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ULCERATED LEG SEVERITY ASSESSMENT SCORE (ULSA) IN PREDICTION OF HEALING OF VENOUS ULCERS OF LEG

Dissertation submitted in partial fulfillment of the regulation for the award of M.S. Degree in General Surgery

(Branch I)

THE TAMILNADU

Dr. M. G. R. MEDICAL UNIVERSITY

CHENNAI – 600 032.

MARCH 2010

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CERTIFICATE

Certified that this is the bonafide dissertation done by **Dr. D.GOPIKRISHNA** and submitted in partial fulfillment of the requirements for the Degree of M.S., General Surgery, Branch I of The TamilNadu Dr. M.G.R. Medical University, Chennai

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Course : M.S General Surgery

Period of Study : 2007 - 2010

College : Coimbatore Medical College

Dissertation Topic : Ulcerated leg severity assessment score (ULSA) in prediction of healing of venous ulcers of leg.

The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation is accepted / ~~Not accepted~~ and you are permitted / ~~Not Permitted~~ to proceed with the above Study.

Coimbatore - 14.

Date : 13.02.08

Secretary
Ethics Committee

DECLARATION

I solemnly declare that the dissertation titled "**ULCERATED LEG SEVERITY ASSESSMENT SCORE (ULSA) IN PREDICTION OF HEALING OF VENOUS ULCERS OF LEG**" was done by me from 2007 onwards under the guidance and supervision of **Prof. Dr.P.M.Nanjundappan, M.S**.

This dissertation is submitted to the TamilNadu Dr. MGR Medical University towards the partial fulfillment of the requirement for the award of M.S Degree in General Surgery (Branch I).

Place : **Dr. D.GOPIKRISHNA**

Date :

CONTENTS

INTRODUCTION

Leg Ulcers are a big problem for both patients and health service resources. Most ulcers are associated with venous disease, but other causes or contributing factors include immobility, obesity, arterial disease, vasculitis, diabetis, and neoplasia.

Most of the venous leg ulcers could he healed if patients were admitted to hospital for continuous leg elevation, shortage of hospital beds, high cost of inpatient care is now rarely practical. Furthermore ulcers often recur when the patient returns home and resumes a lifestyle in which most of the leg is spent with the legs in dependency. Outpatient systems of care that maintain mobility and avoid the complications of bed rest are more cost effective and appropriate. Outpatient and community based care also maintain independence and quality of life. Compression bandaging have dramatically improved healing rates and reduced costs.

ANATOMY OF THE LOWER LIMB VEINS

Each lower limb has three different systems of veins.

They are

1. Superficial venous system
2. Deep venous system
3. Perforator system

1. Superficial venous system

Superficial veins are large with relatively thick walls located just under the skin. It has two major trunks.

1. Long saphenous vein
2. Short saphenous vein.

Long saphenous vein

Saphenous in Greek means easily seen. It is the longest vein in the body and is the pre axial vein of the lower limb bud. It is formed on the Dorsum of the foot by the union of medial end of Dorsal venous arch and digital vein from the medial side of big toe.

It ascends in front of the medial malleolus and then vertically upwards one finger breadth posterior to the medial margin of the tibia in the leg. This part is accompanied by saphenous nerve.

It ascends behind the medial side of the knee joint to reach the thigh. In the thigh it ascends along the medial margin of sartorius. Finally it hooks around the falciform margin of the saphenous opening, piercing the cribriform fascia and ends in femoral vein.

The great saphenous vein contains 15-20 valves which divide the long column of blood into a series of segments to diminish the pressure on the distal parts of the vein.

Tributaries

Just below the knee it receives a major tributary "Posterior arch vein" (Leonardo's vein). It starts behind the medial malleolus and runs posterior to the great saphenous vein, joining it at the knee level. It connects the Cockett perforator to the posterior tibial vein.

A communicating vein comes from the long saphenous vein in the thigh, passes postero-inferorly around the popliteal fossa and joins the short saphenous vein

In the thigh, it receives the posteromedial and antero lateral vein of the thigh. At the saphenous opening it receives, superficial epigastric, superficial circumflex iliac, superficial external pudendal and deep external pudendal vein.

Short saphenous vein

This begins posterior to the lateral malleolus as the continuation of the lateral marginal vein**.** It ascends lateral to the Tendo Achilles and then medially along the midline of the back of the leg.

It perforates popliteal fascia and passes between the two heads of Gastrocnemius in the lower part of the popliteal fossa ends in popliteal vein 3 to 7.5cms above the level of knee joint. Here it commonly gives upwards extension which may run deeply in continuity with profunda femoris vein by its postermedial branch in the upper thigh. In about 50% individuals, it terminates above the popilteal fossa.

Deep venous system

The deep veins below the knee are three pairs, namely, anterior tibial posterior tibial and peroneal veins. Each are associated with the above named arteries. In the upper third of calf, close to the popilteal fossa, they join to form popliteal vein. Other calf veins include the Gastrocnemius veins and the soleal veins.

Proximally, the popliteal vein enters the adductor canal to become the superficial femoral vein. The deep femoral vein joins the superficial femoral veins 5-10cm below the inguinal ligament to form the common

femoral vein which then passes upwards underneath the inguinal ligament to become the external iliac vein.

Perforator system

These veins link the superficial and deep veins by piercing the deep fascia. This linkage may be direct or indirect via sinusoids. There are valves within these veins which under normal conditions allow the blood flow from superficial to deep. There are two groups of perforators.

1. Direct
2. Indirect

1. Direct perforators

These veins directly connect the long saphenous vein or their tributaries to the deep vein. In the long saphenous vein the main perforators are arranged in three sets.

i. Dodd's perforators

In relation to the Hunters canal at the antero medial aspect of the mid thigh.

ii Boyd's perforators

In relation to the calf muscles just below the knee level.

iii Cockett's perforators

It is situated just above the ankle joint over the medial aspect. There are 3 cockett perforators situated in line with posterior border of the tibia 2,4 and 6 inches above the medial malleolus. The upper two perforators enter the posterior tibial vein where an un valved soleus venous sinus enter.

Perforators between short saphenous vein and deep veins include.

i. Bass's perforators

5cms above the calcaneum and connects the short saphenous vein with peroneal veins.

ii. Soleus point perforator

Connects soleal vein with short saphenous vein.

iii. Gastrocnemius point perforator

Connects the gastrocnemius veins with short saphenous vein.

2. Indirect perforators

They are numerous small vessels, which start from the superficial venous system, pierce the deep fascia and communicate with a vessel in an underlying muscle. The latter vessel in turn is connected with the deep vein. These indirect perforators are mostly seen int the upper part of the leg.

VALVES

Fabricus described three valves in 1574. Each valve is made up of two thin cusps. The cusps are supported by the vein walls and their integrity as functioning valves depends on this support having sufficient strength to resist forces dilating the veins and tending to separate cusps. These valves can resist pressures of upto 300mm Hg. Deep veins contain more valves than superficial vein. There are 90-120 valves in both the systems. In the leg the valve cusps are oriented to direct the blood flow centrally and prevent reflux of venous blood.

PHYSIOLOGY AND HAEMODYNAMICS

Limb veins have 3main functions

1. A pathway for return of blood to the heart.
2. Storage of blood.
3. Thermoregulation.

Deep venous system drains about two third of its blood and is the main venous outflow tract

About 70% body's blood is in the venous system at any one time. When the veins are dilated a large amount of blood is trapped in the limbs. Small changes in the caliber of the veins can markedly influence the cardiac output by increasing venous return.

