International Journal of Research in Medical Sciences
Bishnoi RK et al. Int J Res Med Sci. 2020 Sep;8(9):3418-3425
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: http://dx.doi.org/10.18203/2320-6012.ijrms20203706

Review Article

Takayasu arteritis: a comprehensive review of literature

Ravin K. Bishnoi[1]*, Shivani Devi[1], Shivani Rai[2], Gunjot[3]

[1]Department of Nursing Services, All India Institute of Medical Sciences, Rishikesh, Uttarakhand, India
[2]Department of Obstetrics and Gynaecological Nursing, Metro College of Health Sciences and Research, Greater Noida, Uttar Pradesh, India
[3]College of Nursing, All India Institute of Medical Sciences, Rishikesh, Uttarakhand, India

Received: 07 July 2020
Accepted: 31 July 2020

***Correspondence:**
Mr. Ravin K. Bishnoi,
E-mail: bishnoiravin@gmail.com

ABSTRACT

Takayasu arteritis is an inflammatory pulseless disease of large arteries with unknown etiology which advances in three stages i.e. early systemic, vascular, and burnout stage. It is more prevalent in Asian women of childbearing age. Hypertension, fever, weight loss, arthralgia, limb claudication, light-headedness, and arterial pain are common manifestations. Angiography is a gold standard test to evaluate TA. There is no reliable serological marker has been identified. Control on the inflammatory process and hypertension are two imperative angles to treat the disease. Steroids are most used. Reconstructive surgeries are limited to severe and stenotic lesions. This review aims to report comprehensive evidence about Takayasu arteritis. We conducted an integrative review of theoretical and empirical publications reporting epidemiology, etiopathogenesis, classification, diagnostic evaluation, and management of TA. The authors searched PubMed, Embase, and Scopus until March 2020. A total of 1104 records found, we included 37 papers for review after reading the articles. Remaining was excluded because of no innovative content, insufficient details, and no clear endpoints. This review of the literature presents comprehensive evidence in all fields of TA. Still, large areas need to be studied for better management of patients with TA.

Keywords: Aneurysms, Arteritis, Pulse, Pulseless disease, Takayasu's

INTRODUCTION

The first published description of Takayasu's arteritis is from 1908 when two Japanese ophthalmologists, Takayasu's and Onishi discovered retinopathy occurring with absent limb pulses.[1] It is also known as pulseless disease, occlusive thromboaortopathy, and Martorell syndrome.[2] The effect of the inflammatory process of the disease has been seen in the major arteries and aorta. This also includes the renal arteries, carotid arteries of the head and brain, subclavian arteries that furnish the upper limbs, and coronary arteries.[3,4] It is the primary arteritis of the obscure cause, which usually occurs in the women of the reproductive age.[1] Wall thickening, fibrosis, stenosis, and clots prompt to vessel irritation adding to end-organ ischemia as manifestation. Increasingly acute inflammation annihilates the arterial media and leads to aneurysm development.[5] The arterial lesions can lead to secondary hypertension, retinopathy, cardiac involvement, cerebrovascular events, and premature death.[3]

This review aimed to study and report the new findings of epidemiology, etiopathogenesis, clinical presentations, diagnostic criteria, associated bio and serological markers, and management of patients with Takayasu arteritis.

METHODS

We decided to include all the publications describing global and Indian epidemiology, etiopathogenesis, stages, diagnostic criteria, bio and serological markers, and

management of patients with Takayasu Arteritis. We excluded all the articles with other than the English language and with no innovative content and insufficient details. Two reviewers performed a search of PubMed, Embase, and Scopus databases till March 2020 using the keyword: "Takayasu arteritis". Two reviewers independently assessed the title and abstract of the potentially eligible studies. A total of 1104 records found, and we included 37 papers for review after reading the full text of articles. Out of these ten were about epidemiology, five described etiopathogenesis, eleven explained about clinical presentation and manifestations, four were about stages of TA, six explained diagnostic criteria, and 13 described the medical and surgical management of patients with TA.

