DISSERTATION

ON

UNNA'S BOOT IN NON – HEALING ULCER

Dissertation submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfilment of the regulations

for the award of the degree of

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THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

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CERTIFICATE

This is to certify that this dissertation entitled " **UNNA'S BOOT IN NON - HEALING ULCER**" is the bonafide original work of **Dr. K.VINOTH KUMAR** in partial fulfilment of the requirements for M.S. Branch -I (General surgery) Examination of the TamilNadu Dr. M.G.R. Medical University to be held in APRIL - 2013. The period of study was from January2011 to June 2012.

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I, **Dr. K.VINOTH KUMAR,** solemnly declare that the dissertation titled "**DISSERTATION ON THE UNNA'S BOOT IN NON – HEALING ULCER"** is a bonafide work done by me at Thanjavur Medical College, Thanjavur during January 2011 to June 2012 under the guidance and supervision of **Prof. Dr. P.SHANTHINI M.S., D.G.O., FICS** Unit Chief S-V, Thanjavur Medical College, Thanjavur.

This dissertation is submitted to Tamilnadu Dr. M.G.R Medical University towards partial fulfilment of requirement for the award of **M.S. degree (Branch -I) in General surgery.**

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CONTENTS

INTRODUCTION

The lower limb ulcers are one of the challenging diseases among the surgeons. Because of its varied etiology, disease manifestations and different proposal in the treatment, makes it a nemesis for the treating physicians. Venous disease causing venous ulcer in the lower limb is a common finding but usually causes limited morbidity in majority of patients. However the patients develop skin changes and ulcerations leading to pain and discomfort which necessitates treatment at frequent interval. This imposes financial burden on the patient as well as on health care systems. The prevalence of venous disease has been studied in considerable number of epidemiological surveys. However the exact prevalence could not be made out due to various reasons.

Leg ulcers are approximately 0.5% of the world population and 2.5 million in United States. Approximately 70% are caused by Chronic Venous Insufficiency. About 20 to 25% are due to arterial or mixed disease. Exact prevalence and incidence of leg ulcers could not be determined because they are reported as a complication of underlying systemic disease and not reported separately. While the exact prevalence of leg ulcers are unclear, the impact of these ulcers on health care system and patient is becoming increasingly evident. The cost of treatment of

venous ulcer alone is estimated to be one billion dollar per year in United States.

The non healing ulcers of the lower extremity are multifactorial in origin. They usually develop as a result of skin and tissue changes caused by CVI and the associated ambulatory venous hypertension. The prevalence of venous ulcerations increases with age in both men and women. This probably because of fact that as venous diseases progresses throughout the life and hence most of the patients reach the point at which an ulcer develops. The reason for ulcer in old age was due to, limited mobility associated with degenerated joint diseases and poor wound healing.

The usual site of venous ulcer is above the medial malleolus, the gaiter's area because the ambulatory venous pressure is more in this area and the gravity is also high in this area. The severity of impairment of venous physiology of the lower limb is mainly measured by the ambulatory foot vein pressure which helps in assessing the severity of the venous reflux. Management of these venous ulcers include thorough History, clinical examination to put the disease in probable etiology and investigations for proper diagnosis and treatment and to give the best possible outcome of the disease, and more importantly to prevent recurrence in long term disease management.

HISTORICAL REVIEW

The first reference about vein was probably in the Ebers papyrus written, 1550 BC[1], which notes 'Serpentine Windings'. The first known mention of leg ulcers is by Hippocrates (460-377 BC)[3], who noted the association between varicose veins and leg ulcerations and recommended that patient with venous ulcer should avoid standing. He introduced "puncturing and bandaging" for the treatment of leg ulcers.

Aurelius Cornelius Celsus (25 BC-50 AD) advised the use of plasters and linen bandages in the treatment of ulcers and also treated varicose vein by avulsion and cauterisation[2].

Haly, son of Abbas, during the Dark Ages, those with unsightly and ulcerated legs were because of bandaging the varicose vein which might have reintroduced the 'black bile' into the circulation and lead to madness.

Ambroise Pare (1510-1590), after being taken prisoner at the Siege of Hesdin in 1553, he was summoned to attend Lord Vaudeveille, who was suffering from non healing ulcer for 6 to 7 years. After debriding the ulcer and excising the indurate edge, he applied a dressing soaked in plantain juice and bandaged the leg from foot to knee. Ulcer completely healed by 2 weeks later[4].

Richard Wiseman described a laced stocking in 1676 in the treatment of leg ulcers[5]. In 1775 John Hunter wrote about leg ulcers as "the sores of poor people are often in a bad condition from bad living and are often healed by rest in a horizontal position, fresh provisions and warmth in hospitals, and the change is generally very speedy"[6].

During the 18th century it began to be realised that leg ulcers were not necessarily accompanied by visible varicose vein. Baynton in 1797 introduced paste bandaging to heal ulcers and found that some dressings caused sensitivity reactions[8]. In 1868 John gay, described the calf and ankle perforating veins. He also recorded the fact that ulcers could occur in the absence of varicose veins and ulcer may be due to post-thrombotic damage to the deep veins and introduced the term "venous ulcer".

In 1916, John Homans divided ulcers in to those associated with varicose veins, easily cured by stripping of the veins and post-phlebitic ulcers, rapid in development, intractable to palliative treatment, incurable by removable of varicose vein alone[9]. In 1927, Franklin discovered valves in veins and created interest in venous physiology and pathology. In 1937, Edwards and Edwards, showed that venous thrombosis destroyed valves but followed by the recanalization.

AIM OF THE STUDY

- To analyse and study the epidemiology of age, sex, incidence of non healing ulcers of lower limb in Thanjavur Medical College.

- To study about the correlation between the occupation and the type of non – healing ulcers among the patients.

- To study about the incidence of various types of non – healing ulcers and their duration.

- To analyse about the conservative management of Unna's Boot in various non – healing ulcers and their response to the treatment.

MATERIALS AND METHODS

About 60 clinical cases of non – healing ulcer of the lower limb admitted in Thanjavur Medical College and Hospital during the period of 18 months from January 2011 to June 2012 were selected.

INCLUSION CRITERIA:

1. Non – healing ulcer patients of lower limb were selected.
2. Previous interventions either surgical or conservative management were included.
3. Patient should be ambulant.

EXCLUSION CRITERIA:

1. Not giving consent for treatment modality used in the study.
2. Non – healing ulcer turning into malignancy are excluded.
3. Non – healing ulcer of arterial diseases are excluded.
4. Superficial skin infection turning into ulcer are excluded.
5. Patients with heart failure or debilitating general conditions of etiology other than non – healing ulcer are excluded.
6. If patients are of diabetic, if arterial system are involved, then the patients are excluded from the study.
7. Patients allergic to components used in the Unna's boot are also excluded from the study.

MATERIALS REQUIRED:

1. Roller guaze bandage
2. Zinc Oxide, Sorbitol, Gelatin,Glycerin,calamine.
3. Elastic crepe bandage.
4. Gloves

PROCEDURE:

1. Wash hands and put on gloves

2. Wash cloth or scrub sponge with cleaning agent

3. Assess the ulcer and skin.

4. Clean the affected area gently with the sponge and cleaning agent. Rinse with normal saline solution.

5. Position the patient's leg in a slightly flexed position.

6. Put on gloves

7. Apply the unna boot (gauze) in a circular motion from the foot to the knee. The wrap should be snug but not tight to cover the area completely. Be sure each turn overlaps the previous one by half the bandage's width.

8. Continue wrapping the patient's leg up to the knee, using firm, even pressure. Mold the boot with your free hand as you apply the bandage to make it smooth and even.

9. Cover the boot with an ace bandage for compression.

10. Observe the patient's foot for signs of impairment, such as cyanosis, lose of feeling, or swelling.

11. Leave the boot on for 5 to 7 days, or as ordered by the physician.

12. Patient Instruction:

A. Observe for signs of impairment

B. Tell the patient the boot will stiffen, but will not be as hard as a cast

C. To cover the boot with a plastic kitchen trash bag sealed at the knee to avoid getting the boot wet

13. Remove the boot with a sterile tongue blade and bandage scissors or scalpel

PRECAUTION: Do not make reverse turns while wrapping the bandage. (This could create areas of excessive pressure that may cause discomfort)

ANATOMY OF BLOOD VESSELS

The integrity of the skin of the lower limb depends on its micro circulations and the ulceration is the end result of microcirculatory failure. Hence in treating leg ulcers one should have working knowledge of the lower limb circulation which can be discussed under the following:

1. Microcirculation

2. Arteries

3. Veins

Microcirculation

The dermal microcirculation is a network of arterioles, the rete subpapillae, or superficial plexus from which the capillaries loop in to the dermal papillae, before passing back to the venous plexus[9]. Arteriovenous anastomosis are common in the deeper layer of dermis, which are surrounded by the sphincter like smooth muscles and pursue a convoluted course. The exchange of oxygen and other metabolites takes place through the wall of capillaries and across the walls of venules and arterioles. Blood flow in the microcirculation is governed by contraction and relaxation of smooth muscles in the arterioles and veins. The alteration in arteriolar and venous tone are controlled by the sympathetic

nervous system. Leukocytes and platelets accumulate in the microcirculation during venous congestion, and their activation is responsible for some of the complications of chronic venous hypertension.

VEINS OF THE LOWER LIMB

The walls of the veins composed of three coats namely an inner endothelium, a muscular media and an outer fibrous adventitia[7]. The endothelium of intima secretes factor VIII prostacyclins and fibrinolytic activator. The media consists of collagen and elastin fibres and circularly arranged non-striated muscle fibres. The muscle fibres of superficial veins are well developed and contraction is controlled by postganglionic adrenergic sympathetic nerve fibres whereas the media of the deep veins contains relatively little muscle and these veins act mainly as passive blood conduits. The adventitia consists of loose areolar tissue with longitudinal elastic fibres.

SUPERFICIAL VEINS

These are the great and small saphenous veins and the tributaries of the perforating veins.

THE GREAT SAPHENOUS VEIN (Saphena Magna):

The Great Saphenous Vein (GSV) arises from the medial end of the dorsal venous arch of the foot. It passes in front of the medial malleolus and along the medial surface of the leg, enclosed in its own fascial sheath. The GSV lies posterior at knee level and then passes of the thigh and through the foramen ovale and the femoral triangle to join the common femoral vein[9]. It crosses the superficial external pudendal artery at the lower border of the foramen ovale.

The tributaries of GSV in the groin are the anterolateral and posteromedial veins of the thigh. Smaller tributaries are the superficial and deep external pudendal vein, superficial epigastric vein and superficial circumflex vein. Just below the knee are the anterior vein of the leg and posterior arch vein. The important direct perforating vein of the calf communicates with the posterior arch vein and not directly with the GSV. The GSV is accompanied by the saphenous nerve, which is in close approximtiy with the GSV in the lower third of the calf[13]. The dorsal venous arch of the foot communicates with the plantar veins by four perforating veins. The GSV, which arises from the medial end of the dorsal venous arch gives one or two inframalleolar veins triburatries to the posterior arch veins and forms the corona phlebectatica network of malleolar veins, that drain in to the medial calf perforating veins[13].

THE SMALL SAPHENOUS VEIN (Saphena Parva):

The Small Saphenous Vein (SSV) starts at the lateral end of the dorsal venous arch of the foot and passes behind the lateral malleolus to join the popliteal vein in the popliteal fossa. The termination of the SSV is variable. The high termination occurs in 33% of cases. In this variation, the SSV passes up to the posterior surface of the thigh namely the persistent post axial vein, Giocomini vein and either joins the GSV or terminates in muscle vein[14]. The low termination occurs in 9% of cases in which the SSV join the gastrocnemius veins in the calf. It is important to realize that SSV perforates the deep fascia in the lower or middle third of the calf between the bellies of gastrocnemius until it joins the popliteal vein in the popliteal fossa. Ulceration in the lateral surface of the ankle is probably due to incompetence of the SSV. The GSV and SSV have relatively thick muscle coats, but the valves of the tributaries are thin and more likely to dilate and become varicose.

THE PERFORATING VEINS:

The perforating veins are those veins which penetrate the deep fascia passing from superficial to deep fascia other than GSV and SSV. There are about 100 unimportant perforating veins, which enter the muscles before joining the deep veins. They contain numerous valves[13].

THE CALF AND ANKLE PERFORATING VEINS:

There are three direct perforating veins on the medial surface of the ankle and leg and one or two laterally. The medial perforating veins communicate with the posterior arch vein, not the GSV itself. Each perforating vein contains a valve, which directs blood from superficial to deep. The lowest perforating vein lies behind the medial malleolus, the next a hand's breadth above this and the most proximal another hand's breadth higher (cockett's hand's breadth rule)[12].

The direct calf and the ankle perforating veins drain the skin over the medial and lateral malleoli by networks of the venules and small veins at the distal end of posterior arch vein. These veins dilate under increased venous pressure to form “ankle venous flare or corona phlebectatica". Thus, "the ulcer bearing area of the leg" is not directly drained by either GSV or SSV. However GSV communicate with the medial perforating veins through the inframalleolar vein and distal GSV also communicate with the plantar veins through the dorsal venous arch and the transmetatarsal veins[14]. The corona phlebectatica therefore communicates with the deep system by two routes:

1. Directly to the posterior tibial vein through the calf perforating veins.

2. Indirectly to the plantar veins by way of the dorsal venous arch.

THE FOOT PERFORATING VEINS:

The foot perforating veins join the plantar veins to the dorsal venous arch which joins the distal great and small saphenous vein[11]. Hence they are able to transmit the high venous pressures from incompetent deep veins to the distal GSV by inframalleolar veins.

THIGH PERFORATING VEINS:

The Hunterian perforators forms communication between the GSV in lower third of thigh and the superficial femoral vein in the subsartorial canal. About 70% of incompetent thigh perforating veins are found in the region of the adductor canal. Other perforating veins are boyd's perforator[9], which joins the GSV to posterior tibial vein at the level of the tibial tubercle.

THE DEEP VEINS:

The deep veins of the lower leg are the paired venae comitantes of the anterior and posterior tibal and the peroneal arteries, the gastrocnemius veins and the soleus venous arcades. They join to form the poplitial vein. The main calf veins are profusely valved, but the soleus arcades dilate in to large valveless sinusoids[14]. These important vessels with the total capacity of about 140ml act as a reservoirs or ventricles for the calf muscle pump. Contraction of the soleus muscle makes the blood to flow

from soleus sinusoids into the posterior tibial and popliteal veins. The reflux of blood is prevented by the numerous valves. The popliteal vein becomes femoral vein in the lower thigh. Its few tributaries other than hunterian perforator, muscle veins and the profunda femoris vein, joins it to form the common femoral vein. The common femoral vein receives the termination of GSV. The common femoral vein becomes the external iliac vein at the level of inguinal ligament, which in turn receives the internal iliac vein at the level of sacroiliac joint to become the common iliac vein.

TYPES OF ULCERS- BASED ON THE ETIOLOGY:[15,17]

- Ulcers related to venous disorder:

  Varicose ulcer, Venous ulceration, Ischemic ulceration

- Ulcers of mixed arterial and venous origin:
- Arteriovenous fistula
- Venous malformations- Klippel –Trenaunay syndrome
- Rheumatoid and other vasculitic ulcers
- Steroid ulcers
- Hypertensive ulcers- Martorell's ulcers
- Diabetic ulcers
- Neuropathic ulceration
- Traumatic ulceration
- Lymphedema ulcers
- Tropical ulcers
- Infectious ulcers
- Malignant ulcers

  Squamous cell, Basal cell carcinoma, Sarcomas and lymphomas

- ❖ Blood dyscrasias
- ❖ Nutritional ulceration
- ❖ Immunodeficiency causing ulcers
- ❖ Contact dermatitis

VARICOSE ULCER:

It is an ulcer overlying a superficial varicosity. The significant feature of varicose ulcer is, it is a painless ulcer[16].

Examination of ulcer: It is situated on anteromedial of lateral surface of the lower leg of ankle. It appears as Small, shallow, well epithelialised edge with healthy granulation tissue at the base[15].

VENOUS ULCER:

Venous Ulcer is related to perforator, saphenous or deep vein incompetence or combination of these[17]. The following history should be elicited as these are common features in venous ulcers.

- ❖ Local trauma
- ❖ Deep vein thrombosis
- ❖ Calf swelling (different from ankle edema)
- ❖ Calf pain (nature of pain often termed as ‘bursting type’)

The commonest site is over Medial malleolus,1 to 2mm extending down to deep fascia (deeper than varicose).

Venous ulcer are usually accompanied by

1. Ankle venous flares (direct calf perforating incompetence)
2. Pigmentation
3. Lipodermatosclerosis

Most of the venous ulcers are infected which is evident by examining the base and edge. Base is covered by purulent debris and slough, edge may be punched out. The examination of base helps to differentiate from ischemic ulcer. On removing the purulent debris, base is full of healthy pinkish granulation tissue, which is not seen in ischemic ulcer[16].

Examination of limb:

Patient should be examined in lying position. The site and appearance of the ulcer are noted.

- ❖ Signs of iliac vein obstruction should be looked i.e., small dilated groin collateral veins, increase in calf circumference on affected side.
- ❖ Abdominal and rectal examination should be done.
- ❖ With the patient in standing position, venous filling is noted after delay of 30 seconds and there by varicose veins.

- ❖ Patient toes and forefoot should be examined for ischemic signs i.e., cyanosis, slow capillary refilling position.
- ❖ All lower limbs pulses should be palpated and ankle pulse pressure should be noted.

Investigations:

- ❖ Duplex USG – to evaluate venous system
- ❖ Doppler USG – To identify venous incompetence. Venography is preferred in the absence of duplex. (Demonstrates both incompetency / occlusion).

If there is any occlusion, venography should be followed by femoral venous pressure at rest and on exercise to evaluate for collateral circulation.

Ischemic ulcers:

Severe pain at rest is the dictum. Smoking history often present. Most of the patients are old and bedridden[20]

Examination of ulcer:

It is found in the toe or soles, particularly in heel, in bed ridden patients.

It is situated in an area of cyanosed and pregangrenous skin.

In a typical ischemic ulcer

- Base is covered by yellow purulent exudates and necrotic debris
- On removing this, there is no granulation tissue, but with only deep fascia or tendon.
- Edges are very poorly epithelialised and may be punched out.

Examinations of lower limbs pulses are important .Ankle pulse pressure should be measured. Ankle pulse pressure is not reliable in diabetic patients (increased stiffening of arteries due to calcification), in Buerger's disease and vasculitis. In these circumstances, toe capillary refilling time is useful as it is increased greatly in ischemia.

Investigations:-

Duplex of lower limb arteries to find out stenosis or occlusion. Severity of stenosis can be evaluated by measuring peak systolic velocity rate.

Ulceration of mixed arterial and venous origin:

Ulcers may be of both arterial and venous origin. History is much helpful in case of venous origin by means of varicose veins or DVT.

Examination: A complete examination of the ulcer should be done. Ischemic signs should be looked. Careful examination of pulses is very important in all patients with venous ulcers to detect arterial origin.

Investigations:

- Doppler USG to measure ankle pulse pressure
- Duplex ultra sound evaluation of arterial and venous system of lower limb.

Arterio-venous fistula:

Skin ulceration may also be caused by arteriovenous fistula. AV fistula is due to trauma or it may be congenital[30]

Congenital:

Parkes – Weber syndrome**.** Extensive congenital AV fistula of one or both limbs, associated with increased blood flow and limb hypertrophy.

Traumatic:

It may be surgically induced E.g.: 1) Ulceration of hand following dialysis in arm or leg (formation of Brescia cimino arteriovenous fistula). 2) Inadequate ligation of tributaries of great saphenous vein when it is used in femoropopliteal vein grafting. History of increased limb growth may be present

Examination:

Congenital usually affect leg and Traumatic occur anywhere in body

Ulcer is indistinguishable from venous ulcer. The close differential diagnosis is Simple varicose veins. AV fistula is found out by careful examination of surrounding skin which shows pulsation of dilated veins and bruits may be present.

Investigation:

Arteriography – to locate the site of fistula and to evaluate the possibility of embolisation.

Venous malformations :

Ulcers are common in venous malformations and one syndrome associated with this is klippel trenaunay syndrome.

Features are Cutaneous nevus (portwine stain),Venous lymphatic malformations and Limb hypertrophy[21].

In Klippel type, the limb hypertrophy is not present, whereas other two features of classical Klippel trenaunay syndrome are present.

Investigations: Venography

Management: Ultrasound guided foam sclerotherapy.Surgical excisions of these vessels are difficult because of risk of haemorrhage and damage to adjacent structures.

Rheumatoid ulcers:

History of rheumatoid arthritis may present or may not be present. In the latter cases rheumatoid ulceration diagnosis will be the last one.

Examination:

Usually affect the lateral or posterior surface of lower leg. It resembles venous ulcer but without lipodermatosclerosis. It is a shallow serpiginous and multiple ulcers with poorly epithelialised edges with base covered by yellow slough. Examination of hands and other joints should be done to look for signs of rheumatoid arthritis.

Investigations:

- Diagnosis confirmed by presence of rheumatoid factor and elevated systemic inflammatory markers.

Vasculitic Ulcerations:

Vasculitic ulcer are small, multiple and painful They usually don't resemble venous ulcer, like rheumatoid ones.Once the vasculitis origin is suspected, other features pertained to specific conditions should be looked.

Scleroderma: Typical tightness of facial skin, fixed expression with shiny tapered fingers.

Polyarteritis nodosa: Patchy reticular livido on legs.

SLE: Malar rash,Reticulate telangiectatic erythema on toes and lateral border of feet and heels. Examinations of the pulses are mandatory.

