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DAWALI (VARICOSE VEINS): DESCRIPTION IN UNANI SYSTEM OF MEDICINE

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ABSTRACT

The dilatation of veins due to the accumulation of natural *saudavi maadda* (atrabilious matter) is known as *Dawali* (varicose veins).[1] Dilatation of leg veins is known as *Dawali.* It is caused by pooling of *saudavi khoon*.[2,3] Tortuous, dilated and prominent veins in legs are known as *Dawali*.[4,5] *Dawali* is a disease in which veins of the legs become dilated, tortuous and green due to the accumulation of *saudavi khoon*.[6,7] In this disease dilated, tortuous veins of different shapes and colour appear in legs.[8] In *Dawali* the veins in legs become thick due to excess pooling of *saudavi* or *balghami khoon* or *tabai khoon*.[9] It is a disease in which the veins of the legs become dilated, tortuous and green due to the accumulation of *saudavi khoon*.[6,10]

KEYWORDS: Dawali; Varicose Veins; Unani Medicine; Leeching, Alqa, Hirudina Medicinalis.

INTRODUCTION

Varicosity is a disease in which veins of legs and feet become dilated due to excess accumulation of blood which is derived from *saudavi maadda* (atrabilious matter), *ghair saudavi maadda* or *balghame ghaleez* (viscous phlegm).[11] When a vein becomes dilated, elongated and tortuous, the vein is said to be "varicose.".[12] Varicose veins are dilated, tortuous superficial veins that result from defective structure and function of the valves of the saphenous veins, from intrinsic weakness of the vein wall, from high intraluminal pressure, or, rarely, from arteriovenous fistulas.[13,14,15]

Historical Background

The word "varicose" is an old one. The origin of the word comes from the Greek "grapelike". It was probably first used as a medical description by Hippocrates in 460 BC. Indeed, since the beginning of written history, mankind has suffered from and devised many treatments for varicose veins. Methods of treatment have been under development for more than 2000 years, but until the present era, relatively little weight was given to the cosmetic outcome. Varicose veins were first described in the *Ebers papyrus* over 3,500 years ago. This ancient Egyptian work described 'serpentine windings' which were not to be operated on because the patients would be 'head to the ground'. This is the first description of what must have been a failed attempt at surgery to treat varicose veins, implying that the use of incisions leads to fatal haemorrhage. Hippocrates wrote some of the earliest medical descriptions of varicose veins. The Hippocratic Treatises, written in 460 BC, took treatment one step further and whilst he did not recommend the excision of varicose veins, he prescribed compression following multiple punctures. He also believed in cautery and is quoted as saying 'what cannot be cured by medicaments is cured by the knife, what cannot be cured by the knife is cured by the searing iron, whatever this cannot cure must be considered incurable'. The Greek, Paulus Aegineta (625-690 A.D.), favoured ligation of the long saphenous vein long before Trendelenburg 200 years later. He wrote: 'Varices of the leg may be operated upon in a manner similar to that for varicocele.' The leech was used in medicine as a means of "local depletion" (bloodletting) from the ancient days of Greece, Rome and Arabia. In Greece, they were first mentioned by Themison (80-40 B.C.), a pupil of Asclepiades, who lived a century before Christ.[16]

Epidemiology of *Dawali*

Approximately 15% of adults have varicosity of veins.[17] Varicose veins are extremely common in America, one branch of the Framingham study found that the incidence was 2.6% in women and 2.0% in men.[18] A study conducted in London of people aged 35 to 70 years concluded that the prevalence of varicose veins in men

and women was 17% and 31% respectively.[19] A survey to determine the prevalence of varicose veins among railwaymen of identical socio-economic status and doing identical work of sweepers in the North and South of India showed that the overall prevalence was significantly higher among South Indian sweepers (25.08 per cent) than among North Indian sweepers (6.8 per cent).[20]

Types of *Dawali*

Varicose veins can be categorized as primary or secondary.

