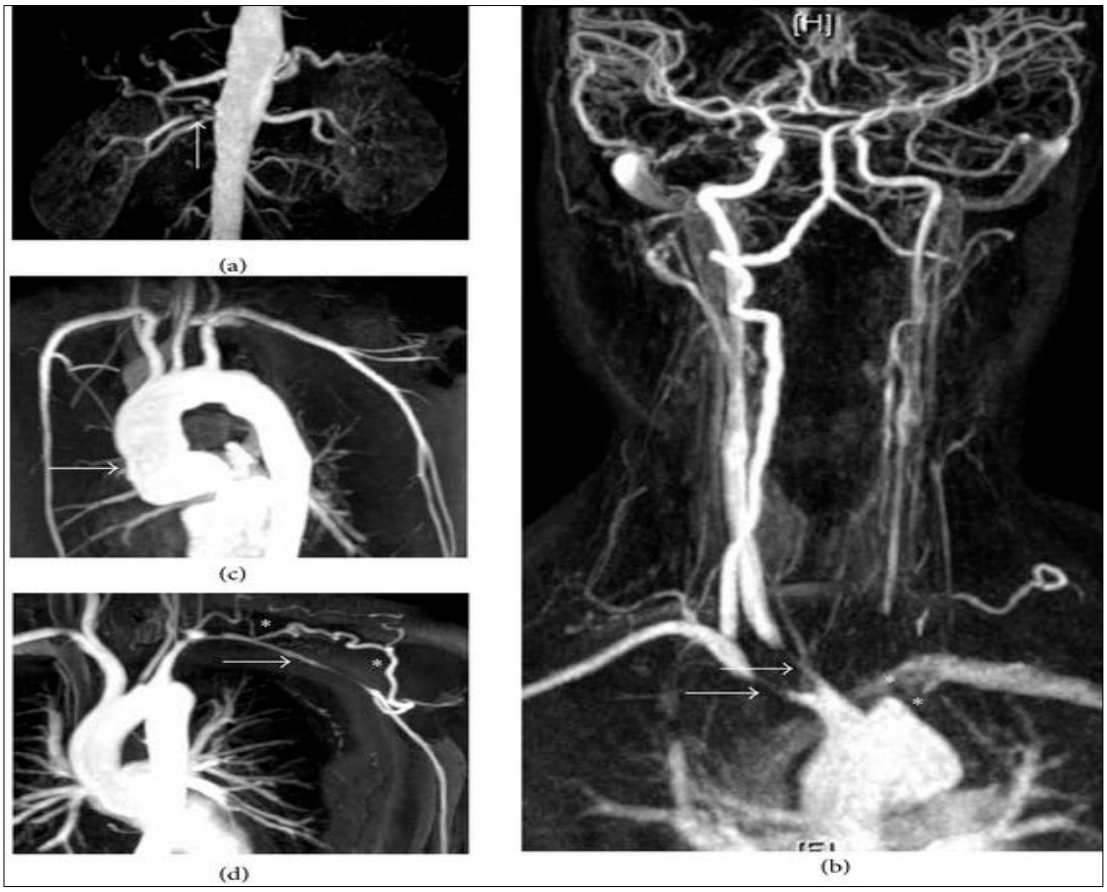

Fig 5: a) A right renal artery stenosis was subsequently successfully treated by percutaneous angioplasty with resolution of hypertension. b) Severe disease of the great vessels including stenoses of the right subclavian and common carotid arteries (Arrows) and occlusion of the left subclavian and left common carotid arteries (Stars). The patient suffered severe cerebrovascular symptoms and underwent successful bypass surgery. (c) A dilated ascending aorta with associated severe aortic regurgitation in a young female patient who required aortic valve replacement. (d) Although patients commonly suffer claudication symptoms as a consequence of subclavian artery stenosis (Arrow), these often improve following the development of collaterals (Stars) as shown here, precluding the need for intervention.

