Review Article

Varicose Veins: A Comprehensive Review of Pathophysiology, Anatomy and Management Strategies

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ABSTRACT

The treatment of varicose veins [VV] and Post-thrombotic Malady the epidemiology, and pathophysiology of the aforementioned conditions are still unclear. As a result, therapy outcomes are typically less than desirable. Varicose veins differentiate themselves from aging veins. Varicose veins exhibit a compact and dense fibrosis between the intima & adventitia, a reduced elastic network, a disordered muscle layer, and thickened and fibrillated collagen fibers. The VV corresponds to a abnormally vein with abnormalities. Pressure-induced distention may become much more prevalent. Dysplastic has been linked to altered pharmacodynamics and histochemistry in VV. VV exhibits a decrease of stypsis. Varicose veins become easily distended due to anatomical and metabolic breakdowns. According to Baron [varicose veins may develop due to weak or nonfunctioning venous valves. This could be caused to a hereditary deficit in vain wall collagen. Numerous studies show a link between inheritance & the emergence of the varicose veins in different populations.

Keywords: Varicose veins, anatomy, pathophysiology, management.

INTRODUCTION

Treating venous reflux and obstruction, whether deep and peripheral, which causes VV[1] and Post-thrombotic syndrome (PTS)[2] Forms a significant portion of the workload of almost all vascular & endovascular specialists which is expected to expand as the population ages.[3] However, the epidemiology,[4,5] genetics,[6] and pathophysiology of the aforementioned conditions are still unclear,[7,8,9,10,11] and many clinicians have an insufficient knowledge of the root anatomy and vascular biology.[12] As a result, therapy out-comes are typically less than desirable.

Anatomy

The lower limb venous system has a pair of channels: one beneath the muscular system along with another superficial to it (Fig. 1).

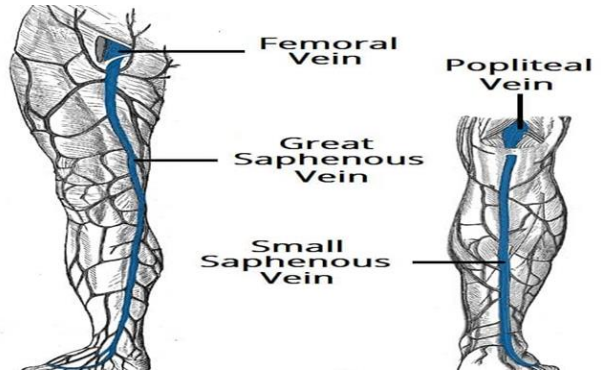

Figure 1: Simplistic diagram of the venous system in the leg.

The femoral and popliteal veins are the main deep veins, which originate in the foot as the plantar digital vein. These have one-way valves every couple of millimeters. The one-way valves guide blood flow to the heart (Fig. 2). Muscular compression causes 90% of venous blood to depart the leg through the deep veins. This system is known as the "calf-pump" or "peripheral heart."[13]

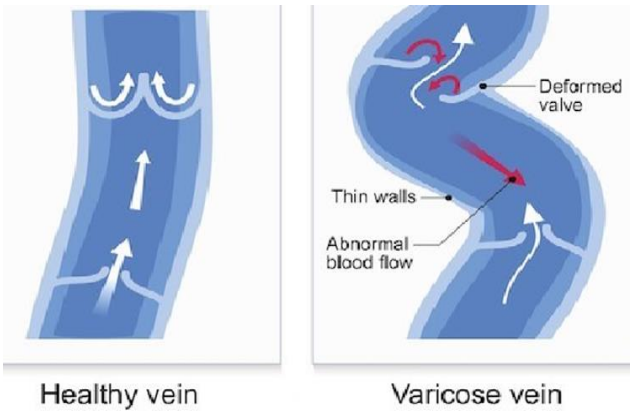

Figure 2: Diagram of blood flow via one-way valves in the vein

The deep venous system has several channels that connect to superficial veins, allowing the blood to circulate back to the heart even if the femoral vein is blocked. These more superficial veins also offer venous return to the cutaneous and subcutaneous systems via one-way valves.[14]

The mighty (long) saphenous vein is the most visible superficial vein. (The term saphenous comes from an ancient Greek word for "visible.") The dorsal venous arch

is where this vein begins on the medial part of the great toe. The vein begins at the medial malleolus & crosses the tibia obliquely. It then travels along the medial portion of the tibia, past the medial and anterior thigh, and ends in the femoral vein.

