A STUDY ON THE EFFICACY OF WELLS CRITERIA

FOR DIAGNOSING DEEP VEIN THROMBOSIS

A DISSERTATION SUBMITTED TO

THE TAMILNADU DR.MGR MEDICAL UNIVERSITY

In partial fulfillment of the regulations for the award of the

Degree of M.S (GENERAL SURGERY)

BRANCH-1

DEPARTMENT OF GENERAL SURGERY

STANLEY MEDICAL COLLEGE AND HOSPITAL TAMILNADU
DR.MGR MEDICAL UNIVERSITY,CHENNAI

MAY 2020

CERTIFICATE BY THE INSTITUTION

This is to certify that dissertation **"A STUDY ON THE EFFICACY OF WELLS CRITERIA FOR DIAGNOSING DEEP VEIN THROMBOSIS"** is a bonafide record of work done by **Dr.PRAVEENRAJU BALA** in the Department of General Surgery, Stanley Medical College, Chennai, during his Post Graduate Coursefrom MAY 2017- MAY 2020. This is submitted in partial fulfillment for the award of **M.S. DEGREE EXAMINATION-BRANCH I (GENERAL SURGERY)** to be held in May 2020 under the **Tamilnadu DR.M.G.R. Medical University, Chennai.**

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DECLARATION

Dr. PRAVEENRAJU BALA solemnly declare that this dissertation titled "**A STUDY ON THE EFFICACY OF WELLS CRITERIA FOR DIAGNOSING DEEP VEIN THROMBOSIS" ,** is a bonafide work done by me in the department of general surgery, Govt. Stanley Medical College and Hospital, Chennai under the supervision of **Prof. Dr.T.SIVAKUMAR,M.S.** This dissertation issubmitted to the Tamilnadu Dr MGR Medical university, Chennai in partial fulfillment of the university regulations for the awardofM.S,degree (General Surgery), branch – 1 examination to be held in May 2020.

DATE:

PLACE:

Dr. PRAVEERAJU BALA

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I am grateful to the Dean ***Prof. Dr.R. SHANTHI MALAR, M.D., D.A.,***for permitting me to conduct the study and use resources of the college.I consider it a privilege to have done this study under the supervision of my beloved professor and head of the department ***Prof. Dr. T.SIVAKUMAR*** **,M.S.**, who has been a source of constant inspiration and encouragement toaccomplish this work.I am sincerely thankful to my guides **Prof.** ***Dr.T.SIVAKUMAR, M.S.,*** for their immense support in completing mywork.I express my deepest sense of thankfulness to my assistant professors***Dr. G.CHANDRASEKAR M.S., Dr. D.S.JIM JEBAKUMAR*** **M.S.,**for theirvaluable inputs and constant encouragement, without which this dissertation could not have been completed. I express my sincere thanks to my fellow post graduates, my beloved senior and junior colleagues for their support and help in completing this dissertation .It is my earnest duty to thank my family without whom accomplishing this task would have been impossible. I am extremely thankful to my patients who consented and participated to make this study possible.

CERTIFICATE BY GUIDE

This is to certify that this dissertation work titled "**A STUDY ON THE EFFICACY OF WELLS CRITERIA FOR DIAGNOSING DEEP VEIN THROMBOSIS"** of the candidate **DR.PRAVEENRAJU BALA** with registration number **221711064** for the award of **M.S General Surgery degree**. I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows 17**%** of plagiarism in the dissertation.

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PRINCIPAL INVESTIGATOR : DR. PRAVEENRAJU BALA,
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The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 11.01.2019 at the Council Hall, Stanley Medical College, Chennai-1 at 10am.

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

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A STUDY ON THE EFFECTIVENESS OF WELLS CRITERIA FOR DIAGNOSING DEEP VEIN THROMBOSIS

INDEX

A STUDY ON THE EFFECTIVENESS OF WELLS CRITERIA FOR DIAGNOSING DEEP VEIN THROMBOSIS

Keywords: Deep vein thrombosis, Wells criteria , Doppler ultrasound

Abstract:

A clinical criteria devised by Wells and co-workers to diagnose deep vein thrombosis namely "Wells Criteria" has been tested in our study. This criteria has been tested in various health care settings in New Zealand and has helped to reduce the dependence on radiological investigations .

Aim of the study:

To test the effectiveness of Wells criteria for diagnosing deep vein thrombosis.
To find the associated co-morbidities causing deep vein thrombosis.

Methods:

In our study the Wells criteria was tested using venous doppler ultrasound to confirm any case of deep vein thrombosis especially post operative cases suspected by Wells criteria. Time duration of the study was from December

2017 to august 2019.The study group included 50 cases of deep vein thrombosis as diagnosed by Wells criteria. Along with this the associated co morbid conditions in each case was also documented.

Results:

Fifty cases of suspected deep vein thrombosis in operated patients, as per Wells Criteria admitted to **Stanley Medical College Hospital** were subjected to the confirmatory test of doppler venous ultrasound out of which 46 were proven to have deep vein thrombosis which amounted to 92% of cases. Higher the score more was the probability of having deep vein thrombosis.

Out of the associated co morbidities analyzed surgery was found to be leading factor responsible which was amounting to 43% of cases analyzed. In this group alone 90% of the surgical cases were post caesarean section which signified the prothrombotic status of pregnant females who were in the age group of 20 and 30 years .

There was also a relation between blood group and deep vein thrombosis in which 47.8% of cases had A positive blood group indicating a higher level of Von Willebrand factor, possibly a mutant variant.

Conclusion:

We can conclude that the Wells criteria is a very efficient indicator in diagnosing deep vein thrombosis in our setup and to classify patients into various risk groups. Thus it would help to reduce delays in diagnosis and helps the clinician to start treatment early for DVT(deep vein thrombosis).

INTRODUCTION

Intraluminal clotting is one of the most important problems in the diseases of the peripheral veins. In 1929, Dencke pointed out that as a result of stasis, soft friable thrombi could develop in the veins of the calf and the foot distinguishion of thrombophlebitis (inflammatory) from phlebothrombosis(non-inflammatory) was done by Oschner and Debakey.

Phlebothrombosis is a disease which remains symptom free till complications like pulmonary embolism becomes obvious. It was considered that the efficient return of blood from the veins of the extremities depends on six factors:

1)The circulation time-directly related to the functional efficiency of the Heart.

2)The absence or elimination of mechanical obstruction.

3) The compressive action of muscle contraction on veins

4) The gravity.

5) The maintenance of normal negative pressure within the abdomen and thorax.

6)An efficient /normal peripheral arterial flow.

Phlebothrombosis may be due to combination of any of the above reasons. Phlebothrombosis of deep veins is clinically silent and manifests only as its complications.

Early recognition of the process of venous phlebothrombosis, its pathophysiology and availability of Doppler study has drastically reduced the incidence of Pulmonary emboli and its consequences by instituting early management.

The human vein is an organ that has been given importance since ancient times. Historians, religious clerics, physicians, politicians even Presidents have quoted about veins during various phases of time.

Rabindranath Tagore, had said “The same stream of life that runs through my veins night and day runs through the world and dances in rhythmic measures.”

Among the Greeks it was claimed that Asclepius the God Of Medicine drew blood from the Veins on the right side of Medusa’s head to bring forward untold healing powers , even giving life back to the dead.

Sir William Osler (1849-1919) ,a physician from Canada had once quoted “Varicose veins are due to improper selection of grandparents.”

As per traditional belief the vein was even believed to be a vein that ran straight from the heart to the fourth finger of the left hand. This was why the fourth finger was initially known as the ring finger where engagement rings are worn.

However all said and done man remains unwary and takes for granted the many magical functions that our veins do quietly in the background until they start malfunctioning.

Deep vein thrombosis (DVT) is a preventable condition that causes significant morbidity and mortality. Recent Statistics shows that complications from DVT kills more people than breast cancer and AIDS combined.

The DVT definition ,as given by national institute of health -- “A condition in which blood clot forms in a vein that is deep inside the body” .DVT is most commonly known to occur in the lower leg and thigh and if it get dislodged can reach the lungs causing pulmonary embolism (PE) which can be potentially fatal. An equally fatal condition can occur when blood clot reaches the brain causing a cerebral stroke.

DVT causing factors are known as
Virchow’s triad—
1) venous stasis ,
2) damage to endothelial wall and
3) state of hypercoagulability.

Other factors associated with DVT include traditional cardiovascular risk factors namely hypertension, obesity and diabetes.

In adults some of the contributing factors for DVT are cancer, old age, prolongedperiod of immobilization , stroke or paralysis , previous history of venousthromboembolism , pregnancy or puerperium, dehydration hormonal treatment, long air travel, congestive cardiac failure, varicose veins, , rheumatoid disease, inflammatory bowel disease and nephrotic syndrome

Though we had knowledge regarding DVT and its dreaded complications ,timing of starting prophylaxis especially in post operative patients due to the fear of profuse bleeding during the post operative period.