Recent advances in takayasu arteritis

Tocilizumab

The TAKT study, a Phase 3 randomized control trial from Japan carried out by Nakaoka *et al*., randomly assigned 36 patients to receive background glucocorticoids in addition to either tocilizumab (TCZ) or a placebo. Though it narrowly missed statistical significance, the tocilizumab group's primary endpoint-the time to relapse-was longer. The majority of the 36 patients in the long-term extension research showed radiological stabilization at 96 weeks, glucocorticoid sparing effect, and clinical improvement while receiving weekly subcutaneous TCZ [26].

TNF- α- inhibitors

Ferfar *et al*. listed 13 studies including 96 TA patients receiving TNF inhibitor therapy (Adalimumab, etanercept, and infliximab). 39% of patients discontinued taking their glucocorticoids, 61% experienced a clinical improvement, and 3 patients' MRA lesions had regressed. During a 24-month follow-up period, 28 relapses were documented. The significant risk of tuberculosis reactivation associated with TNF inhibitors is concerning, particularly in endemic regions. After a meta-analysis of eighteen observational studies, the combined data showed that 81% of patients on TNF inhibitors were able to obtain a partial response in clinical characteristics, 86% had angiographic stabilization, and 32% experienced relapses. However, there was a noticeable disparity in the results between the several investigations [26].

JAK inhibitors

One important mechanism in the pathophysiology of TAK is the JAK/STAT signaling combination. The first JAK inhibitor to be licensed for use in the treatment of RA is tofacitinib as stated by Japan. According to guidelines from the College of Rheumatology, its use is restricted to RA patients for whom methotrexate is not sufficiently effective. Numerous clinical studies have shown their effectiveness and safety in TAK [27].

Rituximab

In a retrospective analysis, 2 grams of rituximab were administered as induction therapy and maintained doses every 6 months to 7 TA patients who had shown resistance to glucocorticoids and other conventional synthetic and biological DMARDs. Of the seven patients, three experienced complete remission; the other four continued to have radiographic progression and chronic disease. The use of rituximab in TA is not supported by the study's findings. On the other hand, previous case studies revealed that eight of nine individuals met the clinical and radiological criteria [26].

Abatacept

In a multicentric double-blind randomized control trial, 26 patients who achieved remission at week 12 were randomly assigned to receive monthly abatacept or placebo with a background glucocorticoid taper. Thirty-six patients got abatacept at a dose of 8 mg/kg. At 12 months, 22% of patients in the abatacept group attained the primary outcome of relapse-free survival, compared to 40% in the placebo arm. The results of the study showed that adding abatacept to a regular regimen had no benefits [27].

Ustekinumab

Ustekinumab is a monoclonal antibody that inhibits both Th1 and Th-17 responses, which are important inflammatory drivers in the pathophysiology of GCA. It targets the components common to IL-12 and IL-23 (p40). In one research, the median daily prednisolone dose was lowered from 20 to 5 mg over a year, and improvement of vasculitis was shown using CT imaging. All 25 patients with resistant GCA who received Ustekinumab in addition to GC remained in remission. Seven of the initial eleven patients in the second study experienced a relapse, leading to an early termination that has not yet been reported in abstract form [28].

Conclusion

In this study, the features of Takayasu arteritis were examined. A summary of radiological findings, together with some physical findings and the values that correspond to them, have been listed. Takayasu arteritis more frequently affects women, with an average onset age of 13 to 38 years, according to our examination and analysis of all case reports on the condition. Most patients present with a variety of symptoms, ranging from fever to claudication indications and related symptoms. The findings demonstrated the variation in blood pressure readings between two different limbs, mainly the radial pulse, that had a diminished or missing pulse. If a patient has elevated CRP and ESR values and is symptomatic, Takayasu may be a differential. Imaging is the primary diagnostic tool since it shows problems such as significant vessel stenosis. There might be some alterations in cardiac function as well as associated effects due to clot formation. This is an overview of Takayasu's arteritis and what medical professionals might anticipate when managing the condition.

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