The accessory saphenous vein connects the lateral knee with the saphenous-femoral junction, the anterior vein connects the lower lateral calf with the medial knee, and a smaller (short) saphenous vein starts at the lateral aspect of the tiny toe and ascends to the lateral malleolus, then ascends to the calf between the gastrocnemius heads and ends in the popliteal fossa. The linkages between superficially and deep venous systems differ from the previous simple model. Raivio's[15] detailed anatomic studies help explain this variation.

Unnamed tributaries of the saphenous vein can become dilated and convoluted with increased venous pressure. Reticular or even connecting branch veins might develop varicose. Distinguishing between them and the primary venous system is crucial for treatment planning.

The deep fascia contains the high-pressure calf muscle pump. The superficial fascia surrounds the saphenous trunk.[16] The saphenous vein tributaries are not supported by the fascia, but rather lie on its surface. Tributaries of the great saphenous vein are typically more dilated than the vein itself, even if the cause is proximal.[17]

Histology

The immature internal saphenous vein is made up of three tunica's: intima, medium, and adventitia (Fig. 3). The intima is a narrow structure sur-rounded by a thin, fractured elastic lamina.[18]

The media consists of three layers of muscular bundles. The inner layer is made up of tiny bundles of longitudinally organized muscular fibers. Muscle bundles are separated by loose connective tissue and tiny elastic fibers.' The middle layer consists of smooth muscle bundles in a circular configuration.[18] Muscle bundles may be separated by thin or extensive layers of elastic fibers.[18] The outer layer is made up of muscle bundles that span thick fibrous tissue.

The adventitia connects itself to the perivenous connective tissue, housing the vasa vasorum and adrenergic nerve fibers.[19]

The intima thickens with age, resulting in a surge and disorientation of elastic fibers.[18] The media causes muscle bundles to become disordered and the outside muscular layer to grow. Elastic fibers become increasingly irregular and dystrophic.[18] The elastic lamina turns fractured, atrophic, thin, and uneven. The adventitia becomes more fibrous. An old vein's lack of elastic support increases its susceptibility to pressure-induced distention.
Varicose veins differentiate themselves from aging veins. Varicose veins exhibit a compact and dense fibrosis between the intima & adventitia, a reduced elastic network, a disordered muscle layer,[18] and thickened and fibrillated collagen fibers (see Fig. 4).[20] Hence, the varicose vein corresponds to a dysplastic vein with abnormalities. As a result, pressure-induced distention may become much more prevalent.

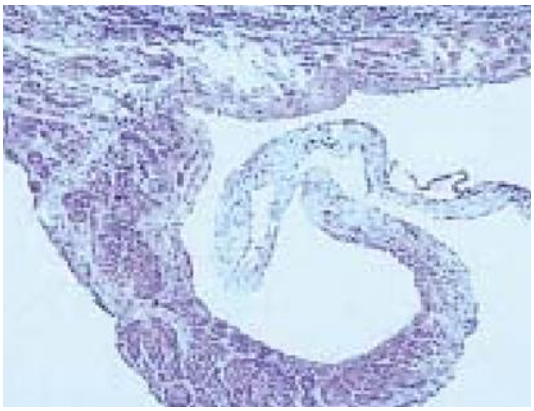

Figure 3: Microscopic cross-section through a normal great saphenous vein (H&E x 150)

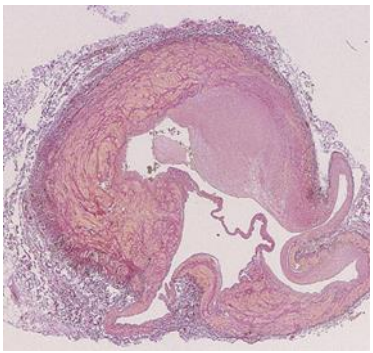

Figure 4: Cross-section through a typical varicose saphenous vein with partial hypertrophy of media and intimal hyperplasia (H&E x 150).

