INTERNATIONAL
AYURVEDIC MEDICAL
JOURNAL

IAMJ

International Ayurvedic Medical Journal, (*ISSN: 2320 5091)* (April, 2017) 5 (4)

A CASE REPORT ON *PAKSHAGHATA* WITH REFERENCE TO *TAKAYASU'S* ARTERITIS

Nithin Krishnan R[1], Advait Vaze[2], Ajay GS[3], Totad Muttappa[4]

[1]Department of Roganidana, [2,3,4] Department of Kayachikitsa
SDM College of Ayurveda, Hassan, Karnataka, India

Email: nithinkris1989@gmail.com

ABSTRACT

Takayasu's arteritis is a rare, chronic, inflammatory, granulomatous large vessel vasculitis predominantly affecting the aorta, its major branches and the pulmonary arteries due to which it presents as pulseless upper extremities. The neurological symptoms vary depending on the degree and nature of blood vessel obstruction. A 28 year old female arrived with a short history of fever and chills followed by partial weakness of left upper and lower limb (hemiparesis) since three days. History of the patient was recorded, examinations were performed which yielded knowledge on absence of radial pulsation on right upper limb and feeble to absent radial pulsation on left upper limb. The condition can be diagnosed as *margaavaranajanyapakshagata*. This article highlights the ayurvedic understanding of pathogenesis of *Takayasu*'s arteritis leading to stroke as a complication. The critical understanding of pathogenesis of the disease has been described on the basis of the concept of *Sahajasatmya* and its de-stability triggering autoimmune reaction. The sequence of autoimmune reaction culminates in granulomatous changes. This process of formation of granulomatous change is the vitiation of *raktha* by *pitta dosha*. Ultimately it leads to *rakthavrittavata* ending in *margaavaranajanyapakshagata* (hemiparesis). The diagnosis is made on the demonstration of vascular lesions in large and middle sized vessels on angiography, CT, magnetic resonance angiography; contrast angiography.The earliest detectable lesion is a local narrowing or irregularity of the lumen, which may develop into stenosis or aneurysms and occlusion.

Keywords: *Takayasu*'s arteritis, *margaavaranajanyapakshagata, Sahajasatmya, raktha*

INTRODUCTION

Takayasu disease or *Takayasu*'s arteritis is a rare[1] chronic progressive idiopathic inflammatory disease characterised as chronic granulomatous vasculitis resulting in occlusive or ectatic changes[2] mainly in the aorta and its immediate branches as well as the pulmonary arteries and its branches[3,4].The classic presentation is characterised by a biphasic pattern of manifestation involving an initial systemic

phase – with a beginning non inflammatory vascular stage followed by an inflammatory vascular stage presenting with nonspecific symptoms such as fever, fatigue, etc. A second occlusive phase is later manifested, depending on the site of occlusion, various areas of the body or various systems are affected exhibiting their respective features. We report a case, which presented straight away with a major catastrophic complication of the disease and was marked by atypical angiographic findings.

Case report:

Presenting concerns:

A 28 year old female, a plantation worker, presented to us with a short history of fever and chills followed by partial weakness of left upper and lower limb since three days.

Historical data:

There was no past history of arthralgia, myalgia, night sweating, anorexia, claudication or other systemic manifestations.

Clinical findings:

On examination patient was moderately built and nourished.

The arterial pulsation in both the upper limbs were not palpable (on left upper limb radial and brachial pulses were feeble to absent, whereas on right upper limb pulses was absent). In lower limb the arterial pulsations were palpable. Carotid pulsations were palpable on both sides. Blood pressure was not able to be recorded in upper limb. Gait was hemiplegic. Cardiovascular examination revealed an apex beat in the fifth intercostal space in the mid clavicularline and a systolic murmur of grade 1 over the mitral area and radiating to the left infraclavicular area. There were no renal vascular bruits. Other symptoms revealed no significant abnormality. The fundoscopic examination was normal.

Higher mental status examination of patient was intact. All twelve pairs of cranial nerves were functioning within normal limits. Motor system assessment revealed hypotonic muscles with a reduced muscle bulk for the left upper and lower limb. Muscle strength was assessed as 3/5 for left upper and lower limb. Sensory system and coordination of the patient was intact. Deep tendon reflexes were brisk on the left side.

Diagnostic focus and Assessment:

Diagnostic results:

The complete blood investigation revealed a raised erythrocyte sedimentation rate, with a borderline reduction of haemoglobin percentage and serum potassium level. Urine analysis showed no cast or other abnormalities. Chest X – Ray showed no abnormalities. The echocardiogram revealed trivial mitral regurgitation with mitral valve prolapse.

