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A review on hetus of varicose veins

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ABSTRACT

The life style changes, obesity, occupational pattern of prolonged standing and pregnancy are considered to be significantly contributing to this situation known as **Varicose veins** wherein veins become enlarged and twisted. Incidence of varicose vein is more in people belonging to the occupation that involved prolonged standing. Varicose vein can be co-related to *Sirajgranthi* as described in Ayurvedic texts. Ayurved formulations are said to be effective in the management of *Sirajgranthi* (Varicose veins)*Sirajgranthi*, treated at an early stage or allows proper maintenance of signs and symptoms reduces the complications and support a better quality of life. In this article we have tried to analyse the anatomical and physiological changes due to long term standing.

Keywords: Hetus, Varicose veins, *Sirajgranthi*

INTRODUCTION

Unless the proper diagnosis is done, it is difficult to provide medicine & cure the disease. To give permanent relief, the root cause has to be removed. The causative factors can be the food, life style or some external factors .In the present era, life

style and the working pattern of the person is showering varieties of diseases. Among which Varicose Veins are troubling the person a lot. Varicose vein is a very common condition in surgical practice. It occurs in people who are habituated for standing for a long time. Abnormal dilated, elongated and tortuous alteration in the saphenous veins and their tributaries is called as varicose veins. Varicose veins are dilated, tortuous, elongated superficial veins that are usually seen in the legs. It can occur in any age group. It is a progressive disease. It is more common in females than males.Varicose vein can be very much correlated to *Siraj granthi*. Being a kind of *Raktadushti Vikara* (Blood involved pathology), Immense attention is to be given in the primary stage itself to prevent further complications. So this article is intended to give a review on *hetus* (causes) of varicose veins for prevention and cure of siraj granthi.

REVIEW OF LITERATURE ON HETUS OF VARICOSE VEINS

Ayurvedic Review

दोषः प्रदुष्टो रुधिरं सिरास्तु सम्पीड्य सङ्कोच्य गत स्त्वपाकम् ॥१५॥

सास्रावमुन्नह्यति मांसपिण्डं मांसाङ्कुरैराचितमाशु वृद्धिम् |

स्रवत्यजस्रं रुधिरं प्रदुष्टमसाध्यमेतद्रुधिरात्मकं स्या त् ॥१६॥ su.ni.11/15-16

According to Acharya Sushruta, the etiological factors play a role in the vitiation of Vata and this vitiated Vata directly affects the Sira Prathana (cluster of veins) by Aakshepa and exposes them to Sampeedana, Samkochana and Vishoshana and produces Granthi which is protruding out. excessive exertion by a person who is weak and emaciated. A weak person who undergoes excessive exercise, the vitiated Vata vitiates the vascular bundles, which in turn compresses (sampeedya), squeezes (samkochya) and dries it up (vishoshya) produces Granthi within less time which is raised, circular Siraja Granthi.

Modern Review

- **Long term standing major cause of varicose veins**

Mostly people working as watchmen, traffic police, beauticians, surgeons, teachers, bus conductors, roadside vendors face this problem.

When standing, gravity pulls the blood downwards to the lower part of the body. Body mechanisms, such as vasoconstriction and valves of the veins, assist in pumping blood upwards. As blood is pumped through the body, the valves within the veins prevent the blood from flowing backwards. After extensive,

prolonged standing, these valves can become weak and eventually fail. When this happens, blood is no longer being prevented from flowing backward. Gravity will pull the blood back into an individual's legs, ankles and feet. This forces the veins to expand or "balloon" to accommodate this extra blood.

The valves of the veins work best in concert with accompanying muscle contractions that force the blood to continue moving up the leg. Standing with some muscles constantly strained weakens these muscles and therefore the strength of the contractions. Varicose veins have also been associated with chronic heart and circulatory disorders and hypertension as well as complications related to pregnancy.

Prolonged standing increases the risk for hospitalization from varicose veins. Among the working age population one out of five hospitalizations from varicose veins are as a result of prolonged standing. Prolonged standing leads to impeded blood flow and stasis in the veins in the lower limbs, which can cause varicose veins.

- **PREGNANCY**

During pregnancy, blood volume increases, while the rate at which blood flows from legs to pelvis decreases. This puts pressure on the veins, which can cause varicose veins. Varicose veins are enlarged veins that commonly occur in the legs, although during pregnancy they can also appear on the buttocks and vaginal area.

- **HORMONAL CHANGES**

Hormonal changes can also lead to varicose veins as increased progestin levels can dilate or open the veins. In addition, during pregnancy the uterus puts pressure on the inferior vena cava (the vein that carries blood from the legs and feet to the heart), further contributing to varicose veins.

- **MENSTRUATION**

About a week or two before menstruation, the hormone progesterone rises within the veins. This progesterone surge loosens the veins walls, allowing the vein to carry more blood. However, valves in the leg veins that transport blood back to the heart may strain from this excess blood flow, causing varicose veins to form or worsen.