Heat exchange is saved by the dilatation of the superficial veins while heat conservation is by vasoconstriction. This mechanism is mainly controlled by sympathetic vasoconstrictor nerves. It exists in normal superficial veins but is abolished in varicose veins.

HAEMODYNAMICS

Venous pressure at the calf level in a normal individual in erect posture is equal to the distance from the right atrium to the foot, if one takes the pressure in the right atrium as zero. This is equivalent to the pressure of 100-130cm H_2O or 75-95mm Hg

On exercise the contracted calf muscles produce 100mm Hg pressure, this compresses the deep veins and propel the blood to the heart provided venous valves are intact. As a result the pressure within the superficial veins is reduced to about 30-49 mm Hg. If the valves are incompetent the blood returns to the superficial veins and produce venous hypertension.

PATHOLOGY OF VARICOSE VEINS

Etiopathogenesis of primary varicose veins

The venous return is mainly governed by

1. Calf muscle pump
2. Competent valves in the veins.
3. Negative intrathoracic pressure.
4. Vis-a-tergo produced by arterial pressure.
5. Venae comitantes
6. Gravity.

Pathophysiology of primary varicose veins

The most essential factor in the development of primary varicose vein is failure of valves. This failure of valves may be due to

1. Degenerative changes in the valve
2. Failure in the valves themselves.
3. Degenerative changes in the vein wall leading to dilatation.

The following mechanisms have been suggested.

I. Hydrostatic pressure

The hydrostatic effect of gravity in the upright position is of great importance in valvular failure. In lying down position pressure in the superficial veins will

be near zero if foot is at the same level of heart, but in standing position, the hydrostatic pressure rises and exerts pressure on the valves, leading to failure

II. Hormonal influences may also play a part in the pathogenesis. The charges in the hormonal level particularly progesterone, have an effect on the

vein wall. These high levels of progesterone will cause changes in the structure of collagen as well as smooth muscle relaxation.

III. Hereditary influences: The early onset of severe and intractable varicose veins in the very young person often indicates that they have relatively few valves in the venous system of the leg. There is an increased risk for children of parents who both had varicose veins.

IV. Ageing: As with ageing there is structural changes within the vein or valve, perhaps to elastin or collagen

SECONDARY VARICOSE VEINS

Etiopathogenesis

1. Mechanical factors eg.: Pregnancy or tumours in the pelvis
2. Deep vein thrombosis leading to damage of the valves
3. Homonal causes: Progesterone may cause varicosity in the multiparous females.

4. Acquired arteriovenous fistula (due to truma or deliberate shunting for dialysis)
5. Extensive cavernous (Venous) haemangioma
6. Retroperitoneal lymphadenopathy or retroperitoneal fibrosis
7. Iliac vein thrombosis

CAUSES OF SUSTAINED VENOUS HYPERTENSION

a. VENOUS DISEASE

i. Superficial venous incompetence-varicose veins
ii. Deep vein incompetence
iii. Deep vein obstruction
iv. Previous deep vein thrombosis
v. External compression

b. IMPAIRED CALF MUSCLE PUMP FUNCTION

i. Immobility
ii. Joint disease
iii. Paralysis
iv. Obesity-Immobility, Femoral vein compression
High abdominal pressure

c. CONGESTIVE CARDIAC FAILURE

WIDMER CLASSIFICATION

Category	**Description**
Hyphen webs	Venous telengiectasia, spider veins
Reticular veins	Dilated tortuous subcutaneous veins not major trunks or branches
Truncal veins	Dilated tortuous LSV or SSV or main branches
*CVI grade I	Venous flare at the ankle “Corona Phlebectatica”
*CVI grade 2	Hyper or depigmented area in the gaiter area
*CVI grade3	Open or healed venous ulcer

*CVI-CHRONIC VENOUS INSUFFICIENCY

The more recent and comprehensive classification is CEAP classification.

CEAP-CLASSIFICATION

CLINICAL

- **C0 -no disease**
- **C1 -telangiectasias reticular veins**
- **C2 -varicose veins**
- **C3 -Edema**
- **C4a -hyperpigmentation**
- **C4b -lipodermatosclerosis**
- **C5 - Healed ulceration**
- **C6 -Active ulceration**

ETIOLOGY

- **Primary - Ep**
- **Secondary- Es**
- **Congenital-Ec**
- **No etiology found- En**

ANATOMY	PATHOLOGICAL
• **Superificial vein As**	• **Reflux Pr**
• **Deep veins Ad**	• **Obstruction Po**
• **Perforator veins Ap**	• **Obstruction and reflux Pr,o**
• **No venous location identified An**	• **No pathophysiology found Pn**

PREDIPOSING FACTORS

1. Prolonged standing
2. Old age (Peak 50-60yrs)
3. Obesity
4. Athletes
5. Pregnancy

KLIPPEL – TRENAUNAY SYNDROME

It consists of triad of

- Cutaneous nevi
- Congenital varicose veins
- Soft tissue hypertrophy of limbs

PATHOLOGY

Two theories have been proposed:

1. White cell hypothesis

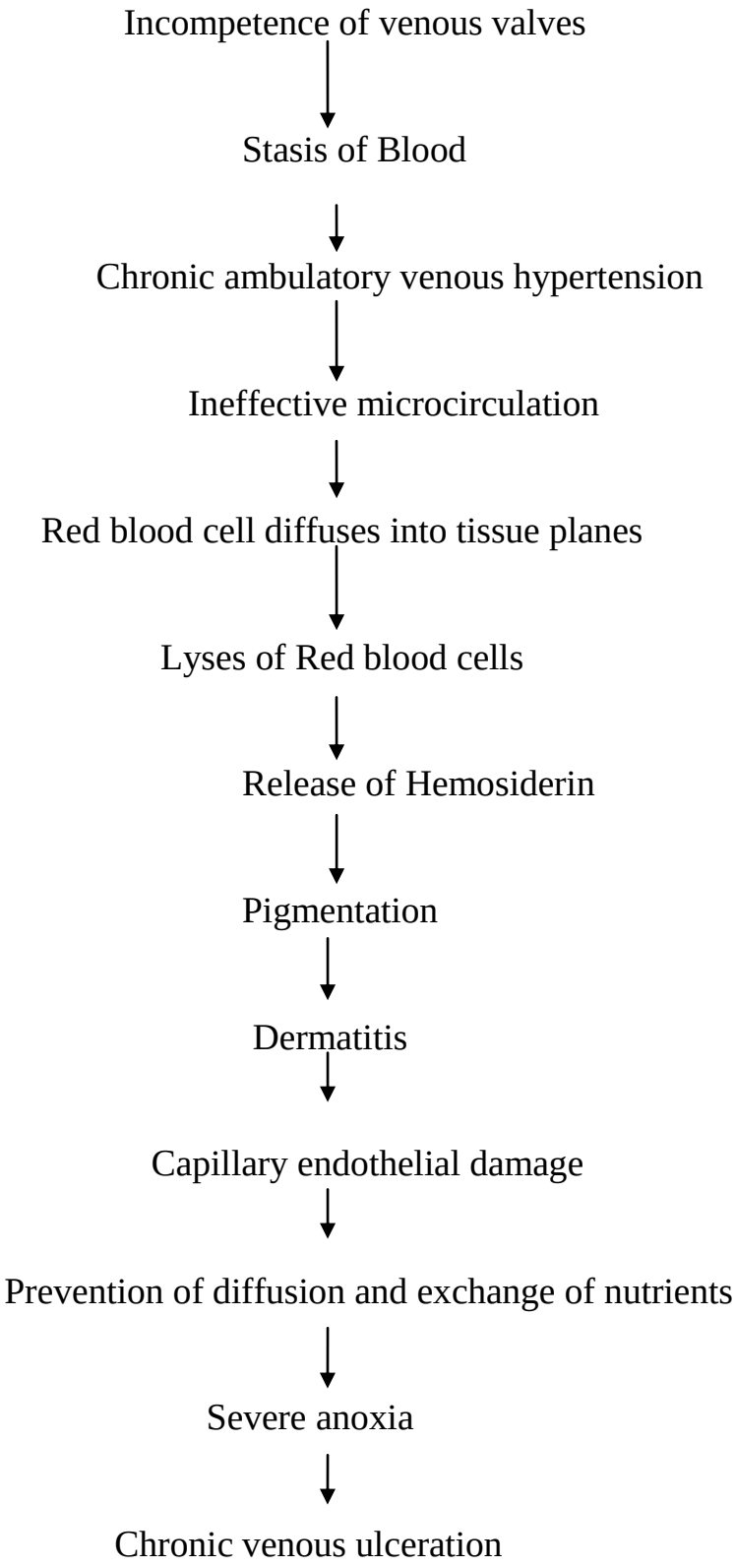
Patients with chronic venous disease resulting in venous ulceration trap more leucocytes than do subjects with normal limbs. These trapped leucocytes become activated and release the proteolytic enzymes that are normally used in defence against infection. This, in turn, causes injury to the capillary endothelium.

