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Comparative Study between Foam Sclerotherapy versus Stab Avulsion in the Treatment of Perforator Incompetence of the Lower Limb

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I. INTRODUCTION

Varicose veins refers to a condition characterized by dilated, tortuous and elongated veins in the leg . It is characterized by reversal of blood through this defective valve. Varicosities of legs were described as early as 1550 BC and in the 1600s AD, the association between the varicose vein with trauma, childbearing age and " standing too much before kings " were proved[1]. The risk factors for varicose veins are female sex, pregnancy, prolonged standing and history of phlebitis[1].

Various treatment modalities for varicose veins are trendelenberg procedure, stripping of veins, stab avulsion, sclerotherapy and minimally invasive methods like radio frequency ablation , endovenous laser therapy available[1].

Varicose veins are classified as primary and secondary based on etiology. Primary varicose veins are due to genetic or developmental defects in the vein wall that causes defect in elasticity and valvular incompetence. Most common etiology for isolated superficial venous insufficiency is primary varicose veins. Secondary varicose veins are due to dysfunction of valves system caused by trauma, DVT, arteriovenous fistula, or nontraumatic proximal venous obstruction like pregnancy, pelvic tumor. Chronic venous stasis occurs When valves of the deep and perforating veins are disrupted[1].(Fig 1)

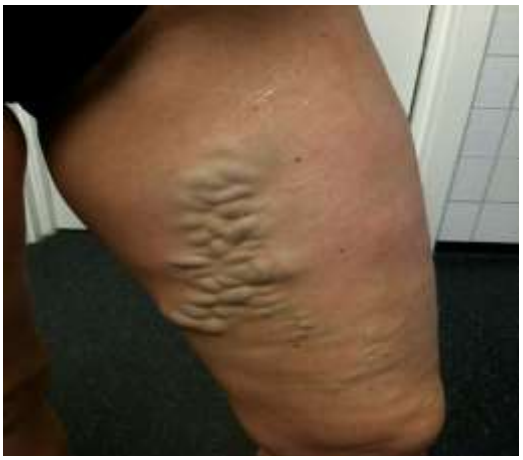

Fig 1 . Dilated veins in the medial thigh

This study was done to compare the results of two treatment modalities, namely foam sclerotherapy and stab avulsion as the treatment for perforator incompetence.

II. AIMS AND OBJECTIVE

The study is aimed at comparing the results of foam sclerotherapy versus stab avulsion as the treatment for perforator incompetence.

III. REVIEW OF LITERATURE

ANATOMY OF VEINS OF LOWER LIMB[1,2]

The venous system of lower limb can be divided into the superficial venous system which is located within the superficial tissues and the deep venous system, that lies beneath the deep fascia of leg. The superficial veins drain into the deep system, either at junctions or via fascial perforating veins and the deep veins then return blood to the right atrium of heart[3]. (Fig 2)

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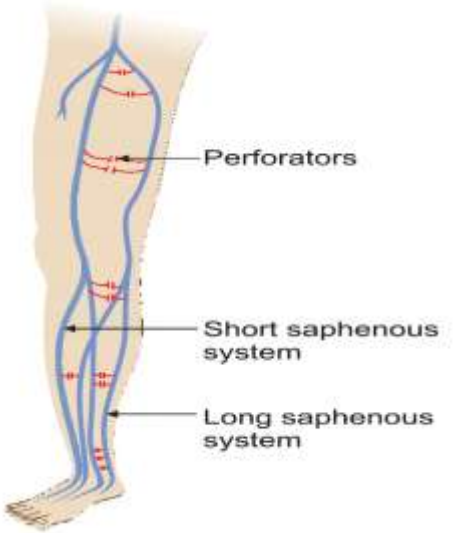

Fig 2. Anatomy of lower limb venous system

Veins are thin-walled, highly distensible, and collapsible. Their structure specifically supports the transport blood toward the heart and serve as a reservoir to prevent intravascular volume overload. The venous intima is composed of a nonthrombogenic endothelium with an underlying basement membrane and an elastic lamina. The endothelium produces endothelium-derived relaxing factors such as nitric oxide and prostacyclin, which aids in maintaining a non-thrombogenic surface by inhibiting the platelet aggregation and promotion of platelet disaggregation. Circumferential rings of elastic tissue and smooth muscles are located in the media of the vein allow the changes in venous caliber with minimal changes in venous pressure. The adventitia is most prominent in large veins and consists of collagen, elastic fibers, and fibroblasts. In the axial veins, unidirectional blood flow is achieved with multiple venous valves. The inferior vena cava (IVC), common iliac veins, portal venous system, and cranial sinuses are valveless. In the axial veins, valves are more numerous distally in the extremities than proximally. Each valve consists of two thin cusps of a fine connective tissue skeleton covered by endothelium. Venous valves shuts in response to cephaladto- caudal blood flow at a velocity of at least 30 cm/s.2[4].

DEEP VENOUS SYSTEM

The deep veins of the lower limb comprises of three pairs of venae commitantes and three crural arteries (anterior and posterior tibial and peroneal arteries). These six vessels communicates with each other and meet in the popliteal fossa to form the popliteal vein and unites with the soleal and gastrocnemius veins. The popliteal vein enters the subsartorial canal as superficial femoral vein, which receives the deep (profunda) femoral vein (or veins) in the femoral triangle to become the common femoral vein, which forms external iliac vein as it passes behind the inguinal ligament. The internal iliac vein joins with the external iliac vein in the pelvis to form the common iliac vein. The left common iliac vein passes behind the right common iliac artery to join the right common iliac vein on the right side of the abdominal aorta to form the inferior vena cava[3]. (Fig. 3)

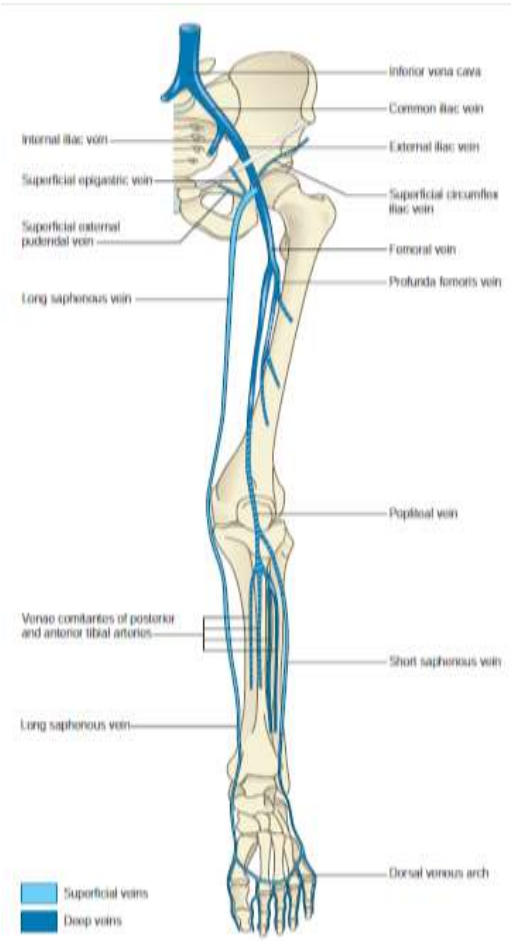

Fig .3 Anatomy of Deep venous system

SUPERFICIAL VENOUS SYSTEM

The long saphenous vein (great saphenous vein) , the longest vein in the body, is the continuation of the medial marginal vein of the foot, and ends in the femoral vein. It ascends immediately anterior to the tibial malleolus, crosses the distal third of the medial surface of the tibia obliquely in an anteroposterior direction to reach its medial border, and then ascends a little behind the knee. Proximally, it is posteromedial to the medial

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tibial and femoral condyles (lying the breadth of the subject's hand posterior to the medial edge of the patella), and then ascends the medial aspect of the thigh. It passes through the saphenous opening and finally opens into the femoral vein. The 'centre' of the opening is often said to be 2.5–3.5 cm inferolateral to the pubic tubercle. However, the saphenous opening varies in size and disposition so that this 'centre' is not a reliable surface marking for the saphenofemoral junction[2].

In its course through the thigh, the long saphenous vein is accompanied by the medial branches of the anterior cutaneous branches of the femoral nerve. At the knee, the saphenous branch of the descending genicular artery (the saphenous artery) and, in the leg and foot, the saphenous nerve all lie anterior to the vein. The vein is often duplicated. It has 10–20 valves, which are more in the leg than in the thigh. One is present just before the vein pierces the cribriform fascia, another at its junction with the femoral vein. In almost its entire extent the vein lies in subcutaneous tissue, but it has many connections with the deep veins, especially in the leg[2]. (Fig.4)

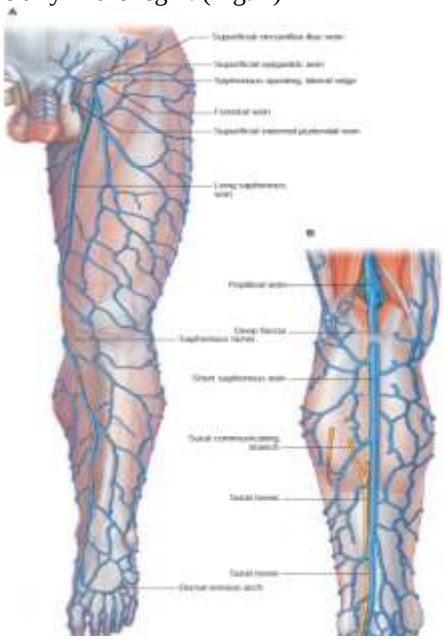

Fig.4 Anatomy of Superficial Venous system

TRIBUTARIES OF GREAT SAPHENOUS VEIN

At the ankle, the long saphenous vein drains the sole by medial marginal veins. In the leg, it often connects with the short saphenous vein and with deep veins via perforating veins. Distal to the knee, it receives three large tributaries from the front of the leg, the tibio - malleolar region (connecting with some of the 'perforating' veins) and the calf (communicating with the short saphenous vein). The tributary

draining the tibio malleolar region is formed distally from a fine network of delicate veins over the medial malleolus, and then ascends the medial aspect of the calf as the posterior arch vein (Dodd and Cockett 1976). This vein was first illustrated by Leonardo da Vinci, whose name is sometimes given to it. It connects with posterior tibial venae comitantes by a series of perforating (communicating) veins. There are usually three, equally spaced between the medial malleolus and the mid-calf. More than three such perforators are uncommon, and an arch vein perforator above mid-calf is only very rarely found[2].

Above the posterior crural arch vein, perforating veins join the long saphenous vein or one of its main tributaries at two main sites. The first is at a level in the upper, the tibial tubercle perforator; the second is in the lower/intermediate third of the thigh, where it perforates the deep fascia roof of the subsartorial canal to join the femoral vein[2].