Current contraindications for initiation of prophylaxis against DVT (with low molecular weight heparin(LMWH) include intracranial bleed, internal bleeding from the raw wounds and operated sites bleed , spinal bleeding following spinal anesthesia and spinal injury due to hematoma.

While in hospital , DVT patients have so much sufferings. This leads to significant raise in the expense to the patient due to prolonged period of stay in the hospital and also ends in wastage of manpower and precious hospital resources.

The clinical diagnosis of DVT is not only challenging but may mimic other conditions as well, thereby making it risky to start empirical therapy with anticoagulants. Advanced imaging facilities such as Doppler venous ultrasound may not be always available in peripheral hospitals. Due to delay in the diagnosis, the treatment will also gets delayed resulting in wastage of precious time. Assessment based on these clinical signs alone may be inadequateas there are many instances in which clinical assessment with Homan's and Mose's sign have yielded false positives .

From the history and clinical signs we may develop a scoring system which is known as wells score which helps the doctors to arrive at a diagnosis.So that we can avoid unnecessary investigations saving time and money .

So my aim is to test the application of the Wells Score in our clinical set up and to see how effectively we can diagnose DVT .

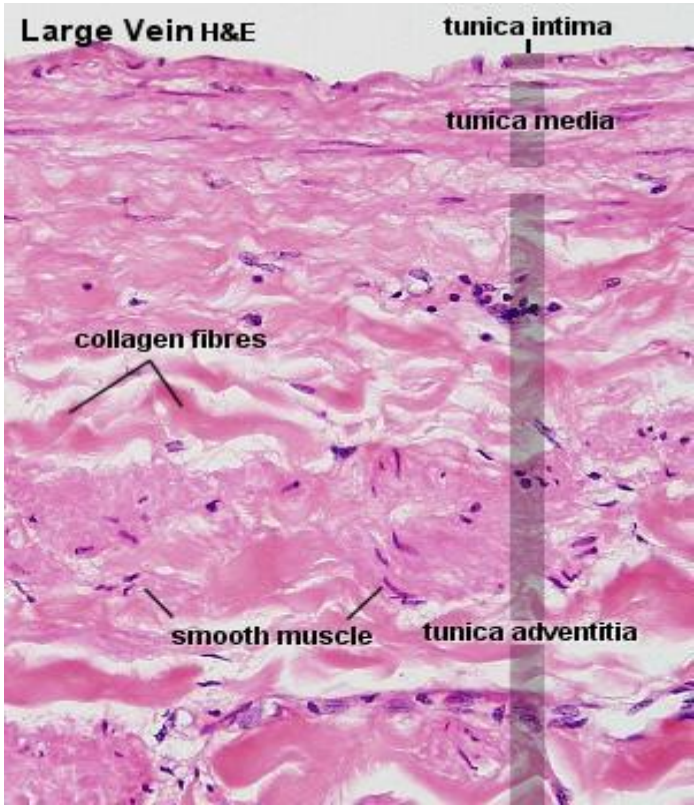
REVIEW OF LITERATURE

The Vein:

Venous histology and functions

Vein walls have less smooth muscle, elastin compared to their arterial counterparts. Intimal layer hasan endothelial cell layer which rests on basement membrane. Intima media is composed of elastin connective tissue and smooth muscle cells. Tunica adventitia contains adrenergic fibres especially in the cutaneous veins. Brainstem thermoregulatory centres and central sympathetic discharge can alter venous tone like other stimulus such as emotional stimuli, temperature changes , volume changes and pain.

The histologic features vary depending on caliber of veins . The venules, the smallest veins, 0.1 to 1 mm and contain mostly smooth muscle cells whereas the larger extremity contain few smooth muscle cells. These larger caliber veins has limited capacity of contractility comparing to thicker walled great saphenous vein . venous valves prevent retrograde flow; its failure of valvular incompetence leads to reflux and asscociated symptoms. Venous valves are most prevalent in the distal extremity, and if we proceed proximally , the valve number decreases to the point no valves are present in inferior vena cava and superior vena cava.



Venous system acts as capacitance of vascular tree. Since veins do not have large amount of elastin they can withstand large volume shifts with small changes in pressure. Veins normally has elliptical configuration till its capacitance limit is reached ,at which it assumes round configuration.

Calf muscle augment venous return by functioning as pump. Resting venous pressure in foot is the sum of residual kinetic energy minus the resistance in arterioles and precapillary sphincters. A pressure gradient is generated to right atrium of approximately 10 to 12 mm hg in supine position . whereas in upright position resting venous pressure of foot is reflection of hydrostatic pressure from the upright column of blood extending from right atrium to foot .

Fig.2

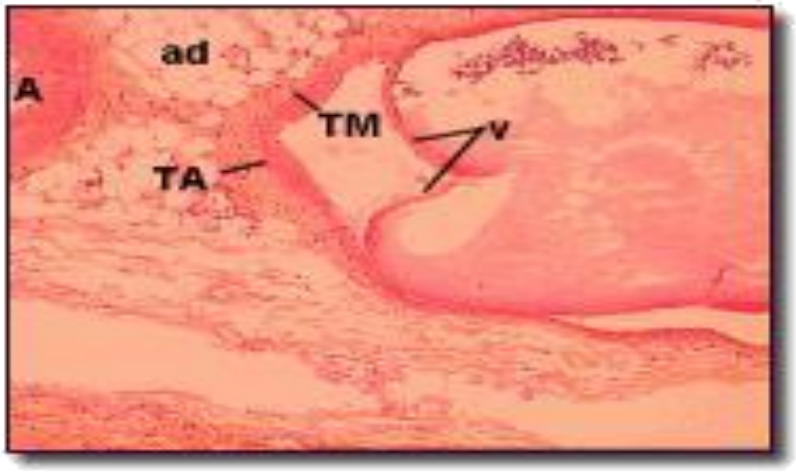
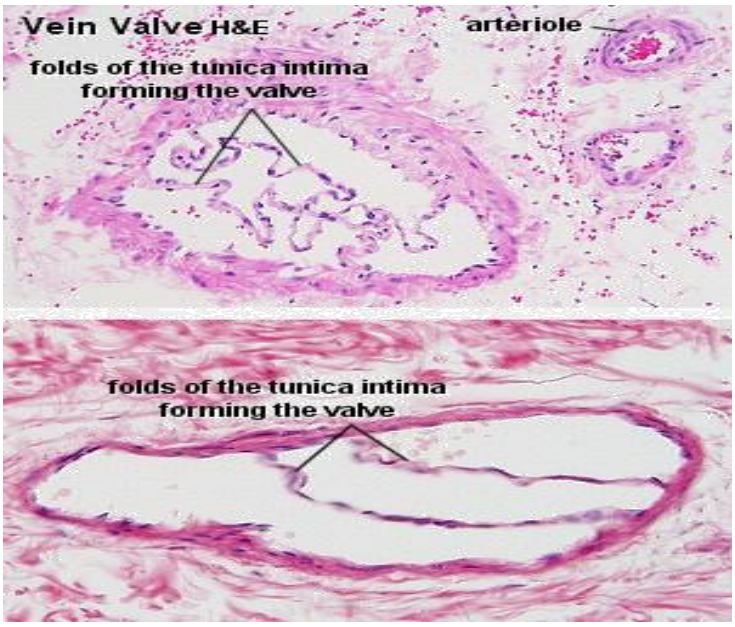


Fig. 19 Medium vein with valve.

The return of blood to the heart from lower extremity is by pumping action of calf muscle, compresses the gastrocnemius and soleal sinuses and propels the blood towards heart. The normal functioning valves in venous system prevent retrograde flow ; when these valves become incompetent, symptoms of venous insuffiency develops. The venous pressure of foot and ankle drop dramatically during calf muscle contraction. The pressure developing in muscle compartments duringexcerciase vary from 150 to 200 mm hg and when there is failure of perforating veins, these high pressure are transmitted to superficial system.

Fig. 3



Superficial venous system

The superficial veins of lower extremity form a network that connects superficial dorsal veins of foot and deep plantar veins. The dorsal venous arch , into which dorsal metatarsal veins empty, is continous with great saphenous vein medially and small saphenous vein laterally . The great saphenous vein is in close proximity to saphenous nerve , ascends anterior to medial malleolus , crosses and then medial to knee. It ascends in superficial compartment and empties into common femoral vein after entering fossa ovalis. Before its entry it receives medial and lateral accessory saphenous veins , as well as small tributaries from inguinal region, pudendal and abdominal region. The posterior arch vein drains the area around the medial malleolus while ascending in up the posterior medial aspect of calf , it receives medial perforating veins termed cockett's perforators, before joining the great saphenous vein at or below the knee. The small saphenous vein

arises from dorsal venous arch at the lateral aqspect of foot and posteriorly ascends to lateral malleolus, rising cephalad in midposterior calf, and then terminates into popliteal vein. Exact entry of small saphenous vein into popliteal vein is variable . thesural nerve is parallel to small saphenous vein .