2. Fibrin Cuff hypothesis

Fibrinogen is driven out of the Venules, and act as a barrier to the diffusion of oxygen and other nutrients into tissues, leading to atrophy of the overlying skin, which breaks down following some trivial injury.

Venous hypertension is the main factor in the genesis of ulcer and it is aggravated by erect posture.

Fibrin cuff theory



COMPLICATIONS OF VARICOSE VEINS

Haemorrhage: It may occur from minor trauma to the dilated vein. The bleeding may be profuse due to high pressure within the incompetent vein.

Thrombophlebitis: This may occur spontaneously or secondary to minor trauma. Mild phlebitis may be produced by the sclerosing fluid used in the injection treatment.

Eczema: Due to extravasation and breaking down of the RBC's in the lower part of the leg, the skin may itch. The patient scratches which may lead to eczema formation.

Lipodermatosclcerosis: The skin becomes thickened, fibrosed and pigmented. This is due to high venous pressure which causes fibrin accumulation around the capillary.

Calcification: This is dystrophic in nature and may occur in long standing varicose vein.

Equinus deformity: This results from the patient trying to walk on the toes to avoid pain, so he continuous to do so and ultimately the Achilles tendon become shorter to cause this defect.

Ankle flare: In case of long standing varicosity, the small veins from the sole

of the foot and the ankle which drain into the long saphenous vein through the medial marginal veins these become dilated and this gives rise to swelling of the ankle.

Periosteitis: This is usually seen if there is a long standing ulcer over the medial surface of the tibia penetrating into the periosteum producing a reaction.

Malignant transformation: Chronic nonhealing ulcer may predispose into squamous cell carcinoma (marjolin's ulcer. Because of the extensive fibrosis of the base involving neural and lymphatic channels they are painless and slow to metastasise to the regional nodes.

VENOUS ULCER

Venous ulceration may be defined as the break in the skin,

present for more than six weeks, between the malleoli and tibial tuberosity,

that is presumed to be wholly or partly due to venous disease.

PATHO PHYSIOLOGY OF VENOUS ULCERATIONS

Chronic venous insufficiency has been widely assumed to be due to deep venous obstruction or incompetence and the term “post phlebitis limb” has been used even though most people with venous ulcers have no history of deep vein thrombosis. Venous ulceration is clearly associated with age and younger patients with severe chronic venous insufficiency may avoid ulceration by virtue of their mobility which maintains function of the calf muscle pump. Whether the final mechanism in ulceration is a fibrin cuff, leukocyte trapping or chronic inflammation due to a repetitive ischaemia reperfusion injury the treatment for venous ulceration is based almost entirely on avoiding venous hypertension.

Sustained venous hypertension results in edema. Within the dependent lower limb, which increases the distance over which metabolites must diffuse

from the microcirculation to tissue cells. The tissues around the ankle becomes ischemic during dependency with reperfusion on walking or elevation. This chronic reperfusion injury results in an inflammatory process with further oedema, tissue fibrosis and formation of a cuff of extra cellular matrix proteins around capillaries. These changes results in the features of chronic venous insufficiency which include aching, heaviness of the legs, itching, lipodermatosclerosis, pigmentations, swelling, eczema and ultimately ulceration.

INVESTIGATIONS

1). DOPPLER ULTRASONOGRAM

The piezoelectric crystal in the probe emits a continuous ultrasound signal. According to the speed and direction of the movement of the RBC's there is a Doppler shift in the phase of the signal which is recognized by the machine. A Doppler ultrasonogram utilizes the Doppler probe to determine the presence of venous flow, phasic changes with respiration, increased flow with distal compression (Augmentation) and reverse flow with proximal compression (Bidirectional)

2). DUPLEX IMAGING

This is a combination of Doppler ultrasound with B mode ultrasound. Doppler gives the flow patterns in the vein while B mode ultrasound gives anatomical delineation. It has a 95% accuracy in detecting a lower limb deep vein thrombosis and is the first investigation of choice. Color coding has added to the value of the test and one can visualize the direction of flow of blood.

3). ULTRASOUND AND CT SCAN of the abdomen should be performed to rule out the causes for secondary varicose veins.

4). CULTURE SENSITIVITY

5)ANKLE BRACHIAL INDEX

This test is done by measuring blood pressure at the ankle and in the arm while a person is at rest. Measurements are usually repeated at both sites after 5 minutes of walking on a treadmill.

The ankle-brachial index (ABI) result is used to predict the severity of peripheral arterial disease (PAD). This test is done to screen for peripheral arterial disease of the legs.

The ABI result can help diagnose associated peripheral arterial disease (PAD).

Normal A normal resting ankle-brachial index is 1 or 1.1.

Abnormal

A resting ankle-brachial index of less than 1 is abnormal. If the ABI is:

- Less than 0.95, significant narrowing of one or more blood vessels in the legs is indicated.
- Less than 0.8, pain in the foot, leg, or buttock may occur during exercise
- Less than 0.4, symptoms may occur when at rest.
- 0.25 or below, severe limb-threatening PAD is probably present.

6. VENOGRAPHY

It consists of

1. Ascending Venography.
2. Descending Venography.

ASCENDING VENOGRAPHY

It is the gold standard for demonstrating venous occlusion and the pattern of collateral flow. About 5ml of contrast material (Iohexol) is injected into peripheral vein with visualization of the proximal venous anatomy. Thrombi are identified as globular filling defects.

DESCENDING VENOGRAPHY

Retrograde flow following femoral vein injection is used to grade venous valvular insufficiency.

Grading of descending venogram

Level 0	-	Competent valve
Level 1	-	Saphenofemoral valve incompetancy
Level 2	-	Antegrade iliac flow with reflux into distal thigh
Level 3	-	Reflux through popliteal vein to calf
Level 4	-	reflux to the calf without any ante grade iliac flow.