In the thigh, the long saphenous vein receives many tributaries. Some open independently, while others converge to form large named channels that frequently pass towards the basal half of the femoral triangle before joining the long saphenous near its termination. These may be grouped as follows: one or more large posteromedial tributaries,

one or more large anterolateral tributaries, and four or more periinguinal veins. The posteromedial vein of the thigh, large and sometimes double, drains a large superficial region indicated by its name; it has (as have the other tributaries) radiological and surgical significance. One of its lower radicles is often continuous with the short saphenous vein. The posteromedial vein is sometimes named the accessory saphenous vein, though some restrict the term accessory to a lower (more distal) posteromedial tributary when two (or more) are present. Another large vessel, the anterolateral vein of the thigh (anterior femoral cutaneous vein), usually commences from an anterior network of veins in the distal thigh and crosses the apex and distal half of the femoral triangle to reach the long saphenous vein. As the latter traverses the saphenous opening, it is joined by the superficial epigastric, superficial circumflex iliac and superficial external pudendal veins. Their mode of union varies. Superficial epigastric and circumflex iliac veins drain the inferior abdominal wall, the latter also receiving tributaries from the proximolateral region of the thigh. The superficial epigastric or the femoral vein may connect with the

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lateral thoracic veins by means of a thoracoepigastric vein that runs superficially on the anterolateral aspect of the trunk. This vein connects the inferior and superior caval areas of drainage and may be dilated and visible in cases of inferior caval obstruction. Superficial external pudendal veins drain part of the scrotum/labia; one is joined by the superficial dorsal vein of the penis/ clitoris. The deep external pudendal veins join the long saphenous vein at the saphenous opening[2].

SHORT SAPHENOUS VEIN

The short saphenous vein (small saphenous vein) begins posterior to the lateral malleolus as a continuation of the lateral marginal vein . In the lower third of the calf, it ascends lateral to the calcaneal tendon, lying on the deep fascia and covered only by subcutaneous tissue and skin. Inclining medially to reach the midline of the calf, it penetrates the deep fascia, within which it ascends on gastrocnemius, only emerging between the deep fascia and gastrocnemius gradually at about the junction of the middle and proximal thirds of the calf (usually well below the lower limit of the popliteal fossa). Continuing its ascent, it passes between the heads of gastrocnemius and proceeds to its termination in the popliteal vein, 3–7.5 cm above the knee joint[2].

TRIBUTARIES OF SHORT SAPHENOUS VEIN

The short saphenous vein connects with deep veins on the dorsum of the foot, receives many cutaneous tributaries in the leg, and sends several communicating branches proximally and medially to join the long saphenous vein. Sometimes a communicating branch ascends medially to the accessory saphenous vein: this may be the main continuation of the short saphenous vein. In the leg, the short saphenous vein lies near the sural nerve and contains 7–13 valves, with one near its termination. Its mode of termination is variable: it may join the long saphenous vein in the proximal thigh or it may bifurcate, one branch joining the long saphenous vein and the other joining the popliteal or deep posterior femoral veins. Sometimes it drains distal to the knee in the long saphenous or sural veins[2].

SAPHENOUS NERVE ANATOMY

The saphenous nerve is the largest and longest cutaneous branch of the femoral nerve and the longest nerve in the body .

It descends lateral to the femoral artery in the femoral triangle and enters the adductor canal, where it crosses anterior to the artery to lie medial to it. At the distal end of the canal, it leaves the artery and emerges through the aponeurotic covering with the saphenous branch of the descending genicular artery. As it leaves the adductor canal, it gives off an infrapatellar branch that contributes to the peripatellar plexus and then pierces the fascia lata between the tendons of Sartorius and gracilis, becoming subcutaneous to supply the skin anterior to the patella. It descends along the medial border of the tibia with the long saphenous vein and divides distally into a branch that continues along the tibia to the ankle and a branch that passes anterior to the ankle to supply the skin on the medial side of the foot, often as far as the first metatarsophalangeal joint. The saphenous nerve connects with the medial branch of the superficial fibular nerve. Near the mid-thigh, it provides a branch to the subsartorial plexus. The nerve may become
entrapped as it leaves the adductor canal[2].

PERFORATOR SYSTEM

As their name suggests, PVs perforate the deep fascia of the leg, which separates the superficial and deep compartments; they can be classified as direct or indirect. Direct perforators connect the superficial to the deep venous systems, whereas indirect perforators join the venous sinuses of the calf muscles. Furthermore, PVs connect to each other via communicating veins above and underneath the deep muscle fascia. The majority of PVs are accompanied by perforating arteries and nerves that provide blood supply and innervation to the skin. Within the fascial orifice the artery is usually located proximal to the vein, but the topography of the subfascial and suprafascial segments of perforator arteries varies significantly.

Duplex ultrasound is used to identify the vessels and perforators especially sclerotherapy is being considered as a treatment option[2].

The International Interdisciplinary Consensus Committee on Venous Anatomical Terminology recommends classifying PVs into six groups according to the segment of the lower extremity in which they are found: Perforators of the foot (venae perforantes pedis)
Perforators of the ankle (venae perforantes tarsalis)
Perforators of the leg (venae perforantes cruris)
Perforators of the knee (venae perforantes genus)
Perforators of the thigh (venae perforantes femoris)
Perforators of the gluteal muscles (venae perforantes glutealis)

The most important perforators are the direct medial calf perforators, which cross the superficial posterior compartment. The posterior tibial PVs originate from the posterior accessory

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saphenous vein of the calf (posterior arch vein in the old terminology). The most distal posterior tibial perforators are located behind the medial malleolus, whereas the middle and upper posterior tibial perforators are located more proximally in the calf (at 7–9 cm and 10–12 cm from the medial malleolus, respectively) and about 1 inch medial to the tibia; these PVs connect the posterior arch vein to the posterior tibial veins (Cockett perforators). More proximal direct PVs are the paratibial direct perforators or "24-cm perforators," located closer to the tibia and 18 to 22 cm from the medial malleolus, as evident in anatomic cadaveric studies. Another group of medial calf perforators, found just below the knee, is known as Boyd's perforators. Boyd's perforators connect the GSV and its tributaries to the tibial or popliteal veins. Clinical importance are the posterolateral or peroneal perforators, which connects the short saphenous vein to the peroneal veins. Among these, the most important are Bassi's perforator, located at 5 to 7 cm from the lateral aspect of the ankle, and the "12-cm perforator," located at 12 to 14 cm. Thigh perforators are poorly developed than calf PVs. The main instances are the Dodd perforators and the Hunterian perforators, which are located in the medial aspect of the thigh and connect the GSV to the popliteal or femoral veins. Other PVs connect the superficial system to the profunda femoris vein[6]. (Fig.5)

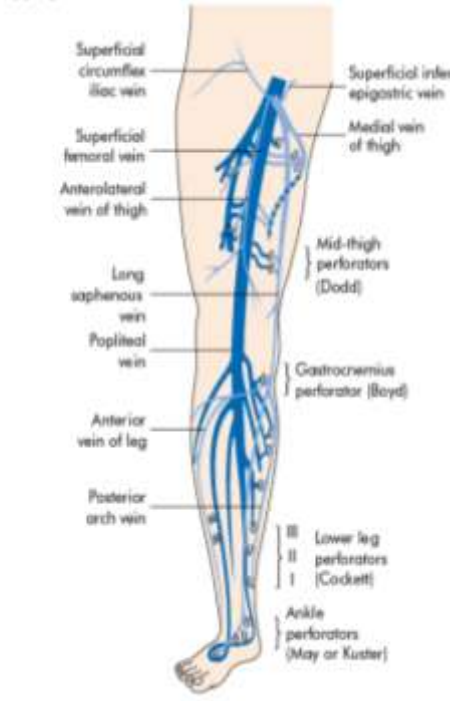

Fig .5 Anatomy of Perforators in lower limb

NORMAL VENOUS HISTOLOGY AND FUNCTION

The venous wall is composed of three layers, the intima, media, and adventitia. Vein walls have less smooth muscle and elastin than their arteries. The venous intima has an endothelial cell layer resting on a basement membrane. The media is composed of smooth muscle cells and elastin connective tissue. The adventitia of the venous wall contains adrenergic fibers, particularly in the cutaneous veins. Central sympathetic discharge and brainstem thermoregulatory centers can alter venous tone, such as temperature changes, pain, emotional stimuli, and volume changes[5].

The histologic features of veins vary, depending on the caliber of the veins. The venules, the smallest veins, range from 0.1 to 1 mm and made up of mostly smooth muscle cells, whereas the larger veins contain relatively few smooth muscle cells. These larger caliber veins have limited contractile capacity in comparison to the thicker walled great saphenous vein. The venous valves prevent retrograde flow; it is their failure or valvular incompetence that leads to reflux and its associated symptoms. Venous valves are most prevalent in the distal lower extremity, whereas as one proceeds proximally, the number of valves decreases to the point that no valves are present in the superior vena cava and inferior venacava (IVC)[5].

The calf muscles increases venous return by functioning as a pump. In the supine state, the resting venous pressure in the foot is the sum of the residual kinetic energy minus the resistance in the arterioles and precapillary sphincters. Thus, a pressure gradient is generated in the right atrium of approximately 10 to 12 mm Hg. In the upright position, the resting venous pressure of the foot is a reflection of the hydrostatic pressure from the upright column of blood extending from the right atrium to the foot[5].

The return of the blood to the heart from the lower extremity is helped by the muscle pump function of the calf, a mechanism whereby the calf muscle, functioning as a bellows during exercise, compresses the gastrocnemius and soleal sinuses and propels the blood toward the heart. The normally functioning valves in the venous system prevent retrograde flow; when one or more of these valves become incompetent, symptoms of venous insufficiency can develop. During calf muscle contraction, the venous pressure of the foot and ankle drops dramatically. The pressures increases in the muscle compartments during exercise range from 150 to 200 mm Hg, and when there is failure

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of perforating veins, these high pressures are transmitted to the superficial system[5].

PATHOPHYSIOLOGY OF VARICOSE VEINS

The pathophysiology of varicose vein development is probably related to changes in the vein wall (dysfunctional smooth muscle cell proliferation, collagen deposition, decreased elastin content and increased matrix metalloproteinases) leading to venous dilatation and secondary valvular incompetence rather than to a primary valvular defect, which occurs in a small group of patients who have total lack of venous valves. Secondary varicose veins can develop in patients with post-thrombotic limbs and in patients with congenital abnormalities such as the Klippel–Trenaunay syndrome or multiple arteriovenous fistulae[5].

Classification

The CEAP (clinical – etiology – anatomy – pathophysiology) classification for chronic venous disorders is widely utilised.

Clinical classification

- C0: no signs of venous disease
- C1: telangectasia or reticular veins
- C2: varicose veins
- C3: oedema
- C4a: pigmentation or eczema
- C4b: lipodermatosclerosis or atrophie blanche
- C5: healed venous ulcer
- C6: active venous ulcer

Each clinical class is further characterised by a subscript depending upon whether the patient is symptomatic (S) or asymptomatic (A) e.g. C2S.