Investigations:

Apart from the routine investigations for ulcer like Doppler and duplex USG, specific investigations for vasculitis should be done.

Eg: SLE: Anti double stranded DNA binding capacity.

Polyarteritis nodosa – Antineutrophil cytoplasmic antibody.

Scleroderma – Barium swallow to show abnormal oesophageal and jejunal peristalsis.

Biopsy of the ulcer edge and immunohistochemistry may be helpful when no specific cause is identified.

Erythrocyanosis:

Occur in women with fat legs.

Examination: Areas of skin mottled and cyanotic, painful ulcer present.

Investigations: Routine investigations to rule out arterial and venous disorders and vasculitis.

Management: Warm stockings and calcium antagonists. Chemical sympathectomy can be done.

Steroid ulcer:

Develops in patients on long term steroid treatment. E.g.: Asthma, Rheumatoid arthritis.

Examination:

Ulcers are large, shallow and serpeginious with poorly epithelialised edges and the surrounding skin is thin and fragile. They are known for their slow healing[32].

Diabetic ulcer:

Diabetic ulcer may be due to

1. Peripheral neuropathy
2. Ischemia
3. Infection

History: History of diabetes or history to diagnose diabetes should be asked.

Ischemic – Toes or forefoot or heel (Particularly in bedridden)

Neuropathic –sole (pressure on Charcot foot)

Less common condition called necrobiosis lipoidica can occur that is ulceration resulting from infection and fat necrosis of the lower leg skin. Ulcer is usually covered by ischemic slough and surrounding skin

anaesthesia may present. Complete neurological examination should be done. Examination of lower limb pulses are mandatory.

Investigations:

- Blood investigations for diabetes
- Venous and arterial disorders should be excluded by Doppler /duplex.
- Arteriography is necessary as ankle pressure is unreliable.

Neuropathic ulceration:

Common in patients with paraplegia or peripheral neuropathies.

Investigation: All neuropathic ulcer need not be of neuropathic origin[33].

So, arterial or venous causes should be excluded by both examination and investigations.

Treatment: Standard cleaning and dressing.

Foot wear (poorly fitted calipers / any tight strap) should be corrected.

Traumatic Ulceration:

Trauma may be due to

- Accident
- Self induced

- Surgically induced

(Injection sclerotherapy due to extravasations of sclerosing solution causes subcutaneous fat necrosis and ulceration of overlying skin)

Preventive measures:

- Any patient with abrasion / laceration should be looked for signs of venous reflex or arterial insufficiency.
- Diabetes should be excluded.

In case of injecting sclerotherapy

- Inject small varices with low concentration of polidocanol (eg. Sclerovein) rather than strong ones.
- Confirming whether the needle is in lumen before injecting the solution.

Other alternative are

- Foam sclerotherapy (less sclerosant per unit volume than liquid sclerosant)
- Low concentration of STD (0.2%)
- Scleremo (Chromated glycerol) But should be carefully used in patients allergic to heavy metals
- High concentration glycerol (72%)

Factitious ulceration:

Common in elderly persons, who feel lonely and want to communicate with other persons and to enjoy the company of others in hospital. These ulcers are usually Bizarre in site, slow to heal and recur repeatedly.

Lymphedema ulcer:

Ulceration in lymphedema is very rare. Usually this ulceration may be primary or secondary to filarial leg or malignant involvement of the nodes.

History: Long history of swelling of leg and foot present.

Examination: Anterior surface of ankle or lower leg. In cardiac origin, the ulcer is scattered over all surface of the leg. It appears as small, indolent and painless ulcer.

Investigations:

- Doppler USG / Duplex USG
- Cardiac causes should be excluded.
- Most effective method for lymphatic functions is Quantitative isotope scintigraphy.

Treatment:

Correcting the primary conditions.

Tropical Sores:

It is a chronic ulcer of dorsum of foot. It is common in underdeveloped countries, among poor populations and is of mixed bacteriology. Common in males with male female ratio (2:1)[29].

Most common pathogens are

- Pseudomonas aeurginosa
- Proteus mirabilis vulgaris.
- Treponema vincenti
- Bacillus fusiformis

Pre disposing factors:

- Tropical environment
- Poverty
- Malnutrition
- Chronic anaemia
- Poor educational standard
- Lack of hygiene
- Poor medical facilities

Appearances: Single, with well defined raised edges and edema. Base is covered by greenish – grey foul slough, on removing this, bleeding granulating base present. Long standing ulcer may change to malignancy.

Other infectious ulcers:

- Tubercular skin ulcer (scrofula)
  - Characterized by undermined edges which is irregular, bluish and friable.
  - It is usually Multiple.
- Bazin's disease / Erythema induratum scrofulosorum
  - Tuberculous ulceration of calf.
- Syphilitic ulcer.
  - Painless, circular punched out ulcer with wash leather slough in base.

Osteomyelitis:

Chronic Osteomyelitis of tibia discharges to form sinus and mimics venous ulceration, particularly in the lower third of medial surface of leg. History of local trauma or pulmonary or abdominal tuberculosis may present.

Investigations:

- Venous or arterial disorders should be excluded
- X ray examination shows bone destruction and sequestrum formation.

Malignant ulcers:

Squamous cell carcinoma:

Squamous cell carcinoma developed in established venous ulcer is known as Marjolin's ulcer which is remarkably rare. Malignancy is suspected if there is overgrowth of tissue at the base or at edge. Diagnosed by Biopsy and Histological examination[23]

Basal cell carcinoma:

- Mimics venous ulceration.
- Diagnosed by Biopsy.

Sarcoma and lymphoma:

- Kaposi's sarcoma and cutaneous B cell and T- cell lymphoma manifest as skin ulcers when they are associated with pyoderma gangrenosum.

Blood dyscrasias:

Leg ulceration may occur in blood dyscrasias such as, Sickle cell anemia, Thalassemia, Polycythemia rubra vera (Canes ischemia),Leukemia, Cryoglobulinemia and macroglobulinemia.

It causes rouleaux formations of RBC and thereby occlusion of small vessels.

Nutritional Ulceration:

Ulcer may be caused by malnutrition on conditions like kwashiorkor, beriberi and scurvy. Zinc deficiency is also a contributory factor.

Contact dermatitis:

Contact dermatitis is a common complication of ulcer dressings, which leads to failure of existing ulcer to heal or in the development of new ulcers.

On examination:

Skin is red, scaly, shallow with yellow base.

Common allergens are:

Balsam of peru, Neomycin, Fragrance mix, Wood tars, Benzocaine Cetrimide, Cetyl alcohol, Ammoniated mercury.

Prevented by

- ❖ Using cotton compression bandages and stockings
- ❖ To avoid lycra, used in the manufacture of stockings, which is potential thing for causing allergy.

CLASSIFICATION AND GRADING OF LOWER LIMB CHRONIC VENOUS DISEASE:

The CEAP method of classification of chronic venous disease is most widely used as well as an accepted classification. It will not only tell the etiology but also the pathophysiology in a concise manner. Hence it helps in understanding the disease well and treat them well. It also allows one to score the disease as well and categorizes them accordingly[23].

CEAP CLASSIFICATION OF LOWER LIMB CHRONIC VENOUS DISEASE[33]

C -	For Clinical signs (grade 0-6) Supplemented by (a) for asymptomatic and (s) for symptomatic presentation
E -	For Etiologic classification –congenital (E_C), Primary (E_P), Secondary (E_S)
A -	For Anatomic distribution –superficial (A_s) deep (A_D) or perforator (A_P) alone or in combination
P -	For Pathophysiologic dysfunction – reflux (P_R) or obstruction (P_O) alone or in combination

Clinical Classification

Class 0	No Visible or palpable sings of venous disease
Class 1	Telangiectases or reticular veins
Class 2	Varicose veins
Class 3	Edema
Class 4	Skin changes ascribed to venous disease (e.g. pigmentation, lipodermatosclerosis, venous eczema)
Class 5	Skin changes as in class 4 with healed ulceration
Class 6	Skin changes as in class 4 with active ulceration

Etiologic Classification

* Congenital (E_c)

* Primary (E_p) –with undetermined cause

* Secondary (E_s) – with known cause

-Post thrombotic

- Post traumatic

- other

Anatomic Classification

Superficial veins (As_{1-5}):

Segment 1	Telangiectases/reticular veins Great saphenous vein
Segment 2	Above knee
Segment 3	Below knee
Segment 4	Small saphenous vein
Segment 5	Non-saphenous vein

Deep veins (A_{D6-16}):

Segment 6	Inferior Vena cava
Segment 7	Common Iliac
Segment 8	Internal Iliac
Segment 9	External Iliac
Segment 10	Pelvic – gonadal. Broad ligament, other

Segment 11	Common Femoral
Segment 12	Deep Femoral
Segment 13	Superficial Femoral
Segment 14	Popliteal
Segment 15	Crural –Anterior tibial , posterior tibial, peroneal (all paired)
Segment 16	Muscular –gastrocnemial, soleal, other

Perforating veins ($A_{P17,18}$)

Segment 17	Thigh
Segment 18	Calf

Pathophysiologic classification

* Reflux (P_R)

* Obstruction (P_O)

* Reflux Obstruction ($P_{R,O}$)

CLINICAL SCORING OF THE SIGNS AND SYMPTOMS OF LOWER LIMB VENOUS DISEASE

Pain - 0 - no pain; 1- moderate; 2 - severe

Edema - 0 - no pain; 1- mild or moderate; 2 - severe

Claudication(venous) - 0 - no pain; 1 - mild/moderate; 2 - severe

Pigmentation - 0 - no pain; 1 - localised; 2 - generalised

ULCER CHARACTERISTICS:

Size(the biggest one)- 0 - none; 1 - <2cm; 2- >2cm

Duration - 0 - nil; 1- less than 3 months; 2- more than 3 months

Recurrence - 0 - nil; 1 - one time; 2 - two or more times

Number - 0 - nil; 1 - one; 2 - Multiple

DISABILITY SCORE

0 – No symptoms

1 – can function without support device but symptomatic

2 – can work but with help of support device upto 8 hours

3 – cant work even with support device.

PATHOPHYSIOLOGY OF VENOUS ULCERS

An ulcer is a local defect or excavation of the surface of an organ or the tissue which is produced by sloughing of the inflammatory necrotic tissue. The base of an ulcer is made up of necrotic tissue and inflammatory exudate with fibroblast proliferation and scarring. In an infected ulcer the surface consists of bacteria surrounded by an inflammatory exudate[25]. But in healing ulcer consists of a granulation tissue which is nothing but the proliferating capillary loops.

The edges are composed of epidermal cells which attempt to cover the healing granulation tissue over the base. The non healing of an ulcer occurs where the epithelisation of the edge and fibroblast proliferation at the base are opposed by factors preventing the healing process. The usual factor in impending the wound healing is vascular in origin. Healing is poor or almost nil if there is no arterial supply, as the blood is important to meet the need of growing granulation tissue by providing the necessary and adequate metabolites for their growth. In case of venous ulcer, it is the venous hypertension which is the main culprit in wound healing. Healing is also inhibited by other factors like infection, diabetes, anti inflammatory effects of steroids, vitamin c deficiency, anaemia and malnutrition.

Venous ulcers mostly occur over the medial malleolus and rarely over the lateral malleolus. It may vary in size from few cm to giant ulcers covering the whole gaiters area. Venous ulcers usually get infected in the initial stage but responds well with dressing, compression bandaging and appropriate antibiotics. However venous ulcers due to deep vein or perforating vein pathology are difficult to manage and they are refractory to the usual conservative management.

A clean venous ulcer has a base composed of pink granulation tissue, edges of pink epithelium. The pink granulation tissues are nothing but the capillary loops. Venous ulcer is usually surrounded by hyper pigmented skin. It may also be indurated (lipodermatosclerosis). These skin changes are due to pericapillary fibrin and hemosiderin deposition.

The veins are the capacitance vessels of the circulation. It contains two third of the circulating blood volume. The changes in the blood volume are controlled by changes in the venous tone mediated by the sympathetic system. The most important function of the venous system is the thermoregulation. The deep veins have less muscle coat and they mainly act as passive blood conduits. Venous ulceration is a direct impact of ambulatory venous hypertension.

All veins are made up of one- way valves that help in the unidirectional flow of blood towards the heart. Moreover these valves prevent the

backflow of blood from the high pressure deep venous system to the low pressure superficial venous system. Hence competences of these valves are very important in the pathophysiology of the venous diseases[26]. In addition to these, the course of the perforating vein, which takes an oblique course through the fascia and the muscle layers provides additional support to the connecting veins and their valves.

The returning of blood from the feet and the leg to the heart is a major physiological challenge for the lower limb venous system. This is because the blood as to flow uphill against the gravity. In an individual, in standing position, the gravitational force exerts a hydrostatic pressure equivalent to about 90mmHg at the ankle[26]. The mechanism by which the blood is returned to the heart is by three mechanisms:

1. Smooth muscle tone within the venous walls
2. Contraction of the calf muscles (gastrocnemius and soleus)
3. Negative intrathoracic pressure created during inspiration

Of all the above mechanisms, the calf muscle pump is the most important mechanism involved. The calf muscle pump and the one-way valve mechanism work synchronously to propel the blood from feet and leg to the heart[24].

Blood is returned to the heart by following muscle pumps:

1. Foot pump
2. Calf muscle pump
3. Abdominal pump
4. Respiratory pump

Foot venous pump:

Gardner and Fox first demonstrated the foot venous pump. According to them the blood is expelled from the plantar veins by their intermittent stretching during foot movement, rather than by the direct pressure of sole of the foot over the ground. This physiology is applied in preventing the deep vein thrombosis in bed ridden patient who were asked to do a passive foot movements while lying on the couch itself.

Calf muscle pump:

The capacity and pressure profile of the calf muscle pump is something unique and it is greater than other pump mechanisms in returning the blood to heart. The disordered calf muscle pump is considered to be the single most important factor in the etiology of venous ulceration. The calf muscle pump is often termed as the 'Peripheral Heart'[28]. This is because it is interesting to note that the total amount of blood of the lower limb in

erect posture is about 100 -140 ml which is equal to the amount of blood in the ventricle of the heart. Moreover the calf muscle has a powerful pumping mechanism. The intramuscular pressure varies from 140mmHg to about 250 mmHg.

There are four important components of the calf muscle pump. They are:

1. The dilated valveless sinusoids within the soleus and the gastrocnemius muscle.
2. The direct calf perforating veins.
3. Numerous valves in the communicating and deep perforating veins which direct the blood flow from the superficial to deep venous system.
4. The layer of very tough deep fascia which surrounds the calf muscles.

PRESSURE SYSTEM IN THE CALF:

In deep veins, the pressure are rather lower than the calf muscle. This is because of the deep vein lies within the deep fascia. The pressure changes are more markedly seen in the posterior tibial vein. During the calf muscle contraction, i.e., the systolic phase of the calf muscle contraction, the pressure in the deep venous system reaches a peak to about 120-250mmHg, the one way valves of the perforating veins get closed. Thus

the pressure is contained within the deep system and the blood is pushed into the deep veins from the dilated soleus sinusoids. During the diastolic phase of the calf muscle i.e., the relaxation of the calf muscle, the pressure falls back to 30mmHg, as well as the valve of the perforating vein opens and the blood from the superficial system flow into the deep venous system[26]. Thus high resting pressure (muscle contraction) and low walking pressure (emptying) characterize normal venous function.

DISORDERED CALF MUSCLE PUMP:

The calf muscle pump may become ineffective due to following:

1. Sedentary life style
2. Occupation that require prolonged standing like bus conductor, tea master, work supervisor, manual labourer etc
3. Musculoskeletal conditions like paraplegia, multiple sclerosis, etc.
4. Advanced age which is usually associated with decreased elasticity of the calf muscle tendon.
5. Reduced mobility
6. Altered shuffling gait that fails to induce effective calf muscle contraction.

The venous disorders responsible for reduced calf muscle functions are:

1. Direct calf perforating vein incompetence.
2. Superficial system incompetence.
3. Deep venous system incompetence.

DIRECT CALF PERFORATING VEIN INCOMPETENCE:

The high pressure in the deep vein system is unlikely to have direct effect on the skin and subcutaneous tissue. The pathways by which these high venous pressure are conducted to the superficial and dermal venous plexus are by the perforating vein system. These calf perforating vein valve incompetence are the sole reason for the venous ulceration in the ankle. Cockett, after the demonstration of the venous flow from deep to superficial venous system through incompetent perforating vein, coined the term ankle blowout[22]. He too considered the fact that calf perforating vein incompetence is an important factor in the venous ulceration.

Dodd and Cockett also made an important point that the direct perforating vein of the lower leg and ankle communicate with the deep veins of the calf close to the soleal arcades. Incompetence of these perforating vein will therefore transmit a very high intramuscular pressure directly to their tributaries. Hence result in ankle flare. These malleolar veins visible as the ankle venous flare or corona phlebectatica when distended by high

pressure resulting from perforating vein incompetence. High pressure and flow in the malleolar vein are responsible for the dilatation of corona phlebctatica, which results in microcirculatory changes underlying lipodermatosclerosis and ulceration in the overlying skin.

SAPHENOUS VEIN INCOMPETENCE WITHOUT PERFORATING VEIN INCOMPETENCE:

Many studies deal with venous ulcer states that saphenous vein incompetence is more important than perforating vein incompetence provided the deep veins are intact. Surgical treatment for saphenous vein alone is enough for the healing of venous ulcers associated with both saphenous and perforator incompetence. A randomized controlled study has demonstrated that superficial venous surgery in addition to compression therapy promotes wound healing better than compression therapy alone.

SAPHENOUS AND PERFORATING VEIN INCOMPETENCE:

The contraindication that the perforator incompetence is necessary for venous ulceration could be explained by the basic hemodynamic as follows[14]. Although the great saphenous vein does not communicate directly with the calf perforating vein, it does communicate with posterior arch vein and the corona phlebectatica which are tributaries of the

perforating veins. Venous distension is mainly due to the intraluminal volume and pressure changes. The pressure changes in volume are a result of venous wall stretching.

The corona phlebectatica which underlies the ulcer bearing area of the leg, communicates with the distal saphenous vein through the inframalleolar vein distally and incompetent perforating vein proximally. The distal venous blood flow through the great saphenous vein is around 300 -600ml/min. the ambulatory mean venous pressure in the presence of incompetent great saphenous and perforating vein is of the order of 60 mmHg. The systolic pressure of about 140mmHg has been measured in the posterior tibial veins. This pressure will be transmitted to the corona phlebectatica through the incompetent perforating vein via the posterior arch vein.

During calf muscle relaxation, blood flows distally through the great saphenous system. Some diverges at knee to flow down into the posterior arch vein directly into the incompetent perforating vein of the calf. However most of the venous blood flow distally down the great saphenous vein into the inframalleolar veins then into the posterior arch vein from below to pass proximally through dilated calf perforating vein .

When the calf muscle contracts, the distal flow down the great saphenous vein continuous to flow, but proximal flow along the posterior arch vein

into the perforating vein is impeded by the outward pressure of about 140mmHg transmitted through the deep fascia from the deep vein by the incompetent perforating vein. Hence the resulting high pressure in the posterior arch vein are then transmitted to the tributaries of the posterior arch vein which dilate to form the corona phlebectatica. These high venous pressure in turn are then transmitted to the venules which drain the skin capillaries and these dilate and elongate resulting in the microcirculatory changes which underlie lipodermatosclerosis and ulceration.

DEEP VEIN INCOMPETENCE WITHOUT PERFORATING VEIN INCOMPETENCE:

This condition is very uncommon simple reason that as soon as the deep vein becomes incompetent, the perforating vein has to become incompetent. The explanation for that is as follows:

1. The thrombosis responsible for deep vein incompetence also damages the perforating vein valves.
2. Since the pressure in the deep veins are transmitted to the perforating vein, and the deep vein is already dilated much because of its incompetence, the venous hypertension in the deep veins also dilate the perforating veins and hence the valves of the perforating vein become secondarily incompetent.

Patient with deep vein incompetent alone will suffer from the heaviness of the lower limb, swelling especially in the calf muscle region and also ankle edema. However lipodermatosclerosis and venous ulceration does not develop until the perforator become incompetent.

DEEP VEIN INCOMPETENCE WITH PERFORATING VEIN INCOMPETENCE:

This combination results in the worst form of venous ulcers. Mostly the resulting venous ulceration is refractory to most of the surgical treatment. However various studies have shown that the surgical management along with compression bandage preferably inelastic bandaging have shown promising result in ulcer healing. The possible explanation could be:

1. The inelastic compression bandaging helps in compressing the distal great saphenous vein on the foot and ankle, thus preventing the high pressure in the deep veins to be transmitted to the ankle flare veins through the metatarsal veins and the dorsal venous arch.

2. Compression also has the direct protective effect over the skin in patients with skin changes and venous ulceration

3. Compression also minimizes the transmural pressure in the microcirculation and accelerates the flow in these vessels.

DEEP VEIN OBSTRUCTION:

The deep vein obstruction most commonly the iliac vein occurs due to the failure of the iliac vein to recanalise following thrombosis. This results in swelling of the affected limb. Patient also suffers from severe bursting type of pain on walking which is referred to as venous claudication. The occlusion of the proximal vein leads to dilatation of the deep vein and the perforating vein, which ultimately leads to venous ulceration. But this takes place at least 10-15 years after iliac vein thrombosis.