Primary varicose veins: Primary varicose veins originate in the superficial system and occur two to three times as frequently in women as in men.[13] The fundamental defect in primary varicosity is incompetent venous valves. This may be inherited. Incompetent valves of perforating veins in thighs and lower limbs, communicate the pressure from deep venous system to superficial venous system.[17]

Secondary varicose veins: Secondary varicose veins result from deep venous insufficiency and incompetent perforating veins or from deep venous occlusion causing enlargement of superficial veins that are serving as collaterals.[13]

Aetiology of *Dawal*

- Varicose veins develop due to continuous and prolong walking, and in people who stand during their work.[1,2,4,6,9,10] During prolonged standing long column of blood along with gravity puts pressure on the weakened valves of the veins. This causes failure of the valves quickly giving rise to varicosity of the long or short saphenous vein. During prolonged standing the calf muscles also do not work quit often so the calf pump mechanism also cannot push the venous blood upwards.[12]
- It occurs frequently in weight lifting people.
- Continuous and excessive use of sauda producing foods.[1,2,4,6,9,10,11]
- It occurs in peoples who live in hot and moist places. It is common in Bengal (India).[21]
- Usually it occurs after acute diseases specially diseases of *Tihal* (Spleen) or it may occur as primary disease.[1,6,10,11]
- *Dawali* occurs due to accumulation of non-purulent *balghami* (phlegmatic), *saudavi* (atrabilious) or *damvi* (sanguineous) matter in leg veins.[2,6,8,9,10,21]
- Weakness in the walls of the superficial veins. This weakness may be inherited. Over time, the weakness causes the veins to lose their elasticity. They stretch and become longer and wider. To fit in the same space that they occupied when they were normal, the elongated veins become tortuous, with a snake like appearance they cause a bulge in the skin over them. Widening causes the valve cusps to separate as a result, the veins rapidly fill with blood when the person stands, and the thin walled, tortuous veins enlarge even more. The enlargement also affects some of the connecting veins, which normally allow blood to flow only from the superficial veins into the deep veins. If the valves of these connecting veins fall, blood squirts backward into the superficial veins when the muscles squeeze the deep veins, causing the superficial veins to stretch further.[17]
- **Obesity** excessive fatty tissue in the subcutaneous tissues offer poor support to the veins. This leads to the formation of varicosity.
- **Pregnancy** is said to predispose the formation of varicose veins. Varicose veins are often noticed in multiparous women. Pregnancy acts in 2 ways.
- **Progesterone** causes dilatation and relaxation of the veins of the lower limb. This makes the valves incompetent. This hormonal effect is more in the first trimester of pregnancy.
- **Pregnant uterus** causes pressure on the inferior vena cava, thus causing obstruction to the venous flow. This effect is mostly seen in the last trimester of pregnancy. After each pregnancy both hormonal and mechanical effects are removed and there is improvement of varicosity. During the subsequent pregnancy these factors again cause the varicosities to develop in a bigger way. That is why varicose veins are commonly seen in multiparous women.
- **Old age** causes atrophy and weakness of the vein wall. At the same time with aging the valves in the veins become gradually incompetent.
- **Athletes**, sometimes varicose veins are noticed among athletes. Forcible contraction of the calf muscles may force blood through the perforating veins in reverse direction. This will cause destruction of the valves of the perforating veins and ultimately lead to formation of varicose veins. Similarly, Rickshaw pullers often suffer from varicose veins.[22]

Clinical Features of *Dawali*

1. Veins become long dilated and tortuous.[1,6,10]
2. **Tiredness and pain:** Veins become heavy.[21] The commonest symptom is tiredness and aching sensation in affected lower limb, particularly in calf, at the end of the day. The severity of symptoms depends mostly on the extent of the high back pressure; it is relieved with leg elevation.[12,13,17,22,23,24] Pain may be bursting or severe in nature and may be particularly localized to the site of the incompetent perforating veins, such bursting pain while walking indicates deep vein insufficiency.[12]
3. **Cramps:** Some patients may suffer from cramps in the calf shortly after retiring to bed. Such cramp is usually due to sudden change in the caliber of communicating veins which stimulates the muscles through which they pass.[12]
4. **Difficulty in walking**
5. **Ankle swelling (*Oedema*):** The legs feel heavy, and occasionally mild ankle oedema develops.[12,13] During standing venous pressure at the ankle is 85-

