Dissertation
On

CLINICAL CASE STUDY OF VARICOSE VEINS

Submitted to

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI.

M.S. BRANCH II
GENERAL SURGERY

DEPARTMENT OF GENERAL SURGERY
KILPAUK MEDICAL COLLEGE
CHENNAI – 600 010.

FEBRUARY 2006

CERTIFICATE

Certified that this dissertation is the bonafide work of **Dr.P.KATHIRVEL KUMARAN on CLINICAL CASE STUDY OF VARICOSE VEINS** during his M.S(General Surgery) course from march 2003 to February 2006 at Kilpauk Medical College Hospital

Prof.P.KULOTHUNGAN,M.S.,
Professor of surgery
Department of general surgery
Government Kilpauk Medical
College, Chennai-600010.

Prof.Dr.R.THIRUNARAYANAN
M.S.,FICS
Head of the department
Department General Surgery
Government Kilpauk Medical
College, Chennai-600010.

Prof.G.ELANGOVAN, MD.,DD.,DIH.,Ph,D.,
DEAN
Kilpauk Medical College and Hospital
Kilpauk chennai-600010

ACKNOWLEDGEMENT

I sincerely express my gratitude to my professors **Prof. P.KULOTHUNGAN M.S.,** and my head of the department **Prof. THIRUNARAYANAN M.S.,** and to my old Prof. **A.NATARAJAN M.S.,** for their encouragement, guidance and timely advice during the course of study.

I'm greatly indebted to my assistant professors **Dr. R.DAMODARAN M.S., Dr. S.SELVA KUMAR M.S., Dr. AFFEE ASMA DGO., M.S., Dr BHOOPATHY M.S.,**

Their able guidance and involvement has been indispensable in conducting the study.

I honour the help and assistance rendered by my fellow postgraduates and all the patients involved in the study.

Last but not the least I express my sincere thanks to the Dean, KMC for permitting me to conduct the study.

CONTENTS

	Page No.
INTRODUCTION	1
AIM OF STUDY	3
REVIEW OF LITERATURE	4
DIAGNOSTIC WORK-UP	19
VARIOUS MODALITIES OF TREATMENT	27
MATERIALS AND METHODS	36
OBSERVATION AND ANALYSIS	39
CONCLUSION	59
MASTER CHART	
BIBLIOGRAPHY	

INTRODUCTION

The term varicose is derived from latin word varix meaning 'bent'. varicose limb which has lost its valvular efficiency and as a product of the resultant venous hypertension in standing position becomes dilated, tortuous, elongated and thickened .

Varicose vein are known from antiquity since hippocrates and treatment in its length and associated with varicose ulcer.

Varicose veins of the lower limb are the most common vascular disorder affecting the human beings. Its almost certainly the price we pay for the erect posture. Delicate valves that evolved through million of years of ambulation of four legs are unable to increased gravitational pressure upright posture – ALEXANDER.C.J.

Although mortality is minimal , morbidity due to the disease causes much misery and suffering as it occurs in the prime of life .As can be expected there is enormous loss of man power and productivity. Interestingly this disease has an effective treatment and hence my selection of varicose veins as a dissertation topic.

HISTORICAL REVIEW

The **papyrus of ebers** (approx- 1550 BC) mention varicose vein and advise against operation for this condition.

Hippo crates (460-377 BC) associated ulcer of the leg with varicose veins and he also noted it does not arise before puberty.

Aureluis Cornelius celsus (258BC-25 AD) advised the use of plasters and linen roller bandage for leg ulcers .varicose vein were treated with by exposure followed by avulsion with blunt hook or by cauterisation.

Claudisus galen likewise treated with blunt hook and he applied wine to leg ulcers and opposed to frequent change of dressings.

First ligation of varicose vein seems to have been done by **zatane physician Aetius of amidia on the tigris** (502-575 AD).

Harvey studied about venous valves and thought to and fro movement was not possible.

Wiseman (1676) realized the valvular incompetence results from dilatation of the vein and he was the first to consider that ulcer might be direct result of the circulatory defect. He accordingly used the term varicose ulcer.

AIM OF THE STUDY

1. To analyse the age and sex incidence

2. To analyse the correlation between occupation and disease.

3. To study mean age of presentation.

4. To analyse the various clinical presentations, complications of varicose veins and various modalities of treatment.

5. To analyse incidence of varicose ulcer.

6. To analyse the incidence of recurrent varicose veins.

REVIEW OF LITERATURE

ANATOMY OF THE LOWERLIMB

Each limb has three set of anatomically and functionally distinguishable system of veins. They are :

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

SUPERFICIAL SYSTEM

They are subcutaneous. It has two major trunks namely great saphenous vein and long saphenous vein.

A.Great saphenous vein:

Saphenous in Greek 'easily seen'. It is the longest vein in the body & preaxial vein of the lower limb.

It is formed on the dorsum of the foot by the union of the medial end of dorsal venous arch and digital vein from the medial side of the big toe. It ascends about 2.5 to 3 cm in front of the medial maleolus and runs upwards crossing the lower part of the medial surface of the tibia obliquely to gain its medial border about finger breath (approx 10 cm from the patella). It passes upwards on the posterior parts of the medial condyle of the tibia and femur. On

its way to saphenous opening it ascends upwards and laterally, pierces the cribriform fascia to enter into femoral vein.

Saphenous opening lies about 2.5 to 3.5 cm below and lateral to pubic tubercle. In thigh some branches of medial femoral cutaneous nerve accompany it. At the knee by the saphenous branch of descending genicular artery and in foot by saphenous nerve which is placed in front of the vein.

TRIBUTARIES

In the leg posterior arch vein (**Leonardo vein**) is a constant vessel, which arises from a series of small venous arches connecting the medial perforating veins. An anterior vein runs up the front of the leg front from the ankle region to join the saphenous below the knee.

A short saphenous vein sends a tributary to it from its upper part frequently to long saphenous vein namely **giacomani vein.**

Antero-lateral and postero-medial veins are the large vessels in the thigh, which joins the saphenous in the upper part of thigh. The accessory saphenous vein is an occasional large trunk, which drains skin, fat of upper and medial thigh.

The superficial veins (external pudental vein, inferior epigastric vein, circumflex iliac vein) enter into saphenous in variable manner at the fossa ovalis.

It contains 15-20 valves which divide into columns to diminish pressure on the vein walls, they are present large numbers in the distal than proximal.

The short saphenous system

This begins posterior to lateral maleolus as a continuation of the marginal veins it ascends lateral to the tendo-achilles then medially to midline of the calf where it pierces the deep fascia. Then it ascends in between the heads of the gastrocnemius to terminate in popliteal vein 1-3 inches above the knee joint. It has a variable mode of termination as it may join great saphenous vein in the proximal thigh. It may bifurcate with one joining the great saphenous other joining the popliteal vein or deep femoral vein. Because of its variable termination venous hypertension of the one system can transmitted to other system.

THE DEEP SYSTEM

They correspond to main arteries. In the upper part of calf close to the popliteal fossa tibial and peroneal join to form the popliteal vein. It enters the adductor canal to become superficial femoral vein and join with deep femoral vein 5-10 cm below the

inguinal ligament to form common femoral vein. It continues as external iliac vein above the inguinal ligament. These veins lie among and they are supported by powerful muscle of the leg.

COMMUNICATING SYSTEM

These are communicating vessels between the superficial and deep vein. They show predilection for intermuscular septum, on either side of sartorius and peronial group, between vastus lateralis and hamstrings, and along anterior border of soleus. There are valves, which allow only unidirectional blood flow from superficial to, deep and distal to proximal. There are two groups

Direct perforators

Indirect perforators

DIRECT

These veins directly connect the saphenous veins or their tributaries to deep veins .a few of these are constant in no and size. They are

In adductor canal (DODD'S Perforator) between long saphenous and femoral vein.

In leg there are 3 groups:

a) MEDIAL Perforator

COCKETT Perforators: There are 3 constant perforators on the medial side of leg which is situated on the posterior border of tibia 2" 4" 6" inches above the medial malleolus. Upper two enter into posterior tibal vein opposite to where the unvalved soleal plexus of veins enter it. So the clots arising from the soleal plexus can enter posterior tibial vein and then into perforator veins that by destroying the valves.

BOYD Perforator: In relation to calf muscle, just below the level of the knee.

MAY or KUSTER Perforator: Ankle perforator at the level of gaiters area to enter into posterior tibial and plantar veins.

PERFORATOR OF SHORT SAPHENOUS

b) CENTRAL PERFORATORS : one or two veins connect the short saphenous and veins of gastrocnemius and soleal muscle . lower is situated at the junction of the calf muscle and tendoachilis ,other is situated in the higher up in calf. they are called as soleus point and gastrocnemius point perforators respectively.

c) LATERAL ANKLE Perforators:

BASSI Perforators-they are situated at 2" 5" 7" inches above the lateral maleolus

INDIRECT PERFORATORS:

They are numerous small vessels, which start from the superficial system and drain into the underlying muscular veins, which in turn connected to deep veins.

FUNCTIONAL ANATOMY AND PHYSIOLOGY OF VENOUS RETURN

1. PRESENCE OF VALVES: Fabricus 1574

Two frail cusps attached to a point of the vein wall (valve ring). They tend to lie immediately distal to the point of entry of a major tributaries and the orifice of major tributaries bear valves. Usually they direct the blood flow from distal to proximal and superficial to deep. Valves occur in greater frequency distally in a limb than proximal. Each valve is made up of gossamer thin cusps, which are delicate and strong. When these become incompetent or destroyed varicose vein occurs.

2. THE VIS A TERO

It means "force from behind" ie-from heart.

3. NEGATIVE PRESSURE WITH IN THORAX

Negative pressure in chest during inspiration is also a factor augmenting venous blood towards the chest.

4. MUSCULOVENOUS PUMP

The soleal plexus of the large vein is designed to be the main peripheral heart. When the muscle contracts pressure in the muscle raise up to 250 mm hg. Intramuscular vein completely empties and the valves in perforators and deep vein direct a flush of blood in deep vein upstream. There is modest raise of pressure in the deep vein.

During the phase of relaxation there is a sharp fall in the pressure in deep vein blood refill in the soleal plexus from the superficial veins. Mean pressure in the superficial system range from 100 to 30 mmhg.

The recognizable effect of musculovenous pump are:-

a). They constitute the genuine form of peripheral heart.

b). They minimize the accumulation of tissue fluid in the lower limb. It occurs only when outflow is obstructed (deep vein obustruction) or if there is incompetence of valves in the superficial or the perforating veins.

5. VENAE COMITANTES

Veins lie beside the artery and may be helped by the arterial pulsation to propel the blood towards heart.