POST THROMBOTIC SYNDROME:

The 'valveless syndrome', is the primary deep vein incompetence which is very rare in deed. Usually the deep vein incompetence occurs after the thrombosis of the deep vein[31]. Immediately after thrombosis, the fibroblats, mast cells polymorphs and histiocytes occlude the vein lumen. The occluded lumen is then restored by the process of recannalisation and retraction of the clot. Unlike the primary varicose vein, where the vein wall becomes thinner and weak, in post thrombotic leg, the veins are thicker and less distensible. This is because of mainly of the deposition of collagen by the fibroblasts.

In the process of recanalisation, the valves become incompetent by two ways:

1. The valves are destroyed in the process of recanalisation

2. The valve become permanently adherent to the adjacent vein wall.

Homans described that most of the patient with venous ulcerations which is non healing in nature, has past history of deep vein thrombosis. Some patient had thrombophilia which is also an important factor for valve damage[31]. The deep vein incompetence is usually associated with perforating vein incompetence also. The perforating vein becomes incompetent by the following ways:

1. Long standing primary varicose vein with long or short saphenous incompetence, the perforating vein dilates and become incompetent

2. Following deep vein incompetence, by the effect of the back pressure and the dilated perforating vein, the perforator also become incompetent.

3. Following local thrombophlebitis and in the process of recanalisation, valve damage may occur to the perforating vein.

Deep vein incompetence with perforating vein incompetence are very difficult to treat and most of the non healing venous ulcer, the primary etiology may be because of this worst combination of deep vein and perforating vein incompetence.

MICROCIRCULATION AND VENOUS ULCERATIONS:

The venous ulcer is said to be because of the venous stasis, hence eventhough the term chronic venous insufficiency is now commonly used, the older term is venous stasis. Hommans is the first person to demonstrate that there was a causal relationship between venous ulceration and venous stasis[28]. He also demonstrated that there is shortage of oxygen to skin and this leads to tissue hypoxia, tissue necrosis and ulceration. But many studies have proved that the shortage of oxygen is not the cause for venous ulcers. It had been hypothesized that the presence of cutaneous arteriovenous fistula has further deprived the oxygen to the cutaneous tissue and lead to tissue hypoxia.

Coleridge Smith, have demonstrated that, the oxygen was increased in the skin of the patients with varicose ulcers rather than decrease in oxygen content[26]. In addition to this he also insisted that there is no oxygen barrier created by fibrin cuff. Histology and immunocytochemistry also suggest that there is no significant oxygen barrier. He insisted that the lesions in chronic venous insufficiency are due to the various inflammatory processes. The inflammatory processes are the first pathological steps that lead to remodelling of the skin by fibrosis and sclerosis leading to ulceration. The target of the inflammatory reaction is the vascular network of the superficial layer of the skin.

Capillary density also varied in the lipodermatosclerotic skin. Some areas are devoid of capillaries, Atrophie Blanche, while in other areas shows, dilated, tortuous, elongated capillaries. Endothelial abnormalities in the capillaries include, irregularity in the intraluminal surface, intracellular edema, and increased intracytoplasmic vesicles. Basement membrane appears to fuse with the surrounding tissues. Pericapillary spaces are filled with cell fragments and proteins. Fibrin cuffs are also observed in the pericapillary spaces. T- lymphocytes, plasma cells, monocyte macrophage, neutrophils are found in the endothelial surfaces.

Lymphocytes, plasma cells, macrophages, histiocytes and fibroblasts are found in the sub epithelial layer of the epidermis. The collagen fibres which lost their complete orientation are also found to be deposited in the epidermis. These lesions which are because of the inflammatory and post inflammatory process are responsible for the process of tissue fibro sclerosis – liposclerosis.

In the study of Moyses and Thomas, they have shown that leukocyte trapping to occur in the lower limbs with venous insufficiency. This attributes to the inflammatory process in the pathology of venous ulceration. Leukocyte trapping and its activation has been the important etiology in the venous hypertension and venous ulcerations.

In addition to venous hypertension, the other factors which lead to leukocyte activation are studied. The hemodynamic force plays a crucial role in the activation of leukocytes. Blood flow cessation and blood stasis leads to lower shear stress and it may activate the leukocytes. Once they are activated, they adhere to the endothelial surface with the help of the intercellular adhesion molecules. The intercellular adhesion molecules which help in the adherence of the leukocyte to the endothelial surface are intercellular adhesion molecule -1(ICAM - 1), vascular cell adhesion molecule (VCAM - 1), E and L selectins, binding to the specific leukocyte receptors. These leukocyte receptors belong to a special group called β-integrins[27]. The expressions of these markers are found in the every stage of venous dermatitis.

The other factor whose expressions are either up regulated or enhanced in the pathological process of venous ulceration and their hindrances in the healing of venous ulcers are as follows:

1. Tumor necrosis factor-α (TNF-α), is up regulated in venous ulcers and are drastically reduced once healing of the ulcer starts.

2. Vascular endothelial growth factor (VEGF), whose levels are very high in patients with venous insufficiency and with skin changes. They enhances the inflammatory process by inducing the expression of intercellular adhesion molecules.

3. Venous ulcers exudates itself have been found to inhibit the growth of human endothelial cells.

4. Urokinase plasminogen activator (uPA) and plasmin, its product, inhibits the angiogenesis and ulcer healing.

5. Transforming growth factor β (TGF β), is a cytokine whose expressions are up regulated in venous ulcers. They are related to tissue remodelling which they exhibit by stimulating the formation of granulation tissue, proliferation of fibroblasts and synthesis of collagen fibers.

6. The role of anti endothelial antibodies has also been demonstrated in the venous ulcers. Their activation leads to activation of leukocytes and hence the other process continues as discussed.

THEORIES IN VENOUS ULCERATIONS:

There are various theories in the pathophysiology of venous ulcerations. They are as follows:

1. Fibrin cuff theory
2. The white blood cell activation theory
3. Trap hypothesis
4. Combination theory

FIBRIN CUFF THEORY:

Browse and Burnand in 1982, postulated that the distended capillaries lead to leakage of large molecules like fibrinogen from the capillaries. These fibrinogen then polymerize to form fibrin cuffs in the pericapillary region. They also demonstrated that there are reduced fibrinolytic activities[27]. These fibrin cuff acts as a physical barrier for diffusion of oxygen and other nutrients into the tissues and the overlying skin. However many studies have disproved this theory and they in fact demonstrated that the transcutaneous oxygen levels are unchanged in venous ulcers in the presence of fibrin cuff.

WHITE BLOOD CELL ACTIVATION THEORY:

This is the most acceptable theory in the pathophysiology of venous ulceration. Venous hypertension causes reduce in the velocity of blood flow through the capillaries. These hemodynamic changes cause less shear force on the endothelial surface of the capillaries. Hence the venous stasis causes aggregation and activation of leukocytes. They in turn invite the intercellular adhesion molecules and the inflammatory process is activated. Leukocyte aggregation itself causes plugging of capillaries and tissue ischemia. Leukocyte margination causes migration of activated leukocytes into the surrounding tissue and which is the ultimate cause for tissue sclerosis and ulceration.

TRAP HYPOTHESIS:

The leaking capillaries leak the macromolecules like fibrin into the dermis[31]. These macromolecules then trap the growth factors and matrix proteins which are needed for tissue repair and remodelling. Hence by trapping this, they are unavailable for maintenance of healthy tissue and wound repair.

COMBINATION THEORY:

In 2003, Kalra and Gloviczki, came to a conclusion that venous ulceration is a complex process which occurs by combination of above said theories[31]. The initial event in the ulceration and skin changes are because of the extravasations of RBC's and protein molecules especially the fibrin. The release of these substances attract the WBC's. It is the migration and activation of the WBC's is the single most important pathological event for the venous ulceration. These activated WBC's release the inflammatory mediators and cause tissue inflammation and dermal fibrosis. Once the tissues are inflamed, they are susceptible to ulceration which occur spontaneously or to any trivial trauma.

INDICATORS OF VENOUS DISEASE:

1. Edema: This is because of capillary distension and elevated capillary pressures. Initially the edema is pitting involving upto knee which gets relieved by leg elevation above the level of heart. With the prolongation of disease, the gradual fibrosis of the tissue occurs and the edema become non pitting and known as 'Brawny'.

2. Hemosiderosis: The hyperpigmentation of the skin in venous disease is because of the extravasations of the RBC's and the release of their breakdown product, hemosiderin.

3. Varicosities: Varicosities are caused by the combination of venous reflux as well as venous hypertension and a predictor of venous ulceration.

4. Ankle Flare: These are nothing but the distended small veins inferior and distal to the medial malleolus, again because of the venous hypertension.

5. Atrophie Blanche : These are defined as white plaques of thin atrophic tissue speckled with tortuous vessels on the ankle with hemosiderin pigmented borders. Identifying this lesion is very crucial as these areas are high risk for venous ulceration in future.

6. Lipodermatosclerosis: It is known as ‘hypodermitis sclerodermiformis’. It denotes fibrosis or hardening of the soft tissue in the lower limb. The fibrotic changes are confined to the gaiter area and results in inverted ‘champagne bottle’ appearance. These fibrotic changes are because of fibrin deposits, reduced fibrinolytic activity, deposition of collagen in response to growth factors produced by the activated leukocytes.

7. Venous dermatitis: As discussed before, the venous dermatitis are results of the deposition of the inflammatory mediators especially the β integrins.

8. Ankle Blowout Syndrome: These refer to rupture of the small veins around the medial malleolus and usually present as a cluster of small and acutely painful ulcer[26]. It is because of underlying venous hypertension which gets reversed to some extent by applying compression bandage.

MANAGEMENT OF NON HEALING ULCER

General Investigations:

1. Hemoglobin estimation – anemia
2. Complete blood count and differential blood count
3. Serum Albumin
4. Renal function test
5. Liver function test
6. ESR: > 70mm/hr - infection, prognosis for tuberculosis.
7. CRP more sensitive than ESR
8. Blood glucose level and Glycosylated hemoglobin level (HbA_1C), fasting and post prandial for Diabetes
9. Urine sugar for diabetes
10. Wound swabs and tissue culture
11. X-ray chest for tuberculous foci
12. VDRL – syphilis
13. Local X-ray for sequestrum, tissue edema, gas in tissue planes and foreign body.

NON INVASIVE METHODS:

PULSE VOLUME RECORDING (PVR) OR PLETHYSMOGRAPHY[50]:

It measures the volume changes in a segment with each pulse beat by using pneumatic cuffs around thighs, calves, ankles feet and sometimes toes. The resultant tracings provide information about the hemodynamic effects of arterial disease at each level[36].

TRANSCUTANEOUS TISSUE OXYGEN ($TcPO_2$):

It is a non invasive measure of local oxygen supply to the tissues. Measurement of $TcPO_2$ < 30 mmHg is considered to be incompatible with healing[48].

NERVE CONDUCTION STUDY:

Estimation of nerve conduction velocity reveals 60% incidence of peripheral neuropathy in diabetes as compared to 10-20% detected clinically.

WEDGE BIOPSY:

Wedge biopsy from the edge of the ulcer is taken. Microscopically, 80% are well differentiated characterised by central structureless mass of keratin surrounded by normal looking squamous cells which are arranged

in concentric manner like onion skin. Appearance is called epithelial pearl or cell nest. 20% are undifferentiated with numerous mitosis, without keratinisation.

IMAGING MODALITIES:

CONTINUOUS WAVE DOPPLER ULTRASOUND (CWDU):

Principle: Frequency of signals reflected from moving red blood cells shift in proportion to the velocity of the cells. Output present as audible signals so that sound is heard whenever there is movement of blood in the vessel being examined. Augmentation manoeuvre is performed by compressing and then releasing the underlying veins and muscles below the level of the probe[35].

- Compression – Audible forward flow in direction of valves.
- Release – Audible backward flow through the incompetent valves.

To detect:

1. DVT
2. Saphenofemoral, saphenopopliteal incompetence.
3. Perforator incompetence.
4. Uniphasic signals: Signifies flow in one direction.
5. Biphasic signals: Signifies reversal flow with incompetence.

DUPLEX SCANNING:

It is the Gold standard modality. It uses B-mode ultrasound to image vessels and a second type of ultrasound namely Doppler ultrasound to sound the vessels. A 4 to 7 MHz multifrequency transducer is used[43]. With duplex ultrasound the 2 dimensional anatomic images are displayed, a particular spot in the image can be selected for Doppler shift measurement of flow direction and velocity[34].

In normal veins, cephalad flow phase with inspiration is indicated by the blue colour in the lumen. This is enhanced with distal thigh or calf compression. On release of compression, reflux is shown in red colour that lasts for 0.5 seconds. In the absence of reflux, the lumen is black. Examination should start from foot to the IVC. Veins are assessed for the

1. Presence of spontaneous flow
2. Compressibility
3. Respiratory phasicity
4. Distal augmentation should be seen within.

Flow cessation with valsalva manoeuvre is also studied.

COLOUR DOPPLER IMAGING:

DVT is well assessed by colour Doppler imaging.

Acute phase of DVT: Shows an abnormally large, non compressible vein with no colour flow within. Proximal extent should be assessed because the thrombus progresses proximally and hence there is an increase in chance of pulmonary embolism [35].

Chronic phase of DVT: It shows cord like, poorly distending vein with loss of respiratory phasicity. Intraluminal synechiae may be present. Destruction of venous valves lead to deep vein reflux with flow reversal seen in vein on valsalva manoeuvre.

Saphenofemoral or saphenopopliteal junction incompetence may be well visualised with dilated tortuous superficial veins. Superficial varicosities are also well marked out[36].

Perforator incompetence can be well made out. Perforator more than 4 mm in diameter is considered as incompetent and surgically also it is significant.

Collaterals may be well visualised. Endovenous laser treatment of long or short saphenous vein could be done under USG guidance.

VENOGRAPHY:

Lower extremity venography: This technique is used to be the gold standard in the diagnosis of deep vein thrombosis.[37]

Ascending venography: It is the common investigation done before the Doppler ultrasound. A tourniquet is applied above the malleoli and vein of the dorsal venous arch of the foot is cannulated. Water soluble dye is then injected and it flows into the deep veins. X-rays are taken below and above the knee level. It detects:

1. Any block in the deep vein
2. Extent of the block
3. Perforator status

Descending venography: It is done when ascending venogram is not possible and also to visualise the incompetent veins. Here the contrast material is injected into the common femoral vein while patient is in standing position. X-rays are taken to visualise the deep vein and the incompetent veins. It is used to:

1. Distinguish the primary valvular incompetence from thrombotic disease
2. Level of deep vein reflux
3. Morphology of venous valves.

MAGNETIC RESONANCE VENOGRAPHY (MRV):

Sensitivity and specificity of MRV are about 100% and 96% respectively. It is the most sensitive and specific test for deep and superficial venous disease in the lower limb and the pelvis. Injection of Gadolinium is useful for determining the age of the thrombus[38].MRV is particularly useful when unsuspected non vascular causes of leg pain and oedema may often be seen on the scan image when the clinical presentation erroneously suggests venous insufficiency or venous obstruction.

PHLEBOGRAPHY:

Ascending phlebography: It is a specific phlebographic technique. It identifies the level of venous obstruction. Here the contrast medium is injected into a foot vein and the following interpretation can be made out:

1. Status of the deep veins
2. Detects the incompetent communicating or perforating veins.

Descending phlebography: It identifies the valvular incompetence. Here the contrast medium is injected into the femoral vein and the amount of reflux towards the periphery is assessed and graded. It shows abnormal reflux in the deep veins as well as in the great saphenous veins[38].

Phlebography should be considered only when the ultrasound examination does not give the necessary information required for decision making.

Disadvantages:

1. Invasive modality
2. Costly
3. Non repeatable
4. Exposes the patient to radiation
5. Nearly 15% of the patient undergoing venography develops new thrombosis in the due course of the time.

VARICOGRAPHY:

Here non ionic, iso-osmolar, non thrombogenic contrast media is injected directly into the variceal vein. It investigates the site, extent and connections of superficial and deep varices. It is helpful in the recurrent varicose vein.

RADIONUCLIDE SCANNING:

Tecnitium 99m – apatide binds the Glycoprotein IIb/IIIa receptor on the activated platelets and it is specific for acute thrombus. It is helpful in patients with indeterminate ultra sonogram examinations and contraindicated for any contrast medium[38].

D-DIMER:

It is a plasmin mediated breakdown product of cross linked fibrin. It indicates the presence of intravascular fibrin. It is highly sensitive but not specific for deep vein thrombosis. Various methods of measuring d-dimer levels are:

1. Conventional membrane ELISA
2. Micro plate ELISA
3. Latex agglutination
4. Whole blood agglutination assays.

PLETHYSMOGRAPHY:

It is a non invasive method which measures the volume changes in the leg. It is of two types;

1. Photo Plethysmography
2. Air Plethysmography.

Photo Plethysmography: Using a probe transmission of light through the skin, venous filling of the surface venules which reflects the superficial venous pressure is measured. Initially patient performs dorsiflexion of the ankle for 10 times to empty the venules and then the pressure tracing falls on the photo Plethysmography. Patient takes rest and refilling occurs. In normal people it occurs through arterial inflow in 20-30 seconds. In vein incompetence, filling time is faster because of backflow from vein.

Air Plethysmography: The patient is initially in supine position with veins emptied. Air filled plastic pressure bladder is placed around the calf to detect the volume changes. Minimum volume is recorded, then patient is turned to upright position and venous volume is assessed.

Venous filling index=$\frac{\text{maximum venous volume}}{\text{Time required to achieve max. venous volume}}$

Venous filling index is the measure of the reflux.

Ejection Fraction: It is the volume changes measured prior and after single tip toe manoeuvre which is a measure of the calf pump action. Increased venous filling index and decreased ejection fraction in a patient will benefit from surgery.

AMBULATORY VENOUS PRESSURE (AVP):

It is an invasive method. Needle is first inserted into the dorsal vein of the foot,connected to a transducer which measures a pressure which is equivalent to the pressure in the deep vein of the calf. Ten tip toe manoeuvres are done by the patient. Initially there will be a rise in the pressure, which then decreases and eventually stabilises with a balance. Pressure now is called the ambulatory venous pressure. Raise in ambulatory venous pressure signifies venous hypertension. Patient with AVP more than 80mmHg has got 80% chances of developing venous ulcer.

TREATMENT

Primary Strategies for correction of venous insufficiency and hypertension includes.

1) Compression Therapy
2) Limb elevation
3) Surgical procedures

Adjuvant measures are:

1) Physical therapy and Exercise
2) Pharmacologic agents
3) Routine leg elevation

COMPRESSION THERAPY

It is the application of externally applied pressure or static support to the lower extremity as a means of facilitating normal venous flow.

Mechanism of Action:

Compression therapy products are designed to provide graduated pressure from the ankle to the knee and to support calf muscle pump during ambulation and dorsiflexion to augment venous return.

Compression Therapy products

Compression of superficial tissue

Increase interstitial tissue pressure & collapsing of superficial veins

Opposition of leakage of fluid into the tissue.

Return of interstitial fluid into blood stream.

No Edema

Compression of superficial veins promotes coaptation and normal function of the valves and increases the velocity of blood flow.

LEVELS OF COMPRESSION:

An important factor in compression therapy is the level of compressions, which is the amount of pressure exerted against the underlying tissue. The level of compression provided by currently available devices ranges from less than 20mm Hg the more than 60mmHg at the ankle[46].

High levels:

Therapeutic amount of compression for venous ulcer management is 30-40 mm hg at the ankle. It is effective in controlling venous hypertension of preventing edema formation in patients with venous disease.

Lower (Modified) levels:

Lower level of compression therapy is used in patients who are unable to tolerate optimal levels (300mm Hg) of compression or who have consistent arterial disease.

Levels of compression guide for patients with LEVD [49]:

Therapeutic level upto 30 mmHg at ankle:

1. ABI greater than 0.8
2. No evidence of acute heart failure.

Modified (low level) upto 23 mmHg at ankle:

1. ABI greater than 0.5 to less than 0.8

2. No evidence of acute heart failure.

No static compression

1. ABI less than 0.5
2. Clinical evidence of acute heart failure.

CLASSIFICATION OF COMPRESSION DEVICES[52]:

1. Sustained or Static compression devices. Elastic and inelastic.
2. Intermittent or Dynamic compression devices.

Sustained or Static:

Elastic:

1. Layered compression wraps
2. Reusable single layer elastic wraps (long stretch)
3. Therapeutic compression stockings.

Inelastic:

1. Unna's boot
2. Short stretch wrap
3. Orthosis

Intermittent or Dynamic compression devices:

Pneumatic compression devices.

High compression options:

1. Layered compression wraps
2. Reusable single layer elastic wraps(long stretch)
3. Therapeutic compression stockings

Modified, reduced compression options:

1. Therapeutic compression stockings(less than 30mmHg)
2. Modified layered compression wraps (Profore light)
3. Unna's boot
4. Short stretch wrap
5. Orthosis.

Sustained compression:

Compression wraps are one of the most commonly used compression products especially during early therapy when limb volumes are changing rapidly as a result of oedema reduction. They are designed based on Laplace's law of Physics.

Laplace's Law:

It states that, sub-bandage pressure is directly proportional to tension and number of bandage layers and inversely proportional to the leg circumference and the bandage width.

Sub-bandage = Tension X Number of layers

Pressure Leg Circumference X Width of the bandage

This law only explains the application of a wrap with constant tension will create graduated pressure. The reason is that the bandage tension is held content while the circumference of the leg increases steadily from ankle to knee. Thus the self – bandage pressure will be highest at the ankle and lowest at the knee.

Most commonly used wraps:

1) Nonelastic paste wrap (Unna's boot)
2) Layered Elastic wraps.(U.K)
3) Single layer short stretch bandages (Europe & Australia)

Non- elastic compression:

These devices augment the function of calf muscle pump. They are indicated in patients who are actively ambulating and are safe for patients with co existing arterial disease.