Deep venous system

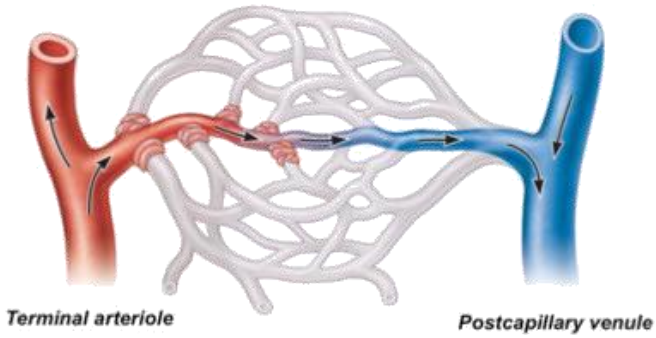
The plantar digital veins in foot empty into a network of metatarsal veins that compose the deep plantar venous arch . it continues into medial and lateral plantar venous arch, which then drains into posteriortibial veins. The dorsalispedis veins on dorsum of foot form the paired anterior tibialveinsat the ankle.

The paired posterior tibial veins, adjacent to the posterior tibial artery , run under the fascia of deep posterior compartment. These veins enter soleus and join popliteal vein. There are large venous sinuses within the soleus muscle- soleal sinuses- empty into posterior tibial andperoneal veins. Bilateral gastrocnemius veins empty into popliteal vein distal to point of entry of small saphenous vein into popliteal vein.

The popliteal vein enters a gap in adductor magnus at which point it is called the femoral vein . the femoral vein ascends and receives venous drainage from the profundafemoris vein or deep femoral vein, and after this confluence , it is common femoral vein . while common femoral vein crosses the inguinal ligament , it becomes external iliac vein.

Perforating venous system

They connect the superficial venous system to the deep venous system by penetrating the fascial layers of lower extremity. These perforators run in perpendicular fashion to axial veins . perforators vary in number ,range upto 100. The perforators enter at various points in leg - foot, medial and lateral calf, and mid and distal thigh. They are named as cockett'sperforators , which connect the posterior archaand posterior tibial veins, boyd's perforators, which connects the great saphenous and gastrecnemius veins, hunterian and dodd's perforators , which connects the great saphenous and superficial femoral veins. The perforator veins have important function. Their valve system aids in preventing reflux from deep to the superficial system, particularly during periods of standing and ambulation.



This flow is sometimes boosted up by other supportive back pressures such as the increased right atrial pressures of congestive heart failure.

There is no motive force involved which causes such efficient venous return. If all these factors are considered and put together the very mode of functioning of the venous system almost feels magical.

The veins of the lower extremity that are primarily involved in collecting blood are passive, and are thus thin-walled reservoirs that can be easily distended. Most of these veins are in the suprafascial compartment, which basically consists of only fatty and loose alveolar tissue and thus easily displaced . These veins in the suprafascial compartment can in turn dilate due to the nature of the surrounding tissue to accommodate large volumes of blood with very less increase in the back pressure, so in turn the quantity of blood passing within the venous system at any given point of time will vary by a factor of two or more without any significant alteration of the normal venous function.

The superficial venous system is represented by the veins in the suprafascial compartment. The flow from the collecting veins is via conduit veins whose walls are thicker and thus much less distensible.

These conduit veins are mostly in the subfascial compartment and are thus surrounded by dense connective tissue. As a result it is understandable that these subfascial veins belong to the deep venous system.

Categories of peripheral veins:

1)Suprafacial (superficial)

2)Perforating Veins

3)Subfascial Veins (Deep)

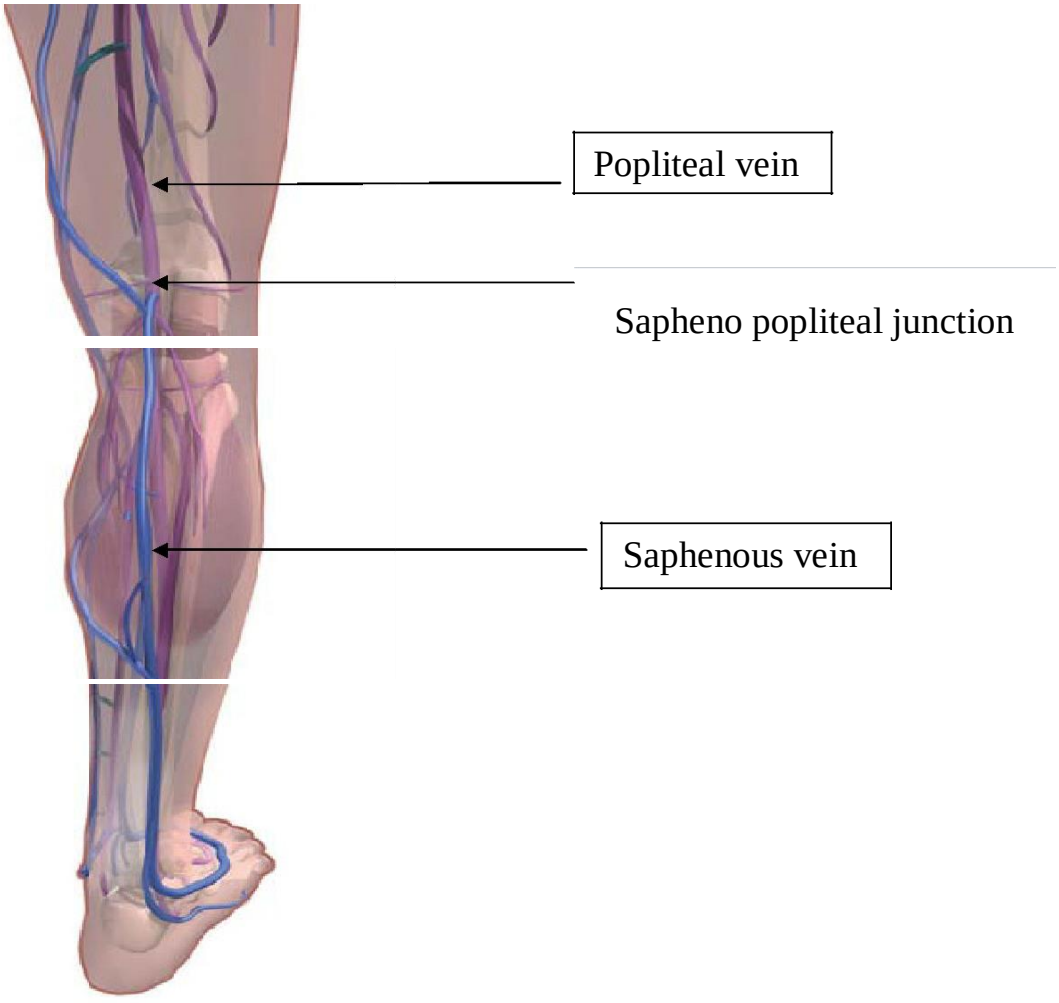
The Superficial (suprafascial) system of veins:

The suprafasical venous system consists of a web like network of veins which are interconnected at various places and intricately arranged and the veins that form this system are mostly unnamed . But there consist of a few superficial veins ,the position of which fairly remain constant and like the deep veins A few larger superficial veins are fairly constant in location; like the deep veins, these superficial veins serve like a passageway to channel blood centrally and

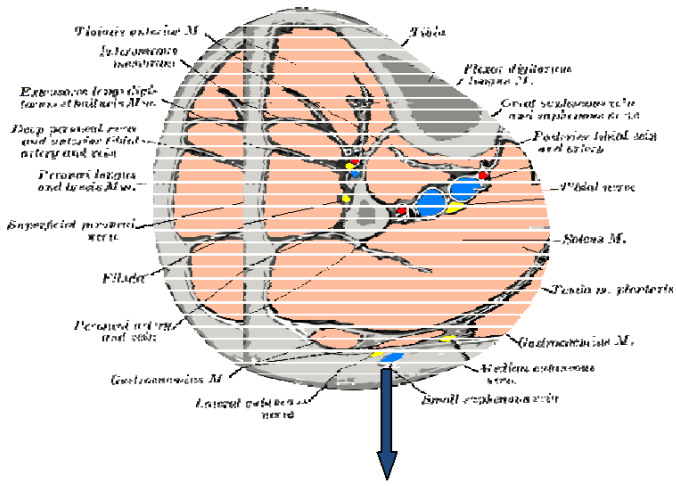
finally into the deep venous network. The most important superficial veins of the lower extremity are the short saphenous vein (SSV) that usually runs from the level of ankle to the knee and there is the great saphenous vein which runs from the medial aspect of the ankle to the groin.

The Small Saphenous vein

The origin of the small saphenous vein is in the lateral aspect of the foot. Its course is postero-lateral to the Achilles tendon in the lower aspect of the calf muscle. The small saphenous vein is usually situated directly superficial to the deep fascia over the midline as it reaches the upper calf, where it in turn enters the popliteal space between the two heads of the gastrocnemius muscle.



The small saphenous vei n joins the popliteal vein above the kneee joint in two-thirds or the cases and in the remaining one-third of cases, it joi ns with the other veins (mostly the GSV or the deep muscular veins of the thigh). In some of the cases, the SSV may have two or three different termination sites .