Veins form the part of the post capillary reservoir, which contains 70% of the total blood volume. Because of its venous capacitance there is little change in central venous pressure even though there is large variation in blood volume.

AETIOLOGY

Varicose vein may be

a). Primary

b). Secondary

Although the true aetiology for the primary varicose vein are unknown but certainly there is a hereditary factor as noted by all researchers in the field.

As noted in the incidence F: M ratio 3:1 female hormones are incriminated as the cause . Veins become more prominent during pregnancy and increasing in size during the succeeding childbirth. Since estrogen hormone dilate smooth muscle, its not undesirable to assume that smooth muscles of the veins are affected as that of uterus.

During pregnancy enlarging uterus-increased blood volume and hormones may be a contributing factor. The river of blood from the uterus impedes blood from the periphery.

SECONDARY VARICOSE VEIN

1) The most common cause is post phelebotic syndrome. After the venous thrombosis vein is recannalised at the cost of destruction of valves. Varicose vein results as a result of transmission of high pressure in the deep veins to the superficial veins through communicating veins whose valve becomes incompetent.

2) Gross varicose vein before the age of 15 may be due to congenital arterio-venous malformation.

3) Juvenile varicose veins –due to congenital absence of deep veins.

4) KLIPPEL TRENAUNAY SYNDROME

5) Any pelvic tumor pressing the Iliac veins or IVC.

6) Retroperitoneal Disorders.

PATHOLOGY

Oschner and Mahovier have tabulated the nine features that grossly distinguish full borne varicose vein.

1. Elongation & tortuosity
2. Loss of elasticity
3. Ectasia or dilatation
4. Thickening of intima
5. Increased or decreased thickness of veins
6. Hypertrophy or atrophy of muscularis.
7. Disappreance or atrophy of valves.
8. Thrombosis or calcification.
9. Enlargement of collaterals.

Microscopically the most marked change in the thick walled veins is an increase in thickness of media. Muscle at-times become tremendously hypertrophied and has relatively little intramural connective tissue, which resembles as that of arteries. Thin walled veins show atrophy of the media and replaced by the connective tissue. Thrombosis occurs spontaneously in varicose veins, the process is rapid and eventual recanalisation reduces the original lumen. Calcification is an occasional finding in the thrombus. Pathogenesis of primary varicose vein is obscure. In most instances process appears to spread downwards in the saphenous vein. This makes probable high pressure above the valve stretches the vein until the valve becomes incompetent.

PATHOGENESIS OF SECONDARY VARICOSE VEIN

Venous obstruction in the deep gives abnormal dilatation and narrowing of the veins. Recanalisation will cause destruction of venous valves. This produce incompetence of superficial veins which in turn gives rise to

a. Venous hypertension
b. Passive congestion of tissue and cyanosis
c. Opening of collaterals.

PATHOLOGY OF VEIN WALL IN INCOMPETENT VEINS

Failure of vein wall may be attribute to a problem with regulation of connective tissue. There was reduced amount of collagen and elastin in the varicose vein. Valve failure starts with depression of the commissure of the valves with subsequent widening of the space between the free edges of the valve. There is also hyperplasia of the collagen fibre of the valve cusps. There is infiltration with tissue macrophage and class II MHC antigens were present on the endothelium. Smooth muscles of the tunica media is thickened.

VENOUS ULCER

Venous ulcer occurs either in connection with varicose vein or follows deep vein thrombosis, in which recanalisation of deep veins has occurred but the valves are destroyed or incompetent. Long

standing varicose vein lead to venous stasis favouring local anoxia and edema are the underlying causes of ulcer. But lipolysis of the subcutaneous fat is an important factor. Its important to be sure that ulcer is not due to ischemia or arteritis. Varicose ulcer responds promptly to ambulatory treatment ligation. Where as post thrombotic ulcer is usually refractory to treatment and requires bed rest curettage and split skin graft.

HYDROSTATIC THEORY

Homan attributed leg ulceration to hypoxia. Venous hypertension is the fundamental cause.

FIBRIN CUFF THEORY (BROWSE)

As a result of long standing raised venous pressure, there is an increased capillary leakage result in blood and fibrin getting into subcutaneous tissue. These changes are called lipodermatosclerosis. Fibrin is deposited around the capillaries leading to formation of barriers, which impedes oxygen transfer to skin leading to breakdown and ulceration.

WHITE CELL TRAPPING THEORY (DORMANDY):

In patients with venous hypertension arteriovenous gradient decreases. Blood slows down in capillaries and white cell marginates and trapped. There is decrease in blood flow through the capillaries leading to hypoxia. Cytokines activated proteolytic

enzymes and oxygen free radicals are liberated causing tissue damage.

Ulceration occurs in gaiters area of lower 1/3 of leg and is almost always posterior and superior to medial malleolus of the ankle. They may occur in corresponding area on the lateral side of the ankle and in severe state may encircle the foot. Often ulcers are multiple & scars of healed ulcer can be seen. Hyper pigmentation and dermatitis surrounding the ulcer that is very characteristic. Ulcers are not painful till they get infected. Marjolins ulcer may develop in long standing ulcer.

CLASSIFICATION OF CHRONIC VENOUS INSUFFIENCY

WIDMER CLASSIFICATION

CATEGORY	DESCRIPTION
Hypen webs	venous telengiectiasis or spider veins
Retinacular veins	dilated tortuous subcutaneous not Major trunks and branches
Truncal veins	dilated tortuous SSV or LSV or main branches
CVI grade -1	venous flare at ankle

	"corona pheblectatica"
CVI grade-2	Hyper or depigmented area in gaiter area
CVI grade-3	open or healed venous ulcer.

CEAP CLASSIFICATION

CLINICAL

CLASS-0	No visible or palpable signs of venous disease
CLASS-1	Telangiectasia or reticular veins
CLASS-2	Varicose veins
CLASS-3	Odema
CLASS-4	Skin changes (lipodermatoscelerosis, atrope blanche, and eczema.
CLASS-5	Healed ulceration
CLASS-6	Active ulceration

ETIOLOGICAL

Ec	Congenital
Ep	Primary

Es	Secondary

ANATOMICAL

As	Superficial veins
Ad	Deep veins
Ap	Perforating veins

PATHOPHYSIOLOGICAL

Pr	Reflux
Po	Obstruction
Pr,o	Both

DISABILITY SCORE

1	Asymptomatic
2	Symptomatic but function with out supportive device
3	Can work 8 hours a day but only with supportive device
4	Unable to work even with supportive device

DIAGNOSTIC WORK UP

The preoperative evaluation requires the differentiation between primary and secondary varicose veins and to evaluate the patency of deep venous system. Patient was examined in supine and standing.

CLINICAL EXAMINATION

1. BRODIE TRENDLENBURG TEST:

To determine the in competency of the sapheno-femoral and communicating system. Test consists of two parts.

A) The patient is placed in supine position and leg is raised to empty the veins. The sapheno-femoral junction is compressed with thumb and the patient is asked to stand quickly with thumb released quickly. If the sapheno-femoral valve is incompetent the varices will be filled quickly.

B) In this test after emptying the veins with occluding the SF junction. Patient is asked to stand pressure is NOT released. If the perforator is incompetent varices will be filled slowly.

2. TORNIQUET TEST:(OSHNER MAHONER TEST)

Superficial veins are emptied and tourniquets are tied around the thigh and leg at different levels. Patient is now asked to stand up. A vein filled above or below a torniquet represents the presence

of an incompetent vein. By altering the tourniquet down insteps, position of the incompetent perforator can be identified.

3. PERTHE'S TEST

Veins are emptied and elastic bandage is applied to the lower limb. Patient is asked to move around and exercise. Severe crampy pain occurs in DVT.

4. MODIFIED PERTHE'S TEST

Tying a tourniquet in thigh so as to prevent the reflux .if the deep vein and the perforators are normal the varicose vein will shrink. If blocked, they will become more distended.

5. SCHWARTZ TEST

In long standing case, if a tap is made on the lower part of the leg. Impulse can be felt at the saphenous opening with the other hand.

6. PRATT TEST

Tying an elastic bandage empties veins; tourniquet is applied at the groin. Bandage is now removed & applied from groin downwards. At the site of perforators blow out can be seen and marked.

7. COUGH IMPULSE TEST: (MORISSEY'S)

Veins are emptied, on coughing an impulse is felt in the long saphenous vein if sapheno-femoral valve is incompetent. A bruit may heard on auscultation.

8. FEGAN'S TEST

In standing posture, places of excessive bulge were marked. Veins are emptied in recumbent position. At the marked site gaps are pits may be felt in the deep fascia, which transmits the imperforate vein.

TEST FOR DVT

9. HOMAN'S SIGN

Forceful dorsiflexion of the foot with knee extended will elicit tenderness in calf.

10. MOSSES SIGN

Squeezing the calf from side to side causes pain in the thrombosed deep vein.

ABDOMINAL EXAMINATION

To look for secondary causes.

PERIPHERAL PERFUSION ASSESSMENT

INVESTIGATIONS

NON INVASIVE

A) BIDIRECTIONAL DOPPLER VELOCITY STUDY

10MHz and 5 MHz are suitable for superficial and deep venous systems. Audible signals and their directions are observed in rest, abdominal compression, during Valsalva maneuver, thigh and calf compression.

Superficial venous insufficiency can differentiate from deep vein insufficiency by comparing saphenous and posterior tibial vessel at the supra malleolar level. Superficial venous insufficiency is defined as abnormal reflux in saphenous vein alone. In deep vein insufficiency there is reflux in both the veins.

Incompetent communicating or valves are detected by auscultation with Valsalva maneuver or augmentation there is abnormal reflex towards probe.

Retrograde flow detected by bi-directional probe detects the valvular incompetence. When there is absence of spontaneous flow or diminished augmentation by distal compression indicates obstruction.

B) DUPLEX IMAGING / COLOUR FLOW IMAGING

Duplex imaging permits examination of the short and long saphenous veins as well as the perforating veins in the superficial system. Femoral, popliteal, and calf veins are the deep veins imaged.

Saphenofemoral competence, saphenous vein diameter, wall thickness, diameter of the saphenous tributaries, status of perforators, valvular competence with in the saphenous vein & patency of the deep veins should be assessed. Valvular competency is assessed by three phenomenon. A) Direction of blood. B) Valve thickness. C) Valve cooption.

INDICATIONS

1. To R/O deep vein thrombosis
2. To R/O anatomical variation of the superficial system
3. Short saphenous varicosities
4. 4.Recurrent varicosities

LIMITATIONS

1. Operator dependent.
2. Takes long time.

C) PHOTO PLETHYSMOGRAPHY

Used to demonstrate an “abnormal brief recovery time” after exercise, is restored to normal when the suspected pathway of incompetence is selectively occluded by finger. An unequivocal response is accepted as an evidence of incompetence.