E.g.: Non- elastic paste bandage, orthotic devices.

Non elastic paste Bandage (Unna's boot)[51,52]:

Unna's Boot was named after a German Dermatologist Dr. Paul Gerson Unna, who introduced the use of a Zinc paste bandage to provide a comfortable but inelastic boot around the leg.

Most of the inelastic compression dressing that are used commonly is impregnated with zinc oxide, glycerine and gelatin. Some contain additional ingredients such as calamine. The bandage should be applied with the foot in dorsiflexion, beginning at the base of the toes to the tibial tuberosity below the knee.

As the paste layer gets dried when left open, most commonly it is covered with an elastic bandage or a self adherent wrap, to prevent the soling of cloth with the zinc paste bandage.

Unna's boot should be changed every 3 to 7 days or as it get loosened or wrap becomes saturated with the drainage.

ORTHOTIC DEVICES:

The Circ-Aid Thera- Boot is an orthotic device that works by augmenting calf pump function to provide a level of continuous compressions. It consists of multiple velcro straps that can be adjusted. though it is bulky, the advantage is that it can be early adjusted remould which permits more frequent bathing and wound care.

ELASTIC COMPRESION:

There devices provide compressions both at rest and during ambulation. They are used in patients who are relatively sedentary and who have a shuffling gait that fails to engage the calf muscle.

Layered bandage system:

This combines both elastic and non elastic layers to provide sustained compressions at rest and during activity. There systems are available as two layer, three layer and four layer systems.

All the system consist of one or two inner layers that afford padding of bony prominences and absorption of exudates, and 1 or 2 elastic layers that afford sustained compression. The most critical element of effective application is in the correct degree of tension.

Single layer Reusable compression wraps

Non Elastic short stretch

Compression warps

1. Provide high level support for the calf muscle pump.

2. Effective in actively ambulating patients.

Elastic long stretch

Compression wraps

1. Provides sustained Compression.

2. Used for sedentary & actively ambulating patients.

Advantage: Washable & reusable

Disadvantage:

- ❖ Limited conformability.
- ❖ Possibility of pressure injury over bony prominences.

Support stockings:

Most commonly used for patients with stable venous insufficiency to prevent ulceration. They can be used in patients with an existing ulcer once the edema has been controlled & limb circumference has stabilized.

They are not used during initiation of therapy & in severe lipodermatosclerosis.They are available in various levels of compression, in variety of colors size & styles.

Stockings are the main stay of long term compression.

Disadvantage:

Difficulty with application

Most discomfort

Cost factor

Elastic stockinette type of sleeves:

It provides consistent low level compression known as elastocrepe or Tubigrip. It can provide a compression pressure of about 18-20mmHg when applied as a double layer.

CONTRAINDICATIONS TO SUSTAINED COMPRESSION:

1) Uncompensated heart failure
2) Co-existing peripheral arterial disease

As mobilization of edema fluid into the systemic circulation could increase pre load volume and precipitate pulmonary edema.

ABI of 0.5 or less is a contraindication, as high levels of sustained tissue pressure exerted by the compression devices could further compromise tissue perfusion and cause ischemic death of the tissues.

Hence before recommending, a thorough evaluation of cardiac history, ABI measurement and any indications of heart failure should be done.

ANTIEMBOLISM HOSE /SUPPORT (ACE) BANDAGES:

They provide low levels of compression and are not used in patients with LEVD and venous ulceration.

As they stretch when the calf expands, they fails to provide calf muscle support during ambulation. They are user dependent, and are frequently applied incorrectly.

INTERMITTENT (DYNAMIC) PNEUMATIC COMPRESSION (IPC):

They are used for patients who are immobile or who need higher level of compression that can be provided with stockings on wraps. It involves the use of an air pump to intermittently inflate a sleeve applied to the lower extremity. It can be used as an adjuvant to sustained compression therapy.

It should be applied once or twice daily for 1-2 hours each time. These devices vary in terms of inflation - deflation cycle, amount of pressure exerted against the leg and the number of compartments in the sleeve.

Single compartment sleeves inflate and deflate on a cyclic basis, whereas multicompartment sleeves provide for a sequential compression in a distal to proximal milking compression wave.

Benefits of the IPC therapy:

- ❖ Mobilisation of interstitial fluid (edema) back into the circulation.
- ❖ Enhanced venous return.
- ❖ Increased arterial inflow.
- ❖ Exert antithrombotic and vasodilator effects, as a result of the marked increase in the velocity of blood flow and the resultant shear stress at the level of the endothelial cells.

They contribute to healing of long- standing venous ulcers which failed in other modalities of compression therapy. It is an appropriate therapy for most of the patients with mixed arterial and venous disease and for patients with pure arterial disease.

It is not generally used for patients with an active thrombus because of the potential for embolism and for the patients with uncompensated heart failure.

Limb elevation:

It is a simple but effective strategy for improving venous return by the use of gravitational forces. It is an important component of management for any patients with venous insufficiency.

It is an essential element of therapy for those who are unable to adhere to compression therapy regimens.

In this, the affected limb should be elevated above the level of the heart for at least 1 to 2 hours twice daily as well as during sleep. They should avoid prolonged standing or prolonged sitting with their legs in dependent position.

"Legs up" chart should be maintained and reviewed at each visit to reinforce the importance of leg elevation.

TABLE 1 - AGE RELATED INCIDENCE

S .NO	AGE GROUP	NO. OF PATIENTS	PERCENTAGE
1.	Upto 30 – 40 years	8	13.3%
2.	40 – 60 years	42	70%
3.	Above 60 years	10	16.67%

The incidence of non healing ulcer was maximum in the age group of 40 – 60 years. About 70% of the people were in this age group. This implies the fact that non healing ulcer increases with age.

TABLE – 2 SEX - WISE INCIDENCE

TYPE OF ULCER	MALE		FEMALE	
	NUMBER	PERCENTAGE	NUMBER	PERCENTAGE
1.VENOUS	32	53.3%	2	3.33%
2.DIABETIC	10	16.67%	6	10%
3.TROPICAL	6	10%	-	-
4.OTHERS	4	6.67%	-	-

About 86.67% of the patients were male and the remaining 13.33% of the patients were female in this study. The incidence of non healing ulcer is low in female. Among this 53.3% of male patients were having venous ulcer whereas 10 % of the female patients were suffering from diabetic ulcer.

TABLE – 3 DISTRIBUTION OF CASES

S.NO	SIDE	NO. OF CASES	PERCENTAGE
1.	UNILATERAL	48	80%
2.	BILATERAL	12	20%

About 80% of the patient had non healing ulcer in the one lower limb. Remaining 20% had non healing ulcer in both the lower limbs. Most of the non healing ulcer occurring bilaterally are of diabetic in origin.

TABLE – 4 DISTRIBUTION OF CASES

S.NO	SIDE	NO .OF CASES	PERCENTAGE
1.	RIGHT	14	23.33%
2.	LEFT	34	56.67%
3.	BOTH	12	20%

About 56.67% of patients has ulcers on the left lower limb whereas 23.33% of patients had the ulcer on right lower limb. Again the incidence is more of venous ulcer. The reason for that is the venous pressure on the left is higher than the right. It is because:

1. Left common iliac vein joins the IVC at an angle
2. The right common iliac artery crosses left common iliac vein immediately after its bifurcation, causing compression of the vein.
3. Loaded sigmoid colon compresses the vein in pelvic brim.

TABLE – 5 INCIDENCE OF OCCUPATION

OCCUPATION	NO. OF PATIENTS	PERCENTAGE
1.FARMER	15	25%
2.COOLIE	15	25%
3.COOK	6	10%
4.TEA MASTERS	5	8.33%
5.CONDUCTORS	5	8.33%
6.HOME MAKER	8	13.33%
7.WATCH MAN	6	10%

Since the geographical area belongs to an agricultural land, most of the people are agricultural labour. In this study about 50% of the patients were agricultural labourer and manual worker. The rest 50% of the population are tea master, conductors, cook, etc.

TABLE – 6 CORRELATION OF ULCERS WITH OCCUPATION

S.NO	OCCUPATION	VENOUS	DIABETIC	TROPICAL	OTHERS
1.	FARMER	11(73.33%)	2(13.33%)	1(6.67%)	1(6.67%)
2.	COOLIE	6(40%)	2(13.33%)	5(33.33%)	2(13.33%)
3.	COOK	2(33.33%)	3(50%)	-	1(16.67%)
4.	TEA MASTERS	5(100%)	-	-	-
5.	CONDUCTORS	5(100%)	-	-	-
6.	WATCHMAN	3(50%)	3(50%)	-	-
7.	HOME MAKER	2(25%)	6(75%)	-	-

About 50% of the population is of agricultural and manual workers. The incidence of venous ulcers are high in these population in contrast to conductors, tea master etc whose job is to stand for longer period. This is because the principle population is constituted by the agricultural and labourers. However among the tea master, conductor, 100% of them had only venous ulcers.

TABLE – 7 DURATION OF ULCERS

S.NO	YEARS	NO.OF PATIENTS	PERCENTAGE
1.	< 1 YEAR	10	16.67%
2.	1-5 YEARS	32	53.33%
3.	6-10 YEARS	16	26.67%
4.	> 10 YEARS	2	3.33%

About 53.33% of the patients had ulcers for duration of 1 -5 years. Most of the long duration ulcers are of venous ulcers. Diabetic ulcers also fall within five years of duration.

TABLE – 8 INCIDENCES OF VARIOUS ULCERS

TYPE OFULCERS	NO. OF PATIENTS	PERCENTAGE
1.VENOUS	34	56.67%
2.DIABETIC	16	26.67%
3.TROPICAL	6	10%
4.OTHERS	4	6.67%

In this study, venous ulcer constitutes about 56.67% of the non healing ulcer. Next comes the diabetic ulcer. The venous ulcers are known for their long duration and recurrences.

TABLE – 9 INCIDENCE OF INFECTIVE ORGANISMS

S.NO	ORGANISMS	NO. OF PATIENTS	PERCENTAGE
1.	KLEBSIELLA	8	13.33%
2.	E.COLI	9	15%
3.	PSEUDOMONAS	4	6.67%
4.	STAPHYLOCOCCUS	6	10%
5.	STREPTOCOCCUS	5	8.33%
6.	FUSIFORM	2	3.33%
7.	ANAEROBES	4	6.67%
8.	MIXED	5	8.33%

The incidence of infection is higher in ulcer due to diabetes. Among these about 15% is due to E. Coli. Next comes Klebsiella with 13.33%. In this study, E.coli and Klebsiella is the most common infective organism as against most study where Staphylococcus and anaerobes is the major infective organisms.

TABLE – 10 SENSITIVE DRUGS

S.NO	SENSITIVE DRUGS	NO. OF PATIENTS	PERCENTAGE
1.	CIPROFLOXACIN	14	23.33%
2.	CEPHALOSPORINS	17	23.33%
3.	AMIKACIN	12	20%
4.	GENTAMYCIN	7	11.67%
5.	PENICILLIN	9	15%
6.	METRONIDAZOLE	5	8.33%

The most sensitive drugs are fluoroquinolones and cephalosporins. About 23.33% ciprofloxacin drug is sensitive. Next to fluoroquinolones and cephalosporins, about 20% sensitivity is there for Amikacin. Metronidazole has only 8.33% sensitivity.

TABLE – 11 FOLLOW UP

S.NO	FOLLOW UP WEEKS	VENOUS	DIABETIC	TROPICAL	OTHERS
1.	0-4	20-30%	UPTO 20%	40%	0-30%
2.	4-8	60-70 %	10-30%	75%	30-60%
3.	8-12	75-90%	30-45%	75-90%	60-80%
4.	12-16	90-95%	60%	UPTO95%	80-100%
5.	16-20	99%	70-90%	-	-

Patients were followed up for over a period of five months. About 99% of the venous ulcer wound gets healed by 16 – 20 weeks. Most of the venous ulcer wound gets healed by 15 weeks. There is delay in the diabetic wound ulcer and they also needed some other treatment like skin grafting. Tropical and other non healing ulcer heals in the same period as that of venous ulcer by around 16 weeks. During this follow up, venous ulcers doesn't recur whereas some diabetic ulcer were in the stage of non healing, and they are also associated with poor glycemic control during this follow up period.

CONCLUSION

❖ The analytical study revealed that among the 60 patient studied, most of the patients were in fourth and fifth decade. It is found that, eventhough the patient become symptomatic somewhat earlier, the ulcer manifest in the late stage. Hence it is become evident that the prevalence of leg ulcer increases with advancing age.

❖ The incidence of non healing ulcer is greater in men than women in contrast to the other western studies where there is female predominance.

❖ The mainstay of occupation of the patient population in this study is agriculture and manual work. Hence the incidence of ulcer appears to be more in this group of occupation.

❖ The occupation correlation with ulcer etiology holds good in this study also as most of the venous ulcer patient are tea master, conductor, cook etc., whose occupation needs prolonged standing.

❖ Most of the patients had ulcer in one lower limb, among them most of the patients had on left side are of venous etiology.

❖ Most of the patients have ulcers for duration of around five years. The ulcers with longer duration are probably has venous etiology.

❖ In the present study most of the diabetic ulcers are infected with organisms. The prevalent organisms are E.coli and Klebsiella. The sensitive antibiotic was mostly fluoroquinolones and cephalosporins.

- ❖ Unna’s boot was applied in all the patients in the study and followed up for around six months. Patient with non healing venous ulcer showed superior and quick healing.
- ❖ Patient with diabetic ulcer responded little later. However there is a complete wound control and required additional treatment like split skin graft to cover the rest of wound in some patients.
- ❖ Patient’s compliance was poor in the initial part of the treatment, but once the patients were educated properly, the compliance improved.
- ❖ Since daily dressing or frequent interval of dressing is not needed, it becomes more appropriate for patients with low intelligent quotient, who keep dressing for long time.
- ❖ Follow up of patients revealed that there is no recurrence of ulcer, implying the importance of compression therapy for non healing ulcers. For diabetic patients, the glycemic control along with compression therapy is still more helpful in managing and preventing the recurrence.
- ❖ In case of non healing ulcer, the management should include not only to heal the ulcer but also to prevent the recurrence. Unna’s boot except for its poor compliance and sensitivity to skin, it plays a major role in wound healing. Patients need to be on periodical review for prevention of recurrence and proper management.

BIBLIOGRAPHY:

1. The healing hand: Man and wound in the ancient world Majino. G ; 1975;90; Cambridge University Press.
2. Celsus . A.C. Medicine London; Renshaw, 1838.
3. Genius works of Hippocrates, London Sydenham society; 1849.
4. A treatment on ulcers of legs, London; Mathews, 1783.
5. Several Surgical Treatments by Wisemen. R; 1676
6. Theory and Management of ulcers. Bell. B;1789
7. Gray's Anatomy, Churchill Livingston.
8. Baynton T. Descriptive account of treating old Ulcers of the leg Bristol; Biggs, 1797
9. The Pathology and Surgery of the Veins of the lower limb. Dodd H, Cockett, 2nd edition.
10. The Return of Blood to the heart, Gardner, Fox. London, Libby, 1989
11. Aspects of venous functions in the lower limb;Ludbrook J. Springfield;Thomas, 1966.
12. Cockett, Elgan Jones. The Ankle Blow Out Syndrome, a new approach to the varicose vein problem. Lancet 1953.
13. The communicating veins of the lower leg and the operative technique for their ligation. Linton. R. R. Ann Sur 1938: 107: 582-583

14. Perforating vein interruption in the Post – phlebitic Syndrome. Negus.D. In: Bergen JJ.edn New York, 1985;195.

15. Bradbury, MacKenzie, Fegan, Chronic venous ulceration Eur J Vas Endovas Sur 2002;24: 97-104

16. Robbins Textbook of Pathology, 8th edition.

17. Humans J. The etiology and treatment of varicose ulcer of the leg Br J 2003; 24: 300-11

18. Cotton L. Varicose veins; Gross Anatomy and Development.

19. Cooper DG. Baker. Primary Varicose vein: the sapheno – femoral junction, distribution of varicosities and pattern of incompetence.

20. Negus D. Friedgood A. The effective management of venous ulceration. . Br J Sur 1995; 70:143-151.

21. Klippel-Trenaunay p, Arch Gen Med 1900; 180: 641-72.

22. Martorell F. Hypertensive Ulcers of the leg. Angiology 1950; 1: 1331- 140.

23. Sabiston. Textbook of surgery. 17th edition.

24. Coleridge Smith PD. Treatment of microcirculation disorders in venous leg ulcer. Progress in microcirculation. Vol 23 1999: 129-41.

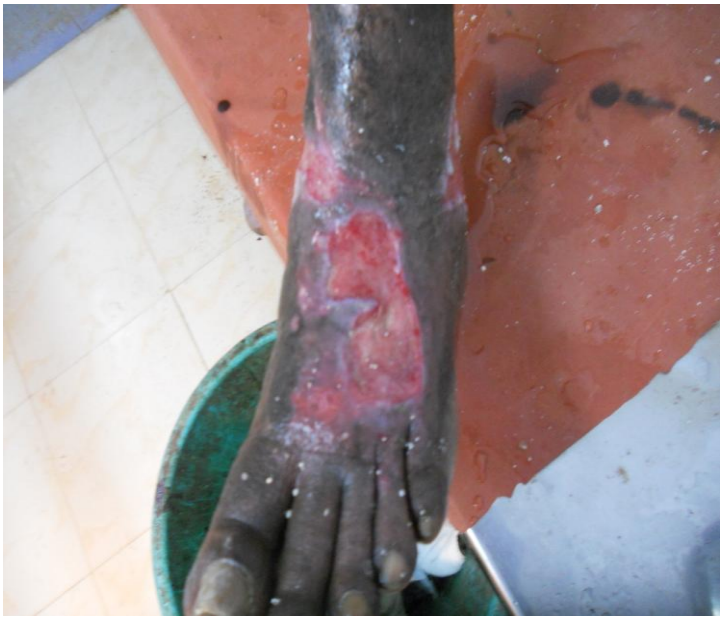
25. Franzeck, Bollinger A. Microvascular changes in chronic venous insufficiency - a review. Cardiovas Sur 1995; 3: 237-45.

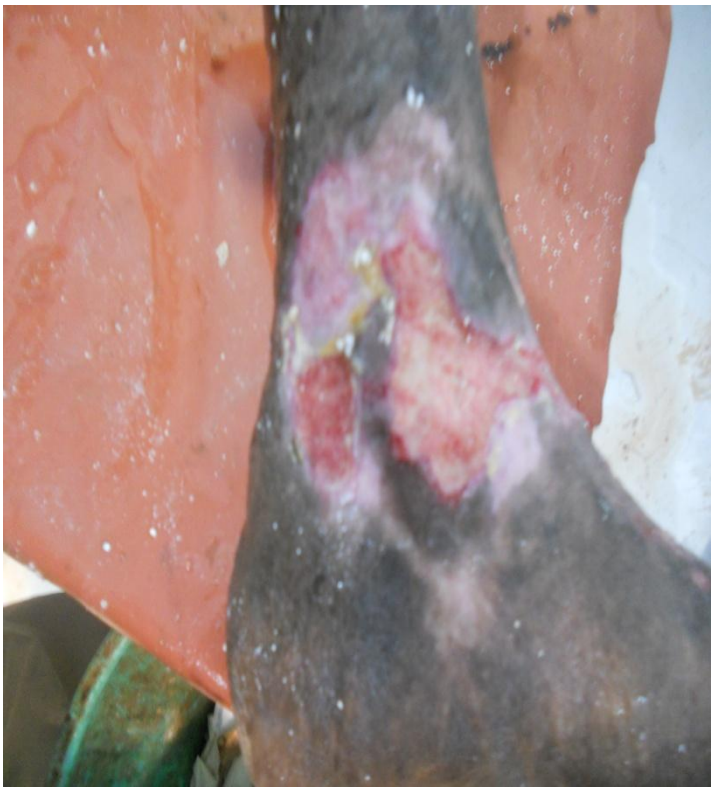
26. Shields, AbheySinghe RD et al. Neutrophil activation in experimental ambulatory venous hypertension Phlebology 1994;9: 119-24.
27. Peshen, Henning B et al Expression of intercellular adhesion molecule; 1989: 104; 345-12.
28. Cheatle TR, McMullin, Farah et al, Treatment of microcirculation disorders in venous leg ulcer. Progress in applied Microcirculation Vol 23;1999: 121-41.
29. Thomas, Nash, Dormundy, Whit cell accumulation in the dependent legs of patient with venous hypertension: a possible mechanism for trophic changes in the skin:Br Med J 1988:296;1726-7
30. Brewew , Arteriovenous Shunt, Br Med J 1990;ii:270.
31. Cederholm, Williams, Hemoconcentration and accumulation of white cells in the feet during venous stasis, Int J micro cir Clin Exp 1987:5;311-20.
32. Shami SK. Sarin S. Cheatle TR. Venous ulcers and the superficial venous system.1994;11:119-24.
33. Rutherford. Textbook of vascular surgery, 6th edition.
34. Gaitini D. Torem S. Image directed Doppler ultrasound in the diagnosis of lower limb venous insufficiency.Br Med J 1999;vol 2;89-92.