Etiologic classification

- Ec: congenital
- Ep: primary
- Es: secondary (post-thrombotic)
- En: no venous cause identified

Anatomical classification

- As: superficial veins
- Ap: perforator veins
- Ad: deep veins
- An: no venous location identified

Pathophysiological classification

- Pr: reflux
- Po: obstruction
- Pr,o: reflux and obstruction
- Pn: no venous pathophysiology identifiable[3]

Epidemiology

The prevalence of visible varicose veins is 25–30 per cent in women and 15 per cent in men. Factors affecting prevalence include:

- Gender: the vast majority of studies report a higher prevalence in women than men, the Edinburgh Vein Study being the main exception
- Age: the prevalence of varicose veins increases with age. In the Edinburgh Vein Study, the prevalence of trunk varicosities in the age groups 18–24 years, 25–34 years, 35–44 years, 45–57 years and 55–64 years was 11.5, 14.6, 28.8, 41.9 and 55.7 percent, respectively;
- Ethnicity: seem to influence the prevalence of varicose veins;
- Body mass and height: increasing body mass index and height may be associated with a higher prevalence of varicose veins;
- Pregnancy: appears to increase the risk of varicose veins;
- Family history: evidence supports familial susceptibility to varicose veins;
- Occupation and lifestyle factors: there is inconclusive evidence regarding increased prevalence of varicose veins in smokers, patients who suffer constipation and occupations which involve prolonged standing[3].

VENOUS INSUFFICIENCY

There are three categories of venous insufficiency—congenital, primary, and secondary. Congenital venous insufficiency comprises predominantly anatomic variants that are present at birth. Examples of congenital venous anomalies include venous ectasias, absence of venous valves, and syndromes such as Klippel-Trénaunay syndrome. Primary venous insufficiency is an acquired idiopathic entity. This is the largest clinical category and represents most of the superficial venous insufficiency encountered in the office. Secondary venous insufficiency arises from a postthrombotic or obstructive state and is caused by a deep venous thrombus or primary chronic obstructive process[5].

Primary Venous Insufficiency

There are three main anatomic categories of primary venous insufficiency—telangiectasias, reticular veins, and varicose veins. Telangiectasias, reticular varicosities, and varicose veins are similar but varies in caliber. Telangiectasias are very small intradermal venules that are too small to demonstrate reflux. They measure less than 3 mm. They are idiopathic in nature. However, leg telangiectasias of multiple causes may be a

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manifestation of a systemic disease. Some of these disorders include autoimmune diseases (such as lupus erythematosus and dermatomyositis), exogenous causes, and xeroderma pigmentosum. Reticular veins are vein branches that enter the tributaries of the main axial, perforating, or deep veins. The axial veins, the great and small saphenous veins, represent the largest veins of the superficial venous system[5].

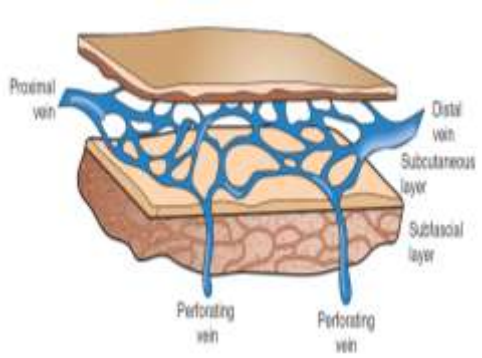

Fig .6 Dilatation of veins causing increase pressure in perforators

Pathology

The precise pathophysiologic mechanism of venous insufficiency has yet to be elucidated. This describes some of the areas in which research has started to reveal its multifactorial pathogenesis[5].

Mechanical abnormalities.

Anatomic differences in the location of the superficial veins of the lower extremities may contribute to the pathogenesis. Primary venous insufficiency may involve both the axial veins (great and small saphenous), either vein, or neither vein. Perforating veins may be the sole source of venous

pathophysiologic changes, perhaps because the great saphenous vein is supported by a well-developed medial fibromuscular layer and fibrous connective tissue that bind it to the deep fascia. In contrast, tributaries to the small saphenous vein are less supported in the subcutaneous fat and are superficial to the membranous layer of superficial fascia. These tributaries also contain less muscle mass in their walls. Thus, these veins, and not the main trunk, may become selectively varicose. For example, failure of a valve protecting a tributary vein from the pressures of the small saphenous vein allows a cluster of varicosities to develop.

Pressure studies have shown that there are two causes of venous hypertension. The first is gravitational and is a result of venous blood coursing in a distal direction down the linear axial venous segments. This is called as hydrostatic pressure and is the weight of the blood column from the right atrium. The highest pressure generated by this mechanism is seen at the ankle and foot, where measurements are expressed in centimeters of water or millimeters of mercury. The second source of venous hypertension is dynamic, it is due to the force of muscle contraction, usually contained within the compartments of the leg. If a perforating vein fails, high pressures (range, 150 to 200 mm Hg) developed within the muscular compartments during exercise are transmitted directly to the superficial venous system. The sudden pressure transmitted causes dilation and lengthening of the superficial veins. This can result in Progressive distal valvular incompetence. If proximal valves such as the saphenofemoral valve become incompetent, systolic muscular contraction is supplemented by the weight of the static column of blood from the heart. Blood flowing proximally through the femoral vein spills into the saphenous vein and flows distally. As it refluxes distally through progressively incompetent valves, it is returned through perforating veins to the deep veins. Here, it is conveyed once again to the femoral veins, only to be recycled distally.

Regardless of the precise source of the elevated hydrostatic pressure, the ultimate end result is increased ambulatory hypertension. The inflammatory processes that occur throughout the venous circulation have been demonstrated within the vein wall as well as within the vein valves. It is unclear as to which abnormality occurs first, that is, whether the vein wall becomes distended from increased pressure and then causes vein wall abnormalities, or vice versa. The resulting increased ambulatory venous pressure affects the endothelium as well as the venous microcirculation[5].

This activation is again caused by changes in shear stress and mechanical stress of the vein wall and vein valves.Altered shear stress causes the endothelial cells to release a varietyof agents, including chemokines and inflammatory molecules,which precipitates the inflammatory cascade. In particular,cytokines and metalloproteinases play a prominent role in themechanical and inflammatory process of venous hypertension. The inflammatory process involves many different pathways that result in elevations of inflammatory modulators and cytokines, growth factors, and metalloproteinase activity. Fundamental defects in the strength and characteristics of the venous wall have been

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identified. Varicose vein walls demonstrate decreased amounts of elastin and collagen, suggesting a contributing role toward venous pathophysiology[5].

Risk Factors

Risk factors for the development of varicose veins include advancing age, female gender, multiparity, heredity, and history of trauma to the extremity. Additional risk factors include obesity and a positive family history. Advancing age appears to be the most significant risk factor. Venous function is undoubtedly influenced by hormonal changes. In particular, progesterone liberated by the corpus luteum stabilizes the uterus by causing the relaxation of smooth muscle fibers. This directly influences venous function. The result is passive venous dilation, which in many cases causes valvular dysfunction. Although progesterone is implicated in the first appearance of varicosities in pregnancy, estrogen also has profound effects. It produces the relaxation of smooth
muscle and a softening of collagen fibers. Furthermore, the estrogen-to-progesterone ratio influences venous distensibility.

This ratio may explain the predominance of venous insufficiency symptoms on the first day of a menstrual period, when a profound shift occurs from the progesterone phase of the menstrual cycle to the estrogen phase. Autosomal dominant penetrance has been identified as the underlying genetic risk factor for subsequent development of varicose veins[5].

Symptoms

Varicose veins frequently cause symptoms, the most common being aching or heaviness, which typically increases throughout the day or with prolonged standing and is relieved by elevation or compression hosiery. Other less common symptoms include ankle swelling and itching while complications (bleeding, superficial thrombophlebitis, eczema,

Lipodermatosclerosis and ulceration) represent important indications for investigation and intervention. The Edinburgh Vein Study failed to show any evidence that the extent of valvular incompetence was related to the severity of symptoms[3].

This syndrome is termed venous claudication and is a clinical manifestation of venous outflow obstruction, secondary venous insufficiency. Predominant causes of venous claudication includes prior deep venous thrombosis (DVT) and May Thurner syndrome. Multiparous female patients in their childbearing years may present with a group of symptoms that involve varicosities of the leg in conjunction with chronic pelvic pain. Additional symptoms include a feeling of bladder fullness with standing, dyspareunia, and chronic pelvic pain. This clinical picture is similar to pelvic congestion syndrome. As the differential diagnosis for pelvic pain is extensive, the diagnosis of pelvic venous congestion tends to be one of exclusion; diagnostic investigation to confirm includes magnetic resonance venous imaging (MRVI) of the pelvis and conventional pelvic venography, which can be both diagnostic and therapeutic[5].

SIGNS

The presence of tortuous dilated subcutaneous veins are usually clinically obvious. These are confined to the long and lesser saphenous systems in approximately 60 and 20 per cent of cases, respectively. The distribution of varicosities may indicate which superficial system is defective; medial thigh and calf varicosities suggest long saphenous incompetence , posterolateral calf varicosities are suggestive of short saphenous incompetence, whereas anterolateral thigh and calf varicosities may indicate isolated incompetence of the proximal anterolateral long saphenous tributary . Percussion over the varices may elicit an impulse tap by the fingers placed over the dilated trunk.

Other signs commonly found include:

• Telangectasia, which are dilated intradermal venules <1 mm in diameter. These may be mild or severe. Synonyms include spider veins, thread veins and hyphen webs.

• Reticular veins are dilated, subdermal veins, 1–3 mm in diameter. The presence of telangectasia and reticular veins are of dubious significance, are not necessarily associated with major varicose veins and are purely a cosmetic problem.

• In saphena varix , there is a large groin varicosity which presents as a (usually painless) swelling, emergent when standing and disappearing when recumbent. Gentle palpation over the varix during coughing may elicit a thrill.

• Atrophie blanche are localised white atrophic skin frequently surrounded by dilated capillaries and hyperpigmentation, usually seen around the ankle

• Corona phlebectasia are fan-shaped patterns of small intradermal veins on the medial or lateral aspects of the ankle or foot. Synonyms include malleolar or ankle flares

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• Pigmentation is usually a brown discolouration (because of haemosiderin deposition) of the skin, most
frequently affecting the gaiter area, and may be associated with phlebitis and ulceration.
• Eczema ; this is an erythematous dermatitis which may progress to blistering, weeping or scaling eruption of the skin, not to be confused with contact dermatitis
• Dependent pitting oedema as a result of increase in volume of fluid in skin and subcutaneous tissue characteristically increases throughout the day, and is relieved by elevation and compression hosiery/bandaging. The oedema is usually confined to the ankle area but may extend to the foot and rest of the leg.
• Lipodermatosclerosis is a localised chronic inflammation and fibrosis of the skin and subcutaneous tissues of the leg, a sign of severe chronic venous disease. (Fig.7)

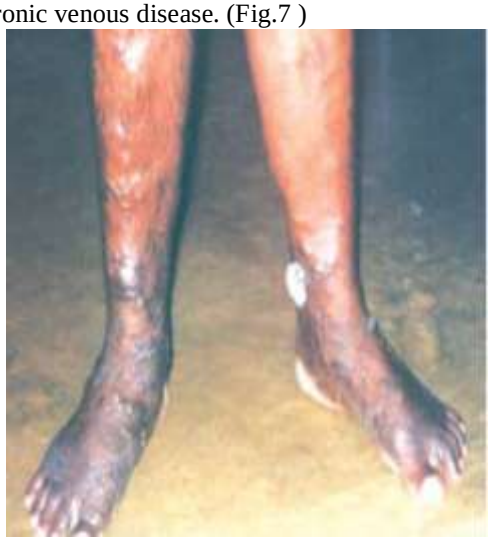

Fig .7 Bilateral varicose veins with skin changes

• Ulceration : a full thickness epidermal defect, most frequently affecting the gaiter area[3]. (Fig .8)