Magnetic resonance angiogram was done, indicating major cervical vessel occlusion, except right vertebral artery. Gross wall thickening of left subclavian artery was observed and both common carotid, where right common carotid occlude more than the left common carotid. Magnetic resonance imaging of brain revealed acute infarct on right M1lenticular striate territory.

Diagnostic reasoning

A diagnosis of Takayasu disease was done, as the patient satisfied four of six criteria mentioned according to the diagnostic criteria framed by the American

College of Rheumatology **[Table 1]**. An initial phase treatment was given from an allopathic institute – antipyretics. But, unfortunately after two days, patient succumbed with stroke due to an acute infarct. Hence, second phase of treatment was given with anti-inflammatory drugs, antilipidemic and blood thinners.

Therapeutic focus

Rehabilitation from strokeresulted as a complication from Takayasu disease.

DISCUSSION

Takayasu disease, also known as pulse less disease, Martorell's syndrome, Young female arteritis, occlusive thromboaortopathy or Ruder – Harbitz syndrome[5]is a chronic inflammatory arteritis affecting large and small vessels predominantly the aorta and its main branches.

Gian Bathista in 1830 published the description of arteritis[6]. Morgagni in 1761, described the entity[7]. The disease is named after Dr. Mikito *Takayasu*, who in 1908 described it as a wreath like appearance of retinal vessels with absence of radial pulse, supported by Dr. Onishi and Dr. Kagoshima[8].

Table 1: American College of Rheumatology criteria for classification of Takayasu disease[9]

Sl. No	Diagnostic criteria
1.	Age of disease onset less than or equal to 40 years of age
2.	Claudication of extremities
3.	Decreased brachial artery pulse
4.	Blood pressure difference of greater than 10mmHg
5.	Bruits on subclavian arteries or aorta
6.	Arteriogram abnormality

Takayasu disease has worldwide distribution but prevalent in Asian population especially in Japan and India[10].Takayasu disease is the commonest cause of reno vascular hypertension in India[11].

Etiology of *Takayasu* disease is still unknown. *Takayasu* continues to be an enigma even a century after its discovery. The exact cascade of pathogenic process is yet to be established. Till now the aetiologies have been postulated based on a post infection (especially tubercular infection) based on studies conducted in India[12] and Japan[13]. Genetic link to the predominance of disease in Asian population is due to HLA B52 haplotype[14]. HLA B52 link possess more with aortic regurgitation, ischemic heart disease and pulmonary infarction are much more common[15]. Renal artery stenosis occurs more frequently in HLA B39 positive patients[16,17,18]. Also, involvement of microbes and other abnormalities in cellular mediated immunity can trigger the pathogenesis. But, they have not been absolutely proved.

Symptoms are initially often non – specific and may include the different stages of the diseases. Initial stage symptoms include fatigue, rapid, unintended weight loss, muscle or joint pain and low – grade fever. It is possible that swelling could damage arteries for months or even years before the next phase symptoms occurs[19].

In the second stage, inflammation has caused arteries to narrow enough to reduce the amount of blood, oxygen and nutrients reaching to certain tissues or organs. The second phase symptoms include arm or leg weakness or pain with use, light headedness, dizziness, fainting, headache, difficulty thinking and remembering, visual disturbances, high blood pressure, difference in blood pressure between both arms, diminished or absent pulse in the wrists and sometimes ankles, anaemia, which may make you feel tired or weak, chest pain, shortness of breath and fatigue[20].

Repeated inflammation (swelling and healing) of the arteries can lead to the following complications like – hardening and narrowing of blood vessels, inflammation of the heart – either of the heart muscle itself (myocarditis) or sack surrounding the heart (pericarditis)on the heart valves (valvulitis), heart failure, aneurysm in the aorta, higher blood pressure, ischemic stroke or transient ischemic attack (a temporary stroke that has all the symptoms of an ischemic stroke without causing damage), heart attack and pulmonary artery problems[21].

Table 2: Angiographic classification of Takayasu'sarteritis (Takayasu conference, 1994)[22]

Type	Vessel involvement
Type I	Branches from aortic arch
Type IIa	Ascending aorta, aortic arch, and its branches
Type IIb	Ascending aorta, aortic arch 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 5	Combined features of Type IIb and IV.