MANAGEMENT OF VARICOSE VEINS

The treatment options for varicose veins are

1. Conservative treatment
2. Sclerotherapy
3. Surgical treatment

I. CONSERVATIVE TREATMENT

INDICATIONS

1. Very early case of varicosity
2. Those who are pregnant
3. Those who do not want operation
4. Those who are waiting for operation
5. Patients with superficial varicosity and acute deep vein thrombosis.
6. Klippel Trenaunay syndrome

Treatment

1. Avoidance of prolonged standing
2. A crepe bandage or elastic stocking is applied from the toes to the thigh throughout the day and is only taken off during sleep.
3. Sleep with the limb above the heart level
4. Exercises like bi-cycle riding in the air.

I. SCLEROTHERAPHY

INDICATIONS

1. Varicosities mostly confined to below knee
2. Recurrent or residual varicosity after surgery
3. Telangiectasia
4. Patient unsuitable for standard anesthesia/surgery
5. Patient not willing for surgery.

CONTRAINDICATIONS

1. Deep vein thrombosis
2. Saphenofemoral incompetence

The aim of Sclerotherapy is to inject a small volume of an effective sclerosant

into the lumen of the veins in order to destroy the venous intima.

COMPLICATION

1. Excess concentration of sclerosant, if injected outside the vein wall will lead to the formation of deep and painful ulcer.
2. Thrombophlebitis
3. Anaphylactic reactions to sclerosant.
4. Accidental intra-arterial injection.
5. Neurological damage.

III. SURGERY

Various options of surgery available are

i. Simple high ligation of saphenofemoral or saphenopopliteal junction.

ii. Ligation and stripping

iii. Ligation and multiple avulsions

iv. Subfascial ligation of perforators.

INDICATION

1. Symptoms of aching like

   Heaviness and cramps.

2. Complications of venous stasis

   a. pigmentation

   b. dermatitis

   c. induration

   d. ulceration

   e. thrombosis of varicosities

3. Large varicosities subjected to trauma

4. Cosmetic

MANAGEMENT OF VENOUS ULCERS

I. DRESSING AND TOPICAL AGNTS

1. Antiseptics:

Hypochlorite, Povidone iodide and Hydrogen peroxide. These agents are toxic to the tissues as well as the bacteria, which are believed anyway to be largely harmless.

2. Enzymatic debridement : (Streptokinase- Streptodornase)

These agents digest the constituents of 'slough'

3. Hydrocolliod dressings.

4. Bead dressing: (Cadexomer iodine and dextronomer)

These are hydrophilic, polysaccharide materials that absorb large amount of fluid and slough.

5. Paste dressing:

These comprise a plain weave cotton fabric impregnated with zinc oxide paste either alone or with calamine or icthamol. These additives are designed soothe the venous eczema.

6. Alginate dressings:

These are dressing produced from sea and contain calcium and sodium alginates. On contact with the wound these dressings become

hydrophilic gel, which absorb exudates and create a moist environment.

7. Biological dressing: Cultured human epithelium or fibroblasts are under trial.

II DRUGS

1. Veno active drugs:

Pentoxyphyline improves the pliability of RBC's and has been shown to be of benefit in the treatment of venous ulcer.

Other groups of drugs like calcium dobesilate, Tribenoside,

Flavonoides are used in management. There is not much benefit from these drugs.

2. Antibiotics:

When there is no evidence of clinical infection, systemic antibiotics are not indicated.

III.BANDAGES

1. Four layer bandages

This technique is developed at Charing Cross Hospital in London.

a. Orthopaedic wool
b. Crepe bandage
c. Elasticated bandage
d. Cohesive bandage

This method achieves pressure of 45mm Hg at the ankle and has been shown to produce healing of 70% of venous ulcers within 12 weeks.

2. Paste Bandage

Always used in conjunction with compression bandages.

IV PHYSICAL THERAPY

1. **Bisgaard's method**

   Which consists of

   a. Massage and elevation of the whole leg, particularly to increase the flow to the indurated area around the ulcer.
   b. Passive movements to maintain the mobility of the foot and ankle.
   c. Active movements to the calf muscles in elevation and in standing posture (with bandages on).
   d. Teaching the correct method of walking by placing heel down first and using the calf muscles to the heel of the back foot, giving a spring to the walk and improving the venous pump.
   e. A firm elastic bandages is applied spirally from the base of the toes to the knee, by graded compression so that the movement in walking alternatively stretches and relaxes the bandage and pressure adds to the pumping effect.

EFFECTIVE TREATMENT FOR VENOUS LEG ULCERS

1. Compression Bandaging
2. Limb elevation
3. Improve mobility
4. Reduce obesity
5. Improve nutrition
6. Skin grafting in selected patients
7. Venous surgery in selected patients

V. SURGICAL MANAGEMENT

Patients with venous ulceration can be divided on the basis of duplex ultrasonography into

1. Isolated superficial venous reflux
2. Combined deep and superficial reflux
3. Isolated deep venous reflux
4. Deep venous obstruction

Surgery is useful in isolated superficial venous reflux and isolated deep venous reflux.

Surgical options are

1. Perforator surgery-SEPS
2. Deep venous reconstruction

Other surgical options are

- Ulcer debridement
- Skin grafting

AIM

The study is on the utility of Ulcerated Leg Severity Assessment Score (ULSA) in the prediction of healing and in the management of venous ulcers in patients with primary varicose veins.

OBJECTIVE

The study would identify the effectiveness of the ULSA score in predicting venous ulcer healing with conventional modes of treatment such as compression and dressing under antibiotic cover and to surgically intervene in patients with high ULSA scores

REVIEW OF LITERATURE

A study was conducted in the Cheltenham hospital. United Kingdom. The aim of the study was to create a reliable scoring system for the prediction of venous ulcer healing in patients with primary varicose veins. A prospective baseline study was undertaken to identify the risk factors that affect healing and the line of management in patients with varicose vein ulcers. The study was conducted between march 1999 And August 2001. A number of were related to healing rates over a three month period. A Cox pattern regression model was used identify risk factors that predicted ulcer healing, from which a scoring system was developed and validated prospectively between February 2004 and March 2005. In the baseline study of 229 patients, patient age, ulcer chronicity were identified as risk factors. Using these factors, the following formula was devised: Ulcerated Leg Severity Assessment (ULSA) score=(Age + Chronicity)-50. Patients with an ULSA score of 50 or less had higher 24-week ulcer healing rates than those with higher scores in both the performed in 86 patients. The conclusion was that the ULSA score may help to identify patients with venous ulcers unlikely to respond to conventional treatment who could be offered alternative therapy

MATERIALS AND METHODS

50 cases of varicose veins of the lower limb with venous ulcers were studied using ULSA score and were prospectively analyzed over a period of 24 weeks in surgical wards of Coimbatore medical college hospital during the period of AUGUST.2007- SEPTEMBER 2009.

DESIGN

A prospective study

INCLUSION CRITERIA

Patients with primary varicose vein of the lower limb and venous ulcer were selected on the basis of Age, Chronicity, Size

EXCLUSION CRITERIA

The cases associated with DVT, HT,peripheral vascular disease, Malignancy, Ascites, DM, vascular malformations, secondary causes for varicosity and Recurrent varicose veins after surgery were excluded,.

COLLECTION OF DATA

The sources of the data were the patients presented to the surgical OPD with venous ulcers. All patients were routinely examined clinically and also investigated with duplex scan. Peripheral vascular system was also examined. The ULSA score was applied at the time of presentation and prospectively analyzed over a period 24 weeks All Patients were followed regularly at 8 Wks, 12Wks, 24Wks to assess healing rates.