D) AMBULATORY VENOUS PRESSURE

It is defined as superficial venous pressure in the ankle in standing position after 10 tip toe movements.

Patient stands motionless by holding the frame. Superficial venous pressure measured by cannulating dorsal venous arch, is usually 90 mmhg and it depends upon the height of the individual. Then the patient performs 10 tiptoe movements, the pressure usually falls to 15- 30 mmhg.

Once again patient stands still. Venous pressure again falls to base line. Time taken to regain 90% of the baseline level is called Refill Time 90. This is usually 18-40 seconds.

This is a functional test, which assess the efficiency of the venous systems.

INVASIVE

ASCENDING PHLEBOGRAM

It's the 'gold standard investigation' for demonstrating venous occlusion and pattern of collateral flow. It is only used when Doppler can't clearly exclude deep vein occlusion.

INDICATIONS

1. To demonstrate deep vein thrombosis of calf, pelvic varicose veins and IVC.
2. Investigate secondary and recurrent varicose vein.
3. Suspicion of venous malformation.

In presence of varicose veins the appearance of the deep veins before and after the exercise is same as normal veins but superficial vein may be visualized when it is associated with perforator incompetence.

They show dilation and become tortuous following DVT. When obstructed numerous valveless collaterals can be visualized. Following exercise there is poor emptying of the dye from the deep veins, increased filling of the collaterals, perforators and superficial veins. Clots will be seen as filling defects and if recanalised will have irregular margins.

Radiographic signs of DVT are abrupt termination of contrast medium above or below the obustruction, Nonfilling of the entire system, diversion of the flow through the collaterals.

FALSE POSITIVE – Turbulence around the valves, arterial impaction, mixing defects, entry of non-opacified blood, air bubble and overlying band.

Multiple planes and films, Valsalva maneuver, large dose contrast, semi-erect position helps in reducing false positive results.

RADIO NUCLEIDE PHLEBOGRAPHY

Injection of technetium 99m free or albumin labeled the superficial vein and applying compression and diverting the contrast predominately into the deep veins thrombosis can be identified.

- Delayed appearance time.
- Dilution or absence of portion of normal course of major venous segment.
- Presence of collateral pathway.
- Delay in disappearance of radio-nucleide (hot spots).
- bnormal dilution effect associated with excessive collateral flow from venous segments that don't contain radio-nucleide.
- Rate of passage of isotope is equal and rapid in both the limbs. This doesn't occur in venous thrombosis.

VARIOUS MODALITIES OF TREATMENT

TREATMENT

1. CONSERVATIVE TREATMENT.
2. MEDICAL TREATMENT.
3. SURGICAL TREATMENT.

CONSERVATIVE TREATMENT

- GRADED CPMPRESSION STOLKINGS.
- UNNA BOOT
- MULTILAYER COMPRESSION DRESSINGS.
- BISGARD METHOD

BISGARD METHOD

- Massage in elevation of the whole limb particularly to soften the indurated area around the ulcer .
- Passive movement to maintain the mobility of the ankle and foot.
- Active movement to the calf in elevation and sitting (with compression bandage).
- Teaching correct method of walking placing heel down first and using the calf muscle to lift the heel back. Thus giving spring to the walk, therefore improving the venous pump.

- A firm elastic bandage is applied spirally from the base of the toe to the knee. So that movements in walking produces alternatively stretch and relax. Thus the bandage produces add on venous pumping effect.

COMPRESSION THERAPY

Numerous mechanisms have been proposed to explain the benefits of compression therapy.

MACROVASCULAR

- Decrease wall tension and thus further damage to elastin and collagen structure in the wall.
- Increase the velocity.
- Decrease the force of reflux in the perforators.
- Abolish the refluxing blood and thus reducing the venous load.
- Reduce edema and skin tension.
- Reduce AVP.
- Improve refill times.

MICROVASCULAR

Return of starling forces, haemostasis, and leucocytes Margi nation to normal.

- Prevents excessive fluid and protein filtration.

- ❖ Decreases pressure in post capillary venules.
- ❖ Augmenting lymphatic clerance.
- ❖ Augmenting relase of prostacyclin and plasminogen activator from the endothelium.

Compression hosiery

Class-I <25 mmhg at ankle. Thromboembolic prophylaxis and early varicose veins.

Class –II 25-35 mmhg. Advanced varicose veins, odema,

early CVI.

Class-III 35 –45 mmhg. Moderate to severe CVI.

Class-IV >45 mmhg severe CVI and lymphodema.

MEDICAL TREATMENT

1. PENTOXIPHYLLINE- Cytokine antagonist having some fibrinolytic activity.
2. CALCIUM DOBESILATE- Increases lymphatic flow with macrophage-mediated proteolysis, hence reducing edema.
3. DIOSMIN-Flavaniod RUTIN, which increases venous tone and improves lymphatic drainage and platelet microcirculation.
4. PROSTAGLINDIN -PGE1- Reduces WBC activation, platelet aggregation and vaso-dilation.

5. FREE RADICAL SCAVENGERS.
6. ACETYL SALICYLIC ACID- To reduce platelet adhesion.
7. ZINC- In venous ulcer there is decreased level of drugs. Hence supplementation to promote healing.

SCLEROTHERAPHY

First described by MEPHECTERS, popularised by FEGAN (1963).

INDICATIONS

1. Confined to below knee, which are not truncal veins.
2. Recurrent or residual veins
3. Telangiectasia
4. Patient not willing for surgery

CONTRAINDICATIONS

DVT AND SF Incompetence

PRINCIPLE

Sclerosant produces chemical phlebitis and on apposition produces obliteration of the lumen.

SCLEROSANTS

3% Sodium tetra decyl sulphate, ethanolamineoleate, sodiummorruate polidocanol, hypertonic saline.

DISADVANTAGE

Not suitable for large veins, allergic problems, extravasations may cause skin necrosis, local pain, pheriplebitis, intra arterial injection and rarely DVT.

PROCEDURE

Patient sits in a waist height couch with lower limb horizontal. Needle with sclerosant is inserted into the skin marks, which are made previously. Vein is emptied by elevating the limb. Sclerosant is injected into the vein, which is compressed from above and below. As soon as the injection is over, the site is compressed with cotton ball. To prevent refilling of vein and compressive bandage is applied. Patient is encouraged to walk immediately. Bandage is applied for 3 weeks. Regular follow up and further injections may be needed for residual and recurrent varicosities.

SURGICAL MANAGEMENT

INDICATIONS

Symptoms like aching, heaviness, edema , which are relived by compression therapy.

1. Chronic venous insuffiency.
2. Large varicosities subjected to trauma.
3. Cosmetic.

Preoperative MARKING is the most important step in surgical procedure .

SURGERIES FOR SUPERFICIAL VARICOSITES:

- Simple high and flush ligation of sapheno-femoral or/and sapheno popletial junction. (trendelen-burg procedure)
- Ligation and stripping.
- Multiple phlebectomies by rivilin.
- Cockett and dodd's subfacial ligation of perforators.
- Linton's radical subfacial ligation of perforators

Vohra and odogwn introduced endoscopic venous surgery.

Contraindications for surgery

1. DVT.
2. Arterial ischemia.
3. Skin and soft tissue infection.
4. Lymphoedema.
5. Bleeding diathesis.
6. Pelvic tumor.
7. Poor anaesthetic risk.
8. Patient is on OCP is relative contraindication.

VENOUS RECONSTRUCTIVE SURGERY

It is only when there is chronic obustruction and to correct the reflux.

OBSTRUCTION

DE-PALAMA

It is designed to by pass unilateral ILIAC VEIN OCCLUSION. Contra lateral LSV is tunneled supra-pubically and anastamosed to patent common femoral or superficial femoral.

MAY HUSNI OPERATION

Obstructed femoral segment may be bye-passed by anastamosing transected competent LSV to the side of popliteal vein.

REFLUX CORRECTION

The edge of the floppy wall cusps can be sutured to the vein wall.

KISTNER OPERATION

Involves longitudinal venotomy directly through commissure.

VALVULOPLASTY

It is of two types.

External- Vein diameter around the incompetent valve is reduced by vein wall plication and synthetic cuff around the vein.

Internal-Transverse incision above the level of the valve and commissural reefing is performed.

VEIN VALVE TRANSPLANTATION

Autologous valve transposition interposes a segment of axillary or brachial vein, contining a competent valve into an incompetent deep veins. Procedures using synthetic , mixed and animal valves are in experimental stage.

VEIN TRANSPOSITION

An incompetent superficial vein can be transected and end to end or end to side to profunda femoris or long saphenous vein which has competent valve.

NEWER MODALITIES OF TREATMENT

Endovenous laser surgery

Endo-venous cryo surgery

COMPLICATION OF VARICOSE VEINS

1. Odema.
2. Pigmentation.
3. Eczema.
4. Ankle flare.
5. Thrombophlebitis.
6. Varicose ulcer.
7. Haemorhage.
8. Periostitis.
9. Calcification.
10. Equinus deformity.

CAUSES FOR RECURRENCE

- inaccurate pre op assesment.
- incomplete operative technique.
- continuing same pattern of life style

MATERIALS AND METHODS

All the cases were studied in Kilpauk Medical College Hospital during the period between “March 2003 to August 2005”.

All the cases underwent detailed evaluation regarding presenting symptoms, duration and previous modality of treatment. Clinical examination was made as to which system was involved and to identify which superficial valves and perforators were incompetent. Deep vein patency was assessed. Abdominal and pelvic examination was made to search for secondary causes. Cardiovascular and peripheral pulses were assessed to exclude arterial disease.

Routine investigations and selective duplex assessment was done. Compressive therapy was advised for symptomatic groups. Ulcer was treated with dressing till healthy granulation tissue was seen.

Most patients underwent surgical treatment. Choice of surgery is determined by the extent of disease and associated pathology. Post op crepe bandage was applied. These patients were reviewed 2weeks after discharge and followed till 6 months.

PROFORMA

NAME AGE SEX

UNIT OP/IP NO-

OCCUPATION INCOME

PRESENTING COMPLAINTS

1. ASYMPTOMATIC
2. PAIN
3. ABDOMINAL SYMPTOMS
4. ULCERATION AND ITS COMPLIANTS

PAST HISTORY

SCLEROTHERAPHY/ COMPRESSION THERAPHY/ SURGERY

PERSONAL HISTORY.