35. Polak Diagnostic ultrasound. Vol 1 1982:92
36. Eisenberg, Harrison. Vascular disease of the extremities. Diagnostic imaging. 1985.
37. Manual of vascular surgery; 2005: 112;114-45.
38. Joseph, McTaggart, essentials in diagnosis of lower extremity. 2008: 29; 753-96.
39. Chuter T et al. J vas Surg 2008; 47-6
40. Ganchi et al. case report and review of literature in lower extremity venous disease. Ann Plast Surg 2001;46:647. Review
41. Thompson, Gregharty, Lee. Basic mechanism and clinical implication in venous disorders. Cuu Probl Surg.2002; 39:110
42. Mattle HP et al, management of patients with arteriovenous malformations. J neurol 2000; 247: 917.
43. Bradbury et al. the relationship between lowerlimb symptoms and superficial and deep venous reflux on duplex ultrasonography: the Edinburgh vein study. J Vas Surg 2000;32:921.
44. Doughlas. Sumnar: duplex scanning for deep vein thrombosis. Sem Vas Surg 1996;9:3
45. Prandoni P et al. The long term clinical course of acute deep vein thrombosis. Ann Med J 1996;125:1
46. Bailey and Love's Short Practise of surgery, 25th edition.

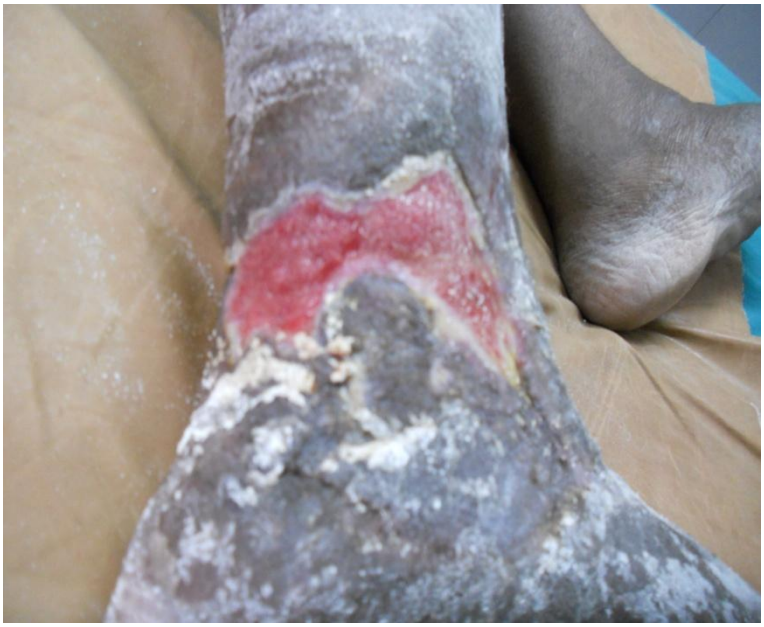
47. Hanrahan LM, Araki CT, Fisher JB et al, Evaluation of the perforating veins of the lower extremity using high resolution duplex imaging.2003;349:1695.
48. Cushieri, Essential Surgical Practice, 4th edition.
49. David Negus, Coleridge Smith,. John J. Bergan, Diagnosis and Management of Leg ulcers, 3rd edition.
50. Sakaguchi S, Tomita, Ishitoba K, Functional segmental plethismography: a new venous function test. J Vas Surg ;2008: 119; 42-72.
51. Schwartz, Principles of Surgery, 9th edition.
52. Ruth A. Bryant, Denise P. Nix. Current Management Concepts of Acute and Chronic Wounds, 3rd edition.

