DISSERTATION

ON

"EPIDEMIOLOGY, NATURAL HISTORY, TREATMENT AND OUTCOME OF PATIENTS PRESENTING WITH DEEP VEIN THROMBOSIS IN THANJAVUR MEDICAL COLLEGE"

M.S.DEGREE EXAMINATION
BRANCH – I
GENERAL SURGERY

THANJAVURMEDICALCOLLEGE AND HOSPITAL

THE TAMILNADU DR.M.G.R.MEDICALUNIVERSITY

CHENNAI

MAY – 2018

CERTIFICATE

This is to certify that dissertation entitled

"EPIDEMIOLOGY , NATURAL HISTORY, TREATMENT AND OUTCOME OF PATIENTS PRESENTING WITH DEEP VEIN THROMBOSIS IN THANJAVUR MEDICAL COLLEGE " is a bonafide record of work done by **Dr.HRIDYA VASUDEVAN,** in the Department of General Surgery, Thanjavur Medical College, Thanjavur, during her Post Graduate Course from 2015-2018 under the guidance and supervision of

PROF.DR. M.ELANGOVAN, M.S, F.I.C.S. This is submitted in partial fulfilment for the award of **M.S. DEGREE EXAMINATION- BRANCH I (GENERAL SURGERY)** to be held in MAY 2018 under the **Tamilnadu Dr. M.G.R. Medical University, Chennai.**

PROF AND UNIT CHIEF S I
Department of General surgery,
Thanjavur Medical College,
Thanjavur

PROFESSOR AND HEAD
Department of General Surgery,
Thanjavur Medical College,
Thanjavur.

DEAN
Thanjavur Medical College,
Thanjavur.

DECLARATION

I declare that this dissertation entitled **"EPIDEMIOLOGY, NATURAL HISTORY, TREATMENT AND OUTCOME OF PATIENTS PRESENTING WITH DEEP VEIN THROMBOSIS IN THANJAVUR MEDICAL COLLEGE"** is a record of work done by me in the department of General Surgery, Thanjavur medical college, Thanjavur, during my Post Graduate Course from 2015-2018 under the guidance and supervision of my unit chief **Prof. DR.M.ELANGOVAN, M.S, F.I.C.S.** It is submitted in partial fulfilment for the award of **M.S. DEGREE EXAMINATION- BRANCH I (GENERAL SURGERY)** to be held in MAY 2018 under the **Tamilnadu Dr.M.G.R. Medical University, Chennai.** This record of work has not been submitted previously by me for the award of any degree or diploma from any other university.

Date : **DR.HRIDYA VASUDEVAN**

Place: Thanjavur

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INTRODUCTION

Deep vein thrombosis (DVT) refers to the formation of one or more blood clots (a blood clot is also known as a "thrombus," while multiple clots are called "thrombi") in one of the body's large veins, most commonly in the lower limbs (e.g., lower leg or calf).

The clot(s) can cause partial or complete blocking of circulation in the vein, which in some patients leads to pain, swelling, tenderness, discoloration, or redness of the affected area, and skin that is warm to the touch. However, approximately half of all DVT episodes produce few, if any, symptoms. For some patients, DVT is an "acute" episode (that is, the symptoms go away once the disease is successfully treated), but roughly 30 percent of patients suffer additional symptoms, including leg pain and swelling, recurrent skin breakdown, and painful ulcers. In addition, individuals experiencing their first DVT remain at increased risk of subsequent episodes throughout the remainder of their lives.

The most serious complication that can arise from DVT is a pulmonary embolism (PE) which occurs in over one-third of DVT patients. A PE occurs when a portion of the blood clot breaks loose and travels in the bloodstream, first to the heart and then to the lungs, where it can partially or completely block a pulmonary artery or one of its branches. A PE is a serious, life-threatening

complication with signs and symptoms that include: shortness of breath, rapid heartbeat, sweating, and/or sharp chest pain (especially during deep breathing). Pulmonary embolism frequently causes sudden death, particularly when one or more of the vessels that supply the lungs are completely blocked by the clot. Those who survive generally do not have any lasting effects because the body's natural mechanisms tend to resorb (or "lyse") blood clots. However, in some instances, the blood clot in the lung fails to completely dissolve, leading to a chronic serious complication that can cause chronic shortness of breath and heart failure. DVT and PE are commonly grouped together and sometimes referred to as "venous thromboembolism" (VTE)[16]

AIMS AND OBJECTIVES

1. To study the Natural history of Acute and Chronic DVT.

2. To study the various treatment modalities in Acute DVT.

3. To study the outcome following treatment in Acute DVT.

REVIEW OF LITERATURE

HISTORY

Deep vein thrombosis (DVT) is a common disease. The first well-documented case of DVT was reported during the middle Ages: in 1271, Raoul developed a unilateral oedema in the ankle, which then extended to the leg. The number of

reported DVT cases steadily increased thereafter, particularly in pregnant and postpartum women.

During the first half of the 20th century, well before the discovery of anticoagulants, many therapeutic approaches were used, and arose from the pathologic hypotheses that prevailed at their time.

Despite the development of anticoagulants, and the fact that they were thought to dramatically decrease DVT mortality, numerous complementary treatments have also been developed during the last 50 years: they include vena cava clips and surgical thrombectomy, and are intended to decrease mortality or to prevent late complications. Most of these treatments have now been abandoned, or even forgotten. The first description of a case truly compatible with a DVT first appears during the Middle age. In the manuscript of Guillaume de Saint Pathus entitled 'La vie et les miracles de Saint Louis', it is reported that, in 1271, Raoul, a 20-year-old Norman cobbler suffered unilateral pain and swelling of the right calf that subsequently extended up to the thigh Thus, this first reported case of effective treatment of DVT might not be the most reproducible. Thus, although venous thrombosis is a frequent disease, it appears that cases clearly compatible with the diagnosis of DVT were reported before the description of the case of Raoul[17]. After this first unquestionable description by Guillaume de Saint Pathus, the number of reported cases of DVT increased rapidly and the first pathologic hypotheses arose, leading to the first treatment attempts. During

the Renaissance, physicians hypothesized that pregnancy-related DVT, which was the leading, or even only, cause of reported DVT at that time, was the consequence of retention of ‘evil humors’. It was also thought that postpartum DVT was caused by retention of unconsumed milk in the legs (‘milk leg’). Thus, in the late 1700s, breast-feeding was encouraged to prevent DVT. Of course, the most frequent and popular method among physicians to discharge evil humors during the 17th century was bloodletting .This technique was used to treat DVT and many other diseases until the end of the 19th century. In 1676, Wiseman suggested that DVT was the consequence of an alteration of blood, and then, in 1793, Hunter hypothesized that it was an occlusion of the vein by blood clots. In 1784, well before Virchow demonstrated the relationship between DVT and fatal PE (1856), Hunter had performed venous ligations above thrombosis, to prevent extension of clots. In the absence of any other truly effective treatment for preventing fatal PE, this technique became more widely used at the end of the 19th century. It was assumed to be of ‘immense value in reducing the incidence of PE. The ligation could be performed at the femoral, common femoral, iliac and inferior vena cava (IVC) levels in cases of proximal thrombosis, and more rarely in cases of distal thrombosis, although this latter therapeutic intervention remained controversial. This surgical treatment was still widely used until the mid-20th century in association with, or instead of, anticoagulants. For fear of thrombus migration, strict bed rest was prescribed, and constituted, at least from the end of the 19th century, the

cornerstone of DVT treatment. Thus, in cases of DVT, the patient's lower limbs were set in iron splints to prevent any movement. Special, reclining, orthopaedic beds were also used to favour venous return .However, during the 19th century, the most commonly accepted underlying mechanism for DVT was the inflammation of the vein wall provoked by and/or provoking an infectious phenomenon. This was consistent with the observation that DVT is, in many cases, associated with fever, and frequently occurred postpartum, after – septic – surgical procedures, or during bed rest for an infectious disease. Consequently, the treatments prescribed involved anti-inflammatory medication and the prevention and treatment of infection. Overall, prior to the 1930s, before the introduction of anticoagulants, the most common treatment for DVT mainly relied on:

(i) Bed rest to fix the thrombus in place;

(ii) Elevation of the extremity involved to favour venous return.

(iii) Application of heat with warm compresses to reduce vasospasm and to increase collateral circulation.

Because major risk factors for DVT had already been identified, most currently used thromboprophylactic measures were already known and applied in hospitalized cases.

By the middle of the 19th century, the major pathologic mechanisms of venous thrombosis had been discovered. They were first summarized in the famous Virchow's triad (1856), theorized by Andral in 1831 and which Virchow probably never described.

However, it was only towards the 1920s that a consensus appeared regarding the three factors contributing to thrombosis: stasis, vessel wall alteration, and hypercoagulability. During this period, a number of therapeutic breakthroughs, most of them discovered by accident, revolutionized DVT treatment

ANATOMY OF VEINS OF LOWER LIMB

The lower limb consists of two main types of veins[4]

a) Superficial vein

b) Deep vein

c) Perforators

SUPERFICIAL VEIN:-

They include great and small saphenous veins and their tributaries. They are located within the subcutaneous tissue of superficial facia, on the surface of deep fascia. They are thick walled because of the presence of smooth muscle and some fibrous and elastic tissues in their walls. Valves are numerous in the distal parts of these veins than their proximal parts. A Large Proportion of their blood is drained into deep veins through perforating veins.

DEEP VEIN:-

The deep veins accompany the major arteries and their branches and are usually paired. They contain valves to prevent reflux of blood distally.

Deep veins are,

- Medial & Lateral plantar
- Dorsalis pedis
- Anterior &Posterior Tibial
- Peroneal
- Popliteal and femoral veins and their tributaries.

They are surrounded by powerful surrounding muscles. The valves are numerous in the deep veins than in the superficial veins. They are more efficient channels than superficial veins because of the driving force of muscular contraction.

PERFORATORS:-

The superficial and deep veins are connected by perforator veins. They have valves which permit only unidirectional flow of blood from superficial to deep veins. They are mentioned below.

Indirect Perforating veins:-

These veins connect the superficial with the deep veins through the muscular veins.

Direct Perforating veins:-

These will connect the superficial veins directly to deep veins.There are about five perforators along the great saphenous vein and one along the small saphenous vein.

The small direct perforating veins are follows

1. In the Thigh:- The Adductor canal Perforator connects the great saphenous vein with the femoral vein in the lower part of the adductor canal.

2. Below the Knee:-One Perforator connects the great saphenous vein or the posterior arch veins with the posterior tibial vein.

3. In the Leg:-A lateral perforator is present at the junction of the middle and lower thirds of the leg.it connects the small saphenous vein or one of its tributaries with peroneal vein.

Medially there are three perforators which connect the posterior arch vein with the posterior tibial vein.

a) The upper medical perforator lies at the junction of the middle and lower thirds of the leg.

b) The Middle medical perforator lies above the medical malleolous.

c) The Lower medial perforator lies in the posteroinferior to the medial malleolus.

Factors helping the Venous return:-

General factors: _

1. Negative intrathoracic Pressure, which is made more negative during inspiration

2. Arterial pressure and overflow from capillary bed.

3. Compression of veins accompanying arteries by arterial pulsation.

4. The presence of valves which support and divide the long column of blood in to shorter columns. These also maintain a unidirectional flow.

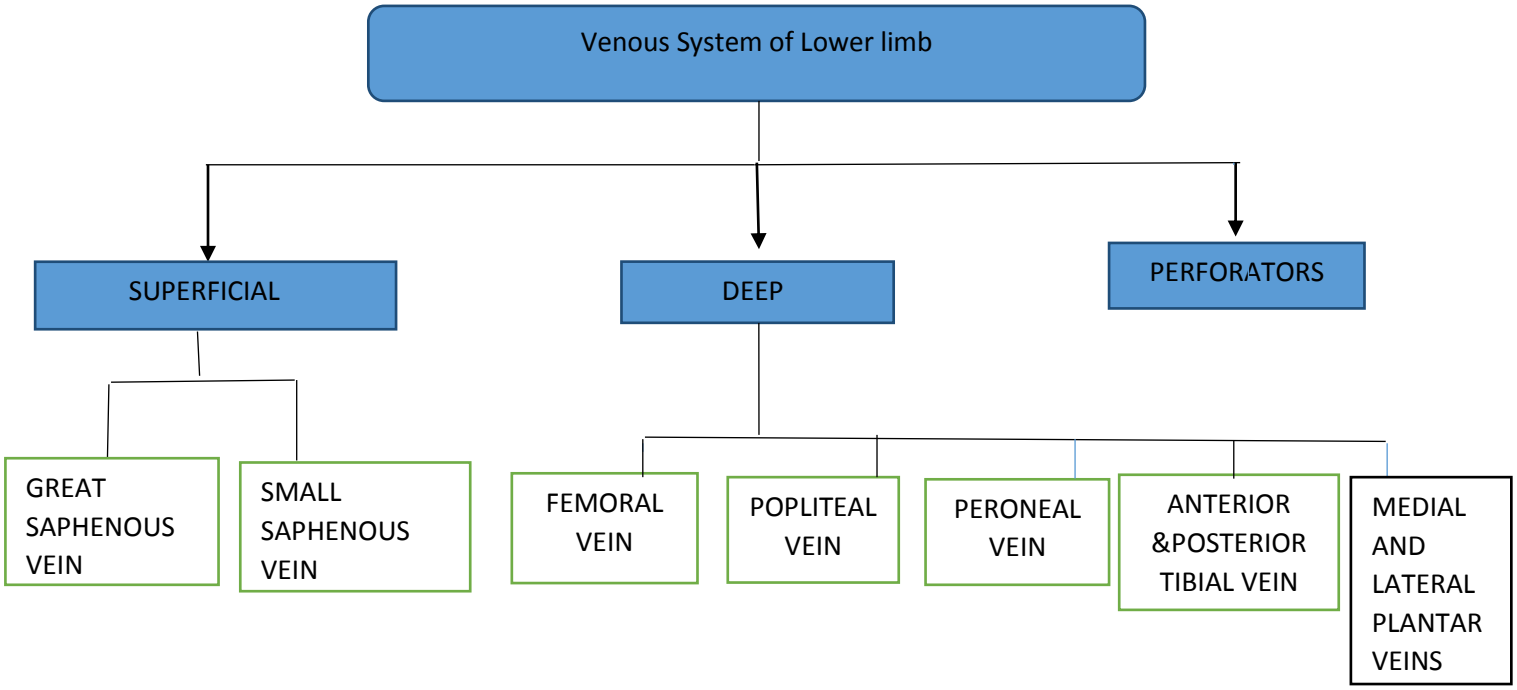
Local factors:-

These are Venous, Muscular, and Fascial

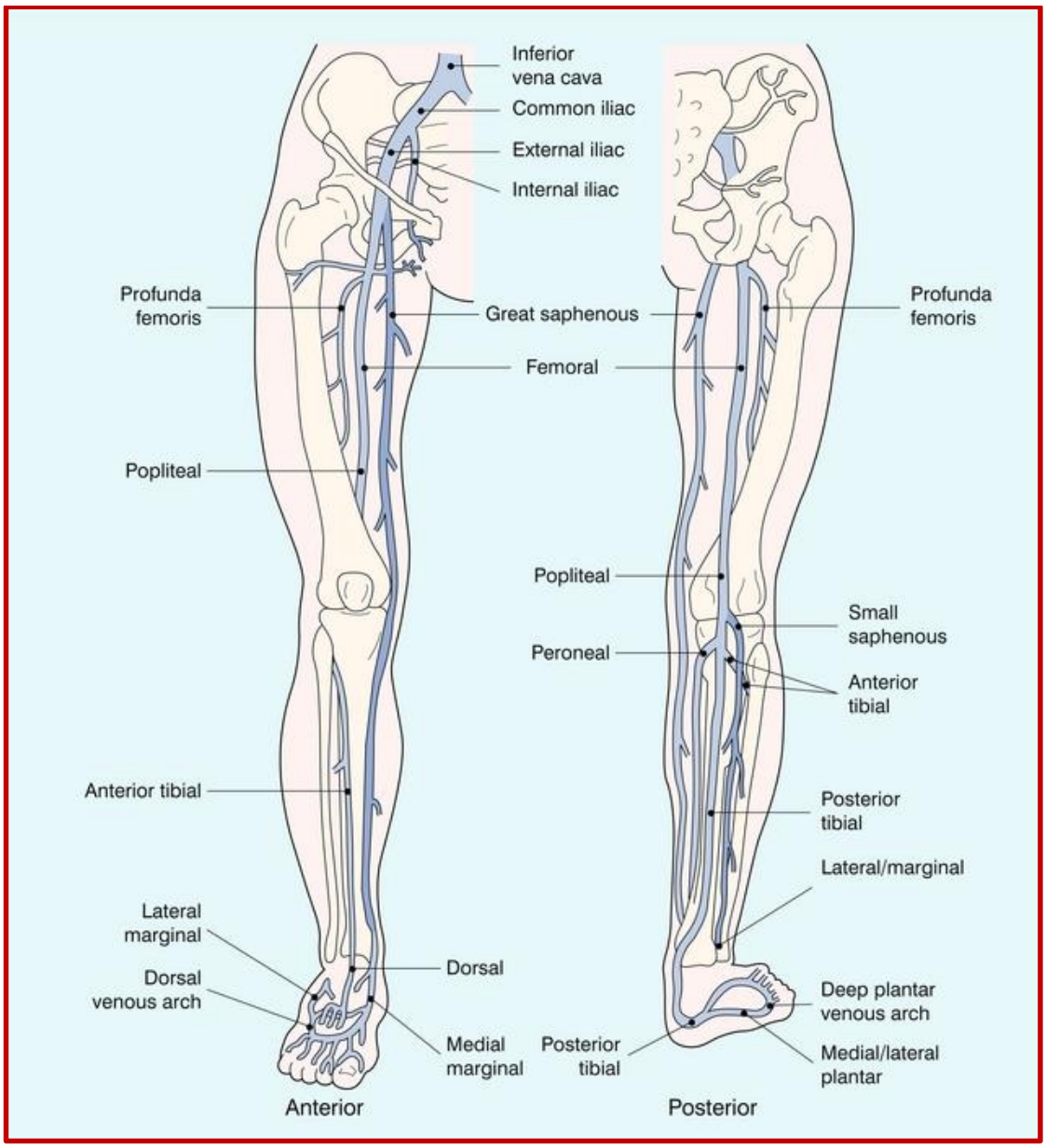
Venous:-The veins of the lower limb are more muscular than the veins of any other part of the body. They have greater number of valves. Superficial veins are connected to deep veins by perforators.