REVIEW OF LITERATURE

Epidemiology

Besides having worldwide spread, TA found as often as possible in Asia.[6] The average age of appearance of TA is commonly in the second and third decades of life with prominent occurrence in Asian women. [1]Although both sexes may be affected, 80-90 percent of patients are female.[4]

Because of the rare occurrence of TA (2-3 per million), limited data is available on its epidemiology. The estimated incidence of TA was 2.2/million in Kuwait and 1-2/million in Japan. The highest ever prevalence of TA at 40 per million was observed in Japan and the least at 0.9 per million in the US. In the European population, the published prevalence varies from 4.7/million to 33/million. This variation may be due to different geographical and heredity contrast.[7]

Furthermore, Renovascular hypertension is most caused by TA in India, Japan, Korea, China, and different nations of South East Asia.[6]

Indian scenario

The incidence and prevalence of data from Indian literature are insufficient and unreliable.[8] The reported ratio of male to female is 1:1.58 in Indian patients. Indian female patients have greater involvement of aortic curve including its branches, whereas male patients have a greater occurrence of hypertension and involvement of abdominal aorta.[9] Pregnant women with TA are more likely to cause serious complications for both maternal and fetal life.[10] Most patients in India presented with hypertension at the time of the visit.[6,9] The probable reason for hypertension is the association of renal arteries which found in 20 to 90% of the cases.[11] As globally, Indian patients also present it in the third decade of life.[2,9] Jain et al reported in their study that hypertension is the commonest mode of presentation and was found in 77.4% at the time of presentation.[12]

Etiopathogenesis

Takayasu's arteritis is an inflammatory disease of large arteries with unknown etiology.[13] The inflammatory lesions emerge from the vasa vasorum followed by cellular infiltration that chiefly comprised of T cells, natural killer cells, dendritic cells, monocytes, and granulocytes which intruding the outer layer of tunica media or to its neighbouring adventitia. By inducing lymphocyte infiltration and promoting endothelial cells activation, the inflammatory cells release interleukin-6, interleukin-1, and RANTES (Regulated on Activation, Normal T Expressed and Secreted) within the damaged cells.[14]

The HLA-B*52 is the only genetic factor that is known to be consistently associated with TA. Various Asian studies confirmed that the main risk factor which is associated with a high prevalence of TA in the Asian population is the HLA-B*52. Again, it may depend on the ethnicities and geographical differences. The European population has less prevalence of TA with a frequency of HLA-B*52 is <2%. Whereas in the Japanese population TA is more prevalent (40/million) with the frequency of HLA-B*52 is 10%.[13] An unknown stimulus triggers the expression of the Heat-shock protein (hsp-65) (produced in stress) in the aortic tissue which produces major histocompatibility on vascular cells by the release of perforin, resulting in acute vascular inflammation. Pro-inflammatory cytokines are released and increase the mononuclear cells in the vascular walls.[15]

BCG immunization is also considered as an activating factor for TA in a vulnerable population. A heat shock protein (hsp-65) is a component of both Mycobacterium tuberculosis and BCG, which is responsible for injury to the vessels. Active tuberculosis infection is reported in 60 % of autopsies of non-specific aortitis which explains the infectious etiology.[9] Mainly because of sensitivity to infective agents, autoimmune disturbances, the mycobacterium tuberculosis have been involved in its pathogenesis.[16]

Clinical features

Symptoms and signs

The onset of symptoms is sub-acute.[17]Approximately 10% of Takayasu arteritis patients show no symptoms. [18]The common symptoms reported by patients are pain in arms or legs i.e. limb claudication, lightheadedness, arterial pain, or tenderness because of narrowing, occlusion, or reduced blood flow.[17] TA includes the following symptoms:

Constitutional symptoms

Clinical vascular involvement may be preceded by constitutional symptoms. These symptoms are common in the early phase of TA which includes weight loss (10-

18%), malaise (35-65%), headache (50-70%), low-grade fever (9-35%), and arthralgia (28-75%).[18]

Cardiac and vascular features include the following

Hypertension

Hypertension occurs in more than one-half (28-53%) of the cases due to the narrowing of one or both renal arteries and decreased elasticity of the aorta and branches.[18] This is most commonly occurring by three mechanisms like atypical coarctation of aorta due to high resistance, aortic arch baroreceptors readjustment, and renal hypoperfusion. [19]Pulmonary hypertension is a rare manifestation of TA. This mainly occurs because of the involvement of the distal aorta and pulmonary artery which leads claudication in lower extremities and pulmonary hypertension.[4]