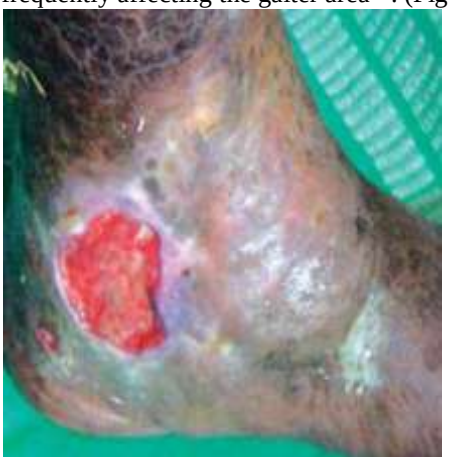

Fig .8 Venous ulcer

CLINICAL EXAMINATION

1. Visible dilated veins in the leg with pain, distress, nocturnal cramps, feeling of heaviness, pruritus.
2. Pedal oedema, pigmentation, dermatitis, ulceration, tenderness, restricted ankle joint movement.
3. Bleeding, thickening of tibia occurs due to periostitis.
4. Positive cough impulse at the sapheno-femoral junction. Saphena varix – a large varicosity in the groin, which becomes visible and prominent on coughing
5. **Brodie-Trendelenburg test**: Vein is emptied by Elevating and milking the limb and a tourniquet is tied just below the sapheno-femoral junction (or using thumb, sapheno-femoral junction is occluded). Patient is asked to stand quickly. When tourniquet or thumb is released, rapid filling from above signifies saphenofemoral incompetence. This is Trendelenburg test I. In Trendelenburg test II, after standing tourniquet is not released. Filling of blood from below upwards rapidly can be observed within 30-60 seconds. It signifies perforator incompetence
6. **Perthe's test:** The affected lower limb is wrapped with elastic bandage and the patient is asked to walk around and exercise. Development of severe cramp like pain in the calf signifies DVT.
7. **Modified Perthe's test**: Tourniquet is tied just below the sapheno-femoral junction without emptying the
vein. Patient is allowed to have a brisk walk which precipitates bursting pain in the calf and also makes superficial veins more prominent. It is used to detect DVT.
DVT is contraindicated for any surgical intervention of superficial varicose veins. It is also contraindication for sclerosant therapy.

8. **Three tourniquet test**: To find out the site of incompetent perforator, three tourniquets are tied after emptying the vein.
1. at sapheno-femoral junction
2. above knee level
3. another below knee level.
Patient is asked to stand and looked for filling of veins and site of filling. Then tourniquets are released from below upwards, again to see for incompetent perforators.
9. **Schwartz test:** In standing position, when lower part of the long saphenous vein in leg is tapped, impulse is felt at the saphenous junction or at the upper end of the visible part of the vein. It signifies

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continuous column of blood due to valvular incompetence. (Fig .9)

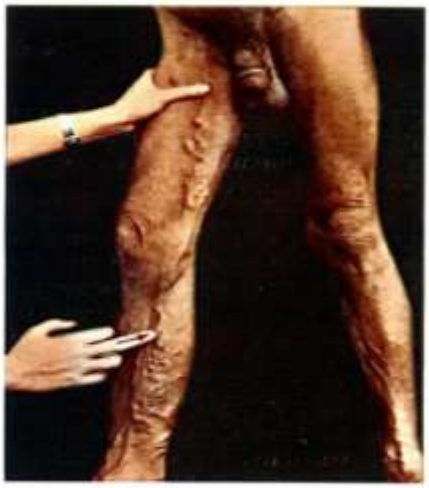

Fig.9 Swartz Test

10. **Pratt's test**: Esmarch bandage is applied to the leg from below upwards followed by a tourniquet at sapheno-femoral junction. After that the bandage is released keeping the tourniquet in the same position to see the "blow outs" as perforators.
11. **Morrissey's cough impulse test:** The varicose veins are emptied. The leg is elevated and then the patient is
asked to cough. If there is sapheno- femoral incompetence, expansile impulse is felt at saphenous opening.
It is a venous thrill due to vibration caused by turbulent backflow.
12. **Fegan's test**: On standing, the site where the perforators enter the deep fascia bulges and this is marked. Then on lying down, button like depression (crescent like) in the deep fascia is felt at the marked out points which confirms the perforator site.
13. **Ian-Aird test**: On standing, proximal segment of long saphenous vein is emptied with two fingers. Pressure
from proximal finger is released to see the rapid filling from above which confirms sapheno-femoral incompetence.
14. Examination of the abdomen has to be done to look for pelvic tumours, lymph nodes, which may compress over the veins to cause varicosity[7]

Grading of clinical signs
0— No visible or palpable signs of venous disease
1— Telangiectases, reticular veins or malleolar flare
2— Varicose veins
3— Oedema without skin changes
4— Skin changes ascribed to venous diseases (pigmentation, venous eczema, lipodermatosclerosis)
5— Skin changes as above with healed ulceration
6— Skin changes as above with active ulceration[7]

INVESTIGATION
1. **Venous Doppler:** With the patient standing, the doppler probe is placed at sapheno-femoral junction and later wherever required. Basically by hearing the changes in sound, venous flow, venous patency, venous reflux can be very well-identified[7].
2. Today, **Duplex imaging** is the first and best modality to assess for the normal function and presence of venous insufficiency of the lower extremities. Duplex technology more precisely shows which veins are refluxing by imaging the superficial and deep veins. The duplex examination is commonly done with the patient supine, but this yields an erroneous evaluation of reflux. In the supine position, even when no flow is present, the valves remain open. Valve closure requires a reversal of flow with a pressure gradient that is higher proximally. Thus, the duplex examination needs to be done with the patient standing or in the markedly trunk-elevated position. There are many advantages of ultrasound imaging. The ultrasound examination is noninvasive, requires no contrast material.
Drawbacks to the investigations include interobserver variability and limitations in imaging in patients with an elevated body mass index and extensive dressings. Imaging is obtained with a 7.5- or 10-MHz probe; the pulsed Doppler consists of a 3.0-MHz probe. The examination begins with the probe placed longitudinally on the groin. First, all of the deep veins are examined. Next, the superficial veins are evaluated.
There are four basic components of the examination that should be included to complete a comprehensive venous evaluation of the lower extremity veins: compressibility, venous flow, augmentation after reflux, and visibility. Reflux can be demonstrated with the patient performing a Valsalva maneuver or by manual compression and release of the extremity distal to the point of the examination. A Valsalva maneuver is performed for the proximal extremity, that is, the thigh and groin, whereas compression is used for the calf. Reflux times of 500 milliseconds or longer are considered significant Perforator veins can be visualized well with the duplex examination. Significant perforator reflux is defined as a diameter of more than 3.5 mm and a reflux time of 500 milliseconds or longer. Demonstration on duplex images of to-and-fro flow, with the

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presence of dilated segments, constitutes findings compatible with a refluxing perforator. In addition, Doppler studies can provide the clinician with information about the deep system. Widespread use of duplex scanning has allowed a comparison of findings between standard clinical examinations and duplex Doppler studies[5]. (Fig .9)

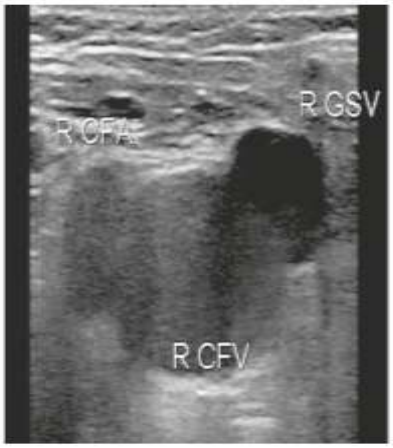

Fig .10 Mickey Mouse sign

3 **Phlebography and venography.** In general, phlebography is unnecessary in the diagnosis and treatment of primary venous insufficiency. In cases of secondary CVI, phlebography has specific usefulness. Ascending phlebography is performed by injection of contrast material into a superficial pedal vein after a tourniquet is applied at the ankle to prevent flow into the superficial venous system. Observation of flow defines anatomy and regions of thrombus or obstruction. Therefore, ascending phlebology differentiates primary from secondary venous insufficiency. Descending phlebography is performed with retrograde injection of contrast material into the deep venous system at the groin or popliteal fossa (femoral vein or popliteal vein). This diagnostic modality identifies specific valvular incompetence suspected on B-mode scanning and clinical examination. These studies are performed only as preoperative adjuncts when deep venous reconstruction is being planned[7].

4. **Magnetic resonance venous imaging.** MRVI is a diagnostic
imaging modality reserved for evaluation of the abdominal and
pelvic venous vasculature. MRVI, unlike venography, is noninvasive and does not require intravenous (IV) administration of contrast material. Studies have documented similar rates of specificity and sensitivity compared with venography. MRVI is used to evaluate pelvic venous outflow obstruction, providing information from the IVC through the iliac venous system. Furthermore, it is an excellent test to evaluate for pelvic congestion syndrome. In some institutions, the computed tomography scan has applications
that can be used similar to the MRVI scan[5].

5. **Ambulatory venous pressure (AVP):** It is an invasive method. Needle inserted into dorsal vein of foot is connected to transducer to get its pressure which is equivalent to pressure in the deep veins of the calf. Ten tiptoe maneuvers are done by the patient. With initial rise in pressure, pressure decreases and eventually stabilises with a balance. Pressure now is called as ambulatory venous pressure (AVP). After stopping exercise, veins are allowed to refill with return of pressure to baseline. Time required for pressure to return to 90% of baseline is called as venous refilling time (VRT). Raise in AVP signifies venous hypertension. Patients with AVP more than 80 mmHg has got 80% chances of venous ulcer formation

6 **U/S abdomen**, peripheral smear, platelet count, other relevant investigations are done depending on the cause of the varicose veins 7 If venous ulcer is present, then the **discharge is**
collected for culture and sensitivity, biopsy from ulcer
edge is taken to rule out Marjolin's ulcer.

8 **Plain X-ray** of the part is taken to look for periostitis

9 **Arm-Foot venous pressure:** Foot pressure is not more than 4 mmHg above the arm pressure

10 **Varicography**: Here non-ionic, iso-osmolar, nonthrombogenic contrast is injected directly into the variceal vein to get a detailed anatomical mapping of the varicose veins. It is used in recurrent varicose veins[7].

TREATMENT

NON OPERATIVE

Symptoms of primary venous insufficiency are manifestations of valvular incompetence. Therefore, the objective of conservative management is to improve the symptoms caused by venous hypertension. The first measure is external compression using elastic hose, 20 to 30 mm Hg, to be worn during the daytime hours. Although the exact mechanism whereby compression is of benefit is not entirely known, a number of physiologic alterations have been observed with compression. These include reduction in ambulatory venous pressure, improvement in skin microcirculation, and increase in subcutaneous pressure, which counters transcapillary fluid leakage. Patients are instructed to wear the hose during the day only, but to put the stockings on as soon as the day begins; swelling with standing will make stocking placement difficult. Care must be

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taken with patients who have concomitant arterial insufficiency because the compression stockings may exacerbate arterial outflow to the foot. Therefore, these patients require less compression—in some cases, no compression whatsoever—depending on the severity of the arterial disease. In general, an ankle-brachial index of less than 0.7 contraindicates the use of 20 to 30 mm Hg compression stockings[5].