Muscular:- When the limb is active muscular contraction compress the deep veins and drives the blood in them upwards.

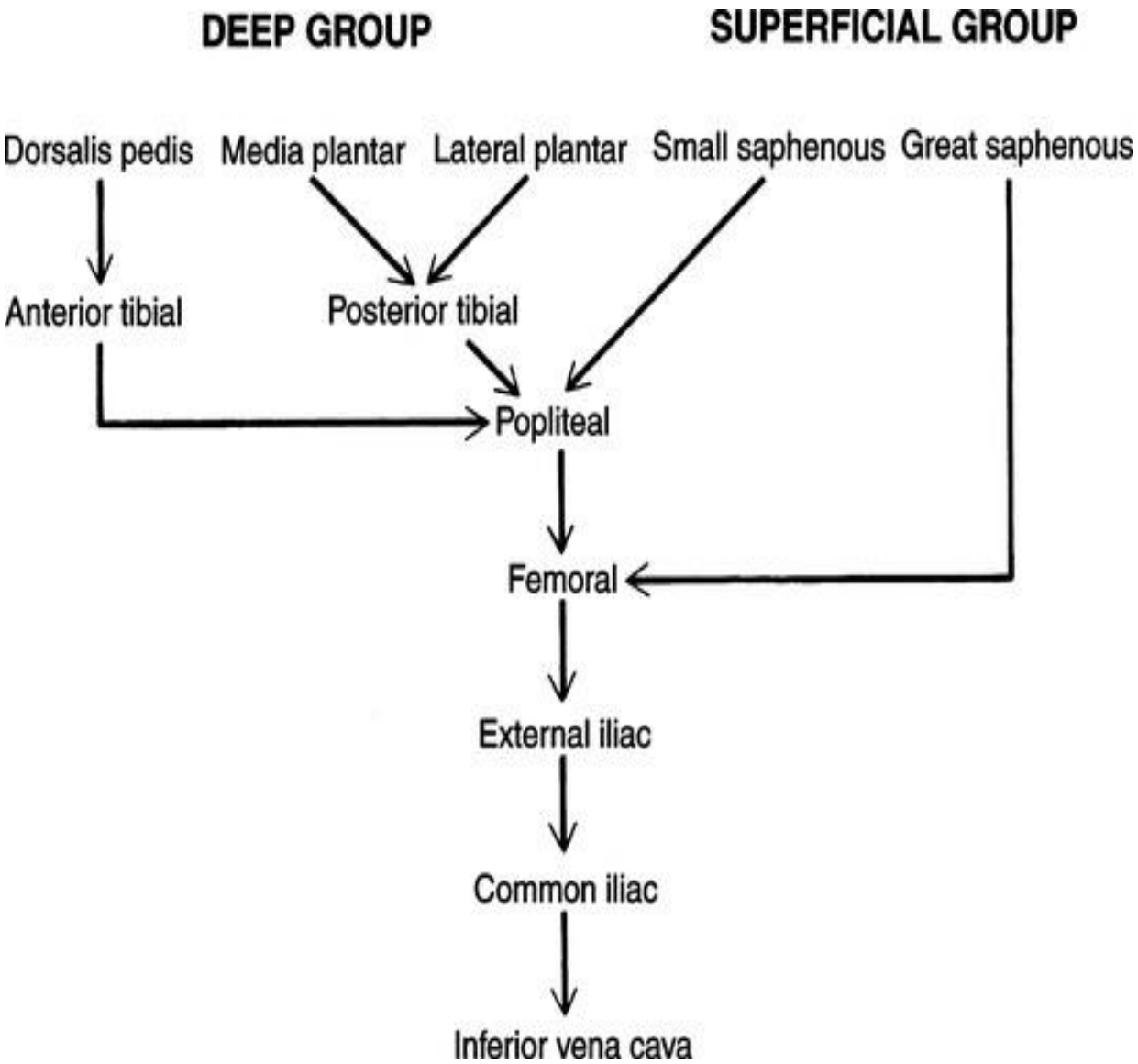
Fascial:-The tight sleeve of deep fascia makes muscular compression of the veins much more effective by limiting outward bulging of the muscles.


Venous System of Lower limb
SUPERFICIAL
DEEP
PERFORATORS
GREAT SAPHENOUS VEIN
SMALL SAPHENOUS VEIN
FEMORAL VEIN
POPLITEAL VEIN
PERONEAL VEIN
ANTERIOR &POSTERIOR TIBIAL VEIN
MEDIAL AND LATERAL PLANTAR VEINS

VENOSUS SYSTEM OF LOWERLIMB



VENOUS DRAINAGE OF LOWERLIMB



EPIDEMIOLOGY

INCIDENCE:

Incidence of lower extremity DVT is highly dependent on the population studied, their underlying risk factors and the means by which DVT is documented. True estimates of the incidence of DVT are limited by the few population based studies, the clinically silent nature of most thrombosis and the need for objective documentation of the diagnosis. Even the interpretation of methodologically sound studies is complicated by inconsistent inclusion of DVT and or pulmonary embolism, differing exclusion or inclusion of recurring DVT and variable age ranges, community based studies of hospitalised patients have suggested firs episode of DVT to have a crude annual incidence of 56/1 Lakh.[2]

AGE:

Age has been most consistently associated with an increased risk of DVT. The incidence of DVT increases exponentially with age. This increased risk appears related to several age- associated factors, including decreased mobility, an increased number of major thrombotic risk factors, age- related hypercoagulability, and changes in the venous system.[2]

GENDER:

Gender differences in the incidence of DVT have been variable and may be related to other risk factors. Although Coon and associates found higher frequency if DVT in young women, one half of Thromboembolic events in women less than 40 years old were associated with pregnancy. Some have noted no significant differences in incidence between men and women. However, incidence rates in young populations are higher in women during the child bearing years and may be higher in men over 45-60 years of age[1,5,7]

AETIOPATHOGENESIS

Aetiology:-

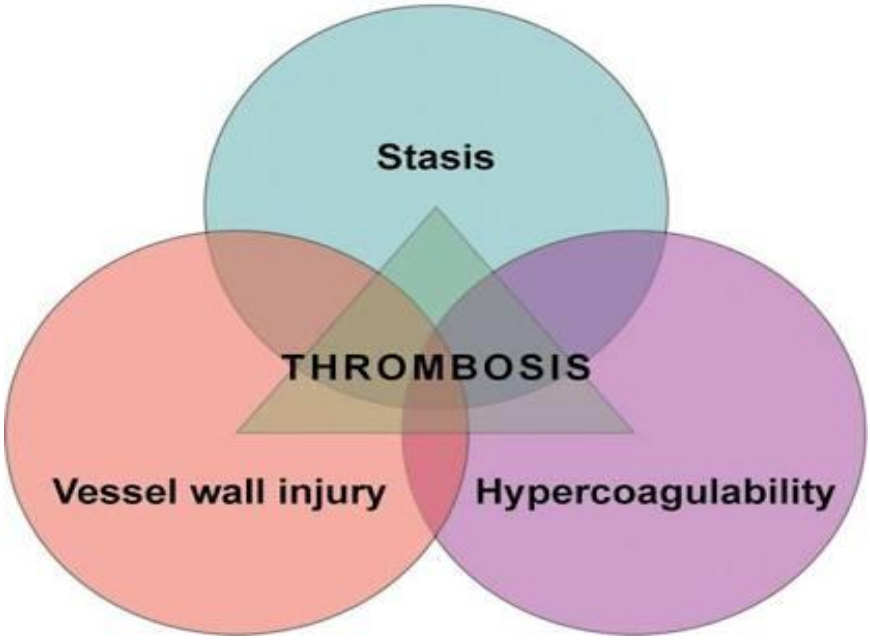
The three factors described by Virchow over a century ago are still relevant in the development of venous thrombosis. They are

a) Changes in the vessel wall (endothelial damage)

b) Stasis, which is diminished blood flow through the veins[3]

c) Coagulability of blood (thrombophilia)

These are together known as Virchow triad.



The most important factor is hospital admission for the treatment of a medical or surgical condition. Injury, especially fractures of the lower limb and pelvis, pregnancy and the oral contraceptive pill are well recognised predisposing factors.

Endothelial damage is now known to be critically important, the interaction of the endothelium with the inflammatory cells or previous deep vein damage, renders the endothelial surface hypercoagulable and less fibrinolytic. Biological injury to endothelium may have very important role in the origin DVT. Venus endothelium is normally antithrombotic, producing PGI 2, Thrombomodulin, TPA and Glycosaminoglycan CO factors of antithrombin. Under conditions favouring thrombosis the endothelium becomes prothrombotic producing tissue factor Von Willebrand Factor and fibronectin. Leukocytes may be a key mediator of both endothelial injury and hypercoagulability, with the early

phases of thrombosis marked by increases in permeability followed by leukocyte adhesion migration and endothelial disruption.

Stasis is a predisposing factor seen in many conditions, especially in the postoperative period, in patients with heart failure and in those with atrial ischemia. Stasis alone is inadequate stimulus in the absence of low levels of activated coagulation factors. Although stasis may facilitate endothelial leukocyte adhesion and cause endothelial hypoxia leading to a pro coagulant state, its most important role may be in permitting the accumulation of Activated coagulation factors in areas prone to thrombosis.

A number of conditions are associated with hypercoagulability (Thrombophilia).deficiencies of antithrombin. Activated protein C &S have all shown to predispose to venous thrombosis in young patients. A thrombophilic cause should be sought in any patient presenting with an episode of venous thrombosis who gives a family history of DVT or in whom there is no other predisposing factor.

All though the development of DVT is probably multifactorial, immobility (and hence stasis) remains one of the most important factors. DVT is recognised as a complication of long-haul flights and other forms of travel.

RISK FACTORS

PATIENT FACTORS:-

a) Age

b) Obesity

c) Varicose veins

d) Immobility

e) Pregnancy

f) Puerperium

g) High dose oestrogen therapy

h) Previous DVT or Pulmonary embolism

i) High dose oestrogen therapy

j) Thrombophilia (Deficiency of Anti thrombin III, Protein C or S, factor V Leiden mutation)

DISEASE OR SURGICAL PROCEDURE:

a) Trauma or surgery, especially of pelvis, hip and lower limb.

b) Malignancy, especially pelvis, hip and lower limb.

c) Heart failure

d) Recent MI

e) Paralysis of lower limb

f) Inflammatory bowel disease

g) Nephrotic syndrome

h) Polycythaemia

i) Paraproteinaemia

j) Paroxysmal nocturnal haemoglobinuria

k) Homocystinaemia

PATHOLOGY:-

A thrombus often develop in the soleal veins of the calf, initially as a platelet aggregate. Subsequently, fibrin and red cells form a mesh until the lumen of the vein wall occludes. The coralline thrombus then progress as a propagated loose red fibrin clot containing many red cells. This is likely to extend up to the next large venous branch and it is possible for the clot to break off and embolise to the lung as a pulmonary embolism. In this situation the embolus arising from the lower leg veins become detached, passes through the large veins of the limb and vena cava, through the right heart and lodges in the pulmonary arteries. This may totally occlude perfusion to all or part of one or both lungs (pulmonary embolism).Acute Right heart obstruction may lead to sudden collapse and death. Lung infarction is

rare as the lung has a dual blood supply(Bronchial and Pulmonary arteries).Moderate sized emboli can cause Pyramidal-shaped infarcts.

CLINICAL PRESENTATION AND NATURAL HISTORY OF ACUTE DVT:-

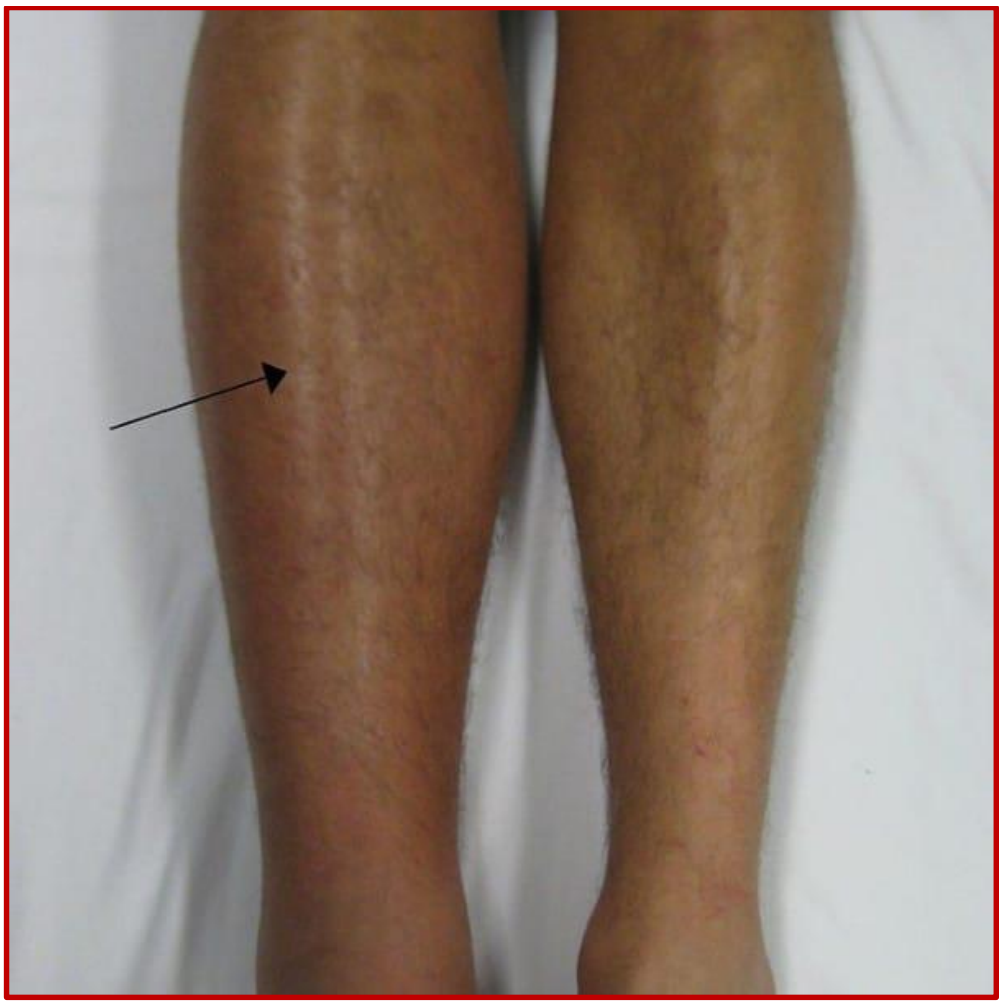
Introduction:-

The spectrum of venous thromboembolism (VTE) includes both deep venous thrombosis (DVT) and pulmonary embolism (PE).Many episodes are asymptomatic and the symptoms of acute DVT includes oedema, pain and erythema are non –specific. At least three quarters of patients having lower extremity symptoms consistent with DVT have non thrombotic cause of their symptoms. Confirmatory test is therefore always required both to ensure appropriate treatment of those with confirmed DVT and to prevent the complications of inappropriate anticoagulation in those with other disorders. The treatment of DVT is aimed at preventing its complications-PE, recurrent DVT, Post-thrombotic syndrome and Death.

Clinical Presentation of Acute DVT:-

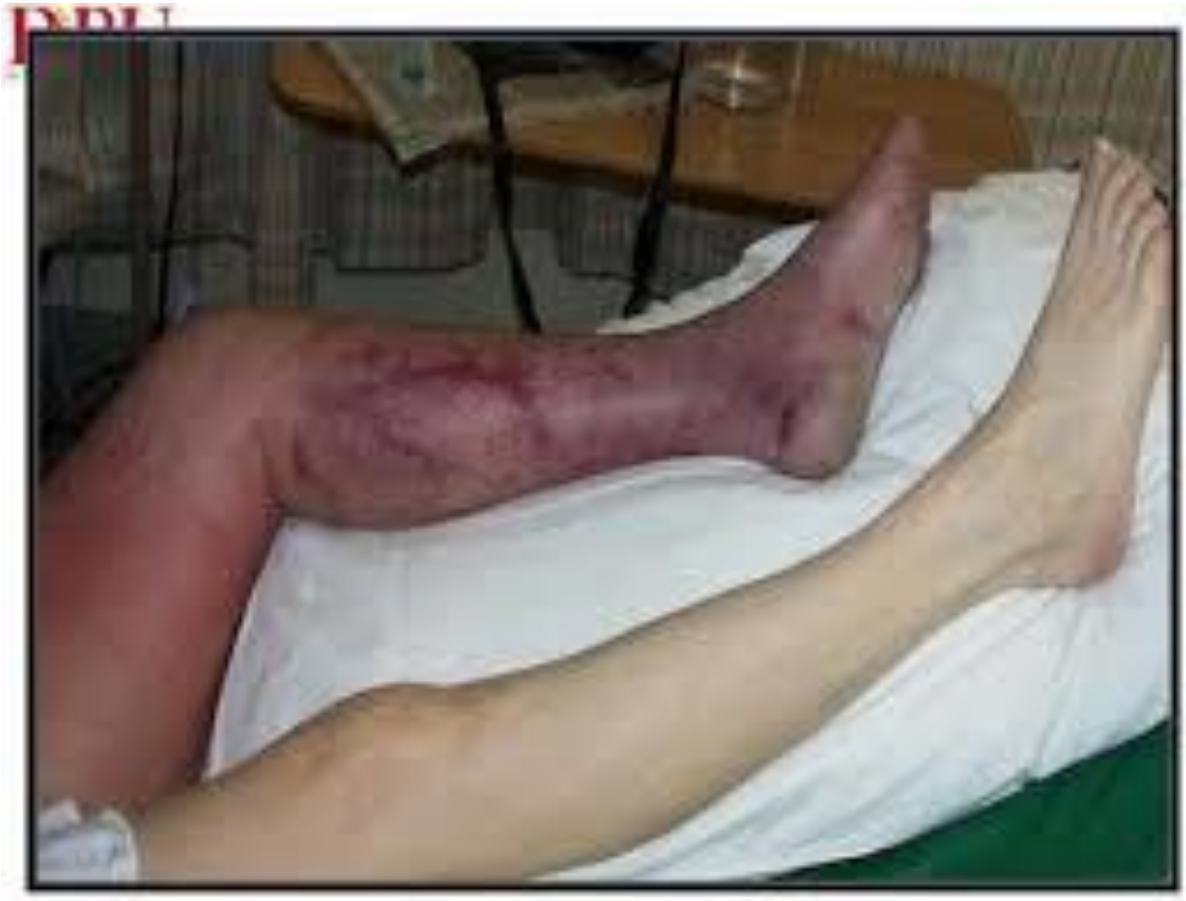
Clinical presentation of acute DVT varies with the anatomic distribution extend and degree of occlusion of thrombus. Symptoms may accordingly range from their absence to massive swelling and

cyanosis with impending venous gangrene (phlegmasia cerulea dolens)[6,8].