AFTER UNNA' BOOT APPLICATION – FOLLOW UP PICTURES

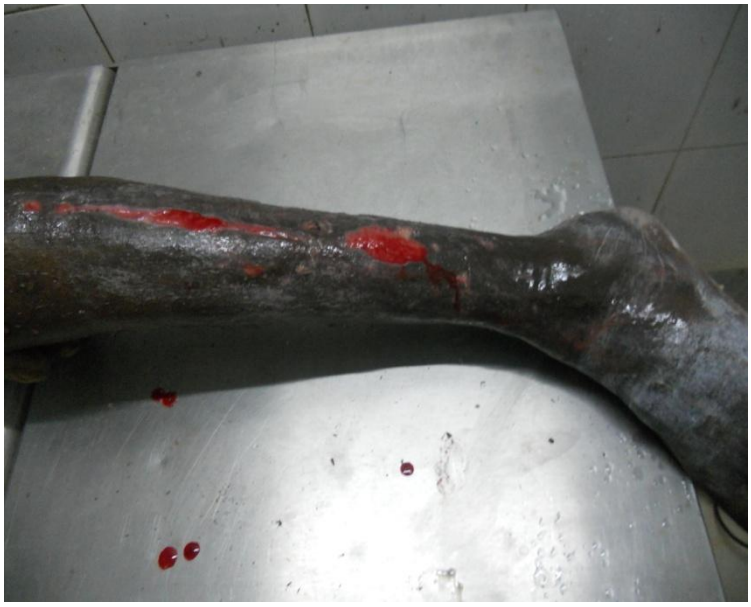




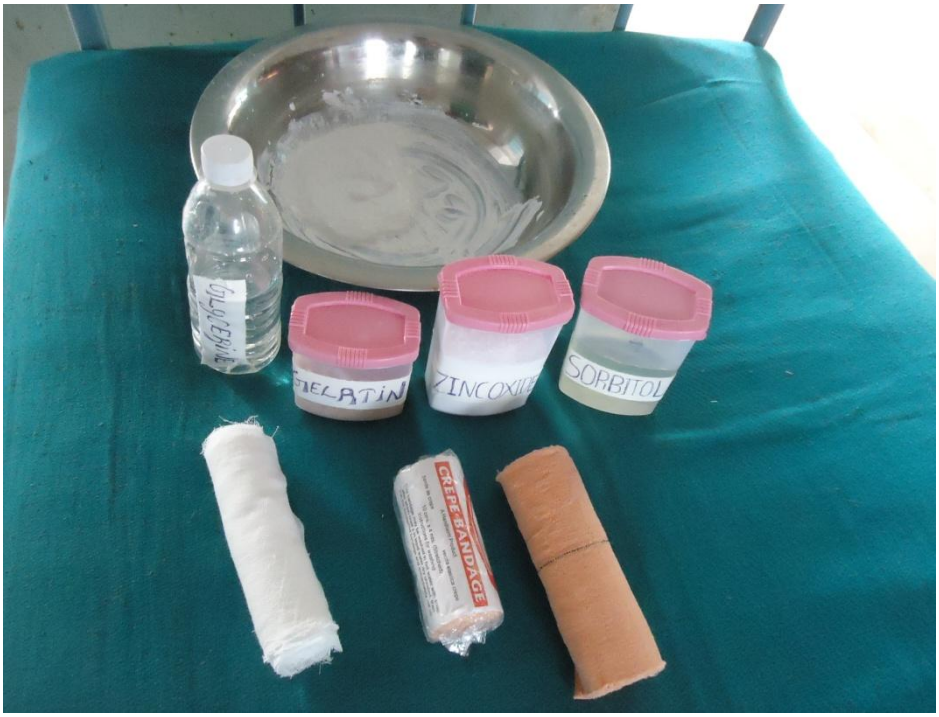
VENOUS ULCER – FOLLOW UP IN 8 WEEKS



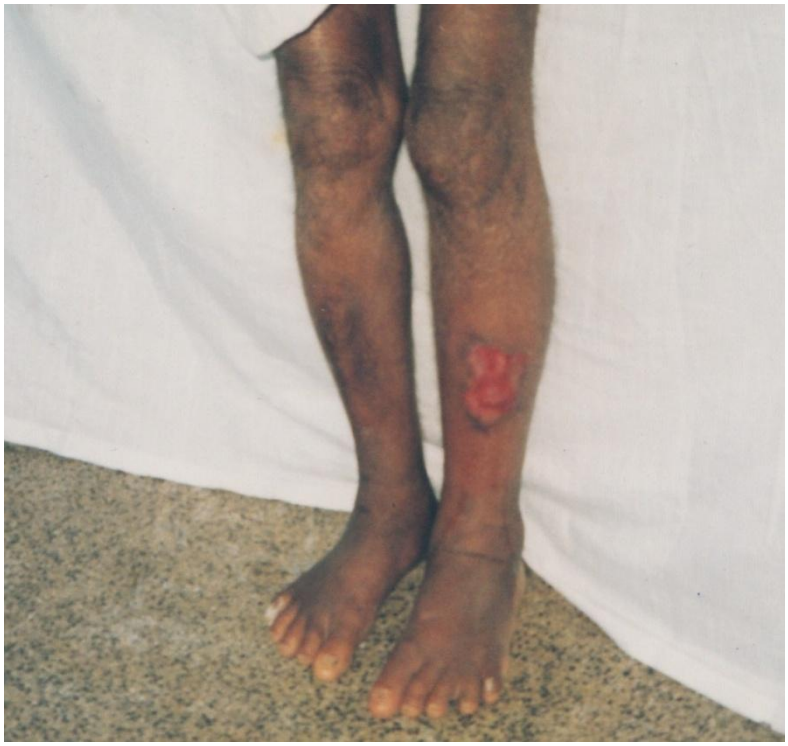
VENOUS ULCER – FOLLOW UP IN 12 WEEKS



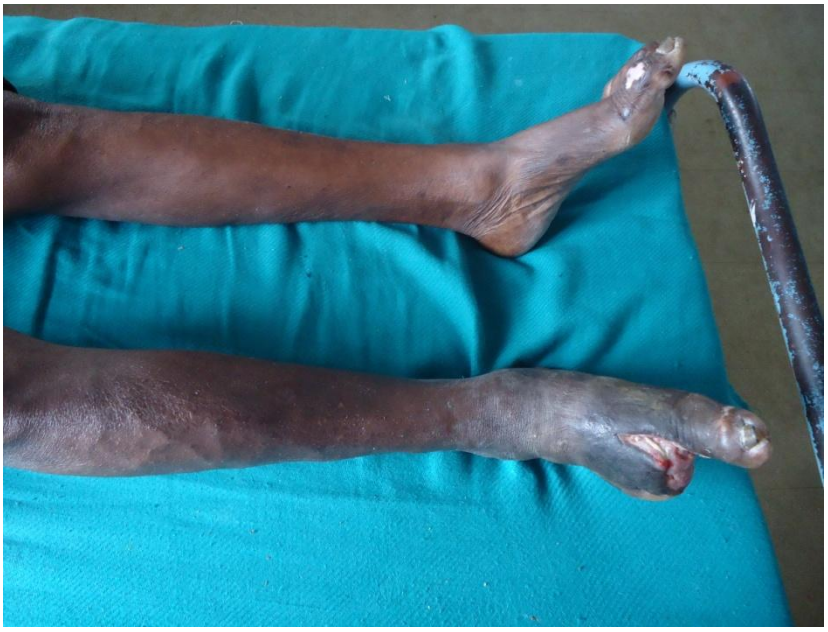
COMPOSITION USED IN UNNA'S BOOT



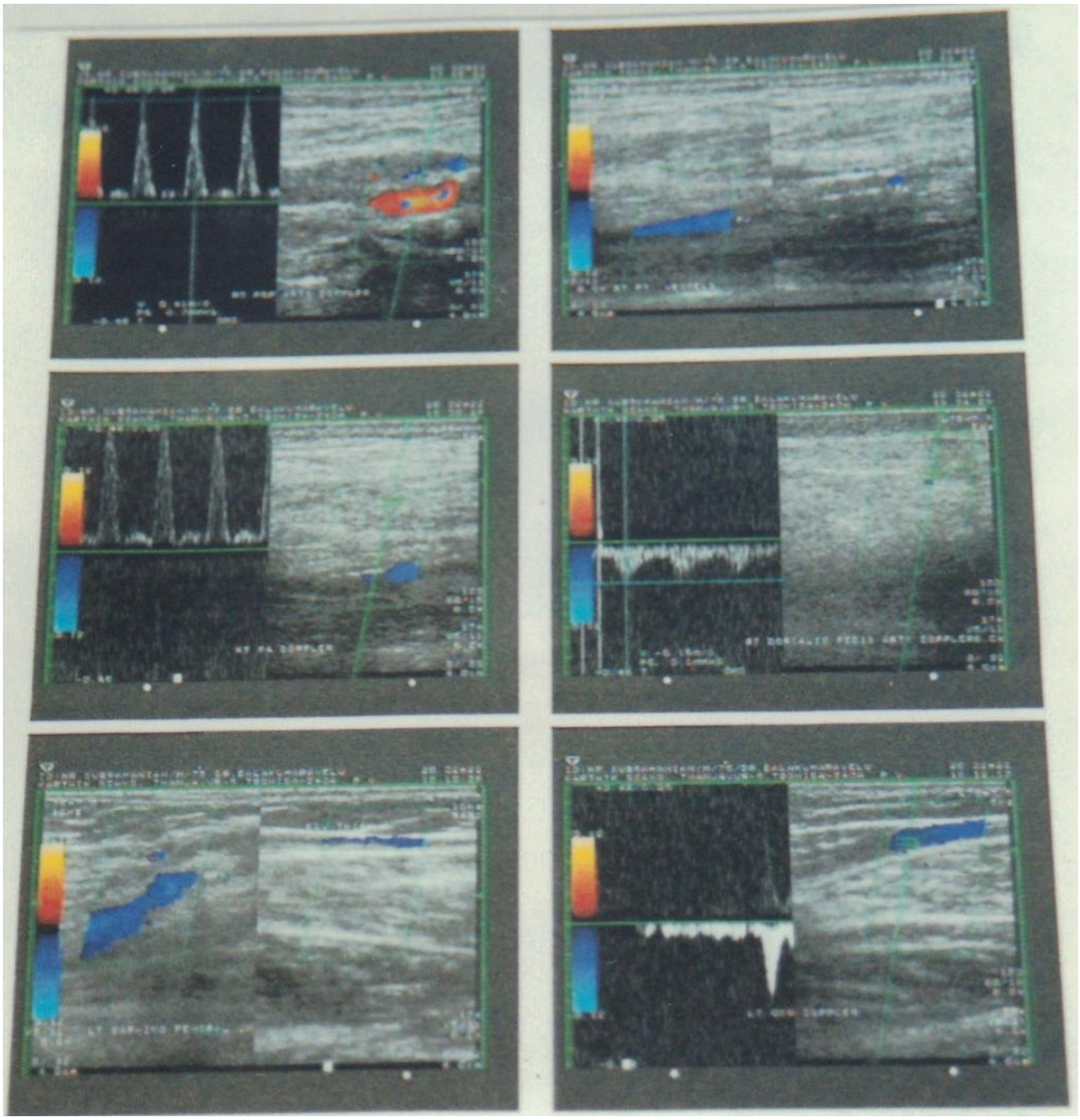
ULCER IN CONGENITAL HEMOLYTIC ANEMIA



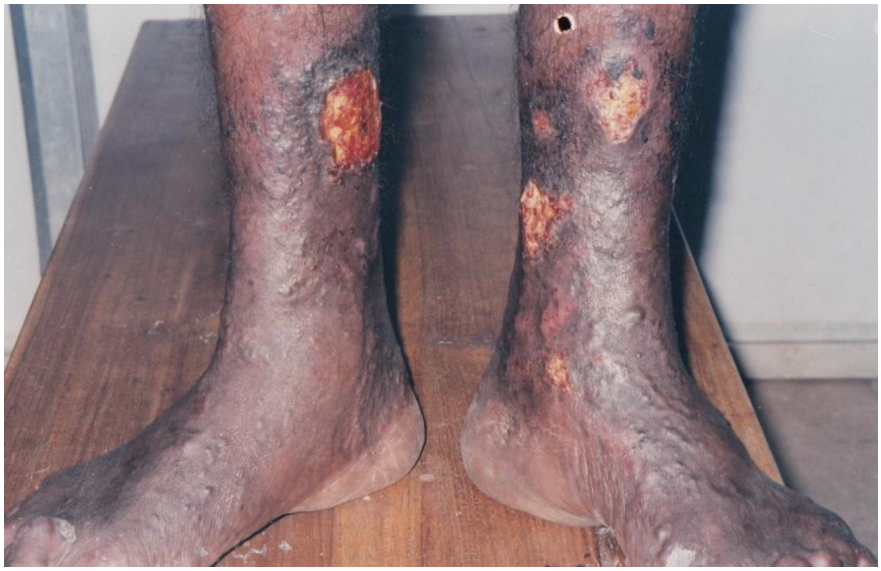
DIABETIC ULCER



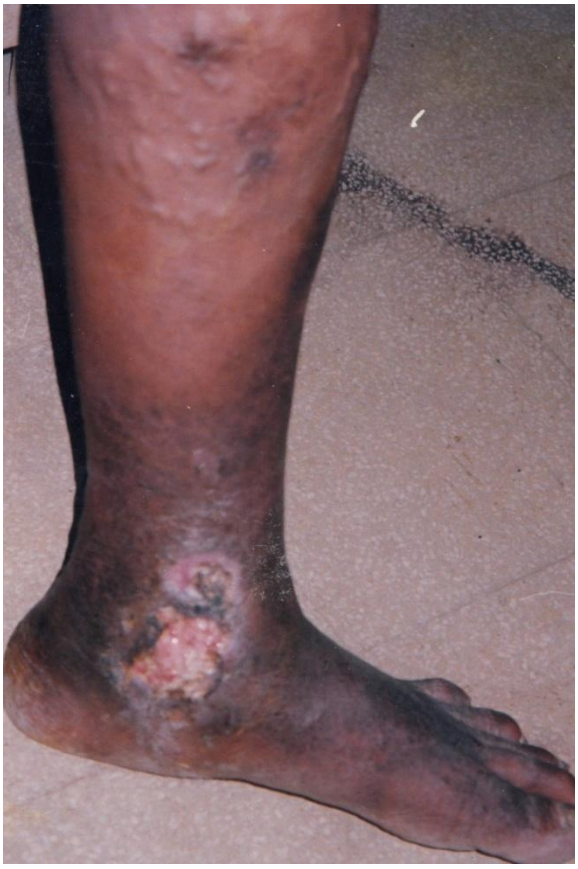
COLOUR DOPPLER USG



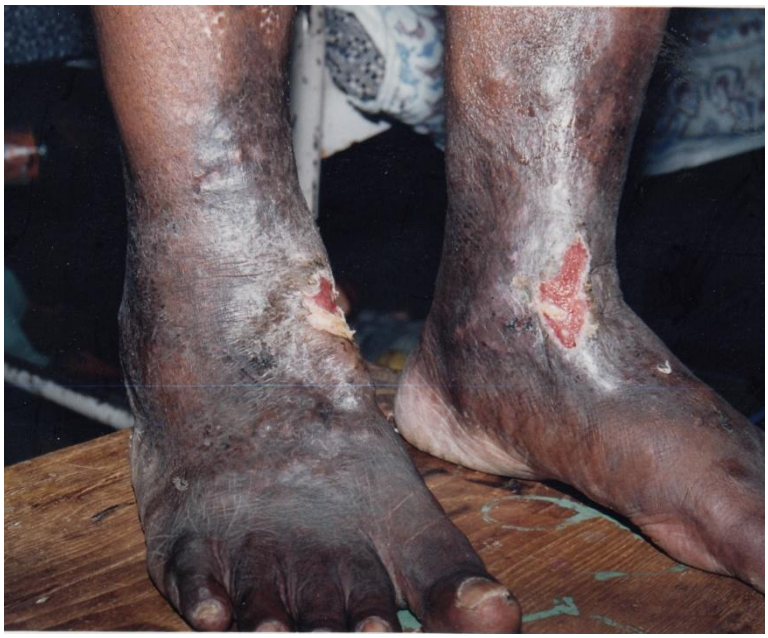
VARICOSE ULCER- BILATERAL



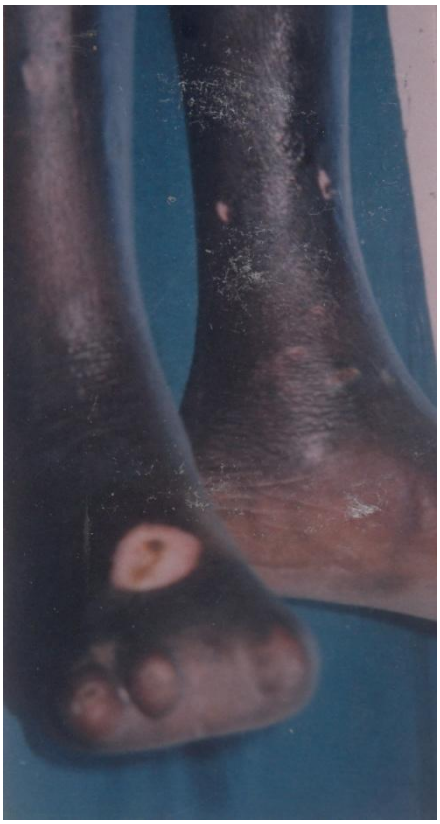
VARICOSE ULCER- UNILATERAL



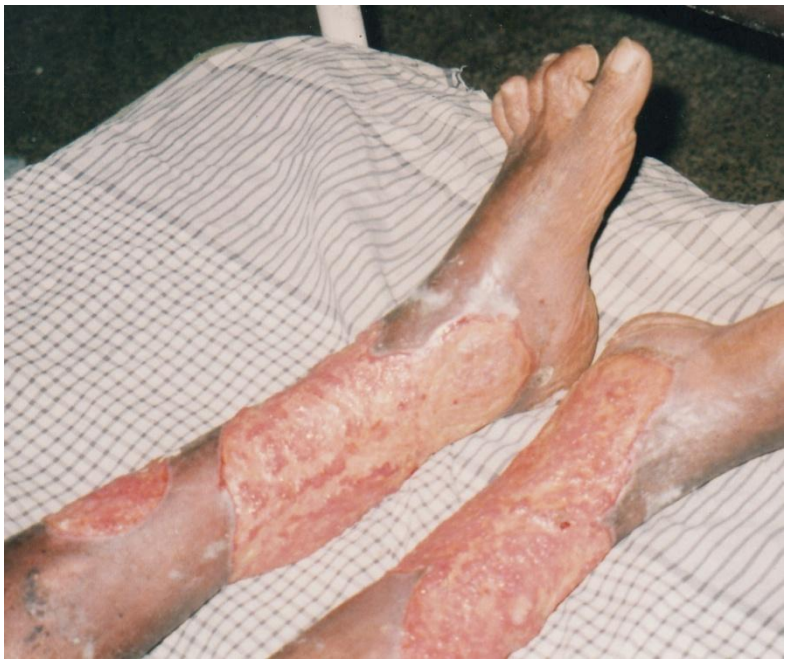
VENOUS ULCER-BOTH LEGS



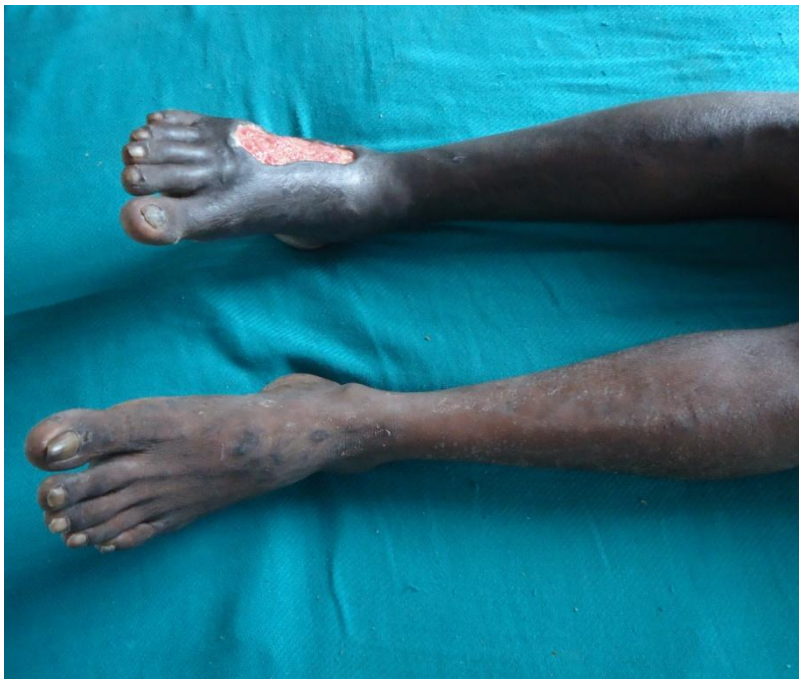
VENOUS ULCER - DORSUM OF FOOT



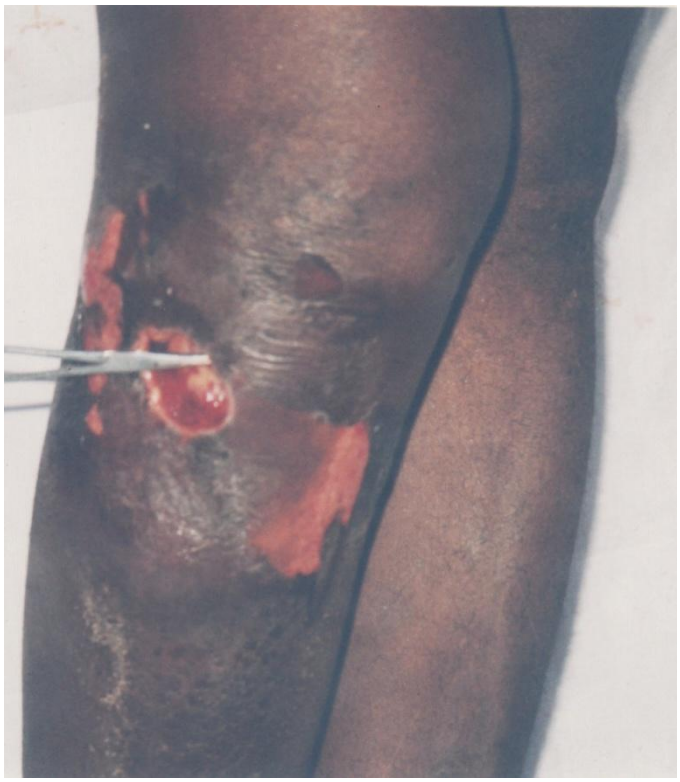
HEALING ULCER



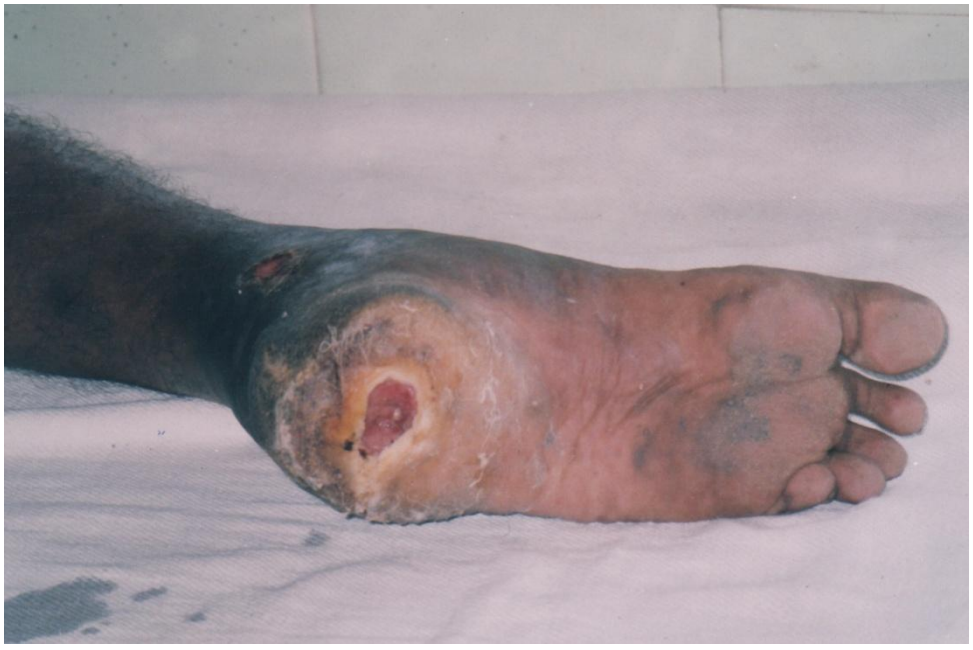
TRAUMATIC ULCER



TROPICAL ULCER



TROPHIC ULCER



HEMODYNAMIC CIRCULATION OF VEINS

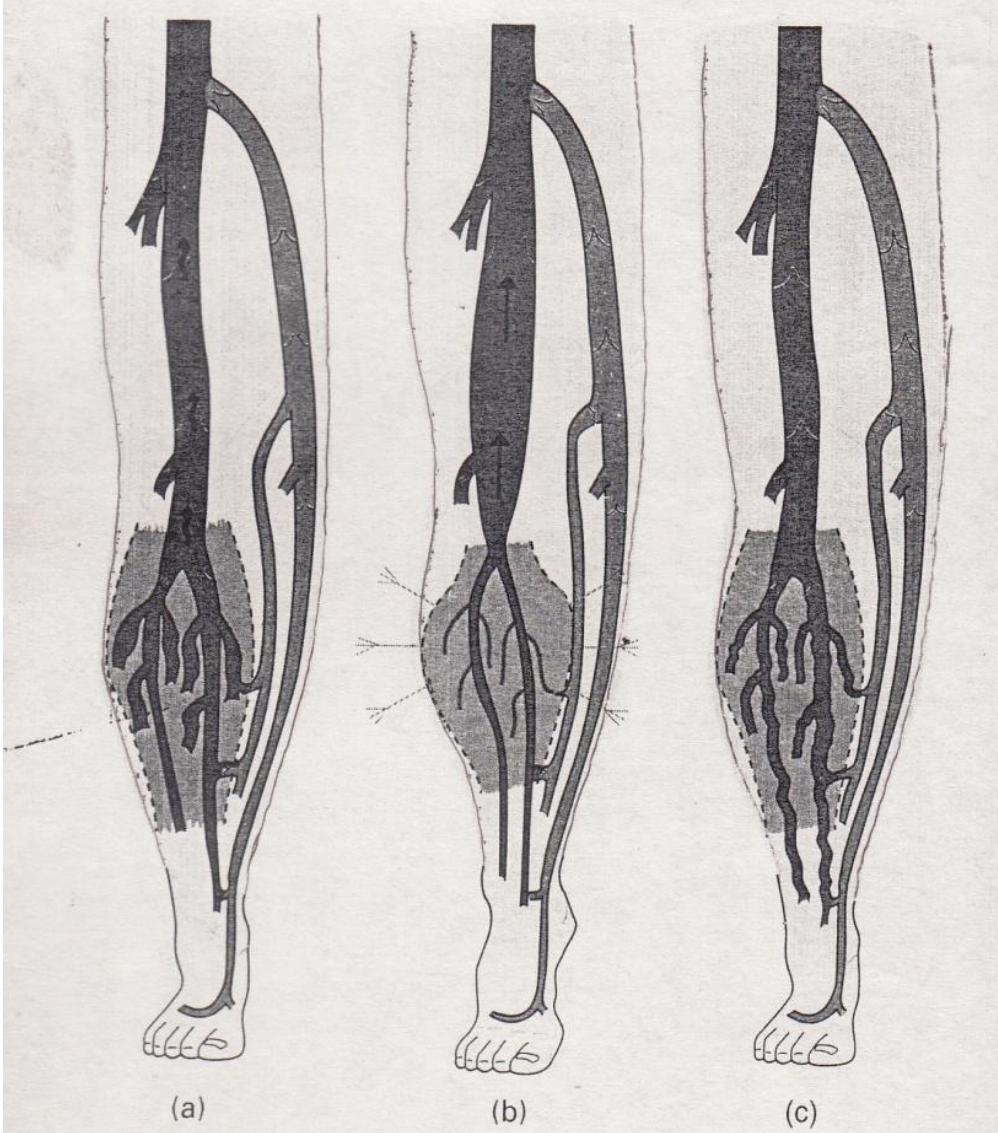
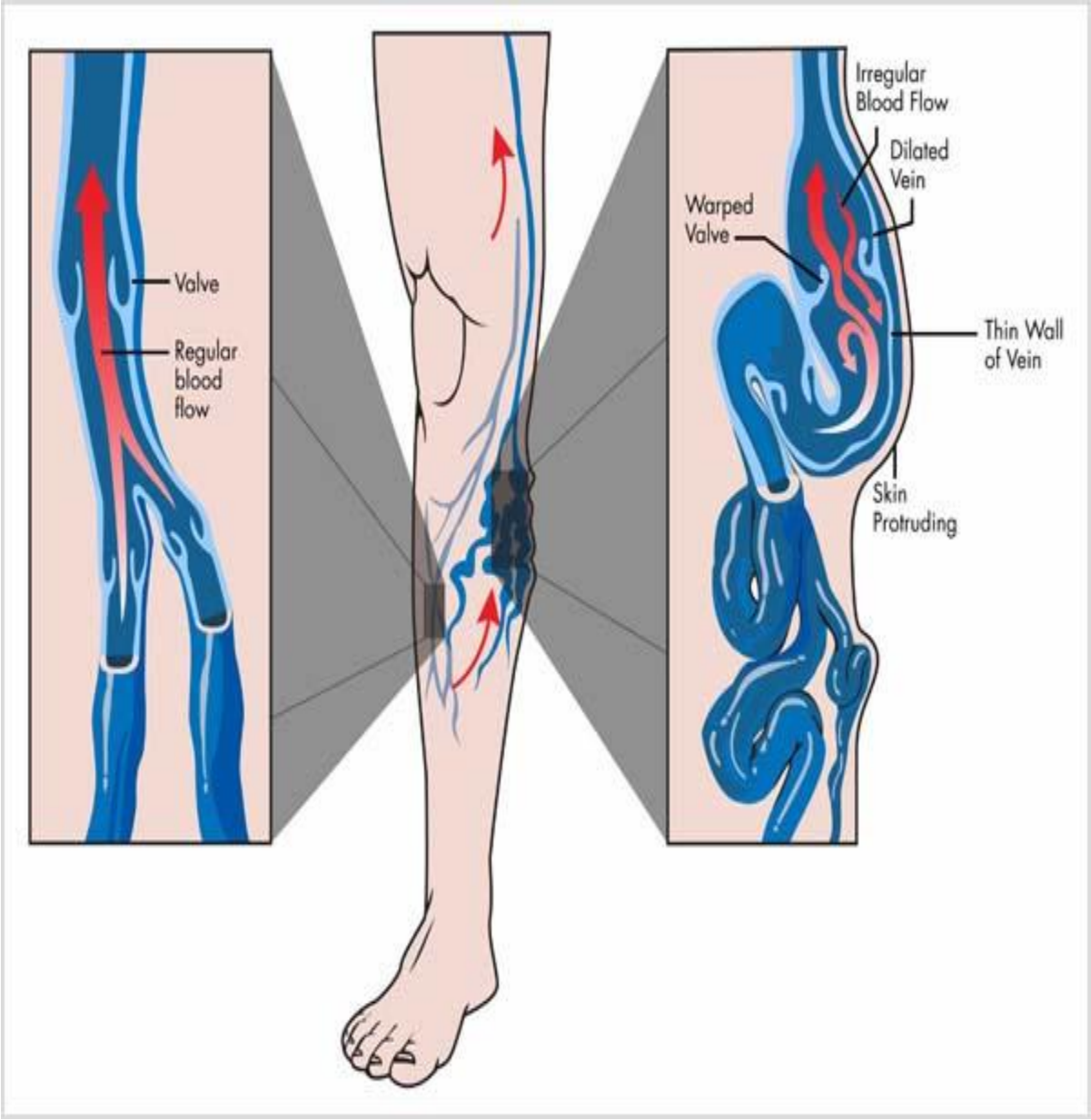


Fig. 8. Normal venous return against gravity in the upright position: (a) standing still the veins fill to capacity in about 30s; (b) with contraction of leg muscles the deep veins are compressed and empty upwards, the only direction permitted by valves; (c) with relaxation of muscle, the veins, protected from reflux by valves, are slack and refill slowly by arterial flow across capillary beds.