The second part of conservative therapy is to practice lower extremity elevation for two brief periods during the day, instructing the patient that the feet must be above the level of the heart, or "toes above the nose." With good compliance, these measures may ameliorate symptoms so that patients may not require further intervention. Third, patients are encouraged to participate in activities that activate the calf musculovenous pump, thereby decreasing ambulatory venous hypertension. These activities include frequent ambulation and exercise. Patients who exhibit venous stasis ulceration will require local wound care . A triple-layer compression dressing, with a zinc oxide paste gauze wrap in contact with the skin, is used most commonly, from the base of the toes to the anterior tibial tubercle with snug graded compression. This is an example of what is generally known as an Unna boot. A 15-year review of 998 patients with one or more venous ulcers treated with a similar compression bandage demonstrated that 73% of the ulcers healed in patients who returned for care . The median time to healing for individual ulcers was 9 weeks. In general, snug, graded pressure, triple-layer compression dressings result in more rapid healing than with compression stockings alone. For most patients, well-applied, sustained compression therapy offers the most cost-effective and efficacious therapy in the healing of venous ulcers. After healing, most cases of CVI are controlled with elastic compression stockings to be worn during waking hours. On occasion, older patients and those with arthritic conditions cannot apply the compression stocking required, and control must be maintained by triple-layer zinc oxide compression dressings, which can usually be left in place and changed once a week. In addition to compression, wound care, and surgery, large chronic venous ulcers may benefit from venoactive medications, in particular, pentoxifylline and micronized purified flavonoid fraction[5].

INDICATION FOR INTERINVENTION

Indications for interventional treatment are symptoms refractory to conservative therapy, recurrent superficial thrombophlebitis, variceal bleeding, and venous stasis ulceration. After clinical and objective criteria have established the presence of symptomatic varicose veins, the next step is to plan a course of therapy. The efficacy of conservative versus surgical treatment for varicose veins was studied in the Randomised Clinical Trial, Observational Study and Assessment of Cost-Effectiveness of the Treatment of Varicose veins (REACTIV) trial. The authors concluded that surgical treatment was more cost-effective and patients had a higher quality of life benefit than the group who had maintained conservative management alone with compression therapy[5].

SURGICAL MANAGEMENT

Vein stripping It has been more than a century since surgeons began to develop techniques to treat superficial axial venous reflux. Keller introduced saphenous vein invagination and stripping, and Mayo pioneered use of an external stripper to remove the saphenous vein. Babcock described stripping the saphenous vein intraluminally from the ankle to groin. High ligation of the great saphenous vein briefly gained popularity as a method for treating venous reflux without removing the great saphenous vein. Enthusiasm for high ligation of the great saphenous vein quickly faded as it proved to be ineffective because the reflux in the axial vein was not eliminated. Today, traditional surgical treatment of superficial venous reflux involves high ligation as well as stripping of the great saphenous vein from the knee to the groin. Stripping at the ankle has been largely abandoned because of a high incidence of saphenous nerve injury. High ligation and vein stripping usually require general or spinal anesthesia. A transverse or oblique groin incision is made just medial to the femoral artery pulse and inferior to the inguinal crease. Sharp dissection allows identification of the proximal great saphenous vein and other venous tributaries that can be ligated and divided. A brief exploration to identify the presence of a duplicate saphenous system should be performed. The great saphenous vein can then be brought up into the surgical field with gentle traction on the saphenofemoral junction. This maneuver affords further visualization of any missed tributaries that require ligation. The great saphenous vein should be ligated with a nonabsorbable suture and transected near its confluence with the femoral vein[5].

Attention is then directed to the below-knee segment of the great saphenous vein by making a small transverse incision on the proximal, medial calf. The great saphenous vein is identified, ligated distally, and transected. The Codman stripper is then advanced proximally through the

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great saphenous vein to exit the transected vein in the groin incision. The bulb is attached to the end of the Codman stripper that exits the groin incision, and a handle is attached to the other end (exiting the calf incision). The saphenous vein should be secured to the bulb of the stripper and inverted onto itself. Forcefully pulling on the handle of the Codman stripper removes the great saphenous vein from the groin to the knee. Before stripping, the lower extremity should be wrapped circumferentially to aid in hemostasis and to prevent postoperative edema and permanent hyperpigmentation due to
blood extravasation[5].

Complications

Neovascularization refers to the development of new venous tributaries and varicose veins around the previously ligated and divided saphenofemoral junction. The incidence of neovascularization after high ligation and stripping of the great saphenous vein exceeds 30% according to some reports. Interestingly, neovascularization does not occur after endovenous ablation procedures, which obviate the need for a groin dissection or venous tributary ligation. This observation challenges the longheld tenet of varicose vein surgery that stressed the importance of a thorough groin dissection with ligation of all visible venous tributaries. Rather than being beneficial, surgical dissection and tributary ligation may actually trigger neovascularization and varicose vein recurrence. Monitoring for this complication usually involves periodic duplex ultrasound examination[5].

Saphenous nerve injury is a well-documented complication that occurs more frequently when the great saphenous vein is stripped from the ankle to the groin. The saphenous nerve runs close to the great saphenous vein in the calf compared with the thigh, where the nerve and vein have more separation. This anatomic detail may explain why stripping from the knee to the thigh only reduces the risk of nerve injury Although axial venous stripping was considered the "gold standard" of therapy for several decades, several disadvantages to the technique have been realized. Patients required general anesthesia and a hospitalization. In addition, once discharged, patients experienced a prolonged convalescence before resuming baseline activity. Also, the problems of nerve injury and neovascularization were frustrating to surgeons and patients[5].

2 **Trendelenburg operation:**

It is juxta-femoral flush ligation of long saphenous vein (i.e. flush with femoral vein), after ligating named (superficial circumflex, superficial external pudendal, superficial epigastric vein), deep external pudendal vein and unnamed tributaries. All tributaries should be ligated, otherwise recurrence will occur. Double saphenous vein is the commonest anomaly occuring near sapheno-venous junction[7].(Fig .11)

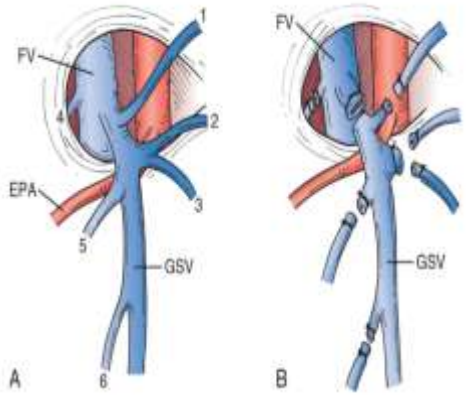

Fig .11 Trendelenburg Procedure

Radiofrequency ablation (RFA) method (VNUSclosure method) (VNUS medical technologies Inc; Sunnyvale, CA, USA) (by Goldman 2000): This procedure is done under general or regional anaesthesia. A RFA catheter is passed into long/ short saphenous vein near sapheno femoral or sapheno-popliteal junction under guidance. 85°C temperature is used for longer period of time to cause endothelial damage, collagen denaturation and venous constriction. Phlebectomy is done
while withdrawing the catheter. Wall of the vein is destroyed through its full thickness. Vein forms a cord, which gets dissolved by macrophages and immune cells[7].

Endo venous laser ablation (EVLA):

It is done as an outpatient procedure or as day- care surgery. Patient lies supine with diseased leg flexed, hip externally rotated and knee flexed. With aseptic precaution, under U/S guidance LSV is cannulated above the knee and a guide wire is passed beyond SFJ and 5- French catheter is passed over guide wire and tip is placed 1 cm distal to the junction. 200 ml of 0.1 % lignocaine (crystalloid with local anaesthetic) is infiltrated along the length of the LSV. Laser fibre is inserted up to the tip of the catheter and catheter is withdrawn for 2 cm and laser fibre protrudes for 2 cm. Laser fibre is fired step by step using diode laser, one mm

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withdrawal in 2 seconds. Once procedure is over catheter is removed and pressure bandage is applied for 2 weeks. Heat produced (729°C – 1000°C at tip) by the laser produces steam bubbles with thermal damage of endothelium leading into occlusion of the vein. Laser energy acts on the blood within the vein rather directly through the wall and heats the blood and in turn heats the vein wall. Drawback of laser therapy is inability to create flush occlusion allowing tributaries to open up to cause possible recurrence[7].

SCLEROTHERAPY

Sclerotherapy can be used to treat a myriad of vein types and sizes, although it is most commonly used to treat smaller vessels such as the reticular veins and telangiectasias. It is best defined as the introduction of a chemical into the lumen of a vein to induce endothelial damage that results in thrombosis and eventually fibrosis. The method used to deliver the sclerosing agent depends on the diameter of the target vein. For smaller veins such as telangiectasias, venulectases, and small reticular veins, liquid sclerotherapy is used.60 Larger reticular veins and varicosities may also be treated by liquid sclerotherapy with a higher concentration of sclerosing agent or by foam sclerotherapy. Foam sclerotherapy involves the addition of air to a detergent sclerosing agent by means of agitation to produce a foam-like consistency, which allows for enhanced contact with the vein wall[7].(Fig .12 , Fig .13)

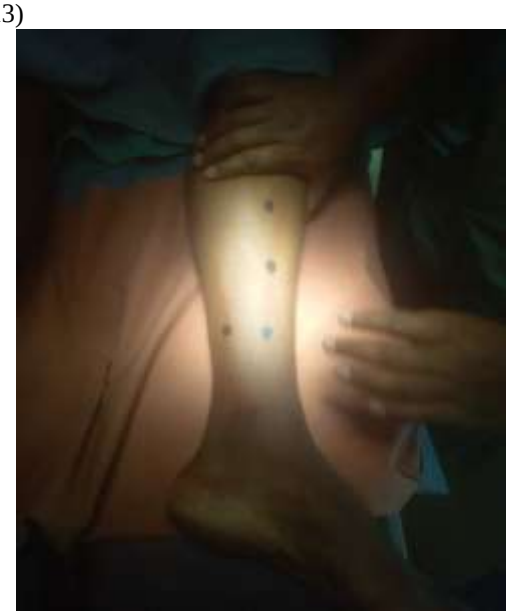

Fig .12 Iv cannulation of marked perforators

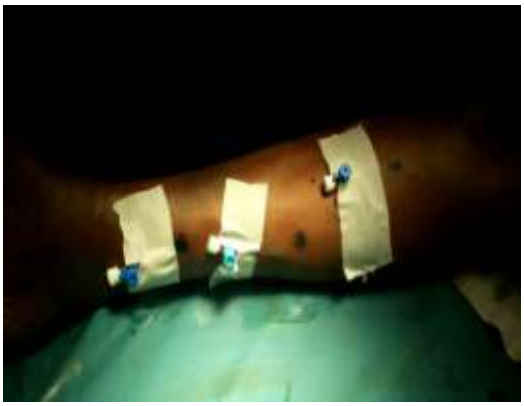

Fig.13 After IV cannulation

Mechanisms of action

• Causes aseptic inflammation
• Causes perivenous fibrosis leading to block
• Causes approximation of intima leading to obliteration by endothelial damage
• Alters intravascular pH/osmolality
• Changes surface tension of plasma membrane[7]

SCLEROSING AGENTS

• Sodium tetradecyl sulphate 3% (STDS)– commonly used
• Sodium morrhuate
• Ethanolamine oleate
• Polidocanol 1%
• Hypertonic saline[7].

