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STRUCTURAL & FUNCTIONAL ASPECT OF SIRAJ GRANTHI IN RELATION TO VARICOSE VEINS – A COMPREHENSIVE REVIEW

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ABSTRACT

Varicose veins are the veins which are permanently elongated, dilated, & whose path has become tortuous inducing pathological circulation. It is common in lower limb that take many forms, ranging from a non-pathologic condition to an invalidating chronic disorder. In *Ayurveda siraj granthi* & *siragat vata rogas* which are synonymous with modern system disease of varicose veins. Aim of the study is to study the structural & functional changes in *siraj granthi* in relation to varicose veins. Varicose veins are not life threatening but it affects day to day activities & condition worsen with chronicity. Hence it is necessary to do a review study on structural & functional damage of veins in varicose veins.

KEYWORDS: Varicose veins, *siraj granthi*, *siragat vata*, *vata vyadhi.*

INTRODUCTION

Modern lifestyles and work patterns have lead to an increase in various diseases, varicose vein is one of them. It is very common condition in surgical practice, in which saphenous veins and their tributaries become tortuous, elongated and dilated. It affects approximately 25% of women and 15% of men. Varicose veins affects around 3 out of 10 adults.[1] There may be involvement of superficial perforating veins or deep veins. The common site of varicosity are superficial venous system of lower limbs, it affects either the long or short saphenous vein or both. Varicose vein may appear blue or dark purple and are often lumpy, bulging, or twisted. Varicose veins can be primarily caused by incompetence of the veins or weakened walls of the veins that causes venous insufficiency or by inflammation in the veins. It is more common in females than males and can occur in any age group.[2] In *Ayurveda* it

can be very much correlated to *Siraja granthi*. According to *Sushruta Samhita Siraja granthi* is condition in which etiological factors like excessive exertion by weak person (*Abalasya Vyayamjaate*), immersing lower limb in cold water just after walking long distance (*Padatte sahasaambho avgahana*) vitiates Vata dosha. This vitiated *Vata dosha* enters in *sira* and causes *Aakshep, Samkochana* and *Vishoshana* and in turn produces *granthi.*[3] According to *Acharya Vagbhata Vata dosha* plays major role and exerts its influence on *sira* and *Rakta dhatu*. Further creates *Samkochana* (constriction), *Vakreekaran* (Tortuosity), *Vishoshana* in sira and finally causes *Siraja granthi*.[4] In normal condition factors responsible for venous return of lower limb are negative intra thoracic pressure, arterial pulsation pressure on venae comitantes, unidirectional flow of blood due to valve, pressure of muscular contraction on veins.[5] As per modern medicine varicose veins occurs when valve of the veins become incompetent. Due to valve incompetency blood becomes stagnated in the superficial veins and veins becomes tortuous, elongated and dilated.[6]

AIMS AND OBJECTIVES

To study the structural and functional changes in *siraj granthi* in relation to varicose veins.

MATERIALS AND METHOD

Various *ayurvedic* texts, published review articles, research papers, and from the internet have been reviewed for this article.

ANATOMY OF VEINS

Veins are thin walled than arteries and have larger lumen than accompanying arteries. Veins have valves which maintain the unidirectional flow of blood even against gravity. (Venous pressure is low as compare to Arteries so valves have utmost value in the veins). The muscular and elastic tissue content of the venous walls is much less than that of arteries. Large veins have dead space around them for their dilatation. Venae comitantes are a pair of veins on the side of the artery in the forearm or leg region. Pulsation in the arteries are transmitted to veins in the venae comitantes & helps in return of blood towards heart.[7]

PHYSIOLOGY OF VENOUS RETURN

Negative intra thoracic pressure sucks the blood into heart from all over the body. Venous valves prevent any regurgitation of the blood. Arterial pulsation press on the venae comitantes intermittently and helps in draining blood towards the heart. Muscular contractions (Muscle pump) press on the veins and helps for the venous return.[8]

ANATOMY OF VEINS IN THE LOWER LIMB

Lower limb consists of three types of veins that are

- Superficial veins,

-Deep veins and

- Perforator veins.[9]

Superficial veins of the leg

It consists of two main veins the long and short saphenous veins as well as their tributaries. The word Saphena means easily visible. These veins are subcutaneous hence easily accessible and visible.

Long Saphenous vein

The medial dorsal vein and veins from the medial side of the foot combine to form long saphenous vein. This vein is subcutaneous from its beginning point to the posteromedial thigh area, but has to go deep in the anterior thigh to drain into deeper veins. In anterior thigh it pierces cribriform fascia then passes from fossa ovalis (Opening in fascia Lata) and at the end enters in femoral sheath and drains into femoral vein. This saphenofemoral junction is located 2.5 cm below inguinal ligament and 4 cm inferolateral to pubic tubercle. The Great Saphenous vein has about 20 valves to prevent backflow of blood.