Ayurvedic Perspective on *Takayasu* Disease leading to Stroke:

Aetiology:

Daiva as*hetu* (unknown factors or due to past deeds) or *svabhavikahetu* (genetic or natural causes or acquired from present life deeds)[23]. In this case the initial development of patient was with Takayasu's Arteritis. The proposed etiological factors or predisposing factors derived from studies conducted throughout the world specifically in Indian sub-continent and Asian countries include –Genetic involvement (HLA B52 haplotype) in the individuals suffering from Takayasu disease, occurrence of *Takayasu* disease in patients with post infection especially tuberculosis and other abnormal cell mediated immune responses to vessels.

Still even after a century of discovering the syndrome the precise etiological factors are unclear. But, the condition is understood as an autoimmune disorder causing granulomatous intimal fibrosis.

Premonitory symptoms:

Headache (in headache*vata* dominated *dosha* accumulate in head, later vitiate *raktha* – blood and *snayu*– nerves present in head to produce headache)[24].

Clinical symptoms:

Fever *(Rasa pradoshalakshana)*, giddiness (produced due to *raja – manodosha*, *pitta* and *vatadosha*), tiredness (*margavaranajanya*), pain (describes as a symptom of hemiplegia – pricking type of pain in affected limb)[25], spasticity (seen in patient

on the final phase of elbow extension of left upper limb due to *snayupraptavata*, where disordered *snayu* (tendons) produces stiffness and contraction[26].

Pathogenesis:

In normal state, in a healthy human body – *vata*, *pitta* and *kaphadosha* irrespective of opposite qualities co-exist each other rather than contradicting each other. This is termed as *sahaja satmya*[27], which the conventional system coined as the so called immune tolerance. Hence, this is the reason why the body's own immune cells are not attacking self from Ayurvedic point of view. But, due to the influence of *daiva* (past deeds or unknown factors) or due to *svabhavika karanas*[28] (in which we can include all the probable theories or mechanism of initiating autoimmune reaction such as microbial, genetic, immunological) potentially paving way to imbalance or disequilibrium or destability to the *sahajasatmya*, there by the immune cells (leucocytes) lose the ability to identify self from non – self or foreign agents.

To be more specific, on looking into the features told in *vataprakruti* individuals, depicting the features of *vata* itself, it includes certain *gunas* like the *sheegra guna*[29], by virtue there are other two *gunas* mentioned in the literature as *alpasmruti* (lesser remembrance – abnormality to WBCs) and *sheegragrahita*[30] (early identification of tissue). The above mentioned features occur at the level of *raktha* i.e. to be precise at the level of WBCs in the recognition of body tissues. This mechanism leads to the mistaken judgement of the body tissues as an external antigen whereas, while describing the *pitta pradhanalakshana* by Charaka, it has been mentioned the feature of *pitta* as *teekshna guna*[31] of *pitta* by virtue of its leading to *teekshnaagni* and *teekshnaparakrama*[32]. This causes the attack of immune system, here to the body's own tissues[33]. This cascade of abnormal and noxious reaction drives an autoimmune disorder sequence. This is nothing but the impaired functioning of *dosha* (*ojovisramsa* – as it is the malfunctioning and displacement of *doshas*[34]). Then due to the repeated action of *pitta* i.e. repeated inflammation. There is vitiation of blood induced by *pitta dosha* – ultimately leading to *sthyayathi* or *ghanambhavathi* (granulomatous formation)[35] as mentioned in the context of describing the features of blood vitiated by *pitta*. This is nothing but repeated inflammation and its failure leading to formation of organized mass of macrophages, where organized mass refers to a tight ball of mass and is termed as Granuloma.

This mode of pathology in Ayurveda is nothing but "*Raktavrittavata*" limiting its manifestation to certain regions of the body (*mandalani cha*)[36]. This pathogenesis resembles the pathogenesis of *vatarakta*, where the complications such as *moorcha*, *mada*, *bhrama* and *vedana* resemble to an extent[37]. So, in this case the site of *Raktavrittavata* is on to different *dhamani* (artery – based on the definition given in classics). And finally the occlusion in these arteries cause aggravation of *vata* in turn leading to the manifestation of *Pakshagata* (hemiplegia). According to Acharya Susrutha, he clearly mentions about the manifestation of contralateral hemiplegia due to aggravated *vata* in *urdhwa* (superior), *adhoga* (inferior)and

tiryaggata (oblique) *dhamani* (vessels)[38], to an extend we can understand it as circle of villus.