Dysplastic has been linked to altered pharmacodynamics and histochemistry in varicose veins. Varicose veins exhibit a decrease of contractility.[21] Histochemical analysis shows a significant rise in the activity of lysosomal enzymes[22] such as acid phosphatase, P-glucuronidase, & lactodehydrogenase in primary varicose vein.[23,24] Enzyme profiles indicate reduced energy consumption and increased cellular damage in varicose veins. Varicose veins collect and process norepinephrine less efficiently compared to normal veins.[25] Varicose veins become easily distended due to anatomical and metabolic breakdowns.

Pathophysiology

The venous blood pressure is influenced by the heart's output and the amount of energy lost before reaching the veins due to arteriole resistance.

Hydrostatic gravity forces are influenced by body posture, blood volume, venous wall anatomy, one-way valves, smooth muscle contraction, and ambient temperature.

At repose in the erect position, the saphenous vein's pressure is predominantly obtained from a column of blood from the right atrium. Measure the leg pressure (about 120 mmHg at the ankle). Muscular activity lowers

the pressure in the saphenous vein at the malleoli by 45-68 mm Hg and reduces the pressure in the posterior tibia vein from 80 to 40 mm Hg.[26] Blood flows through the superficial venous system into the deep venous system through connecting or perforating channels, which likewise have one-way valves. The venous blood flows to the heart.

In a prone state, blood flows uniformly via all vessels, both superficial and deep, to the heart, propelled by tiny vis a tergo through capillaries[27] and respiration-induced sucking into abdominal & thoracic veins. Compared to deep veins, superficial veins exhibit smooth muscle walls. This allows vessels to contract in response to cold, medicines (e.g., dihydroergotamine[28,29]), and dilate in response to alcohol, estrogen, and mild physical stress.[27] As previously established, a part of the pathophysiology of varicose veins might be caused by a reduced responsiveness of this smooth muscle contraction.

Irrespective of the source, chronic venous hypertension in lower extremities causes a surge in venous diameter, which leads to valvular dysfunction. Incompetent communicating veins cause blood flow reversal in the superficial venous system. McPheeters and Rice[30] studied the trajectory of venous flow in varicose veins using fluoroscopy. During sclerotherapy, the reversal of flow is advantageous since it redirects superficial venous flow to smaller branching veins[30], preventing the production of emboli.

When pressure increases, superficial veins dilate to allow for more blood flow. Valvular incompetence develops, and varicosities arise.[31] The motion of the lower limbs transmits high venous pressure from the calf to both superficial veins & subcutaneous tissues, which are emptied by connecting veins.[32,33] In the erect position, venous pressure across the cuticular venules can exceed 100 mmHg.[27] This induces venular dilation, leading to capillary dilatation, increased permeability,[34,35,36,37] and elevated blood volume across the subcutaneous capillary bed.[38]

A unique condition arises at the medial malleolus. In this location, there is no deep or considerable superficial fascia around the perforating veins.

As a result, the elevated deep venous pressure passes directly from the perforating vein to the superficial connecting vein. This causes increased cutaneous pressure and extracellular fluid transudation. Perivascular fibrin deposition can cause decreased oxygenation of cutaneous as well as supporting tissues, resulting to cutaneous ulcers.[35,39,40]

The impact of ambient temperature affecting the venous system is well understood.[41] Strain gauge venous occlusion plethysmography recently verified an increase in venous distensibility in response to temperature elevation.[42]

Varicose veins refer to unusually big and convoluted veins. This term refers to both the huge projecting veins underneath the superficial sub-cutaneous fascia & the smaller "spider veins" found just beneath the epidermis. Varicose veins can be caused by four factors, which may overlap: exacerbated deep venous pressure, intrinsic or secondary valvular incompetence, & fascial weakness.

Increased Deep Venous Pressure

An increase in deep venous pressure might be proximal distal in origin. Pelvic obstruction (which leads to indirect venous obstruction), necessary saphenofemoral incompetence, & venous obstruction are all proximal causes. Distal etiologic factors include communicating either perforating vein valvular incompetence, arteriovenous anastomosis, and venous blockage.

Pelvic obstruction is not a prevalent cause of varicose veins. Extravascular abdominal malignancies like ovarian and uterine cancer or teratoma may be the reason.

During the third trimester of pregnancy, abdominal or pelvic blockage is a typical cause of blood flow restriction. During pregnancy, hormonal stimulation causes venous dilatation in around 70% of cases during the first trimester (when the uterus only becomes slightly enlarged), 25% in the second trimester, and 5% in the third.[43]

Wearing girdles or tight-fitting garments can also cause proximal blockage outside of the body. Women who wear corsets had significantly more varicose veins than those who wear less constrictive apparel.[44] Women who additionally stand at work have a higher incidence than those who stroll or sit more frequently. Crossing one's legs can reduce venous return due to extracorporeal compression.[44] However, this "common knowledge" has not been properly explored.