Arterial bruit

Studies found that patients with TA may have loud S2 at the base and systolic bruit at supraclavicular fossa.[4] Bruit is present in most of the cases (80%) with the most common location being the carotid artery.[18] Bruits are usually audible over the subclavian artery, brachial arteries, carotid arteries, and abdominal aorta.[17]

Pulseless disease

Limb claudication and blood pressure discrepancies of extremities cause diminished or absent pulses in about 84-96% of patients.[18] The salient features distinctly lower blood pressure and weaker pulses in the upper extremities with coldness or numbness of the fingers may occur. Therefore, TA is also named as a "pulseless disease".[2]

Congestive cardiac failure associated with aortic regurgitation, hypertension, and pericarditis.[4]

Neurological symptoms

Neurological manifestations are common symptoms in TA patients in the chronic phase.[20] These symptoms occur mainly due to stenosis of carotid and vertebral arteries which causes decreased cerebral blood flow and leads to cerebral ischemia which is characterized by headache in 50-70% of cases, vertigo and convulsions in 0-20% and stroke in 5-9% of cases.[21] Visual disturbance is a late manifestation and strongly associated with common carotid and vertebral artery disease.[3] Neurological symptoms are secondary to hypertension, postural dizziness, and amaurosis.[22]

Dermatological manifestations

These symptoms include mainly skin lesion resembles erythema nodosum or pyoderma gangrenous and ulcerated sub- acute nodular lesions found over the legs in minority cases.[3,17]

Respiratory symptoms

Symptoms related to the pulmonary system are less common. Pulmonary arteries are involved pathologically in up to 50% of cases. Aortic regurgitation, aortic dilation, or malignant hypertension results in chest pain, dyspnea, hemoptysis, and pulmonary hypertension.[17]

Ocular manifestation

The prevalence of ocular involvement in TA is varied ranging from 8.1% to 68%.[23] Uyama and Asayma had given four stages of ocular involvement.

- Stage 1- Vein distension.
- Stage 2- Formation of microaneurysm.
- Stage 3- Formation of arterio-venous anastomoses.
- Stage4- Ocular complications like cataract, neovascularization, anterior ischemic optic neuropathy, central retinal artery occlusion vitreous haemorrhage, and ocular ischemic syndrome.[24]

Takayasu arteritis in pregnancy

In pregnancy, blood pressure measurement may not be possible due to pulselessness. In this situation, calf pressure needs to be obtained. Where increased blood pressure may lead to subsequent seizures, eye changes, subarachnoid/intracranial haemorrhage, aortic regurgitation, preeclampsia, fatal complications, syncope, and nephrotic syndrome. Pregnancy does not enhance inflammatory activities but the perinatal period may become complicated by the associated symptoms.[18]

STAGES OF TAKAYASU ARTERITIS

Takayasu arteritis progresses through three stages. Symptoms can occur early or late in the progression of the disease.[18] Stages progress with a "pre-pulseless" phase with non-specific inflammatory characteristics to the chronic progression of vascular insufficiency.[2]

Following are the stages of TA.

- Early systemic stage
- Vascular inflammatory stage
- Settles down or burns out stage

Early systemic stage

In this stage patients have signs and symptoms of active inflammatory illness.[25] These include constitutional symptoms like unintended weight loss, fatigue, mild fever, muscle ache, and joint pain, sometimes accompanied by night sweats. Most of the patients have elevated ESR in this stage.[2] Some patients do not experience such symptoms for months or years even as inflammation causes damage to blood vessels and organs.[26] This stage is also considered as a pre-vasculitis stage.[18]

Vascular inflammatory stage

In this stage patients start to develop symptoms due to the narrowing of involved arteries.[25] Signs and symptoms characterizing vascular inflammation and vascular insufficiency include fatigue, palpitation, dyspnea, headaches, pain in joints and extremities, hemoptysis, ulceration, weight loss, bruits, blurred vision, dizziness, fainting, lightheadedness, and seizures. Claudication in legs and arms during activity is rare. Thoracic and lumbar spine pain may occur in this stage but in very rare cases.[18,25,27] Vascular and constitutional symptoms can occur simultaneously.[18]