Diagnostic focus and assessment:

The etiological diagnosis is *shonithadushti* – vitiation of blood (post infection or cell mediated immunity abnormality), anatomical diagnosisis impact on *urdhwagadhamani – superior branch of vessels* (M1 Segment of MCA), pathological diagnosisis *margavarana* – occlusion (*raktavrittavata* – obstruction of movement of *vatadosha* by *raktha*) i.e. granuloma formation causing stenosis or even obstruction and clinical diagnosisis *pakshagata* (hemiparesis on left side) i.e. left sided contralateral hemiparesis).Treatment for*avarana* should be followed by intervention of hemiparesis. Here, the case is hemiparesis with the predominance of *vatadosha* as patient is devoid of any symptoms like heaviness or burning sensation.

CONCLUSION

The case is relatively common when overlooking the gender (female) of the patient and the age of incidence (28 years). But, the presence of short duration of non – specific symptoms in patients such as the fever and chills, occlusion of all the major cervical vessels except the right vertebral artery irrespective of absence of any major symptoms elicited in the history and the complication of *Takayasu* disease ending with stroke made the case relatively rare. Ayurvedic understanding of pathology in *Takayasu* disease ending in stroke has not been discussed in any of the scientific articles, but through this case the pathogenesis has been plotted with authentic references. *Takayasu*'s arteritis often called as Aortic Arch Syndrome is a group of features arising from the granulomatous intimal fibrosis. The condition is difficult to be diagnosed though mere clinical features and examinations. Radiological evidence of wreath like appearance in the wall of arteries is confirmatory for this particular condition.In Ayurveda, this syndrome is nothing but a pathological state of *raktavrittavata* – blood causing obstruction in the path of *vatadosha* and not a proper disease diagnosis. This particular pathology has the potential to end in stroke or *pakshagata*; which the modern science acknowledges as a rare complication to Takayasu Disease.

REFERENCES

1. Johnston SL, Lock RJ, Gompels MM. Takayasu's arteritis: A review journal of clinical pathology1002;55:481 – 6
2. VK Bahl, Sandeep Seth. Takayasu's arteritis revisited: Indian heart journal 2002; 54:147 – 151
3. Ishikawa K. Diagnostic approach and proposed criteria for the clinical diagnosis of Takayasu'sarteriopathy. JAM Collcardiol 1988; 12:964 – 72
4. Johnston SL, Lock RJ, Gompels MM. Takayasu's arteritis: A review journal of clinical pathology1002;55:481-6
5. Patient. info/ doctor/ Takayasu arteritis. 1 – 5. (Cited 20 June, 2016).Available on patient.info/in/doctor/takayasus – arteritis
6. Numanof, Okawara M, Inomata H, et al. Takayasu's arteritis. Lancet 2000; 356:1023 – 5
7. Massimo Vanoli, Ginlia Bacehiani, Laura Orgi, Raffaella Scorza. Takayasu's arteritis: A changing disease. Journal Neprol 2001; 14:427 – 505