OBSERVATION AND DISCUSSION

In this study, 50 cases of the venous ulcers were studied from the period of August 2007 to September 2009

DISTRIBUTION OF CASES

Side	No of patients	Percentage
Unilateral	42	84%
Bilateral	8	16%

Systems involved

Of the 50 cases studied, 18 cases were long saphenous system with perforators. 13 cases were perforators alone, 6 cases were long saphenous system and 6 cases were short saphenous system. 4 cases were a combination

of long saphenous system,short saphenous system with perforators. 2 cases were long saphenous system with short saphenous system, while 1 case was short saphenous system with perforators

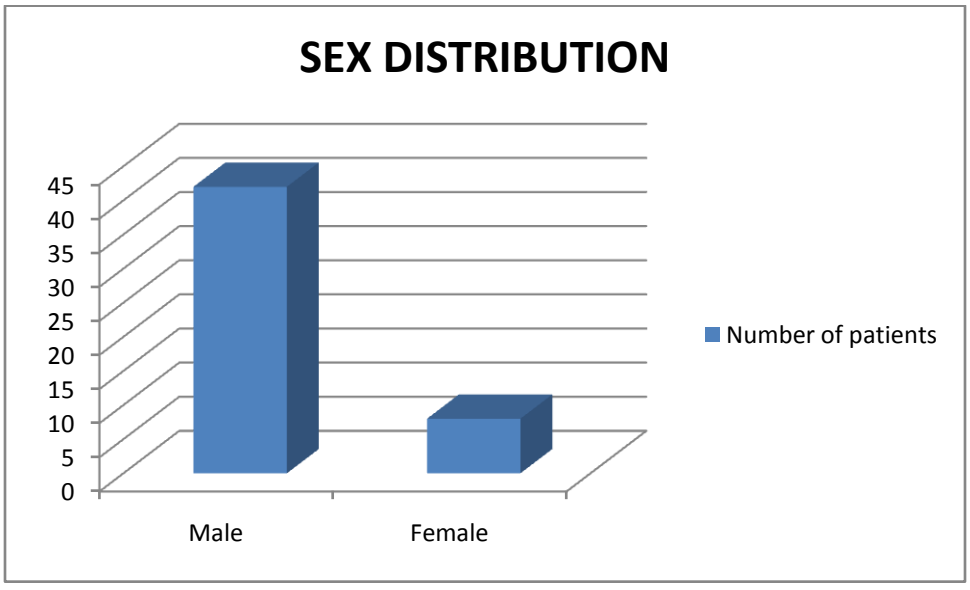
S.No.	**Systems involved**	**No. of patients**	**Percentage**
1.	Long saphenous vein with perforators	18	36%
2.	Perforators	13	26%
3.	Long saphenous vein	6	12%
4.	Short saphenous vein	6	12%
5.	Long saphenous vein with short saphenous vein with perforators	4	8%
6.	Long saphenous vein with short saphenous vein	2	4%
7.	Short saphenous vein with perforators	1	2%

SEX INCIDENCE

Among the 50cases, 42 patients were males and 8 patients were females

Male:female ratio-5.25:1

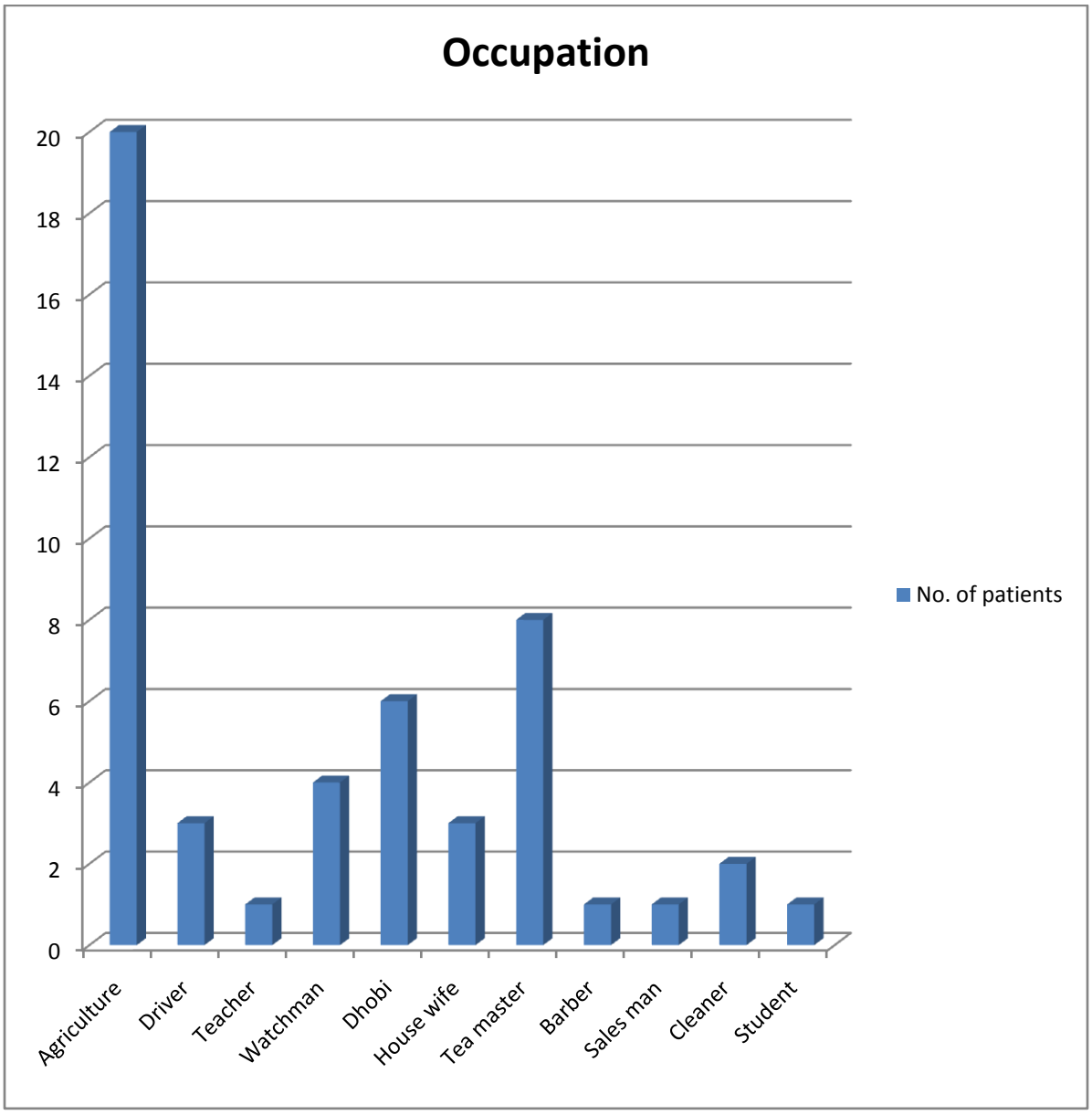
Sex of patient	Number of patients	Percentage
Male	42	84%
Female	8	16%



OCCUPATION AND VARICOSITY

There was a definite relationship between the type of occupation and varicosity. Of 50 cases 20 cases (40%) are agricultural workers and 8 cases tea master and others. During prolonged standing the long column of blood and gravity exert pressure on the weakened valves of the veins giving rise to varicosity.