HT / DM / OBESTETRIC / CONTRACEPTIVE

FAMILY HISTORY

CLINICAL EXAMINATION:

INSPECTION

VARICOSE VEINS

LSS

SSS

SWELLING

LOCALISED /GENERALISED

SKIN OVER THE LIMB

COLOUR TEXTURE

EDEMA

ECZEMA

PIGMENTATION

ULCERATION SITE SIZE SURFACE, SCAR

SIGNS OF ISCHEMIA

PALPATION

TEMPERATURE

TRENDELENBURG - 1

- 2

TORNIQUET TEST

MODIFED PERTHES TEST

COUGH IMPULSE TEST

SCHWARTZ TEST

REGIONAL NODES

COMPLICATIONS

ABDOMINAL EXAMINATION

INVESTIGATIONS

TREATMENT

CONSERVATIVE/ SCLEROTHERAPHY/ SURGICAL TREATMENT

SURGICAL PROCEDURE

POST OPERATIVE PERIOD FOLLOW UP

OBSERVATION AND ANALYSIS

AGE-SEX DISTRIBUTION OF THE PATIENTS

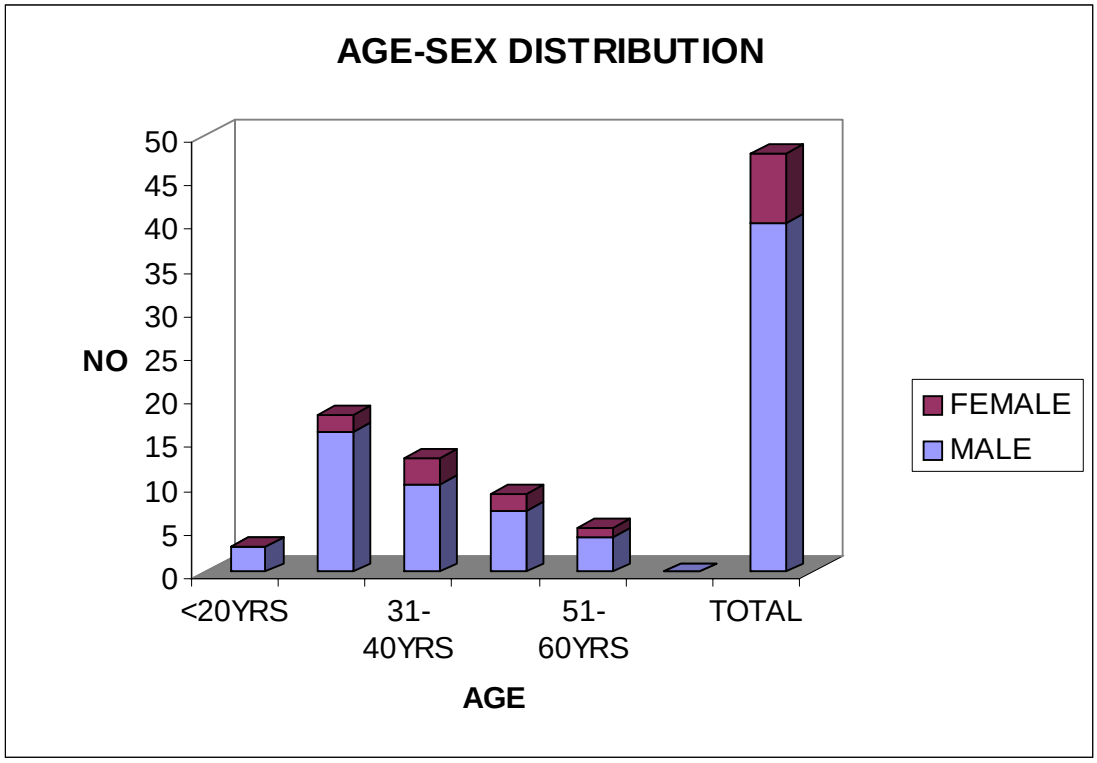
AGE YEARS	MALE No	%	FEMALE no	%
<20 YRS	3	6.2 %	0	0 %
21- 30	16	36.5 %	2	4.2 %
31-40	10	20.8 %	3	6.2 %
41- 50	7	14.5 %	2	4.2 %
51-60	4	8.3 %	1	2.1 %
> 60	0	0 %	0	0 %
TOTAL	**40**	**83.8 %**	**8**	**16.7 %**

In my study male constitute 83% and female 17 % .in contrast to the literature male female ratio was approx 5:1.

Female are distributed around 25 to 40 yrs . males 56% are in the age group around 21- 40 yrs.

Hence this disease mostly affects the economically productive group.

Surprisingly as the age goes on disease is less prevalent.


AGE-SEX DISTRIBUTION
NO
50
45
40
35
30
25
20
15
10
5
0
<20YRS
31-40YRS
51-60YRS
TOTAL
AGE
FEMALE
MALE

MEAN DURATION OF PRESENTATION

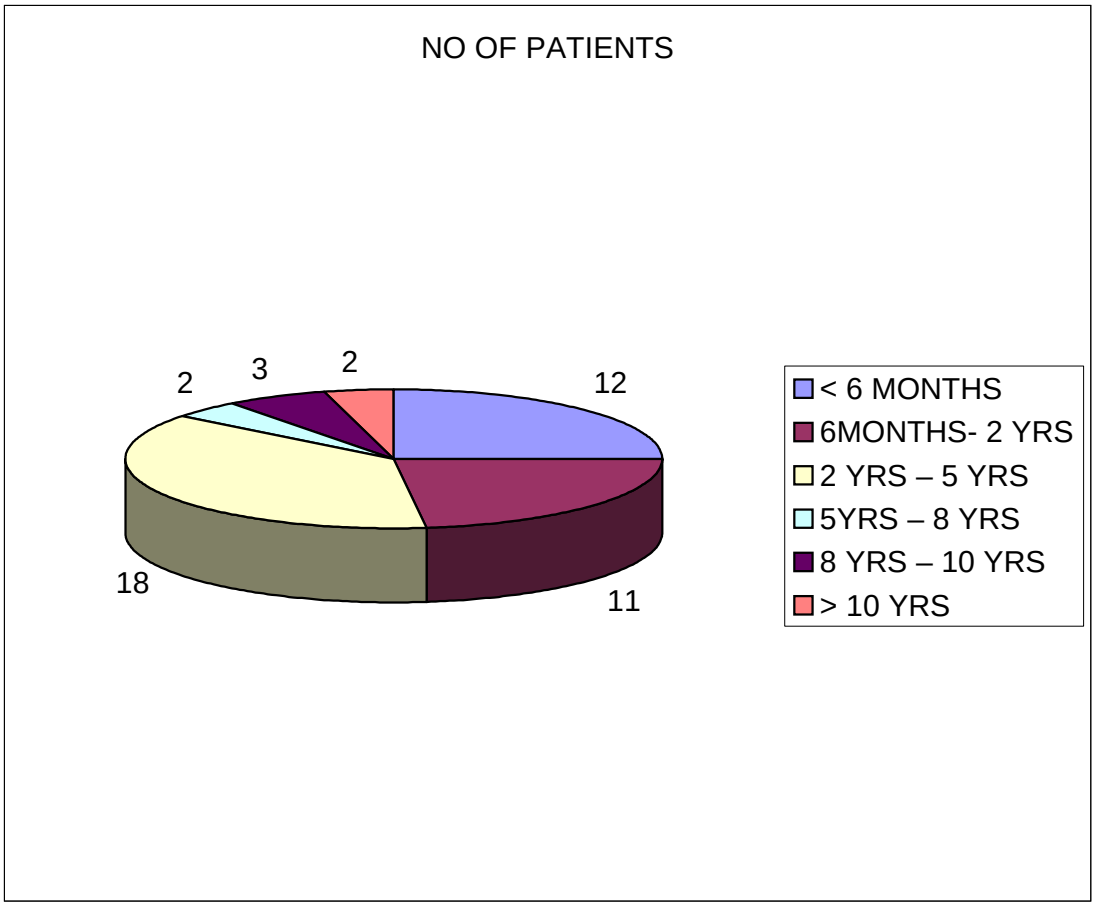
PRESENTATION	NO OF PATIENTS	PERCENTAGE
< 6 MONTHS	12	25.0%
6MONTHS- 2 YRS	11	22.9%
2 YRS – 5 YRS	18	37.5%
5YRS – 8 YRS	2	4.2%
8 YRS – 10 YRS	3	6.2%
> 10 YRS	2	4.2%
TOTAL	**48**	**4.2%**

Most of the people presented with symptoms persists more than 2 yrs before they seek the medical attention.

25% presents with in six months duration and another 23% take for about at least 2 yrs to medical consultation.

Those who take 2-5 yrs constitutes 37% .those who takes more than 5 yrs constitutes 14 %.

Time duration for the presentation between 3 months minimum and 20 yrs maximum.