Settles down or burns out stage

This stage usually occurs when fibrosis sets in. It is associated with remission. This stage does not occur in all patients, even not in suspectable cases of remission and relapse. [18]

DIAGNOSTIC CRITERIA

The diagnosis of TA is based on clinical manifestations and arteriography. The first-ever diagnostic criterion was published by Ishikawa in 1988. In a Japanese study, these criteria showed a sensitivity of 84% and specificity of 100%. In 1995, a modification of Ishikawa's diagnostic criteria was proposed by Sharma et al (Table 1).[14]

A high possibility of the presence of TA is determined by the presence of obligatory criteria and two major criteria or one major and two or more minor criteria or four or more minor criteria. ESR: erythrocyte sedimentation rate.

Diagnostic criteria given by American College of Rheumatology (1990)

It requires at least 3 out of the 6 criteria should meet to diagnose TA. It had a sensitivity of 77.4% and specificity of 95%.

Table 1: Ishikawa's proposed set of diagnostic criteria for TA.[14]

Criteria	Definition
Obligatory criterion	
Age ≤40 years	Age ≤40 years at the time of diagnosis or onset of ''characteristic signs and symptoms of Takayasu arteritis for one-month in patient history.
Two major criteria	
Lesions of the left mid subclavian artery	The most severe occlusion or stenosis present in the central portion from the point one centimetre proximal to the left vertebral artery orifice to that three centimetres distal to the orifice determined by angiography.
Lesion of the right mid subclavian artery	The most severe occlusion or stenosis found in the central portion from the right vertebral artery orifice to the point three centimetres distal to the orifice determined by angiography.
Nine minor criteria	
Increased ESR	Unknown constant increased ESR 20 mm/hour (Westergren) at the time of diagnosis or presence of the evidence in patient history.
Common carotid artery tenderness	Unilateral or bilateral tenderness of common carotid arteries identified by palpation; neck muscle tenderness is undesirable.
Hypertension	Constant high blood pressure of more than 140/90 mmHg (brachial) or 160/90 mmHg (popliteal) at age ≤ 40 years or presence of the history in this age.
Aortic regurgitation or annuloaortic ectasia	Diagnosed by auscultation or Doppler echocardiography or angiography. Diagnosed by two-dimensional echocardiography or angiography.
Lesions of the pulmonary artery	Segmental or lobar arterial stenosis or equivalent identified by angiography or perfusion scintigraphy; or presence of an aneurysm, stenosis, luminal irregularity, or any combination of these in unilateral or bilateral pulmonary arteries or pulmonary trunk identified by angiography.
Lesions of the left mid common carotid artery	Most severe occlusion or stenosis in the central portion of five centimetres in length from the point two centimetres distal to its orifice diagnosed by angiography.
Lesions of the distal brachiocephalic trunk	Most severe occlusion or stenosis in the distal third identified by angiography.
Lesions of the thoracic aorta	Dilation or aneurysm, narrowing, luminal irregularity, or any combination of these diagnosed by angiography; tortuosity alone is unacceptable.
Lesions of the abdominal aorta	Dilation or aneurysm, narrowing, luminal irregularity, or any combination of these and absence of lesion in the aortoiliac region consisting of 2 cm of the terminal aorta and bilateral common iliac arteries identified by angiography; tortuosity alone is undesirable.

Table 2: American College of Rheumatology (1990).[22]

Criteria	Definition
Age at disease onset <40 years	The appearance of symptoms related to TA at the age <40 years
Claudication of extremities	Appearance and aggravating of fatigue and discomfort in muscles of one or more limbs during activity, mainly in upper limbs.
Decrease brachial artery pulse	Decreased pulsation of one or both brachial arteries.
The difference in Blood pressure of more than 10 mm Hg	The difference in systolic blood pressure of more than 10 mm Hg between both arms.
Bruit over the aorta or subclavian artery	Audible bruit on auscultation over the abdominal aorta or one or both subclavian arteries.
Arteriogram abnormalities	Arteriogram showing occlusion or narrowing of the entire aorta and its primary branches or large arteries of the upper and lower limbs which is not caused by fibromuscular dysplasia, arteriosclerosis, or similar factors.