PREOPERATIVE PREPARATION

Digital photographs of the target veins should be obtained to document their appearance before sclerotherapy is performed. Larger target veins such as varicose veins should be traced with a surgical marker with the patient standing because they may be difficult or impossible to identify with the patient recumbent. Preoperative marking is typically not required for smaller veins such as telangiectasias and reticular veins. When sclerotherapy is combined with endothermal ablation of the saphenous veins, sclerotherapy below the knee should be performed second. If a staged approach is used, the GSV or SSV should be treated first, with sclerotherapy following several weeks later. Closure of truncal veins may decompress the varicosities and smaller veins, reducing the need for sclerotherapy[7].

Foam sclerotherapy by Tessari-

STDS taken in a syringe is passed rapidly into another syringe which contains air to result in formation of foam. This foam in much larger quantity is injected into the superficial vein. Air get absorbed between foam and endothelial lining is

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destroyed. Foam minimises thrombosis by pushing the blood out of the site of the vessel where action is needed[7]. (Fig .14)

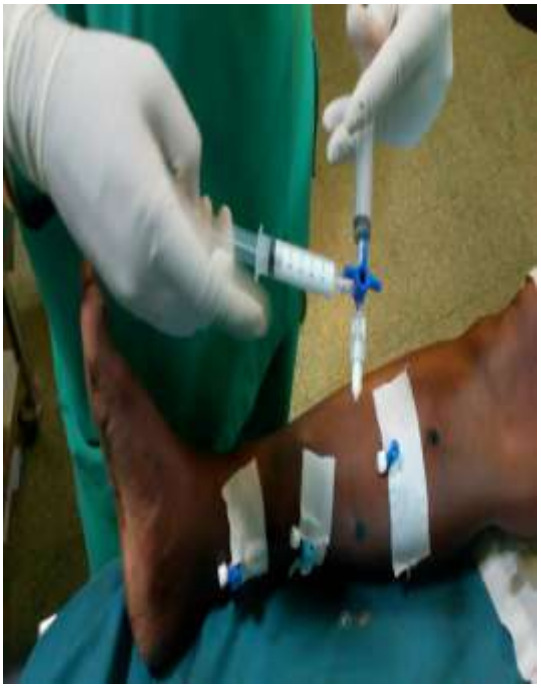

Fig.14 Foam created by TESSARI method

Ultrasound-Guided Foam Sclerotherapy

The method widely used today involves the use of a three-way stopcock connected to two syringes; it was developed by **Tessari** in 1999. One of the main criteria for foam to be viable is that bubble size must be 100 μm or less The pure form of the sclerosing agent is contained on the bubble surface; therefore, concentration is related to bubble size and the air-to-liquid ratio. For the Tessari method, a ratio of one part liquid to four or five parts air is highly effective The amount of foam to be injected can be calculated using the formula $V = \pi \times (D/2) \times L$ (where V is volume, D is diameter, and L is length). Other factors which contribute to success with foam sclerotherapy are stability and longevity. Using the Tessari method, significant coalescence does not begin until after the first 1 to 2 minutes.

Ultrasound-guided sclerotherapy using a Tessari-like method is performed in a similar fashion to foam sclerotherapy without ultrasound. Target vein segments should be marked before the procedure. After the treatment area is mapped, access to the first vein to be treated is achieved with a needle or butterfly under ultrasound guidance. Access is confirmed by return of blood, and the needle/butterfly is taped to the patient's leg. The foam solution is created by a rapid mixing of the air and chemical back and forth between two syringes connected via a three-way stopcock for a total of 20 cycles. After most of the solution has been moved to one syringe, the filled syringe is connected to the needle, and intravascular positioning is reconfirmed with ultrasound. A small amount of foam should be injected initially, under ultrasound, to confirm needle placement within the vein. The amount of foam delivered is determined during injection with the use of ultrasound to visualize when the targeted vein is filled with foam. Upon completion, full-length graduated compression stockings (30 to 40 mm Hg) are applied[5].

Complications

The majority of complications from sclerotherapy are minor and transient. They include hyperpigmentation, telangiectatic matting, pain, and urticaria. Hyperpigmentation occurs in 10% to 30% of patients and is believed to depend on the concentration of sclerosant and to a lesser degree the vessel size and agent used . Spontaneous resolution is observed in 70% and 99% of cases at 6 months and 1 year, respectively. Telangiectatic matting occurs in 15% to 20% of patients, but usually resolves in 3 to 12 months. Pain on injection is largely related to the sclerosing agent used. Detergent agents cause little or no pain, whereas hypertonic saline is the most painful to inject. Urticaria is very common but fades within the first 24 hours. Rarely observed more serious complications include cutaneous necrosis, superficial thrombophlebitis, nerve damage (saphenous, sural), allergic reaction (anaphylaxis), DVT, PE, and inadvertent arterial injection. Necrosis is rare and most often caused by extravasation with hypertonic saline. Superficial phlebitis is usually a result of direct injury to the vein and typically occurs 1 to 2 weeks after the procedure. It is characterized by pain, tenderness to touch, heat, and erythema and can be treated by removal of the coagula via puncture extraction. The incidence of DVT is low after sclerotherapy, with less than 2% of patients affected. Bradbury et al. evaluated one of the largest contemporary series for foam sclerotherapy used in the treatment of truncal reflux. A total of 1252 limbs were treated, 3 patients experienced DVT, and there was 1 PE. Five patients experienced neurologic sequelae in the form of transient visual disturbances, presumably from embolization of foam. Treatment for neurologic sequelae consists of administration of 100% oxygen with the selective use of hyperbaric oxygen[7].

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STAB AVULSION FOR PERFORATOR INCOMPETENCE

Ambulatory phlebectomy is performed by the stab avulsion technique . The patient's varicosities are marked after standing to allow dilation and visualization of affected veins. under local anesthesia with tumescence and IV sedation. First, 1-mm incisions are made along Langer skin lines, and the vein is retrieved with a hook. Continuous retraction of the vein segment affords maximal removal of the vein, and direct pressure is applied over the site. Incisions are made at approximately 2-cm intervals. The extremity is wrapped with a layered compression dressing, and patients are instructed to ambulate on the day of surgery. The postoperative course is short and may require acetaminophen or nonsteroidal anti-inflammatory drugs for discomfort. Compression stockings are worn for 2 weeks after the procedure. Complications are unusual but include bleeding, infection, temporary or permanent paresthesias, and phlebitis from retained vein segments. Recurrence can be a complication. (Fig .15 , Fig .16)

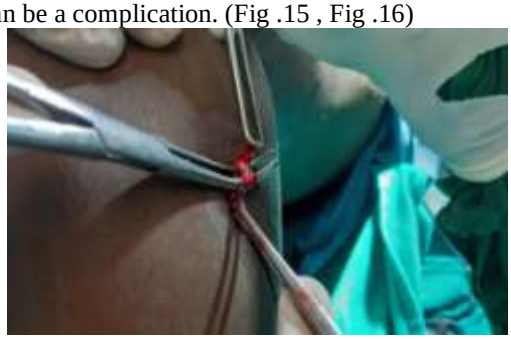

Fig .15 Stab avulsion (hooking out the veins)

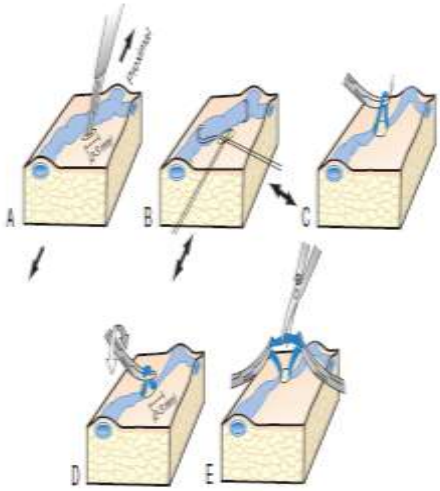

Fig .16 stab avulsion technique

IV. MATERIALS AND METHODS

PRIMARY OBJECTIVES:

- To derive conclusions about efficacy of treatment in Perforator incompetence between stab avulsion versus foam sclerotherapy in GRH,Madurai.

ELIGIBILITY CRITERIA

A.Inclusion criteria:

1. Patients more than 25 years of age groups in both sexes presenting with varicose veins in GRH Madurai.
2. Patients with small varicocities like thread veins and telangiectasia
3. Patients with recurrent varicocities
4. Patients unfit for surgery and aged
5. Patient with uncomplicated perforator incompetence

B. Exclusion criteria:

1. Patients less than 25 years of age
2. Patient not consented for inclusion in the study.
3. Patient with deep vein thrombosis
4. Allergy to sclerosing agents
5. Patients with peripheral arterial diseases
6. Patients with venous ulcer and Large varicocities
7. Patient with saphenofemoral incompetence

V. METHODOLOGY:

From November 2017 to July 2019 patients presenting with varicose veins in GRH Madurai will be recruited in this study.

A total of 100 patients with varicose veins will be included in the study and classified according to CEAP classification. Following consent, a questionnaire will be filled to record the patient's demographic data, duration of illness,occupation,factors predispose to increase intraabdominal pressure,female sex and associated illness.

In all patients, varicose veins graded according CEAP Classification. patient prepared under local anesthesia foam created by **TESSARI method**.Sodium Tetradecyl Acetate3%(STDA) taken in a syringe is passed rapidly into another syringe which contains air to result in formation of foam.1 ml of STDA with 4 ml of air taken in syringe to create foam.Foam injected in the superficial veins.Usually the injection is started at the ankle and proceeded upwards along the length of veins at different points.Later pressure bandage applied for 6 weeks.

Usually the sclerosant causes aseptic inflammation of veins leads to perivenous fibrosis.It also cause approximation of intima leading to obliteration by endothelial damage.

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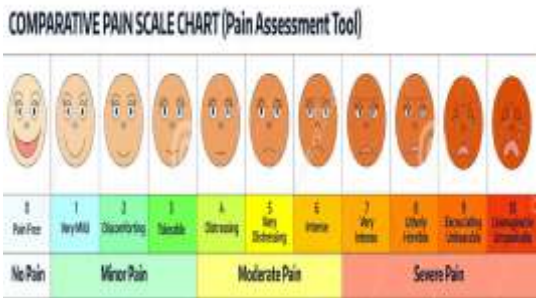
Patients followed for 6 months look for any recurrence. At the same time it compared with stab avulsion of varicose veins. Both groups analyzed for post op complications and recurrence .