8. MikitoTakayasu. A case with peculiar changes of the central retinal vessels. ActaSocietatisophthalmologicaeJaponicae, Tokyo 1908, 12: 554
9. Krishna MV, Rudresh, Namratha S. Takaysu's arteritis – stroke as an initial presentation. JIACM 2004; 5(3): 274 – 6
10. VK Bahl, Sandeep Seth. Takayasu's arteritis revisited: Indian heart journal 2002; 54:147 – 151
11. Chug KS, Jain S, Sakhuja V, Malik N, Gupta A, Sehgals, Jha V, Gupta KL. Renovascular hypertension due to Takayasu's arteritis among Indian patients. Q. J. Med, 1992 Nov – Dec; 85 (307 – 308): 833 – 43
12. San PK, Kinare SG, Kelkar MD, Nanivadekar SA.Non-specificstenosing arteritis of the aorta and its branches: a study of possible etiology. Mt Sinai J Med 1972; 39: 221 – 242
13. Lupi – Herrera E, Sanchez – Torres G, Marcushamer J, Mispireta J, Horwtiz S, Vela JE. Takayasu's arteritis. Clinical study of 107 cases. Am Heart J 1977; 93: 94 – 103
14. VK Bahl, Sandeep Seth. Takayasu's arteritis revisited: Indian heart journal 2002; 54:147 – 151
15. Ibid
16. Dong RP, Kimura A, Numano F, Nishimura Y, Sasazuki T. HLA – linked susceptibility and resistance to Takayasu's arteritis. Heart vessels 1992; 7 (suppl):S73 – S80
17. Kimura A, Kobayashi Y, Takahashi M, Ohbuchi N, Kitamura H, Nakamura T, et al. MICA gene polymorphism in Takayasu's arteritis and Buerger's disease INt. J. Cardiol 1998; 66 (suppl): S107 – S113, discussion S115
18. Kitamura H, Kobayashi Y, Kimura A, Numano F. Association of clinical manifestations with HLA – B alleles in Takayasu's arteritis. Int. J. Cardiol 1998; 66 (suppl): S121 – S126
19. Vasculitis foundation.org, Takayasu's arteritis. (Cited 29 June, 2016) Available on www.vasculitisfoundation.org
20. Ibid
21. Ibid
22. Moriwaki R, Moda M, Yajima M, et al. Clinical manifestations of Takayasu's arteritis in India and Japan –new classification of angiographic findings. Angiology 1997; 43: 369 – 79.
23. Ayurveda Dipika on Agnivesha, Charaka Samhita with Ayurveda Dipika Commentary, Ed. Acharya Y.T, Chaukhambha Surbharati Prakashan, Varanasi, 2009 (Reprint), Sutra Sthana.17/62: p103
24. Agnivesha, Charaka Samhita with Ayurveda Dipika Commentary, Ed. Acharya Y.T, Chaukhambha Surbharati Prakashan, Varanasi, 2009 (Reprint), Sutra Sthana.17/11 - 18: p99
25. Agnivesha, Charaka Samhita with Ayurveda Dipika Commentary, Ed. AcharyaY.T, Chaukhambha Surbharati Prakashan, Varanasi, 2009 (Reprint), Chikitsa Sthana.28/56: p619
26. Agnivesha, Charaka Samhita with Ayurveda Dipika Commentary, Ed. AcharyaY.T, Chaukhambha Surbharati Prakashan, Varanasi, 2009 (Reprint), Sutra Sthana.28/21 - 18: p179
27. Ayurveda Dipika on Agnivesha, Charaka Samhita with Ayurveda Dipika Commentary, Ed. Acharya Y.T, Chaukhambha Surbharati Prakashan, Varanasi, 2009 (Reprint), Sutra Sthana.17/62: p103

28. Ibid
29. Agnivesha, Charaka Samhita with Ayurveda Dipika Commentary, Ed. Acharya Y.T, Chaukhambha Surbharati Prakashan, Varanasi, 2009 (Reprint), Vimana Sthana.8/98: p277
30. Ibid
31. Agnivesha, Charaka Samhita with Ayurveda Dipika Commentary, Ed. AcharyaY.T, Chaukhambha Surbharati Prakashan, Varanasi, 2009 (Reprint), Vimana Sthana.8/97: p277
32. Ibid
33. Amit Nakanekar, Sunanda Bhople, Harshad Gulhane, Suraj Rathode, Jayanth Gulhane, Pravin Bonde. An ayurvedic in the management of Guillian – Barre syndrome: A case study. AncSciLife. 2015 July – Sep; 35 (1): 52 – 57. doi: 10.4103/0257 – 7941.164540.
34. Byadgi PS, Editor. Concept of immunity in Ayurveda; Journal of Applied Pharmaceutical Sciences, 11 July, 2011
35. Ayurveda Dipika on Agnivesha, Charaka Samhita with Ayurveda Dipika Commentary, Ed. AcharyaY.T, Chaukhambha Surbharati Prakashan, Varanasi, 2009 (Reprint), Sutra Sthana.24/20: p125
36. Agnivesha, Charaka Samhita with Ayurveda Dipika Commentary, Ed. Acharya Y.T, Chaukhambha Surbharati Prakashan, Varanasi, 2009 (Reprint), Chikitsa Sthana.28/63: p619
37. Agnivesha, CharakaS amhita with Ayurveda Dipika Commentary, Ed. Acharya Y.T, Chaukhambha Surbharati Prakashan, Varanasi, 2009 (Reprint), Chikitsa Sthana.29/31: p629
38. Susruta, Susruta Samhita with Nibandha Samgraha and Nyayachandrika commentaries, Ed. Vd. Y.T Acharya, Chaukhambha Orientalia, Varanasi, 2009 (Reprint), Nidana Sthana.1/60: p266

Source of Support: Nil
Conflict Of Interest: None Declared

How to cite this URL: Nithin Krishnan R: A Case Report On Pakshaghata With Reference To Takayasu's Arteritis. International Ayurvedic Medical Journal {online} 2017 {cited April, 2017} Available from: http://www.iamj.in/posts/images/upload/1345_1352.pdf