Occupation	**No. of patients**	**Percentage**
Agriculture	20	40%
Driver	3	6%
Teacher	1	2%
Watchman	4	8%
Dhobi	6	12%
House wife	3	6%
Tea master	8	16%
Barber	1	2%
Sales man	1	2%
Cleaner	2	4%
Student	1	2%


Occupation
20
18
16
14
12
10
8
6
4
2
0
Agriculture
Driver
Teacher
Watchman
Dhobi
House wife
Tea master
Barber
Sales man
Cleaner
Student
No. of patients

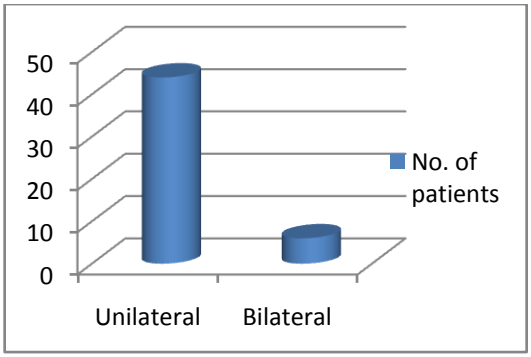
VENOUS ULCER

The ulcer was situated over the superomedial aspect of the medial malleolus in 38 cases. In 12 cases ulcer was seen on medial and lateral aspect of the lower part of the leg.

Site	No. of patients	Percentage
Supero medial aspect of the medial mallelous	38	76%
Medial and lateral aspect of the leg	12	24%

In this series bilateral ulcer was present in 6 cases.

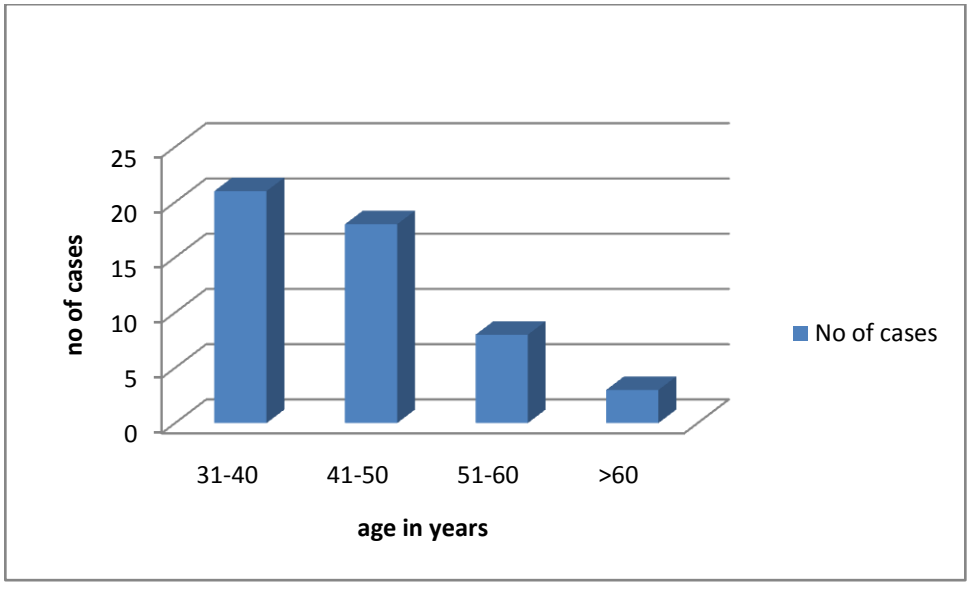
Ulcer	No. of patients	Percentage
Unilateral	44	88%
Bilateral	6	12%



In 14 patients the ulcer was oval in shape and in the rest had irregular margins. All the ulcers were surrounded by black pigmentation and margins were sloping in nature. Most of the ulceration occurred in the gaiter area.

