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Klinika cievnej chirurgie NUSCH, a.s.

Prednosta: MUDr. Jan Tomka, PhD., MHA

Ulcus cruris venosum

Písomná práca ku špecializačnej skúške

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MUDr. Dimitrios Papastavrou

Oddelenie cievnej chirurgie UNB Staré Mesto

Školiteľ: prim. Roman Slysco PhD., MPH

INTRODUCTION- Abstract

Ulcus cruris is the most advanced state of varicose veins. It's a big socioeconomic problem with a significant cost and challenging treatment. The aim of this work is to treat the vein ulcers. Epidemiologic studies show that vein disease affects around 50-80% of populations globally, vein ulcer costs work days and billions of dollars around the world to be treated and sometimes has a cost even bigger than arterial defects. To manage the venous wounds, firstly we have to understand the underlying venous anatomy, pathology and pathophysiology, how the inflammation process and genetic factors affecting pathological changes in CHVI. The blood stasis affects the vein wall, causes disease and later on changes to elastic degradation of the vein wall and causing venous valve insufficiency, these changes lead to venous insufficiency and later cause venous hypertension. Progress through inflammation process or after obstruction of the vein by DVT can lead to edema, chronic pigmentation changes called lipodermatosclerosis. The blood stagnation and further to venous stasis causing venous Ulcers. Principle is to diagnose and differentiate the vein ulcer from the other etiology ulcers. The methods we use to differentiate is history taking, clinical examination, USG examination and classification according to CEAP chart. Methods for treating the vein ulcer are various, usually the most classical non-operative methods use is to reduce edema and venous hypertension by the use of compression therapy with bandages or compression stocking specialized for the vein ulcers. Invasive therapeutics method like operation techniques cross-section and stripping or EVLT procedures, deep venous reconstruction could be choice according to venous pathology. Wound debridement and new non-surgical techniques like Foam sclerotherapy and V.A.C. technology or autologous PRP therapies show good results and make the treatment of the venous ulcers heal quicker. In these work we include the results of the own practice, 4 year follow up and treatment of 20 patient with venous disease, this patient has it first takes medical anamnesis, include previous DVT, genetic inheritance and hematological disorders, male female gender. All of them was scanned with DUS to underlying the venous pathology results than had it venous insufficiency at the VSM and venous

perforating pathology associated with the venous ulcers, all of the patient was treated with multidisciplinary approached surgery/ sclerotherapy and compression therapy 50% the 35% percent with foam and compression techniques, and on case with PRP therapy and compression therapy only. All ulcers need individual approach beginning with clinical examination evaluation and correct classification of the, continue with ultrasound investigation techniques. After is the assessing the medical treatment most possible using surgical and not surgical methods, with combinations of medical treatment and wound dressing and bandage technique or using of negative pressure we succeed to have great results to treat majority of them. Although we have all these advance techniques we do not succeed to treat all of them that the reason we need investigate more all the pathological, physiological and pathophysiological changes and improve the outcomes.

I would like to thanks for his support my chief Slysco Roman PhD. and all my colleagues from the department of vascular surgery , for providing me help to treat patient, I would like also to thanks professor Sefranek and primar Tomka for the support during my studies in vascular surgery.

CONTAIN

INTRODUCTION

GLOSSARY

DECLARATION OF THE WORK

2	Definition	8
3	Epidemiology.....	8
4	History of the venous ulcer.....	9
5	Anatomy of the venous system.....	11
6	Pathophysiology of venous ulcers.....	20
6.1	Changes in superficial and deep veins	20
6.2	Incompetent perforators (IPV).....	22
6.3	Molecular mechanisms affecting venous wall.....	22
6.4	Microcirculation changes.....	23
6.5	Pathophysiology of stasis dermatitis and dermal fibrosis.....	23
6.6	Cytokine regulation and tissue fibrosis.....	24
6.7	Dermal fibroblast function.....	24
6.8	Role of matrix Metalloproteinases (MMP) and their inhibitors in CVD.....	25
7	Diagnosis of venous ulcer	26
7.1	C.E.A.P. Classification	26
7.2	Diagnostic techniques	29
7.3	Ultrasound examination of vein ulcer	32
7.4	Invasive Diagnostic techniques.....	36
8	Differential diagnosis of leg ulcers.....	37

8.1.	Arterial leg ulcers.....	37
8.2	Mixed leg ulcers.....	38
8.3	Diabetic leg ulcer.....	39
8.4	Vasculitic Leg ulcer.....	39
8.5.	Leg ulcers of other etiology.....	40
9	Non- operative management of venous ulcers.....	42
9.1	Pharmacotherapy.....	42
9.2	Compression therapy in venous leg ulcer.....	47
9.3	Compression bandages.....	49
9.4	V.A.C therapy negative pressure in ulcer.....	52
9.5	Prevention of post thrombotic chronic disease.....	54
10	Surgical treatment.....	56
10.1	Surgery in superficial varicose veins and surgical ligation of perforators.....	56
10.2	Deep vein valve reconstruction in severe venous insufficiency.....	57
10.3	Endovascular techniques EVLA, SEPS.....	61
10.4	Endovenous ablation procedures – cyanoacrylate procedures.....	62
10.5	Surgical debridement.....	63
10.6	Foam sclerotherapy.....	64
10.7	Skin grafts.....	67
11	Obstruction relief Deep venous interventions.....	69
12	Alternative therapy	73
13	Prevent of recurrence	74
14	Abstract own results	77

15 Conclusions

References- Literature

Glossary

AASV: anterior accessory saphenous vein

bFGF :fibroblast growth factor

CVI chronic venous insufficiency

CVUs: Chronic venous ulcers

DVT: deep venous thrombosis

DUSG: Doppler ultrasound

ECM: extracellular cellular matrix

EVLA: endovenous laser ablation

EVLV: endovenous laser treatment

GSV: great saphena magna

LRR: Light Reflection rheography

MMPs: matrix metalloproteinases

PA: Plasminogen activator

PPG: Photoplethysmatography

PV: incomplete perforators

RF: Radiofrequency

SSV: short saphena magna

SFJ: saphenofemoral junction

SMC: smooth muscle cells

TIMPs: Tissue inhibitors to metalloproteinases

USG: ultrasound

VAD: Venoactive drugs

V.A.C: vacuum assisted closure

VH: venous hypertension

2 Definition

Venous ulcers is the ulceration of the skin present in the ankle and leg (1) and is due to venous hypertension. Venous ulcers is serious socioeconomic problem, statistical counts the 70 % of wounds are venous etiology. Venous Ulcers has a clinical appearance in the lower part of leg mostly at the ankle they are painful, odorous, and most of the times takes time to be healed. Several guidelines now days the most recent presented from the European vascular society and American venous forum shows the way for more successful treatment (2).

3 Epidemiology

Vein ulcer are the most common venous ulceration in lower leg, observation studies shows incident of 0,8 to 2% among population, several studies like Edinburg studies show prevalence of vein ulcer is between 0,6-2 % but with increase of age we have higher prevalence. In Slovak population at Triangle study refer between 2-4% of population has venous ulcer in active form or healed CEAP C5- C6 classification. At the Bonn in 2003 vein longitudinal study from 3000 participated study patient shows 0.6 % healed and 0.1 % without healing. Prevalence of the disease depends from the ages in the younger population between 30-49 the prevalence in low around 0.2%, at ages 50-60 the prevalence in 0.6 % but between 60-69 % the increase 1.1 % and the age 70-79 % has significant prevalence of 2,4 % per year. At the elderly people < 80 the incidence is around 3%. At age and sex distribution females are most often affected from vein ulcers especially from the age of 60 and over. One very important factor at the has genetic predisposition, patient from previous DVT/PTS prevalence has 22% of trophic changes and venous C5/C6 findings. Another population commonly meets with are obese people we have a prevalence around 0,1-1,0% of population affected by venous ulcer.

Guidelines European ,angiology 2013, vein ulcer book p. 30- 37

4 History of the vein ulcer

Venous ulcers always a plague in the community, a lack of sanitations, hygiene and ever seen human always was trying to find a solution to battle human gravity, pain and ulcer- wounds in legs. In history ulcers was described from prehistoric ages with sigma and decreased quality of life (5). Egyptian at Smith papyrus prescribe ulcers in the ankle region and suggest treatment modality with bee honey and bandaging. At ancient Greek documents Hippocrates observes the varicose veins and ulcer suggest medical treatment of chronic vein ulcer with hygiene of wound and honey dressings. At middle ages we see some beginning to understand the pathological and pathogenesis of venous ulcer. During 18- 19th. Century, we start not only to understand the pathogenesis but also they prescribed from epidemiological point. Commonly ulcer and wound could find on elderly people and female population, at young men especially to the soldiers man whom often had it leg injuries during work and had it low hygienic standards ,often suffer from hypovitaminosis and they was prescribed like a scorbutic disease. This nutrition problems was very common between people. During 18th century doctors accepted the theory that chronic ulcers is affected by blood from the opened wounds. These adverse conditions in the blood that flushed out through the open sores. If such a closed ulcer, disease state levels stays could accumulate and overwhelm the lungs and other vital organs and cause severe disease .Some surgeons warned prior to the conclusion of such ulcers .The discharge of harmful fluids incisions were made in different parts of the body to maintain the wound openly conservative surgery is kept advocates maintain all wounds openly. Surgeon holding advocates kept all open wounds that can faster be cleaned. Until the late 18th century persisting term menstrual ulcer, for which, it was assumed that the name break removes harmful conditions that previously flushed out in menstruation.

In the second half of the 18th century, they began to promote new ideas .English surgeon D. Ingram wrote that concerns the healing of these ulcers were more excuse those surgeons who were not able to heal.

In the 19th century, they have already begun to promote ideas that ulceration can and should be treated by general and local treatment. The total treatment focused on the cause of the ulcer (Lues, scurvy, etc.). Local treatment consisted of application of various inorganic and organic materials, bandages and surgical procedures on adjacent varices. The list of substances that are recommended in the treatment of venous ulcers, is huge: mercury, zinc, silver, vitriol, coal, balsams, decoctions of various flowers, leaves, fruits and vegetables. Assuming that these ulcers are due to poor nutrition, they administered the many different types of diet soups and porridge containing flour, chalk, tragacanth, acacia.

More and more surgeons had various results of these local preparations gradually grown to the point that it is a dead end that cannot lead to success. They began to treat with bed rest and leg elevation, leading to healing leg ulcers is better when the legs rest. But soon as the patients dismissed the hospital, their sores reopened. Therefore it matured time to find such a method to be allowed to treat these patients ambulatory and would guarantee long lasting results.

In England the T. Baynton (6), began with the use of compression bandages circulation adhesive textile strips. His followers improved that method further, by using dressings soaked in antiseptic solution. The meaning of varicose veins in the pathogenesis of venous ulcers, however, still not clear. Some patients with leg ulcers had varices, others with large varices had no ulcer, or even just more pronounced changes in the skin. Notwithstanding that more and more surgeons had suspected some connection varicose veins and leg ulcers and began to focus on varicose veins in the area of chronic ulcers. Everard Home saphenous vein ligation performed at the medial area of the knee. Thomas Baynton subcutaneously cut in a safe at the tibia by a slim curved scalpel. Velepeau introduced under the saphenous varicose ligation with thick needle under the skin whereby the vein closed. However, after several such operations were septic phlebitis, and patients died, these trial surgeons ceased. In the second half of the 19th century, more and more surgeons began to use the term "varicose ulcer", accepting that a relationship between varices and leg ulcers. In England, J Gay focused on the anatomy and pathology of superficial and deep veins of the legs. Based on his

study he emphasized that venous ulcers associated with pathological changes in deep veins. He described the perforators of the distal tibia and realize their importance, however, the pathogenesis of leg ulcers. This chapter opened up F.B. Cockett(7) a hundred years later and in 1955 paper The Pathology and Treatment of Venous Ulcers Of The leg, the foundations of modern surgical treatment of leg ulc

5 Anatomy of the venous system

The anatomy of venous system divided in superficial and deep anatomic structures:

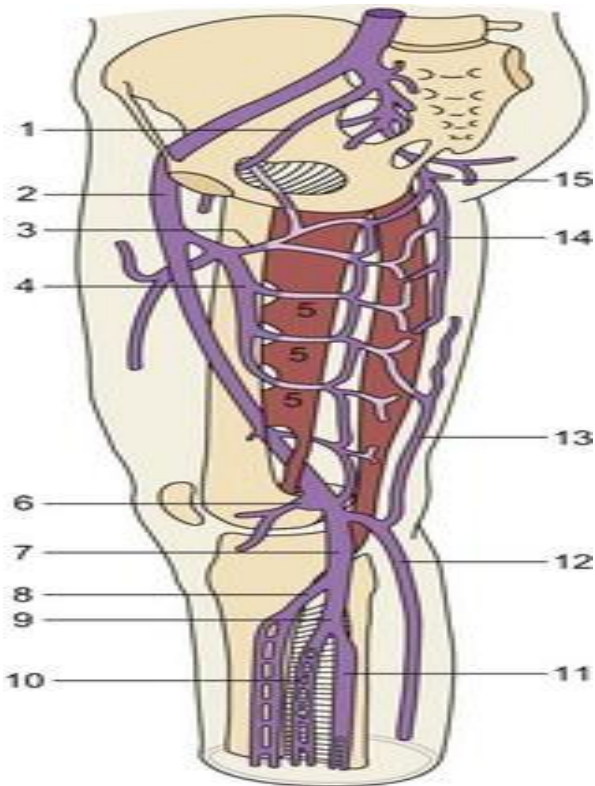
Deep Venous System

The structure of the deep venous system is consist. There are at least two deep veins for each of the three arteries (anterior and posterior tibial arteries and peroneal artery), mutually communicating by transverse bridges (like a ladder). The extremely rich muscular plexus (also connected to the superficial veins) drains into these axial veins placed parallel to arteries. At the foot, axial veins are prevalent in the plantar region, where the first pump mechanism is present (Léjars sol). In the leg two deep veins for each of the three arteries communicate by means of transverse bridges. At the knee and thigh, deep veins flow into the collecting system 'popliteal-femoral veins'. Several other secondary veins are present that can ensure a natural bypass when obstruction occurs to the femoral vein. 1: obturator vein, 2: common femoral vein, 3: medial circumflex femoral vein, 4: profunda femoris vein, 5: perforating veins, 6: descending genicular vein, 7: popliteal vein, 8: posterior tibial vein, 9: proximal portion of the posterior tibial venous axis (common trunk), 10: posterior tibial veins, 11: peroneal (interosseous veins), 12: short saphenous vein, 13: posterior subcutaneous femoral vein, 14: ischial vein, 15: inferior gluteal vein.(picture 1) At the foot, axial veins are prevalent in the plantar region, where the first pump (although not the most important) mechanism is present (Léjars sole).At the soleus and gastrocnemius sites the veins are even larger in number and arranged in a spiral shape, due to the

longitudinal excursion amplitude of the muscles between contraction and relaxation. This creates a volume reservoir (pump chamber), and the relative muscles (soleus and gastrocnemius) are responsible for both movement/standing position as well as pump function (the second and most important pump). This system is correctly termed the calf muscle pump or peripheral heart. In contrast, posterior deep compartment veins (posterior tibial and peroneal) and anteroexternal compartment veins (anterior tibial) are rectilinear, as the surrounding muscles lean against the bones and have a limited shortening during contraction.