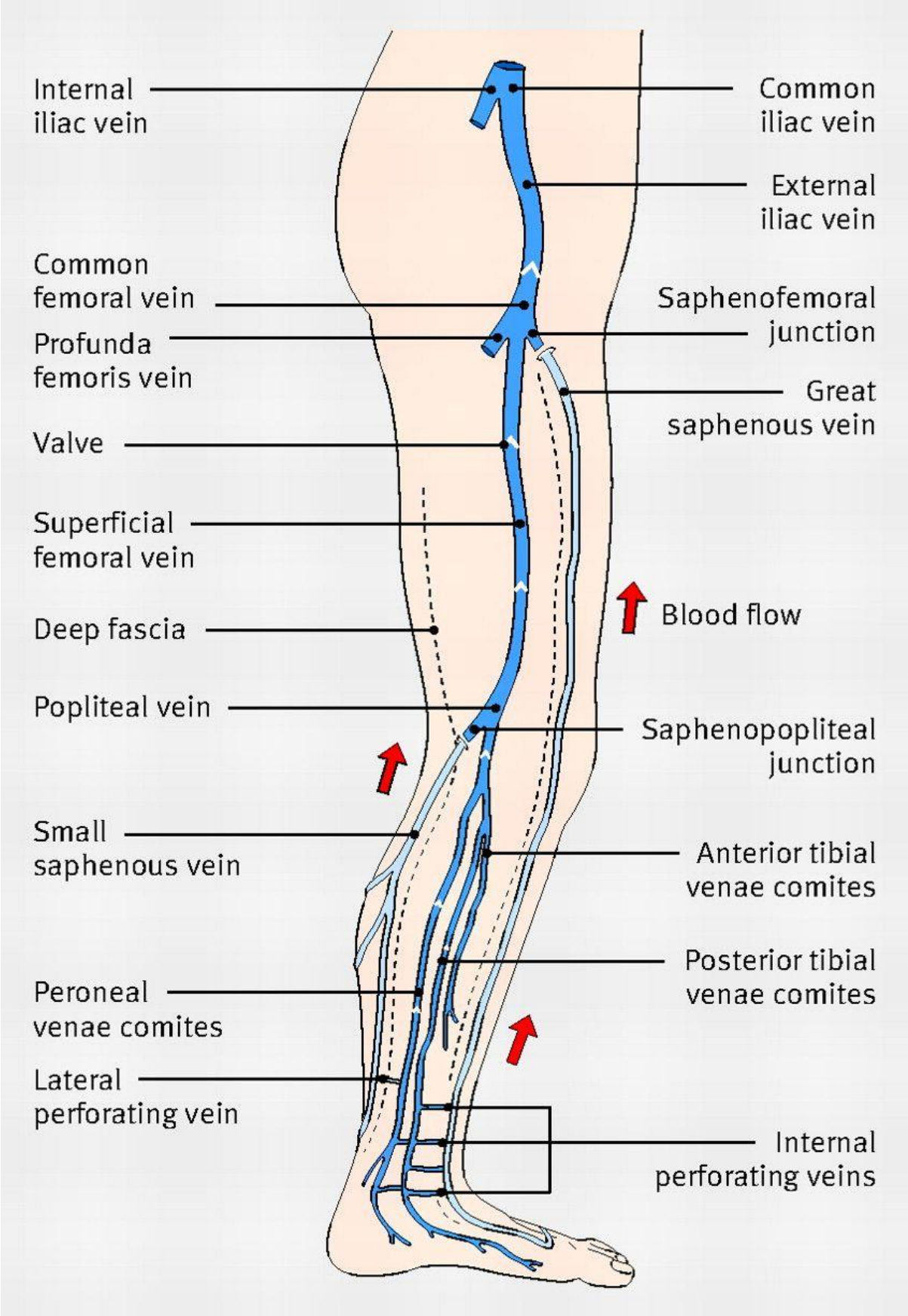


Position of small saphenous vein in the leg

The Great Saphenous Vei n (GSV)

The GSV arises from the medial aspect of the foot and passes o nto the anterior part of the medial malleol us followed by which it, then crosses the medial aspect of tibia posteriorly to rise medially across the knee joint. Above the level of the knee, it continues antero medially, superficial to the deep fascia,

Lower limb vein anatomy



passes through the foramen ovale joining the common femoral vein at the groin crease at a site termed the saphenofemoral junction (SFJ).

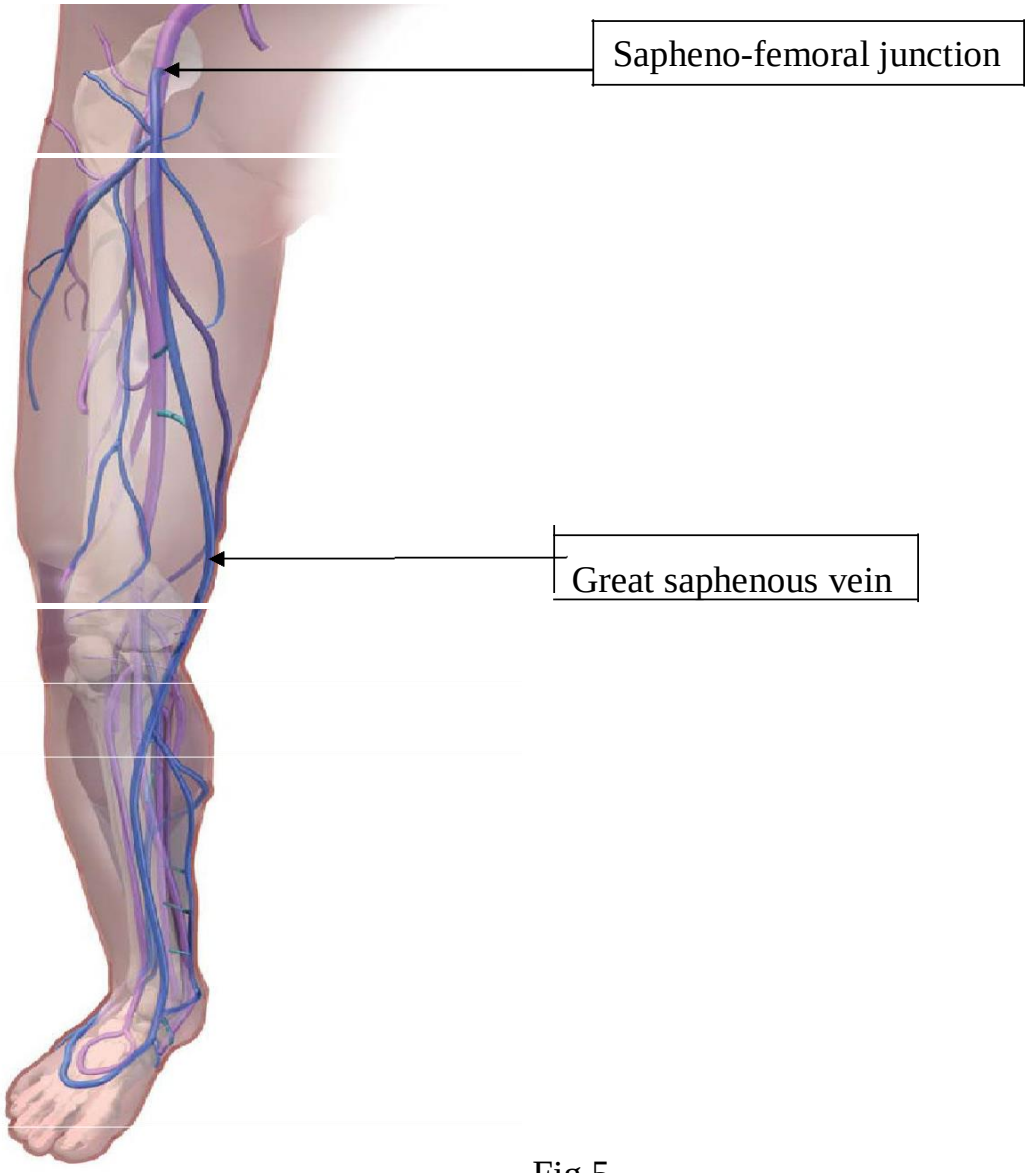


Fig.5

Large tributaries of the GSV are usually mistaken for the main trunk. Majority of patients have at least two major tributaries below the knee ie .the anterior and posterior tributaries, the latter known as the posterior arch vein.

Along with this there are also two above the knee which are the anterior circumflex and posterior circumflex tributaries. They usually drain into the GSV distal to the SFJ; but they are also likely to have a direct connection to the femoral vein. Additionally there are three pelvic veins that commonly drain into the GSV at the SFJ: the superficial inferior epigastric, the superficial external pudendal, and the superficial circumflex iliac veins.