NO OF PATIENTS
2
3
2
12
18
11
< 6 MONTHS
6MONTHS- 2 YRS
2 YRS – 5 YRS
5YRS – 8 YRS
8 YRS – 10 YRS
> 10 YRS

VARIOUS OCCUPATIONS OF THE PATIENTS

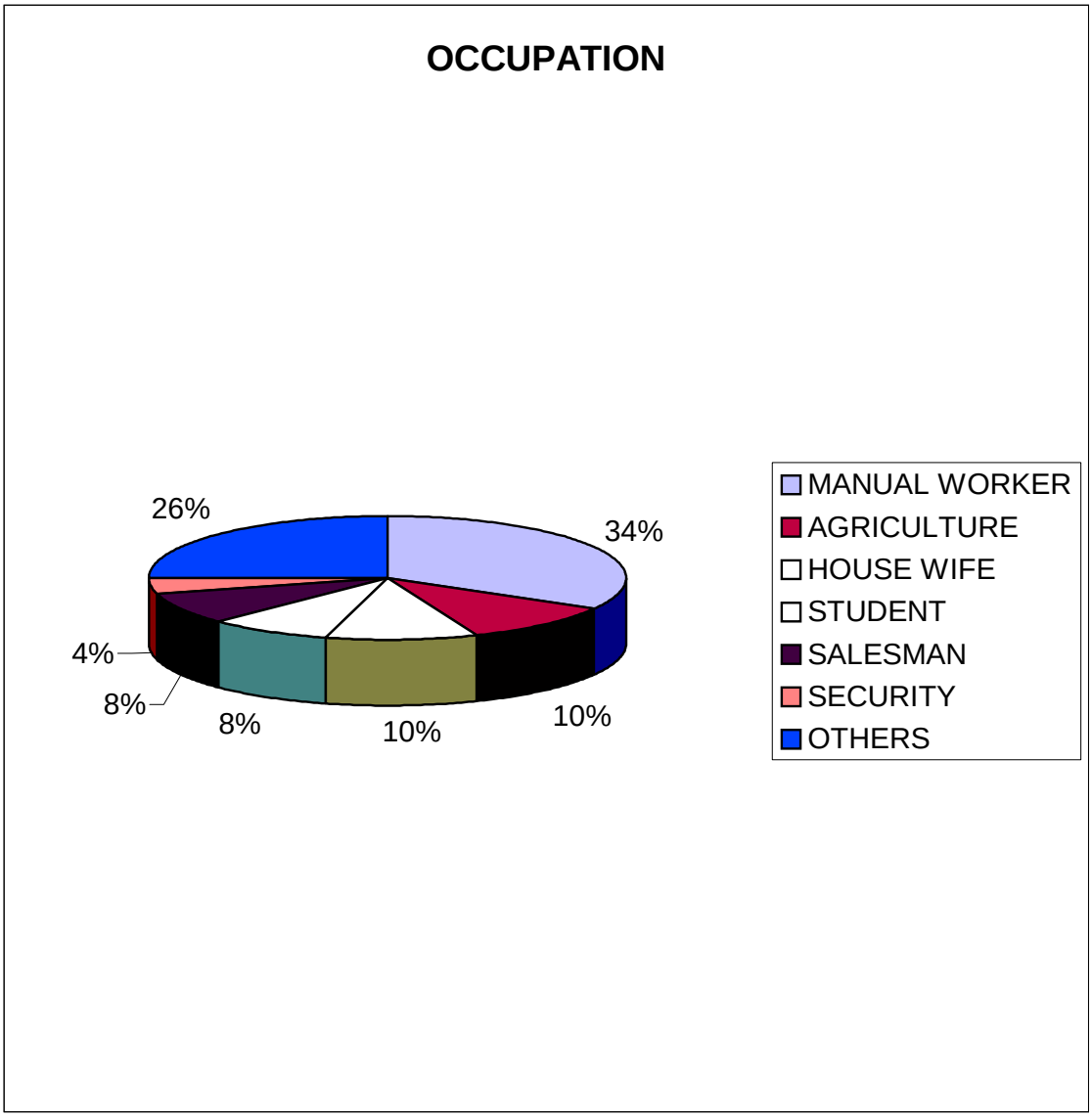
OCCUPATION	NO	PERCENTAGE
MANUAL WORKER	16	33.3 %
AGRICULTURE	5	10.4 %
HOUSE WIFE	5	10.4 %
STUDENT	4	8.3 %
SALESMAN	4	8.3 %
SECURITY	2	4.2 %
OTHERS	12	25.0 %
TOTAL	**48**	**100.0 %**

Manual labourer forms the largest proportion around 33%.

Agriculturists and housewife forms 10% each.

This disease affects mostly lower socioeconomic classes and daily wagers.

Those who stand for prolonged periods constitutes salesman security, constables manual labourers forms around 60%.

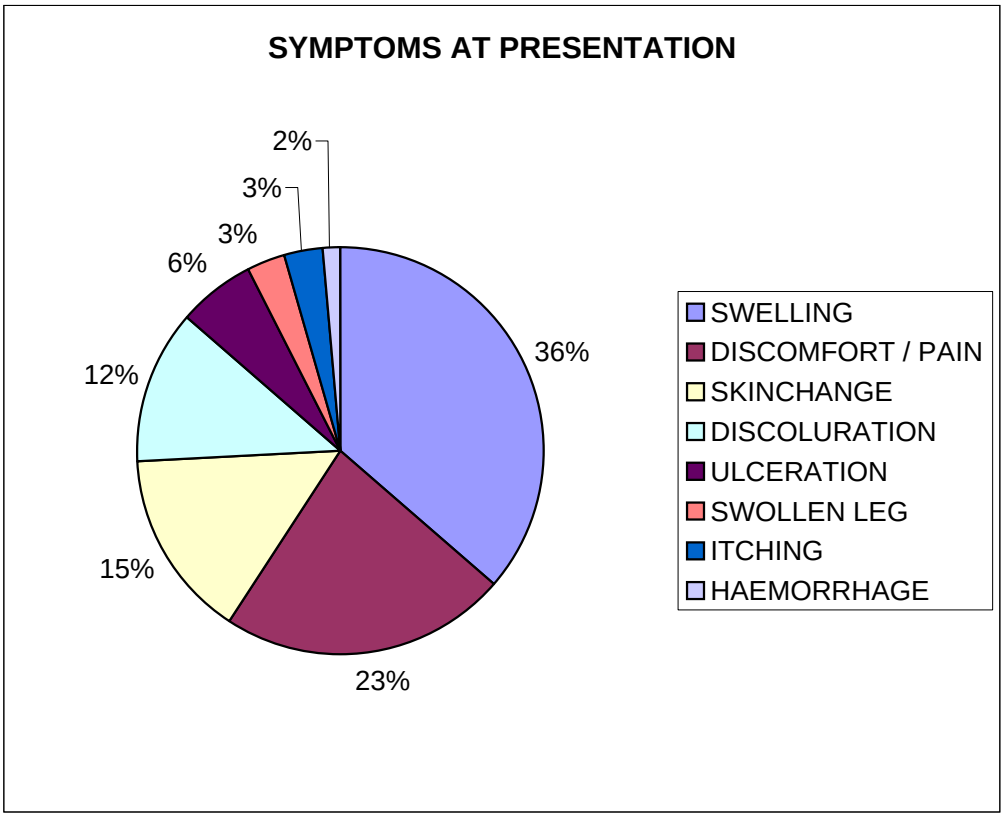

OCCUPATION
26%
34%
4%
8%
8%
10%
10%
MANUAL WORKER
AGRICULTURE
HOUSE WIFE
STUDENT
SALESMAN
SECURITY
OTHERS

DIFFERENT SYMPTOMS AT PRESENTATION

SYMPTOM	NO	PERCENTAGE
SWELLING	24	50.0%
DISCOMFORT / PAIN	15	31.2 %
SKINCHANGE	10	20.8 %
DISCOLURATION	8	16.7 %
ULCERATION	4	8.3 %
SWOLLEN LEG	2	4.2%
ITCHING	2	4.2 %
HAEMORRHAGE	1	2.1 %

50% of the people presents with swelling and pain around 30% which are related primarily to the dilated varicose veins.

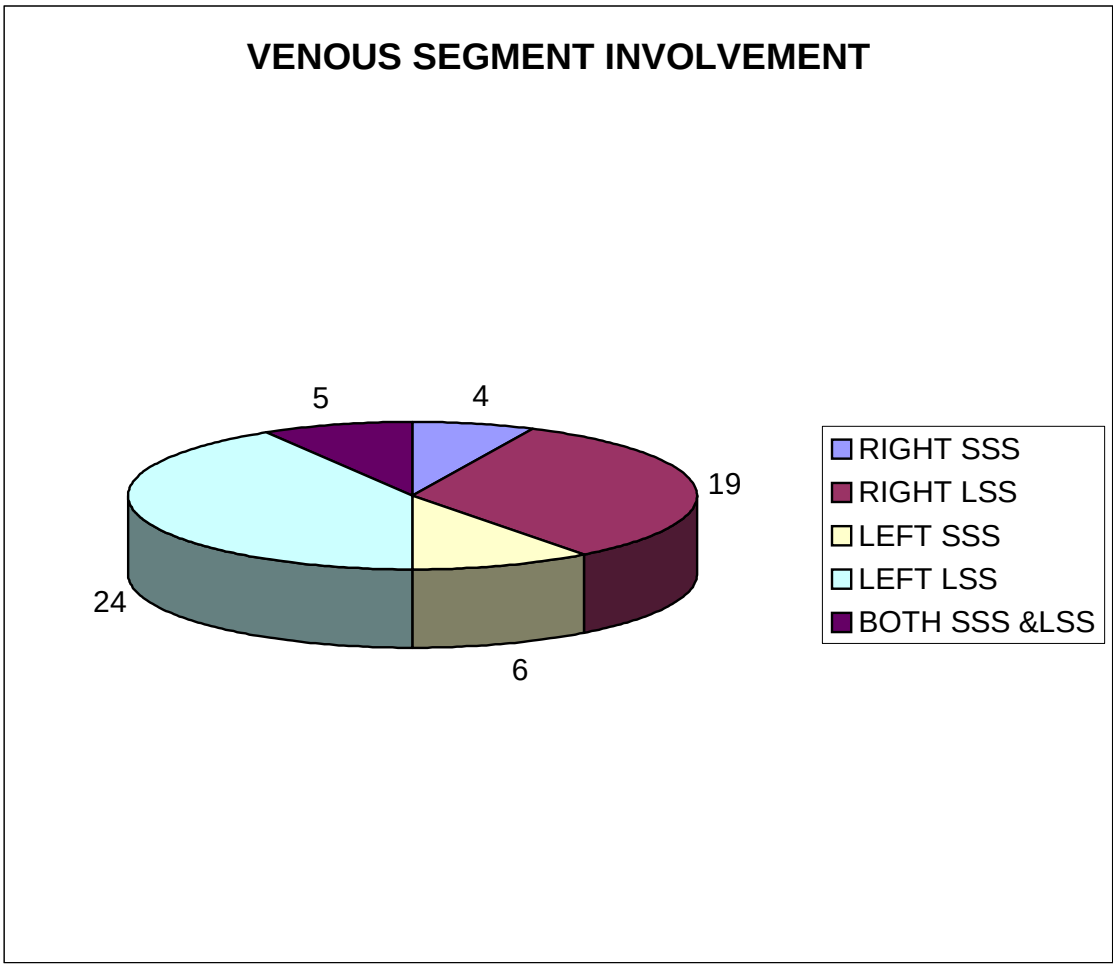
Symptoms due to chronic venous congestion like skin change, discolouration swollen leg and ulceration was presents with 15, 12, 3 and 6 % respectively.


SYMPTOMS AT PRESENTATION
2%
3%
3%
6%
12%
15%
23%
36%
SWELLING
DISCOMFORT / PAIN
SKINCHANGE
DISCOLURATION
ULCERATION
SWOLLEN LEG
ITCHING
HAEMORRHAGE

MODALITIES OF SEGMENT INVOLVEMENT

SIDE	SEGMENT	NO	PERCENTAGE
RIGHT	SSS	4	8.3 %
	LSS	19	39.6 %
LEFT	SSS	6	12.5 %
	LSS	24	50.0 %
BOTH	SSS &LSS	5	10.4 %

LSS is commonly involved around 89% and short shaphenous around 20%. Left sided lesions constitute 62% and right-sided lesions constitute only 48%. Both LSS and SSS make around 10 %.

