

Varicose veins and *Siragranthi*: An Ayurvedic Insight into Etiopathogenesis

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Abstract— Varicose veins are dilated, elongated and tortuous superficial veins, most frequently affecting the lower limbs. Their etiopathogenesis is multifactorial, involving genetic predisposition, valvular incompetence and altered hemodynamics. This condition may progress to complications such as oedema, pigmentation, eczema and venous ulceration. In Ayurveda, this condition corresponds to *Siragranthi*, resulting from deranged *vyana vayu*, *swedavaha srotodushti* and *vitiation of ranjakapitta*. This article highlights a striking convergence between Ayurvedic and modern concepts, particularly regarding venous stasis, wall weakness and microcirculatory dysfunction. Understanding varicose veins through an integrative Ayurvedic approach enhances insight into disease progression and supports improved prevention strategies, early diagnosis and targeted therapeutic interventions.

Keywords: Varicose veins, *siragranthi*, etiopathogenesis.

I. INTRODUCTION

Varicose veins are dilated, tortuous and elongated superficial veins that occur due to valve incompetence and venous wall weakness¹. The term “varicose” is derived from the Latin word “varix,” meaning “twisted,” aptly describing the appearance of these veins². These veins are most commonly seen in the lower limbs, particularly involving the long and short saphenous veins and their tributaries, due to the effect of gravity and the upright posture of humans. Although not life-threatening, it can cause significant discomfort, swelling, pain, heaviness and cosmetic concern. It may progress to complications such as pigmentation, eczema and venous ulcers, if left untreated.

Globally, varicose veins affect 10–30% of adults, with a higher incidence among women, particularly due to hormonal and occupational factors³. In *Ayurveda* the signs and symptoms of varicose veins can be correlated with *Siragranthi*. It is a condition characterized by swelling of the *siras*, primarily due to *vata* imbalance. Understanding the etiopathogenesis from an *Ayurvedic* perspective helps in designing comprehensive management strategies involving *samana*, *sodhana* and parasurgical measures such as *siravyadha* and *jalukavacharana*. The present article attempts to critically analyse the pathogenesis of varicose veins through *Ayurvedic* principles.

Modern understanding of etiopathogenesis

Varicose veins are a common chronic venous disorder resulting from structural and functional abnormalities of the superficial veins of lower limbs. Modern literature highlights a multifactorial etiopathogenesis involving genetic, mechanical, lifestyle-related and hormonal influences, all of which converge to impair venous return and weaken venous structure. A

hereditary predisposition also plays a significant role in the development of the condition. Varicose veins are frequently observed among individuals whose professions require prolonged standing - such as teachers, nurses, conductors, surgeons, traffic police, vendors etc. Additional contributing lifestyle factors include smoking, chronic constipation and obesity, all of which increase venous pressure and impair venous return. The pathogenesis begins with persistently elevated venous pressure, which progressively damages the venous walls, leading to stretching, loss of elasticity, hyperlipodermato-sclerosis and ultimately ulcer formation⁴. Varicose veins of the lower limbs occur due to morphological factors, as veins must drain against gravity and superficial veins lose fatty support.

Clinical features⁵

- Visible dilated and tortuous veins.
- Tired, heavy or aching sensation in the affected lower limb.
- Calf cramps, especially after lying down.
- Other associated symptoms include ankle swelling in the evening, itching or pigmentation of the overlying skin, eczema and venous ulceration.

Clinical examination

- Inspection and palpation of the lower limbs in standing and supine positions to assess varicosity distribution, skin changes, oedema and ulcers.
- Functional tests such as the Trendelenburg, Perthes and tourniquet tests help evaluate valve competence, deep vein patency and the contribution of perforator veins⁶.
- Diagnosis is confirmed using duplex ultrasonography, which accurately assesses vein patency, valve function, reflux, obstruction and thrombotic changes⁷.