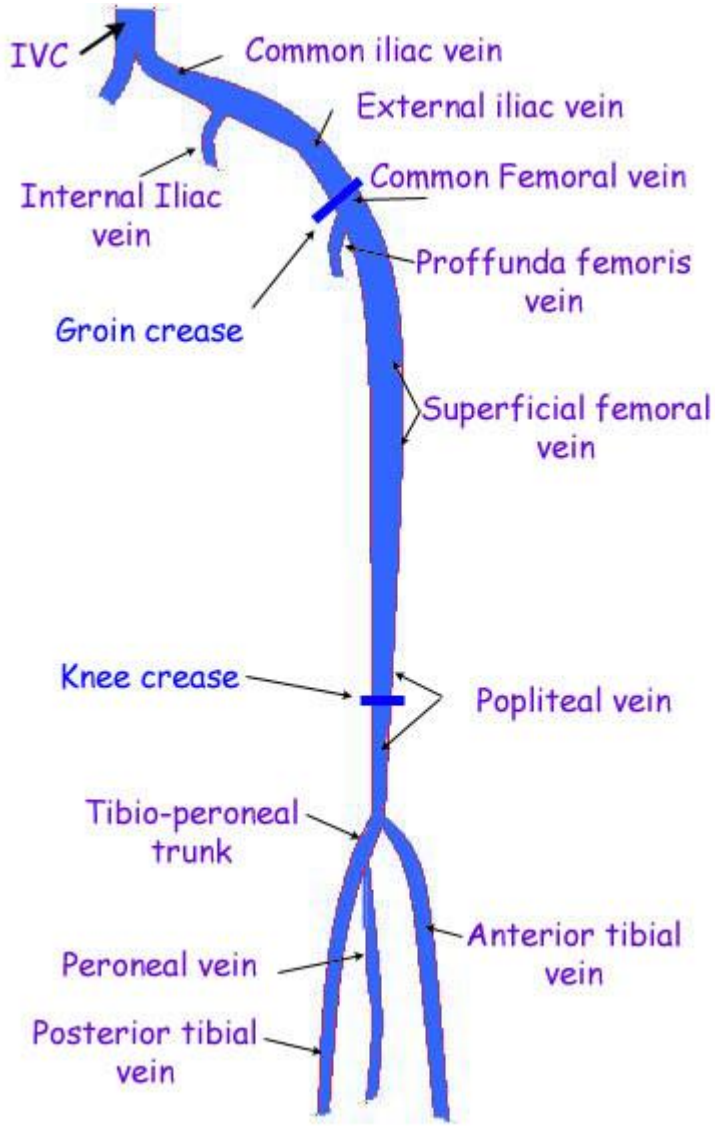
Many cases can also have a duplicated main GSV trunk in the thigh and some may have three or even four veins, known as anterior or posterior accessory veins, which parallel the main GSV trunk and either reconnect with it usually just above or below the knee or traverse more superficially in the distal thigh.

Perforating veins

Most of the veins in the superficial compartment collect and deliver their blood into the great as well as the small saphenous veins, following which these in turn supply most of their blood into the deep venous system via the SFJ and the saphenopopliteal junction (SPJ).

However, there are many alternate channels other than the SPJ and SFJ from the superficial system to the deep system. There are a variable number of perforating veins that connect the superficial veins through various openings in the deep fascia to join directly with the deep veins of the calf or thigh.

Sites for DVT



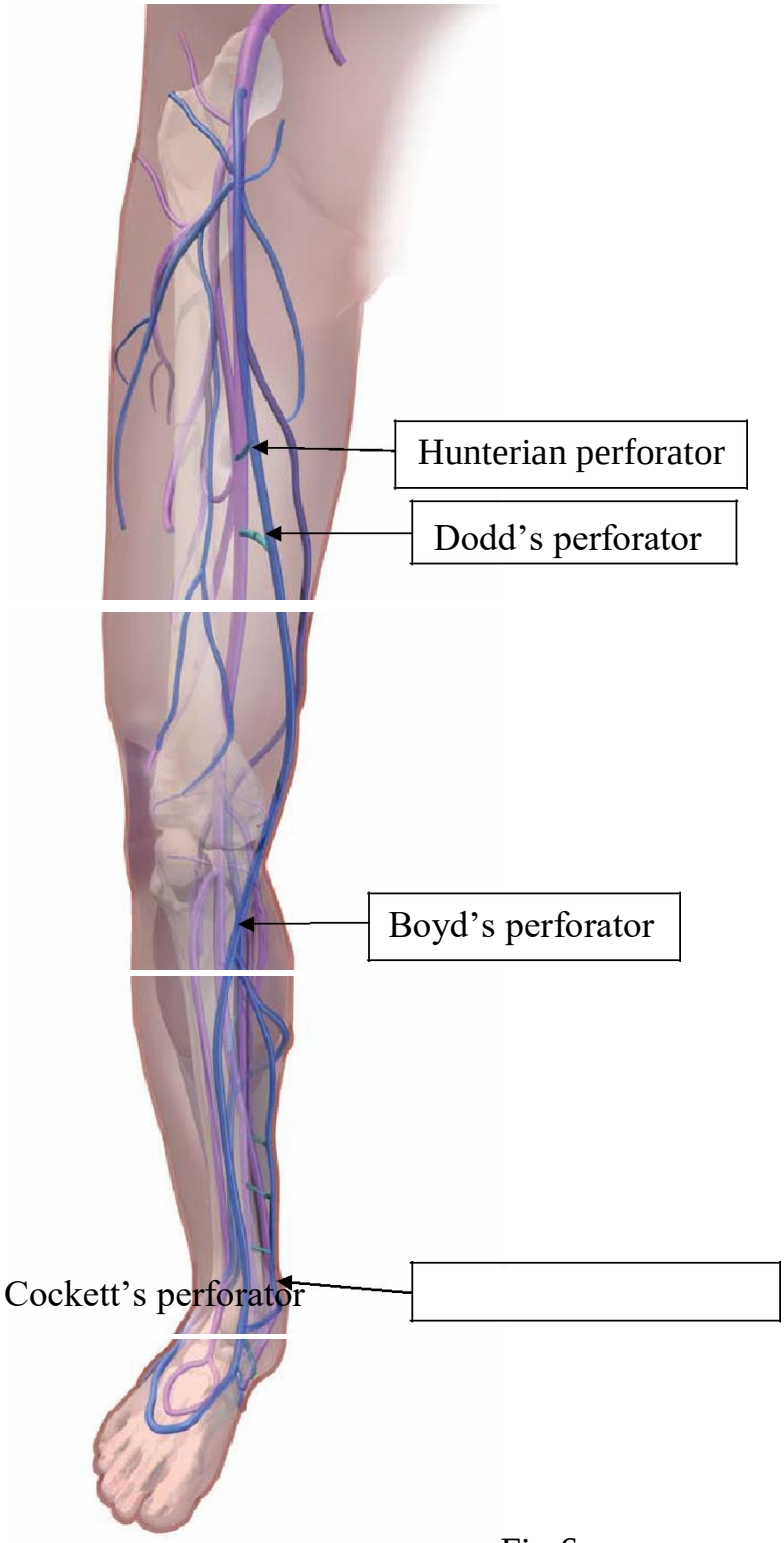

Hunterian perforator
Dodd’s perforator
Boyd’s perforator
Cockett’s perforator

Fig.6

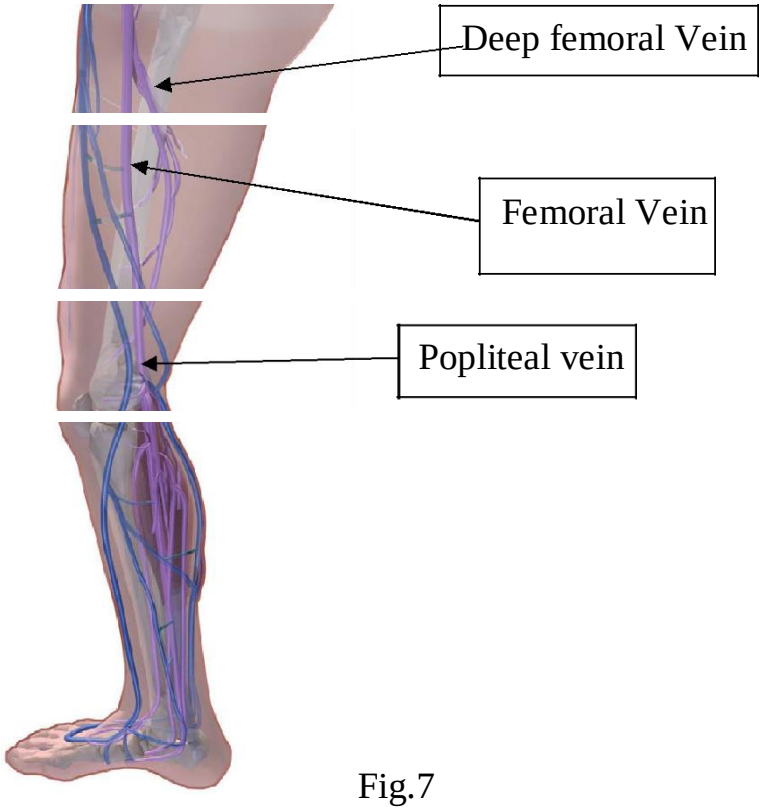


Fig.7

Deep veins of the calf

In the lower leg, there are three pairs of deep veins : the anterior tibial vein (ATV), draining the dorsum of the foot; the posterior tibial vein (PTV), which drains the medial aspect of the foot. Finally there is the peroneal vein, which drains the lateral aspect of the foot.