Signs and symptoms include pain oedema erythema tenderness, fever prominence superficial veins pain with passive dorsiflexion of foot (Homans sign) and peripheral cyanosis. Phlegmasia cerulea dolens is characterised by the triad of massive swelling cyanosis and pain, it is the most severe form of DVT and results from near complete thrombosis of an extremities and venous outflow. Advanced cases it is

marked by severe venous hypertension with collateral and micro vascular thrombosis leading to venous gangrene.



Three patterns of thrombosis are commonly recognised they are

1) Isolated calf vein thrombosis

2) Femoro popliteal

3) Illeo femoral

Calf veins are the most common site of origin, although 40% of the proximal thrombi arise primarily in the femoral or iliac veins presumably in the regions behind the valves.

Investigations:-

- D dimer
- Doppler
- Duplex
- MRI Venography
- Impedance Plethysmography
- Contrast Venography

D –dimer [9]

Elevated D- dimer is not necessarily a risk factor causing VTE , but it should be used and interpreted as a marker of hypercoagulability. D-dimer is formed when fibrin is proteolysed by plasmin. The presence of elevated levels of D-dimer in the circulation signifies that endogenous fibrinolysis of a venous thrombus has yielded cross-linked fibrin. The degree of D-dimer elevation with VTE may depend on the extent of disease , the duration of symptoms , and the use of anticoagulants , with lower D-dimer levels associated with less extensive disease , longer duration of symptoms, and anticoagulant

use. Using D-dimer to preselect patients likely to have DVT has gained considerable interest in an effort to reduce costs and expedite patient work-up. The D-dimer assays currently available are turbidimetry, ELISA, latex particle agglutination, fluorescence immunoassay and immunofiltration tests. Each assay has a corresponding normal reference range which is typically not interchangeable. There are situations where D-dimer assay may be falsely positive. These situations include pregnancy, malignancy, recent postoperative state and total bilirubin greater than 2mg/dl. Further confounding factors may include the age of the clot as significant declines in the D-dimer level may occur with time, position of the clot and heparin use. Despite its limitations, D-dimer is a useful tool to rule out DVT as long as the threshold is set low enough to keep the sensitivity high.

DUPLEX[18,27]- Duplex ultrasound combines compression using real-time B-mode ultrasound with Doppler venous outflow detection .Its lack of radiation, portability, non-invasiveness and cost-effectiveness has made it superior to contrast venography. It also has the ability to distinguish non-vascular pathology such as inguinal adenopathy, Baker's cyst, abscesses and hematomas. The sensitivity of

compression sonography is high for proximal DVT and lower for non-occluding or isolated calf vein thrombosis. The pitfalls of venous duplex imaging include misidentification of veins ,duplicated vein systems, systemic illness or hypovolemia decreasing venous distension , suboptimal imaging in obese or oedematous patients , or areas not amenable to compression such as the iliac veins and adductor canal.

Normal venous flow assessment:-

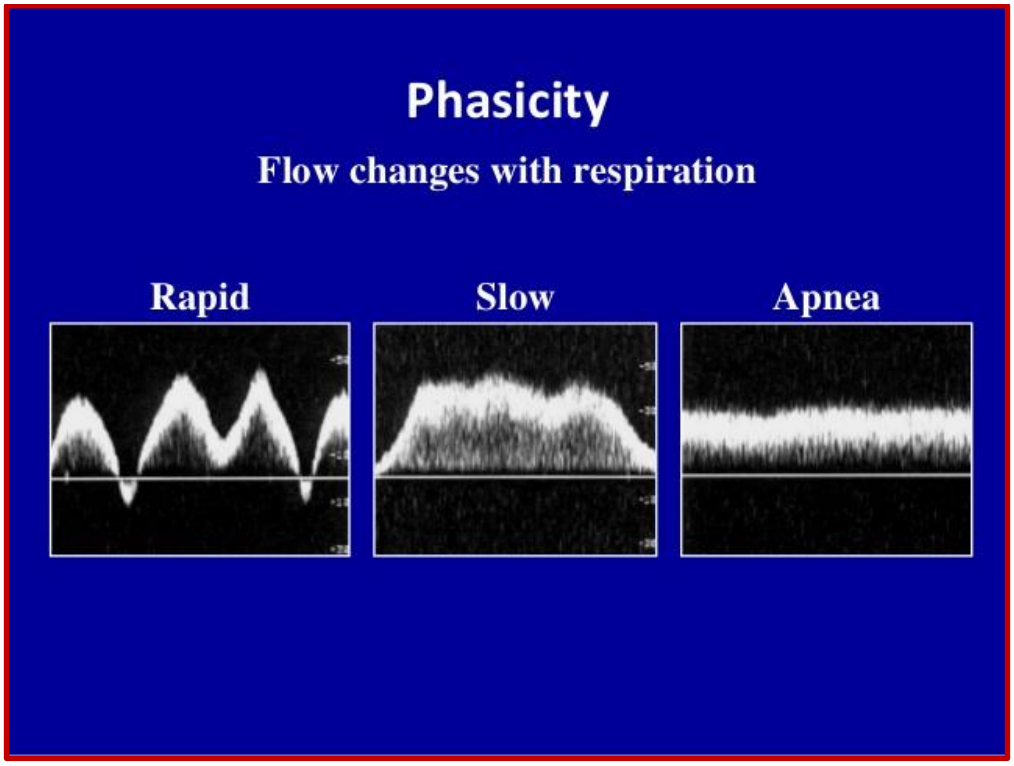
Spontaneity – Spontaneous flow without augmentation

Phasicity – flow changes with respiration

Compression – Transverse plane

Augmentation – compression distal to site of examination, patency below site of examination

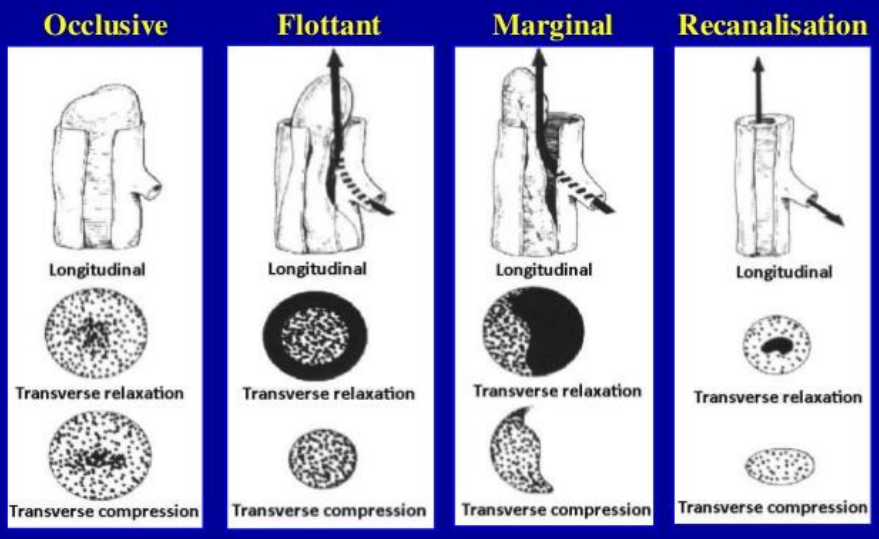
Valsalva –deep breath, strain, while holding the breath patency of abdominal and pelvic veins


Phasicity
Flow changes with respiration
Rapid
Slow
Apnea

US diagnostic criteria of DVT

Direct signs	Indirect signs
• Intramural thrombus	• Loss of phasicity:
• Incompressibility +++	Proximal thrombosis
• ↑ in vein diameter	Venous compression
• No flow in pulsed Doppler	• Loss of augmentation:
• No flow in color Doppler	Distal thrombosis

Types of thrombus



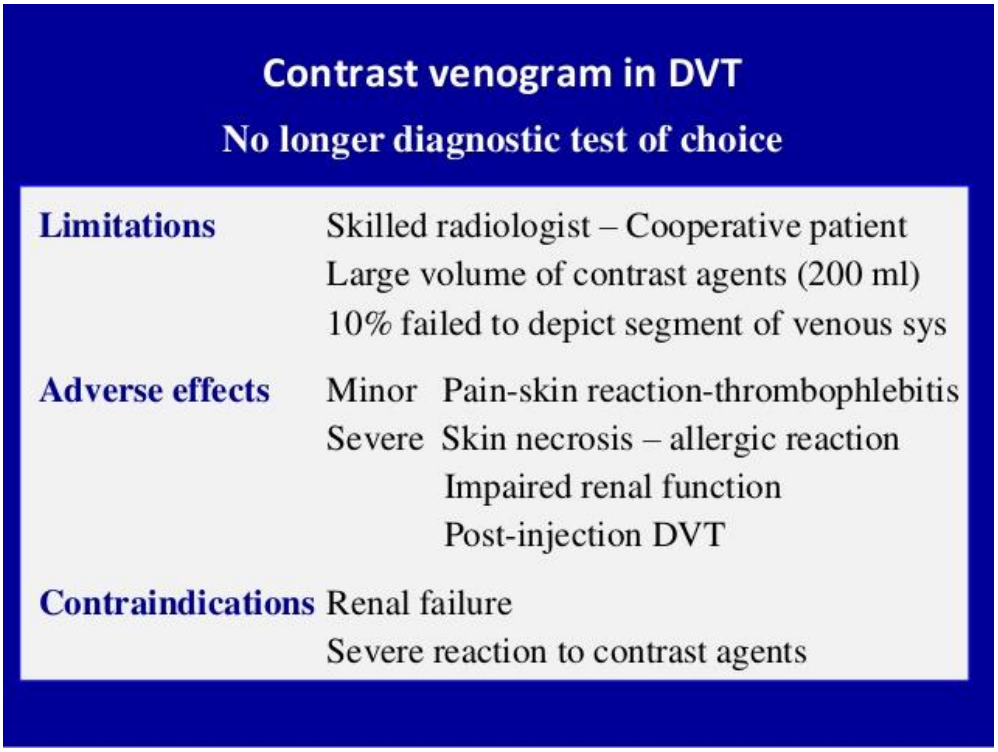
MAGNETIC RESONANCE VENOGRAPHY[20]

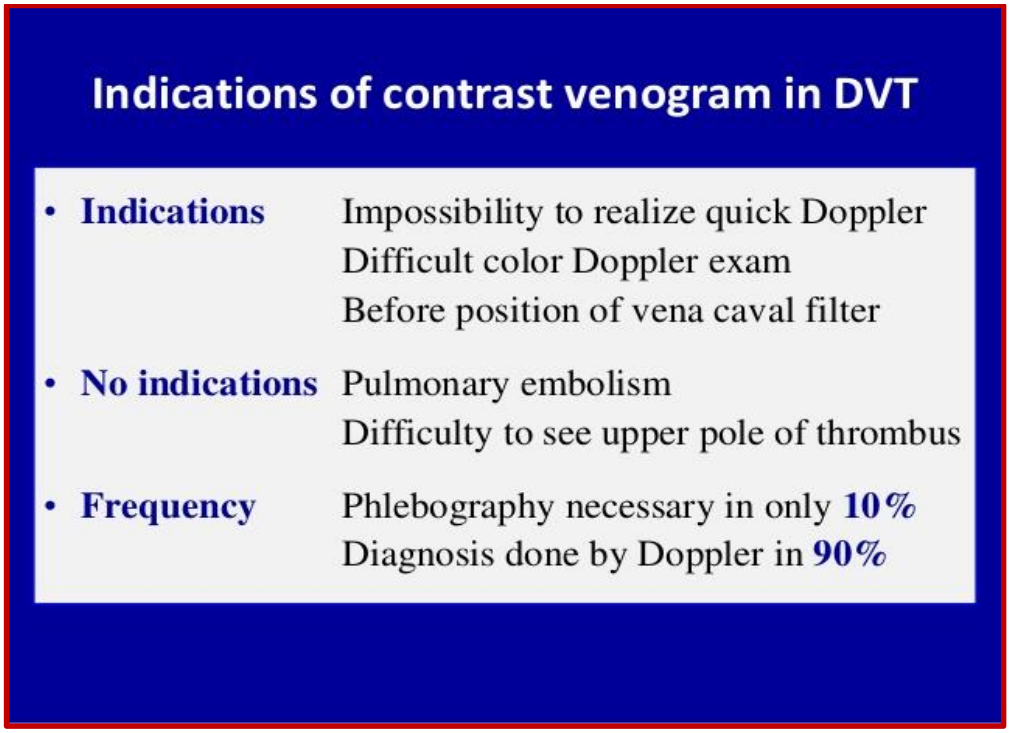
MRV has gained momentum in recent years for the detection of DVT. In addition to been less invasive than contrast venography MRV overcomes some of the limitation of Duplex and Impedance Plethysmography. Since MRV directly visualises the thrombus even non flow limiting thrombi should be detectable unlike with impedance Plethysmography. MRV should also be able to detect thrombus proximal to the inguinal ligament, an area which has been problematic for duplex in the past. MRV results are also independent of the technologists experience and availability. Its limitation includes high cost, patients with implants and renal insufficiency since gadolinium is associated with nephrogenic systemic fibrosis.

CONTRAST VENOGRAPHY:-

Contrast venography has by default been long hailed as the gold standard for detection of symptomatic DVT.As of late, its current role in diagnosis of DVT has been largely relegated to one of historical interest[25]. The study is limited in its practicality by both the availability of highly sensitive non-invasive studies and by its own disadvantages, including the risk of phlebitis, IV contrast load with associated risk of nephrotoxicity and allergic reaction, Increased cost

and the need for adequate IV access. Of the available methods of performing venography 2 techniques have been emerged as dominant. First technique described by rabenow-paulin, involves spot films whereas the second technique involves long-leg films. Rarely is CV a first line study, a role for CV is still exist when non-invasive studies are unavailable , non-diagnostic or in the presence of a clinical condition known to produce false results(e.g. –D dimer levels post operatively or during pregnancy, compression of the iliac veins in pregnant or recently postpartum women on a MRV study etc.



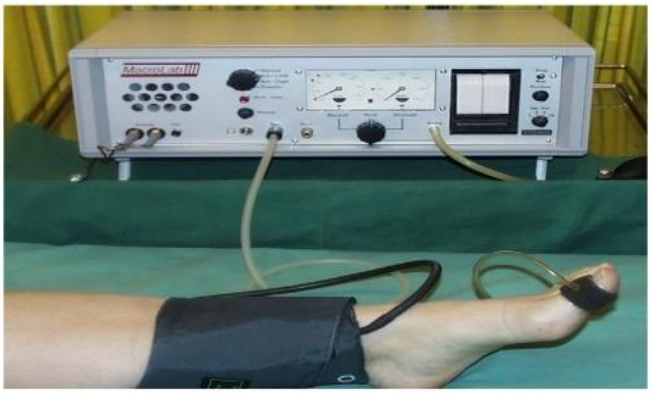


IMPEDENCE PLETHYSMOGRAPHY (IPG):[22]-

IPG is based upon the physiological principle that the impedance between 2 points on the skin of an extremity will decrease as the volume of blood contained in the extremity increases. The technique examines great at which venous outflow occurs, thereby deducing the presence or absence of venous outflow obstruction. The presence of DVT in the major vessels of the lower extremity including the popliteal vein and proximally, should reduce the rate of venous outflow and subsequently affect the tracing. In the instance of non-flow limiting thrombi, the study will be negative. Its limitation include inability to detect DVT distal to popliteal vein.

PLETHYSMOGRAPHY

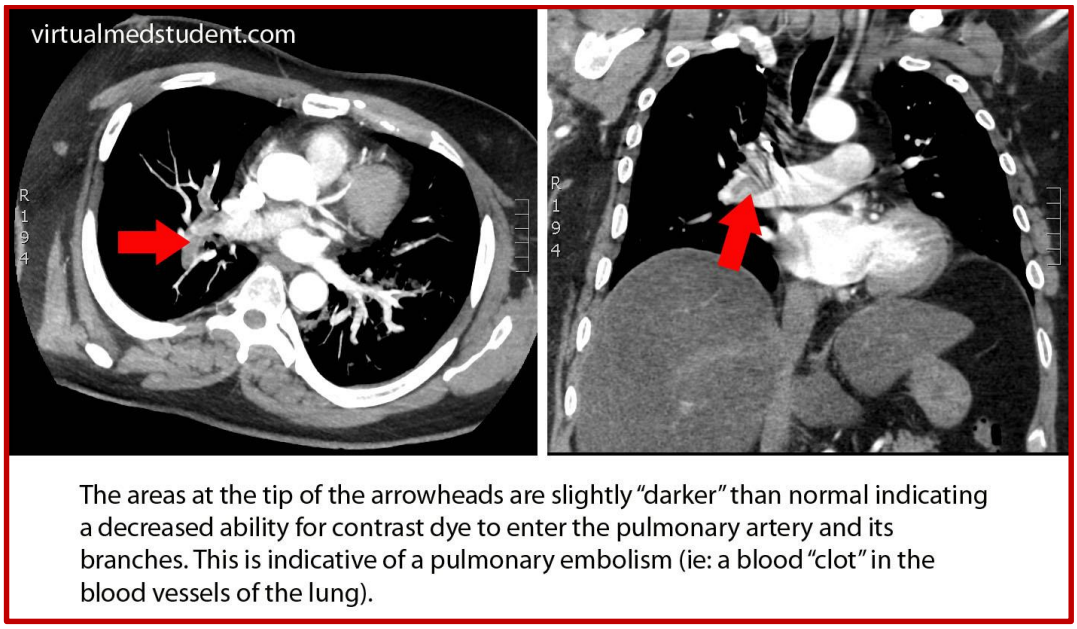
- Plethysmography measures change in lower extremity volume in response to certain stimuli.