Anatomic anomalies in the saphenofemoral triangle are rare causes of essential saphenofemoral incompetence. Pelvic tributary veins and supplementary saphenous veins may confluence, preventing effective flow to the femoral vein.[45] Additionally, iliac venous incompetence can be caused by congenital absence or valve damage from thrombosis.

Venous blockage can occur near or far from the varicose vein. The obstruction is usually caused by a thrombus that spreads both near and far from its source. Thrombosis may also involve communicating as well as perforating veins. Venous blood can be pushed to superficial veins in a retrograde either lateral manner (Fig. 5).

Distal anomalies are the most common cause of elevated venous pressure. According to Fegan[46], Hobbs[47], Lofgren[48], and Benison,[49] treating varicosities alone may not reduce superficial venous pressure. Treatment of the connecting or even perforating veins which drain the ankle & lower leg area is crucial.[32,50,51,52] These latter vessels might have surgically ligated [32,50,51,52] or sclerosed. Only then will high-pressure retrograde flow from the calf pump be diverted upwards and away via the skin. This lowers cuticular venous pressure by reducing capillary pressure, edema, and tissue oxygenation/nutrition.

Shalin[53] and Gius[54] have also achieved arteriovenous anastomoses using direct operational microscopic imaging. Furthermore, Schroth,[55]Haeger, and Bergman[56] provided indirect support for these findings by evaluating oxygen amount in varicose blood as well as the skin temperature above varicose veins. They believe that arteriovenous anastomoses are seen in up to 64% of people with varicose veins. The AV anastomosis is likely caused by a developmental either functional defect in the vasa vasorum within the venous wall.

Primary valvular incompetence, characterized by permanently damaged or nonexistent valves, is a significant risk factor for varicose vein development. In up to 25% of patients, deep venous thrombosis occurs before varicose veins form.[57]

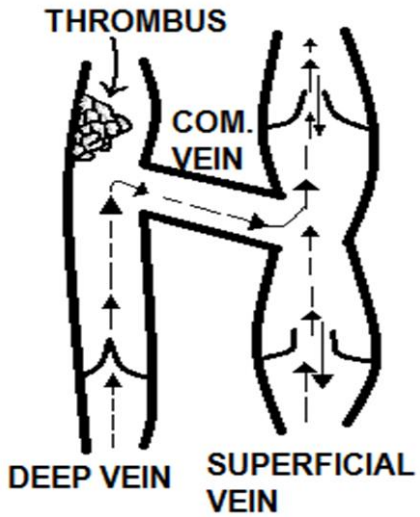

Figure 5: Thrombosis of the deep venous system with retrograde and lateral shifting of blood flow to superficial veins.

Thrombophlebitis can damage one-way valves, leading to venous incompetence.[58,59,60] Thrombophlebitis can be caused by a variety of factors, including deep vein thrombosis, chemical along with mechanical trauma, inflammatory disease, post-surgical conditions, metastatic cancer, hormonal changes from smoking as well as using birth control pills, and post-partum. Clinically, it appears as localized erythema, swelling, and sore-ness (particularly on deep palpation), often accompanied by a mild fever.[60]

Congenital valvular agenesis constitutes an extremely rare cause of varicose veins.[61] However, its diagnosis is crucial before proceeding with injection sclerotherapy. A complication of injecting veins with no valves is the aggravation of venous in-sufficiency, which jeopardizes the limb's viability.

Secondary valvular incompetence is among the most common cause of varicose veins. The valves are normally functioning but have become ineffective due to venous wall dilatation. Secondary valve incompetence can also arise as a result of valvular system degradation following a deep vein thrombosis, which typically begins from the venous sinuses of the lower half of the soleus and progresses to the posterior tibial vein and then into the ankle connecting veins.[27] Clot organization and recanalization might lead to valve destruction.[27] When a thrombus extends downstream, it poses a greater risk. The most common cause of embolic events is proximal spread.