90 mm Hg and rhythmic contraction of leg muscles lower the venous pressure in the leg to less than 30 mm Hg by propelling blood towards heart. This heart ward movement of blood is decreased in patients with varicose veins because their valves are incompetent. These patients may develop stasis or ankle oedema. Interference with the return of the blood to the heart also contributes to oedema. This is seen in case of localized venous thrombosis.[25]

6. Itching (Pruritus)

The lower part of the leg and ankle may itch, especially if the leg is warm after the person removes socks or stockings. Itching can lead to scratching and cause redness or a rash, which is often incorrectly attributed to dry skin. The symptoms are sometimes worse when varicose veins are developing than when they are fully stretched.[12,17,23]

Anatomy of Veins of lower limb

The veins may be classified into 3 groups: superficial, deep and perforators.

Superficial Veins

They include the **Great and Small Saphenous veins, and their Tributaries.** They lie in the superficial fascia, on the surface of the deep fascia. They are thick-walled because of the presence of smooth muscle and some fibrous and elastic tissues in their walls. Valves are more numerous in the distal parts of these veins than in their proximal parts. A large proportion of their blood is drained into the deep veins through the perforating veins.

Long or Great Saphenous Vein

Saphenous opening lies 4 cm below and 4 cm lateral to the pubic tubercle. It is about 2.5 cm long and 2 cm broad, with its long axis directed downwards and laterally. Great Saphenous Vein can be marked by joining the following points, although it is easily visible in living subjects.

- 1st point on the dorsum of the foot at the medial end of the dorsal venous arch.
- 2nd point on the anterior surface of the medial malleolus.
- 3rd point on the medial border of the tibia at the junction of the upper 2/3rd and lower 1/3rd of the leg.
- 4th point at the adductor tubercle.
- 5th point just below the center of the saphenous opening.

Long saphenous vein begins on the dorsum of the foot, runs along the entire length of the lower limb, and terminates into the femoral vein. It contains about 10-20 valves. There is 1 valve that lies just before the vein pierces the cribriform fascia and another at its termination into the femoral vein.

Tributaries

- At the commencement: medial marginal vein from the sole.
- At the leg: it communicates freely with the small saphenous vein and with deep veins.
- Just below the knee: (1) the anterior vein of the leg runs upwards, forwards and medially, from the lateral side of the ankle. (2) The posterior arch vein is large and constant. It begins from a series of small venous arches which connect the medial ankle perforators, and runs upwards to join the great saphenous vein just below the knee. (3) A vein from the calf: this vein also communicates with the small saphenous vein.
- In the thigh: (1) the accessory saphenous vein drains the posteromedial side of the thigh. It may communicate with the small saphenous vein. (2) The anterior cutaneous vein of the thigh drains the lower part of the front of the thigh.
- Just before piercing the cribriform fascia: (1) superficial epigastria (2) superficial circumflex iliac and (3) superficial external pudendal.
- Just before termination: deep external pudendal vein.[26]

Small or Short Saphenous Veins

It can be marked by joining the following points, although this vein is also easily visible in its lower part.

- 1st point on the dorsum of the foot at the lateral end of the dorsal venous arch.
- 2nd point behind the lateral malleolus.
- 3rd point just lateral to the tendocalcaneus above the lateral malleolus
- 4th point at the center of the popliteal fossa.

It commences on the dorsum of the foot, runs along the back of the leg, and terminates into the popliteal vein. Just before piercing the popliteal fascia, it may give a communicating branch to the accessory saphenous vein. Sometimes, the whole of the small saphenous vein opens into the great saphenous vein through the accessory saphenous vein. Occasionally, the small saphenous vein ends below the knee either in the great saphenous vein, or in the deep muscular veins of the leg.[26]

Deep Veins

These are the **Anterior and Posterior Tibial, Peroneal, Popliteal, and Femoral veins and their Tributaries.** They accompany the arteries, and are supported by powerful surrounding muscles. The valves are more numerous in deep veins than in superficial veins. They are more efficient channels than the superficial veins because of the driving force of muscular contraction.