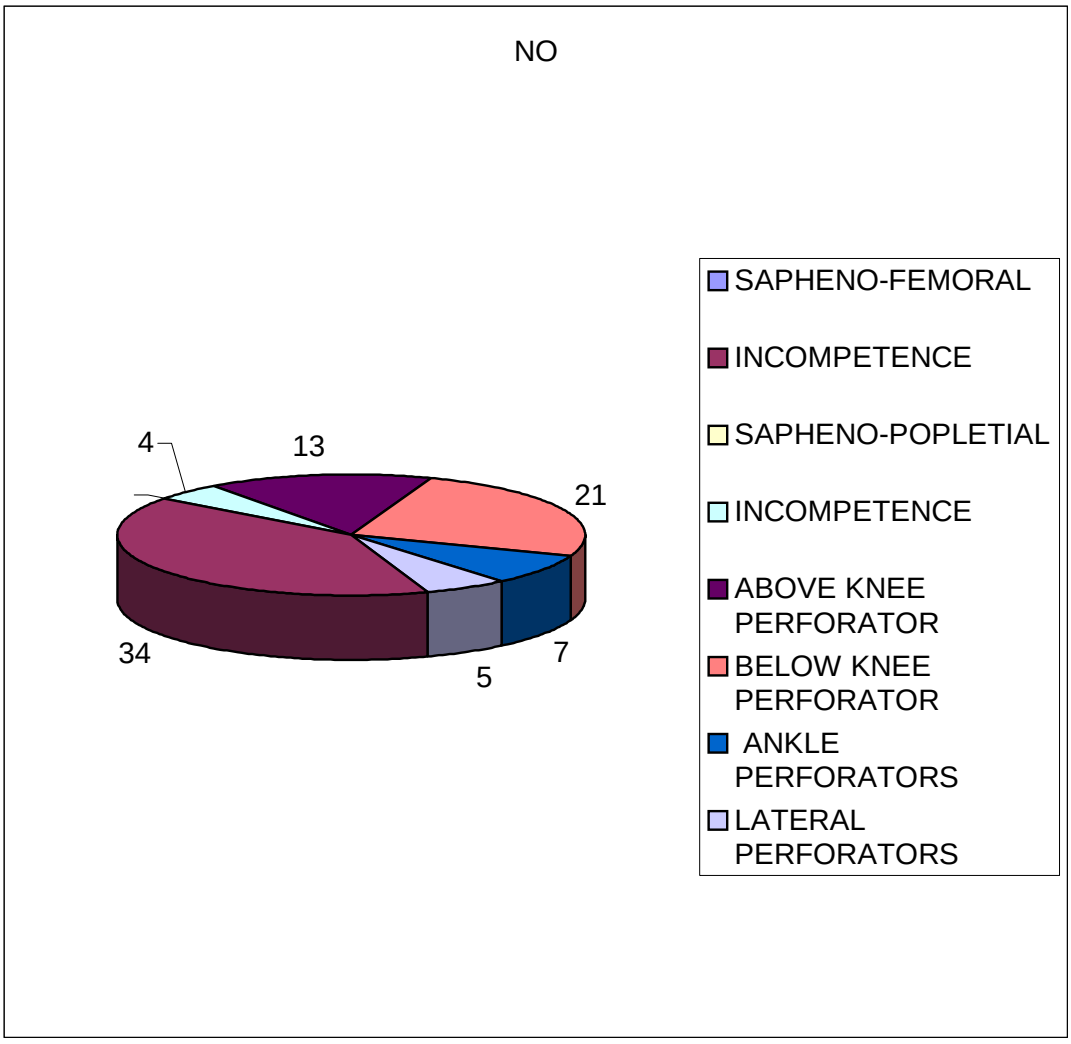

VENOUS SEGMENT INVOLVEMENT
5
4
19
24
6
RIGHT SSS
RIGHT LSS
LEFT SSS
LEFT LSS
BOTH SSS &LSS

DIFFERENT PATHOLOGY OF VARICOSE VEINS

PATHOLOGY	NO	PERCENTAGE
SAPHENO-FEMORAL INCOMPETENCE	34	70.8 %
SAPHENO-POPLETIAL INCOMPETENCE	4	8.3 %
ABOVE KNEE PERFORATOR	13	27.1 %
BELOW KNEE PERFORATOR	21	43.7 %
ANKLE PERFORATORS	7	14.5 %
LATERAL PERFORATORS	5	10.4 %

Sapheno-femoral incompetence constitutes the bulk of the disease 70 % of the in my study group. Sapheno-popletial incompetence occurs in 8 % of the people.

Below knee perforator incompetence occurs in 43%. Above knee perforator incompetence occurs in 27%. Lateral ankle perforator incompetence is lest common only 10%


NO
4
13
21
34
5
7
SAPHENO-FEMORAL
INCOMPETENCE
SAPHENO-POPLETIAL
INCOMPETENCE
ABOVE KNEE PERFORATOR
BELOW KNEE PERFORATOR
ANKLE PERFORATORS
LATERAL PERFORATORS

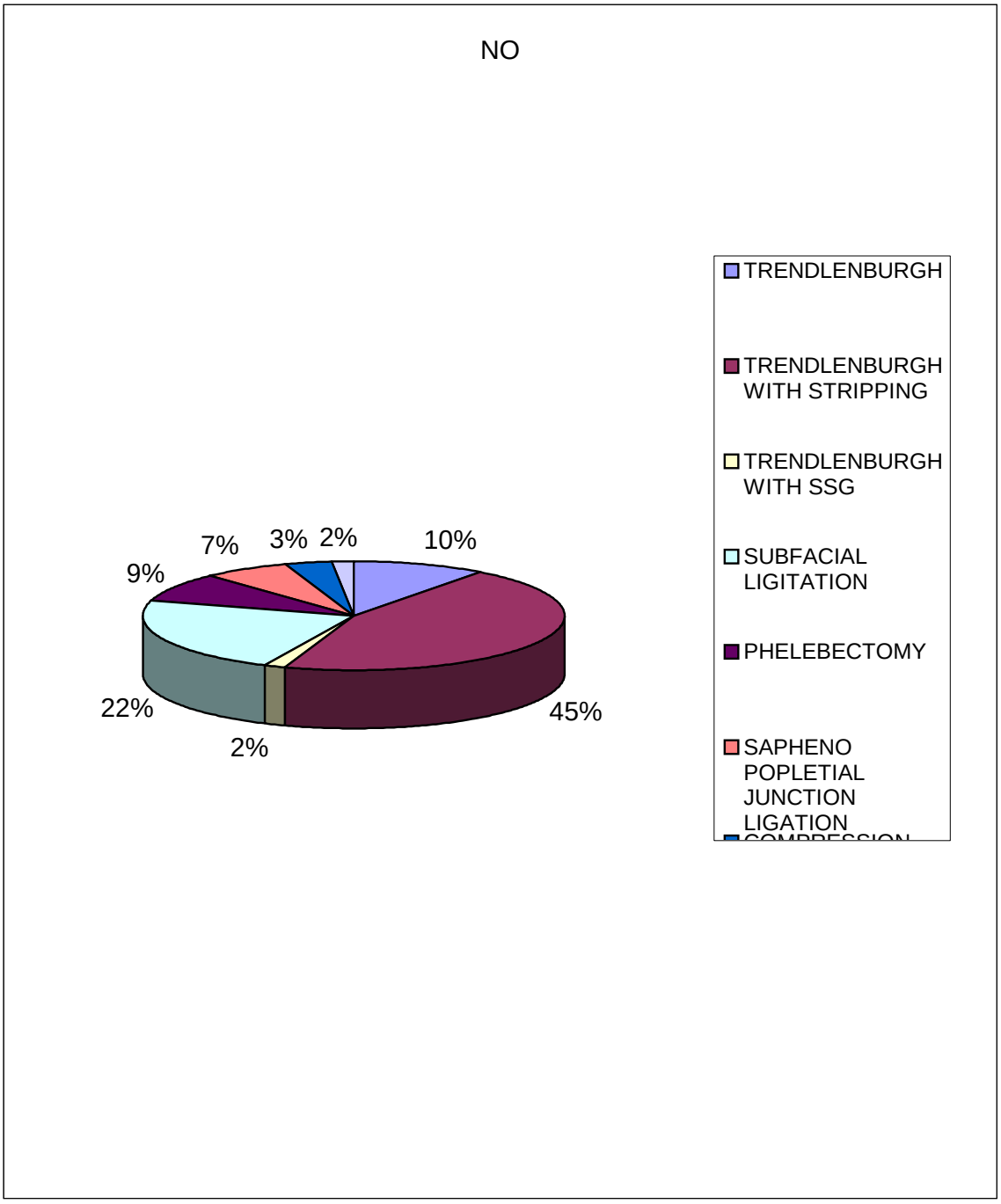
DIFFERENT MODALITY OF TREATMENT

Treatment	No	Percentage
Trendlenburg	6	12.5 %
Trendlenburg with stripping	26	54.2 %
Trendlenburg with SSG	1	2.1 %
Subfacial ligitation	13	27.1 %
Phelebectomy	5	10.4 %
Sapheno popletial junction ligation	4	8.3 %
Compression therapy	2	4.2 %
Suprafacial ligation	1	2.1 %

Trendlenburg with stripping constitutes 54 % and subfacial ligation done in 27%.