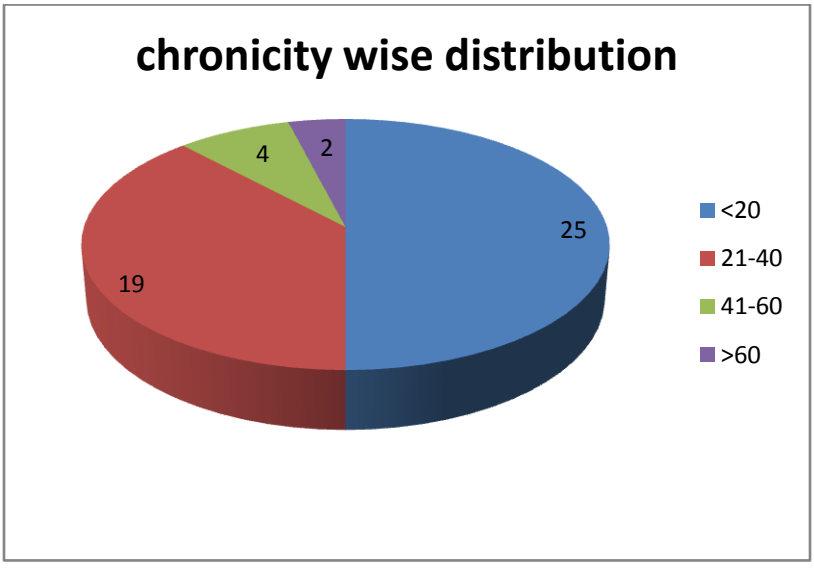
Age wise distribution

Age in years	No of cases
31-40	21
41-50	18
51-60	8
>60	3



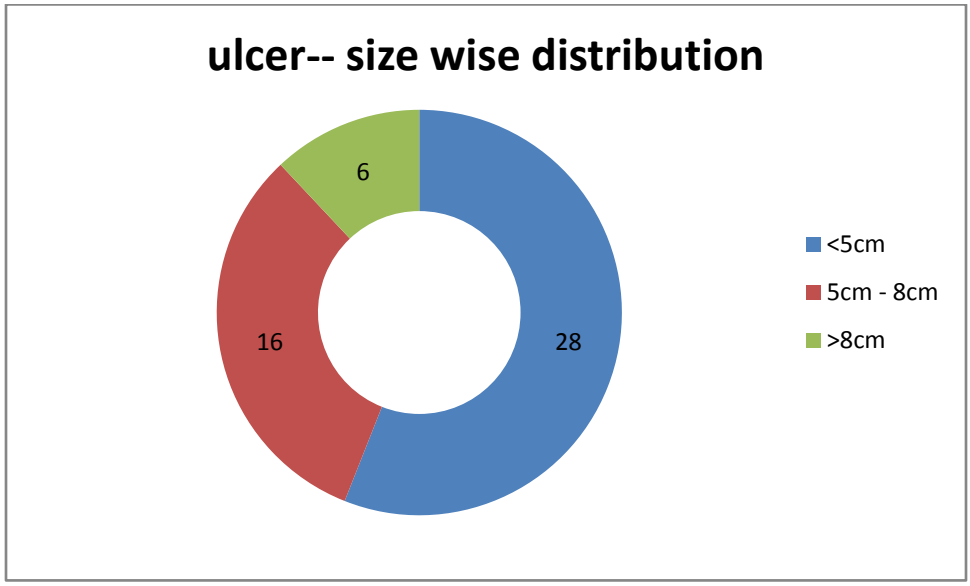
Chronicity wise distribution

Duration in weeks	No of cases
<20	25
21-40	19
41-60	4
>60	2



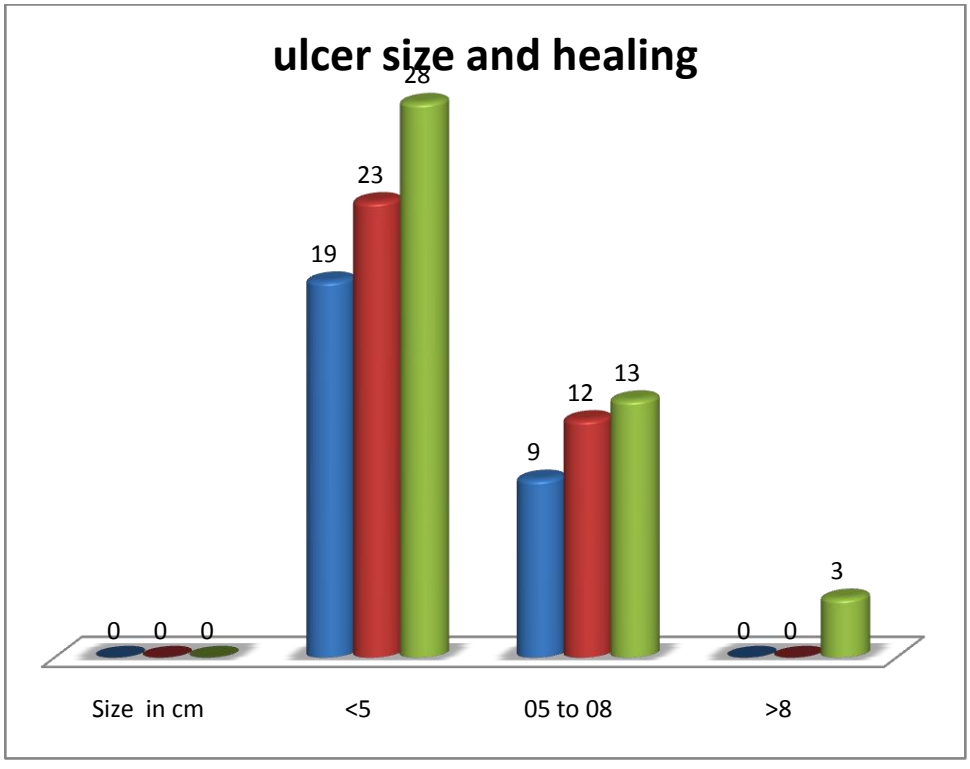
Size of ulcer

Size of ulcer in cm	No of cases
<5cm	28
5cm - 8cm	16
>8cm	6



Size of ulcer and healing

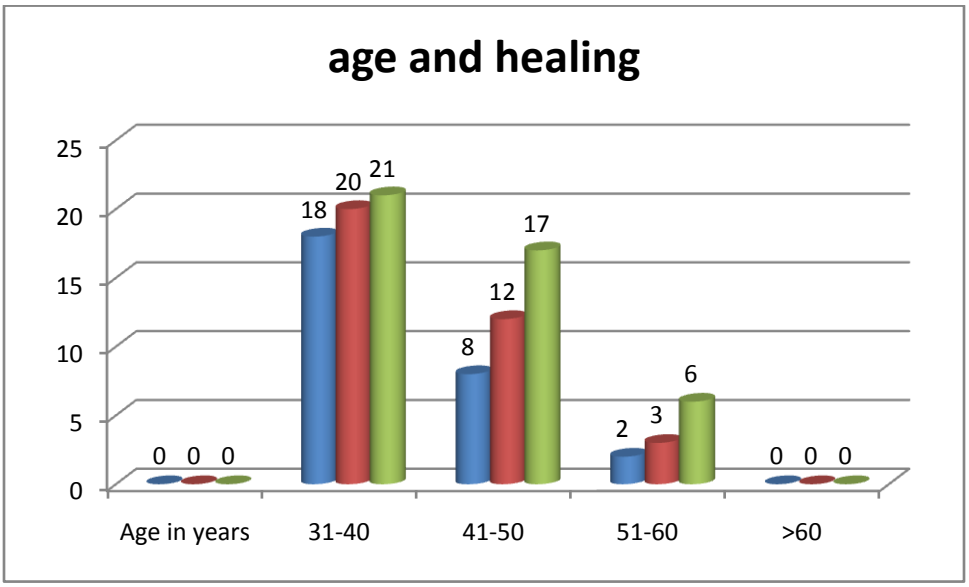
Size in cm	8 weeks	12 weeks	24 weeks
<5	19	23	28
5-8	9	12	13
>8	0	0	3



A large ulcer took a longer time to heal

Age of patient and healing

Age in years	8 weeks	12 weeks	24 weeks
31-40	18	20	21
41-50	8	12	17
51-60	2	3	6
>60	0	0	0

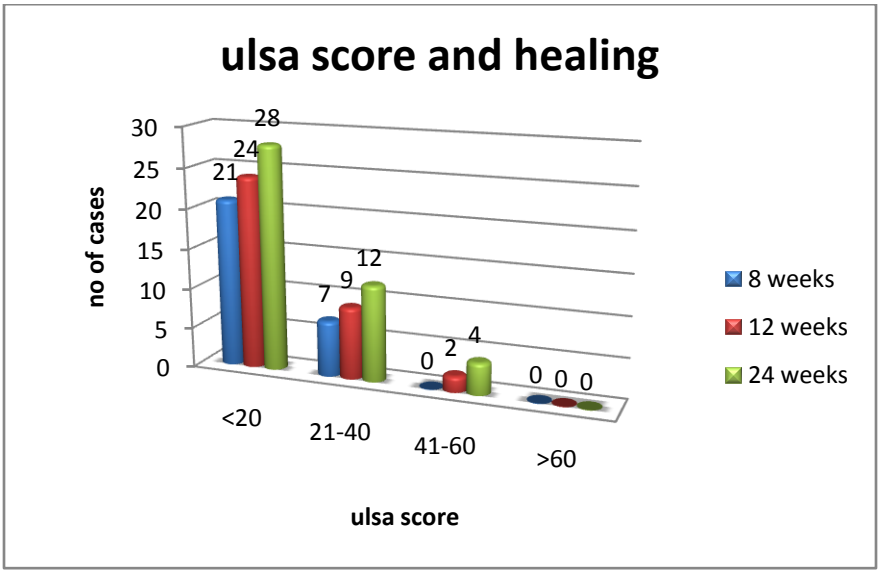


Patients in the higher age group had slower healing rates.

ULSA SCORE AND HEALING OF ULCER

Ulsa score	8 weeks	12 weeks	24 weeks
<20	21	24	28
21-40	7	9	12
41-60	0	2	4
>60	0	0	0

Patients with a low ULSA score had faster healing rates when compared to those with high scores. Majority of cases with ULSA score less than 20 healed within 24 weeks, while those with more than 60 never healed at 24 weeks.



CONCLUSION

Among the 50 cases of varicose veins with venous ulcers of the lower limb studied in this series from August 2007 to September 2009, those with a low ULSA score at the time of presentation healed earlier than those with high scores.

Patients with ULSA score less than 20 had completely healed in 24 weeks and ulcers with scores more than 50 did not heal completely by conservative management .The result of this study correlates with the study undertaken at Cheltenham hospital, United Kingdom where ULSA score less than 50 was associated with complete healing by conservative management.