At the knee and thigh, deep leg veins flow into the collecting system (popliteal-femoral veins). They run in the popliteal crease and adductors canal, and are not enwrapped by a muscular layer as the blood flow to the abdominal cavity has not been held back by compression. The other thigh veins (profunda femoris and circumflex) are still deep intramuscular veins. The popliteal vein is also connected by anonymous muscular veins to the profunda femoris and the sciatic nerve vein, creating a natural bypass when obstruction occurs to the femoral vein (thrombosis, extrinsic compression, bone fracture). Thanks to this autonomy, the femoral vein is used as an alternative conduit when other more accessible superficial veins are unavailable. The common femoral vein collects the backflow of the lower limb and sends it to the pelvis (iliac veins and inferior vena cava), where aspiration pleurodiaphragmatic forces prevail, together with vis-a-tergo of the renal veins. The common femoral vein in particular receives the GSV below the inguinal ligament where it becomes the external iliac vein. A potential alternative way of discharge in this area is due to the obturator vein (normally draining part of the muscles of the medial thigh) and the sciatic vein, often not macroscopically evident (first embryonic vein, secondarily replaced by the femoropopliteal axis, which can be activated in certain conditions). Together with the superficial veins they can contribute to limb drainage in case of femoral thrombosis by their connection to the hypogastric vein. However, the same system may be the cause of varices when endopelvic hypertension is transmitted

to the superficial limb veins. The sciatic vein may also be involved in congenital venous malformations, typically KlippelTrenaunay syndrome.



Deep venous anatomy picture 1 sclerotherapy book 5 th edidion picture 1

Old terminology	New terminology
Femoral Vein	Common femoral vein
Superficial femoral vein	Femoral vein
Sural veins	Sural veins
	Soleal veins
	Gastrocnemius veins

Hunterian perforator	Mid thigh perforator
Cocketts Perforator	Paratibial perforator
Mays Perforator	Ankle lateral and medial perforator
Gastrocnemius point	Intergemellar perforator

Anatomy of the Superficial Veins

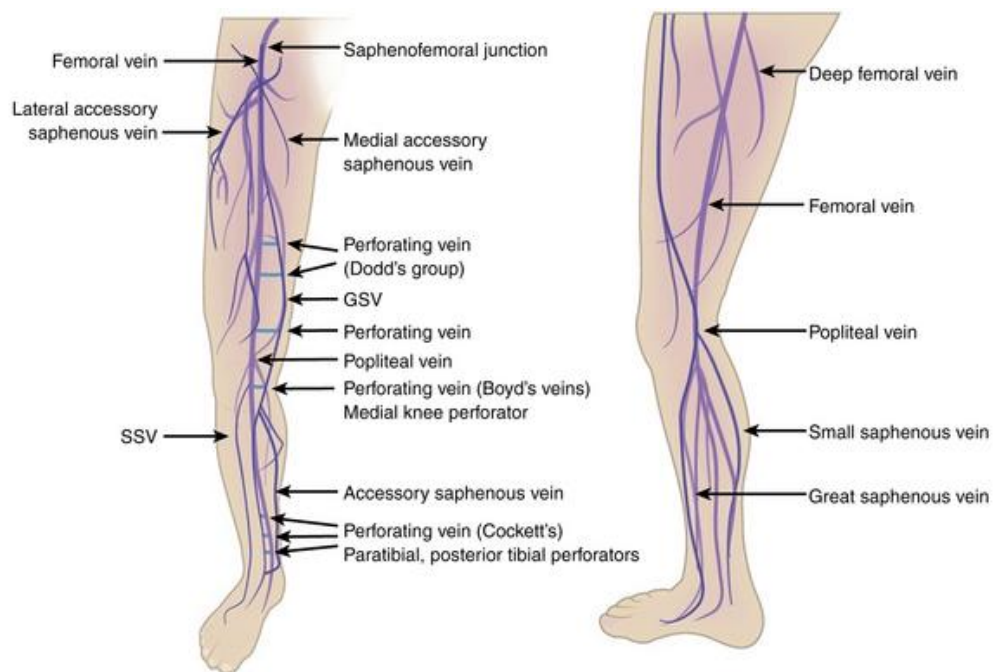
The most important superficial veins are the GSV and the SSV. It is generally thought that the term saphenous is derived from the Greek word *saphenes*, meaning evident, but it could also come from the Arabic words *el safin*, which mean hidden or concealed. Of course, these terms were important in the practice of blood letting (9).

Great saphenous vein

This vein begins on the dorsum of the foot as a dorsal venous arch and internal marginal vein. It passes anterior (10–15 mm) to the medial malleolus, crosses the tibia at the distal third and runs along the tibial internal edge. At the knee the vein it bends posteriorly, running around the condilus femoralis, in contact with the anterior edge of the sartorius muscle, then ascends in the anteromedial thigh, crosses the sartorius and adductor brevis and enters the Scarpa triangle to empty into the common femoral vein .This termination point is referred to as the saphenofemoral junction (SFJ) but is also known as the *crosse*, which is the French description for its appearance as a shepherd's crook. The average diameter of a normal GSV is 3.5–4.5 mm (range 1–7 mm).

Traditional anatomical terms for the lower limb, medial aspect. 1: superficial epigastric vein, 2: pampiniform plexus, 3: external pudendal vein, 4: superficial dorsal vein of the penis, 5: superficial medial circumflex femoral vein, 6: accessory posterior saphenous vein of the thigh, 7: femoropopliteal vein, 8: great saphenous vein, 9: sartorius muscle, 10: anastomoses between the great and small

saphenous veins, 11: posterior arcuate vein (posterior saphenous vein of the leg or vein of Leonard), 12: medial marginal communicating veins, 13: plantar sole, 14: superficial dorsal metatarsal veins, 15: superficial dorsal venous arch of the foot, 16: venous plexus of the dorsal surface of the foot, 17: anterior vein branch (anterior saphenous vein of the leg), 18: superficial femoral vein, 19: perforating veins of Dodd, 20: accessory small saphenous vein (anterior accessory saphenous vein), 21: superficial inguinal lymph nodes, 22: superficial lateral circumflex femoral vein, 23: common femoral vein, 24: superficial circumflex iliac veins. **B**, The great saphenous vein (GSV) and its tributaries are occasionally well displayed on thin legs. ALTV, anterolateral thigh vein; AVL, anterior vein of the leg (accessory saphenous vein); PAV, posterior arch vein.



Picture 2

The GSV receives multiple tributaries along its course. These usually lie in a less supported, more superficial plane above the membranous fascia. The posterior arch vein, the anterior superficial tibial vein and the medial superficial pedal vein join the GSV in the lower leg. The posterior arch vein (known as the vein of Leonardo, but now classified as the posterior accessory saphenous vein) is a

major tributary to the GSV. It enters the GSV below the knee and otherwise communicates with the deep venous system through multiple perforating veins. These are, in ascending order: the Cockett I, Cockett II and Cockett III perforators and the 24-cm perforating vein, now called the upper, middle and lower posterior tibial perforators.

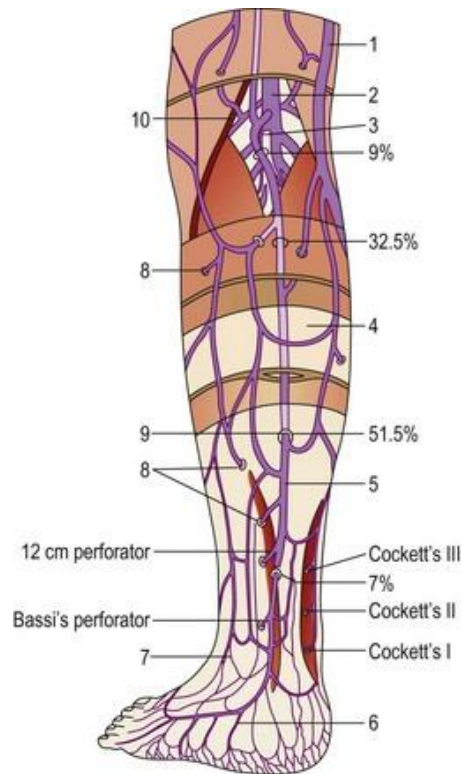
In the thigh, two main clusters of perforating veins connect the saphenous vein to the deep system. Just above the knee, there is the Dodd group, and in the mid-thigh, the Hunterian perforators (now called mid-thigh perforators).

Two large tributaries in the upper third of the thigh – the posteromedial and anterolateral tributaries – join the GSV proximally. These veins usually enter the GSV before it dives posteriorly to penetrate the deep fascia at the fossa ovalis. Both the medial and lateral superficial thigh veins may be so large that they are mistaken for the GSV itself. A variable number of perforators connect the GSV to the femoral, posterior tibial, gastrocnemius and soleal veins.

Small saphenous vein

The SSV is the most prominent and physiologically important superficial vein below the knee. Like the GSV, the SSV has a thick wall and usually measures 3 mm in diameter when normal. It begins at the lateral aspect of the foot and ascends posterior to the lateral malleolus as a continuation of the dorsal venous arch. It continues up the calf between the gastrocnemius heads to the popliteal fossa, where it usually enters the popliteal vein.

Traditional anatomical terms for the lower limb, posterior aspect. 1: Great saphenous vein, 2: popliteal vein, 3: tibial nerve, 4: deep fascia, 5: small saphenous vein, 6: lateral marginal communicating veins, 7: lateral malleolus, 8: perforating veins, 9: gastrocnemius point, 10: common peroneal nerve.



Picture 3 SSV sclerotherapy book p.

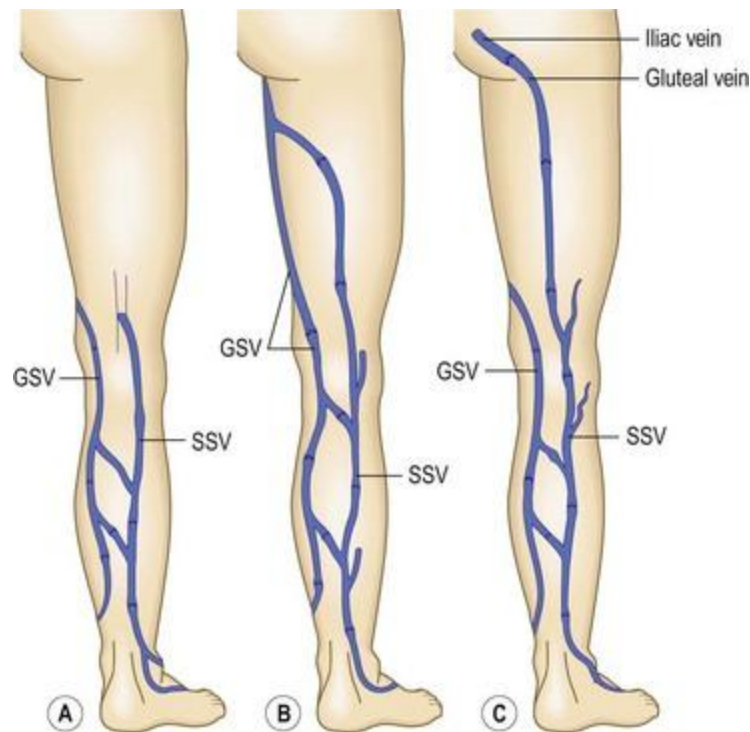
The termination of the SSV is quite variable, usually occurring in the popliteal vein, as stated above. However, in 27% to 33% of the population, it terminates above the level of the popliteal fossae, either directly into the GSV or into other deep veins. In 15.3% of patients, the SSV communicates with the popliteal vein, then continues terminating in the GSV. In 9% to 10%, the SSV empties into the GSV or the deep veins below the popliteal fossae. The SSV may also join the GSV in the thigh through an oblique epifascial vein (the Giacomini vein), or it may continue up under the membranous fascia of the thigh as the femoropopliteal vein, joining the deep veins in the thigh at various locations.

Variations in the termination of the small saphenous vein (SSV). **A**, Termination into the saphenopopliteal junction; **B**, termination into the great saphenous vein (GSV); **C**, termination into the gluteal vein.

Like the GSV, the SSV runs on or within the deep fascia, usually piercing the deep fascia just below the flexor crease of the knee as it passes into the popliteal fossa. Gross incompetence of the SSV usually occurs only in areas where the SSV and its tributaries are superficial to the deep fascia, on the lateral calf and lower third of the leg behind the lateral malleolus. The SSV often receives substantial tributaries from the medial aspect of the ankle, thereby communicating with the medial ankle perforators. The SSV may also receive a lateral arch vein that courses along the lateral calf to terminate in the SSV distal to the popliteal fossa. It may also connect directly with the GSV.

Other superficial veins and collateral veins

The superficial collateral or communicating venous network consists of many longitudinally, transversely and obliquely oriented veins. These originate in the superficial dermis, where they drain cuticular venules. These veins are normally of lesser diameter, but when varicose they can dilate to more than 1 cm. They are thin walled and are more superficial than the superficial fascia that covers the saphenous trunks. They drain into deep veins through the saphenous veins, directly through perforating veins or through anastomotic veins in the abdominal, perineal and gluteal areas. Therefore, collateral veins may become varicose either in combination with truncal varicose veins or independently.



Picture 5, sclerotherapy book

Schematic diagram of subcutaneous venous anatomy showing four types of flow from subcutaneous veins (SCV). SCV to GSV/SSV to SFJ/SPJ to deep system; SCV to GSV/SSV to perforator to deep system; SCV to perforators to deep system; SCV to deep system. GSV, great saphenous vein; SSV, small saphenous vein; SFJ, saphenofemoral junction; SPJ, saphenopopliteal junction.