COMPLICATION;-

1) **Pulmonary embolism**[11]: - The potentially life threatening consequences of PE make it the most important short term complication of acute DVT. Symptomatic PE accompanies approximately in 10% of DVTs and hospital discharge data suggests 23 per 1 lakh population. Most common signs of PE are Tachypnoea and tachycardia. Less common signs include syncope, Hypoxemia and sudden hypotension. The use of a validated pretest probability score is recommended for as the first step in the work up of patients suspected to have PE.

CT SHOWING PULMONARY EMBOLISM



The areas at the tip of the arrowheads are slightly "darker" than normal indicating a decreased ability for contrast dye to enter the pulmonary artery and its branches. This is indicative of a pulmonary embolism (ie: a blood "clot" in the blood vessels of the lung).

RADIOLOGICAL SIGNS IN PULMONARY EMBOLISM

- Fleischner sign – enlarged pulmonary artery
- Hampton hump- peripheral wedge air space opacity and implies lung infarction
- Westermark sign – regional oligaemia
- Palla sign- enlarged right descending pulmonary artery
- Chang sign- dilated right pulmonary artery with sudden cut off

WELLS CLINICAL DECISION RULE:-

Clinical signs and symptoms of DVT, minimal swelling of the leg and pain on palpitation of the deep leg veins	3.0
Pulmonary embolism more likely than an alternative diagnosis	3.0
Heart beat frequency >100 beats per minute	1.5
Recent immobilization or surgery within < 4 weeks	1.5
Documented history of DVT and or PE	1.5
Haemoptysis	1.0
Recent history of malignancy <6 months (treatment or palliative treatment)	1.0

Clinical score for Pulmonary embolism:-

Low <_ 2

Moderate 2.0-6.0

High >_6

POST THROMBOTIC SYNDROME

Affects 43% patients with DVT within 2 years.

Higher incidence in those with proximal (iliofemoral) DVT.

Characterised by painful oedematous legs, paraesthesia, cramps, pruiritis, hyperpigmentation, varicosities, Ulceration and recurrent infection.[20]

Inflammation is thought to play a role as well as damage to venous valves from the thrombus itself. Valvular incompetence combined with persistent venous obstruction from thrombus increases the pressure in veins and capillaries. Venous hypertension induces rupture of small superficial veins, subcutaneous haemorrhage, and an increase in vascular permeability.

RISK FACTORS-Age >65 , proximal DVT , second DVT in same leg as first DVT , persistent DVT symptoms within one month of DVT diagnosis, obesity , poor quality of anticoagulation control during the first 3 months of treatment.

PREVENTION – early treatment of acute and recurrent DVTTREATMENT – ambulation, compression stockings, anticoagulants, and catheter directed thrombolysis.

RECURRENT VENOUS THROMBOSIS

Recurrent Thrombotic events compete with recanalization early after an acute DVT. Most clinicical studies have included both symptomatic recurrent DVT and PE, with rates depending on treatment, proximal or distal location of Thrombus and duration of follow up.

Fortunately standard anticoagulation is very effective in preventing recurrent VTE while patients are being treated. The risk of recurrence is highly related to the underlying Thrombotic risk factors.

Recurrent VTE in the setting of Thrombosis isolated to the calf veins requires special consideration. Limited data suggest that isolated calf vein Thrombosis is

associated with less extensive activation of coagulation than proximal venous Thrombosis, perhaps implying some difference in Pathophysiology[10].

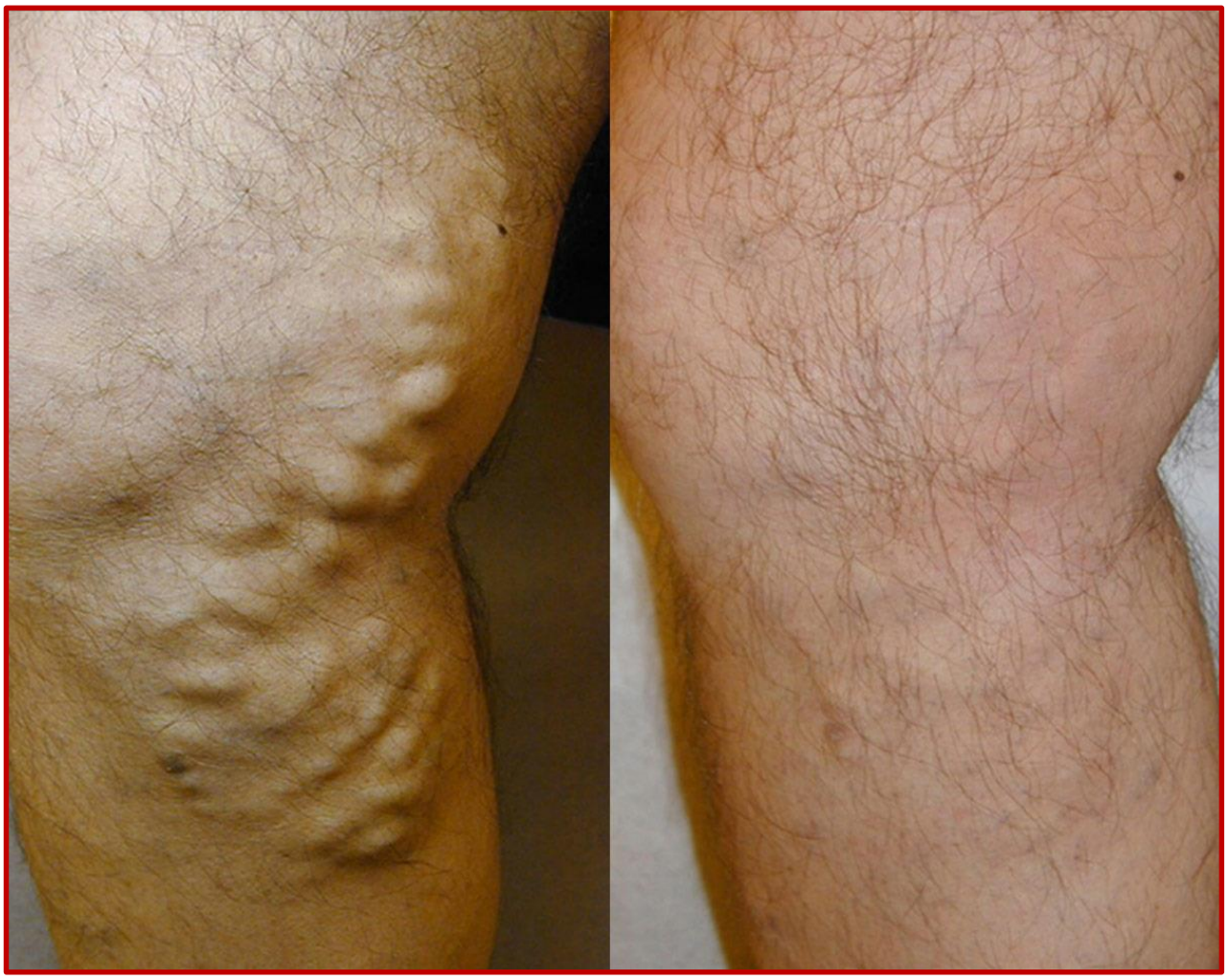
At least two types of calf vein Thrombosis may be differentiated – those with involvement of the paired posterior tibial and peroneal venae commitantes and those isolated to the veins draining the gastrocnemial and soleal muscles and their natural history may be different.

Recognised Thrombophilic states, particularly the factor 5 Leiden mutation, lupus anticoagulant and Homocystinaemia have been associated with recurrent Thrombo embolic events.

Recanalization and recurrent Thrombosis are related and failure of recanalization is now recognised as an independent predictor of recurrent DVT.

The presence of ongoing hypercoagulability is in fact, perhaps a better predictor of the risk of recurrent VTE.

VARICOSE VEIN



Varicose vein occurs as a complication of Deep vein Thrombosis due to destruction of valves by thrombus.

TREATMENT:-

A) MEDICAL :-

- HEPARIN
- LMWH

- ORAL ANTICOAGULANTS

B) ENDOVASCULAR INTERVENTION

C) SURGICAL THROMBECTOMY

The objectives of treatment in patients with VTE are

a) To prevent death from pulmonary embolism

b) To prevent recurrent VTE

c) To prevent post-phlebitic syndrome

The mainstay of medical therapy has been anticoagulation since the introduction of heparin in the 1930s.Other anticoagulation drugs have subsequently been added to the treatment armamentarium over the years, such as vitamin K antagonists and low-molecular-weight heparin (LMWH).

Anticoagulant therapy is recommended for 3-12 months depending on site of thrombosis and on the ongoing presence of risk factors. If DVT recurs, if a chronic hypercoagulability is identified, or if PE is life threatening, lifetime anticoagulation therapy may be recommended. This treatment protocol has a cumulative risk of bleeding complications of less than 12%.

CONTRAINDICATION OF ANTICOAGULATION:-

Absolute contraindications include

Intracranial bleeding

Severe active bleeding

Recent brain, eye, or spinal cord surgery

Pregnancy

Malignant hypertension.

Relative contraindications include

Recent major surgery

Recent cerebrovascular accident

Severe thrombocytopenia

HEPARIN THERAPY:-

Heparin is a heterogeneous mixture of polysaccharide fragments with varying molecular weights but with similar biological activity[21].

The larger fragments exert their anticoagulant effect by interacting with antithrombin III (ATIII) to inhibit thrombin. ATIII, the body's primary anticoagulant, inactivates thrombin and inhibits the activity of activated factor X in the coagulation process.[23,24]

Heparin prevents extension of the thrombus and has been shown to significantly reduce (but not eliminate) the incidence of fatal and nonfatal pulmonary embolism and recurrent thrombosis.

Heparin Protocol:-

Administer initial intravenous heparin bolus 5000U

*Administer continuous intravenous heparin infusion: commence at 42 ml/h of 20000U (1680U/h) in 500 ml of two-thirds dextrose and one-third saline (a 24 hour heparin dose of 40320U), except in following patients in whom heparin

infusion is commenced at a rate of 31 mL/h (1240U/h, a 24 hour dose of 29760U)

*Patient who have undergone surgery within the previous 2 weeks

*Patients with a previous history of peptic ulcer disease or gastrointestinal and genitourinary bleeding.

*Patient with recent stroke (thrombotic stroke within 2 weeks previously)

*Patients with a platelet count <150 x 10*9/L

*Patients with miscellaneous reasons for a high risk of bleeding (e.g.:-hepatic failure, renal failure, or Vitamin K deficiency)

*Adjust heparin dose by use of aPTT. The aPTT test is performed in all patients as follows,

*4-6 hours after commencing heparin, the heparin dose is then adjusted

*4-6 hours after the first dose adjustment, then as indicated by the nomogram for the first 24 hours of therapy.

Thereafter once daily, unless the patient is sub therapeutic (< 1.5 times the mean normal control value for the thromboplastin reagent being used) in which case the aPTT test is repeated 4-6 hours after the heparin dose is increased.

Although there is a strong correlation between sub therapeutic aPTT values and recurrent Thromboembolism, the relationship between sub therapeutic aPTT and bleeding is less definite. Indeed bleeding during heparin therapy is more closely related to underlying clinical risk factors than to aPTT elevation above

the therapeutic range. Studies confirm that weight and age > 65 are independent risk factors for bleeding on heparin.

Complications of Heparin therapy:-

- Bleeding
- Thrombocytopenia
- Osteoporosis
- Heparin induced thrombocytopenia

Low Molecular weight Heparin:-

Low-molecular-weight heparins (LMWHs) are a new class of anticoagulants derived from unfractionated heparin (UFH). They have a number of advantages over UFH that have led to their increasing use for a number of thromboembolic indications[13].

They differ from UFH in many ways, of particular importance are the following:-

*Increased bioavailability

*Prolonged half life

*Predictable clearance enabling once or twice daily injection, and *predictable antithrombotic effect based on body weight permitting treatment without laboratory monitoring.

*Ability to inactivate platelet bound factor Xa, resistance to inhibition by platelet factor IV, and their decreased effect on platelet function and vascular

permeability, possibly accounting for less haemorrhagic effects at comparable antithrombotic doses.

Though there are complications like bleeding, Heparin induced thrombocytopenia, osteoporosis, with LMWH the incidence is much lower when compared to UFH.

If heparin induce thrombocytopenia occurs LMWH should be stopped and an alternative such as argatraban or hirudin derivative should be commenced.

ORAL VITAMIN K ANTAGONIST:-

There are 2 distinct chemical groups of oral anticoagulants

4-hydroxy coumarin derivatives (e.g.; warfarin)

Indanedione derivatives (e.g.; Phenindione)

The coumarin derivatives are the anticoagulants of choice because they are associated with fewer non haemorrhagic adverse effects than the Indanedione derivatives[10].

Anticoagulation effect of warfarin id mediated by the inhibition of Vitamin K dependant coagulation factors, II,IV,IX, and X.

The anticoagulant effect of warfarin is delayed until the normal clotting factors are cleared from circulation, and the peak effect does not occur until 36-72 hours after drug administration. Heparin and warfarin treatment should overlap by 4-5 days.

The lab test most commonly used to measure the effects of warfarin is the one stage PT test. To promote standardization of the PT monitoring, WHO (World

Health organization) enveloped an international reference thromboplastin from human brain tissue and recommended at the PT ratio to be expressed as the International normalized ratio, or INR. The monitoring and dosing of the oral anticoagulant therapy is done according to the INR value.

Warfarin is administered in an initial small doses for the first 2 days, then the dose adjusted according to the INR value. Heparin therapy is discontinued in the 4th or 5th day following initiation of warfarin therapy, provided the INR is in the therapeutic range (INR 2-3)

The dose response relationship of warfarin therapy varies widely between individuals and therefor the dose must be carefully monitored to prevent under dosing or over dosing. Patient should be warned against taking any new drugs without the knowledge of their attending physician in view of drug interaction.

Once the anticoagulant effect and patients warfarin dose requirements are stable, the INR should be monitored at regular intervals throughout the course of warfarin therapy for VTE for maintenance of the narrow therapeutic range.

COMPLICATIONS OF WARFARIN

- Major adverse effects are bleeding
- Coumarin induced skin necrosis – it is a serious complication, most commonly seen in women. It occurs between 3 to 10 days after therapy has commenced. Most common locations are areas with abundant subcutaneous tissue such as the abdomen, buttocks, thighs and the breast.

The antidote used for vitamin K antagonists is Vitamin K1.

FACTOR XA AND DIRECT THROMBIN INHIBITORS:-

Fondaparinux

Fondaparinux, a direct selective inhibitor of factor Xa, overcomes many of the aforementioned disadvantages of low-molecular-weight heparins(LMWHs). Pharmacokinetic studies of fondaparinux reveal that only a single-daily subcutaneous dose is required. Furthermore, a single dose of 7.5mg is effective over a wide range of patient weights between 50 and 100 kg. Daily doses of 5 mg or 10 mg are appropriate for patients who weigh less or more than that weight range.

Heparin-induced thrombocytopenia (HIT) has not been reported. Therapeutic monitoring of laboratory parameters such as the prothrombin time or activated partial thromboplastin time (aPTT) is also not required.

In some regions, the cost of therapy

With fondaparinux is less than enoxaparin when it is being used to bridge therapy to a vitamin K antagonist (VKA).

The combination of two factor Xa inhibitors may be an effective treatment strategy for acute venous thromboembolism (VTE).

Rivaroxaban

Rivaroxaban (Xarelto) is an oral factor Xa inhibitor approved by the FDA in November 2012 for treatment of DVT or pulmonary embolism (PE) and for reduction of the risk of recurrent DVT and PE after initial treatment.

Approval for this indication was based on studies totalling 9478 patients with DVT or PE. Participants were randomly assigned to receive Rivaroxaban, a combination of enoxaparin and a VKA (eg, warfarin), or a placebo. Study endpoints were designed to measure the number of patients who experienced recurrent symptoms of DVT, PE, or death after receiving treatment. Data from a pooled analysis of the EINSTEIN-DVT [7] and EINSTEIN-PE [8] trials suggested that Rivaroxaban is as effective in preventing VTE recurrence as enoxaparin followed by a VKA and may be associated with less bleeding in addition, the data suggested that there are no grounds

for avoiding Rivaroxaban use in high-risk groups [26](eg, fragile patients, cancer patients, and patients with a large clot).

Approximately 2.1% of patients treated with rivaroxaban experienced recurrent DVT or PE, compared with 1.8-3% treated with the enoxaparin and VKA combination.

Additionally, results from extended treatment demonstrated a reduced risk of recurrent DVT and PE. Approximately 1.3% in the rivaroxaban group experienced recurrent DVT or PE, compared with 7.1% in the placebo group.

Apixaban

In March 2014, the FDA approved apixaban (Eliquis) for the additional indication of prophylaxis of DVT and PE in adults who have undergone hip- or knee-replacement surgery.

Support for this new indication was a result of the ADVANCE 1, 2, and 3 clinical trials that enrolled nearly 12,000 patients.

Apixaban was originally approved by the FDA in December 2012 for the prevention of stroke and systemic embolism in patients with

nonvalvular atrial fibrillation.

In August 2014, apixaban was approved for treatment of DVT and PE. The approval for treatment of PE and prevention of recurrence was based on the outcome of the AMPLIFY (Apixaban for the Initial Management of Pulmonary Embolism and Deep-Vein Thrombosis as First-Line Therapy) and AMPLIFY-EXT (extended treatment) studies, in which apixaban therapy was compared with enoxaparin and warfarin treatment.

The AMPLIFY study showed that, in comparison with the standard anticoagulant regimen, apixaban therapy resulted in a 16% reduction in the risk of a composite endpoint that included recurrent symptomatic venous thromboembolism (VTE) or VTE-associated death.

Data from the AMPLIFY-EXT trial showed that extended anticoagulation (12 months) with apixaban shortened hospital stays, reduced symptomatic recurrent

venous thromboembolism or all-cause death without an associated increase in major episodes of haemorrhage when compared with placebo.