Perforating Veins

They connect the superficial with the deep veins. Their valves permit only one-way flow of blood, from the superficial to the deep veins. There are about 5 perforators along the great saphenous veins, and 1 perforator along the small saphenous vein.[26] These are classified as follows.

Indirect perforating veins connect the superficial veins with deep veins through the muscular veins.

Direct perforating veins connect the superficial veins directly with the deep veins. The great and small saphenous veins are the large direct perforators. The small direct perforating veins are summarized below.

- **In the thigh:** The abductor canal perforator connects the great saphenous vein with the femoral vein in the lower part of the adductor canal.
- **Below the knee:** 1 perforator connects the great saphenous vein or the posterior arch vein with the posterior tibial vein.
- **In the leg:**
- A lateral perforator is present at the junction of the middle and lower thirds of the leg. It connects the small saphenous vein, or one of its tributaries with the peroneal vein. Medially there are 3 perforators which connect the posterior arch vein with the posterior tibial vein.
- The upper medial perforator lies at the junction of the middle and lower thirds of the leg.
- The middle medial perforator lies above the medial malleolus.
- The lower medial perforator lies posteroinferior to the medial malleolus.[26]

Physiology of the venous system in the lower limb

The primary and most obvious function of the venous system is to return the blood to heart, the blood that has passed through arteries and capillaries. Four aspects of normal venous hemodynamic are of concerned:

1. **Venous pressure**

- **Effect of right atrial pressure on peripheral venous pressures:** Blood from all the systemic veins flows into the right atrium; therefore, the pressure in the right atrium is called *central venous pressure.* When the right atrial pressure rises above its normal value of o mm Hg, blood begins to back up in the large veins and open them up. The pressure in the peripheral veins does not rise until all the collapsed points between the peripheral veins and the large central veins have opened up. This usually occur when the right atrial pressure rises to about +4 to +6 mm Hg. Then as the right atrial pressure rise still further, the additional increase in pressure is reflected by a corresponding rise in peripheral venous pressure.
- **Effect of intra-abdominal pressure on venous pressures of the leg:** The normal pressure in the abdominal cavity of a recumbent person averages about 6 mm Hg, but it can rise to 15-30 mm Hg as a result of pregnancy, large tumours, or ascites in the abdominal cavity. When this happens, the pressure in the veins of the legs must rise above the abdominal pressure before the abdominal veins will open and allow the blood to flow from the legs to the heart. Thus, if the intra-abdominal pressure is 20 mm Hg, the lowest possible pressure in the femoral vein is 20 mmHg.

2. **Effects of gravitational pressure on venous pressure:** Gravitational pressure also occurs in the vascular system of the human being because of the weight of the blood in the vessels. When a person is standing, the pressure in the right atrium remains about 0 mmHg because the heart pumps into the arteries any excess blood that attempts to accumulate at this point. However, in an adult who is standing absolutely still, the pressure in the veins of the feet is about + 90 mmHg simply because of the gravitational weight of the blood in the veins between the heart and the feet. The venous pressures at other levels of the body lie proportionately between 0 & 90 mmHg.[27,28]
3. **Venous volume and its relationship with compliance, pressure flow phenomenon in collapsible tubes:** The volume of the vein increases strikingly when the transmural pressure is raised. However, this phenomenon is not so much due to any stretching of the wall as to the geometric change in the cross-sectional profile as the transmural pressure rise. The cross-sectional area will influence the pressure-volume response of the vessel so that simply changing the profile from elliptical to circular by slightly increasing the transmural pressure will considerably raise the volume accommodated per unit length. Only when the cross-sectional profile is circular will a further increase of pressure cause stretch of the distensible elements of the venous wall. The collapsibility of the thin-walled veins is of the utmost importance in allowing striking changes of venous capacity in response to quite slightly changes of transmural pressure over a certain stage. If the transmural pressure becomes zero or even negative extra vascular pressure exceeds intravascular pressure, the vein collapses completely and resistance to flow increases sharply. Such changes occur in veins above heart level-the neck veins are collapsed in normal individuals (when sitting or standing) above a level which is 5-7 cm higher than that of the heart. Quiet standing considerably increases the volume of blood in the leg veins. On the other hand, the resistance offered by the leg veins to venous return is less, for the veins is all of circular profile.[29]
4. **Effect of musculovenous pump:** The gravitational pressure effect would cause the venous pressure in the feet always to be about +90 mm Hg in standing adult. However, every time one moves the legs, one tightens the muscle and compresses those veins in the muscles and adjacent to them, and this squeezes the blood out of the veins. The valves in the veins are arranged so that the direction of the blood flow can be only towards the heart. Consequently, every time person moves the legs or even tenses the leg muscles, a certain amount of blood is propelled toward the heart, and the pressure in the veins is lowered. This pumping system is known as the venous pump or muscle pump, and it is efficient enough that under ordinary circumstances, the