Short Saphenous Vein

The short saphenous vein connects the dorsal venous arch at the lateral border of the foot, behind the lateral malleolus. It travels laterally into the leg then travels posteriorly to the calf, draining into the popliteal vein by piercing the roof of the popliteal fossa. It enters the popliteal vein between the heads of the gastrocnemius muscle. There are several connections between the long and short saphenous veins in the knee area. The short saphenous vein has about 13 valves to prevent backflow of blood.

Deep veins of the leg

Anterior tibial vein, Posterior tibial vein, Peroneal vein, Popliteal vein, Femoral vein, Profonde/Deep femoral vein, external iliac vein are the deep veins of the leg**.** The posterior tibial vein, placed posterior to the medial malleolus, is formed when the medial and lateral plantar veins of the plantar venous arch combine. Peroneal vein runs laterally and joins to posterior tibial vein. Anterior tibial vein joins to Posterior tibial, peroneal vein and forms popliteal vein. The popliteal vein connects to the femoral vein in the Adductor canal after

crossing the adductor hiatus. From adductor canal femoral vein connects to the femoral triangle. Here, the profound femoral vein meets the femoral vein which connects to the inguinal ligament and then continues as the external iliac vein. The common iliac vein is formed by joining the external and internal iliac veins. The right and left common iliac veins unite to form the inferior vena cava.

Perforator veins

Perforator veins connect the deep and superficial venous systems, allowing passage of blood in between them. Lower extremity perforators are named depending on their topographical location. Thigh level perforators are named Hunter veins; perforators located just above and below the knee are named Dodd and Boyd veins respectively; and calf level perforators are named Cockett veins.[10,11] Small anatomic series in cadavers have reported an average of 64 perforating veins between the ankle and the groin.[12]

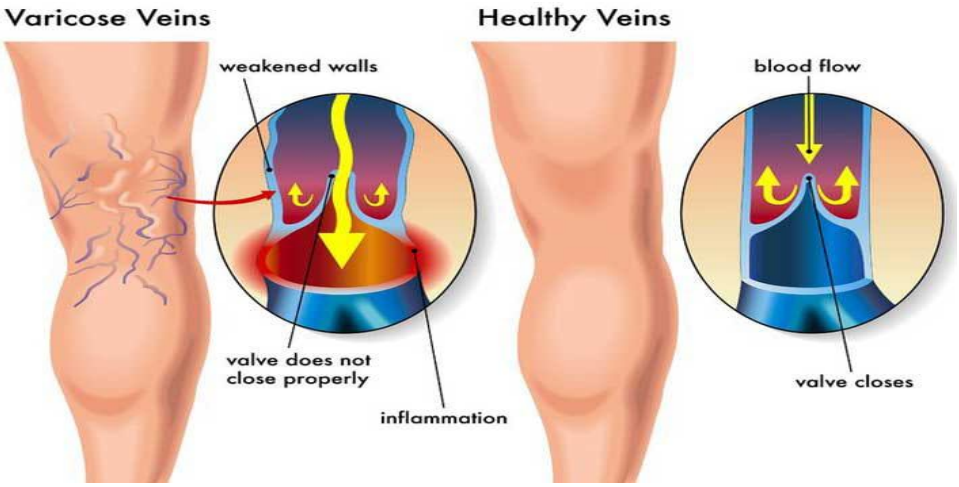
ABNORMAL VENOUS ANATOMY

Varicose veins are usually defined as dilated, palpable tortuous veins greater than 4 mm in diameter that do not discolor the overlying skin.[11] The histologic features associated with varicose veins are diverse and vary in different regions of the vein. However, characteristic changes include irregular thickening of the intima, fibrosis between the intima and adventitia, atrophy and disruption of elastic fibers, thickening of individual collagen fibers, and disorganization of the muscular layers.[13,14,15,16,17,18] These abnormalities are heterogeneously distributed through the great saphenous vein and its tributaries,[15] with some areas appearing hypertrophic while others appear atrophic or normal. The saccular dilations constituting the varices are consistently located just to the distal (upstream) side of valve cusps.[19] Studies of surgical specimens suggest that varices, rather than being initiated at the saphenofemoral junction, can occur anywhere along the course of the great saphenous vein. These observations are supported by ultrasound studies showing primary valvular incompetence to be a multicentric disease that develops simultaneously in discontinuous venous segments.[20] Varicose saphenous veins show an increased collagen and reduced elastin content,[21] and decreased venous elasticity has been demonstrated both in limbs with overt varices and in those without varices but at high risk for their development.[22] These findings suggest that abnormalities in the vein wall architecture precede the development of both overt varicosities and valvular incompetence. Reflux is presumed to occur when the weakened vein wall dilates, causing stretching of the commissure between the valve cusps and separation of the

valve leaflets.[23] Proposed mechanisms about varicose veins have included hypoxia-mediated endothelial changes,[24] cell cycle dysfunction with inhibition of programmed cell death,[18] changes in enzyme activity,[25] and underlying defects in venous tone.[15,26]