Dabigatran

Dabigatran (Pradaxa) inhibits free and clot-bound thrombin and thrombin-induced platelet aggregation. This agent was FDA approved in 2010 to reduce the risk of stroke and systemic embolism in patients with nonvalvular atrial fibrillation. In April 2014, it was approved for the treatment of DVT and PE in patients who have been treated with a parenteral anticoagulant for 5-10 days. Additionally, it was approved to reduce the risk of DVT and

PE recurrence in patients who have been previously treated. Approval was based on results from 4 global phase III trials that showed Dabigatran was non inferior to warfarin and had a lower risk of major or clinically relevant bleeding compared with warfarin. There have been reports of severe and fatal bleeding in users of the drug.

The RE-COVER and RE-COVER II trials included patients with DVT and PE who were treated with parenteral anticoagulant therapy for 5-10 days. Results showed Dabigatran was non inferior to warfarin in reducing DVT and PE after a median of 174 days of treatment with a lower risk of bleeding compared with warfarin.

The RE-SONATE trial and RE-MEDY trials included patients (n=2856) with acute DVT and PE who had completed at least 3 months of anticoagulant therapy. Results from this trial showed Dabigatran was non inferior to warfarin

in the extended treatment of VTE and carried a lower risk of major or clinically relevant bleeding than warfarin.

Edoxaban

Edoxaban (Savaysa) was approved by the FDA in January 2015 for the treatment of DVT and PE in patients who have been initially treated with a parenteral anticoagulant for 5-10 days. Approval was based on the Hokusai-VTE study that included 4,921 patients with DVT and 3,319 patients with PE. Among patients with PE, 938 had right ventricular dysfunction, as assessed by measurement of N-terminal pro-brain natriuretic peptide (NT-proBNP) levels. There was a 3.3% rate of recurrent VTE in this subgroup in those who received Edoxaban compared to 6.2% in the group that received warfarin. The investigators concluded that Edoxaban was not only non inferior to high-quality standard warfarin therapy but also caused significantly less bleeding in a broad spectrum of patients with VTE, including those with severe PE.

Reversal of Anticoagulation

Anticoagulation-related major bleeding is associated with an increased risk of death and thrombotic events, independent of the class of anticoagulant used. Although older agents of anticoagulation and their reversal are well studied, the newer agents lack similar antidotes. With the increasing use of non–vitamin K antagonist oral anticoagulants (NOAC), the number of patients who require reversal of their anticoagulant effects can be expected to rise.

The following section describes the reversal agents for both older and new anticoagulants.

Heparin

Heparin has a relatively short half-life of about 60–90 minutes and, therefore, the anticoagulant effect of therapeutic doses of heparin will mostly be eliminated at 3-4 hours after termination of continuous intravenous administration. For a more immediate neutralization of heparin, protamine sulfate can be administered at a dose of 1 mg for every 100 units of heparin.

Lower molecular weight heparins

Currently, there are no specific antidotes to low molecular weight heparins. Recombinant FVIIa (rVIIA) has been shown to stop bleeding in patients anticoagulated with fondaparinux; however, no randomized controlled trials on such patients have been conducted.

Warfarin

Vitamin K

In patients with clinically significant bleeding, vitamin K can be used to reverse the anticoagulant effect of vitamin K antagonists (VKA). Vitamin K can be given orally or intravenously. The parenteral route has a more rapid onset; however, it is associated with a slightly increased risk of allergic reaction.

Fresh frozen plasma (FFP)

In case of a life-threatening emergency, FFP can be used for the reversal of VKA. FFP contains all the coagulation factors in normal concentrations.

However, FFP should be used with caution, as it has the potential to cause volume overload, allergic reaction, and transfusion-related reactions.

Prothrombin complex concentrates (PCCs)

In the case of serious and life-threatening bleeding, immediate correction of the international normalized ratio (INR) can be achieved by the administration of PCCs. These contain 3 or 4 of the vitamin K–dependent coagulation factors, as well as proteins C and S. In a prospective study, administration of PCCs has been shown to result in sustained haemostasis in patients using VKA.

Non–vitamin K antagonist oral anticoagulants (NOACs)

The new oral anticoagulant factor Xa or IIa inhibitors have numerous advantages over traditional VKAs, including rapid therapeutic effectiveness, ease of dosing, and lack of monitoring. Until recently, there were no approved drug-specific reversal agents for the NOACs.

A number of drugs are currently under development. [145, 146]

Due to the short half-life of FXa inhibitors, discontinuation of the drugs suffice in clinical situations in which there is time to await spontaneous clearance.

Currently, PCCs can be used to address severe bleeding in patients taking NOACs when administered in high enough dosages. Some guidelines suggest an initial dose of 25 to 50 U/kg of PCCs in life-threatening emergencies, to be repeated if necessary.

Idarucizumab (Pradbind)

Idarucizumab is a humanized antibody fragment directed against Dabigatran. This agent has been shown to completely reverse the anticoagulant effect of Dabigatran within minutes; on October 16, 2015, it was approved by the FDA as an antidote for Dabigatran. [147, 148, 149, 150]

Andexanet alfa

Andexanet alfa is a recombinant, modified FXa molecule that acts as a decoy protein that is catalytically inactive but has a high affinity for FXainhibitors. It is being developed as an antidote for apixaban, Edoxaban, and Rivaroxaban. Andexanet alfa has been shown to reverse the anticoagulant effects of apixaban and Rivaroxaban in human volunteers, and more studies are ongoing.

Aripazine(PER977, ciraparantag)

Aripazine is a synthetic small molecule that has broad activity against both old (Heparin, low molecular weight heparin) and new oral anticoagulants (Dabigatran, Rivaroxaban, apixaban, Edoxaban). A 2014 study of human volunteers demonstrated that administration of Aripazine reversed the prolonged clotting time caused by Edoxaban.

ENDOVASCULAR INTERVENTION:-

Percutaneous transcatheter treatment of patients with deep venous thrombosis (DVT) consists of thrombus removal with catheter-directed thrombolysis[30], mechanical thrombectomy, angioplasty, and/or stenting of venous obstructions.

In some cases, patients may also be given pulmonary embolism (PE) prophylaxis by means of filter placement in the inferior venacava[27].

Indications for Percutaneous Transcatheter Treatment **include the relatively rare**

Phlegmasia or symptomatic inferior vena cava thrombosis that responds poorly to anticoagulation alone, symptomatic iliofemoral or femoropopliteal DVT in patients with a low risk of bleeding. In the last groups, the goal is to reduce the high risk of PTS or to achieve symptomatic relief in conjunction with angioplasty or stent placement.

Phlegmasia cerulea dolens treatment

Phlegmasia cerulea dolens is an indication for emergency catheter-directed thrombolysis in patients with moderate or low bleeding risks. This recommendation is based on reports of limb salvage, which stand in contrast to the high rates of limb amputation and death seen with standard therapies. Surgical thrombectomy remains an effective option in patients at high risk for haemorrhagic complications, although it often results in incomplete thrombus removal, recurrent DVT, and an increased incidence of systemic complications.

Inferior vena cava thrombosis treatment

Acute or subacute inferior vena cava thrombosis that causes at least moderate pelvic congestion, limb symptoms, or compromised visceral venous drainage warrants catheter-directed thrombolysis. Involvement of the suprarenal cava,

renal veins, and/or hepatic veins may precipitate acute renal or hepatic failure. Thrombus that involves the upper inferior vena cava may make it impossible to place an inferior vena cava filter for PE prophylaxis.

Subacute and chronic Iliofemoral DVT treatment

Subacute and chronic iliofemoral DVT is accompanied by moderate to severe pelvic or limb symptoms with a low bleeding risk. Because recanalization of the iliac vein is unlikely, iliofemoral DVT often produces valvular reflux. This combination of outflow obstruction and reflux is associated with the most symptomatic forms of PTS. In this situation, patients have venous damage, and the alternative is venous bypass. In these instances, catheter-directed thrombolysis is seldom expected to completely clear the vein, but it is often used to remove any acute component of thrombus and to uncover chronic stenoses or underlying anatomic abnormality as an adjunct to angioplasty or stent placement. Compared with systemic thrombolysis, catheter-directed thrombolysis improves the preservation of valve competence (44% vs 13%).

Acute iliofemoral or femoropopliteal DVT treatment

Whether catheter-directed thrombolysis is indicated in the relatively common event of acute iliofemoral or femoropopliteal DVT is somewhat controversial. Catheter-directed thrombolysis may be superior to anticoagulation with regard to decreasing the incidence of recurrent DVT and PTS. However, the evidence is not conclusive.

Catheter-directed thrombolysis improves thrombus clearance compared with systemic thrombolysis.

Asymptomatic DVT

Asymptomatic DVT is not considered an indication for endovascular intervention at this time. The incidence of PTS at 5 years after asymptomatic calf or proximal DVT is low, at 5%. The absence of symptoms may reflect the lack of the obstructive effect that is proposed to initiate the syndrome. On the other hand, while the incidence of PTS may not warrant endovascular treatment, some reports suggest that treatment of asymptomatic DVT may be necessary to prevent most cases of PE that are diagnosed at autopsy. Asymptomatic proximal DVT had a mortality risk of 13.7% versus 2% in patients without DVT

Contraindications for percutaneous transcatheter treatment they are the same as those for thrombolysis in general.

Absolute contraindications include:-

Active internal bleeding or disseminated intravascular coagulation,

A cerebrovascular event, trauma, and neurosurgery within 3 months.

Relative contraindications

They include major surgery within 10 days, obstetric delivery, major trauma, organ biopsy, intracranial or spinal cord tumour, uncontrolled hypertension, major GI haemorrhage (within 3 months), serious allergic reaction to a

thrombolytic agent, known right-to-left cardiac or pulmonary shunt or left-heart thrombus, and an infected venous thrombus.

Unfortunately, most patients with DVT have absolute contraindications to thrombolytic therapy. Thrombolytic therapy is also not effective once the thrombus is adherent and begins to organize. Venous thrombi in the legs are often large and associated with complete venous occlusion. In these cases, thrombolytic agents act on the surface of the clot but may not be able to penetrate and lyse the entire thrombus.

Technique:-

Access to the iliofemoral venous circulation is usually obtained via the popliteal vein, using ultrasonographic guidance, although the common femoral, tibial, or internal jugular veins are also used. When thrombolysis is planned, use of ultrasonography and a micropuncture 21-gauge needle are recommended to minimize bleeding risk.

Diagnostic venography is used to identify the extent of DVT. Fluoroscopic guidance is the most accurate and straightforward means of placing infusion catheters or devices. A sheath is placed, and a multiple–side-hole catheter or wire is used to maximize delivery of the thrombolytic agent to the surface area of the thrombus.

During thrombolysis, patients remain on bed rest, with frequent monitoring of vital signs and puncture sites performed. Pericatheter oozing, enlarging hematoma, or evidence of gastrointestinal or genitourinary bleeding warrant

immediate attention. Additional punctures, particularly arterial or intramuscular ones, should be avoided.

A separate IV access facilitates blood sampling, which is performed at 6-hour intervals to monitor the patient's haematocrit; platelet count; activated partial thromboplastin time (aPTT), if concomitant heparinization is used; and possibly fibrinogen values. Monitoring of fibrinogen levels is controversial, although levels < 4.4 µmol/L (150 mg/dL) might indicate a clinically significant systemic effect.

Comparison of plasminogen activators and dosages

Plasminogen activators include streptokinase, uPA, tissue-type plasminogen activator (tPA; alteplase), tenecteplase (TNK), and recombinant tPA (r-tPA; reteplase).

The FDA has approved only streptokinase for systemic thrombolytic therapy of DVT. However, this agent is not currently recommended because of high rates of allergic reaction and bleeding complications and because of the availability of lower-risk agents. In the 1980s and 1990s, uPA was used extensively, but when it was temporarily taken off the market, tPA and r-tPA subsequently became the agents of choice.

In a retrospective analysis of catheter-directed thrombolysis for DVT, no significant differences were observed between uPA, tPA, and r-tPA with regard

to success rate (>97%) or major complications (3-8%), although tPA and r-tPA were significantly less expensive than uPA. [16]

Recommended continuous dosages for catheter-directed thrombolysis of unilateral leg DVT are as follows:

- tPA - 0.5-1.0 mg/h
- r-tPA - 0.25-0.75 U/h
- TNK - 0.25-0.5 mg/h

Other dosing options include an initial lacing dose, which entails an initial bolus given throughout the target thrombus, and a front-loaded dose, which is a high concentration given for the first few hours. No advantage to either approach has been demonstrated.

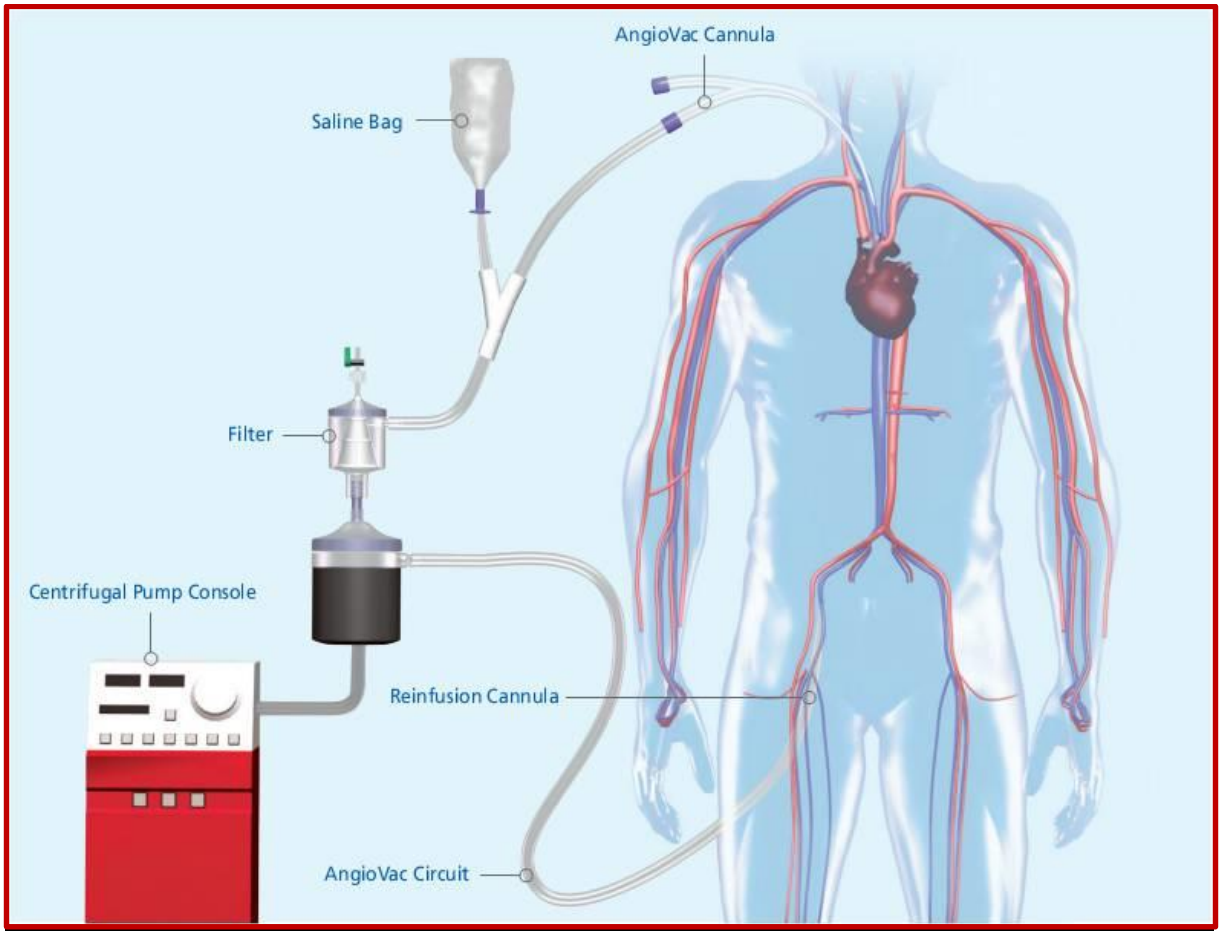
Concomitant heparinization

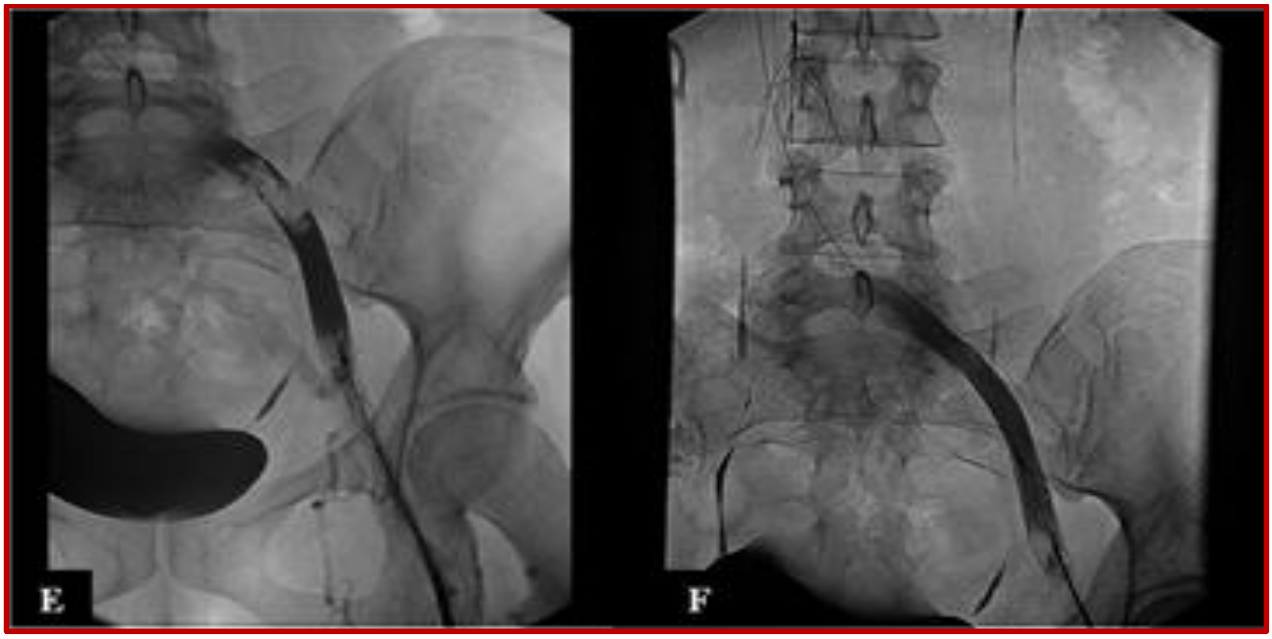
Most practitioners use concomitant heparinization. Full heparinization was commonly used in conjunction with uPA, whereas the current trend is to administer subtherapeutic heparin with tPA. Low-molecular-weight heparin (LMWH) has not been studied in this setting

COMPLICATIONS:-

The haemorrhagic complications of thrombolytic therapy are formidable (approximately 3 times higher than that of anticoagulant therapy) and include the small, but potentially fatal, risk of intracerebral haemorrhage. The

uncertainty regarding thrombolytic therapy is likely to continue. Currently, the American College of Chest Physicians (ACCP) consensus guidelines recommend catheter-directed thrombolytic therapy only for selected patients with extensive acute proximal DVT (eg, those with iliofemoral DVT, symptoms for less than 14 days, good functional status, and life expectancy of >1 year) who are at low risk of bleeding





INFERIOR VENACAVA FILTERS

Inferior vena cava filters are not recommended in patients with acute venous thromboembolism (VTE) on anticoagulant therapy.