Classification of Takayasu Arteritis

According to anatomic distribution, the disease can be classified into 5 or 6 types (Table 3).[14]

Serological marker and laboratory findings

No definitive serological marker has been distinguished till now for the TA.22As TA is the rare disease of large vessels, its diagnosis is difficult in the early stage. However, a distinct collection of [18F]-FDG in the thoracic and abdominal aorta was observed in a 19-year-old female patient with a complaint of back pain which was examined by [18F]-FDG-PET for detection of the source of inflammation and was diagnosed to have TA. 28 Previously for the remission criterion, ESR normalization was utilized, but currently, researchers found that it has low sensitivity and specificity because ESR has been found normal in 28% of the active patients with TA, while it is increased in approximately half of the TA patients who were in clinical remission.[22] Various serological investigations have been performed including C reactive protein, von Willebrand factor, ESR, tissue factor, tPA (tissue plasminogen activator), thrombomodulin, E-selectin, ICAM-1, VCAM-1 and PECAM-1 but no investigation was found to be reliable to differentiate healthy volunteers from TA patients.[29]

Table 3: Angiographic classification of Takayasu arteritis.[22]

Type	Feature
5 types-based classification of TA (2010)	
Type I	Arch of aorta, subclavian and carotid arteries, and brachiocephalic trunk.
Type II	Descending thoracic and abdominal aorta.
Type III	Arch of the aorta and abdominal aorta.
Type IV	Pulmonary artery and any of the above types.
Type V	Coronary arteries involvement and any of the above types.
6 types-based classification (1994)[27]	
Type I	Arch of aorta, carotid and subclavian arteries, and brachiocephalic trunk.
Type II-a	Ascending aorta, arch of the aorta and its branches.
Type II b	Ascending aorta, arch of the aorta and its branches, thoracic descending aorta.
Type III	Thoracic descending aorta, abdominal aorta, and/or renal arteries.
Type IV	Abdominal aorta and/or renal arteries.
Type V	Combination of type II b and type IV.

Serum autoantibodies i.e. anti-endothelial antibodies: Serum biomarkers like IL-6, 8 and 18, and matrix metalloproteinase-9 are suggested biomarkers. Currently pentraxin 3 (PTX3) is advised as a biomarker for disease activity in TA patients, which is released by the vascular and immune cells in response to pro-inflammatory signals. [21]

Radiological findings

American College of Rheumatology has added arteriogram abnormalities as diagnostic criteria to diagnose the disease. The gold standard criterion is Angiography for assessment of vascular injuries and involvement; specifically, pan-angiography permits a correct evaluation of the expansion of the disease, which relates to its severity. A few researchers focus on the high rate (15%) of coronary inclusion in TA and suggest performing a coronary graphic exam.[22]

MANAGEMENT

Medication treatment helps to control the inflammation and avoid damage to blood vessels. Sometimes treatment of Takayasu's arteritis is troublesome because the disease may remain active even after symptoms subside. [7]

The basis of medical management is its disease activity and developing complications. Some patients may have an only mild form of TA, whereas others may worsen significantly. Control on the inflammatory process and hypertension are two imperative angles to treat the disease.[17]

Corticosteroids

Corticosteroids are used to control the inflammation. In the active phase of the disease, initially, a high dose (1-2 mg/kg/day) of prednisolone or its equivalents is given to mostly TA cases as standard treatment. Usually, two-thirds of the total dose may be given early in the morning and remaining in the evening after dinner.[30] Systemic symptoms usually suppressed by mostly steroids and these also hamper the disease progress. Doses of steroids should be tapered when symptoms get reduced. Prednisone dosage tapered by 5mg/week until it reaches to a dose of 20 mg/day. Steroids can be stopped in remissions or can be increased in the exacerbations of the disease.[2] If the patient has resistance to glucocorticoids, then combination therapy with cyclophosphamide or methotrexate (MTX) is usually used. European League Against Rheumatism (EULAR) recommends that a starting dose of glucocorticoids should be 1mg/kg bodyweight for 4 weeks for the disease remission and as an adjunctive therapy it favours immunosuppressive agent. As symptoms subside taper off the dose.[22]