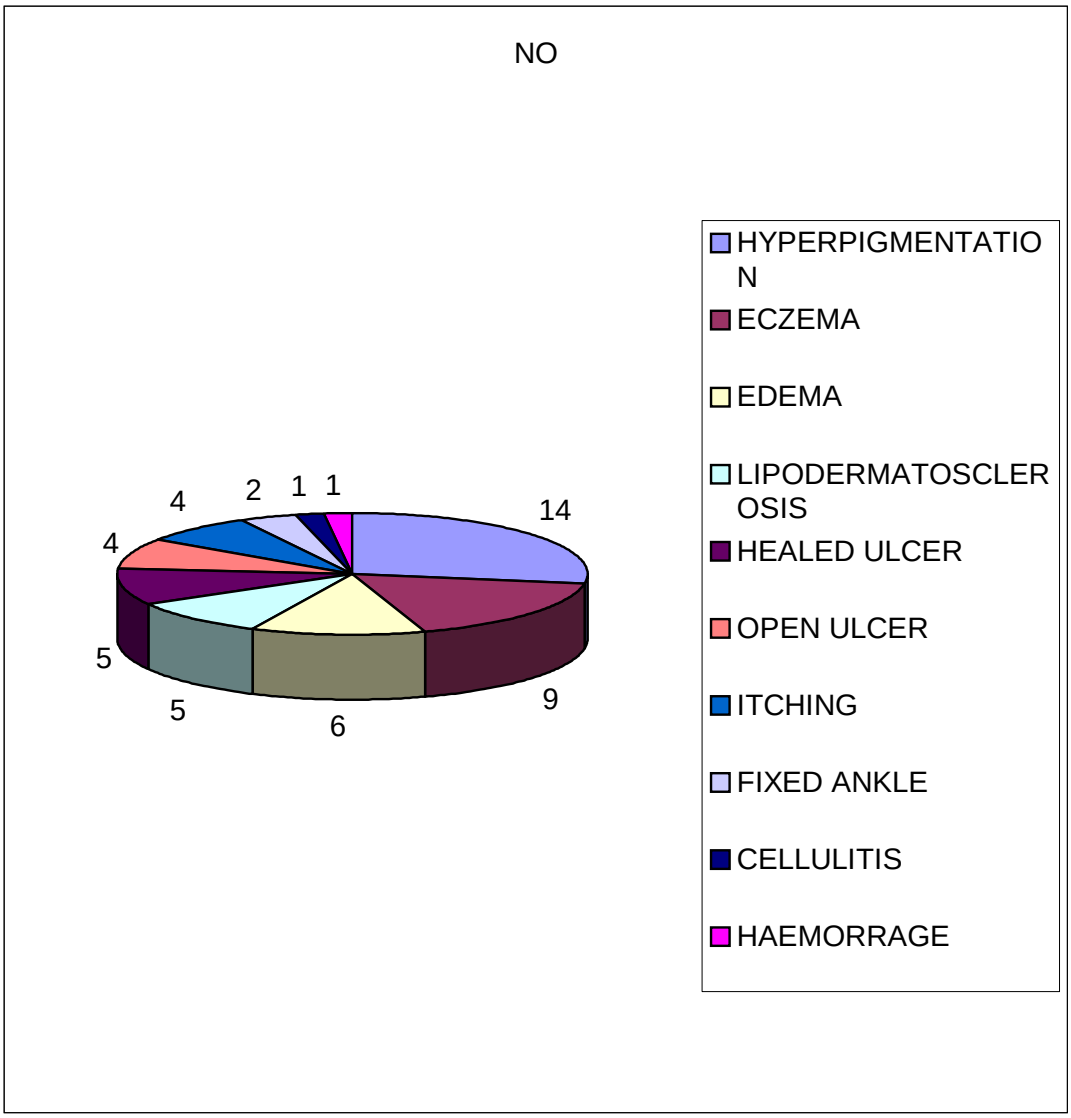
Trendlenburg procedure done in 12%. Hence saphenofemoral junction ligation in 68 %.

Plebectomy and sapheno-popletial done in 10 percent each.


NO
10%
45%
2%
22%
9%
7%
3%
2%
TRENDLENBURGH
TRENDLENBURGH WITH STRIPPING
TRENDLENBURGH WITH SSG
SUBFACIAL LIGITATION
PHELEBECTOMY
SAPHENO POPLETIAL JUNCTION LIGATION

COMPLICATIONS OF VARICOSE VEIN

Complications	No	Percentage
Hyperpigmentation	14	29.2 %
Eczema	9	18.7 %
Edema	6	12.5 %
Lipodermatosclerosis	5	10.4 %
Healed ulcer	5	10.4 %
Open ulcer	4	8.3 %
Itching	4	8.3 %
Fixed ankle	2	4.2 %
Cellulitis	1	2.1%
Haemorrage	1	2.1 %

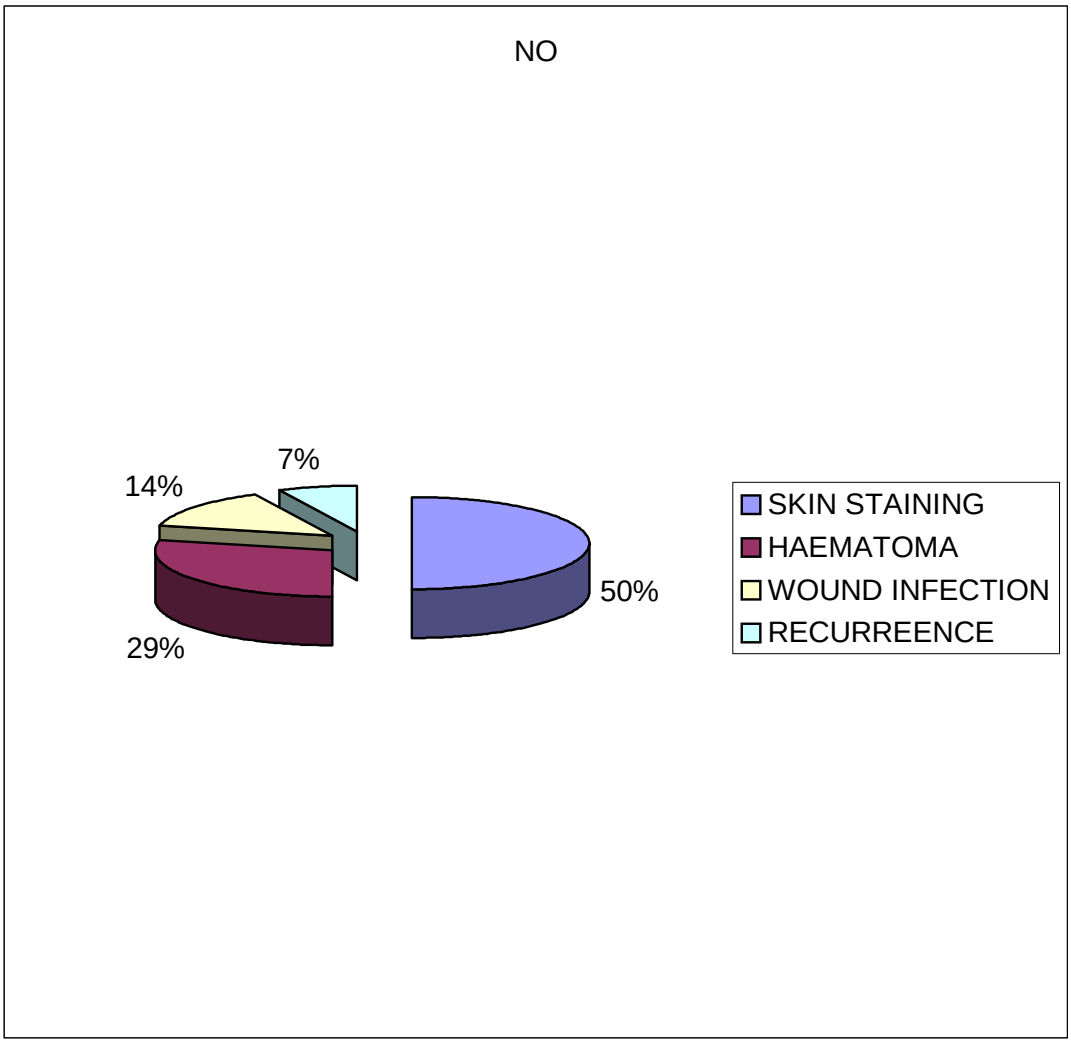

NO
14
9
6
5
5
4
4
2
1
1
HYPERPIGMENTATION
ECZEMA
EDEMA
LIPODERMATOSCLEROSIS
HEALED ULCER
OPEN ULCER
ITCHING
FIXED ANKLE
CELLULITIS
HAEMORRAGE

POST-OPERATIVE COMPLICATIONS

Complications	No
Skin staining	7
Haematoma	4
Wound infection	2
Recurrence	1

Skin staining is the most common complication in 7 patients and recurrence in 1 patient.

Haematoma is next common complication which occurred in 4 patients.


NO
7%
14%
50%
29%
SKIN STAINING
HAEMATOMA
WOUND INFECTION
RECURREENCE

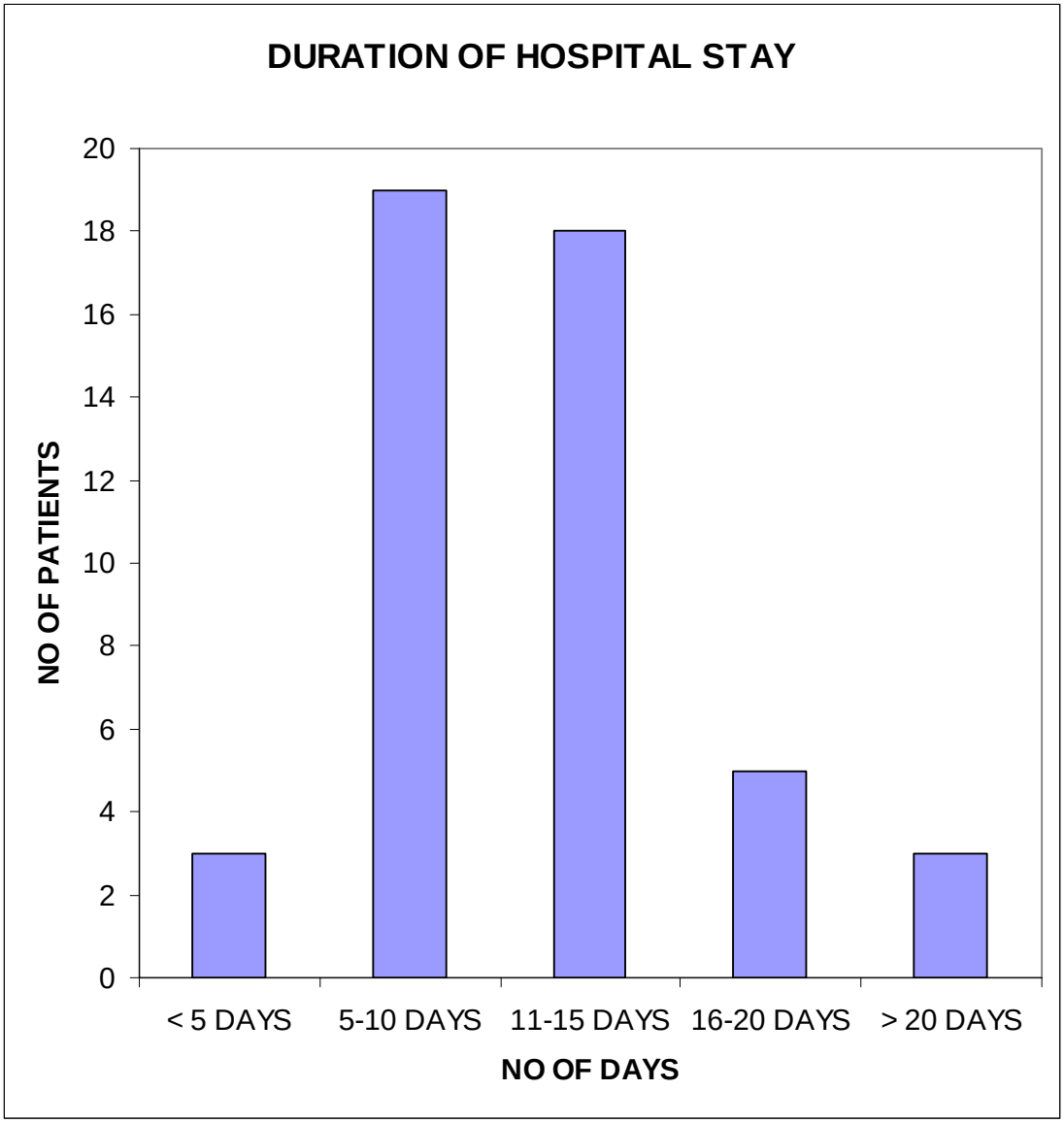
DURATION OF HOSPITAL STAY

Duration of Hospital stay	No	Percentage
< 5 days	3	6.3
5-10 days	19	39.5
11-15 days	18	37.5
16-20 days	5	10.4
> 20 days	3	6.3

40 % of the people stayed mean duration 5-10 days in hospital. 37% people stayed for the duration about 11-15 days.