venous pressure in the walking adults remains less than 25 mm Hg. If a person stands perfectly still, the venous pump does not work, and the venous pressure in the lower part of the leg increases to the full gravitational value of 90 mm Hg in about 30 sec. The pressures in the capillary also increase greatly causing fluid to leak from the circulatory system into the tissue spaces. As a result, the legs swell and the blood volume diminishes. Indeed, 10-20% of the blood volume can be loosed from the circulatory system within the first 15 minutes of standing absolutely still, as often occurs when a soldier is made to stand at rigid attention.[27]

Pathophysiology of *Dawali*

In case of prolong standing, walking and excess production of *sauda*, *dam* or *ghaleez balgham*. The excretory faculty of body propels matter towards the lower limbs to prevent vital organs; the retentive faculty of lower limb retains it in lower limb resulting in *Dawali*. This may be due to weakness of affected organ. The dilatation of vein depends upon the amount of matter reaches to the affected organ.[1,2,9,11]

Uncomplicated *Dawali*

Failure of competence in the venous valves will lead to retrograde flow down the limb when the patient stands up or after exercise movement has resulted in slack veins in the lower part of the leg. In the superficial veins, this is the basis of the most common venous disorder – simple varicose veins.[30] The term "varicose vein" refers to veins that are abnormally large and tortuous. This term applies to both the large protruding veins within the superficial subcutaneous fascia and the smaller "spider veins" that occur just beneath the epidermis.[31] Varicose veins are classified according to the three circumstances of unnatural flow that result in enlarged tortuous veins. Primary varicose veins occur only in the superficial veins of the lower limb and are by far the most common variety of this disease.

The pathogenesis of chronic venous diseases still largely unclear. Some authors believe that varicose vein result from a failure of valves in the superficial veins, leading to venous reflux and vein dilatation.[31] Another theory suggests that the structural integrity of the vein wall is compromised, resulting in dilation of the vein and separation of valve cusps, which subsequently renders a previously competent valve incompetent.[32] Secondary varicose veins are caused by an obstruction in the deep vein system of the lower limb, in which the superficial veins work as collateral vessels. Secondary venous insufficiency may sporadically also occur as a complication of an arteriovenous fistula. In the case of teleangiectases, the dilatations develop between the epidermis and the hypodermis in the subpapillary dermal plexus and usually measure between 0.1 and 1 mm. They may be constituted by veins, arteries or capillaries. In chronic venous insufficiency, all teleangiectases are accompanied by reticular varices, even when not visible on a clinical examination. In most cases, the sources of reflux are distinguishable as incompetent perforating veins and situated beneath teleangiectactic efflorescences.[33] Superficial venous insufficiency is most often caused by reflux via venous junctions or via perforating veins of the superficial veins to the LSV or SSV trunks. Less common sources of retrograde circuit are the ovarian veins via the pelvic veins or the pelvic veins to the upper thigh and the Giacomini branch or gastrocnemius vein via a mid-leg perforator to the calf. Until a few years ago, VV were considered to be a response to a dynamic process secondary to reversed flow and not merely a result of static distension of the veins. However, the suggestion that valvular insufficiency in the LSV or SSV is the principal cause underlying varicosities has now been questioned. Originally, it was thought that the descending valvular incompetence of veins commenced proximally and progressed distally. The results of duplex scanning studies have shown that reflux in the LSV is quite common without SFJ incompetence.[34,35] And the authors have suggested that the development of the primary varicose vein may be an ascending rather than a descending phenomenon.[36] In the CEAP clinical classification, objective signs of CVD are divided into seven categories with further subscripts to indicate the presence or absence of symptoms.[37,38] Patients with uncomplicated CVD (teleangiectasies or varicose veins) are often asymptomatic. The relationship with mild clinical findings (CEAP clinical class 0-3) and leg symptoms is poor. Thus, surgery is unlikely to ameliorate symptoms in the majority of patients with varicose vein.[39]