Immunosuppressive agents

Immunosuppressive (IS) agents are also used widely for the treatment of TA. The first choice in addition to corticosteroids is methotrexate (MTX). It has been found effective in various 3016 patients with TA. Out of sixteen, thirteen patients went into remission and eight patients remain in remission for eighteen months.[31] Another commonly given immunosuppressive agent is AZA (Azathioprine). It can be given with corticosteroids to the patients who previously had not taken any IS agents. The AZA can be initially started with a dose of 2 mg/kg/day with corticosteroid treatment. An Indian study conducted by Valsakumar et al reported that acute phase symptoms were considerably reduced, no progression is shown by control angiography and no adverse events occurred.[32]

IL-6 receptor inhibitor

Interleukin-6 (IL-6) plays a major role in the inflammatory process of large vessel vasculitis. Various studies showed that the humanized monoclonal antibody tocilizumab, which acts by blocking the soluble IL-6 receptor, can produce good clinical improvement. In patients with refractory TA, it has a steroid-sparing effect.[21]

Supportive measures

Calcium, vitamin D, regular exercises, and low salt diet are essential components to reduce the side effects related to corticosteroids agents. Blood pressure measurement should be done on unaffected extremities because of the absence of pulses on affected extremities. Atherosclerosis is also considered as the risk for increased TA so preventive measures should be considered.[30]

Surgical management

There is a need for surgical procedures to bypass or open stenosed or occluded arteries to restore the uninterrupted blood supply. In the active inflammatory phase, it is better to avoid endovascular procedures and surgeries.[22] After interventions, immunosuppressive agents are also recommended.[30]

The principle of surgical management is revascularization of blood vessels either by endovascular procedures like balloon angioplasty, stent, and graft replacement or by surgeries.[33]

The prognosis and success rate of endovascular interventions depend on the stage, site and length of the arterial stenosis.[30] PCTA (Percutaneous transluminal angioplasty) is the most commonly used palliative method with an 80% success rate.[34] PCTA is not able to manage all the lesions. When stenosis is more than 70%, surgical bypass becomes essential. Irrespective of the surgical interventions, the outcome becomes favourable if the disease is dormant.[33] Antiplatelet treatment should be used to decrease the chances of reoccurrence of stenosis before and after endovascular interventions in TA. Usually loading doses of 300 mg of aspirin and clopidogrel given 12 hours before the interventions and then continue with clopidogrel 75 mg/day for four weeks and aspirin 100 mg/day indefinitely post-intervention.[30]

Weaver et al reported renal revascularization procedure in TA associated with stenosis of the renal artery. Thirty-two aortorenal bypass procedures were performed. All the patients were hypertensive before surgery with a mean BP of 167±6/99±5 mm Hg, three patients were dependent on dialysis and two had refractory congestive heart failure. Autologous bypass grafts were used in 20 patients and prosthetic materials in 12 patients. No postoperative death was reported. Three graft stenosis (9%) and three graft occlusions (9%) were documented. Mean blood pressure measurement decreased to (mean 132±4/79±2 mm Hg), no patient longer required dialysis, and congestive heart failure resolved in both patients who had this condition before surgery.[35]

Miyata et al published a review of 106 patients who underwent surgical treatment. There were 12 early hospital deaths. Thirty-one of the remaining 94 patients died after a mean follow-up period of 19.8 years. Congestive heart failure was the major cause of death, accounting for 45% of events. The cumulative survival rate at 20 years was 73.5%.[36]

Jin-Hyun Joh et al reported that Ascendo-bicarotid bypass and Ascendo-right ICA bypass had been performed for patients who had nearly complete occlusion of bilateral common carotid arteries. After the ascendo-carotid bypass surgery, the symptoms of visual disturbances and cerebral ischemia disappeared, and migraine was reported in one patient for three days which was relieved by no specific

medicine. The thoraco-left iliac bypass was done in patients who had nearly complete occlusion of the mid-abdominal aorta. After this surgery, the symptoms of cardiac failure disappeared. No other postoperative complication has been reported so far.[37]