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Proforma *case no:*

Name:

Age/ Sex: **I.P.No**

Diagnosis:

Duration of venous ulcer:

Investigations

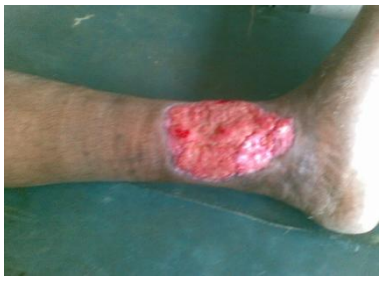
Hb%	Glucose	Urea	Creatinine	Others(specify)

Doppler study

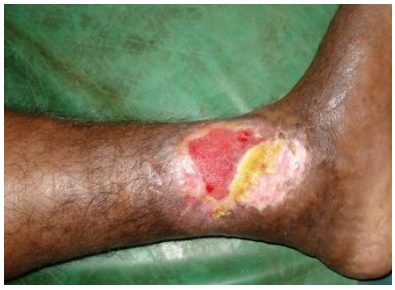
ULSA SCORE = (AGE+CHRONICITY) - 50 =

Healing time in weeks

ULSA SCORE > 60

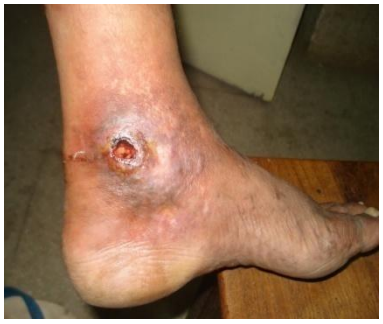


Day 1

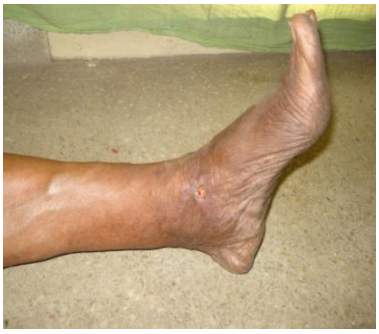


24 weeks

ULSA SCORE 20

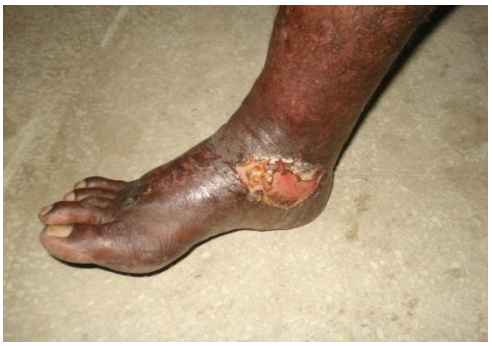


Day 1

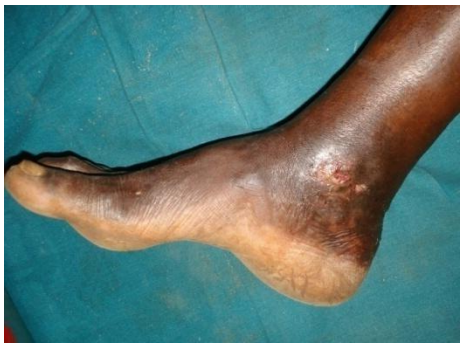


5 weeks

ULSA SCORE (20)

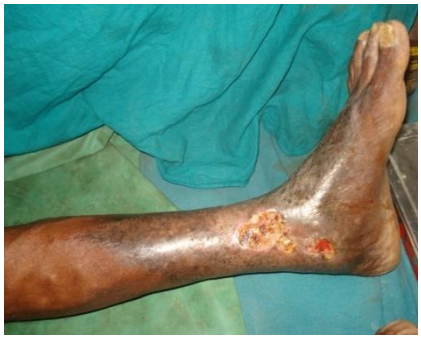


DAY I

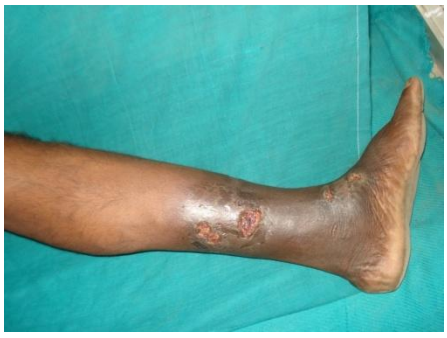


WEEK 6

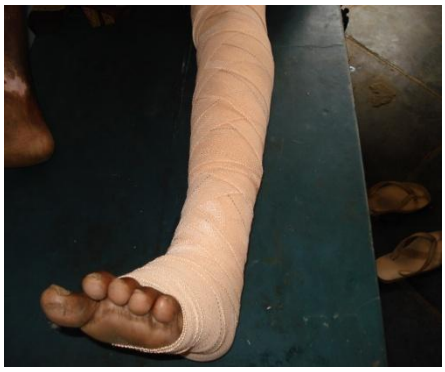
ULSA SCORE 30



DAY I



WEEK 20



ELASTO CREPE COMPRESSION BANDAGE

MASTER CHART

S.No	Name	Age	Sex	IP/OP No	Duration	ULSAscoring
1.	Sivaraman	36	M	6880	15	1
2.	Saminathan	36	M	32716	17	3
3.	Damodharan	37	M	9604	18	5
4.	Bakiyaraj	37	M	2705	12	-1
5.	Satish	37	M	41241	14	1
6.	Chinnasamy	37	M	95900	24	11
7.	Chokalingan	38	M	232558	16	4
8.	Kalaiselvan	38	M	55242	18	6
9.	Sathyam	38	M	56675	14	2
10.	Angappan	38	M	258073	18	6
11.	Gomathinayagan	38	M	6171	16	4
12.	Kuppusamy	38	M	68620	17	5
13.	Veeramuthu	38	M	222881	13	1
14.	Malathy	39	F	7829	14	3
15.	Selvaraj	39	M	222943	18	7
16.	Elangovan	40	M	236492	16	6
17.	Illayaraja	40	M	225701	12	2
18.	Karuppias	40	M	225211	12	2
19.	Arunachalam	40	M	212042	17	7
20.	Thomas	40	M	233151	19	9
21.	Chinappan	40	M	222540	18	8
22.	Ramanathan	41	M	29088	11	2
23.	Alamelu	41	F	238026	14	5
24.	Ragupathy	43	M	230026	12	5
25.	Muthappa	43	M	232102	19	12

S.No	Name	Age	Sex	IP/OP No	Duration	ULSAscoring
26.	Rajangan	43	M	232427	26	18
27.	Selvaraj	44	M	299679	22	16
28.	Nataraj	44	M	218268	32	26
29.	Zaher	45	M	6389	16	11
30.	Somasundaram	45	M	243999	14	35
31.	Md.Ibrahim	45	M	244652	36	31
32.	Sangeetha	45	F	237587	32	27
33.	Palanisamy	45	M	290818	35	30
34.	Nasimabegum	47	F	248114	38	31
35.	Chandran	47	M	280214	27	24
36.	Manoharan	48	M	252692	25	23
37.	Kamala	48	F	237592	42	40
38.	Chinakannan	50	M	250052	28	28
39.	Balu	50	M	250955	34	34
40.	Govindan	51	M	249739	39	40
41.	Johnpeter	51	M	246348	48	49
42.	Akkalu	53	F	461667	54	57
43.	Marimuthu	55	M	270482	33	38
44.	Kala	56	F	262007	37	43
45.	Mani	57	M	259285	72	79
46.	Murugan	57	M	245679	65	72
47.	Velusamy	58	M	358708	46	54
48.	Kanagaraj	62	M	403404	38	40
49.	Anjammal	65	F	376498	34	49
50.	Subramani	69	M	320294	30	49