Complicated *Dawali*

Venous insufficiency is a condition of inadequate venous return and hypertension when the patient is in an upright position. An increase in venous pressure results in a corresponding increase in capillary pressure and characteristic changes in the skin and subcutaneous tissue. Capillary transudation with protein molecules leads to deposition of fibrin, which forms a barrier to nutritional exchange between the capillaries and the surrounding tissues.[40] Leukocytes are trapped in the capillaries causing further damage to the endothelium and the vessel walls and slowing down microvascular circulation.[41] Extravasated haemosiderin gives the characteristic brown skin pigmentation. The outflow of fluid and corpuscles from the capillaries into the interstitial tissue initiates some of the mechanisms leading to symptoms of chronic venous insufficiency,[40] swelling, venous eczema and dermatitis, lipodermatosclerosis, pigmentation and finally venous ulcer take many months, or even years, to develop. Sensory neuropathy is another feature of severe chronic venous insufficiency, and its distribution is coincident with trophic changes.[42] Skin changes ascribed to chronic venous disease may be caused by insufficiency of the deep or the superficial venous system or both. In patients with skin changes and ulceration, the incidence

of reflux in the superficial veins has been found to vary from 17% to 53%.[43,44] In duplex scanning studies, reflux in the whole LSV occurs in approximately 8% of limbs with venous ulcer.[45,46] However, Bello *et al* observed deep venous obstruction in roughly two-thirds of such limbs.[46] At any rate, the role of superficial venous insufficiency seems to be important in the development of venous skin changes

Complications of *Dawali*

- **Thrombosis**
- **Pigmentation:** Veins of calf or leg become, greenish.[1,6,10,21] Venous stasis of long-standing leads to secondary changes in the skin. There may be a network of superficial varices and pigmentation there is actual leakage of blood cells into the tissues of the lower leg. The skin becomes brown in colour due to deposition of haemoglobin within the tissues. This "stasis pigmentation" can be associated with itching and thickening of the skin.[12,17,25]
- **Eczema:** Venous eczema is a complication of varicose veins.[22] This may or may not be associated with the dilatation and tortuousity of veins.[12] Lipodermatosclerosis is a skin and connective tissue disease characterised by inflammation of the layer of fat under the epidermis. It is a form of lower extremity panniculitis,[47] an inflammation of the layer of fat under the epidermis.[48] Pain may be the first noticed symptom of lipodermatosclerosis.[47] People with lipodermatosclerosis have tapering of their legs above the ankles, forming a constricting band resembling an inverted coke bottle.[47,49] In addition, there may be brownish-red pigmentation and induration.[49] Increased blood pressure in the veins (venous hypertension) can cause diffusion of substances, including fibrin, out of capillaries. Fibrotic tissue may predispose the tissue to ulceration. Recurrent ulceration and fat necrosis is associated with lipodermatosclerosis. In advanced lipodermatosclerosis the proximal leg swells from chronic venous obstruction and the lower leg shrinks from chronic ulceration and fat necrosis resulting in the inverted coke bottle appearance of the lower leg.[50]
- **Venous haemorrhage**[12]
- **Venous ulceration:** Extensive venous varicosities may cause skin ulcerations near the ankle.[13] The most serious problem is venous ulceration, which complicates varicose veins in less than 5% of patients. However, it is a troublesome and painful condition which requires careful management if the ulcer is to heal.[22] The skin over a varicose vein may become poorly nourished due to stasis of blood, leading to the formation of varicose ulcers usually on the medial aspects of the leg just above the ankle.[51]

Diagnosis

A full history should always be taken, Patients report a wide range of symptoms associated with venous disease. These include tiredness, aching, tingling and ankle swelling, which get progressively worse towards the end of the day are relieved by elevating the leg. Patients with severe deep vein obstruction may also develop bursting pain in the calf on walking, due to the very high venous pressures that may occur under these conditions.[22]