Although many collateral veins are unnamed, some prominent or consistent superficial veins are, for example, the Giacomini vein, which connects the proximal GSV to the SSV. This vein has been found by duplex examination in 70% of limbs with chronic venous insufficiency. Other examples include the lateral anterior accessory saphenous vein (AASV), which runs from the lateral knee to the SFJ, the anterior crural veins, which run from the lower lateral calf to the medial knee, and the infragenicular vein, which drains the skin around the knee. Geniculate perforators, although small, may contribute significant reflux.

A lateral subdermal plexus of reticular veins, first described by Albanese et al has its origin through perforating veins at the lateral epicondyle of the knee. It has been speculated that it represents a remnant of the embryonic superficial venous system that fails to involute. This system of veins has its importance in the development of telangiectasia. These veins may become varicose even in the absence of truncal varicosities.

6 Pathophysiology of venous ulcers

Chronic venous disease (CVD) of the lower limbs is often characterized by symptoms and signs as result of a structural or functional abnormalities of the veins. Symptoms include aching, heaviness, leg tiredness, cramps, itching, burning sensation, swelling and the restless leg syndrome, as well as cosmetic dissatisfaction. Signs include telangiectasia, reticular and varicose veins, edema, and skin changes such as pigmentation, lipodermatosclerosis, dermatitis and ultimately ulceration. (9)

CVD is usually caused by primary abnormalities of the venous wall and valves or by secondary abnormalities resulting from previous deep venous thrombosis (DVT) that can lead to reflux, obstruction or both, rarely can be also congenital malformations lead to CVD.(10)

6.1 Changes in superficial and deep veins

Varicose veins are a common manifestation of CVD and are believed to result from abnormal distensibility of connective tissue in the vein wall. Veins from patients with varicosities have different elastic properties than those from individuals without varicose veins.(11)

Primary varicose veins result from venous dilatation and valve damage without previous DVT. Secondary varicose veins are the consequence of DVT or, less commonly, superficial thrombophlebitis. Recanalization may give rise to relative obstruction and reflux in deep, superficial and perforating veins. Approximately 30% of patients with deep venous reflux shown by imaging appear to have primary valvular incompetence rather than detectable post-thrombotic damage.

(12).Rarely, deep venous reflux is due to agenesis or aplasia. Varicose veins may also be caused by pelvic vein reflux in the absence of incompetence at the saphenofemoral junction, thigh or calf perforators. Retrograde reflux in ovarian, pelvic, vulval, pudental or gluteal veins may be also associated with clinical symptoms and signs of pelvic congestion.(13)

Following DVT, spontaneous lysis over days or weeks and recanalization over months or years can be observed in 50% to 80% of patients. (14)Rapid thrombus resolutions after DVT is associated with a higher incidence of valve competency. Such resolution depends on thrombus extent and location. Inadequate recanalization following DVT can lead to outflow obstruction. Less frequently, obstruction results from extramural venous compression (most commonly left common iliac artery), intra-luminal changes, or rarely from congenital agenesis or hypoplasia.(15)

Most post-thrombotic symptoms result from venous hypertension due to valvular incompetence and/or outflow obstruction. Venous hypertension increases transmural pressure in post-capillary vessels leading to skin capillary damage lipodermatosclerosis and, ultimately, ulceration.(16)

The reported prevalence of post-thrombotic syndrome following DVT has been variable (35% to 69% at 3 years and 49% to 100% at 5 to 10 years) and depends on the extent and location of thrombosis as well as treatment. Patients with both chronic obstruction and reflux have the highest incidence of skin changes or ulceration. The risk of ipsilateral post-thrombotic syndrome is higher in patients with recurrent thrombosis and is often associated with congenital or acquired thrombophilia. In recent studies, skin changes or ulceration have been less frequent (4% to 8% in 5 years) in patients with proximal thrombosis treated with adequate anticoagulation, early mobilization, and long-term elastic compression.(17)

6.2 Incompetent perforators (IPV)

Perforators are the veins which connects the superficial and deep veins they cross the fascia, they can have bidirectional flow during muscular contraction outward and during relaxation inward. In the normal patient with the venous hypertension are flows inward from the superficial to deep. However in the pathological cases of deep venous damage after DVT the flows goes outward. IPV can be due to superficial or deep venous reflux but they cannot be isolated. The prevalence of IPVs their diameter, volume and flow can increase the clinical picture of chronic venous disease, or the coexisting deep venous damage. Studies show that in 10% of woman we can see there no reflux but only IPVs. (18)

6.3 Molecular mechanism affecting the venous wall

Varicose veins have different elastic properties in contrast with normal veins. The collagen I and collagen III is altered suggesting systemic disorder with genetic basis. (19)

Leucocyte activation, adhesion and migration through the endothelium as a result of altered shear stress contribute to the inflammation and subsequent remodeling of the venous wall and valves. Reduction in shear stress also stimulates production of tumor growth factor – $\beta 1$ (TGF- $\beta 1$) by activates endothelial cells and smooth muscle cells (SMCs) inducing SMC migration into the intima and subsequent proliferation. Fibroblasts proliferate and synthesize matrix metalloproteinases (MMPs) overcoming the effect of tissue inhibitors of metalloproteinases (TIMPs). The MMP/TIMP imbalance results in degradation of elastin and collagen. This may contribute to hypertrophic and atrophic venous segments and valve destruction as observed in varicose veins. Remodeling of the venous wall and abnormal venous distension prevents valve leaflets from closing properly resulting in reflux. (20)

6.4 Microcirculation changes

With help of various techniques laser Doppler or interstitial capillaroscopy we notice the changes in skin microcirculation in legs with CVD. At patient with venous hypertension capillaries can become dilated elongated tortuous, especially at the skin site where lipodermatosclerosis and hypertension presents. These associated with high overall of microvascular blood flow at dermis and decrease flow of nutritional capillaries. Can be observed under capillaroscopy a striking feature at skin and dilatated capillaries. This is the cause associated with micro edema, precapillary fibrin and other proteins which prevent the normal nutrition of the skin cells causes' venous ulcer. Microlymphangiopathy and outward migration of leucocytes exacerbate inflammation and edema. These phenomenon cause capillary thrombosis successively lead to reduction in nutritional skin capillaries and transcutaneous oxygen. (21)

6.5 Pathophysiology of stasis dermatitis and dermal fibrosis

Mechanism modulating leukocyte activation, fibroblast function and dermal extracellular matrix alterations have been the focus of investigation in the 1990s. As stated above, CVD is caused by persistent venous hypertension leading to chronic inflammation. It is hypothesized that the primary injury is extravasation of macromolecules (i.e. fibrinogen and α_2 -macroglobulin) and red blood cells into the dermal interstitium. Red blood cell degradation products and interstitial protein extravasation are potent chemoattractant that represent the initial underlying chronic inflammatory signal responsible for leukocyte recruitment. These cytochemical events are responsible for increased expression of intercellular adhesion molecule-1 (ICAM-1) on endothelial cells of microcirculatory exchange vessels observed in CVI dermal biopsies. ICAM-1 is the activation dependent adhesion molecule utilized by macrophages, lymphocytes and mast cells for diapedesis. (22)

6.6 Cytokine regulation and tissue fibrosis

As indicated above, CVD is characterized by leukocyte recruitment, tissue remodeling and dermal fibrosis. These physiologic processes are prototypical of disease states regulated by TGF- β 1. TGF- β 1 is present in pathological quantities in the dermis of patients with CVD and increases with disease severity. TGF- β 1 is secreted by interstitial leukocytes and becomes bound to dermal fibroblasts and extracellular matrix proteins. Platelet-derived growth factor receptor alpha and beta (PDGFR- α and PDGFR- β) and vascular endothelial growth factor (VEGF) have also been identified in the dermis of CVD patients. It has been postulated that these molecules regulate leukocyte recruitment, capillary proliferation and interstitial edema in CVD by upregulation of recruitment, diapedesis and release of chemical mediators.(23)

6.7 Dermal fibroblast function

Aberrant phenotypic behavior has been observed in fibroblasts isolated from venous ulcer edges when compared to fibroblasts obtained from ipsilateral thigh biopsies of normal skin in the same patients. Collagen production by fibroblasts is increased by 60% in a dose-dependent manner in control skin whereas venous ulcer fibroblasts are unresponsive. Unresponsiveness in ulcer fibroblasts is associated with a fourfold decrease in TGF- β 1 type II receptors. This is associated with decrease in phosphorylation of TGF- β 1 receptor substrates SMAD 2 and 3 as well as p42/44 mitogen activated protein kinases, and decrease in collagen and fibronectin production from venous ulcer fibroblasts when compared to normal controls.(24)

Venous ulcer fibroblasts growth rates become markedly suppressed when stimulated with bFGF, EGF and IL-1 and this growth inhibition can be reversed with bFGF. The proliferative response of CVI fibroblasts appear to become morphologically similar to fibroblasts undergoing cellular senescence. (25)

6.8 Role of matrix Metalloproteinases (MMPs) and their inhibitors in CVD

The signaling event responsible for development of a venous ulcer and the mechanism responsible for slow healing is an orderly process that involves inflammation, re-epithelialization, matrix deposition and tissue remodeling. Matrix deposition and tissue remodeling are processes controlled by matrix metalloproteinases (MMPs) and tissue inhibitors of matrix metalloproteinases (TIMPs). In general, MMPs and TIMPs are induced temporarily in response to exogenous signals such as various proteases, cytokines or growth factors, cell-matrix interactions and altered cell-cell contacts. Gelatinases MMP-2 and MMP-9 as well as TIMP-1 appear to be increased in exudates from venous ulcers compared to acute wounds. However, analyses of biopsy specimens have demonstrated variable results. Herouy *et al.*, reported that MMP-1,2 and TIMP-1 are increased in patients with lipodermatosclerosis compared to normal skin. In a subsequent investigation, biopsies from venous ulcer patients were found to have increased levels of the active form of MMP-2 compared to normal skin. In addition, increased immune-reactivity to extracellular inducer of MMP (EMMPRIN), membrane Type 1 and 2 metalloproteinases (MT1-MMP and MT2-MMP) were detected in the dermis and perivascular regions of venous ulcers. Saito *et al.* were unable to identify differences in overall MMP-1,2,9 and TIMP-1 protein levels or activity in CVD patients with clinical CEAP class 2 through 6 disease compared to normal controls. However, within a clinical class, MMP-2 levels were elevated compared to MMP-1, 9 and TIMP-1 in patients with CEAP class 4 and 5 disease. These data indicate that active tissue remodeling is occurring in patients with CVD. Which matrix metalloproteinases are involved and how they are activated and regulated is currently unclear. It appears that MMP-2 may be activated by urokinase plasminogen activator (uPA). Herouy *et al.* observed increased uPA and uPAR mRNA and protein levels in venous ulcers compared to normal skin. Elevated levels of active TGF β -1 in the dermis of CVI patients suggest a regulatory role for TGF β -1 in MMP and TIMP synthesis and activity but this, needs to be verified by further studies.(26)

7 Diagnosis of venous ulcer

Clinical examination

Correct diagnosis in the venous ulcers should start with the medical history taking. Patient with venous ulcer has to go through detailed examination include family anamnesis taking and occupational history. Patient has to be asked if he had it in the past any DVT or has blood coagulation disorders. We should follow up the physical examination with the inspection both of the legs and leg palpation starts from the lower up identified. Superficially we inspect check for pigmentation, eczema presence, location of wound color, the depth, wound morphology, presence of erythema, infection. The palpation of the lower leg should be done in upright position, we need to have good blood return generally we inspect the legs both for presence of edema or lymphedema, in the palpation techniques we can palpate and identified the presence of dilatation VSM or if there is perforator insufficiency. We should continue the clinical examination asking the patient to perform Valsalva maneuver. In presence of venous insufficiency we can palpate the thrill. Later we have to put the patient at Trendelenburg position and Perth's test should be performed, with the use of esmar we can observe the venous return. All the upward finding has to be documented, and the wound should be photo document, and a wound measurement should be taken. (27)

7.1 C.E.A.P. Classification

The CEAP classification was published in the mid-1990s in 25 journals and books in 8 languages. Several revision by the ad hoc committee of the American Venous forum in conjunction with International ad hoc committee have resulted in the classification summarized below that has been adopted worldwide to facilitate meaningful communication about and description of all forms of CVD. The term CVD includes all morphological and functional abnormalities of the venous system in the lower limb. Some of these like telangiectasia are highly prevalent in the adult population and in many cases the use of the term disease is therefore,

inappropriate. The term chronic venous insufficiency CVI is entrenched in the literature and has been used to imply a functional abnormality (reflux) of the venous system and is usually reserved for patients with more advanced disease including those with edema C3, skin changes C4 or venous ulcers C5/C6. In the revised CEAP classification the previous overall structure of CEAP has been maintained but more precise definitions have been added. The following recommended definitions apply to the clinical C classes in CEAP.

Telangiectasia: a confluence of dilated intradermal venules of less than 1 mm in Caliber: Synonyms include spider veins, hyphen webs, and thread veins.

Reticular veins: dilated bluish subdermal veins usually from 1mm in diameter to less than 3mm in diameter. They are usually tortuous. This excludes normal visible veins in people with transparent skin. Synonyms include blue veins, subdermal varices, and venulectasies.

Varicose veins: subcutaneous dilated veins equal to or more than 3mm in diameter in the upright position. These may involve saphenous veins saphenous tributaries, or non –saphenous veins. Varicose veins are usually tortuous, but refluxing tubular saphenous veins may be classified as varicose veins. Synonyms include varix, varices, and varicosities.

Corona phlebectatica: this term describes a fan shaped pattern of numerous small intradermal veins on the medial or lateral aspects of the ankle and foot. This is commonly thought to be an early sign of advanced venous disease. Synonyms include malleolar flare and ankle flare.

Edema: This is defined as a perceptible increase in volume of fluid in the skin and subcutaneous tissue characterized by indentation with pressure. Venous edema usually occurs in the ankle region, but it may extend to the leg and foot.

Pigmentation: brownish darkening of the skin initiated by extravasated blood, which usually occurs in the ankle region but may extend to the leg and foot.

Eczema: Erythematous dermatitis, which may progress to a blistering, weeping or scaling eruption of the skin of the leg. It is often located near varicose veins but

may be located anywhere in the leg. Eczema is usually caused by CVD or by sensitization to local therapy.