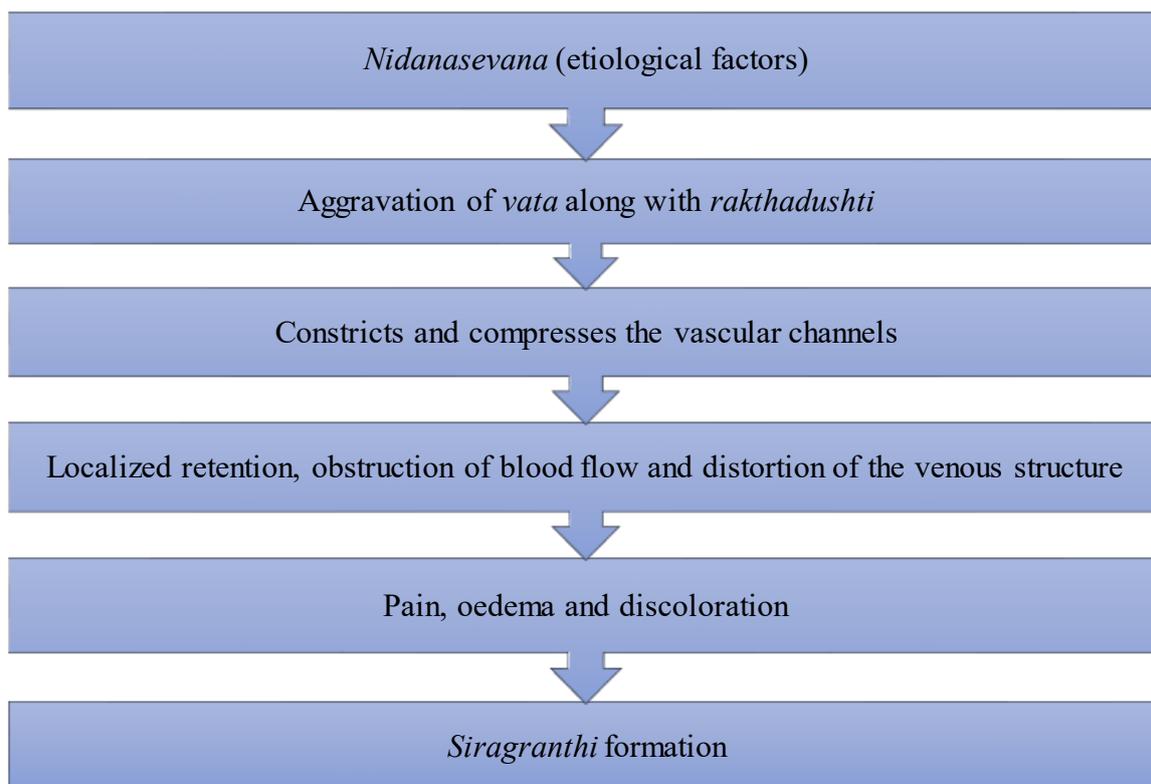
Ayurvedic interpretation of varicose veins

Susruta samhita provides the earliest and most comprehensive description, detailing its causes, pathology, prognosis and clinical features⁸. According to *Ayurveda*, several causative factors contribute to the development of *Siragranthi*.

- *Ativyayama* (excessive exertion)
- *Deerghasthana* (prolonged standing)
- *Adwagamana* (long-distance walking)
- *Seethasamsparsha* (exposure to cold water)
- *Seethoshna akramasevana* (faulty sequence in the use of cold and heat)
- *Bharavahana* (weight lifting)

- Intake of *abhishyandi* foods like curd, lassi, deep fried foods, heavy non-veg foods, junk foods, aerated drinks etc

According to *Acharya Susruta*, *Siragranthi* develops primarily in persons who are weak or those who indulge excessively in physical exertion⁹. These causative factors vitiate *vata*, affecting the *sira prathana* (vein clusters), *sampeedana* (squeezes), *samkochana* (constricts) and *visoshana* (dries up), resulting in *granthi* formation¹⁰. He describes *Siragranthi* as either movable and painful or immovable, enlarged and circular, potentially occurring in *marmasthanas*. When *vata* becomes excessively aggravated, the veins lose their natural elasticity and may fail to pump blood effectively. This impaired circulation predisposes the veins to dilation and tortuosity.



- *Acharya Susruta* describes the painful variety as *krichrasadhya* (difficult to cure) and the painless variety that is bigger in size and presenting at *marmasthanas* as *asadhya* (totally incurable)¹¹.
- *Vagbhatacharya* outlines the treatment options for *Siragranthi* and adds that vitiated *vata* primarily affects *siras* and *raktadhatu*, causing *vakreekarana* (tortuosity) and non-pulsatile, painless swellings¹².
- *Acharya Charaka* notes that vitiated *vata*, *kapha*, *rakta*, and *pitta* can obstruct vessels and cause local oedema.
- *Acharya Bhoja* explains that in weak individuals, *vata* invasion leads to vein proliferation and dehydration, sometimes resulting in painful *sirajala*.