These filters were developed in an attempt to trap emboli and minimize venous stasis. In most patients with deep venous thrombosis (DVT), prophylaxis against the potentially fatal passage of thrombus from the lower extremity or pelvic vein to the pulmonary circulation is adequately accomplished with anticoagulation. An inferior vena cava filter is a mechanical barrier to the flow of emboli larger than 4 mm.[28,29]

The ideal vena cava filter would trap venous emboli while maintaining normal venous flow. Many different filter configurations have been used, but the current benchmark remains the Greenfield filter with the longest long-term data. Patency rates greater than 95% and recurrent embolism rates of less than 5%

have been demonstrated by numerous studies. The conical shape allows central filling of emboli while allowing blood on the periphery to flow freely. Numerous other filters with similar track records have since been developed, including filters that can be removed.

Regardless of the type of filter placed, the technique remains the same. Local anaesthetic is used to anesthetize either the groin for a femoral vein approach or the neck for a jugular vein approach.

A single wall needle is used under ultrasonic guidance to enter the target vein, and a 0.035-inch guide wire is passed into the inferior vena cava.

A venogram is performed to identify the renal veins and measure the diameter of the vena cava to ensure the cava is not too big for the filter. Intravascular ultrasound (IVUS) can also be used for this purpose. It has the added benefit of not only allowing for bedside filter placement in sick intensive care unit (ICU) patients, but it also obviates the need for IV contrast. The correct filter location traditionally entails an infra-renal fixation with central filter extension to the level of the renal veins. Placement in the suprarenal inferior vena cava or superior vena cava may be indicated in some situations.

American Heart Association recommendations for inferior vena cava filters include the following:-

*Confirmed acute proximal DVT or acute PE in patient with contraindication for anticoagulation (this remains the most common indication for inferior vena cava filter placement)

*Recurrent thromboembolism while on anticoagulation

*Active bleeding complications requiring termination of anticoagulation therapy

Relative contraindications include the following:

*Large, free-floating iliofemoral thrombus in high-risk patients.

*Propagating iliofemoral thrombus while on anticoagulation

*Chronic PE in patient with pulmonary hypertension and corpulmonale

*Patient with significant fall risk

Complications:-

Migration

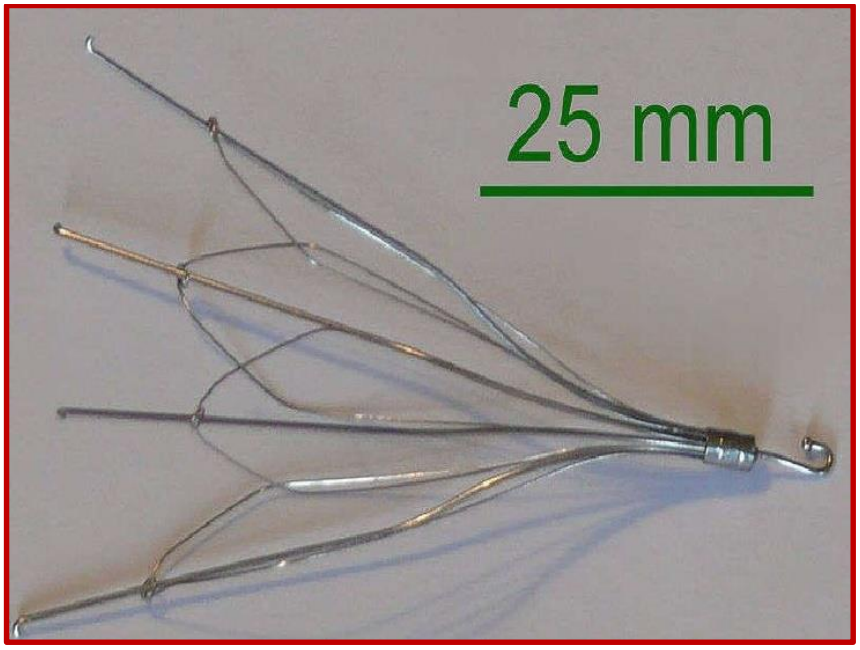
Thrombosis

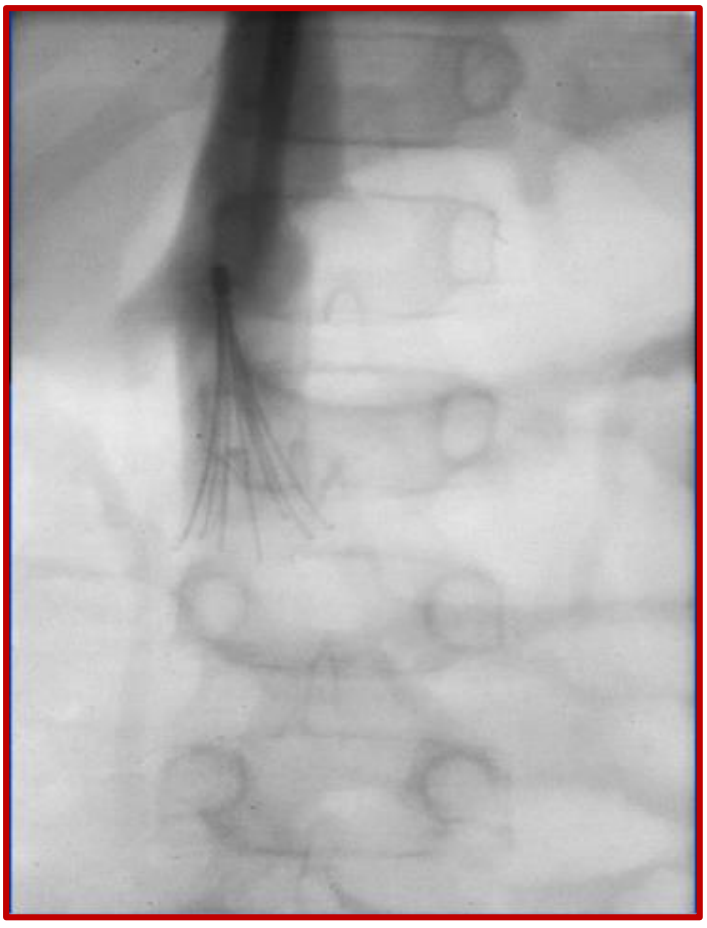
Filter fracture

IVC perforation

Pulmonary embolism

Device infection


25 mm



SURGICAL THROMBECTOMY:-

Surgical thrombus removal has traditionally been used in patients with massive swelling and phlegmasia cerulea dolens. In many patients, fibrinolysis alone is highly effective, and it has become the primary treatment of choice for many forms of venous and arterial thrombosis.

Unfortunately, when thrombosis is extensive, fibrinolysis alone may be inadequate to dissolve the volume of thrombus present. Even when the bulk of the thrombus is not excessive, many patients with thrombosis are poor candidates for fibrinolysis because of recent surgery or trauma involving the central nervous

system or other noncompressible areas. Precisely defining the location and extent of thrombosis before considering any surgical approach to the problem is important.

Duplex ultrasonography may sometimes be sufficient for this purpose, but venography (including routine contralateral iliocavography) is a more reliable guide to the anatomy and the particular pathology that must be addressed.

The patient must be heparinized before the procedure. Traditional venous thrombectomy is performed by surgically exposing the common femoral vein and sapheno femoral junction through a longitudinal skin incision. A Fogarty catheter is passed through the clot, and the balloon is inflated and withdrawn, along with the clot. However, care must be taken to avoid dislodging the clot or breaking it into small fragments because pulmonary embolus will result.

A proximal balloon or a temporary caval filter may be used to reduce the likelihood of embolization. Venography is mandatory to confirm the clearance of the thrombus. Back bleeding does not indicate clot clearance because a patent valve can block flow, or flow can be present with patent tributaries.

Venous valves may sometimes prevent the passage of a catheter in a retrograde direction down the leg. When this happens, the leg may be wrapped tightly with an Esmarch bandage in an attempt to force clot extrusion. After the thrombus has been removed, construction of a small arteriovenous fistula may assist in maintaining patency by increasing the flow velocity through a thrombogenic iliofemoral venous segment and promoting collateral development. The fistula is usually performed between the saphenous vein and the femoral vein.

To reduce the likelihood of rethrombosis, heparin anticoagulation is usually initiated before surgery, continued during the procedure, and maintained for 6-12 months afterward. Leg compression devices are useful to maintain venous flow.

Outcomes from multiple studies have shown rethrombosis rates around 12% when a temporary arteriovenous fistula is used. Optimal results were found in thrombosis less than 7 days, clearance of thrombus from the external and internal iliac veins, intraoperative venography, early ambulation, and religious use of compression stockings.

In a prospective randomized study from Sweden comparing surgery with anticoagulation, at 5 years, 37% of operated patients were asymptomatic, compared with just 18% in the anticoagulation group. Vein patency was 77% in the surgical group compared with just 30% in the anticoagulation group.

PROPHYLAXIS

The patient who has undergone major abdominal or orthopaedic surgery, has sustained major trauma , or has prolonged immobilisation (> 3 days) represents an elevated risk for the development of venous thromboembolism[12,13]. The methods of prophylaxis can be mechanical or pharmacological. The simplest method is for the patient to walk. Activation of the calf pump mechanism is an effective means of prophylaxis , as evidenced by the fact that few active people without underlying factors develop venous thrombosis.

The most common method of surgical prophylaxis has traditionally revolved around compression devices, which periodically compress the calves and essentially replicate the calf bellows mechanism. This has clearly reduced the incidence of venous thromboembolism in the surgical patient. The most likely mechanism for the efficacy of this device is prevention of venous stasis.

Another traditional method of thromboprophylaxis has been the use of low-dose unfractionated heparin. The dosage traditionally used was 5000 U of

unfractionated heparin every 12 hours. More recently , a number of studies have revealed the efficacy of fractionated low-molecular weight heparin for the prophylaxis and treatment of venous thromboembolism.

Comparison of LMWH with mechanical prophylaxis has demonstrated the superiority of LMWH for reduction of the development of venous thromboembolic disease [13,14,15.] LMWH is considered the optimal method of prophylaxis for moderate- and high-risk patients. Even the traditional reluctance to use heparin in high-risk groups , such as the multiply injured patient and head-injured patient , must be re-examined , given the efficacy and safety profile of LMWH in multiple prospective trials.

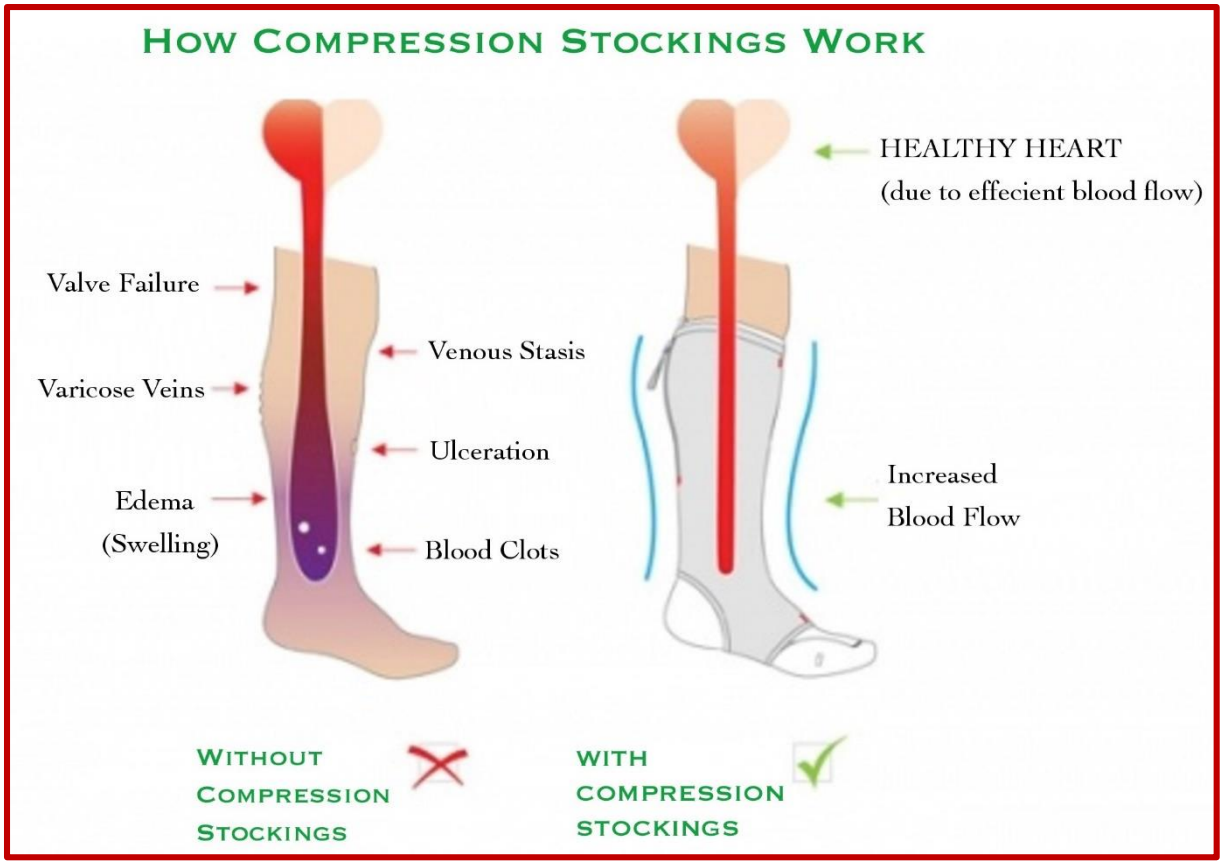

HOW COMPRESSION STOCKINGS WORK
Valve Failure
Varicose Veins
Edema
(Swelling)
Venous Stasis
Ulceration
Blood Clots
HEALTHY HEART
(due to effecient blood flow)
Increased
Blood Flow
WITHOUT
COMPRESSION
STOCKINGS
WITH
COMPRESSION
STOCKINGS

Table 1

Compression-Hosiery Classification and Indications[a]

Class	Pressure	Indications for Use
	8-15 mmHg (support)[b]	Tired/aching feet and legs, slight edema Spider veins, early varicose veins
1	14-17 mmHg[c] 15-20 mmHg[b]	Varicose veins, mild edema DVT prevention
2	18-24 mmHg[c] 20-30 mmHg[b]	Moderate varicose veins, mild edema Prevention of venous-ulcer recurrence
3	25-35 mmHg[c] 30-40 mmHg[b]	Severe varicose veins Prevention and treatment of venous ulcers Lymphedema Postphlebitic limb Chronic venous insufficiency

[a] Choice of type and level of compression is patient-specific. Patient considerations include arterial status and ability to tolerate and put on the stocking.
[b] U.S. recommendation.
[c] British standard.
DVT: deep vein thrombosis.
Source: References 5, 7, 27, 28.

MATERIALS AND METHODS

THIS STUDY WAS CONDUCTED IN THANJAVUR MEDICAL COLLEGE FROM PERIOD OF SEPTEMBER 2016-2017.

The study includes 100 patients presenting with complaints of lower limb pain, swelling admitted in surgical wards during the above mentioned period. Detailed history was taken and thorough physical examination was done. All details were recorded in the proforma designed prior to the commencement of the study.

The recorded details include patients particulars, duration of condition, predisposing factors, details about previous medical or surgical illness, vitals monitored, basic blood investigations done. All patients were subjected to Doppler USG and involved segments noted.