DISCUSSION

Takayasu arteritis is an inflammatory disease of the large arteries which includes the aorta, great vessels, renal and pulmonary arteries. Arterial lesion can lead to secondary hypertension, retinopathy, cardiovascular involvement, and premature death. It has worldwide distribution, but most observed in the Asian population than in North America. Women have affected more than men. Etiopathogenesis remains unknown for TA but active tuberculosis and HLA-B*52 were found associated with non-specific aortitis. About 10% of patients with TA are asymptomatic where constitutional symptoms occur in the early phase. Hypertension occurs in more than half of the patients due to the narrowing of one and both renal arteries. Angiography is considered as a gold standard for evaluation in vascular lesions. The treatment of TA is to control the inflammatory process using medication to prevent further damage to blood vessels. Whereas sometimes it is difficult to treat TA because symptoms get improve but disease might still be active. If it is not cured with medical management then surgical revascularization is needed like balloon angioplasty, stenting, and graft replacement.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Vitthala S, Misra P. Takayasu' s Arteritis and Pregnancy : A Review. Internet J Gynecol Obstet. 2007;9(2):1-5.
2. Johnston SL, Lock RJ, Gompels MM. Takayasu arteritis: a review. J Clin Pathol [Internet]. 2002;55(7):481–6.
3. Singh N, Tyagi S, Tripathi R, Mala YM. Maternal and fetal outcomes in pregnant women with Takayasu aortoarteritis: Does optimally timed intervention in women with renal artery involvement improve pregnancy outcomes? Taiwan J Obstet Gynecol . 2015;54(5):597-602.
4. Alibaz-Oner F, Aydin SZ, Direskeneli H. Recent advances in Takayasu's arteritis. Eur J Rheumatol. 2015;2(1):24-30.
5. Numano F, Okawara M, Inomata H, Kobayashi Y. Takayasu's arteritis. Lancet. 2000;356(9234):1023-5.
6. Chugh KS, Jain S, Sakhuja V, Malik N, Gupta A, Gupta A, et al. Renovascular hypertension due to Takayasu's arteritis among Indian patients. Q J Med. 2019;85(307,308):833-43.
7. Onen F, Akkoc N. Epidemiology of Takayasu arteritis. Presse Med. 2017;46(7–8):e197-203.
8. Jain S, Pondaiah SK. Takayasu's arteritis: Review of epidemiology and etiopathogenesis. Indian J Rheumatol, 2015;10:S22-9.
9. Parakh R, Yadav A. Takayasu's Arteritis: An Indian Perspective. Eur J Vasc Endovasc Surg. 2007;33(5):578-82.
10. Seyahi E. Takayasu arteritis: An update. Curr Opin Rheumatol. 2017;29(1):51-6.
11. Subramanyan R, Joy J, Balakrishnan KG. Natural history of aortoarteritis (Takayasu's disease). Circulation. 1989;80(3):429-37.
12. Jain S, Kumari S, Ganguly NK, Sharma BK. Current status of Takayasu arteritis in India. Int J Cardiol. 1996;54:S111-6.
13. Espinoza J, Ai S, Matsumura I. New Insights on the Pathogenesis of Takayasu Arteritis: Revisiting the Microbial Theory. Pathogens. 2018;7(3):73.
14. Serra R, Butrico L, Fugetto F, Chibireva MD, Malva A, De Caridi G, et al. Updates in Pathophysiology, Diagnosis and Management of Takayasu Arteritis. Ann Vasc Surg. 2016;35:210-25.
15. Arnaud L, Haroche J, Mathian A, Gorochov G, Amoura Z. Pathogenesis of Takayasu's arteritis: A 2011 update. Autoimmun Rev.2011;11(1):61-7.
16. Sagar S, Ganguly NK, Koicha M, Sharma BK. Immunopathogenesis of Takayasu arteritis. Heart Vessels. 1992 Mar;7(S1):85-90.
17. Markel, Peter A; Matteson, Eric L; Curtis MR. Clinical features and diagnosis of Takayasu arteritis - UpToDate.2019
18. Maffei S, Di Renzo M, Bova G, Auteri A, Pasqui AL. Takayasu's arteritis: a review of the literature. Intern Emerg Med. 2006;1(2):105-12.
19. Sadurska E, Jawniak R, Majewski M, Czekajska-Chehab E. Takayasu arteritis as a cause of arterial hypertension. Case report and literature review. Eur J Pediatr. 2012;171(5):863-9.
20. Li-xin Z, Jun N, Shan G, Bin P, Li-ying C. Neurological manifestations of Takayasu arteritis. Chinese Med Sci J = Chung-kuo i hsueh k'o hsueh tsa chih. 2011;26(4):227-30.
21. Kim HJ, Suh DC, Kim JK, Kim SJ, Lee JH, Choi CG, et al. Correlation of neurological manifestations of Takayasu's arteritis with cerebral angiographic findings. Clin Imaging. 2005;29(2):79-85.
22. Setty NSN, Vijaykumar HS, Nagesh JR, Patil CM, Jadav SS, Raghu SR, et al. Takayasu's arteritis-a comprehensive review. J Rare Dis Res Treat. 2017.2
23. Peter J, David S, Danda D, Peter JV, Horo S, Joseph G. Ocular manifestations of takayasu arteritis: A cross-sectional study. Retina. 2011;31(6):1170-8.
24. Chauhan A. Takayasu Arteritis-Ocular Perspectives. EC Ophthalmol. 2016;4(5):592–3.
25. Takayasu's Arteritis : Johns Hopkins Vasculitis Center. https://www.hopkinsvasculitis.org/types-vasculitis/takayasus-arteritis/. Accessed on 10 June 2020.
26. Takayasu's Arteritis - Vasculitis Foundation. https://www.vasculitisfoundation.org/education/forms/takayasus-arteritis/. Accessed on 10 June 2020.