22 % people stayed either less than 5 days or more than 15 days in the hospital.

Minimum 4 days maximum 29 days is duration of hospital stay for the patient.


DURATION OF HOSPITAL STAY
NO OF PATIENTS
20
18
16
14
12
10
8
6
4
2
0
< 5 DAYS
5-10 DAYS
11-15 DAYS
16-20 DAYS
> 20 DAYS
NO OF DAYS

CONCLUSION

Males constitute 83.8 % of our study and females 16.7% . male: female ratio 5:1.

In males it affects predominately economically productive people between 20 to 40 yrs.

Mean age of presentation is around 2 years. Only three patients less than six months and longest duration are 20 years. Varicose vein being a benign disorder hence people don't seek medical attention early.

Manual worker constitutes 33% of our study. Occupations like manual labourer, sales man, farmers, security guards constitute around 60%.

Half of the people seek medical attention due to varicosities and one third of patient complaints discomfort or pain. Ulceration and hemorrhage was about 8 % and 2 % respectively.

Left sided lesions are common than right side.

Long saphenous segment is more commonly than short saphenous segment. Both systems are involved in 10% of people.

Sapheno-femoral incompetence is the most commonly observed pathology. Of the perforators below knee perforator is commonly involved and lateral perforator least.

Trendlenburg procedure with stripping is the most commonly done procedure. SSG is done only 2% with the above procedure. Next commonly done procedure is subfacial ligation.

Hyperpigmentation is common complication. Edema healed ulcers and lipodermatosclerosis occur in 10 % of people.

Cellulitis and hemorrhage in 2% of the people.

Skin staining is the common complication. Recurrence in 2% of people.

Mean hospital stay is 11 days' minimum-4 days and maximum – 29 days.

BIBLIOGRAPHY

1. Oxford text book of surgery.
2. ESSENTIAL SURGICAL PRACTICE higher surgical training in general surgery 4th edition.
3. Sabiston TEXT BOOK of SURGERY 17th edition.
4. BAILEY & LOVES SHORT PRACTICE OF SURGERY 24 edition
5. Vascular surgery by Robert Rutherford.
6. SCHWARTZ principle of surgery 7th edition.
7. A manual of clinical surgery S.Das 1994.
8. HAMILTON BAILEY'S DEMONSTRATION OF CLINICAL SIGNS in general surgery 17th editon.
9. A synopsis of surgical anatomy Lee McGrecor.
10. BROWSE N.L BURNAD K.G. Causes of venous ulceration.
11. COCCKETT F.B Pathology and treatment of venous ulcer of the leg.
12. COCCKETT F.B JONES B.E Ankle blow out syndrome a new approach to the varicose problem.

13. DAVIS TIBISS Venous disorder, venous malformation and chronic ulceration in the lower limb.

14. HOMAN.J The aetiology and treatment of varicose ulcer in the leg.

15. LINTON R.R The post thrombotic ulceration of the lower extremity its aetiology and treatment.

16. RIVILIN.S the surgical cure of primary varicose vein.

17. STEPHEN D.BLAIR ET AL Sustained compression and healing of venous ulcer.

MASTER CHART

SL	IPNO	NAME	Age/Sex	Dura	occupa	SEGM	PATHOLOGY	PRESENTA	COMPLI	SURGERY	POST-	Hos	RE
1	2189/03	LOGANATHAN	34/M	2 Mn	sales	LLS	SFI	sk+pa		tr+st		12	
2	3424	SRIDAR	55/M	3.5 Y	manual	LLS	BK+AK	sk+it		tr+st		6	
3	7344	SIVAKUMAR	21/M	9 Mn	student	RLS	SFI	sw		tr+st		8	
4	10618	CHINNA THAMBI	50/M	14 Y	manual	LLS	SFI	sw	it	tr+st		6	
5	13859	SUNDARAMOORTHY	48/M	2 Mn	securit	RLS	SFI +BK	pa+sw		tr+st	sk	13	
6	14285	NAGARAJ	35/M	2.5Y	office	BOTH	SFI+BK+AK+LA	swelling		tr+st		21	
7	14649	RAMESH	21/M	5 Y	student	RSS	SPI+LA	sk	hp+ec	spj	sk	12	
8	15424	MUNNUSAMY	27/M	3 Mn	manual	LLS	SPI+AK+BK	sk+pa		ple+sfl		13	
9	18187	VENKUTTAIAN	48/M	4Mn	auto	LLS	SFI	pa		tr+st		21	
10	21184	HARIRISHNAN	38/M	9 Y	manual	LSS	SFI+BK	sk+pa	hu+ld	tr+st		6	
11	1343/04	PANDU	50/M	4 Y	manual	LLS	SFI	sw+pa		tr+st		9	
12	2503	POONGKODI	58/F	12 Y	house	BOTH	SFI+BK+AA	sw+pa		tr+st+sfl		10	
13	2629	DAMODARAN	20/M	6 Mn	student	LSS	SFI	sw		tr+st		12	
14	2659	OSCAR	25/M	2.5 Y	student	RLS	SFI+AK	sw		tr+st	sk	7	
15	2921	SUBASH	27/M	5 Y	industry	RSS	LA	sw		spl+sfl		9	
16	4902	SHANKAR	26/M	2 Mn	agricul	LLS	SFI	pa		tr+st		7	
17	11564	MEENAKSHI	40/F	5 Y	agricul	RLS	SFI	sk+sw	fa+hp	tr	wi ha	8	
18	16826	LOGANATHAN	52/M	8 Y	securit	LLS	SFI+BK	sk	hu+hp+ec	tr+st	sk	10	
19	17447	SURESH	20/M	3 Y	student	LLS	SFI	SW		tr+st		12	
20	17945	MULLAIKUDI	32/F	10 Y	house	RSS	SFI	sw+pa+di	ec+hp	ple		6	
21	18523	NAGAVALLI	32/F	7.5 Y	house	LLS	AK+BK	sw+pa		tr+st+sfl		6	
22	19303	SATISH KUMAR	27/M	2 Mn	agricul	RLS	SFI+AK+BK	ul	ou	ple		12	
23	20006	KRISHNAPPA	55/M	20 Y	manual	RSS	SFI+AK+BK	sw	hu	spl+ssg		18	
24	20522	YASODA	28/M	1 Y	house	RLS	SFI+BK	sw			ha	6	
25	23729	ELUMALAI	32/M	3 Y	ricksha	RLS	SFI	pa	ld+hp	tr		15	
26	26649	MOHAN	41/M	5 Y	police	LLS	SFI	sw		tr+st		5	

SL	IPNO	NAME	Age/Sex	Dura	occupa	SEGM	PATHOLOGY	PRESENTA	COMPLI	SURGERY	POST-	Hos	RE
27	26735	LAKSHMIKANT	28/M	2.5 Y	agricul	RLS	SFI+BK	pa+di	hp+ec+fa	tr+st		8	
28	28724	MOHAN	41/M	4 Mn	manual	LSS	LA	ed	ed	spl		13	
29	28885	SUNDAR	26/M	5 Y	sales	RLS	SFI	dI+PA	hp+ec+ld	tr+st	wi	12	
30	29971	ELUMALAI	35/M	3 Y	police	RLS	BK	fe	hp+ed+cel	sfl		2	
31	30808	BABU	18/M	7 Mn	securit	RLS	SFI	SW		tr+st		7	
32	34037	KUMAR	40/M	4 Y	manual	RLS	SFI	PA		tr+st		6	
33	35540	PALANI	27/M	6 Mn	agricul	LLS	AK+BK	SK	hu+hp	ple		9	
34	1212/05	SASIKUMARI	47/F	10 Y	industry	BOTH	SFI+AK+BK+AA	PA +DI	ec	tr+st	sk	16	
35	1619	RAJENDRAN	35/M	2.5 Y	industry	RLS	BK	SW+DI+PA	ou	com		12	
36	2105	KUPPAN	41/M	3.5 Y	agricul	LLS	SFI+BK+AA	SW	hp	tr		18	rec
37	2728	MOHAMED FAROOQ	50/M	5 Y	manual	BOTH	SFI+BK	DI+SW+PA		tr+st+ple		11	
38	3923	KANNAN	54/M	3.5 Y	cook	LLS	SFI	UL	hu+hp+ec	tr+st		13	
39	4472	RAJENDRAN	27/M	2 Y	agricul	RLS	SFI	sw	ou	com	sk	15	
40	6782	GOPINATH	24/M	6 Mn	sales	LSS	LA+BK	sw		spj+sfl		17	
41	11099	VIMALA	30/F	1 Mn	milk	LSS	SFI+BK	sw		spj		14	
42	15382	DAMODARAN	22/M	6 Y	manual	LLS	SFI+BK	sk	ld+hp+ec	tr+st		20	
43	15314	GNANASELVAN	28/M	5 Y	office	RLS	SFI	sw		tr+st	sk	2	
44	15793	AKBAR	36/M	3 Y	sales	RLS	SFI+AK+BK+AA	hae	ble	spl		15	
45	16233	SEKHAR	32/M	2 Y	manual	LLS	SPI	ul+sk	hp+ou	tr+st		29	
46	16241	SANTHI	40/F	2 Y	manual	LLS	SFI+BK	sw		tr+st+sfl		12	
47	19499	SANKAR	34/M	4 Mn	manual	RLS	SFI+BK	sw	it+ed	tr+st		14	
48	20822	GNANASEKHAR	21/M	7 Y	electric	LLS	SFI+BK+AK	it		tr+sfl		8	