Lipodermatosclerosis: LDS localized chronic inflammation and fibrosis of the skin and subcutaneous tissues sometimes associated with scarring or contracture of the Achilles tendon. LDS is sometimes preceded by diffuse inflammatory edema of the skin which may be painful and which is often referred to as hypodermis. This condition needs to be distinguished from lymphangitis, erysipelas or cellulitis by their characteristic local signs and systemic features. LDS is assign of severe chronic venous disease.

Atrophy Blanche or white atrophy: localized often circular whitish and atrophic skin areas surrounded by dilated capillary spots and sometimes with hyperpigmentation .This is a sign of severe chronic venous disease. Scars of healed ulceration are exclude from this definition.

Venous ulcers: full thickness defect of the skin most frequently at the ankle that fails to heal spontaneously sustained by CVD.(28)

C.E.A.P. classification of severity

Table 1. CEAP Classification - Clinical, Etiologic, Anatomic, Pathophysiologic

C-Clinical Class	Characteristics*	E-Etiology**
0	No clinical findings or symptoms	C Congenital
1	Telangiectasia or reticular veins	S Secondary
2	Varicose veins	P Primary
3	Edema, only due to a venous etiology	A-Anatomy**
4	(a) Pigmentation and/or eczema (b) Lipodermatosclerosis, atrophie blanche	S Superficial (Great and short saphenous systems as well as any branch varices)
5	Prior ulceration, dermatitis	P Perforator (Veins that communicate between the superficial and deep systems)
6	Active ulceration	D Deep (Calf veins and sinuses, popliteal, femoral, iliac veins and vena cava)
A, S	Subscript: Asymptomatic, Symptomatic	P-Pathophysiology**
Date	Date of investigation	R Reflux
Level	Level of investigation (I, II, III)	O Obstruction
		R-O Both
		N** No evident disease**

*Complaints are expected to be related to venous insufficiency and are not classified if another etiology is present (i.e. edema secondary to heart failure).

**The N subscript indicates no evidence of disease. It is applicable to E, A, and/or P of CEAP.



Class 1:
Telangiectasia.

Class 2:
Varicose vein.

Class 3:
Edema.

Class 4:
Pigmentation /
Eczema.

Class 5:
Healed Ulcer.

Class 6:
Venous Ulcer.

7.2 Diagnostic techniques

Venous wave form Doppler

Venous Doppler signals displays five characteristics. At the normal patient have a spontaneous signal over any vessel and the flow should be sound unidirectional. This signal are diminished in intensity during inspiration as descent of the diaphragm causes a rise in intraabdominal pressure, thus decreasing the venous outflow from the leg. It will be augmented similarly with exhalation .This waxing and waning of the intensity of the signal with the respiratory cycle is a phenomenon called phasicity. Venous signal are continuous except for their respiratory variation and are not pulsatile, except if there is a congestive heart failure or tricuspid valve insufficiency. Finally the most important is during leg

elevation of patients with varicose veins venous signal can be present under certain maneuvers. The response to these maneuvers is that what provides the presence of valvular insufficiency and obstruction on venous system. When we compressing the distal and proximal point of the vein the flow sound increase and shows no proximal obstruction. In the presence of the DVT the sound which response are weaker and delayed. When we examined the patient to the upright position release of compression should be followed by silence as the valves close in response to the downward pressure of the blood being pulled by gravity. With the patient at supine position and the release of compression should be normally be followed by the return of the lower intensity spontaneous or silence in the smaller veins. In the presence of valvular insufficiency at the level of the Doppler probe a loud reflux flow signal can be heard on the release of distal compression. This reflux flow also we can quantitate with the help of the pneumatic cuff and duration of the reflux may be read from the tracing obtained. To be considered true reflux and not merely delayed valve closure the duration of the valve should be 0.5 sec. The other method of augmentation is proximal compression and release. Proximal compression produces a transient obstruction to outflow and thus causes an accumulation of blood distally, with an associated interruption of the Doppler signal. During release the large blood flow created loud sound. This has also been found to be more sensitive maneuver in diagnosing the DVT ,even that limited to calf veins, with the diminished or delayed signal indicates of a significant thrombosis, Valvular insufficiency is discovered easily ,because proximal compression yields a loud reflux instead of silence.(29)

Photoplethysmatography PPG

The PPG is simple test are easy and quick to be performed, The principle are an infrared light source and sensor are attached adhesive to the medial aspect of the lower leg to the malleolus space. The infrared light travels to blood and the subdermal venous plexus and is absorbed by hemoglobin in red cell. During examination patient ask to performed dorsiflexion of the knee to activate the calf pump and produces outflow. The photoplethysmatography detects the blood volume and the conclusion of the exercise and provide the degree of reflux either

in deep or superficial system. Normal the blood circuit takes 20-25 sec to return a value less than 200 sec indicates a presence of the abnormal refilling channel namely retrograde flow through incompetent superficial or deep ve

Light Reflection rheography LRR

Light reflection is a form of PPG, The principle is that incorporates three infrared light beams and focus to cover the subcutaneous venous plexus. Dermal pigment such that commonly find to the patient with the chronic venous insufficiency is concentrated more to the superficial layer of the skin and interferes with the light transmit ions ,However this techniques are yielding inaccurate and has variable values. So the LRR is not considered sufficient to make correct diagnosis of the DVT and other test should be performed. (31)

Air Plethysmography

Air plethysmography is one technology that is just a simple to use and potentially supplies a great deal of additional information compared with the conventional PPG. This device consist a tubular polyvinyl- chloride air chamber that surrounds the leg from the knee to ankle. This is inflated to 6mmHg and connects to the pressure transducer an amplifier and a recorder .A smaller bag placed between the air chamber and the leg is used for calibration by injecting a certain volume of air and water and measuring the change in the records associated with the volume. This method has advancing ability to quantitate reflux with the VFI and determine the prognosis and its ability to measured calf muscle pump function through the determination of the EF. This technique should be combined with Doppler or duplex findings and clinical evaluation. However at the clinical practice has achive some validation studies but the test is limited to the laboratory use. But one study of the David Summers shows that plethysmographic measurements of functional venous parameters do not discriminate well between limbs with uncomplicated varicose veins and limbs with the ulcers or stasis dermatitis and the venous filling index correlates poorly with the presence of incompetent veins and their diameters. (32)

7.3 Ultrasound examination of vein ulcers

Duple ultrasound scanning is considered the test of the choice among different diagnostic tools for detailed evaluation of chronic venous insufficiency. It combines color flow imaging with B mode and pulse Doppler. It very well documented that duplex ultrasound scanning is the most reliable and cost effective, quick and noninvasive method to identify venous reflux .This type is also able to defect venous obstruction and assess recurrence of both reflux and thrombosis.

Most recent studies have shown that the superficial and perforating veins are involved in the majority of patients. However, several reports document that the type and duration of treatment and the rate of recurrence of venous ulcers are significantly different when the deep venous system is involved. Therefore, knowledge of the venous anatomy, pathology, and their variations is an important prerequisite for the person who performs the duplex ultrasound in order to identify precisely the etiology and the anatomic distribution of the venous malfunction in each patient.(33)

The investigation of the venous system in the vein ulcers has to be done at stand position and all the weight to the contralateral limb, the leg should be relaxed with the knee slightly flexed, examination at supine position is not recommended .In case of patient cannot stand has to be examined at sitting position.

For evaluation of deep and superficial system we use different transducers based on the depth of the location we will like to performed examination. At superficial veins within 1 cm in the subcutaneous fat can be evaluated with 10 MHz transducers. A 3 MHz transducers is used for evaluation of veins deeper than 6 cm.

The veins to be examined are identified with B mode imaging, manually we start from the groin by rapidly squeezing and suddenly releasing different muscle beds above and below the vein .At the groin we performed the Valsalva maneuver. In obese patient or in patient with edema, dorsal planta flexion can also be used, or the transducer has to swift at different frequencies or to adjust the depth. Manual

compression is used to demonstrate reflux by rapid squeezing and suddenly releasing different muscle beds above and below the vein in question. However when precise measurement are needed regarding flow or duration of reflux, then automatic rapid inflation-deflation cuffs should be used.

All main veins should always be imaged along the entire length. Non saphenous, accessory veins and major tributaries when incompetent should be followed, and their course should be noted. It is important, for the study to be reliable and accurate, that the examiner is aware of the venous anatomy and the different variations such as duplications, hypoplasia, and segmental aplasia. Duplication is usually seen in the popliteal vein in up to 40% of the limbs and in 25 % to 30% in the femoral vein. Duplication of the GSV and SSV are found in <3% while hypoplasia and aplasia are frequently found.

Throughout the exam, color flow imaging is used on top of the B mode ultrasound initially the artery is identified by a red color on the monitor. Then and while applying compression, the adjacent veins are identified by a blue color. Blue color indicates that flow is in the opposite direction than the flow in the artery. If no color is noticed in the vein after the release of the compression, then no reflux is present, implying the veins are competent. The appearance of the red color indicated the presence of reflux. The Doppler waveform will document the presence of reflux.

Retrograde flow behind the valves when is less than 0.5 second consider normal. Pathological it is when the flow last more than > 1,000 ms in the common femoral ,femoral and popliteal veins: >500 ms in the superficial veins ,deep femoral veins and deep calf veins and >350ms in the perforating veins. Reflux can be defined a single venous segment and multisegmental when it is confined to more than one venous segment.(34)

The examination has to include the following parts:

Superficial veins

Imaging of the GSV starts at the groin where the saphenofemoral junction SFJ is identified medial to the common femoral artery. The terminal valve in the GSV is located 1-2 mm distally. Between those two valves, there are tributaries that join the GSV, which are often associated with pathology. They can have reflux together with GSV or be independent to it. In cases of deep vein obstruction, they are dilated and act as collateral pathways. For example reflux in the accessory saphenous vein without the involvement of GSV occurs in about 9% of patients. This is important because only the accessory vein should be treated, and the GSV can be spared. In the thigh GSV is always identified in the saphenous eye, which the fascial sheets create. The upper echogenic layer seen in a transverse scan is the superficial fascia and the lower echogenic layer is the muscular fascia. Any vein outside the saphenous eye is not the GSV, but an accessory saphenous or tributary. In the knee area it is difficult to recognize the saphenous eye and the GSV is usually identified in a triangle formed by the tibia medial gastrocnemius muscle and fascial sheet. Occasionally a large tributary arises above the knee and may be mistakenly identified as the GSV. In general the GSV in the thigh and the leg is often followed by parallel veins that may be confused with the GSV. The anterior and medial accessory veins in the thigh and the posterior and anterior arch vein in the calf are the tributaries most commonly incompetent. (35)

The SSV is found at the popliteal fossa within the triangular fascia over the medial and lateral heads of the gastrocnemius muscle. The vein lies in the leg in a fascial compartment that is identified with the ultrasound as an eye similar to the GSV eye. The SSV may terminate at different levels of the popliteal vein is now collectively termed as thigh extension of SSV. Reflux in non-saphenous veins is found in 10% of the patients with CVD. The location and origin of these patients are usually multiparous women. The location and origin of these veins are variable. Often they are found as an extension of the pelvic veins are variable. Often they are found as an extension of the pelvic veins in the thigh vulvar and gluteal, lateral and posterior thigh, popliteal fossa. About 90% of these patients present with CVD classes 1 to 3 and skin damage is present in 10%. Inaccurate clinical evaluation has been found in one third of cases with this type of reflux.

Therefore DU is very important in identifying reflux in these veins because there are many different ones and there is great variability in the patterns of reflux. (36)

Perforating veins

The role of the perforator veins is controversial in the development of venous ulcers in patient with CVD. They are seen in different planes with the ultrasound because the direction of flow is different planes with the ultrasound because the direction of flow is different in those veins. Transverse and oblique scanning along the course of the superficial veins is more appropriate. The fascia has to be identified by the examiner. The fascia appears white sue to high collagen content and the perforators are identified as they travel though the fascia. About 150 perforating veins have been identified and they are grouped based on their location. However only about 20 of them have been shown to become incompetent and are frequently found in patients with ulceration. Most of these veins are found in the medial and posterior calf, and they are connecting tributaries of the GSV and SSV with muscular and deep axial veins. The examination starts from the medial malleolus going upward to the knee. The posterior arch and GSV are scanned on a transverse plane for incompetent perforator veins. Localized varicose veins are highly suspicious for an underlying incompetent perforator, and that region requires detailed evaluation. Then the SSV is tested starting from lateral malleolus and ending at its termination in the fossa or higher up. In order to determine normal valvular function, distal compression is applied for augmentation of flow, and the perforators are tested in combination with deep and superficial veins. The flow in the calf perforator veins may be bidirectional .The net outward flow from deep to superficial veins is measured to determine reflux. In patients with significant edema, dorsi /plantar flexion is performed to determine reflux in these veins. This is important because manual compression alone may not show reflux in dilated perforator veins. (37)

Deep veins

The common femoral veins is tested from the inguinal ligament to its union with the femoral and the deep femoral veins. Any evidence of continuous flow, poor augmentation, and asymmetrical waveforms to the contralateral CFV should alert the examiner to extend the imaging in the iliac veins and the inferior vena cava. The CFV should also be examined above and below the SFJ in order to determine the presence or absence of reflux at the SFJ in order to determine the presence or absence of reflux at the SFJ level and test the effect of the GSV on the CFV. The femoropopliteal veins are examined along their length from the groin to the tibioperoneal trunk. In the calf the posterior tibial, peroneal and soleal veins are imaged because the frequency of deep vein thrombosis in these veins is high, and it turn can lead to PTS reflux. The anterior tibial veins are imaged in the presence of CVD signs and symptoms in the anterior aspect of the calf. (38)

Ulcer Bed

The ulcer bed should always be evaluated for incompetent veins including the area surrounding the ulcer as far as 2 cm from the periphery of the ulcer .In ulcers over the medial malleolus, the veins that need to be tested are the GSV, the posterior arch ,posterior tibial and peroneal veins.in ulcers on the anterior tibia veins need to be assessed .Sterile technique can be used for the evaluation of the ulcer bed. The surface of the ulcer may be covered with a transparent sterile dressing, or the probe can be covered with a sterile glove that is filled with ultrasonic gel. The prevalence of deep venous reflux has been found to be higher in patients with venous ulcers when compared to patients with less sever forms of CVD. In addition, it was found that the ration of outward flow in these veins is longer in patients with ulcers compared to patients without ulcers. (39)

7.4 Invasive Diagnostic Techniques

Venography

Venography is invasive and to a large extent has been superseded by duplex scanning for the investigations of venous disease. Occasionally MR or VT venography are required .The venography can provide useful anatomical and functional information, particularly when the duplex findings are equivocal or

duplex is limited. Varicography ascending and descending venography may be used in investigation of VUs. (40)

8 Differential diagnosis of leg ulcers

Introduction

The term ulcer is a general use term but the symptomatology can be from different origin. The majority of the leg ulcer presented at the leg or foot. Non-venous ulcers the localization area at the foot are most of the time. At Koerber study from 354 legs they found 75% was venous leg ulcers, 3,66% venous leg ulcers, 14,66% mixed venous or arterial, and 13,5% vasculitic ulcers, in all studies diabetics ulcers was excluded. Statistically the venous ulcers affects mostly women population at 62% and male in 52%. All ulcers need to be clinically and anamnestically examined first and later has to be investigated with the Duplex ultrasound and exclude vascular occlusive disease venous or arterial occlusive disease. Skin biopsies should be of concern to the non-healing ulcers as a diagnostic tool to find the origin of them. (41)

8.1 Arterial ulcers

Arterial ulcers are caused by deficit of oxygen supply in the tissue due to arterial occlusions. The most frequent cause is PAOD arteriosclerosis changes in the arterial wall causes occlusion to the blood arteries. Arterial ulcers appear necrotic and mainly present on the toes, foot or leg may occur in the advanced stage of Fontaine stages IV. In Fontaine stage III stage patient suffers from resting pain or nocturnal pain but we don't have any loss of the tissue. The Fontaine II stage are accompanied with the clinical picture of intermittent claudication and sometimes can be together with chronic venous insufficiency, but during the rest pain is missing.