27. Agarwal R, Sinha RK, Goel A, Lakhani KK, Aggarwal A. Takayasu's arteritis. Journal, Indian Acad Clin Med. 2006;7(3):248-51.
28. Koga T, Nishino Y, Makiyama J, Hayashida T, Miyashita T, Izumi Y, et al. Serum amyloid A is a useful marker to evaluate the disease activity of Takayasu's arteritis. Rheumatol Int. 2010;30(4):561-3.
29. Hoffman GS, Ahmed AE. Surrogate markers of disease activity in patients with Takayasu arteritis: A preliminary report from The International Network for the Study of the Systemic Vasculitides (INSSYS). Int J Cardiol. 1998;66:191-4.
30. Keser G, Aksu K, Direskeneli H. Takayasu arteritis: An update. Turkish J Med Sci. 2018;48(4):681-97.
31. Hoffman GS, Leavitt RY, Kerr GS, Rottem M, Sneller MC, Fauci AS. Treatment of glucocorticoid-resistant or relapsing takayasu arteritis with methotrexate. Arthritis Rheum. 1994;37(4):578-82.
32. Valsakumar AK, Valappil UC, Jorapur V, Garg N, Nityanand S, Sinha N. Role of immunosuppressive therapy on clinical, immunological, and angiographic outcome in active Takayasu's arteritis. J Rheumatol. 2003;30(8):1793-8.
33. Agueda AF, Monti S, Luqmani RA, Buttgereit F, Cid M, Dasgupta B, et al. Management of Takayasu arteritis: A systematic literature review informing the 2018 update of the EULAR recommendation for the management of large vessel vasculitis. RMD Open. 2019;5(2):1-13.
34. Bali HK, Jain AK. Takayasu's arteritis: Current status of angioplasty and stenting. Asian Cardiovasc Thorac Ann. 1999;7(4):339-44.
35. Weaver FA, Kumar SR, Yellin AE, Anderson S, Hood DB, Rowe VL, et al. Renal revascularization in Takayasu arteritis–induced renal artery stenosis. J Vasc Surg. 2004;39(4):749-57.
36. Miyata T, Sato O, Koyama H, Shigematsu H, Tada Y. Long-term survival after surgical treatment of patients with Takayasu's arteritis. Circulation. 2003;108(12):1474-80.
37. Joh JH, Kim DK, Park KH, Kim DI. Surgical Management of Takayasu's Arteritis. J Korean Med Sci. 2006;21(1):20-4.

Cite this article as: Bishnoi RK, Devi S, Rai S, Gunjot. Takayasu Arteritis: A Comprehensive Review of Literature**.** Int J Res Med Sci 2020;8:3418-25.