From above the level of the ankle, the anterior tibial vein courses upward , anterolaterally to the interosseous membrane .The peroneal vein passes superiorly and posteriorly through the calf whereas the posterior tibial vein runs superiorly and posteromedially below the medial edge of the tibia.

Venous sinusoids inside the calf muscle unite to form the intramuscular venous plexi of gastrocnemius and soleus ,which in turn join the peroneal vein at the 28

level of mid calf. In most individuals, there are six named deep veins below the knee each being a pair of veins which flank an artery of the same name.There are four anterior and four posterior tibial veins fuse with the two peroneal veins to ultimately form a single large popliteal vein just below the knee.

Deep veins of the thigh

The popliteal vein runs behind the knee proximally and then passes anteromedially through the adductor canal in the distal aspect of the thigh, from where it is called the femoral vein. The popliteal vein and the femoral vein are continuation the same vein and thus this is the longest as well as the largest deep vein of the lower extremity.

The deep femoral vein (DFV) originates in the lateral thigh within the deep

muscles tributaries is a short and stubby vein and may occasionally communicate with the popliteal vein in up to 10% of patients.

The common femoral vein is formed in the proximal thigh by the union of femoral vein and deep femoral vein. This vein in turn passes upward above the level of the groin crease to become the iliac vein.

Venous valves-

The presence of valves is one of the most important clinical features of veins. These structures are delicate but extremely strong at the same time and lies at

the base part of a segment of vein which expands to form a venous sinus. Due to this arrangement the valves are able to open up properly without coming into contact with the wall. This feature allows rapid closure when flow begins to reverse.

The anterior tibial vein has approximately 9 to 11 valves followed by 9 to 19 in the posterior tibial, peroneal having 7, 1 in the popliteal and 3 valves in the superficial femoral vein . In two thirds of the femoral veins there is a valve present at the upper end at 1 cm proximity to the inguinal ligament. One-fourth of the external iliac veins have valves. No valves are usually present in the common iliac veins.

The number of valves present in the superficial veins are much less, the greater and lesser saphenous veins having around 7 to 9 valves. Venules with a diameter of 0.15 mm have approximately 56 valves.

In most of the areas in the arms and legs valve cusps are oriented in such a manner so as to direct blood flow towards the vena cava and prevent reflux down into the lower extremity.

Usually valves in the perforating veins allow blood to flow from the superficial to the deep venous system , but in the case of the foot the valves also allow flow from the deep to the superficial system.

Venous Circulation [27]

Blood flow that occurs through the blood vessels especially the veins is mainly due to the pumping action of the heart. Flow of blood through the veins is also aided by the rise in the pressure in the thorax during inspiratory phase of respiration , the beating heart as well as compression of the veins by contraction of skeletal muscles which constitutes the muscle pump.

Venous flow and pressure

The normal pressure inside the venules ranges from 12-18 mm Hg. As the caliber of the vein increases the pressure falls steadily to about 5.5 mm Hg inside the great veins that lie outside the thorax.

The intraluminal pressure of the great veins before they enter into the right atrium (central venous pressure) comes to an average of 4.6 mm Hg , but also fluctuates with respiratory movement and heat beat.

Peripheral venous pressure like the pressure in the arteries is altered by gravity. It increases by 0.77 mm Hg for every centimeter below the level of right atrium and decreased by a similar amount for every centimeter above the level of right atrium the pressure is measured.

Thus if the influence of gravity is measured on arterial versus venous pressure there is a greater effect on venous pressure. When blood flow occurs from the venules into the large veins , the average velocity gets increased as the total 31

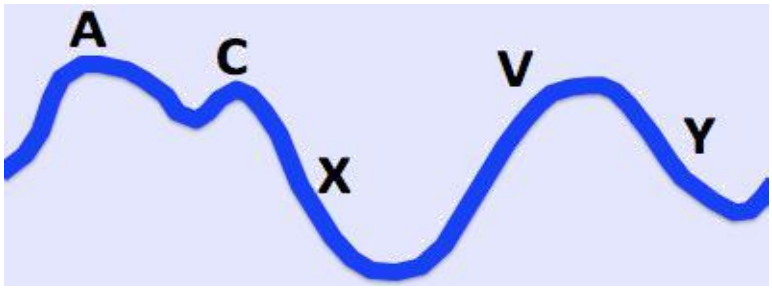
cross-sectional area of the blood vessels decreases .In most of the great veins , the velocity by which the blood flows comes to about one fourth of that in the aorta which averages to 10 cm/s.

Thoracic pump:

While inspiring the fall in intrapleural pressure that occurs is from -2.5 to -6 mm Hg. This fall in pressure is in turn transmitted to the great venous system and to atria in a lesser extent. As a result the fluctuation in central venous pressure ranges from 2mm Hg during inspiration and 6 mm Hg during expiration. This drop in venous pressure during the inspiratory phase aids venous return. Rise in intra abdominal pressure occurs when the diaphragm descends down while inspiring and thus blood is squeezed towards the heart since venous valves prevent backflow into the leg veins.

Effects of heartbeat:

When the variations in atrial pressure are transmitted into the great veins it produces the a,c and v waves of the venous pressure-pulse curve.



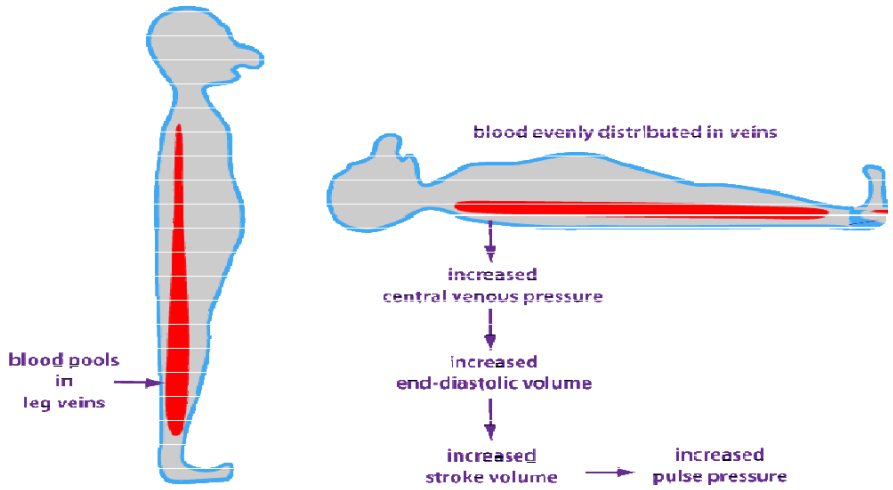
Fig,8

During the ejection phase of ventricular systole the atrial pressure drops sharply because of the pulling down of atrioventricular valves thus increasing the capacity of the atria. As a result of this blood is sucked from the atria from the great veins. This sucking of blood into the atria during systolic phase is mainly responsible for venous return especially when the heart rate is high. Venous flow is pulsatile when in close proximity to the heart. Two periods of peak flow are appreciable especially when the heart rate is slow. One period occurs due to the pulling down of atrio-ventricular valves during ventricular systole and the other during early diastole as part of the rapid filling phase of the ventricle.

Muscle pump

There are various veins in the lower limb which are surrounded by skeletal muscles. When these muscles are put into contraction during any activity this compresses the veins. Veins are also compressed by the pulsations of the adjacent arteries. Blood as a result moves towards the heart due to the valves in the veins that prevent flow in the opposite direction. When standing immobile the full effect of gravity manifests and the venous pressure at the ankle level

becomes 85-90 mm Hg. This results in pooling of blood which reduces venous return as a result of which cardiac output reduces and if this process continues fainting can occur.



.Fig.9

Venous pressure in the legs is reduced to less than 30 mm Hg as a result of contraction of leg muscle s in a regular rhythm and which results in propulsion of blood towards the heart. This movement of blood towards the heart is decreased in varicose veins due to the incompetent venous valves. The patients develop stasis along with ankle edema, When this condition persists for a long duration.

In spite of the incompetent valves flow of blood can still continue towards the heart due to muscle contractions and also since the resistance offered by the

larger veins lying in the direction of the heart is less than the resistance offered by the small vessels which lie away from the heart.

Venous pressure in the head

In the standing position the venous pressure in those parts of the body above the level of the heart is decreased by the force of gravity. At a point above which the venous pressure is close to zero, the neck veins collapse. However the dural sinuses cannot collapse since they have rigid walls. Irrespective of the position whether standing or sitting, the pressure is always sub atmospheric. The quantum of negative pressure in thevein is proportional to the distance vertically above the level of the collapsed neck veins and can be as much as – 10 mm Hg in the superior sagittal sinus.