PATHOPHYSIOLOGY OF VALVE INCOMPETENCE



LOWER LIMB VENOUS ANATOMY

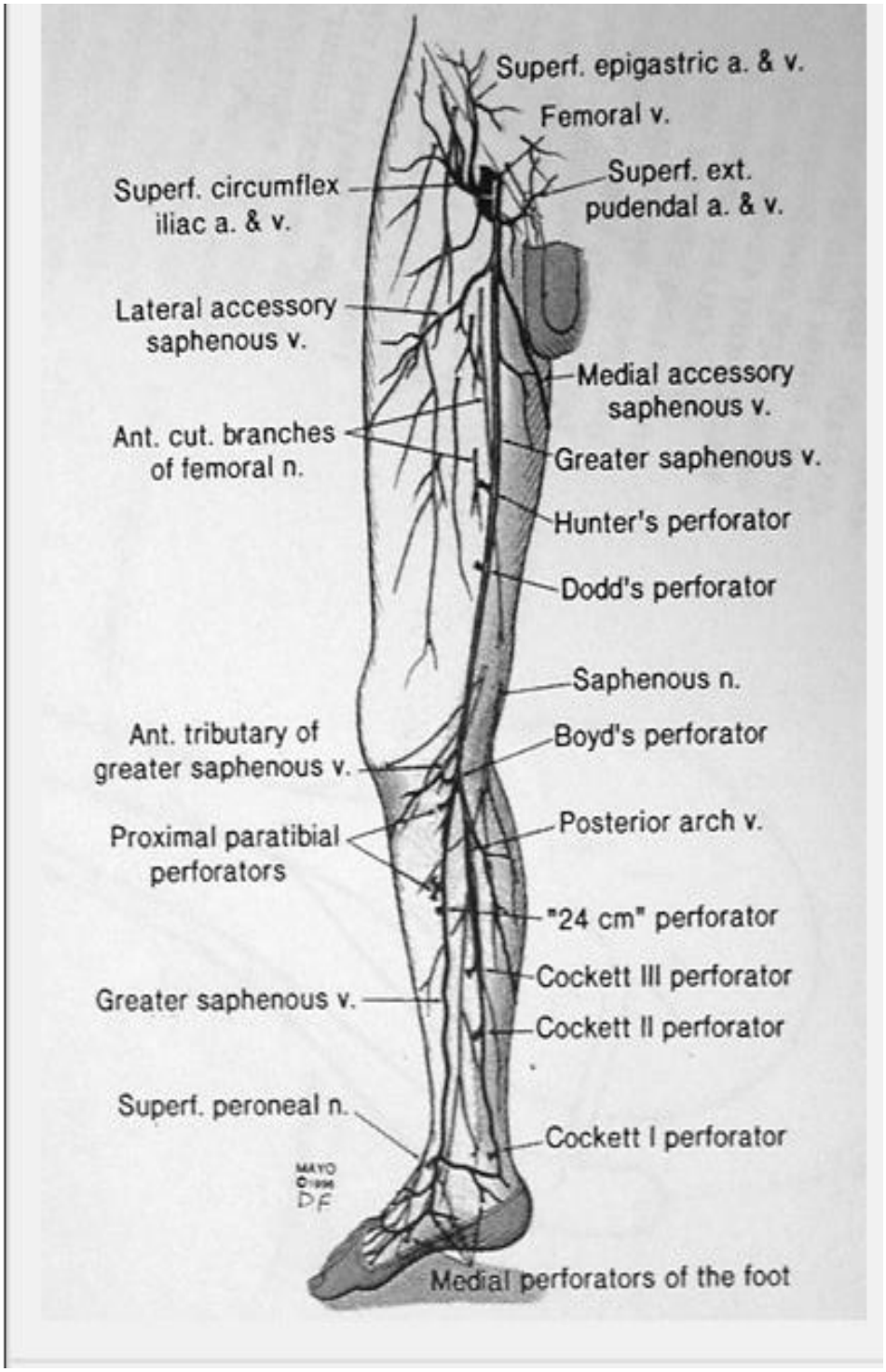


CHART 1 – AGE RELATED INCIDENCE

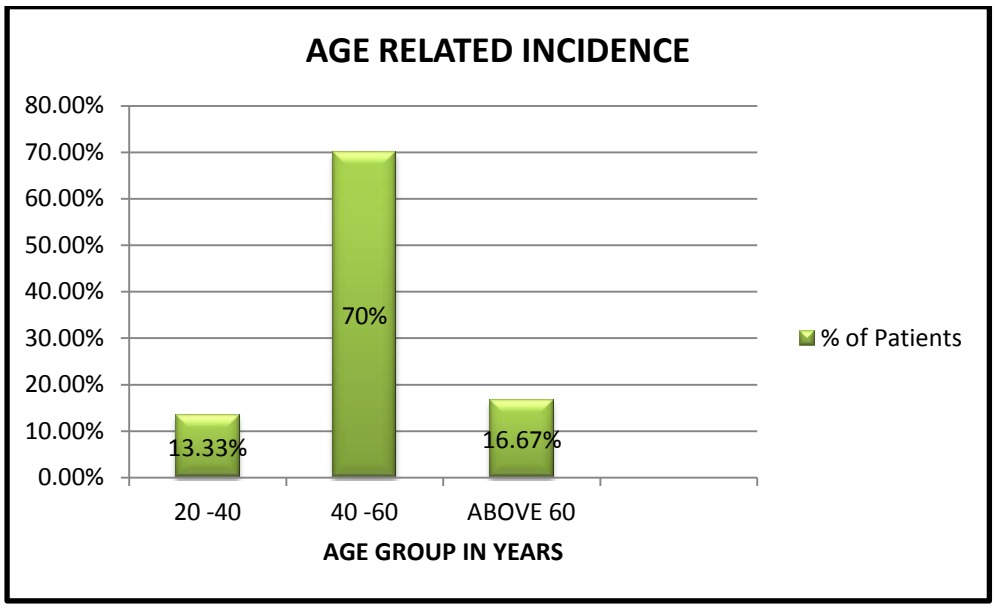


CHART – 2 SEX WISE INCIDENCE OF NON HEALING ULCER

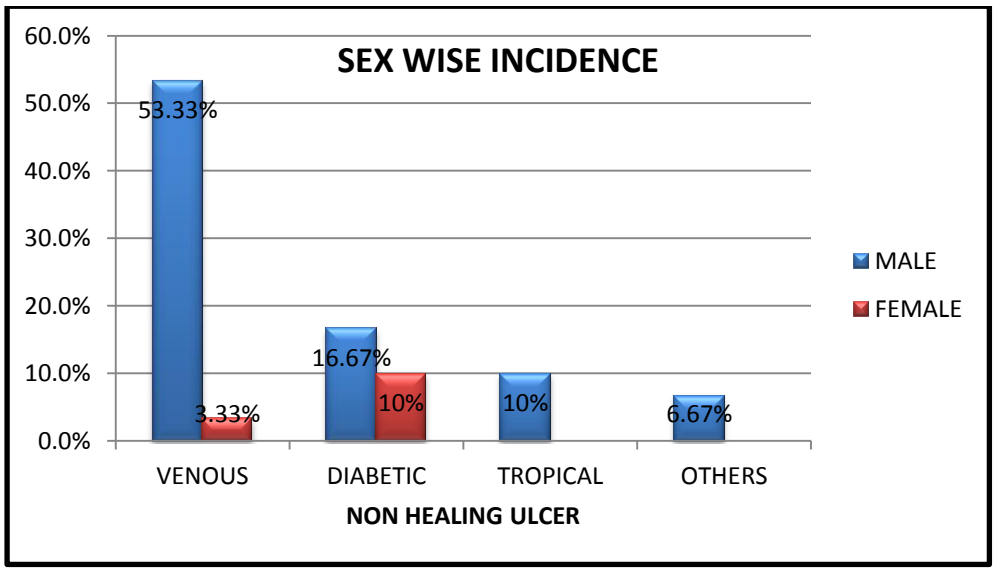


CHART – 3 DISTRIBUTIONS OF CASES

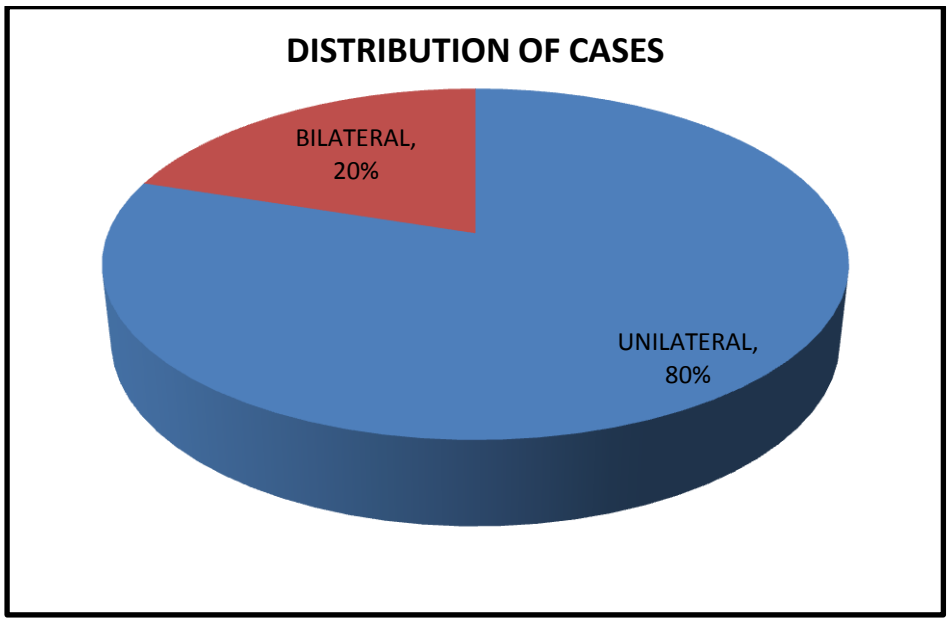


CHART – 4 DISRIBUTION OF CASES

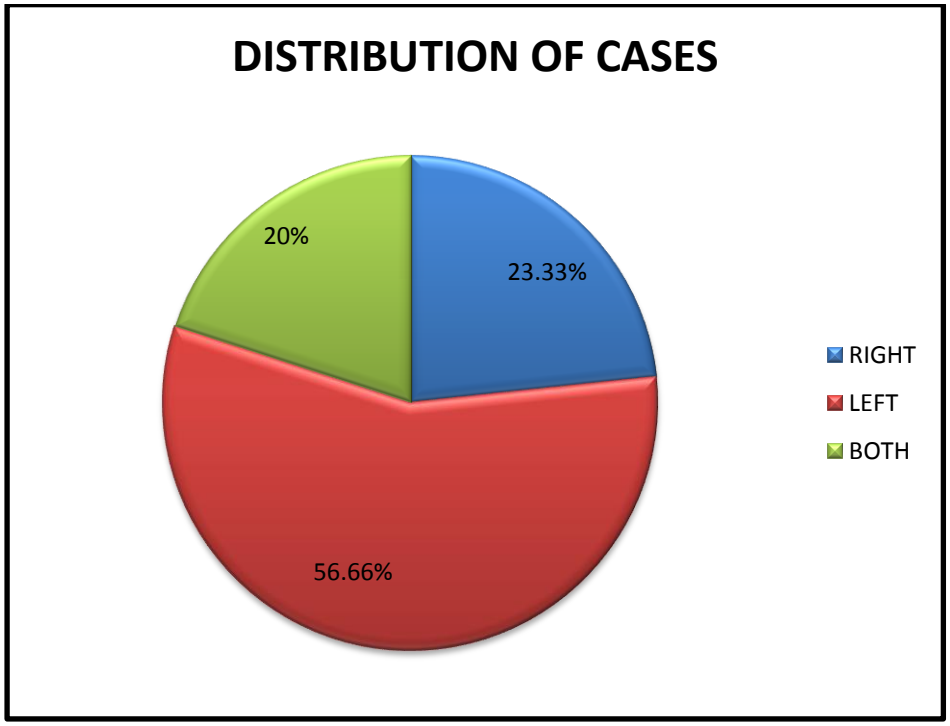


CHART – 5 INCIDENCE OF OCCUPATION

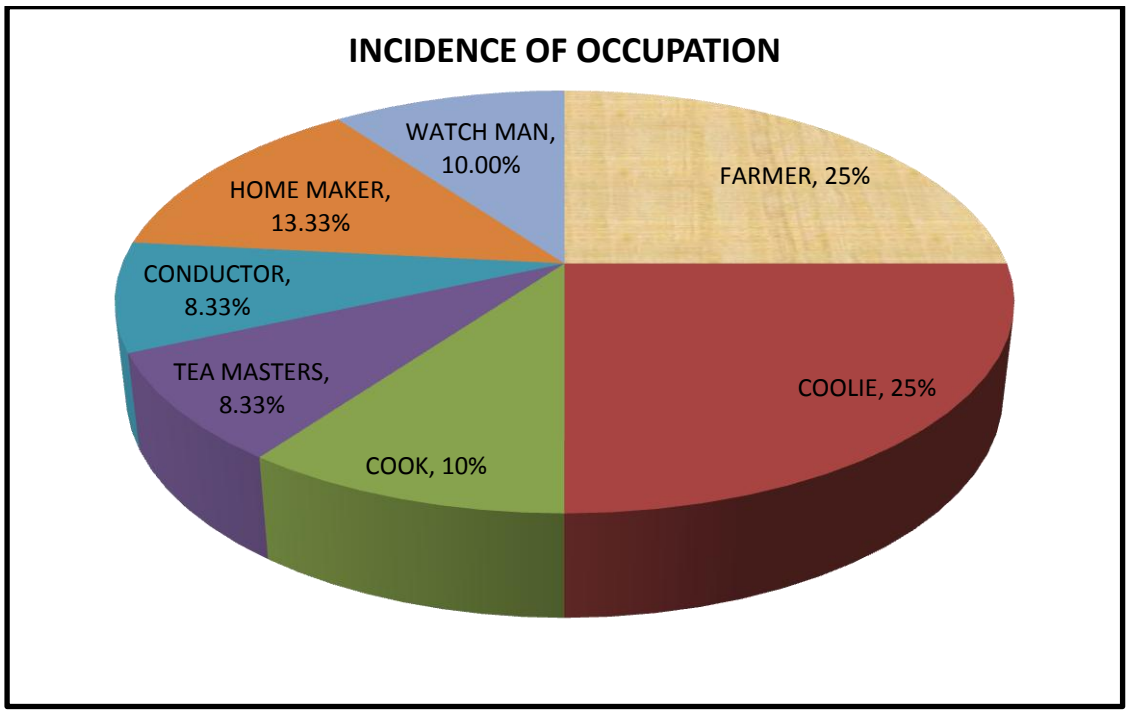


CHART – 6 CORRELATION OF ULCER WITH OCCUPATION

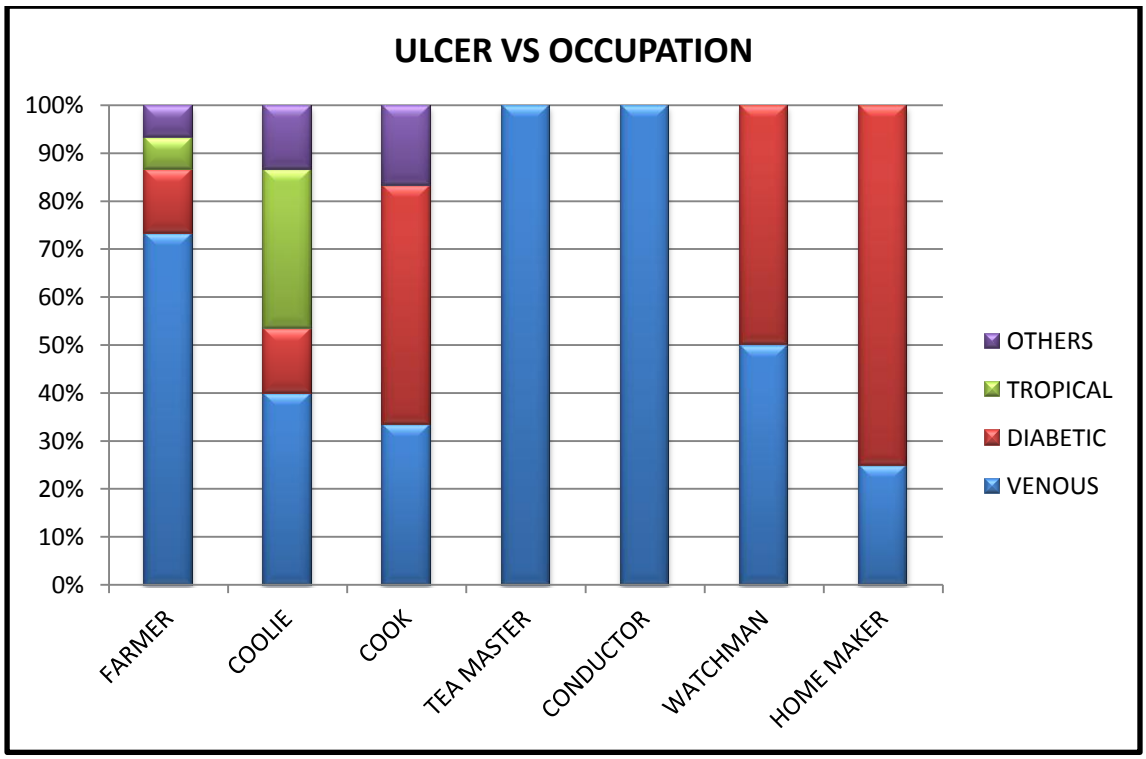


CHART – 7 DURATION OF ULCERS

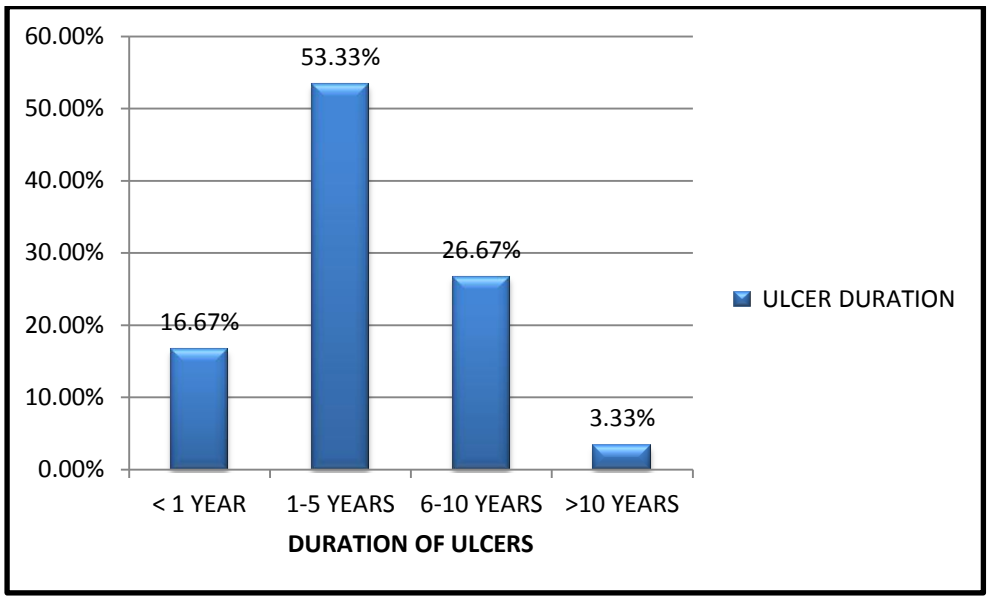


CHART – 8 INCIDENCE OF VARIOUS ULCERS

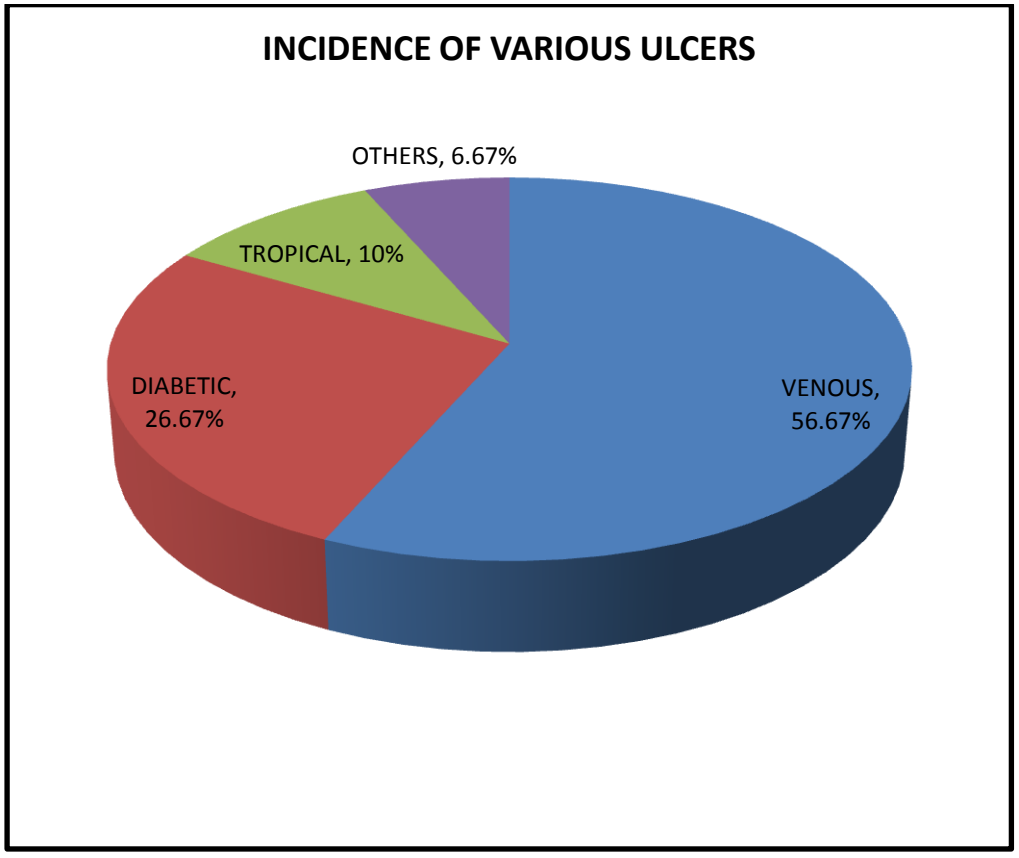


CHART – 8 INCIDENCE OF INFECTIVE ORGANISMS

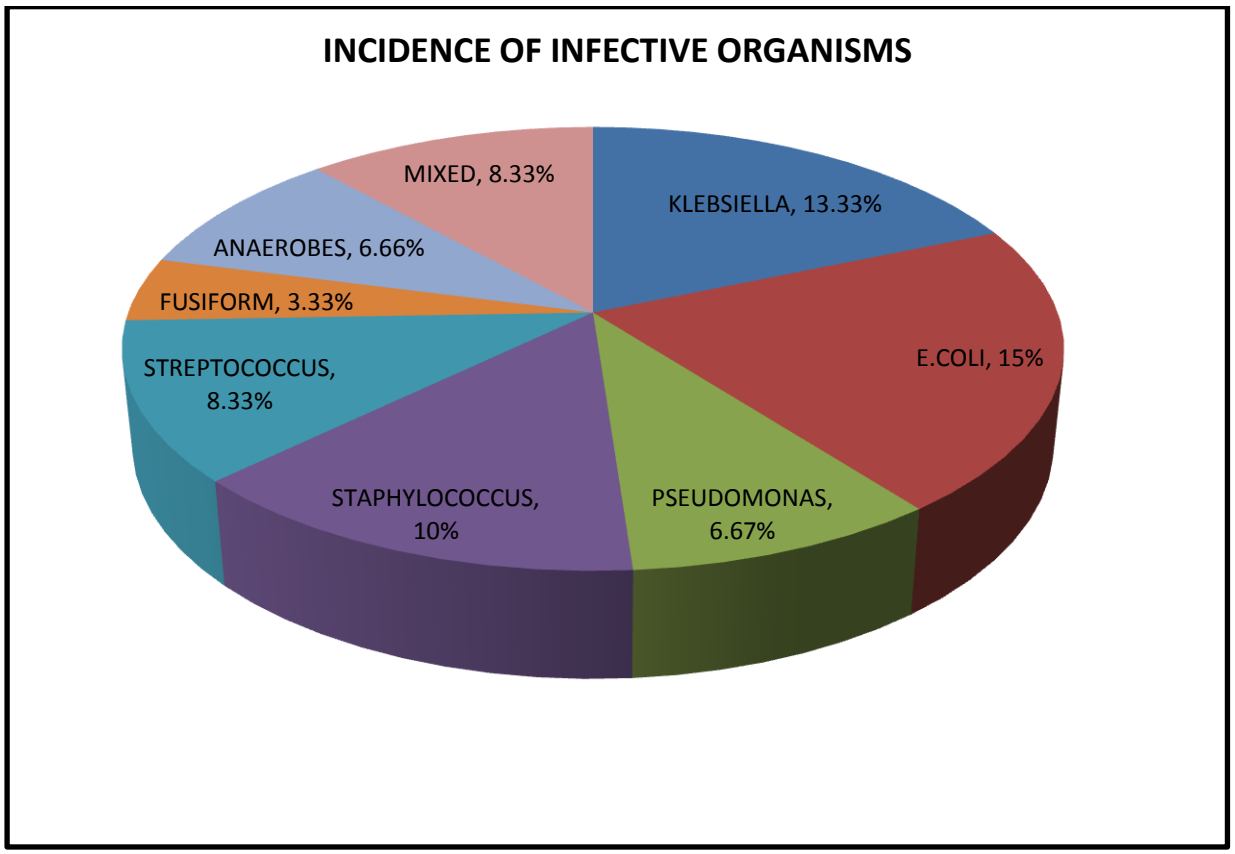
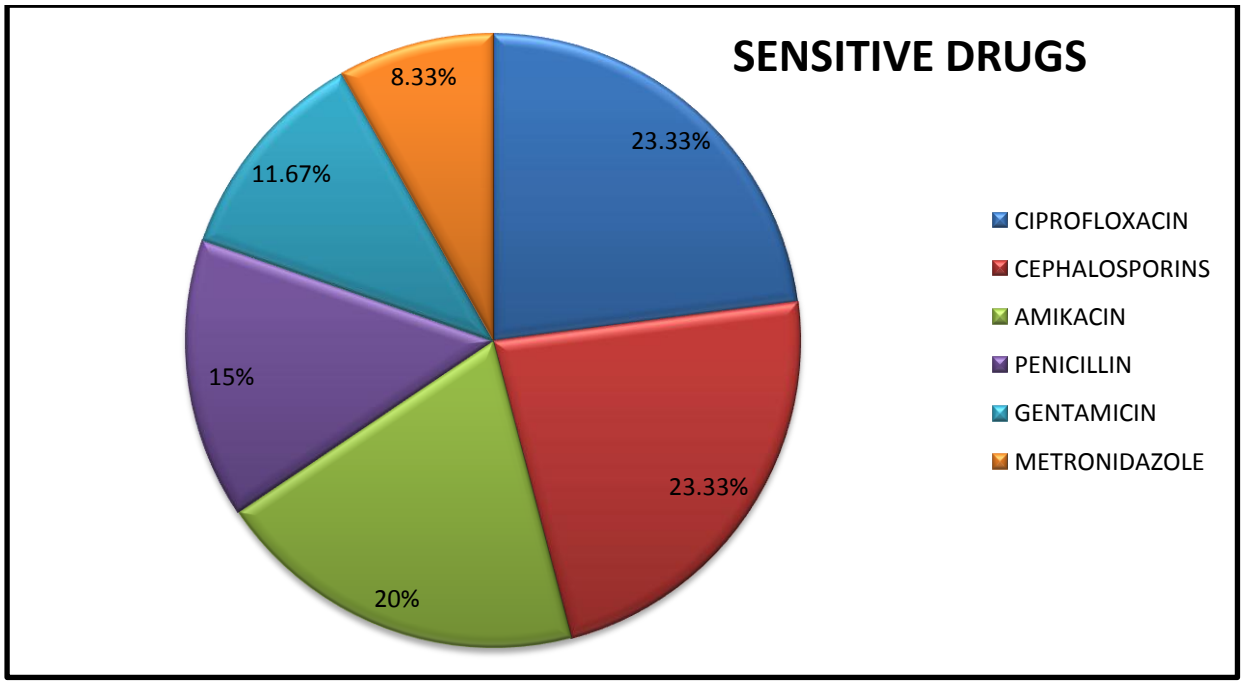