Pathophysiology of varicose veins

In healthy veins, venous blood flows from superficial veins to deep veins through perforator veins and then from deep veins blood reaches to the heart. One-way venous valves are found in superficial, deep and perforator veins. Incompetency in any of these series of valves leads to disturbances in unidirectional flow of blood towards the heart. Incompetency in any of these series of valves leads to disturbances in unidirectional flow of blood towards the heart. Incompetency in the superficial venous system alone commonly results from incompetency of valves located at saphenofemoral junction (SFJ) and saphenous popliteal junction (SPJ). The gravitational weight of the blood column throughout the length of the vein creates hydrostatic pressure which is more harmful for the distal aspect of the vein. If valves in the perforator veins fail then, the pressure generated in the deep venous system due to calf muscle pump is transmitted to superficial system through incompetent perforator veins and pool of blood is formed in the superficial venous system leading to venous dilatation which then causes greater valvular insufficiency. Afterwards due to more local dilatation other valve sequentially fails then a series of valves fails finally entire superficial venous system become incompetent. Once superficial venous system become incompetent, subsequently perforator and deep venous valvular dysfunction occurs.[27]



Risk factors for varicose veins: Obesity, Age- above 40, Pregnancy, prolong standing and family history of varicose veins.

Clinical Features: Pain, tenderness, heaviness, inability to walk or stand for long hours, thus hindering work. Skin conditions / dermatitis which could predispose skin loss. Skin ulcers especially near the ankle, usually referred to as venous ulcer.[33]

SIRAJA-GRANTHI

Siraja Granthi can be very well correlated to varicose veins.[28] Due to various *vata* vitiating factors *vata* gets vitiated in the body and this vitiated *vata dosha* acquires *sthansanshraya* (i.e. localization) in different parts of the body. *Acharya Sushruta* described that *vitiated vata dosha* gets accumulated either in one part or one *dhatu* by producing symptoms described above like *Shotha* (inflammation), *shoola* (painful) etc.[29] If the proper management of *vitiated vata* dosha is not done at the same time then this vitiated *vata dosha* spreads to the other body parts and if still neglected produces the various diseases like *siragranthi* (ie.varicose veins).[30] *Vata* vitiates *rakta dhatu* due to this; *Sira* undergoes *sankochan*, *sampidana* and *vishoshana* causing *Siragranthi* (ie. varicose veins). If vitiated at the site of vein leads to signs and symptoms of varicose veins. The change in the shape and size causes veins to become *sankuchit* i.e. tortousity of veins. Also due to *Ruksha guna* (dryness) veins becomes rough and hard (i.e. *vishoshana*) and finally veins looks like a swelling that is painless and non-pulsatile called *siraj granthi.*

Clinical features of *Siragranthi- Sira Sankocha* (tortous veins), *Sira Vakrata* (irregular surface of veins), *Sira Utsedha* (inflammation of veins), *Vishoshana* (roughness and hardness of veins)[31] According to *Acharya Sushruta* two types of *Siragranthi* are present one which is painful and movable and another which is painless, immovable, and greatly enlarged and can occur in *Marmasthan*.[32] In *Siragranthi Lakshanas* like *sampeedya*, *nisphuram*, and *nirujam* can be correlated to dull aching pain in varicose veins. Signs like *sankochya*, *Vrutta*, *Unnatha*, *Shopham* can be correlated to dilated, elongated, and tortuous veins.

CONCLUSION

Siraja granthi occurs when vitiated *vata dosha* enters the *sira*, causing *Aakshep*, *Samkochana*, and *Vishoshana*, which can be very well correlated to varicose veins. Varicose veins are dilated, elongated, and convoluted veins. It is caused by the weakening of veins and the incompetency of the valve due to which the normal anatomy of the vein is disturbed. Varicose veins are commonly caused by occupations that require prolonged standing, pregnancy, obesity and unhealthy lifestyle. This condition commonly affects lower limbs, particularly saphenous veins and their tributaries, and is frequently seen in females than

males. The present lifestyle imposes a significant physical and mental stress on society, leading to an increase in such diseases. Therefore, we must focus on altering our lifestyle through an *Ayurvedic* regimen to live a life free of illness. Early diagnosis and management of the disease will surely improve the patient's quality of life, improve the prognosis for the condition, and prevent further complications.

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