(exposure to excessive heat), *seethoshna akramasevana* (alternating hot and cold exposure without proper sequence), *krodha*, *shoka* and *bhaya*¹³. These factors disturb the normal functioning of the *tiryak dhamanis*, described by *Acharya Susruta* as transversely spreading arterial capillary network in the skin¹⁴. Excessive and vigorous exercise heats the body and stimulates vasodilation of these capillary networks. *Ashtangahridayam* explains that immersing the feet in cold water immediately after excessive exercise, the warm dilated vessels undergo abrupt vasoconstriction¹⁵. This sudden thermal imbalance disturbs the *swedavaha srotas*, leading to *sanga* (obstruction) within the superficial vascular channels. Modern physiology also supports this: heat raises the hypothalamic set point and promotes vasodilation and sweating, whereas sudden cold triggers intense vasoconstriction, disrupting normal microcirculatory flow.

Swedavahasrotas: *Swedavaha srotas* are responsible for thermoregulation and sweat formation, it becomes vitiated due to factors such as *ati-vyayama* (excessive exercise), *atisantapa*

Vyana vayu: Acharya Susruta has described that *vyana vayu* regulates the circulation of *rasa* into the body¹⁶. When *vyana vata* becomes vitiated, excessive dryness and impaired vascular tone lead to hardening and loss of elasticity of the veins and their valves. This dysfunction, combined with increased intraluminal pressure, causes venous dilatation and valvular incompetence, making it difficult for the musculovenous pump to propel blood toward the heart. Consequently, blood pools in the superficial veins of the legs, which have comparatively less muscular support, resulting in the formation of *Siragranthi* beneath the skin.

Ranjaka pitta: *Ranjaka pitta*, located in the liver and spleen, governs *rakta* formation, purification and nutrient distribution. As a secondary mechanism, the stagnated blood becomes susceptible to *ranjaka pitta* vitiation. When disturbed, it leads to blood impurities and association with *ama*, making the blood thick, sluggish and inflammatory. In varicose veins, this impure *rakta* aggravates local tissue damage and contributes to hyperpigmentation and venous ulcer formation. Thus, varicose veins primarily arise from impaired circulation due to *vyana vata dushti*, while their complications - such as inflammation and ulceration are attributed to vitiated *ranjaka pitta*.

II. DISCUSSION

The etiopathogenesis of varicose veins reveals significant convergence between *Ayurvedic* and modern understanding. Both systems recognize that structural weakness of the venous wall and stagnation of blood are central to disease progression. Modern pathology attributes these to prolonged venous hypertension, valvular incompetence and obstruction to venous return. *Ayurveda* interprets it as *vata prakopa*, *raktadushti* and *srotodaurbalya*, which impair the structural integrity and elasticity of venous channels. *Nidanans* such as excessive exertion, prolonged standing, long-distance walking and sudden exposure to cold aggravate *vata*, leading to constriction, obstruction and subsequent dilatation of veins.

Sudden exposure to cold after intense activity, as described by *Ashtangahridayam*, further provokes *vata* and weakens venous walls. *Swedavaha srotodushti*, caused by excessive heat, sweating and abrupt cold exposure, disturbs *tiryak dhamani*-mediated microcirculation and creates congestion and obstruction, paralleling modern microvascular dysfunction. *Vyana vayu* plays the central role by regulating circulation; its vitiation results in loss of venous elasticity, valvular incompetence and venous pooling. Secondary involvement of *ranjaka pitta*, contributing to hyperpigmentation, inflammation and ulceration seen in chronic varicose veins.

Overall, *Ayurvedic* descriptions of *Siragranthi* offer a comprehensive insight into the etiopathogenesis of varicose veins, integrating structural, functional, hemodynamic and microcirculatory factors. The interplay of *vata* vitiation, *srotodushti*, venous wall weakness and secondary involvement of *ranjakapitta* closely parallels modern pathology, reflecting a deep alignment between classical *Ayurvedic* principles and contemporary vascular science. Integrating *Ayurvedic* principles with modern diagnostics enhances diagnostic accuracy and provides a more comprehensive treatment strategy.

III. CONCLUSION

Varicosity is increasingly prevalent due to lifestyle changes and occupational patterns such as prolonged standing, heavy work and lack of physical activity. The disease arises from the cumulative effect of deranged *vyana vayu*, *swedavaha srotodushti* and vitiation of *ranjaka pitta*, all precipitated by *nidana* such as excessive exertion, prolonged standing, cold exposure, etc. The etiopathogenesis described in classical texts closely aligns with modern concepts of venous reflux, valve incompetence and venous hypertension. Integrating modern clinical methods with traditional *Ayurvedic* interventions offers a comprehensive framework for alleviating symptoms, preventing complications and improving quality of life.

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