Local Examinations

- **Inspection:** When the patient stands up, the veins become prominent. The varicosities may be either wide spread or restricted to a single varix. When such single varix is situated at the saphenous opening, it is called a 'saphena varix'. It must be distinguished from a femoral hernia. One can feel characteristic thrill when the patient coughs. Such varix disappears when the patient lies down. When this varix is tapped with a finger, a fluid thrill may be obtained in the long saphenous vein lower down in the limb. One must assess in inspection whether varicosity has affected the long saphenous vein or the short saphenous vein or the both. The skin of the lower part of the leg should be particularly inspected to exclude oedema, pigmentation, eczema or ulceration.[12]
- **Palpation:** The dilated veins are particularly palpated. The saphena varix is palpated and presence of cough impulse is elicited.
- **Tests:** A few tests are performed to know the details of the varicose vein including the sites of incompetent perforators.[12]
- **Brodie-Trendelenburg test:** This test is performed to determined incompetency of the sapheno-femoral valve and other communicating system. This test can be performed in two ways. In both the methods, the patient is first placed in the recumbent position and his legs are raised to empty the veins. The sapheno-femoral junction is now compressed with the thumb of the clinician and the patient is asked to stand up quickly. In first method, the pressure is released. If the varices fill very quickly by a column of blood from above, it indicates incompetency of the sapheno-femoral valve. This is called a positive Trendelenburg test. To test the communicating system, the pressure is not released but mentioned for about one minute. Gradual filling of the veins during the period indicates incompetency of the communicating veins, mostly situated on the medial side of the lower half of the leg allowing the blood to flow from the deep to the superficial veins. This is also considered as a positive Trendelenburg test.[12]
- **Tourniquet test:** It can be called a variant of Trendelenburg test. In this test the tourniquet is tied around the thigh or the leg at different levels after the superficial veins have been made empty by raising the leg in recumbent position. The patient is now asked to standup. If the veins above the tourniquet fill up and those below it remain collapsed, it indicates presence of incompetent communicating vein above the tourniquet. Similarly if the veins below the tourniquet fill rapidly whereas

veins above the tourniquet remain empty, the incompetent communicating veins must be below the tourniquet. Thus by moving the tourniquet down the leg in steps one can determine the position of the incompetent communicating vein.[12]

Other examinations

- **Examination of the abdomen:** this is essential to exclude pregnancy or presence of any pelvic tumor as the cause of varicosity. Such varicosity is called secondary varicosity. Any dilated collateral veins in the abdomen should be carefully noticed. These veins develop due to inferior vena caval obstruction particularly thrombosis. Such veins are more often seen in the flanks. Communicating with the veins of the chest wall, tributaries of the superior vena cava.
- **Vaginal and rectal examinations** are must to exclude pelvic tumours.
- **Peripheral arterial pulses** should also be examined to exclude presence of arterial insufficiency. Ulcers in the lower limb with presence of varicose veins may not necessarily be the venous ulcers. Such ulcers may occur due to ischaemia from arterial insufficiency and are known as arterial ulcers. In these cases, varicose vein is the second pathology and not the cause of the ulcer.[12]
- **Doppler ultrasound:** A Doppler assessment is now the minimum level of investigation required before treating a patient with venous disease. A Doppler flow probe can be used to exclude arterial disease and to determine the patency of a vein, and a bidirectional flow probe used to detect venous reflux. This investigation is carried out with the patient standing. The Doppler probe is first placed over the SPJ and the blood flow assessed to locate the venous flow in the common femoral vein. With one hand the examiner gently squeezes the calf to produce an acceleration of blood flow in the veins. This is heard as a 'whoosh' from the loudspeaker of the Doppler machine. The calf compression is released and any reverse flow in the veins sought. With practice, it is possible to identify venous reflux in the SFJ reliably. The examination may be repeated with the probe held over the long saphenous vein in the mid-thigh region to confirm that the venous reflux lies in the superficial vessels. Some surgeons use a tourniquet to occlude the superficial veins, in the same way as when performing a Trendelenburg test. The probe may also be held over the SPJ while the calf is compressed and released to test the competence of veins in this region. In the popliteal fossa, it is more difficult to distinguish between deep and superficial venous incompetence. This method is very useful when examining patients with primary varicose veins, especially those which are thought to result from SFJ incompetence. The popliteal fossa contains many veins and, if venous reflux is heard, it is difficult to be certain from which vein it arises. However, in patients with primary varices saphenopopliteal incompetence is usually readily identified. All surgeons who regularly treat patients with varicose veins should be competent at this type of investigation. When the source of recurrent varices or a leg ulcer is sought, duplex ultrasonography is usually more reliable.[22]