An important fact which must be remembered by neurosurgeons especially when neurosurgical procedures are performed with the patient in seated position. Air embolism occurs if one of the sinuses gets opened during a procedure.

Measuring Venous Pressure

Central venous pressure is measured by the insertion of a catheter into the great thoracic veins in a direct manner. In majority of the conditions peripheral venous pressure correlates well with central venous pressure. Peripheral venous pressure is measured by measured by inserting a needle attached to a manometer into a vein in the arm.

The peripheral vein should be kept in level with the right atrium which is a point about half the diameter of the chest from the back while being supine. The readings obtained as the dividing by 13.6 (the density of mercury).

As the distance from the heart along the veins increases the peripheral venous pressure also increases in comparison to central venous pressure. The mean pressure present in the antecubital vein is normally 7.1 mm Hg, as compared with a mean pressure of 4.6 mm Hg which is seen in the central veins.

An approximate estimate of central venous pressure can be measured without any equipment by checking the level to which jugular veins get distended when the subject lies with the head above the heart.

The vertical distance separating the right atrium and the area the vein collapses (which is the place where the pressure inside it is zero) is the level of venous pressure in mm of blood.

Factors that increase CVP include:

- Hypervolemia
- Tension pneumothorax
- forced exhalation
- Heart failure
- Pleural effusion
- Cardiac tamponade
- Decreased cardiac output
- Pulmonary Hypertension
- Pulmonary Embolism
- Mechanical ventilation and the application of positive end-expiratory pressure (PEEP)

Factors that decrease CVP include:

- Hypovolemia
- Deep inhalation
- Distributive shock

Venous dysfunction

Venous dysfunction results due to impairment of venous return for any reason, and can arise from abnormalities within the deep veins, superficial veins, or a combination. It may result from primary muscle pump malfunction, from thrombotic or nonthrombotic venous obstruction or as a result of venous valvular incompetence, which may be limited to a segment or can involve the entire length of the vein. The pressure is extremely low within the veins of the lower extremity immediately after ambulation. The arterial inflow is purely responsible for the inflow to the lower extremity veins. Up to 3-5 minutes of standing is required to fill up the normal venous system. Once entire venous system gets filled, the valves open up and venous pressure increases to a maximum which is exactly equal to the height of a standing column of venous blood starting from the right atrium to the foot. Under this condition there occurs an urge to move the legs, thus resulting in activation of the muscle pumps and thereby emptying the leg veins.

VENOUS DISORDERS

1) VARICOSE VEINS

Varicose veins basically refer to any tortuous, dilated and elongated vein which can be of any caliber.

It includes those conditions causing venous dilatation, the spectrum of which from minor telangiectasia all the way to severely tortuous and dilated varicose veins.

Telangiectasias consist of varicosities which are intradermal small and tend to be cosmetically unappealing but at the same time not symptomatic by themselves in any manner. Reticular veins are dilated veins in the subcutaneous layer that join the tributaries in the main axial or trunk veins.

Trunk veins are those veins with names, such as greater or lesser saphenous veins or their tributaries as well.

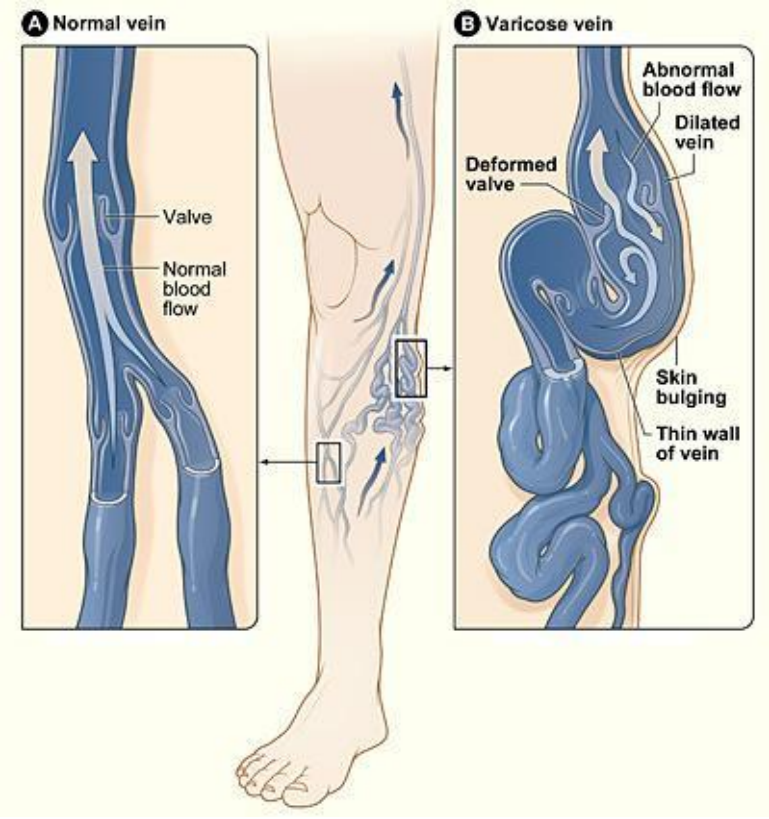


Fig.10

Chronic venous insufficiency towards the end can result in conditions that range from aching, pain, heaviness, and swelling associated with sitting or standing for long durations in the case of varicose veins which are symptomatic. In the other end it may cause severe lipodermatosclerosis along with edema and towards later stages it results in ulceration in those patients with severe chronic venous insufficiency.

Risk factors for varicose veins [4]:

Many risk factors in combination rather than one specific risk factor in general can predict the chances of a patient to develop varicose veins.

Heredity also has a major role to play in the development of varicose veins, without doubt.

Valvular insufficiency and dysfunction-

Gravitational force, hydrostatic force, female sex and hydrodynamic forces resulting from muscular contraction.

Hormonal factor- progestrone is responsible for valvular relaxation.

Pathogenesis of varicose veins:

Pathogenesis of varicose veins is based on the defects in strength and pliability of the venous wall.

Defects in the valves of the communicating veins which connect the deep to the superficial compartment may occur .

According to pressure studies there are two sources of venous hypertension. The first source is gravitational and results from the venous blood flowing in a direction distal to linear axial venous segments situated below. The weight of the blood column from the right atrium is referred to as hydrostatic pressure.

There is a dynamic component to the second source of venous hypertension. It results from the force of muscular contraction, which is usually maintained within the compartments of the leg. In case of failure of a perforating vein, high pressures which ranges from 150 to 200 mm Hg developing within intramuscular compartments during exercise are in turn transmitted directly to the superficial venous system. Since the sudden pressure transmitted directly it causes dilation and lengthening of the superficial veins. Further on there is increase in the distal valvular incompetence.

Capillary proliferation due to the widening of interendothelial cell pore is seen in the distal liposclerotic area and a rise in capillary permeability occurs. Leakage of principal osmotically active particle fibrinogen across the capillaries occurs.

The extravascular fibrin prevents the entry of oxygen and nutrients into the surrounding cells .However there is very little proof that exists for any actual abnormality for the delivery of oxygen into the tissues.

Release of proteolytic enzymes from the extravasated lymphocytes is another factor.

Venous thrombosis

It is a condition which can occur either in the superficial or deep venous systems in the leg. Simultaneous involvement of both systems does occur but usually in such cases it begins as a deep venous thrombosis which later on extends to the superficial venous system as well.

Two terms need to be well understood,In order to get an understanding of venous thrombosis :

Thrombophlebitis refers to the formation of a clot in a vein associated withinflammatory findings such as erythema, pain and tenderness . As a result of the inflammation the blood clot remains firmly adherent to the vessel wall and there are very few chances of it getting dislodged and going to the lungs.

*Phlebothrombosis*is the formation of a clot in the vein without any signs ofinflammation. Here the clot formation is usually asymptomatic but has more chance of getting dislodged and embolising.

Superficial thrombophlebitis-

Thrombophlebitis usually involves the superficial veins of the lower extremities in comparison to phlebothrombosis which mostly involves the deep veins. Varicose veins is also a prerequisite factor in superficial thrombophlebitis of the lower extremities but not an absolute prerequisite. Other contributing factors are venous wall trauma associated with venous catheters, I.V drug abuse, repeated venous punctures, use of strong IV solutions that produce inflammatory response.

Manifestations of thrombophlebitis-

Marked redness along vein

Dull, aching pain over affected area

Increased warmth over area of inflammation

Palpable cordlike structure

More immediate attention is required if edema, chills, high fever; suggests complications of inflammation.

In cases when thrombophlebitis extends above the level of the knee joint, anticoagulant therapy should be considered if conservative measures fail. The role of anticoagulant treatment is mainly for the purpose of preventing thrombembolism and not to treat the primary pathology

DEEP VENOUS THROMBOSIS [13]

Acute deep venous thrombosis (DVT) in hospitalized patients is one of the major causes of morbidity and mortality especially in post operative patients.