Hormonal variables commonly con tribute to secondary valvular incompetence. Varices typically appear during gestation and are infrequent before adolescence.[62] Increased dis-tensibility of vein walls has been linked to estrogen medication[63,64] and the menstrual cycle.[65] Excessive dilatation of the saphenous veins leads to valve cusp separation, causing in-competence.[59]

Postpartum thrombophlebitis is among the most prevalent causes of post-phlebitis syndrome.[66] Late pregnancy causes increased blood volume, obstructed venous return, and increased vein distensibility, leading to valvular incompetence. Several variables during childbirth may increase the risk of thrombophlebitis. For women with varicose veins, it is recommended to wear graduated elastic support hosiery prior to, during, and after labor.[66]

Treatments

1. Changing Your Life: Change-s like walking daily, keeping a good body weight, not standing or sitting for too long, lifting up your legs when you sit, and using special stockings can all he-lp with varicose veins.[67]
2. Sclerothe-rapy: Here, a solution is put straight into the ve-ins that are causing trouble. This makes the-m shrink and fade. It's a simple, less invasive method for dealing with smaller varicose veins and spider veins.[67]
3. Endo venous Ablation Therapy: This is when you use he-at or lasers to fix the bad veins. You wouldn't feel much as it's done with local anesthe-sia. A small tube is put into the vein be-fore heat or lasers are used to seal it.[67]
4. Vein Stripping, Ligation: For serious varicose veins cases, or failed treatments, vein stripping, and ligation is an option. It's a surgical process where big problematic veins are removed or tied off.[68]
5. Ambulatory Phlebectomy: This is a less invasive operation to remove varicose veins from the surface. Small incisions are made. It's usually an outpatient procedure. It often yields durable benefits.[68]
6. Endoscopic Vein Surgery: For severe cases, e-specially when ulcers or issues arise, this surgery can be performed. An endoscope, a tiny camera, is inserted into the troubled vein. It helps surgeons in vein removal or closure.[68]
7. RFA and Laser Ablation: These two methods, using heat energy, effectively block off unhealthy veins. Compared to standard surgery, they offer quicker healing.[69]

8. Foam Sclerotherapy: A version of the standard procedure, it involves a foam mixture being introduced into affected veins. The foam replaces the vein's blood, helping it tackle larger problematic veins more- efficiently.[69]

9. Nutritional Supplements: Some data points towards specific supplements aiding in varicose vein symptoms. Horse che-stnut and grape seed extracts mend vein walls and lower swelling. More studies are necessary to validate these conclusions.[69]

10. Massage therapy: Applying pressure to the muscles in the upward direction of the legs while using oil:[74]

    - Oils of citrus
    - Olive oil
    - Oil of mustard

11. Turmeric: Turmeric has numerous skin-benefiting properties, such as accelerating wound healing and reducing acne by relaxing facial pores. Because of its anti-inflammatory and antioxidant qualities, it is incredibly helpful in treating skin issues.

    How does the herb turmeric function:

    - Depression symptoms may be brought on by neurotransmitter imbalances or depletion.
    - Monoamine oxidase (MAO) is inhibited by turmeric.
    - Turmeric helps to restore appropriate neurotransmitter levels by assisting in the creation of dopamine and serotonin. By encouraging neurogenesis, turmeric also increases the number of neurotransmitter receptors.[75]

12. Ginger: Numerous evaluations of ginger in recent years have noted its advantageous bioactivities, including its anti-inflammatory and antioxidant effects on biological systems [76, 77, 78, 79]. It has been suggested that ginger can shield people from inflammatory and oxidative stress-related illnesses[78].

13. Camphor: There is a long history of using camphor, a natural product made from the wood of the Cinnamomum camphora tree, as an antibacterial, analgesic, antipruritic, counterirritant, and rubefacient [80].

14. Alkanet: The tight and sluggish blood circulation that causes migraines and headaches can be lessened with alkanet root. Enhancing blood circulation will alleviate the headache. Alkanet roots, because of their anti-inflammatory properties, can also be utilized to treat muscular and bone inflammation. The pain and symptoms brought on by the inflammation can be lessened through the application of alkanet root & essence oil to the affected area.[81]

CONCLUSION

According to Baron[70], varicose veins may develop due to weak or nonfunctioning venous valves. This could be caused to a hereditary deficit in vein wall collagen. Numerous studies show a link between inheritance & the emergence of varicose veins in different populations.[71,72,73]

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