- **OBESITY**

Obesity is an important risk factor for all types of lower limb venous disease, and obese patients with lower limb venous disease are more likely to be symptomatic as a result of their lower limb venous disease. Excess weight places every organ

in body under greater stress, including veins. This increased pressure gradually weakens the valves within veins, which soon fail to properly pump blood. The damaged veins are further forced to work even harder to pump blood against the additional weight, creating more opportunities for blood to flow backward and pool in the veins.

- *MUSCLE FATIGUE*

Muscles kept in a constant stress position quickly become exhausted and can result in pain and swelling in the lower back, legs, ankles and feet.

- *VENOUS HYPERTENSION*

Venous hypertension exerted on veins of the lower extremity is considered the principal factor in varicose vein formation. Varicose veins are characterized by symptoms or signs produced by venous hypertension as a result of structural or functional abnormalities of veins. Symptoms may include aching, heaviness, cramps, itching, sensations of burning, swelling, dilatation or prominence of superficial veins, and skin changes. Signs may include telangiectasia, reticular or varicose veins, edema, and skin changes such as pigmentation, lipodermatosclerosis, eczema, and ulceration.

- *DEEP-VEIN THROMBOSIS (DVT)*

Deep-vein thrombosis (DVT) refers to a blood clot that forms in a deep vein, usually in the calf or thigh, but also the pelvis or the arm. Traumatic injury, surgery, and lying in a hospital bed are common triggers. Sitting for long periods, such as during long-distance travel,leads to sluggish blood flow and a greater chance of having a clot.

PATHOANATOMY

The veins have one-way valves to prevent them from backward flow. The correct functioning of the venous system depends on a complex series of valves. It has been known that varicose veins in the legs are caused by weakening of the veins and valves in the great saphenous veins and/or small saphenous veins. Due to the malfunction in the valves, blood begins to collect in the legs resulting in the build up of pressure. The veins become enlarged and knotted and are visible near the surface of the skin as a varicose vein. Major valves which dysfunctions in the caution of varicose vein are saphenofemoral junction (SFJ) and saphenopopliteal junction (SPJ).The

termination point of the GSV into the common

Femoral vein, located proximally at the groin, is called the Saphenofemoral junction. The terminal valve of the GSV is located within the junction itself. In most cases, atleast one additional sub terminal valve is present within the first few centimeters of the GSV. Most patient's have a single sub terminal valve that can be readily identified approximately 1 cm distal to the junctional valve.

PATHOPHYSIOLOGY

The path physiology behind their formation is complicated and involves the concept of ambulatory venous hypertension. In healthy veins, the flow of venous blood is through the

superficial system into the deep system and up to the leg and toward the heart. One-way venous valves are found in both systems and the perforating veins. Incompetence in any of these valves can lead to a disruption in the unidirectional flow of blood toward the heart and result in ambulatory venous hypertension (AVH).

Incompetence in the superficial venous system alone usually results from failure at valves located at the SFJ and SPJ. The gravitational weight of the column of blood along the length of the vein creates hydrostatic pressure, which is worse at the more distal aspect of the length of vein.

Reflux at or near the SFJ does not always come through the terminal valve of the GSV, nor does it always involve the entire trunk of the GSV. Reflux can enter the GSV below the sub terminal valve or even immediately below the junction, passing through a failed sub terminal valve to mimic true SFJ incompetence. Reflux can also pass directly into any of the other veins that join the GSV at that level, or it may pass a few centimetres along the GSV and then abandon the GSV for another branch vessel.

Incompetence of the perforating veins leads to hydrodynamic pressure. The calf pump mechanism helps to empty the deep venous system, but if perforating vein valves fail, then the pressure generated in the deep venous system by the calf pump mechanism are transmitted into the superficial system via the incompetent perforating veins. Once venous hypertension is present, the venous dysfunction continues to worsen through a vicious circle. Pooled blood and venous hypertension leads to venous dilatation, which then causes greater valvular insufficiency.

Over time, with more local dilatation, other adjacent valves sequentially fail, and after a series of valves has failed, the

entire superficial venous system is incompetent. This can then cause subsequent perforator and deep venous valvular dysfunction.

The clinical findings of varicose veins, reticular veins, and telangiectasias are due to the hypertension in the superficial venous system that spreads to collateral veins and tributary veins, causing dilated tortuous structures.

Treatment modalities are geared towards correcting the superficial venous hypertension. In contrast to the superficial veins, the deep veins do not become excessively distended. They can withstand the increased pressure because of their construction and the confining fascia.

CONCLUSION

- The veins have one-way valves so that the blood can travel in only one direction. If the walls of the vein become stretched and less flexible (elastic), the valves may get weaker. A weakened valve can allow blood to leak backward and eventually flow in the opposite direction. When this occurs, blood can accumulate in the vein(s), which then become enlarged and swollen.

- Several factors predispose individuals to developing varicose veins. Sitting and standing for long periods exert pressure on the leg veins. Carrying extra weight can have a similar effect; that's why pregnant women are more prone to varicose veins, especially after multiple births. In addition, there's a genetic component, as varicose veins tend to run in families.

- By avoiding standing, sitting for a long time, maintaining appropriate body weight, Life style modifications, and dietary changes, along with Ayurvedic treatment helps to prevent Sirajagranthi (varicose veins).

- In order to diagnose the disease the physician should work very hard to know the causes of disease.

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