VISUAL ANALOG SCORE USED FOR POSTOPERATIVE PAIN ASSESSMENT



VI. RESULTS

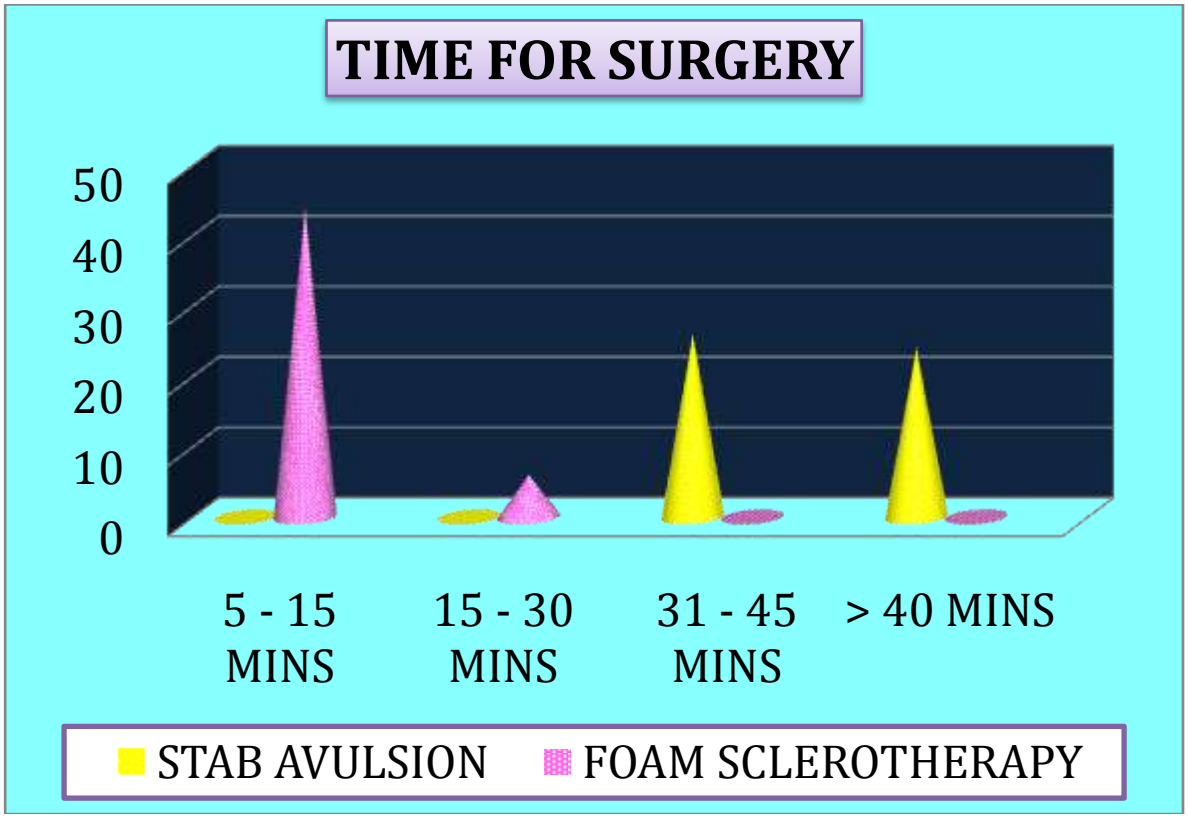
1. AGE DISTRIBUTION :

The mean age group was similar in both groups (43-44 yrs). There was no statistical significance .

2. SEX DISTRIBUTION :

In the Stab avulsion group among 50 patients 32 were male 18 were female . In foam sclerotherapy group 30 were male patients and 20 were females . There was no statistical significance among sex in both groups .

3.TIME TAKEN FOR OPERATION :



The mean time taken for operation in Stab avulsion group was 46.42 minutes and the time for surgery in Foam sclerotherapy was only 14.64 minutes . There was statistically significant .

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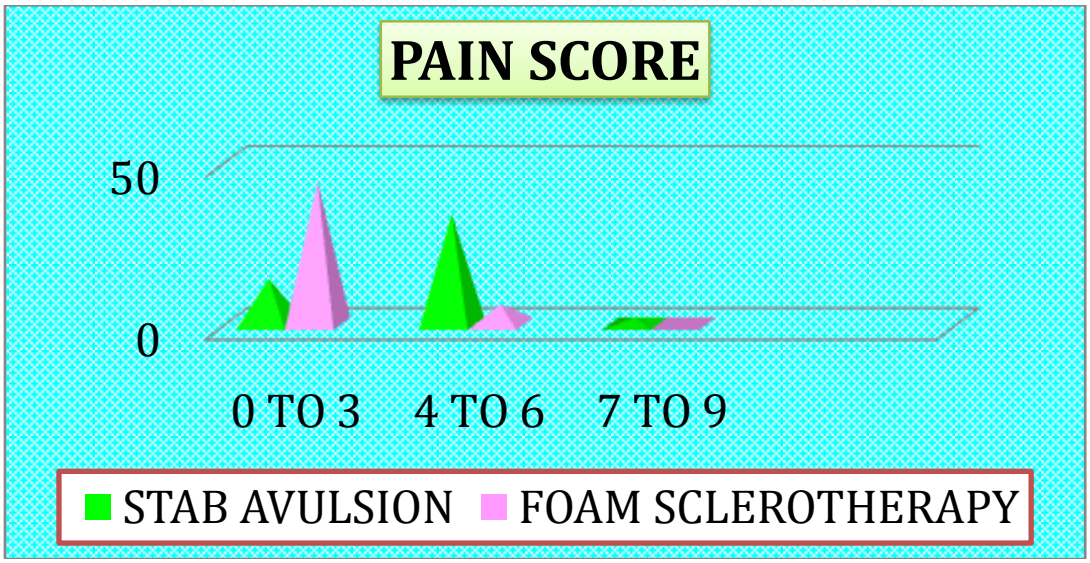
4. POSTOPERATIVE PAIN COMPARED USING VISUAL ANALOG SCORE:

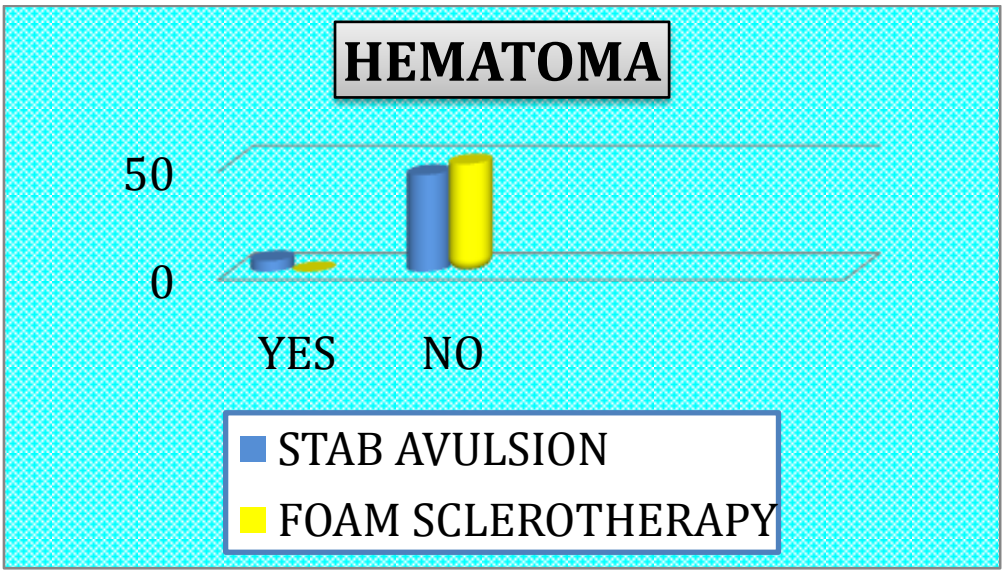


PAIN SCORE	STAB AVULSION	FOAM SCLEROTHERAPY
Oto 3	14	44
4 to 6	34	6
7 to 9	2	0
MEAN	4.5	2.12
MEDIAN	4.5	2
p value	0.00001 (SIGNIFICANT)	

The mean pain score in Stab avulsion group was 4.5 for 9 where it was only 2.12 for 9 in Foam sclerotherapy . p value was 0.00001 , it was statistically significant .

5. HAEMATOMA FORMATION :

It is defined as localized collection of blood at surgical site, found on aspiration of swelling (if present) on incision site or expressed after removal of staplers. It was observed for upto 30 days.



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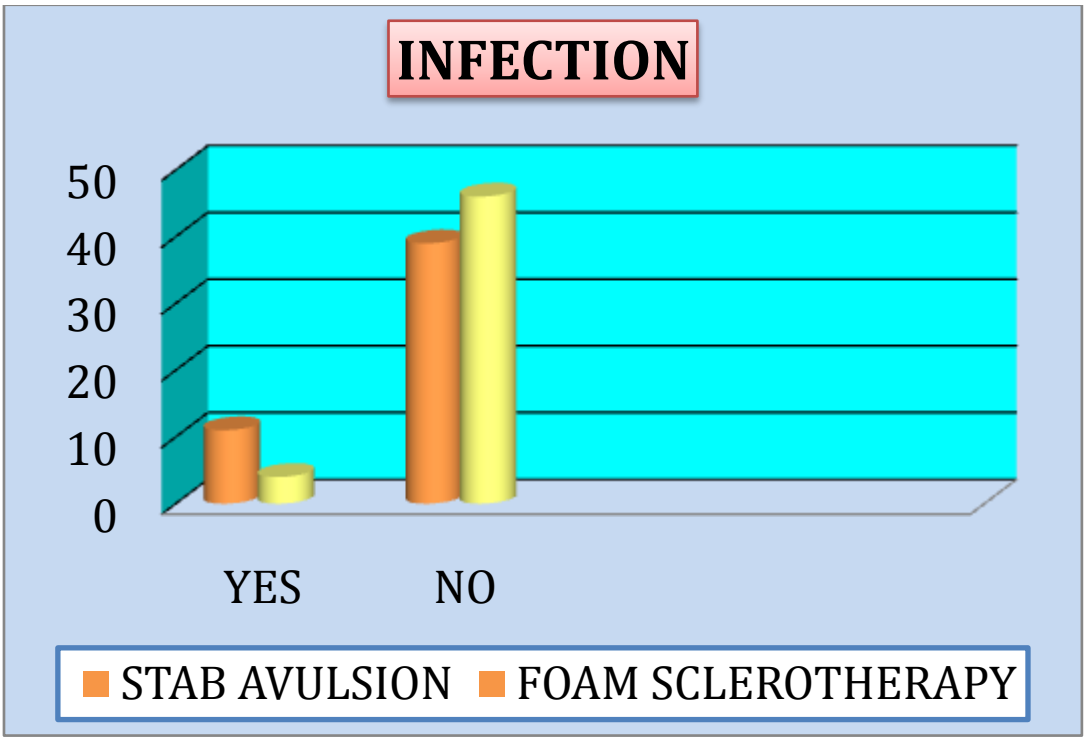
Hematoma	STAB AVULSION	FOAM SCLEROTHERAPY
YES	5	0
NO	45	50
TOTAL	50	50
PERCENTAGE	10%	0%

Hematoma formation occurred in 10 % in stab avulsion group where it was 0 % in foam sclerotherapy , so it was significant .