Prevention & Control of *Dawali*

- **Lifestyle Modifications:** Avoid prolong walking, standing and sitting.[1,2,6,10,12,52] Avoidance of prolong standing relieves symptoms of varicose veins.[13,53] Avoid sitting with leg crossed, it may not be possible in our culture during certain circumstances.[53,54,55,56,57]
- **Leg Elevation:** Elevate the legs regularly,[1,6,10] Elevate the legs by lying down or using a foot stool at least twice a day for 30 minutes at a time.[54] Sitting relieves the symptoms but not prevent varicose veins.[12,13,17,23,57,58]
- **Exercise:** Physical activities improve muscle tone. This helps blood to move through veins. Exercise like "bicycle riding" in the air while lying on the back, walking etc. should be performed to strengthen the calf muscles.[12]
- **Avoid wearing high heels for long periods**: Low heeled shoes can help to tone calf muscles. Toned muscles help blood to move through the veins.
- **Weight reduction:** This will improve blood flow and ease the pressure on veins.
- **Dietary Modifications:** Reduce the quantity of food. Avoid those foods and drinks that produce more and *ghaleez khoon* i.e. *Moallide Sauda wa balgham aghzia*.[1,2,4,6,8,9,10,11,52] All vegetables produce some amount of *sauda* for example *Baqla*, *kabuli matar*, *karnab* (cabbage), *Bazanjan* (Brinjal) and Cereals like *Jau* (Barley), *Ads (*Lentil), *Matar* (Peas) are also *Moallide sauda*.[4,59] Eat fibre-rich food such as *chapattis*, whole wheat bread, vegetables and fruits. Avoid refined foods such as *maida* and pastas.[60]
- **Evacuation of morbid matter:** All the sign & symptoms of *Dawali* develop due to accumulation of *saudavi khoon* in lower limb. To relieve the sign & symptoms of *Dawali* morbid matter should be removed. Unani physicians used emesis, purgation & bloodletting from ancient time for the prevention and control of *Dawali*. Most of the Unani physicians used fasd for the evacuation of diseased matter in *Dawali*. *Taleeq* was used as a substitute of *fasd*, and was preferred over fasd because of definite benefits that it entails. It seems to be effective for the prevention & control of *Dawali*, due to its salivary secretions which it injects during bloodsucking. The saliva of leech contains certain biochemical with vaso-dilating, anti-coagulant, anaesthetic, thrombolytic, antibiotic, analgesic, and anti-inflammatory properties.

CONCLUSION

Prevalence of varicose veins is 15%. Incidence of varicose veins increases with advancing age. It is assumed that aetiology of varicose vein is multifactorial. Complications of varicose veins like venous eczema, venous pigmentation, lipodermatosclerosis, superficial thrombophlebitis, venous ulceration which is more troublesome, distressing and painful condition. Health related quality of life is significantly impaired in individuals with vascular disease.

SUMMARY

Dawali is a disease in which veins of legs and feet become dilated, elongated, tortuous and greenish in colour. The aetiology of varicose vein is still incompletely understood despite the fact that it is a very common disease affecting all ages from teenagers to elderly people. Greeko-Arab physicians postulated that it is caused by accumulation of non-purulent *balghami, saudavi* or *damvi* matter in leg veins or due to weakness. The patient may report aching & restless leg, itching, swelling in leg, pigmentation, venous ulceration, lipodermatosclerosis, eczema etc. health related quality of life is significantly impaired in individuals with varicose veins.

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