The arterial ulcers usually localized in the foot and toe region and in the advanced stages can be also at the malleolar region. These ulcers are very painful and have the appearance of black necrotic areas. (42)

Thromboangiitis obliterans is another cause of the arterial ulcers. It's an inflammatory process disease of the small arteries and veins, by thrombotic occlusion. This has clinical appearance mostly necrotic in the feet and may occur. Arteriovenous fistula is another rare cause of the chronic ischemic leg ulcer.

Hafner et al. all proposed a concept of ischemic subcutaneous arteriosclerosis. Hyperesthetic subcutaneous atherosclerosis may lead to different but similar patterns of ischemic skin necrosis. This includes Martorell's hypertensive ischemic leg ulcers, proximal non uremic calciphylaxis in the normal renal and parathyroid function, distal calciphylaxis in the end stage of renal insufficiency. The Martorell's ulcer skin necrosis localized in the latero-dorsal leg are typically clinical findings. Sometimes these ulcers have to be not misdiagnosed with the pyoderma gangrenosum. (43)



Arterial ulcer own material

8.2 Mixed leg ulcers

Mixed leg ulcer there ulcer of combined and venous etiology there are present around 15% of all the ulcer population. In the majority of the case primary cases are venous origin and the arterial occlusive disease complicating the healing. This

type of ulcers typically present at the older population due to lack of mobility and later on the arterial claudication maybe overseen? The ulcers has typical signs of the venous disease usually are big circular and may have black necrotic areas in addition. We treat this ulcers with the correction of venous insufficiency with surgery and in the same time we can combined with arterial recanalization usually with the endovascular procedures. (44)

8.3 Diabetic leg ulcers

Diabetic mellitus is a risk factor for PAOD. Despite this specific ulcers may occur in diabetic patients. Diabetic microangiopathy may lead to occlusions of small vessels mainly in the toes and consecutive toe necrosis with risk of bacterial infection and later on gangrene. The diabetes also can cause diabetic neuropathy with loss of sensation and ulcerations present in the pressure areas. The neuropathy involves the skin regulation, skin physiology, skin sensitivity and also muscle innervation of the foot. Neuropathy results to pressure distribution in the foot pressure areas. The neuropathic ulceration usually located at pressure zones showing hyperkeratosis surround the ulcer. Sensory loss is also typical in the neuropathic ulcers .A foot ulcer without hyperkeratosis can be another non-neuropathic etiology and should be taken skin biopsies. The histology appearance can show the necrobiosis Lipolitika it's a disease usually occurs in the patient with diabetes but can be also to the patient without. It an inflammation of the skin, and the blood vessel are occlude. The most frequent location is the anterior lower leg ulceration in most of the cases. (45-46)



Diabetic foot own material

8.4 Vasculitic leg ulcers

A big part of the inhomogeneous leg ulcer can be called vasculitis. The Chapel Hill classification distinguishes between large, medium and small vessel vasculitis. In the skin leucocytoclastic small vessel vasculitis with purpura can present in the skin. Multiple skin necrotic ulcers may also have origin vasculitis. Vasculitic ulcers can be present in patients with autoimmune disease or patients with hepatitis C or with myeloma. Another etiology for ulcer can be rheumatoid arthritis for example. Cryofibrinogenemia. Pyoderma gangrenosum can be a specific form of the ulceration with non-leukocytic vasculitis and can be caused due to an auto-inflammatory process. These ulcers are often associated with the inflammatory border of the skin necrosis and usually are due to chronic inflammation. Systemic disease for example ulcerative colitis. Pyoderma gangrenosum also can be occurred after trauma or operation. As all the other vasculitic ulcer has good response to immunosuppressive treatment. (47)



Pyoderma Gangrenosum own material

8.5 Leg ulcer of other etiology

Leg ulcers can occur in patients with Klinefelter's syndrome. These patients with hypogonadism and testosterone deficit slow healing ulcers may occur with or without chronic venous disease. Other etiology can be ulcers caused by infection may include ectymata caused by bacterial infection of the skin, necrotic cellulitis, cutaneous necrosis or subcutaneous leishmaniasis and other infections. Some drugs also cause ulceration the most common is the side effects of hydroxyurea treatment in myeloproliferative disease. Hydroxyurea treatment can cause hyperpigmentation and later ulceration, which can be linked to chronic venous disease.

The most important differential diagnosis of the leg ulcers are ulceration caused by malignant or benign origin. Tumors like Basal squamous cell carcinoma can arise like a complication of the chronic ulceration. In conclusion, each leg ulcer with no tendency to heal should be considered for skin biopsies in order to exclude tumor or tumor transformation.

9 Non operative management of venous ulcers

9.1 Pharmacotherapy

Venoactive (VAD) drugs are a heterogenic group of drugs from vegetal or synthetic trials. Numerous controlled studies demonstrates the anti-edematous effect and the effect to attenuate of symptoms of CVD such as heavy legs, pain and restless legs VAD drugs established in the marked for the all stages of CVD. Some of VAD shows may accentuate the effects of compression and some can accelerate healing of vein ulcers. (48)

Mode of action

VADs have two pathophysiological mechanism of action .They alter macro circulatory changes in the venous wall and venous valves that cause hemodynamic disturbances to produce venous hypertension and they alter microcirculatory effects of venous hypertension that lead to venous microangiopathy. The mode of action varies depending of the drug product. (49)

Classification of the main Venoactive drugs.

Group	Substance	Origin	Dosage	Number of doses
Benzopyrones	Coumarin	Melilot	90 combined with troxerutin	3
Alpha Benzopyrones		Woodruff		
Benzopyrones		Citrus sp.		
Gamma-benzopyrones	Diosmin	Citrus spp.	300-600	1or 2
	Micronized purified flavonoid fraction	Rutaceae aurantiae	1000	1 or 2
	Rutin and rutosides	Sophora japonica	1000	1 or 2
Saponins	Escin	Horse chestnut	Initially 120 then 60	3
	Ruscus extract	Butcher broom	2 to 3 tables	2 to3
Other plant extracts	Anthocynas	Bilbery	116	2
	Proanthocynidins	Martitime pine	100-300	1 to 3
	Extracts Gingo,hetpaminol and troxerutin	Ginko biloba	2 sachets	2
Synthetic products	Calcium dobesilate	Synthetic	1000 to 1500	2 to 3
	Benzaron	Synthetic	400 to 600	2 to 3
	Naftazon	Synthetic	30	1

Action at the macrocirculation level

Till recently the most popular theory was that weakness of the vein wall produced venous dilatation causing secondary valvular incompetence. For this reason, research on VAD was focus only the effects on venous tones. Most of VADS have been shown to increase venous tone by a mechanism related to the noradrenaline pathway. Micronized purified flavonoid fraction (MPFF) prolongs noradrenergic activity, hydroxyethylrutosides act by blocking inactivation of noradrenergic and

ruscus extracts act by agonist on venous α_1 adrenergic receptors. A high affinity for the venous wall was found for MPFF and hydroxyethylrutosides. The precise mechanism by which other drugs increase venous tone is not known. More recently it has been realized that chronic venous disease is related to primary failure of venous valves that affected by inflammation. The available drugs have been shown to attenuate various elements of the inflammatory cascade particularly the leucocyte endothelial interactions that are important in many aspects of the disease. Results of the recent trial shown an anti-inflammatory effect under this acute situation that may result in protection of venous valves in chronic conditions. Action on the microcirculation VAD effects on capillary resistance, lymphatic drainage, protection against inflammation, and blood flow.(50)

Action of Venoactive drugs on venous tone and the venous wall

Group	Compound	Effect on venous tone	Effect on venous wall and venous valve
Benzopyrones	Micrinised purified flavonoid fraction	Increase venous tone by prolonging noradrenergic activity	Protects human saphenous endothelial cells from hypoxia.
	Rutin and rutosides	High affinity for venous wall increase venous tone	---
Association of a benzopyrones and γ -benzopyrones	Coumarine and rutin	Increase of venous flow	
Saponins	Escin	Increases venous wall tone	Protects human saphenous endothelial cells from hypoxia
	Ruscus extract	Venoconstrictive effect and by agonism on venous α_1 adrenergic receptors	Protects human saphenous endothelial cells from hypoxia
Other plant extracts	Proanthocyanidines	-----	Protect endothelial cells against hypoxia
	Pycnogenol	-----	
	Ginkgo biloba	-----	Protects human saphenous endothelial cells from hypoxia
Synthetic products	Calcium dobesilate Naftazone	Increases venous tone	Accelerates endothelial cells proliferation

Capillary resistance:

Numerous studies have shown that VAD are able to increase capillary resistance and reduce capillary filtration. This seen for MPFF rutosides, escin ruscus extracts, proanthocyanides and calcium dobesilate. The capillary protective effect of MPFR may be related to inhibition of leukocyte adhesion to capillaries. This is enhanced micronization. (51)

Lymphatic drainage

The efficacy of coumarin on lymphedema has been described by Casley Smith .Coumarin combined with rutin reduce high protein edema by stimulating proteolysis. MPFF improves lymphatic vessels and calcium dobesilate enhances lymphatic drainage. (52)

Protection against Inflammation

In animal models of skin inflammation VAD appear to attenuate the inflammatory response by various mechanisms. Numerous reports have confirmed free radical scavenging, anti-elastase and anti-hyaluronidase properties of most VADs.(53)

Hemorrhheological disorders

Hemorrhheological disorders changes are constant in CVD appearing as basic trait with increased blood viscosity increase with greater severity of disease. Some VADs limit red blood cell aggregation (ex Gingo, biloba) and decrease blood viscosity MPFF and increase red cell velocity.(54)

Pharmacological treatment in leg ulcers.

Healing the venous leg ulcers CEAP C6 has been shown to be accelerated in several studies using micronized purified flavonoid fractions MPFF. This was confirmed in 2005 by meta-analysis of 5 trial using MPFF as an adjuvant to standard treatment in 723 patients of CEAP c6 classification. Among VADs, the use of horse chestnut seed extract or of hydroxyrutosides failed to demonstrate superiority over compression in advanced chronic venous insufficiency or in preventing venous ulcers recurrence. A small number of other drugs have been seen used with varying success. Stanazolol a fibrinolytic anabolic steroid was expected to break down pericapillary fibrin cuffs but did not increase the ratio of ulcer healing. Abnormalities of coagulation observed in patients with venous

disease have been improved by aspirin but there is a lack of data supporting its use for preventing thromboembolic events in patients with CVD. A thromboxane receptor antagonist (Ifertoban) failed to show benefit over compression therapy in ulcer healing. Several trials have suggested that pentoxifylline may improve venous healing ulcer rates, although the effects appears to be small and the role in patient remain unclear. (55)

Pentoxifylline

Pentoxifylline is a vasoactive drug that reduces leucocyte adhesion and has rheological action on erythrocytes and a mild fibrinolytic action. In a systematic study by Jull et al comparing pentoxifylline or placebo either associated with the placebo or not .They conclude to that our results suggest that pentoxifylline gives additional benefits to compression for venous leg ulcers and possibly effective for patients not receiving compression.(56)

Sulodexide

Sulodexide in the study of M. Kuchareyewski shows the influence of sulodexide in treatment of venous leg ulcers with combination of Unna's boot and surgical treatment. In this study show the venous ulcers healing in 7 weeks in the 35% of the patient and in 10 weeks for the rest population, concludes that the healing process under sulodexide treatment accelerates the healing process. (57)

9.2 Compression therapy in venous leg ulcers

Introduction

Compression therapy is the basic treatment modality in venous leg ulcers that has been shown to be effective for healing ulcers and also for keeping them healed. In every single case, the underlying venous pathology has to be ascertained, preferably by duplex investigation, and methods to correct pathophysiology by surgery or sclerotherapy have to be considered.

Medical compression stockings

Medical compression stockings exerting interface pressure in the gaiter are up to 40 mmHg are effective in preventing and reducing edema. For treating venous ulcers, the use of compression stocking may be considered, if the ulcers are not too large (more than 5 sq cm) and not long standing (history shorter than 3 months). The product has to be able to handle from unskilled elderly patient and has the ability to produce constant pressure for three months and allows the patient to change dressings, clean the ulcer. One of the disadvantages is that the exudates may soil the stocking which then has to be washed and this may weaken. Recently controlled randomized trials report favorable results of ulcers healing using different compression stockings. Usually we prefer to use the double layer stocking the light compression stocking may be kept the local dress in place. A class II compression stocking adds the pressure and increase the stiffness of the kit, several kits are available (ulcer X kit Sigvaris). The basic line also can be kept during the treatment overnight and allows the patient to clean the wound daily.