It is identified by the collection of purulent material at the site of incision, associated with tenderness, erythema and edema at the incision site. It was observed for upto 6 months.

6. WOUND INFECTION:



INFECTION	STAB AVULSION	FOAM SCLEROTHERAPY
YES	11	4
NO	39	46
TOTAL	50	50
PERCENTAGE	22%	8%

Wound infection occurred in 11 cases in stab avulsion where it was only 4 patients in foam sclerotherapy , it was statistically significant .

7.SCAR /PIGMENTATION :

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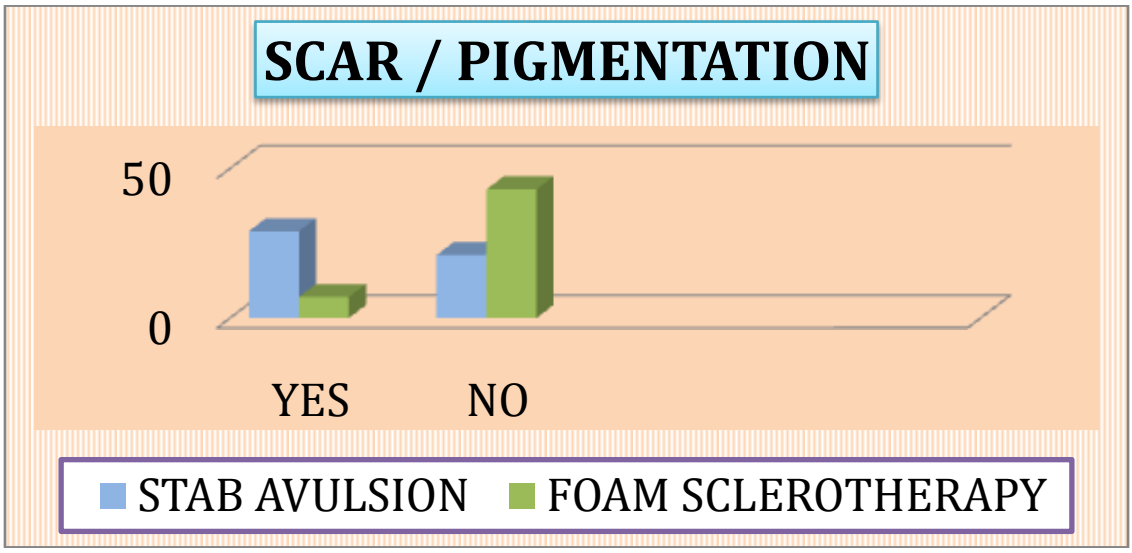
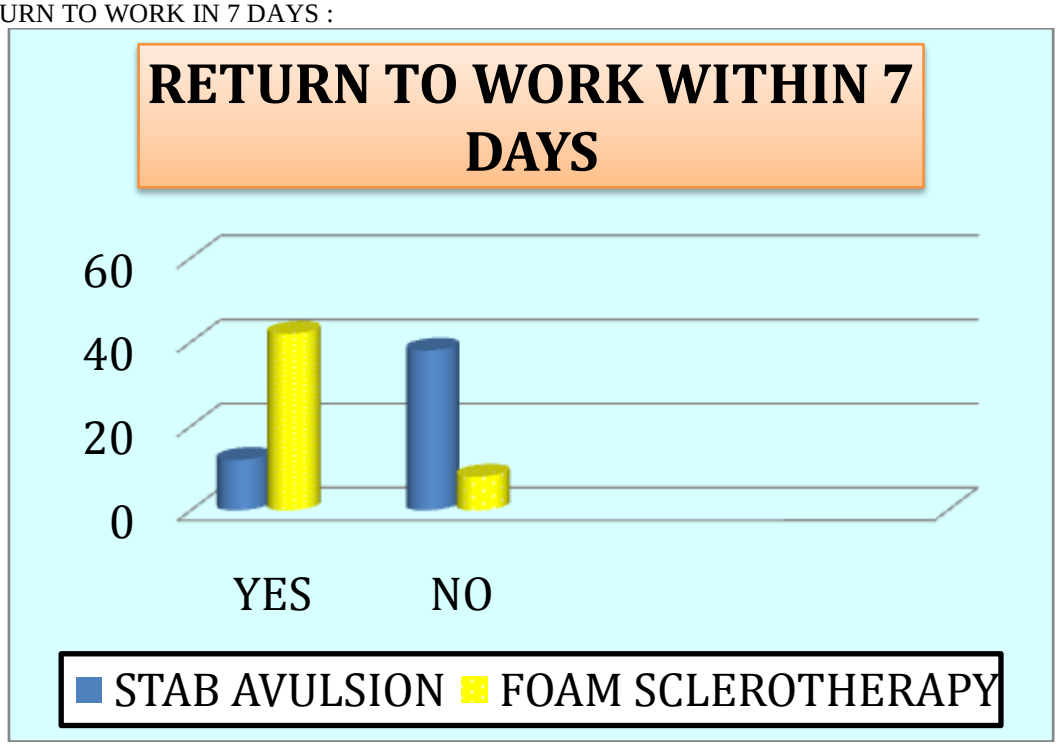


SCAR/PIGMENTATION	STAB AVULSION	FOAM SCLEROTHERAPY
YES	29	7
NO	21	43
TOTAL	50	50
PERCENTAGE	58%	14%

Scar / pigmentation occurred in 58 % of stab avulsion and only 14 % in foam sclerotherapy patients , it was statistically significant.

8.RETURN TO WORK IN 7 DAYS :



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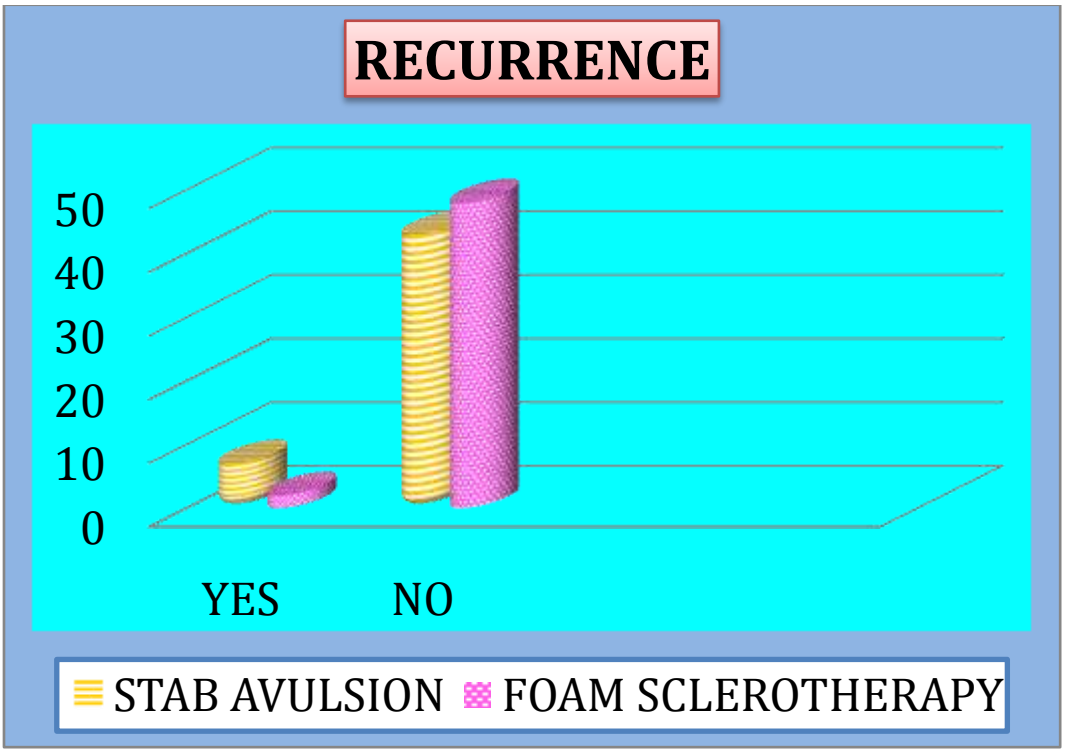
RETURN TO WORK IN 7 DAYS	STAB AVULSION	FOAM SCLEROTHERAPY
YES	12	42
NO	38	8
TOTAL	50	50
PERCENTAGE	24%	84%

In stab avulsion group 24 % of people was returned to work in 7 days but 84 % of people was returned to work in foam sclerotherapy group , it was statistically significant .

9.RECURRENCE :
Recurence is defined as a presence of dilated veins on the operated limb. Our patients were followed up for 6 months .



RECURRENCE	STAB AVULSION	FOAM SCLEROTHERAPY
YES	7	2
NO	43	48
TOTAL	50	50
PERCENTAGE	14%	4%

In 6 months seven patients developed recurrence in stab avulsion group , where only 2 persons developed recurrence in foam sclerotherapy group (significant).

VII. DISCUSSION

- In our study the mean age group of surgery in both groups was 43 years with majority of the cases being males compared to females .
- The mean time taken for operation is 46 minutes in Stab avulsion whereas it is only 14 minutes in foam sclerotherapy. This is understandable since foam sclerotherapy better than stab avulsion.
- There was significant difference in postop pain in the visual analog scale . The mean pain score in Stab avulsion group was 4.5 for 9 where it was only 2.12 for 9 in Foam

sclerotherapy . so it indicate that foam sclerotherapy significantly better than stab avulsion .

- Foam sclerotherapy was associated with a statistically significant less incidence of Haematoma formation, wound infection . This is because foam sclerotherapy is less invasive than the stab avulsion .
- Foam sclerotherapy was associated with an earlier return to normal activities than stab avulsion. This may be due to the foam sclerotherapy is the Opd procedure.
- In 2 years , seven patients of the Stab avulsion group developed recurrence whereas only two patient developed recurrence in the foam sclerotherapy group. This might be because, foam sclerotherapy is technically easier to do with shorter learning curve.

VIII. CONCLUSION

Foam sclerotherapy is associated With shorter operative time, lesser incidence of wound infection,hematoma formation,post operative pain , scar /pigmentation and less recurrence rates. Foam sclerotherapy is an opd procedure also it is suitable for young females where it was give cosmetically better outcome.

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PROFORMA

Name :- I. P. No
Age :-Unit
Sex :-D.O.A
Occupation:-D.O.D

Address :-
Phone no :
DIAGNOSIS:
PRESENTING COMPLAINTS

1) throbbing pain in legs
2) itching
3) Night time cramps
4) Swelling /heaviness in the legs
5) Discoloration /ulceration in the legs
6) Occupation
7) Obstetric history in females
8) Oral contraceptive intake

Co existing co morbidities
Treatment history
GENERAL PHYSICAL EXAMINATION
1. General survey
2. Body build and nourishment
3. Appearance
4. Dehydration: Mild/ Moderate/ Severe/ Nil
5. Anaemia/ Jaundice/ Clubbing/ Cyanosis/ Lymphadenopathy/ Pedal edema
6. Pulse
7. Temperature
8. Respiratory rate
9.Blood pressure

LOCAL EXAMINATION - groin.
1. INSPECTION
2. PALPATION
SYSTEMIC EXAMINATION

Cardiovascular system
Respiratory system
Central nervous system
Abdomen
Genito-urinary system
Per/rectal examination