INCLUSION CRITERA

1. Both sex, with age between 20-80 years
2. Patients presenting with primary DVT
3. Patients presenting with secondary DVT

EXCLUSION CRITERIA

1. Age less than 20 and more than 80
2. Patients not willing to be part of this study.

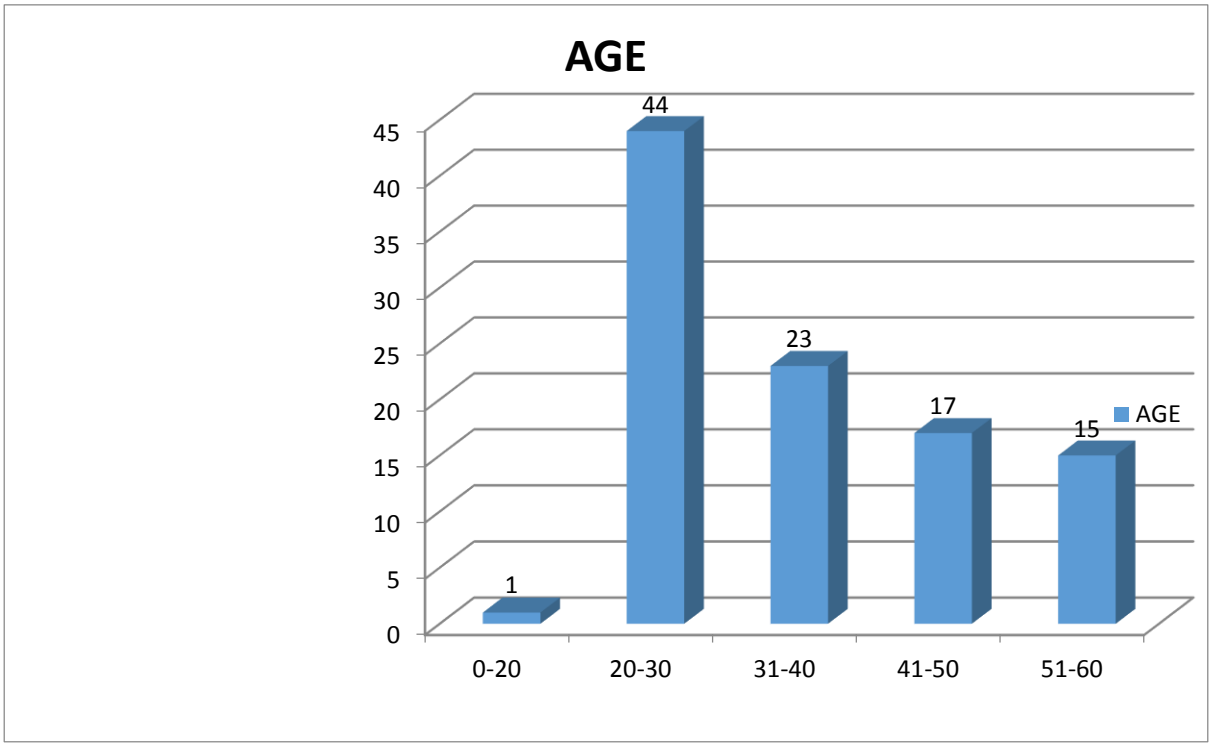
STUDY DESIGN

- This is an observational study conducted in our institution, Thanjavur Medical College during the period September 2016-2017.
- Patients were subjected for Doppler ultrasound and region of thrombus identified .
- All patients were treated with IV anticoagulants and improved. Later changed to oral anticoagulants.
- Young patients presenting within 6 hrs were treated with Catheter directed thrombolysis using UROKINASE and showed improvement.
- Patients were discharged and advised to continue oral anticoagulants.

RESULT

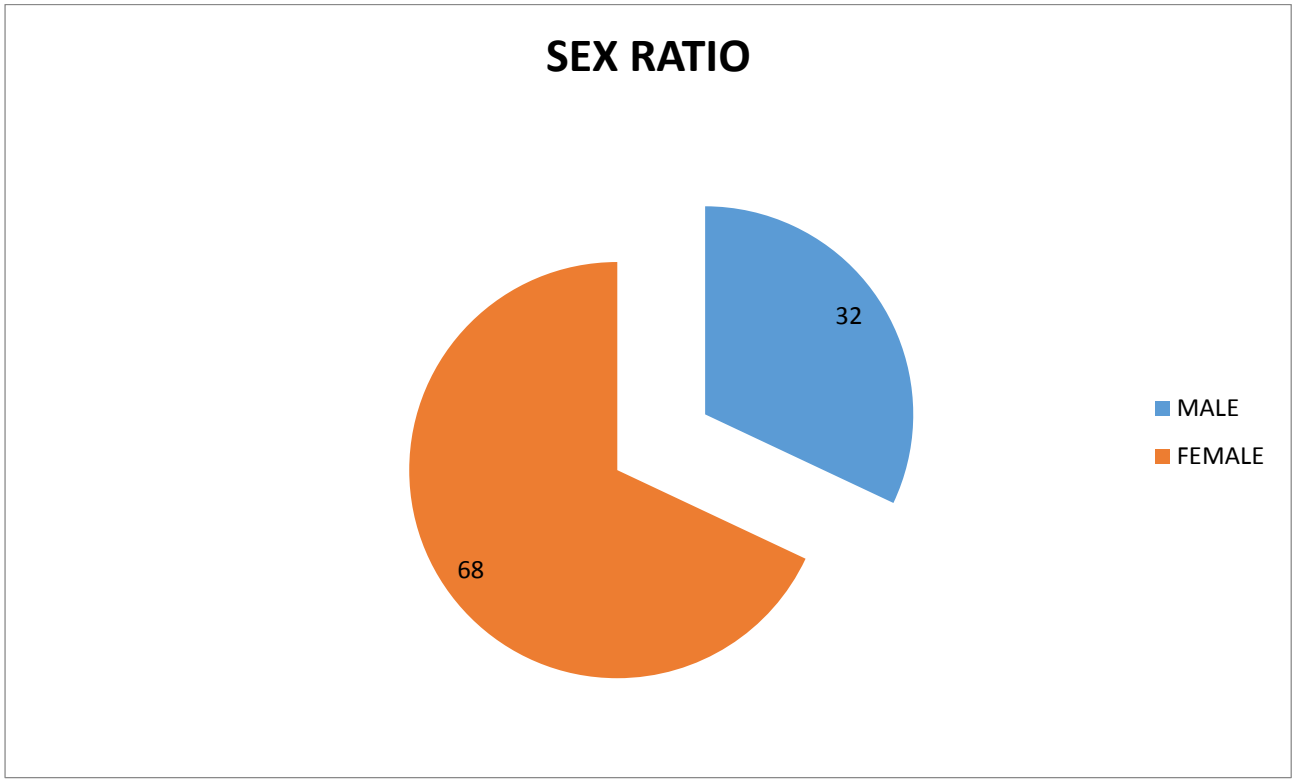
1. AGE

Age Particulars	No.of respondents (n=100)	Percentage (100%)
Below 20	1	1.0
21 to 30	44	44.0
31 to 40	23	23.0
41 to 50	17	17.0
Above 51	15	15.0
Mean:35.98/S.D.:12.192/min.:20/max.:65		



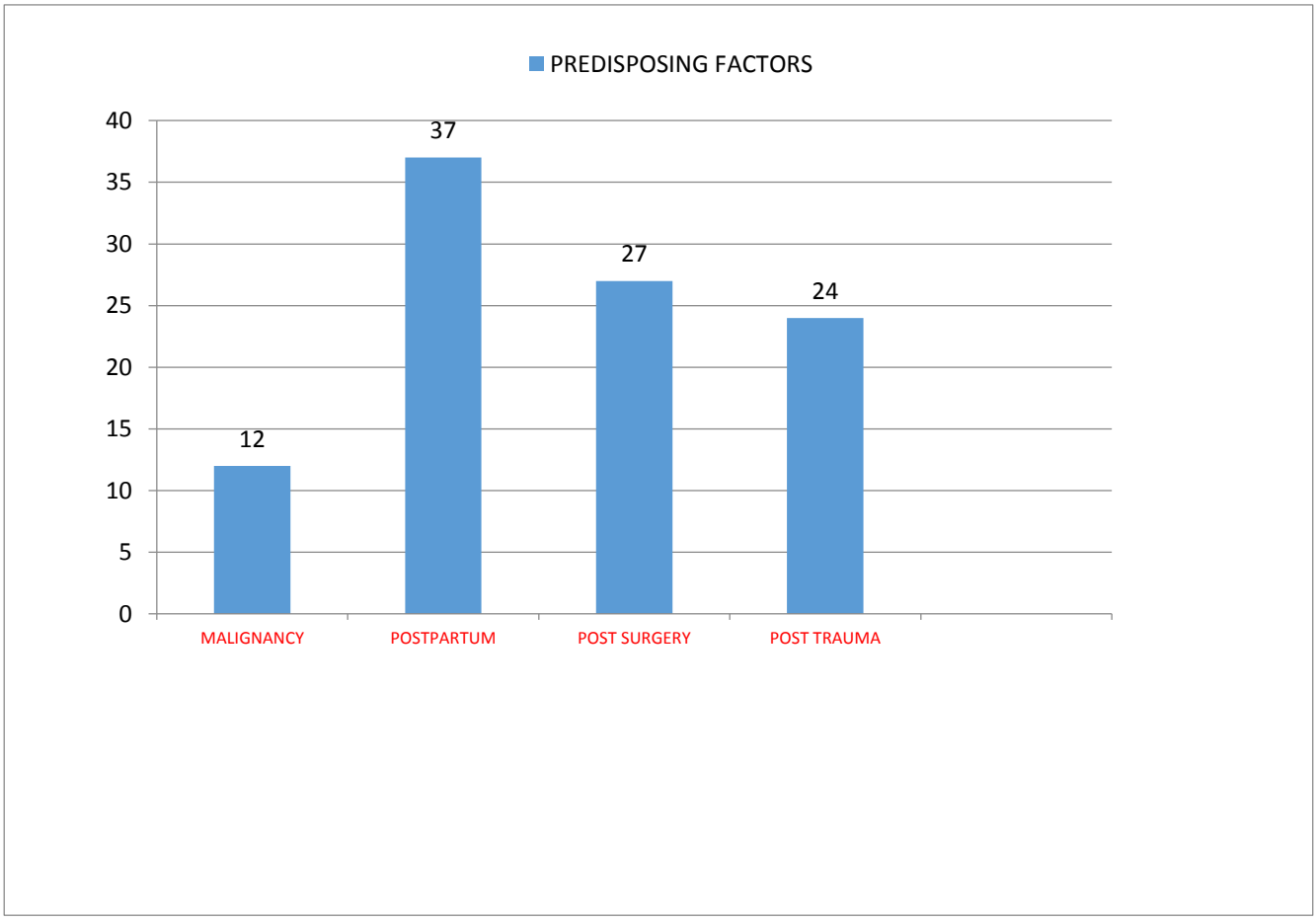
II. SEX RATIO :-

Particulars	No.of respondents (n=100)	Percentage (100%)
MALE	32	32.0
FEMALE	68	68.0



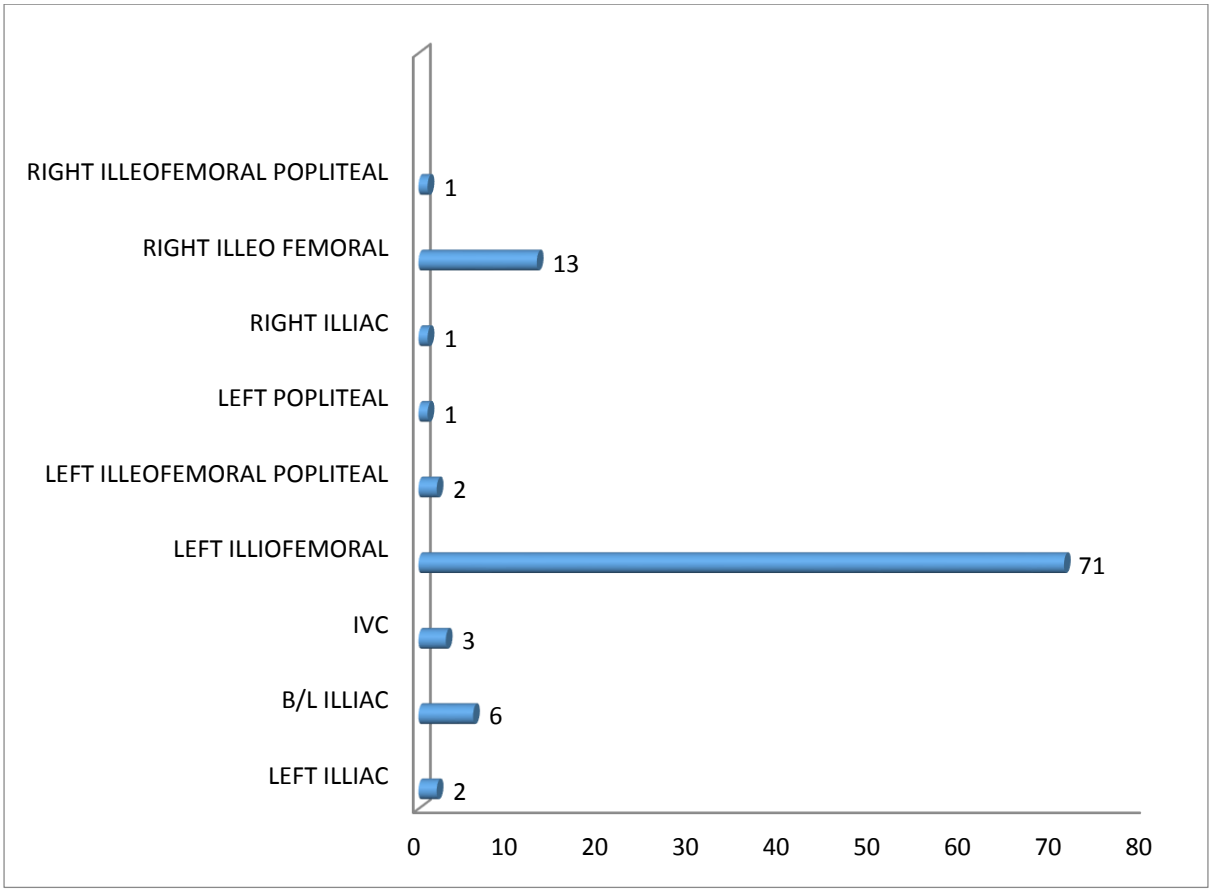
111) PREDISPOSING FACTORS

Particulars	No.of respondents (n=100)	Percentage (100%)
MALIGNANCY	12	12.0
POST PARTUM	37	37.0
POST SURGERY	27	27.0
POST TRAUMA	24	24.0



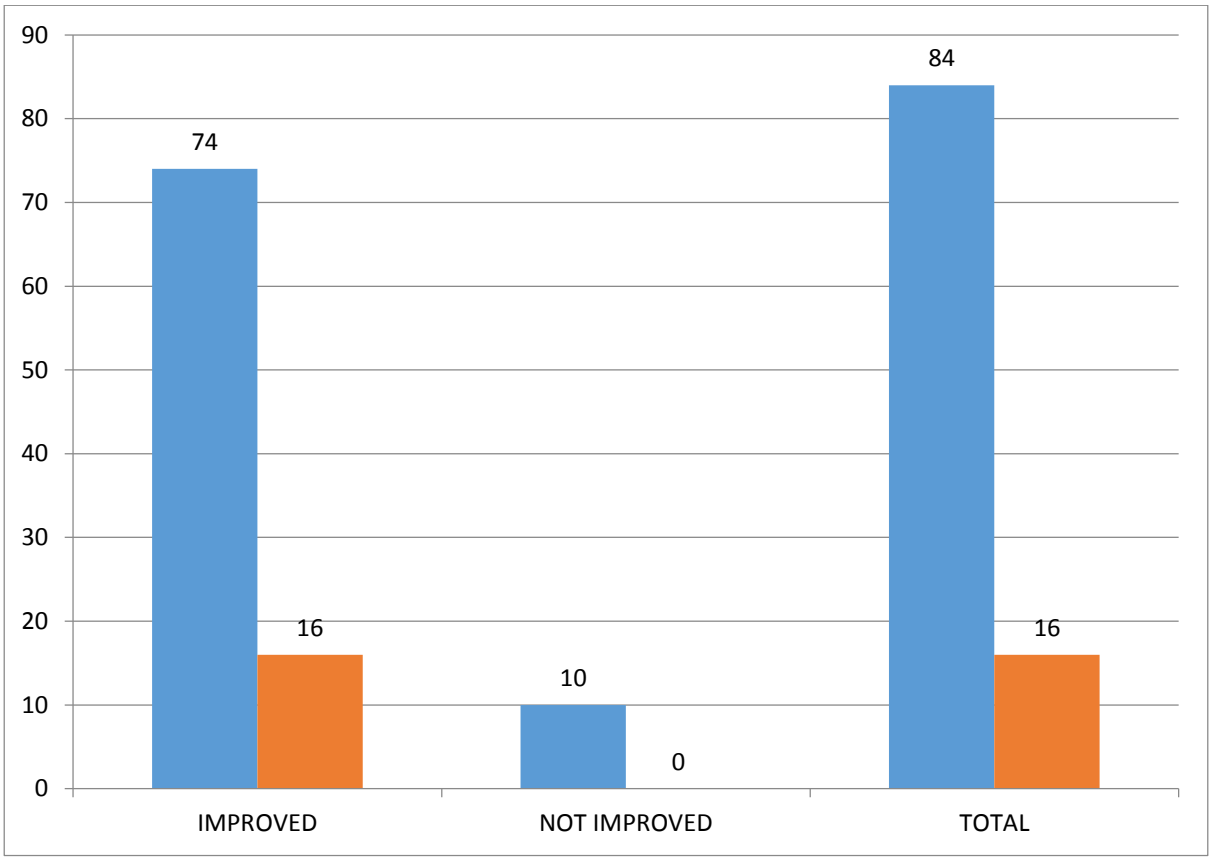
IV REGION

Particulars	No.of respondents (n=100)	Percentage (100%)
LEFT ILIAC	2	2.0
BILATERAL ILIAC	6	6.0
IVC	3	3.0
LEFT ILIOFEMORAL	71	71.0
LEFT ILIOFEMORAL POPLITEAL	2	2.0
LEFT POPLITEAL	1	1.0
RIGHR ILIAC	1	1.0
RIGHT ILIOFEMORAL	13	13.0
RIGHT ILIOFEMORALPOPLITEAL	1	1.0



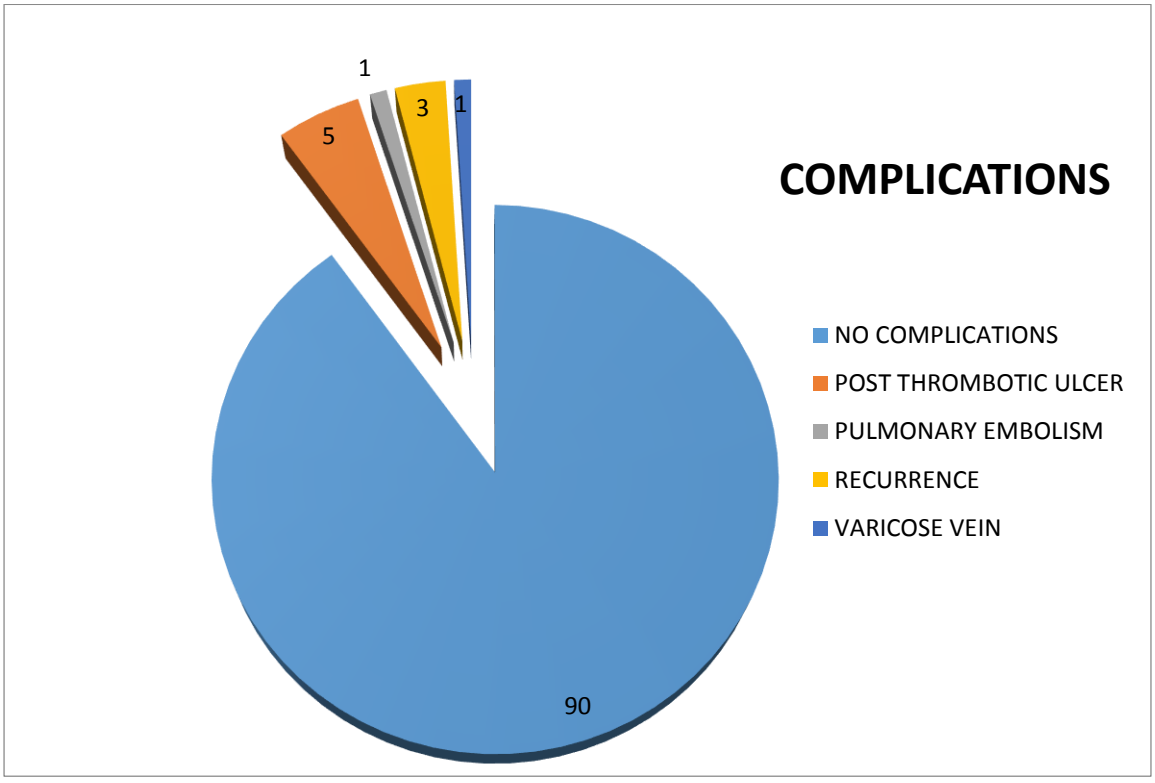
V MANAGEMENT

Particulars	IMPROVED	NOT IMPROVED	No.of respondents (n=100)	Percentage (100%)
MEDICAL	74	10	84	84.0
CDT	16	0	16	16.0



VI.COMPLICATIONS

Particulars	No.of respondents (n=100)	Percentage (100%)
NO COMPLICATIONS	90	90.0
POST THROMBOTIC ULCER	5	5.0
PULMONARY EMBOLISM	1	1.0
RECURRENCE	3	3.0
VARICOSE VEIN	1	1.0



DISCUSSION

In my study, most common age group was found to be between 21-30 years 44%, followed by 31-40 years 23% , 41-50 17% , above 50-15%.