After the ulcers healed compression double layer stocking are important to be used to prevent recurrences, we can use modality of compression techniques but we preferably to be class II stocking and has a pressure in the ankle between 15-20 mmHg of mercury, (medium or moderate class) specialized do not injure the healed ulcer- and lipodermatosclerosis.(58)

Compression class	EU	USA	UK	France	Germany
A	Light 10-14				
I	15-21 (mild)	15-20 (moderate)	14-17 (light)	10-15	18-21 (light)
II	23-22 (moderate)	20-30 (firm)	18-24 (medium)	15-20	23-22 (medium)
III	34-46 (Strong)	30-40 (extra firm)	25-34 (strong)	20-36	34-46 (strong)

IV	>49 (very strong)	40+		>36	>49 (very strong)
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9.3 Compression bandages

For the routine management of the venous ulcers, stockings cannot replace compression bandages, we have two categories of the elastic and inelastic bandages, both of techniques are based on the stiffness and the elasticity of the material used.

Elastic and inelastic bandages

- **Elastic compression bandages:** Bandages that incorporate materials which exert pressure when they are applied with stretch.
- **Inelastic compression bandages:** Bandages that exert pressure which increases when movement causes the calf muscle to contract.

Elastic long stretch material is relatively easy to handle and can be used also by the patients. In contrast to the inelastic material these bandages produce active force by the elastic constriction of their fibers. Therefore such bandages or compression stockings may cause pain and discomfort when the patient sits or lies down, especially when they have applied too tightly. Single component elastic bandages or compression stockings are therefore applied in the morning, preferably before getting up and they are removed before bed at night. During walking, the peak pressure waves are lower than with inelastic material.

Inelastic material produces a much higher increase when the patient is standing up and performing dorsiflexion than does the elastic material. The pressure increase from standing up and the amplitudes during ankle movement are useful parameters for characterization. For a practical differentiation between elastic and inelastic, it has to be defined the difference between standing and supine pressure measured with the small transducer called Static stiffness index. Values are higher

than 10 is indicate of inelastic material, lower than 10 indicates elastic material. A good compression bandage is characterized by well tolerated resting pressure and high pressure peaks during walking. An inelastic bandage achieving pressure peaks of 80 mmHg will compress superficial and deep veins intermittently. This can be demonstrated by compressing the leg with blood pressure cuffs containing an ultrasound permeable window. Using a duplex instrument, it can be demonstrated that in the upright position a pressure of 40-60 mmHg will narrow the leg veins. Dorsiflexion leading to intermittent pressure peaks of 60-80 mmHg causes intermittent occlusion of the veins.

Two main disadvantages of inelastic bandages have to be considered:

- One is the loss of bandage pressure starting immediately after bandage application. After only 1 hour, the initial resting pressure will drop by about 25%, mainly due to an immediate decrease of the volume of the limb.
 - The second disadvantage is the fact that the application of a good inelastic compression bandage is not easy and easy and should be learned and trained. Due to the fast loss of pressure, bandages with inelastic material should be applied with much higher initial tension than elastic bandages. An inadequate bandaging technique with inelastic bandages is likely the main reason for the poorer clinical outcome described in some studies.
- (59)

Single layer and multilayer bandages

The single layer bandages are applied with the overlap of about 50%.The multilayer compression therapy are more effective in the treatment of venous ulcer, multilayer bandage can be consist from one or several component with different compression materials.

Multiple component bandages

Bandages consisting with different materials and called component bandages., usually the companies sell in kits, has to stressed that multiple layers will not only

increase the pressure but it will also change the elastic property toward stiffer final bandage.(60)

Unna's boot bandages

Zinc bandages are examples of completely non stretch material with high stiffness that may remain on the leg for several days up to 2 weeks. During the use of bandages we have reduced the edema and leg circumference this bandages cause a high pressure around 60 mmHg of and increase the strength as the ankle.



Unna's boot own material

Velcro band – Circ aid

Consisting of completely no stretch material, can be applied by instructed patient, pressure can regulate according to need.



Velcro band Circ aid, Medi company own material

Putter bandage

Bandage system consists of several layers of inelastic cotton bandages, is an example of a multilayer single compression bandage. Usually they cover the lower leg and create gradient pressure to the legs.

Prevention of ulcer Recurrences

The leg ulcer management has to be always consist in two phase

1. The healing phase till epithelization
2. The maintenance phase after ulcer healing, and recurrence should be prevented.

In general it is often easier to heal a venous ulcer than to keep it healed. To keep ulcer healed, continuous compression is essential (maintenance phase). Medical compression below-knee stocking 30-45 mmHg, are the preferred method of choice. Patients who are unable to put on the stocking may use elastic bandages instead. Eradication of venous refluxes by surgery or sclerotherapy should be considered in every single patient. (61)

9.4 VAC therapy Negative pressure in ulcers

There are more 80% of ulcers which are venous origin they remain a big socioeconomic problem and in USA alone it cost billions of USA dollars to treat them. Management of these wounds has been problem in medicine for many years. The therapy of debridement of necrotic tissue utilization gauze, leg

elevation, compression medicated wraps and various wound solutions, even with a proper use all these therapy modality still remain a problematic to heal venous ulcers.



V.A.C. system own material

VAC therapy is one of the most modern way to treat wounds, both chronic and acute by use of topical negative pressure. The topical negative pressure sets are consists of an open cell polyurethane sponge with 400-600 μm pores, a transparent adhesive coverings, no collapsing tube and vacuum generating device with the collection reservoir. This device of negative pressure has the ability to provide a wide range of negative pressure and is capable to maintain continuous or intermittent suction. Normal wound healing process consist of healing stages: migrations of cells such as macrophages, fibroblasts and epithelial cells, removal of debris, decrease bacterial burden, angiogenesis, formation of granulation tissue, wound contraction, production of connective tissue. The negative pressure system can enhance the wound healing process needs. Still we don't know the exactly mechanism of action but several trials present that the topical negative pressure has som benefits decrease of edema and increase of local blood flow, decrease of bacterial burden and cause mechanical deformation of wound bed leading to improve the wound healing. It decrease the chronic fluid and has property to mainatnce3 moisture the wound. All these properties effect and

promotes the wound healing increase the cell division and proliferation , an increase the matrix synthesis, and increase the wound healing process.(62)

9.5 Prevention of Post thrombotic chronic disease

CVD is either primary or secondary .Science has not advance to the point where can effectively prevent primary venous disease although it has clarified much of the pathophysiology of secondary CVD. Treatment modalities have demonstrated that the virulence of post-thrombotic CVD can be substantially reduced and in many case avoided. In most of the cases, this must be achieved at the time the patient is managed for acute deep venous thrombosis.

Anticoagulation treatment is the main therapy for acute DVT. Establishing and maintaining a therapeutic level of anticoagulation is important for the best management and to reduce recurrence. This is critical to reduce the severity of DVT increases the like hood of PTS, (43)Randomized trials have shown that the longer the duration of anticoagulation the fewer are the episodes of recurrence.

In addition to anticoagulation ,randomized trials have shown that lower leg compression stocking with an ankle pressure of 30-40 mmHg significantly reduce the severity of the PTS, (41.45.771).The pathophysiology underlying are the CVD is ambulatory venous hypertension. Its two components are venous obstruction and valvular incompetence are found to be present both in the severe PTS syndrome. Although the recanalization of thrombosed venous segment happens may be only a fraction of the original luminal diameter, through that obstruction may not seen in the rest but during the exercise can be present pain or venous claudication.

A study(19) demonstrated the valvular incompetent develops progressively from the time of acute DVT. This study observed that valvular incompetence was more liked to develop in patient with occlusive rather than non- occlusive DVT, and is more likely to occur in the extensive thrombosis.

In other study patient treated for acute DVT with anticoagulation it was found that patient who preserved their valvular function had early lysis of their previously thrombosed veins. Therefore the natural history studies of acute DVT indicate the persistent obstruction increase the severity of the PTS and early clot lysis not eliminate the obstruction but also reduce the severity. In Scandinavian studies patient whose went under treated only anticoagulation drugs versus patient with thrombectomy plus AV fistula had it significant benefit in the follow up treatment.

Successful fibrinolytic therapy for acute DVT may reduce or avoid postthrombotic CVD .From 1990 the catheter directed intra- thrombus thrombolysis is accepted for the treatment of DVT especially with iliofemoral DVT .Large studies performed showed successful outcome in 80-90% ,this patients went under lysis had it vein valves preserved and was functionally. An important fact to improve ultimate success of the thrombolytic therapy is to correct underlying venous stenosis by venous to allow unobstructed venous drainage into the venous cava. Additionally the patient have to be under long term anticoagulation therapeutic treatment to prevent re-thrombosis. Ineffective anticoagulation may lead to recurrent DVT and eliminate the long term benefits of lytic therapy. (63-64)

10 Surgical treatment

The operation techniques in venous ulcers can be divided in four parts

1. Surgical interruption of superficial and perforators communicate veins of the ulcer.
2. Reconstruction or and transplantation of deep venous valves
3. Surgical debridement
4. Subfascial therapy.

10.1 Surgery in superficial varicose veins and surgical ligation of perforators

Surgery crosssection- stripping

The surgery has a goal to relieve presenting symptoms, prevent adverse effects of continuous venous hypertension, and normalize venous physiology by eradicating main stem reflux and removing visible varices. In patients with superficial reflux C2-C6 CVD flush ligation and division of the saphenofemoral junction SFJ combined with the stripping of the GSV to the knee is superior to SFJ ligation alone. Treatment of the superficial reflux involving the SSV usually involves saphenopopliteal junction SPJ ligation and division following pre-operative duplex marking. Stripping of the SSV and the GSV below knee reduces VV recurrence but are associated with the increased risk of sural or saphenous nerve injury .Remaining GSV and SSV varices can be either excised by phlebectomies. (63)



Crossectomy own material

Surgical ligation perforating veins Lintons,

Surgical we can interrupted perforating veins is to be performed ad minimally invasive approach is preferred to reduced morbidity and delayed wound healing and infection, but no consensus as to be the best technique. Paratibial fasciotomy to access the deep compartment is required .There is currently no consensus for these method showing any results preventing CVU.

10.2 Deep vein valve reconstruction in severe venous insufficiency

This is an operative technique which has to be reserved for patient with advanced CVI, especially with C5-C6 changes. Before the operation a good physical examination should be consider with color duplex ultrasounds screening superficial and deep venous system and deep valve reflux and hemodynamics should be monitored .Any previous DVT, and PTS should be documented in the anamnesis. Previous PTS is the result of deep venous thrombosis and the subsequent inflammation of the valve cusps and vein during the process of recanalization. This leads to scaring and shortening of the cusps which turns results in failure o the cusps to achieve normal coaptation and attendant the reflux. Reflux secondary to the PTS in not amenable to direct surgical repair of the

damaged or destroyed valve. Primary reflux is the result of structural abnormalities in the vein wall and valve itself. Redundant, malopposed cusps and venous dilation permit valve prolapse and reflux. In primary reflux, unlike PTS, there is no evidence of the previous thrombosis or inflammation near the valve. It is possible however to have both proximal primary reflux and distal PTS in the same leg. A rare cause of congenital reflux is the complete or partial absence of valves secondary to agenesis.

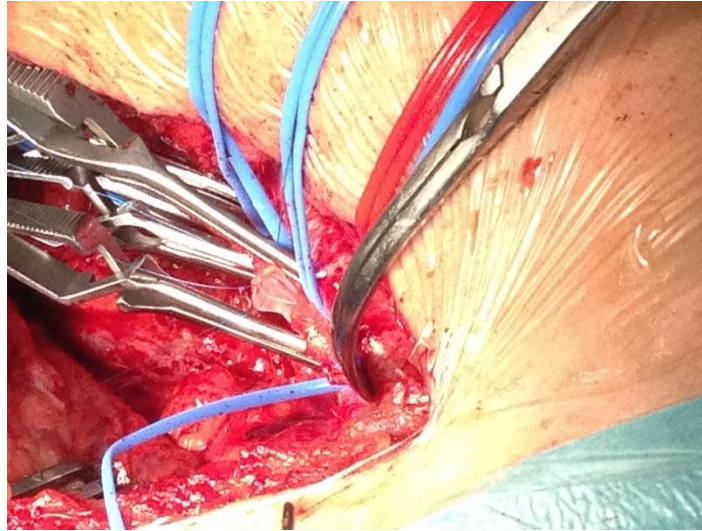
The goal of therapy is to correct the deep venous reflux (DVR) below the level of the inguinal ligament. Infrainguinal reflux leads to permanent venous hypertension, unmitigated by the activity of the calf venomuscular pump. It is important to note however, that all these abnormalities must be addressed in order to correct the venous hypertension

The DVR repair techniques are classified in two groups¹. Those that involve phlebectomy and those that do not involve phlebectomy.

Techniques with phlebectomy.

Internal Valvuloplasty

The first procedure using a longitudinal phlebectomy described by Kistener in 1968 and various procedures have been proposed. The method in all case is the redundant valve cup is applicate to the vein wall using multiple interrupted od continuous 7-0 Prolene reefing sutures. It has been estimated that plication of the leaflet length should restore competence although the best gauge remains visual inspection. (64)



Own material, Raju angioplasty courtesy of Roman Slysco MUDr.,Phd,MPH

Venous segment transfer

The venous segment transfer procedure was designed for patient with a competent great saphenous or deep venous femoral valve in their proximal segment and DVR in the femoropopliteal axis related to PTS changes. The propose of the venous segment transfer is to transpose a component valve bearing venous segments into the axial deep venous system. Several surgical variations of venous segments transfer have been employed using end to end techniques or side to end anastomosis, depending on the competence of the various valves in the veins of the inguinal area. Unfortunately this technique ca be used only in the 20% of patients with PTS because the remaining 80% do not have a competent valve in the proximal segment of the great saphenous or deep femoral vein.

Vein valve transplantation

A transplantation of 2-3 cm segment of the axillary vein is inserted as an interposition graft at the termination of the femoral vein just below the junction of the deep femoral vein with the femoral vein or at popliteal vein.

Neo valve

Various technique for creating a neo valve have been developed .Plagnol constructed a neo bicuspid valve by invagination using the termination of the

great saphenous veins, while Maleti created a valvular cusp by dissecting the femoral venous wall to obtain a single or a bicuspid valve ,these techniques have been used in PTS.(65)

Allograft cryopreserved valve.

Femoral vein allografts with competent valves removed from qualified donors have been prepared, stored and implanted in appropriate hosts.