CHART – 9 SENSITIVE DRUGS



APPLYING UNNA'S BOOT

FIG 1. ROLLER GUAZE DRESSING

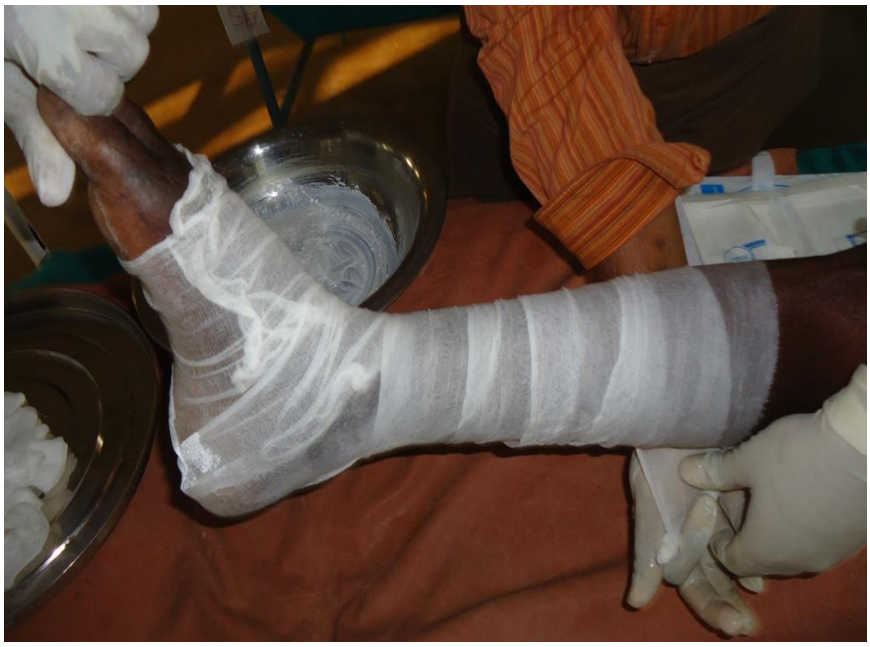


FIG 2. APPLYING INGREDIENTS

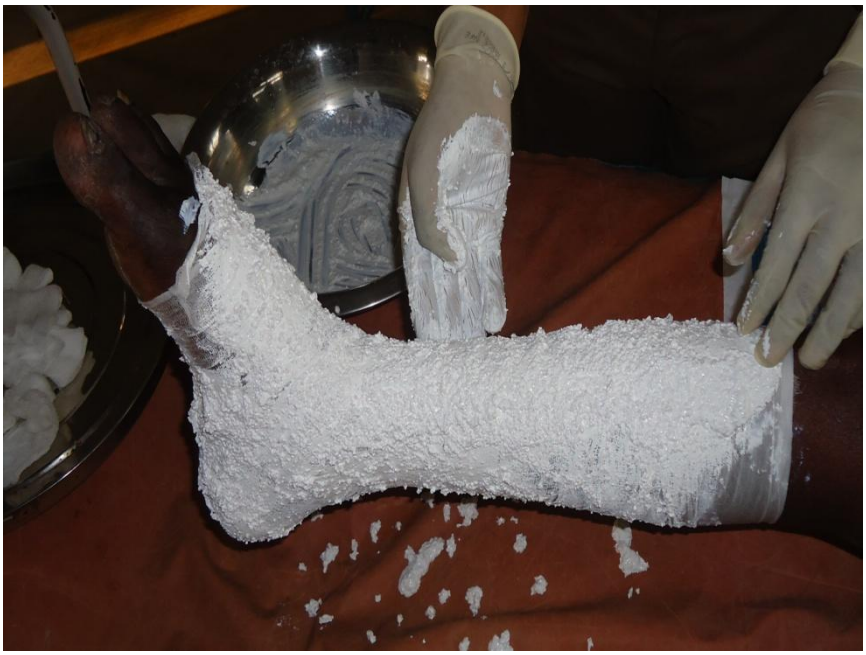


FIG 3. ROLLER GUAZE DRESSING

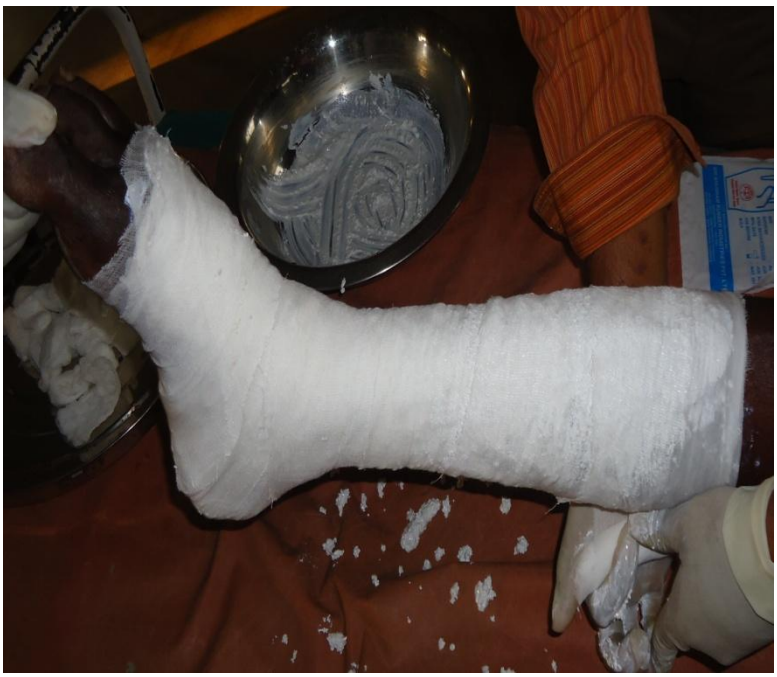
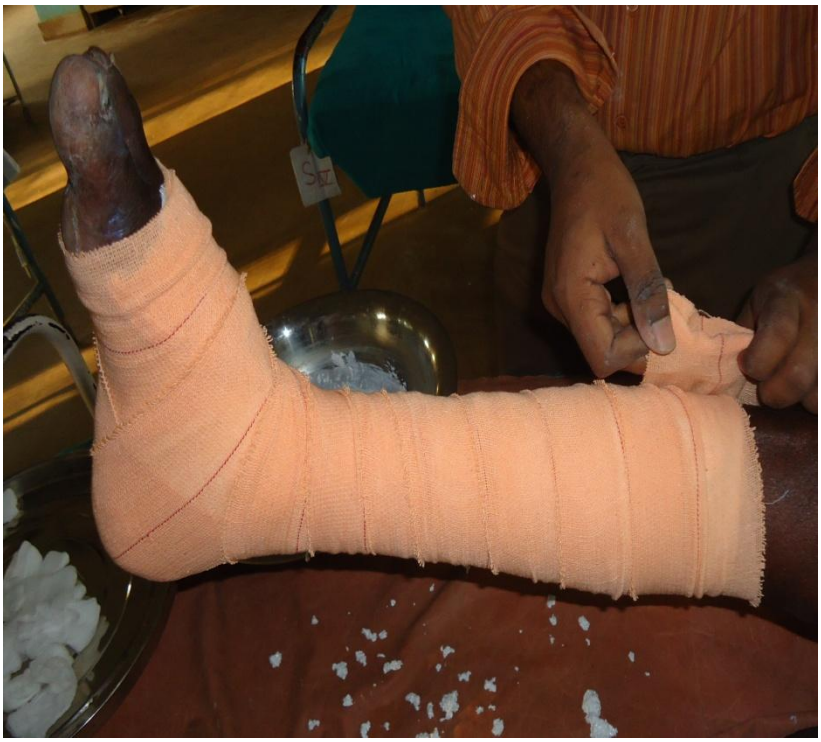


FIG 4. ELASTO CREPE BANDAGE APPLICATION



MASTER CHART

SL. NO	NAME	AGE	SEX	IP NO	OCCPATION	DIAGNOSIS	PUS CULTURE	PUS SENSITIVITY	COMPLICATIONS	HOSPITAL STAY (IN WKS)	FOLLOW UP (IN WKS)
1	RENGASAMY	60	M	1388926	FARMER	VENOUS ULCER	E.COLI, KLEB.	AMIK	WI	6	12
2	RAMAIYAN	56	M	1376831	COOK	DIABETIC ULCER	STAPH	CIPRO	WI	12	22
3	VENKATACHALAM	52	M	1397189	TEA MASTER	VENOUS ULCER	-	-	B	4	8
4	GURUMOORTHY	36	M	1400626	COOLIE	TROPICAL ULCER	-	-	WE	8	16
5	GOVINDAN	50	M	1400829	CONDUCTOR	VENOUS ULCER	ANAERO	METRO	WI, B	6	18
6	RAMAN	62	M	1401861	COOLIE	VENOUS ULCER	-	-	B, WE	14	16
7	ARULMARY	55	F	1403803	HOUSE WIFE	DIABETIC ULCER	E.COLI	CE	GR	16	14
8	NATARAJAN	57	M	1416423	WATCH MAN	VENOUS ULCER	STREP	P	WI/ITC	8	16
9	PARAMASIVAN	50	M	1419268	FARMER	DIABETIC ULCER	ANAERO	METRO	WI/WD	14	16
10	NADESAN	58	M	1426868	FARMER	VENOUS ULCER	-	-	DERM	6	14
11	RAMACHANDRAN	51	M	1427876	WATCH MAN	DIABETIC ULCER	STREP	CE	WD	14	20
12	MUNUSAMY	60	M	1428678	COOLIE	OTHERS	STREP	CE, CIPRO	WI	12	14
13	PITCHAYAMMAL	53	F	1428880	HOUSE WIFE	DIABETIC ULCER	KLEB	AMIK, GM	WD/B	12	18
14	AROKIYARAJ	48	M	1437640	COOK	VENOUS ULCER	-	-	B	8	18
15	KAJA MOHIDEEN	46	M	1438304	COOLIE	TROPICAL ULCER	FUSIFORM	CIPRO, AMIK	WE/B	10	12
16	GANESAN	80	M	1400320	FARMER	VENOUS ULCER	E.COLI, STREP	CE	WI	6	16
17	SAMINATHAN	32	M	1416326	TEA MASTER	VENOUS ULCER	-	-	B	4	10
18	UTHIRAPATHY	53	M	1427961	CONDUCTOR	VENOUS ULCER	E.COLI	CIPRO	GR/WI	4	16
19	KARRUPUSAMY	58	M	1430613	COOLIE	VENOUS ULCER	-	-	B	6	12
20	SENTHIL	47	M	1411813	COOK	DIABETIC ULCER	KLEB	CE	WD/WI	12	18
21	SELVAM	50	M	1406232	COOK	VENOUS ULCER	STREP	P	WI	8	16
22	RAMAMOORTHY	36	M	1426362	COOLIE	TROPICAL ULCER	PSEUDO	CE/P	WE	4	16
23	SUNDARAMBAL	48	F	1426912	HOUSE WIFE	VENOUS ULCER	KLEB	CE	GR	8	20
24	ANJAMMA	60	F	1436232	HOUSE WIFE	DIABETIC ULCER	E.COLI	GM/AMIK	WD	12	16
25	SHANMUGAM	55	M	1437487	CONDUCTOR	VENOUS ULCER	-	-	B	6	12
26	MOORTHY	80	M	1400865	COOLIE	DIABETIC ULCER	STAPH	CE	WI/WD	8	6
27	KANNAN	48	M	1428387	FARMER	VENOUS ULCER	KLEB	CE, CIPRO	WI	4	12
28	BALASUBRAMANY	58	M	1426833	WATCH MAN	VENOUS ULCER	-	-	B	6	14
29	KUBERAN	35	M	1427837	FARMER	VENOUS ULCER	STREP	CE, P, CIPRO	WI	4	8
30	MAHENDRAN	47	M	1408233	COOK	OTHERS	-	-	WE	6	16

31	AMUDAN	50	M	1408726	COOLIE	DIABETIC ULCER	ANAERO	METRO	GR/WI	8	18
32	ANBE	58	M	1406828	TEA MASTER	VENOUS ULCER	-	-	B	4	12
33	SUBRAMANIAN	70	M	1423786	FARMER	VENOUS ULCER	KLEB	GM	WI	6	8
34	NELLAPAN	65	M	1436802	WATCH MAN	VENOUS ULCER	-	-	B	6	12
35	CHANDRAN	48	M	1402336	FARMER	TROPICAL ULCER	PSEUDO	AMIK	WE	4	12
36	GOVINDASAMY	60	M	1426839	FARMER	VENOUS ULCER	E.COLI	CIPRO	WI/B	4	8
37	CHINNASAMY	63	M	1403807	FARMER	VENOUS ULCER	-	-	B	6	16
38	SENGAYEE	70	F	1423783	HOUSE WIFE	DIABETIC ULCER	STREP	CE, CIPRO	WI	4	6
39	BAKIYARAJ	38	M	1403831	CONDUCTOR	VENOUS ULCER	E.COLI	AMIK	B/WI	6	14
40	BABU	53	M	1427800	FARMER	VENOUS ULCER	KLEB	CE	WI	4	16
41	CHANDRAKASAN	40	M	1403666	COOLIE	TROPICAL ULCER	FUSIFOEM	GM	WE	8	16
42	SENTHIL	46	M	1404322	TEA MASTER	VENOUS ULCER	-	-	B	4	12
43	KARTHIKEYAN	60	M	1403672	COOLIE	OTHERS	E.COLI, STAPH	CE/CIPRO	WI	6	12
44	GOVINDARAJ	56	M	1402730	FARMER	VENOUS ULCER	KLEB	P	WI	4	14
45	CHELLAMAL	70	F	1403610	HOUSE WIFE	DIABETIC ULCER	E.COLI	GM	WI/WD	6	6
46	NAVARATHNAM	33	M	1423732	CONDUCTOR	VENOUS ULCER	-	-	B	4	10
47	KRISHNAN	39	M	1404371	WATCH MAN	DIABETIC ULCER	ANAERO, STREP	METRO, P	GR	8	18
48	RAMASAMY	70	M	1432140	COOLIE	VENOUS ULCER	E.COLI	CE/CIPRO	WI	4	6
49	BALAKRISHNAN	54	M	1404331	COOK	DIABETIC ULCER	STREP	CE, AMIK	WD	6	16
50	RAJAN	50	M	1432213	FARMER	VENOUS ULCER	-	-	B	6	14
51	RAJAMANIKAM	52	M	1403230	FARMER	OTHERS	PSEUDO	AMIK	WE/WI	4	12
52	MANI	60	M	1423431	COOLIE	VENOUS ULCER	-	-	B	6	10
53	PALANI	58	M	1404316	TEA MASTER	VENOUS ULCER	STREP	P	B	4	12
54	KANCHANA	50	F	1421310	HOUSE WIFE	DIABETIC ULCER	E.COLI, KLEB.	CE/CIPRO	WI	6	14
55	JAGANATHAN	56	M	1401321	COOLIE	VENOUS ULCER	E.COLI	P/GM	B	8	12
56	VEMBU	65	F	1420306	HOUSE WIFE	VENOUS ULCER	ANAERO	METRO	WD	10	12
57	MARIMUTHU	59	M	1402603	WATCH MAN	DIABETIC ULCER	STAPH	CIPRO/AMIK	WI	8	12
58	ARJUNAN	31	M	1423002	COOLIE	TROPICAL ULCER	PSEUDO	CIPRO	WI	6	12
59	RAJENDRAN	59	M	1402120	FARMER	DIABETIC ULCER	KLEB	GM/AMIK	WD	8	14
60	PANDYAN	43	M	1417237	COOLIE	VENOUS ULCER	E.COLI	CE/AMIK	B/WI	6	12

ABBREVATIONS USED IN THE MASTER CHART

M – Male

F – Female

WI – Wound Infection

WE - Wound Excision

WD – Wound Debridement

B – Bleeding

GR – Graft Rejection

ITC – Itching

DERM – Dermatitis

E.COLI – Escherichia coli

STAPH – Staphylococcus

ANAERO – Anaerobes

STREP – Streptococcus

PSEUDO – Pseudomonas

CIPRO – Ciprofloxacin

AMIK - Amikacin

GM – Gentamicin

METRO - Metronidazole

CE - Cephalosporins

P - Penicillin

PROFORMA

NAME: AGE/SEX: IP. NO:

OCCUPATION: UNIT/WARD: INCOME/MONTH:

D.O.A: D.O.D:

HISTORY:

Leg ulcer: site:

Pain:

Discharge:

Itching:

Skin changes:

Dilated veins:

Bleeding:

Deformity:

Additional complaints:

PAST HISTORY:

1. Diabetes: duration:

   Treatment: medical/surgical
2. H/O surgery for varicose vein
3. Tuberculosis
4. Hypertension

PERSONAL HISTORY: Smoker; alcoholic

MENSTRUAL HISTORY:

FAMILY HISTORY: Congenital haemolytic anaemia

Connective tissue disorder

EXAMINATION:

GENERAL: Built / nourishment

Anaemia

Jaundice PR:

Pedal edema BP:

Fever RR:

Lymphadenopathy TEMP:

LOCAL:

Inspection: Site Palpation: Temperature

Size Tenderness

Shape Edge

Margin Base

Floor Surrounding Area

Surrounding Skin Regional Node

Any Dilated Veins

Toes: Loss of hair/ brittle nail

SPECIAL TESTS:

VENOUS ULCER: system involved

1. GSV/SSV 2. Perforator incompetence 3. DVT

DIABETIC ULCER: sensory testing: motor testing:

Light touch power

Crude touch deformity

Pain sensation

Vibration sensation

INVESTIGATIONS:

URINE: albumin sugar deposits

BLOOD: Hb% TC DC BT CT

Peripheral smear ESR

Urea creatinine sugar – fasting, postprandial

PUS CULTURE AND SENSITIVITY:

FASTING LIPID PROFILE:

X – RAY OF AREA INVOLVED:

ECG:

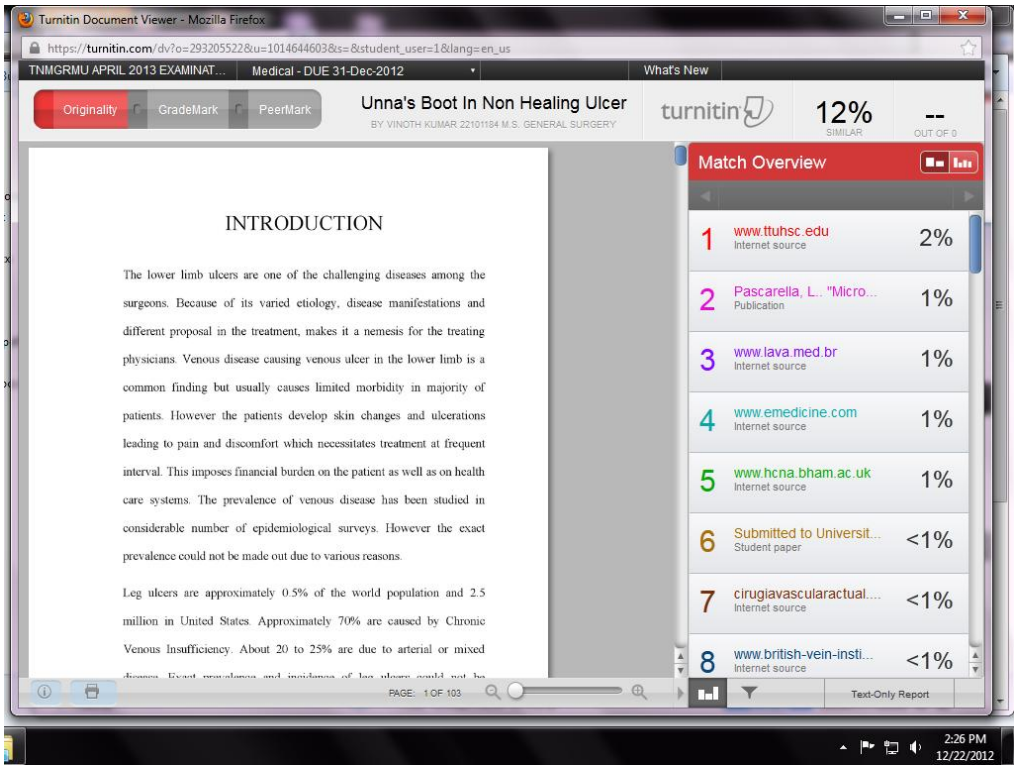
COLOUR DOPPLER STUDY:

DIAGNOSIS:

COMPLICATION:

HOSPITAL STAY:

FOLLOW UP: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TNMGRMU APRIL 2013 EXAMINAT...
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BY VINOTH KUMAR 22101184 M.S. GENERAL SURGERY
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INTRODUCTION
The lower limb ulcers are one of the challenging diseases among the surgeons. Because of its varied etiology, disease manifestations and different proposal in the treatment, makes it a nemesis for the treating physicians. Venous disease causing venous ulcer in the lower limb is a common finding but usually causes limited morbidity in majority of patients. However the patients develop skin changes and ulcerations leading to pain and discomfort which necessitates treatment at frequent interval. This imposes financial burden on the patient as well as on health care systems. The prevalence of venous disease has been studied in considerable number of epidemiological surveys. However the exact prevalence could not be made out due to various reasons.
Leg ulcers are approximately 0.5% of the world population and 2.5 million in United States. Approximately 70% are caused by Chronic Venous Insufficiency. About 20 to 25% are due to arterial or mixed
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INTRODUCTION The lower limb ulcers are one of the challenging diseases among the surgeons. Because of its varied etiology, disease manifestations and different proposal in the treatment, makes it a nemesis for the treating physicians. Venous disease causing venous ulcer in the lower limb is a common finding but usually causes limited morbidity in majority of patients. However the patients develop skin changes and ulcerations leading to pain and discomfort which necessitates treatment at frequent interval. This imposes financial burden on the patient as well as on health care systems. The prevalence of venous disease has been studied in considerable number of epidemiological surveys. However...