Most common in females 68%

Most common in postpartum period 37%, followed by post-surgery 27% , post trauma immobilization 24%, malignancy 12%

Most common site of thrombosis was found to be LEFT ILIOFEMORAL 71% , followed by RIGHT ILIOFEMORAL 13% , BILATERAL ILIAC 6% , IVC 3% ,LEFT ILIOFEMEROPOPLITEAL 2 % , LEFT ILIAC 2 %,RIGHT ILIAC 1%, RIGHT ILIOFEMEROPOPLITEAL 1 % AND LEFT POPLITEAL 1%.

74% CASES RESPONDED WELL TO MEDICAL MANAGEMENT

CDT was done for 16 patients and all of them improved.

90 % patients improved with medical management and CDT, 10 % not improved and went for complications.

COMPLICATIONS found were POST THROMBOTIC ULCER 5 %, RECURRENCE 3 % , VARICOSE VEIN 1 % PULMONARY EMBOLISM 1 %.

CONCLUSION

Venous thromboembolism (VTE) is a serious preventable cause of morbidity & mortality in the world. DVT & pulmonary embolism (PE) are distinct but related aspects of VTE.

Cancer patients undergoing surgical procedures have at least two times the risk of post-operative D.V.T. and greater than three times risk of fatal P.E. than noncancerous patients undergoing similar procedure. In elderly, D.V.T. is associated with 21% one-year mortality rate and P.E. is associated with 39% one-year mortality rate.

P.E. is the leading cause of maternal death associated with childbirths. A woman's risk of developing DVT is six times greater when she is pregnant.

Evaluation and proper management is essential to decrease the burden of V.T.E.

DOPPLER USG can easily find out DVT and anticoagulant therapy can effectively cure and prevent complications due to DVT. CATHETER DIRECTED THROMBOLYSIS when done in early time of presentation was found to be effective and without complications in this study.

SUMMARY

This study was conducted in our institution, Thanjavur Medical College during September 2016-2017 to study the Epidemiology, Natural history, Treatment and Outcome of DVT.

100 patients in the age group of 20-80 years were included in this study. Detailed history and physical examination was done for all the patients. Basic blood investigations were done. Doppler USG was done for all the patients to confirm the diagnosis and to find out the region affected. All patients were treated with anticoagulants. 16 patients underwent Catheter directed thrombolysis with urokinase.

Mean age group was between 21-40 years. Most common in females and during postpartum period.

Most common site was found to be LEFT ILIOFEMORAL VEIN.

74% patients improved with medical management alone.

Patients treated with CDT showed improvement without any complications.

10 % patients treated with medical management experienced complications like post thrombotic ulcer, varicose vein, recurrence and pulmonary embolism.

ABBREVIATIONS

PE	- pulmonary embolism
VTE	- venous thromboembolism
DVT	- deep vein thrombosis
PGI2	- prostaglandin I2
TPA	- tissue plasminogen activator
ELISA	- enzyme linked immunosorbent assay
MRV	- magnetic resonance venography
CV	-contrast venography
Aptt	-activated partial thromboplastin time
LMWH	-low molecular weight heparin
UFH	- unfractionated heparin
PT	-prothrombin time
INR	-international normalised ratio
VKA	- vitamin K antagonist
NT	-proBNP-N-terminal pro-brain natriuretic peptide

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PROFORMA

Serial No :

Name :

Age :

Sex : M/F

Date of Admission :

Op/Ip number :

Co- morbidities : Diabetes/ Hypertension/ Ischaemic Heart Disease/ Others

Vitals at Arrival :

Temperature :

Pulse rate :

Respiratory Rate :

Blood pressure :

Complaints :

Pain- Site :

Duration :

Character :

Aggravating/Relieving Factors:

Swelling

Site :

Duration :

Tenderness :

Skin over the Swelling :Normal / Redness / Ulcer / Dialated Veins

Distal Pulse :Palpable/Not Palpable

Predisposing Factors :Pregnacy / Puerperium / Malignancy / Immobilisation /Unknown Cause

Immobilisation : Duration :

Cause : Surgery / Trauma /Post Partum

Chest Pain Duration :

Character :

Dyspnea :

Haemoptysis :

Blood Investigations :

Doppler- Region :

Complete / Parial Occlusion:

Treatment : Medical / Surgical

Outcome : Improved / Not Improved

Complications of Treatment:

Complications of Dvt :

MASTER CHART

SL.NO	NAME	AGE	SEX	PREDISPOSING FACTOR	REGION	MANAGEMENT	OUTCOME	COMPLICATION
1	SATHYA	25	FEMALE	POSTPARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
2	RAJAMMAL	55	FEMALE	MALIGNANCY	LEFT ILIOFEMORAL	M	IMPROVED	NO
3	BOOPATHY	36	FEMALE	POST SURGERY	LEFT ILIOFEMORAL	M	IMPROVED	NO
4	KUNDHAIVAIDEVI	31	FEMALE	POSTPARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
5	RAJENDRAN	62	MALE	POST TRAUMA IMMOBILISATION	LEFT ILIOFEMORAL	M	IMPROVED	NO
6	SIVAKUMAR	38	MALE	POST TRAUMA IMMOBILISATION	LEFT ILIOFEMORAL	M	IMPROVED	NO
7	JEYALAKSHMI	47	FEMALE	POST SURGERY	LEFT ILIAC	M	IMPROVED	NO
8	RAJASEKAR	52	MALE	POST SURGERY	LEFT ILIOFEMORAL	M	IMPROVED	NO
9	ELAYARAJA	30	MALE	POST TRAUMA IMMOBILISATION	RIGHT ILIOFEMORAL	M	IMPROVED	NO
10	KALAIMANI	33	FEMALE	POST SURGERY	LEFT ILIOFEMORAL	M	IMPROVED	NO
11	CHITRA	30	FEMALE	POST SURGERY	RIGHT ILIOFEMORALPOPLITEAL	M	IMPROVED	NO
12	SUMATHI	38	FEMALE	POST SURGERY	LEFT ILIOFEMORAL	M	NOT IMPROVED	VARICOSE VEIN
13	KAVITHA	34	FEMALE	POST SURGERY	LEFT ILIOFEMORAL	CDT	IMPROVED	NO
14	SHANTHI	43	FEMALE	MALIGNANCY	RIGHT ILIOFEMORAL	M	IMPROVED	NO
15	RUSSIYA	55	FEMALE	MALIGNANCY	LEFT ILIOFEMORAL	CDT	IMPROVED	NO
16	SOORYA	25	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
17	PUNNIYAMMAL	65	FEMALE	POST SURGERY	BILATERAL ILIAC	M	IMPROVED	NO
18	PARAMASIVAM	35	MALE	POST TRAUMA IMMOBILISATION	LEFT ILIOFEMORAL	M	NOT IMPROVED	POST THROMBOTIC ULCER
19	VIVEK	22	MALE	UNKNOWN	LEFT ILIOFEMORAL	M	IMPROVED	NO

SL.NO	NAME	AGE	SEX	PREDISPOSING FACTOR	REGION	MANAGEMENT	OUTCOME	COMPLICATION
20	THANGAPPA	45	MALE	POST TRAUMA IMMOBILISATION	RIGHT ILIOFEMORAL	M	IMPROVED	NO
21	DHAMAYANTHI	22	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	CDT	IMPROVED	NO
22	VIJAYA	22	FEMALE	POST TRAUMA IMMOBILISATION	LEFT ILIOFEMORAL	M	IMPROVED	NO
23	DHANASEKAR	37	MALE	POST SURGERY	LEFT POPLITEAL	M	IMPROVED	NO
24	MEENA	31	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
25	KAVERI	30	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
26	SUBRAMANIAN	51	MALE	POST SURGERY	LEFT ILIOFEMORAL	M	NOT IMPROVED	PULMONARY EMBOLISM
27	VASUDEVAN	42	MALE	POST TRAUMA IMMOBILISATION	RIGHT ILIOFEMORAL	M	IMPROVED	NO
28	SUMATHY	27	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	CDT	IMPROVED	NO
29	JEYALAKSHMI	26	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
30	JEYARAMAN	65	MALE	MALIGNANCY	RIGHR ILIAC	M	IMPROVED	NO
31	VEERAIYIAN	38	MALE	UNKNOWN	LEFT ILIOFEMORAL	M	IMPROVED	NO
32	SELVI	20	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	CDT	IMPROVED	NO
33	SUGANTHI	26	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
34	MUTHU	50	FEMALE	MALIGNANCY	BILATERAL ILIAC	M	IMPROVED	NO
35	KATHIRVEL	35	MALE	POST SURGERY	LEFT ILIOFEMORAL	M	NOT IMPROVED	RECURRENCE
36	BABY	36	FEMALE	POST SURGERY	LEFT ILIOFEMORAL	M	IMPROVED	NO
37	SENTHAMIL SELVI	32	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
38	SHARMILA	30	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
39	POOVANUAMMAL	45	FEMALE	POST SURGERY	LEFT ILIOFEMORAL	M	IMPROVED	NO
40	SARAVANAN	32	MALE	MALIGNANCY	IVC	M	IMPROVED	NO
41	DHANAM	23	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	CDT	IMPROVED	NO
42	RAJINI	42	MALE	POST SURGERY	LEFT ILIOFEMORAL	CDT	IMPROVED	NO
43	VEMKATESAN	45	MALE	POST SURGERY	LEFT ILIOFEMORAL	M	IMPROVED	NO
44	ANJALAI	24	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
45	DHURVA	26	FEMALE	POST PARTUM	RIGHT ILIOFEMORAL	M	IMPROVED	NO

SL.NO	NAME	AGE	SEX	PREDISPOSING FACTOR	REGION	MANAGEMENT	OUTCOME	COMPLICATION
46	KANNIAYAN	51	MALE	POST TRAUMA IMMOBILISATION	LEFT ILIAC	M	IMPROVED	NO
47	MANI	42	MALE	POST TRAUMA IMMOBILISATION	LEFT ILIOFEMORAL	M	IMPROVED	NO
48	SHRUTHI	28	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
49	MANI	28	MALE	UNKNOWN	LEFT ILIOFEMORAL	M	IMPROVED	NO
50	TAMILARASI	47	FEMALE	POST SURGERY	LEFT ILIOFEMORAL	M	NOT IMPROVED	RECURRENCE
51	SWATHY	23	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
52	RAJESHWARI	28	FEMALE	POST PARTUM	RIGHT ILIOFEMORAL	M	IMPROVED	NO
53	SAKTHI	48	FEMALE	POST SURGERY	LEFT ILIOFEMORAL POPLITEAL	M	IMPROVED	NO
54	CHITHRA	30	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	CDT	IMPROVED	NO
55	POORNIMA	28	FEMALE	POST TRAUMA IMMOBILISATION	LEFT ILIOFEMORAL	M	IMPROVED	NO
56	SUNDHARAM	40	MALE	POST TRAUMA IMMOBILISATION	LEFT ILIOFEMORAL	M	IMPROVED	NO
57	MARYSTELLA	25	FEMALE	POST PARTUM	RIGHT ILIOFEMORAL	M	IMPROVED	NO
58	AYISA BANU	25	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
59	JEEVA	43	FEMALE	POST SURGERY	LEFT ILIOFEMORAL	M	NOT IMPROVED	POST THROMBOTIC ULCER
60	MARIYA FRANCIS	55	FEMALE	MALIGNANCY	IVC	M	IMPROVED	NO
61	DHANALAKSHMI	29	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
62	THANIYAARASI	27	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
63	THIYAGARAJAN	50	MALE	MALIGNANCY	BILATERAL ILIAC	M	IMPROVED	NO
64	SEETHA	31	FEMALE	POST TRAUMA IMMOBILISATION	RIGHT ILIOFEMORAL	M	IMPROVED	NO
65	RAVI	49	MALE	POST TRAUMA IMMOBILISATION	LEFT ILIOFEMORAL	CDT	IMPROVED	NO
66	SAVEETHA	24	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	CDT	IMPROVED	NO
67	NATARAJAN	45	MALE	POST SURGERY	LEFT ILIOFEMORAL	M	IMPROVED	NO
68	MANOHARAN	40	MALE	POST SURGERY	LEFT ILIOFEMORAL	M	IMPROVED	NO
69	KUMAR	25	MALE	UNKNOWN	LEFT ILIOFEMORAL	CDT	IMPROVED	NO

SL.NO	NAME	AGE	SEX	PREDISPOSING FACTOR	REGION	MANAGEMENT	OUTCOME	COMPLICATION
70	JAMMIAMMAL	55	FEMALE	MALIGNANCY	RIGHT ILIOFEMORAL	M	IMPROVED	NO
71	SELVI	28	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
72	DHARANI	24	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
73	GOMATHY	22	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
74	NEELAMMAL	30	FEMALE	POST SURGERY	LEFT ILIOFEMORAL	CDT	IMPROVED	NO
75	MENNATCHI	32	FEMALE	POST SURGERY	LEFT ILIOFEMORAL	M	IMPROVED	NO
76	RAJA LAKSHMI	48	FEMALE	MALIGNANCY	BILATERAL ILIAC	M	IMPROVED	NO
77	THAMARAI	34	FEMALE	POST TRAUMA IMMOBILISATION	RIGHT ILIOFEMORAL	M	IMPROVED	NO
78	YOGA LAKSHMI	23	FEMALE	POST PARTUM	RIGHT ILIOFEMORAL	M	IMPROVED	NO
79	KANIMOZHI	23	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
80	ASWINI	27	FEMALE	POST SURGERY	LEFT ILIOFEMORAL	CDT	IMPROVED	NO
81	REVATHY	24	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
82	ANJAMMAL	65	FEMALE	POST SURGERY	LEFT ILIOFEMORAL	M	NOT IMPROVED	POST THROMBOTIC ULCER
83	SEBASTEIN	55	MALE	POST TRAUMA IMMOBILISATION	BILATERAL ILIAC	M	NOT IMPROVED	RECURRENCE
84	MUTHULAKSHMI	32	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
85	MARIMUTHU	65	MALE	MALIGNANCY	IVC	M	IMPROVED	NO
86	VARATHA RAJAN	55	MALE	POST TRAUMA IMMOBILISATION	LEFT ILIOFEMORAL	M	IMPROVED	NO
87	RAMACHANDRAN	42	MALE	POST SURGERY	LEFT ILIOFEMORAL POPLITEAL	M	IMPROVED	NO
88	MUNI	23	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
89	BEGUM	35	FEMALE	POST SURGERY	LEFT ILIOFEMORAL	M	IMPROVED	NO
90	RANI	33	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
91	DHIVYA	25	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
92	KALAYANA SUNDARAM	30	MALE	POST TRAUMA IMMOBILISATION	RIGHT ILIOFEMORAL	M	IMPROVED	NO
93	VANITHA	32	FEMALE	POST SURGERY	LEFT ILIOFEMORAL	M	NOT IMPROVED	POST THROMBOTIC ULCER

SL.NO	NAME	AGE	SEX	PREDISPOSING FACTOR	REGION	MANAGEMENT	OUTCOME	COMPLICATION
94	ANUSUYA	23	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	CDT	IMPROVED	NO
95	PRAKASH	24	MALE	POST TRAUMA IMMOBILISATION	LEFT ILIOFEMORAL	M	IMPROVED	NO
96	AYISABANU	24	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
97	SANGEETHA	23	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	M	IMPROVED	NO
98	BHARATHI	28	FEMALE	POST PARTUM	RIGHT ILIOFEMORAL	CDT	IMPROVED	NO
99	MOORTHY	65	MALE	MALIGNANCY	BILATERAL ILIAC	M	NOT IMPROVED	POST THROMBOTIC ULCER
100	ABIRAMI	30	FEMALE	POST PARTUM	LEFT ILIOFEMORAL	CDT	IMPROVED	NO