Techniques without phlebectomy.

Wrapping banding, cuffing and external stenting

Wrapping, banding, cuffing, and external stenting techniques were initially developed to treat saphenous incompetence but later applied to the treatment of the primary deep veins incompetence.(66)

External valvuloplasty

The first step in external valvuloplasty consist in adventitial dissection until the valves insertion lines are clearly identified as an inverted V. The commissural angle is normally acute but is widened in a refluxing valve.

Transmural Valvuloplasty

Kistner introduced external valvuloplasty in 1990 .In the transmural valvuloplasty technique, an external row of sutures is placed along the diverging margins of the valve cusps in the vein wall. Sutures for external repair are begun at each commissure on both sides of the vein. The interrupted sutures are carried inferiorly until the valve becomes competent by strip testing.

Transcommisural valvuloplasty

Transcommisural valvuloplasty, developed by Raju, differs from transmural valvuloplasty by the use of a transmural suture. A through and through transmural resuspension suture (7-0 Prolene) is placed obliquely across the inserted commissural V, traversing the valve cusps blindly near their wall attachment to pull them up.

Angioscopy assisted external valve repair

In angioscopy assisted external valve repair, an angioscope is introduced through a saphenous side branch and advanced into the proximal femoral vein and positioned directly above the valve. Blood is cleared from the operative field by isolation with vascular clamps and a heparinized solution is infused through the angioscope. Prolene sutures are passed from outside to inside lumen through the angioscope. Prolene suture are passed from outside to inside the lumen, directed

by video – enhanced, magnified angioscopic imaging, allowing for precise approximation of the valve cusps. (67)

10.3 Endovascular techniques EVLA –SEPS

Sub-fascial endoscopic perforator surgery (SEPS)

Numerous uncontrolled series have suggested that SEPS might have beneficial effect upon the natural history of CVD and in particular in chronic venous ulceration. However it is not clear the benefits of SEPS procedures versus saphenous surgery undertaken in most of patients. In addition, it has been suggested by data from uncontrolled series that deep venous reflux might diminish the benefits of SEPS although this has not been a universal finding. In patients with deep post thrombotic or occlusive venous disease, results SEPS in terms of ulcer healing and recurrence in the uncontrolled NASEPS registry have been similar to those that might be expected from compression bandaging alone. The performance of SEPS in patients with PTS syndrome remains controversial.

It has never been shown that interrupting perforators in addition to standard saphenous surgery confers additional benefit in patient with CEAP C2 disease in terms of symptomatic relief, hemodynamic improvement and quality of life or recurrence. This may be because in the absence of deep venous reflux will result in most incompetent perforators regaining competence.

Furthermore, there is no evidence that addition of perforator surgery to standard saphenous surgery confers additional benefit in patients with CEAP C4-6 disease in terms of symptoms relief, hemodynamic improvement, and quality of life ulcer healing or recurrence. This may be because appropriate sub groups that might benefit have not yet been defined. A prospective randomized multicenter trial was conducted to study if ambulatory compression therapy with venous surgery including SEPS and superficial vein ligation was better treatment than compression therapy alone for patients with venous leg ulcers. There was no significant difference in healing rates between the two groups and recurrent ulcers or medially located ulcers in the surgical group had longer ulcer free period than those treated conservatively. (68)

10.4 Endovenous ablation procedures- cyanoacrylate procedures

Introduction

Various electrosurgical devices has been used in a favor to develop minimum invasive techniques for treating varicose veins. In a study by Politowski (527) under Tumescant and ultrasound guidance they use the laser monopolar energy via extra venous or intravenous route. But they has some serious issues with full thickens skin burns, saphenous nerve injury and recurrence were common postoperative complication.

More recently radiofrequency RF ablation using bipolar energy has evolved for endoluminal obliteration of GSV reflux. With growing experience RF can also be used to treat refluxing side branches of the GSV and recurrent varicose veins where an incompetent GSV persists. A new RF catheter named Closure fast in available to the treatment. In addition the laser 810 nm diode laser was approved followed by new laser 940,980 1064 and 1320 nm lasers. In the treatment of varicose veins both of the procedures are used in conjunction with sclerotherapy or phlebectomies.

Mode of action

RF ablation induces resistive heating (85%) causing contraction of collagen fibres with associated circumferential endothelial denudation and muscle necrosis. EVLT uses thermal energy to boil blood producing thermochemical destruction of the venous wall.

Method

RF ablation can be performed under local anesthesia. EVLT is performed under tumescent anesthesia to prevent thermal injury to the skin and saphenous nerve. Both methods involve prograde introduction of catheter through a venepuncture at the ankle or knee level under ultrasound guidance. Duplex ultrasound is indispensable not only to assess the patients suitability for the procedure (sometimes GSV with no tortuous or thrombosed sections)but also as procedural

tool to assess catheter tip position and as post procedural tool to confirm the immediate and long term efficacy of this technique. It should be noted that laser fiber cannot be identified with duplex ultrasound. There are many studies showing the efficacy of laser and RF in one multicenter study of 1222 shows that occlusion in GSV at 87% of the 117 patients in 5 years. The randomized study shows RF ablation was superior to surgery.(69)

10.5 Surgical debridement

The healing ulcer has to have granulated tissue, however necrotic tissues and fluids that excess the ulcers causes the sludge and devitalization of the tissue. This sludge has toxins and bacteria alters the physical healing process, that's the reason we have to perform debridement. The ulcers covered with dry necrotic tissues are derived from the arterial etiology due to PAOD and that indicate inadequate blood supply and needs surgical or endovascular approach this kind of tissue should not be removed before revascularization performed. The debridement of the ulcer can be done with various methods surgical, sharp incision, autolytic, enzymatic, mechanical ex. Water Jet or biological by use of Larvae therapy. From the above techniques we don't have any preference, but the type of debridement should be chosen according to the wound, or to the preference of the surgeon or by the patient comfort and compliance. Surgical debridement is surgical excision of necrotic tissues around the wound. The surgical debridement according to the localization can be performed under local or general anesthesia and by the help of surgical instruments scalpel or surgical spoon can be done quickly and sufficient.

Autolytic method is using the method of Ringer absorbed moisten gases, causes lysis to pathological tissues results to removing and clean the wound. This debridement method is slower but benefits less pain to patient.

Enzymatic debridement is another method of removal of the necrotic tissue in the venous ulcers. The principle is local application of proteolytic agent to the wound disrupt the collagen. Enzymatic debridement shows better results but it's more costly method.

Mechanic debridement

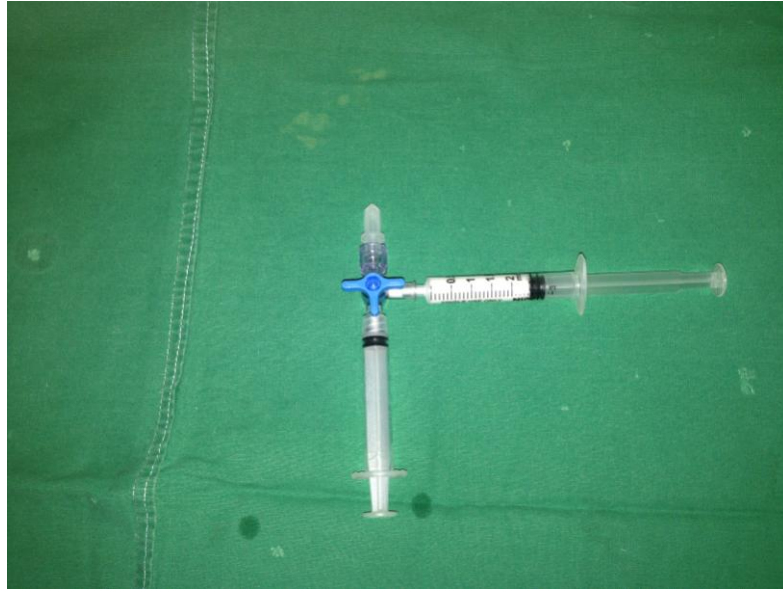
Is the oldest debridement techniques we use wet gaze .This gaze is wet with the acetylosalic cream applied to the dry necrotic tissue. This method doesn't suggest to be applied to the wet ulcers .This methods has a disadvantage that cause significant pain to the patient.

10. 6 Foam Sclerotherapy

Sclerotherapy is the method using to control the venous hypertension and treat the venous perforators round the venous ulcers. In his study Guex reported the direct perforator treatment in the venous ulcer by use of STS or Polidocanol 3% helps to close the venous perforator and improves the healing rate. Later on Cabrera introduce the foam micro sclerotherapy in contrast the sclerotherapy has more advantages ,mechanical action displacing the blood, greater sclerosing ability and volumes than the liquid form, manageability of foam is better has selectivity action ,stability of microbubbles, rapid elimination of gas and real time visibility in ultrasound .Further benefits patients not need of surgery and anesthesia ,and its less costly.

Technique of microfoam sclerotherapy

Liquid sclerotherapy had been demonstrated to be valuable adjust to other therapies but has not been widely accepted as primary therapy for large vein incompetence, which must be treated in most venous ulcers. Microfoam sclerotherapy is pioneered by Cabrera has been shown to be highly effective and safe in the management of incompetent veins of all sizes in the superficial venous system, and these technique can be applied in all of the stages of the disease even in leg ulcers.



Foam sclerotherapy Tessari technique own material

Micro foam sclerotherapy can be performed in the treatment room without anesthesia for non-operative treatment of superficial reflux or incompetent perforator in ulcerated limbs. Duplex ultrasound is used to identify incompetent veins and source of reflux that causes the VH. After polidocanol Microfoam (15-30 ml) has injected under ultrasound guidance into the main incompetent vein to fill the vein up to the source of reflux. The access route is depend from the size of vein. Cannulation with an Abbocath 20 g cannula is performed in great saphenous veins GSV or direct needle puncture (23-25 G) in smaller veins. Duplex ultrasound is used during the procedure to confirm the correct location of needle, the spread of Microfoam and the onset of vasospasm. The ultrasound transducer is placed at the saphenofemoral junction to show the arrival of the injected Microfoam and the occlude of GSV. The Microfoam is the direct distally replacing the probe with a finger to block the proximal saphenous vein. Perforator veins are selectively treated by direct ultrasound –guided injection, placing the needle in a superficial vein that is connected to but 2-3 cm distant from the perforator vein. Injection of Polidocanol Microfoam into this area allows control over the volume, avoiding extension of its action into deep venous system (DVS).A volume of 1-4 ml is usually adequate, and digital or transducer compression is performed to halt any spread of the Microfoam toward to the

DVS. Another method to protect the DVS is to inject the Microfoam with limb elevation. This position creates a gradient whereby the DVS has higher blood pressure, preventing drainage of Microfoam into the DVS. As a safety measure during injection of trunk veins and perforator veins advance should be taken of the blocking of intramuscular veins by calf muscle contraction. Thus the foot is dorsally flexed by using operators hand or cooperative patients voluntarily sustain contraction of the medial gastrocnemius muscle. These close door procedure enhance the safety of the procedure by preventing the Microfoam foam draining into the DVS. After the injection ,the patient should remain supine for 15-20 minutes before standing up and before application of compression in order to avoid dislocation of the Microfoam colum. Treatment is completed by placement of pads and application of a compression stocking, which is worn 7-15 days after injection.



Results in the ESCHAR study show the application of DUS foam sclerotherapy in contrast surgery and its ability to treat venous insufficiency. The study shows the application of compression therapy with combinations of the methods versus compression therapy. Results show that combined techniques foam sclerotherapy with compression therapy has better results in mean of time healing wound and less recurrences. The foam sclerotherapy already has been proven the efficiency to treat venous insufficiency, its safety technique and can be used to treat venous ulcers even to older population with some safety measurement taken. Complication of the treatment can be scotoma, neurological disturbance, visual disturbances. Should be avoided to the patient with presence of history of migraine or known allergy to sclerosant material, all these complications are count less of 1% can be excluded when the doctor are trained and follows the safety guidelines. (70)

10.7 Skin Grafts

Xenografts

Xenografts serve as biologic dressing and protect wounds from bacteria and physical traumas, reduce pain and increase moisture and heat retention, we can use also autologous skin grafts. In the past we used to use from various animals dogs, cats, rats, chicken, sheep cows and pigs. We used till today especially the porcine xenografts for their low cost and wide availability. The xenografts are applied in the wound and they adhered, the treatment modality starts with wound clean, washed of allograft and adhesion of xenograft on the wound. The xenografts or allografts after that has to be closely monitored and changed weekly before rejection occurs. Compression therapy should be applied to reduced edema and patient should be seen once a week. (71)

Allografts

Allografts are donated skin from cadaver or tissue available in tissue banks. This kind of dressing is used to cover burns, but can be used also to cover chronic wounds including venous ulcers. Cadaver tissue works like matrix tissue and growth on granulation tissue. After preparation of wound bed we applied them with local antibiotics applied over graft. The follow up process has to be checked weekly and has to be monitored for rejection or presence of local infection. Allografts have ability to control pain, decrease risk of infection, stimulate neovascularization, reduce healing time, cover bone and tendons and provide good cover in place needed.(72)

Human equivalents

Human equivalents are human skin evolved in by the biologic dressings. The most used widely and researched are HSE is cultured, allogenic, bilayer product called Apligraf treatment approved in USA for treatment of venous ulcer. This dressing has to be used at difficult healing wounds due to cost of the graft. Proper handling of Apligraf is critical has to place at wound by the glossy side. Antibiotics and local treatment with sterile gauze should be taken with combination of compression therapy. Rejection is very low a close up follow should be during healing process. (73)

Collagen dressings

Collagen factors are dressing available for the treatment of chronic wound, they derived from bovine or porcine collagen and it comes in variety of preparation, can be matrix or pure collagen. These dressing can be applied on the wound bed and regenerate faster the collagen and has quicker healing time in vein ulcer.(74)

Growth factor therapy PRP

Growth factor therapy is a combination therapy of growth factor ex Regranix which is combined with blood plasma caused tissue growth and regeneration. The process need blood to be taken from patient and with the help of injection applied in the wound, however this treatment is still under experimented.

11 Obstruction relief Deep venous interventions

Relief of obstruction

Obstruction is the principal cause of symptoms in approximately one-third of postthrombotic limbs. It is associated with reflux in 55% of symptomatic patients with CVD. (75). This combination leads to the highest levels of venous hypertension and the most severe symptoms as compared to either reflux or obstruction, especially in the iliac vein is more likely to cause symptoms than lower segmental blockages. Following iliofemoral DVT, only 20 -30% of iliac veins the remaining veins have residual obstruction and varying degrees of collaterals. (76). The main aim from intervention is to relieve proximal outflow obstruction.

Diagnosis and selection of patients. It is important for the physician to be aware that there may be venous occlusion. Patients presenting with classes C 3-6 should be considered for further studies, particularly those with venous claudication on challenged exercise. Unfortunately, there are no reliable tests to measure what degree of narrowing constitutes an anatomically significant “critical stenosis” in the venous system. This lack of a “gold standard” to assess the importance of chronic outflow obstruction is the major obstacle to selecting limbs for treatment and evaluating outcome. Although a positive noninvasive or invasive test may indicate the need to proceed with further investigations, a negative test should not discourage it. Ascending or ante grade trans femoral phlebography is the standard method to image the venous outflow tract, showing the site of obstruction and the presence of collaterals. Intravascular ultrasound (IVUS) is superior to standard singleplane and multi-plane phlebography for estimating the morphological degree and extent of iliac vein stenosis and to visualize details of intraluminal lesions. Iliocaval obstruction and underlying abnormalities can be detected by MRI and spiral CT venography.(77)

Open surgical reconstruction. Results following open reconstructions are usually presented in series with small numbers of treated limbs and short observation times, usually with poor reporting standards and rarely presenting cumulative patency and success rates. Bypass grafting appears to have relatively poor long-term patency rates, perhaps for several reasons such as low pressure bypass, inherent thrombogenicity of non-saphenous graft material and poor distal inflow due to extensive distal disease.(78)

The cross-over bypass

The autogenous femoro-femoral venous bypass appears to be less thrombogenic with better patency than prosthetic grafts. However, most series have small numbers of patients with inconsistent clinical and venographic follow-up.

The in-line bypass. Anatomic in-line bypass reconstruction can be used in the femoro-iliocaval axial outflow axis with segmental obstruction in the presence of a sufficient venous inflow and outflow of the graft.

The only study presenting cumulative success rates by Jost *et al.* shows a secondary patency rate of 54% at 2 years for prosthetic in-line bypass. This should be compared to 83% patency for saphenous vein femoro-femoral crossover bypass in the same study.(79)

Sapheno-popliteal bypass. Sapheno-popliteal vein bypass is a rarely performed operation for outflow obstruction. The few reported series of patients, show clinical success and patency rates of 31-58% and 56-67% for follow-up at one to five years respectively.

Endophlebectomy of the deep veins. Endophlebectomy may be performed to improve inflow and outflow in association with bypass and stenting procedures.(80)

Femoro-ilio-caval-stenting. The introduction of percutaneous iliac venous balloon dilation and stenting has dramatically expanded the scope of treatment. Complications are minimal and mortality has been nil. Studies of venous stenting in peer review publications often have similar short-comings as reports for open

surgery. Most are case reports and very few are sizable, the follow-up is short-term with patency not reported as cumulative success, stented sites in upper and lower extremities are mixed, and the majority of reported series have not differentiated between etiologies or management of acute and chronic conditions. Patency rates assessed by duplex ultrasound or phlebography in successfully stented limbs of mixed groups of patients are shown results.

Stented limbs with non-thrombotic disease appear to do far better than those with thrombotic disease, with reported primary, assisted-primary and secondary cumulative patency rates of 89%, 100% and 100% and 65%, 85% and 88% respectively at 36 months. (81)

Severe in-stent recurrent stenosis defined as greater than 50% diameter decrease on single plane antero-posterior venogram was infrequent occurring in only 15% at 42 months in one study. Gender and side of limbs involved did not affect outcome. Higher rates of severe in-stent recurrent stenosis were found in thrombotic compared to nonthrombotic limbs, reported as 23% and 4% respectively at 36 months in this study, and 18% and 12%, respectively in the presence of thrombophilia. Long stents (> 13cm) and extension of 25% at 36 months and 40% at 24 months respectively. These three major risk factors of thrombotic obstruction, thrombophilia, and long stents for development of in-stent recurrent stenosis were similar for late occlusion and limbs with these three risk factors show a 61% rate of severe instent restenosis at 24 months post-stenting, while none developed in the their absence.

The reports describing patency rates indicate clicnical improvement in the intermediate term in most patients (>72%). The incidence of ulcer healing after iliac vein balloon dilation and stent placement in 304 limbs with active ulcer was 68% and the cumulative ulcer recurrence-free rate at 2 years was 62%. median swelling and pain severity scores decreased significantly. The frequency of limbs with any swelling decreased from 88% to 53% and limbs with any pain from 93% to 29%. using a quality-of-life questionnaire assessing subjective pain, sleep disturbance, morale and social activities, and routine or strenuous physical

activities, patients indicated significant improvement in all major categories after venous stenting.

Stenting technology is relatively recent so that the follow-up period is limited. Because long-term effects of stents in the venous system are not fully known, monitoring for several more years is required to assess efficacy and safety.(82)

12 Wound care

Wound treatment with Adjuvant therapy

The healing of venous treatment are costly and chronic, it is estimated a great cost to healthcare providers, and may need to be repeated during her/his lifetime. More than a million people in USA are affected, healing process are cost them to loose productive Hours and decrease the quality of life.

Usually the management of wound care are provided by the registered specialized nurses under guidance of the medical team , but till now we don't have certain guidelines to be follow or a methodology proven has provoke quicker healing. We all agree the management of the edema thought (compression therapy, leg elevation cause healing promotion. But in 50% of chronic vein ulcers and especially at elderly people it takes more time. So this kind of people has to be treated with individual approach consider the medical history, systemic disease such heart failure, renal failure, and has to exclude PAD and with use of color Doppler ultrasound identified the cause of pathology .Always we have to well document the wounds by photography and measure and we have to performed aggressive debridement to remove necrotic tissue. We have to perform most of the debridement under local or regional anesthesia, wounds has to be evaluate the presence of infection and in case of cellulitis or infection presence has to be administered precise antibiotic treatment after wound culture. Also can be used local antibiotics or silver of alginates topical treatments. All these treatment has to be used combined with compression therapy ulcer x kits or Unna's boot multilayer bandages. These therapy - trial given, has to be treated with the same treatment modality for 4 weeks at least, progress has to be documented (excellent granulating tissue, wound volume) patient with correctable venous under pathology has to be treated appropriately. When the wound slows down the healing process and the volume still stays without significant reduction more than 50 %, other methods of adjuvant treatment can be used include, xerographs, allografts, growth factor therapy, human skin equivalents and vacuum closure systems, all these systems has to be follow with one goal to promote healing and avoid recurrence.(83)

13 Prevention of leg ulcer recurrence

Prevention of leg recurrence is not well explore most of the studies are dedicated to the heal ulcer, few studies are relate to the problem of leg ulcer recurrence after healing. The incidence of recurrent ulceration after healing with conservative techniques varies in different studies from 26-69% at 12 months. Few studies show recurrence rates between 28%-57% at 2 years, 38% at 3 years and 48% at 5 years. (84)

Compression therapy

Compression therapy is believed to counteract the effects of venous hypertension and to control edema. A recent Cochrane review of compression prevalence didn't find any randomized controlled studies comparing ulcer recurrence rates with and without compression. But still believed that the not wearing compression stocking for various reasons is associated with leg ulcer recurrence. The recurrence rate is 2-3 higher with non-compliant patients during an observation period and we have high rate between 1-156 months. It's difficult to assess a patient daily compliance .Lack of compliance can be due to several factors including lack of cosmetic appeal, discomfort, inability to put stocking on allergy to material, lack of financial resources, and lack of patient understanding and education about their condition and these need to be addressed to improve compliance. Further need to be done to prove the compression therapy leads to satisfactory outcomes in venous ulcer recurrence.

Bed rest and leg elevation

Leg elevation and bed rest have been recommended to control edema, preferably with the leg elevated above the heart level. However there is no supportive evidence that can prevent ulcer recurrence.

Exercise- rehabilitation and body weight

Obesity is an increasing problem in the general population and has been linked to skin changes and ulcers of venous type with or without detection of chronic venous disease. Greater body weight has been shown to be statistically associated with poor healing of venous ulcers and proportionally more patients with ulcer have been found to be obese as compared to the general population shown at study in Sweden.

The function of the calf muscle pump is greatly influenced by the mobility of the ankle joint. It has been shown that ankle range of motion decreases with increasing severity of clinical symptoms of CVD, and is associated with poor calf pump function as measured by plethysmography. It would seem that improvement of the calf muscle pump by exercise would increase venous return and subsequently help the clinical situation. Exercise and weight loss are often recommended to prevent or delay recurrence of venous ulcers but there is no conclusive evidence to show that are effective.

Correction of underlying venous insufficiency

Ulcer recurrence rates have been reported after correcting underlying venous pathology by superficial and deep venous interventions, but few appropriate studies are available to indicate that correction of CVD results in reduced incidence of ulcer recurrence. In a prospective non-randomized study by Mc Daniel shows that significantly less cumulative recurrence rate at 48 months in limbs treated by a variety of operations compared to those treated without surgery. The study found that patient who were not candidates or who elected to forego surgery had 3.4 times higher rate ulcer recurrence. A prospective, randomized study combining compression with or without simple superficial venous surgery showed that the overall 24 week healing rates were similar in the groups, but the 12 th month ulcer recurrence rate was significantly reduced in the group with compression and surgery compared to those with compression alone.

Deep venous insufficiency appears to be major determination for ulcer recurrence. The ulcer recurrence rate after superficial venous surgery or perforator ligation is

markedly increased by associated deep venous disease. Cumulative recurrence rates at 4-5 years 67-100% respectively in limbs with and without deep venous involvement. It seems although we performing deep valve repair should be beneficial but the proof is circumstantial. Long term results from the study Masuda and Kistner after deep valve reconstruction reported 40% ulcer recurrence over a long period but many had long repair were superior for primary disease compared to post-thrombotic disease in some studies, but in Raju study reported 6 years cumulative ulcer recurrence rate after deep venous reconstruction of approximately 40% which was similar in primary and secondary disease.

Treatment should intuitively change underlying pathophysiology to prevent recurrence. A decreased ulcer recurrence rate has been observed in limbs with less reflux as measured by VFI using air plethysmography where limbs with VFI of less than 4,0 ml versus those with more than 4,0 ml/Ls were associated with 28% and 53% recurrence respectively. Another study report that the recurrence rate was only 14% if a venous filling time VFT more than 5s could be maintained compared to 45% when VFT was less 5s.

Ulcer healing outcome data and physiological test results are circumstantial but they support surgery in patients who have recurrence during conservative treatment or in those who are unable to comply with conservative measures.

Prevention of recurrent DVT

Studies to evaluate whether prevention of recurrent DVT decreases the risk of ulcer recurrence have not been performed. Patients with chronic venous ulceration have a 41% prevalence of thrombophilia, similar reported for patients with previous DVT. In series of patients' stented for venous obstruction, 51% of those with postthrombotic occlusion had thrombophilia although thrombophilia was also found in 23% of patients considered to have primary disease. It has been suggested that patients with venous ulceration may have subclinical thrombosis or undetected distal macro and even micro vascular disease due to thrombophilia. It is possible that long term anticoagulation in selected patients may prevent recurrent thrombosis and decrease the risk of recurrent ulceration.

14 Our experience

ABSTRACT

In these study we will like to prove that in venous ulcer we can have better results when we are using multiple techniques to treat venous legs ulcers. In these group of CEAP C6 active or C5 healed vein ulcer .At ulcer prior to treatment went through physical examination, medical history taken and the ulcer was photo document. We diagnose the underlying pathology by use of Ultrasound and we found in the 15 of the cases was SFJ insufficiency more than 5 cm and additional perforator vein insufficient at Cockett I- III, and the other 5 only perforator insufficiency. To treat we use the techniques of Unna's boot treatment to reduce edema in combination of Ultrasound guided foam sclerotherapy at venous perforators, following later on surgical procedures crossectomy with distal stripping of saphenous vein.

Method

Between 1 of June 2013 - 31 December 2016 , 23 patient (28 limbs CEAP C6 24, C5 4),of the median age of 67 years old underwent to Unna's boot compression treatment, by ultrasound guided sclerotherapy at venous perforator, followed by surgery crossectomy and introduce in the end of the healing ulcer compression therapy double layer kit (Ulcer X kit)

Results

The median follow up was 12-32 months, one patient of the C6 group passed away and another one was stopped the treatment. In 21 patient we applied the therapy both ambulatory and during hospitalization. The healing result it was almost 95 % and the average treatment takes 9 weeks patients had a satisfactory results at all patient, at the end of the treatment was introduced to compression therapy ulcer X double layer stocking compression grade II .In additional at 10 patient during the followed up we performed foam sclerotherapy session in average 2 session used both Polidocanol or STS solution 3% Tessari method and

used Cabrera technique catheter applied before or after the surgery. We show that the benefit of treating the patient with multiple approach creates benefits to patient and heals the ulcer more sufficient during the follow up period we had it almost 90% of ulcer healed with 10 % with the recurrence. In these study we show that the more of taking care of vein ulcers treating the pathology of venous insufficiency it proof better outcome results.

Photos Results



case 1.58 yo woman with 2 two year lasting VU. After surgery and USGF results after 6 weeks healing time.



Case 2.55 yo woman with VSM insufincieny and cocket I-II insufincieny after a combination of treatment with surgery and perforator sclerotherapy.



Case 3: 28 yo old men with VU ultrasound of SFJ reflux and coxect I-II perforator insufficiency, after treatment with surgery and foam sclerotherapy.



Case 4: 38 yo women with 5 years lasting Vu after 8 weeks and combination treatment result.

Key words: multiple approach at venous ulcers, Ultrasound foam sclerotherapy, Unna's boot, crossectomy and distal stripping, Ulcer X kits.

Conclusion

Veins ulcers is constant battle against venous hypertension. It need further understanding for the deep venous obstruction and pathophysiology of venous valve insufficiency, to be able to treat the deep venous insufficiency .Old treatment modalities like operation and foam sclerotherapy shows the efficacy to sufficient treat the venous reflux and give results. The biggest problem till now is to prevent the recurrence of the venous ulcer these has patient be introduced to specialized compression therapy as the show has better results.

In the future new technologies like deep venous stent at IVC for relief of the obstruction it shows good results, but further clinical studies has to conduct to prove the treatment of venous insufficiency.