Even though its been a hundred and fifty years since Virchow proposed his triad which consists of endothelial injury, venous stasis and hypercoagulable state this still holds true even today . Although the final credit for the pathological triad in DVT has been granted to Rudolph

Virchow much work has been done in the same field by various physicians.

Wiseman in 1686 had proposed his version of the probable etiology of venous thrombosis which bears a resemblance to Virchows triad.

He had attributed the cause of venous thrombosis to (1) Thickening of blood (2) Coagulation of serum (3) Decreased venous blood flow by impingement of a tumor or any back pressure built up within the venous system. He also had described the increased incidence of venous thrombosis during pregnancy or any malignant conditions.

Van Swieten in 1705 described the increased incidence of clots during puerperium.

White in 1784 had published that phlegmasiaalbadolens is associated with thrombus that develops in the iliac or femoral veins. Phlebitis had been described by Hunter in the same year in his article "Observation of the inflammation of internal coat of veins"

Virchow upon his appointment in Charitehospital ,Berlin was under the direction of Robert Froriep (1804-1861) who appointed him with investigating the assertion given by French pathologist Cruveilhier who had claimed that phlebitis dominates all pathology. He proceeded with clinical and experimental investigations in the process of pursuing

Cruveilhiers statement. Virchow, in a series of 76 autopsies identified 18 cases of venous plugs out of which there were 11 cases of emboli in the pulmonary arteries. Based on this he coined the term emboli and reasoned that majority of the clots developed in the deep veins of the lower extremities, thereby contradicting the belief that thrombosis occurred in the lungs *de novo* due to inflammation in the veins. He substantiated his necropsy observations in a series of studies where he injected various compounds in to the venous circulation of dogs and by ultimately examining the characteristics of the material that was lodged in the pulmonary vasculature.

In conditions such as the anticoagulationabsense or even in the presence in anticoagulation inadequacies the thrombotic process which gets initiated in a venous segment can get propagated and thus involves even more proximal segments in the deep venous system thus causing edema, pain and immobility.

The most dreaded complications of acute DVT which is a potentially lethal condition is pulmonary embolism.

Chronic venous insufficiency can occur as a late consequence of DVT especially in the iliofemoral veins caused by dysfunction of valves along with the presence of luminal obstruction.

, to standardize the protocols to prevent or reduce the occurrence of DVT, and to thereby institute optimal treatment promptly to such cases we need a thorough understanding. All these steps are critical to reduce the incidence and morbidity caused by this unfortunately common condition.

Etiology –

The most common sites where venous thrombosis gets initiated are the soleal sinus. Stasis in the sinuses may contribute to activated platelets getting in contact with the endothelial cellular layer as well as procoagulant factors, thereby leading to deep venous thrombosis.

The Hypercoagulable State

Wiseman (1686) and Andral (1830) had preceded Virchow by suggesting that venous thrombosis was due to the increased coagulating tendency of the blood

Before the 1930's reliable factors that contributing to thrombosis by altering the blood composition werelacking . Although it was known that thrombosis could be precipitated by injection of certain snake venoms only few endogenous factors were known that caused a similar effect.

In 1901 Lotheisen put forward a theory that malignancy, pregnancy and chlorosis along with other factors may affect thrombosis. Following this increase in the levels of fibrinogen, globulin and calcium were also postulated to increase the agglutinability. Later on it was suggested that infection and obesity can potentiate coagulability .

Once any of these conditions favorable for DVT are identified, unless there are specific contraindications,a treatment regimen which consists of anticoagulation is started for life,

From the source being damaged tissues ,Tissue factor may be released after major surgeries into the blood stream in large amounts. Tissue factor is a very potent procoagulant. Increases in the platelet count, changes in coagulation cascade, adhesiveness and finally endogenous fibrinolytic activity all are result of physiologic stress such as any major surgery or trauma and all these put together are known to be associated with an increased risk of thrombosis.

Hypercoagulable States:

■ Factor V Leiden mutation

■ Prothrombin gene mutation

■ Protein C deficiency

■ Protein S deficiency

■ Antithrombin III deficiency

■ Antiphospholipid syndrome

Venous Injury

It has been proven that that venous thrombosis occurs usually in veins that are well away from the site of surgery; for example, it is well known fact that patients undergoing total hip replacement frequently develop contralateral lower extremity DVT.

The notion that damage to the intima causes thrombosis known as the “doctrine of Cruveilhier” was known very well prior to Virchow’s research.

Hodgson in 1815 had proposed that trauma to a vein is a predisposing factor to thrombosis. Likewise Davies (1823), Andral (1830) and Lee (1842) had quoted that inflammation of the endothelium was responsible for this phenomenon.

Baumgarten had demonstrated in 1876 that blood trapped in a double ligated vein failed to clot for months together ,but prompt thrombosis occurs when isolated blood with infectious material was inoculated. Barett in 1924 inflicted aseptic trauma to the veins and noted failure of coagulation even in subsequent

experiments even when bacteria was injected at the site of injury. Only when bacteria inoculated threads were placed in the vasculature a thrombus was able to develop. Along with advances in anesthesia thus enabling prolonged surgeries and casualties from many wars the significance of endothelium as a contributor to venous thrombosis was recognized. Intimal injury was thought to be as a result of direct trauma but more importantly due to damage induced by bacterial toxins. Some workers used to even say that thrombosis cannot occur in the absence of infection. Once we can appreciate the complex biology of the endothelium how various factors govern the dynamic relation as to decide the occurrence of thrombosis or thrombolysis.

In case of thrombosis, multiple microtearsseen within the valve cusps that exposes the subendothelial matrix. The exact mechanism however has not been well understood which causes injury at a site well away from the operated site and the mediators responsible whether they are cellular or humoral , but the very fact that the injury occurs and occurs reliable is clearly evident from these as well as many other studies.

Venous Stasis

Much prior to Virchow's work, Wiseman's work in 1686 establishes stasis as a significant contributor to venous thrombosis was followed by Ballie (1793) ,Davies (1823) , Andral (1830) and finally Bouchut in 1845.
Humphry in 1881 after personally incurring venous thrombosis put forward that clotting of blood occurred as a result of turbulence in blood flow. He said that "eddies" were generated due to the presence of venous valves thus facilitating clotting. It was only 100 years later that the idea of "valve pockets" were popularized. In the 1930's it had come to a consensus that for a thrombus to develop stasis alone was insufficient. In spite of this it was agreed upon that reduced blood flow contributed to thrombosis which is due to the speculation that it results in interaction between the blood components and the vessel wall or by altering the balance of activated clotting as well as inhibiting factors in the blood stream. This hypothesis was additionally supported by the observation of an increased risk of thrombosis in patients paralyzed due to stroke and spinal cord injuries and in patients confined to bed-rest or those who were immobilized. A theory was put forward that in such conditions since the veins were dependent on the pumping action of the adjacent muscles to push back blood to the heart , would develop pooling which would potentiate interaction between the erythrocytes and the vascular endothelium. "Economy class syndrome"is a Recent attention from the media has also revived interest in venous stasis as a contributor in condition especially in passengers travelling in trans-oceanic flights.

Incidence

Venous thromboembolism occurs for the first time in approximately 100 persons/ lakh individuals. This incidence rate goes up with increasing age with an approximate incidence of 0.5% per 100,000 at the age of eighty. Out of this group more than two thirds of cases have DVT alone whereas the rest are known to have evidence of pulmonary embolism. The recurrence rate with anticoagulation measures has been found to be 6% to 7% in the following 6 months .Other than pulmonary embolism, Chronic Venous Insufficiency (CVI) which results from DVT causes a significant increase of cost, morbidity, and limitation of lifestyle. Pulmonary embolism and chronic venous insufficiency are the consequences of deep vein thrombosis considering pulmonary embolism and chronic venous insufficiency, is to be prevented, then there must be an optimization of the measures for the prevention, diagnosis, and treatment of DVT.

Clinical Diagnosis

The diagnosis of DVT requires, to use an overused phrase, a very high index of suspicion. **Homans' sign**, which was initially described by American Surgeon JohnHomans (1877-1954) is basically to pain while dorsiflexing the foot which occurs in the calf. The lack of this sign is not a foolproof indicator of the absence of thrombus is the deep vein, but the once Homans' sign becomes positive this should prompt us to try and confirm the diagnosis of deep venous thrombosis. Most certainly, the extent of venous thrombosis seen in the lower limb is the primary factor responsible for the manifestation of symptoms of deep venousthrombosis . For example, most of the calf thrombi may remain asymptomatic unless it starts propagating proximally. In patients with deep venous thrombosis only 40% of patients end up with clinical manifestations. Venous thrombosis that involves the major veins such as the iliofemoral venous system will end up with a severely swollen leg associated with pitting edema, blanching and pain, a condition referred to as phlegmasia alba dolens. With progression of disease, the edema becomes so massive that after a point there will be a compromise in arterial inflow. As a result a painful blue leg will occur due to this condition, which is called as phlegmasiaceruleadolens.

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PhlegmasiaCeruleadolens

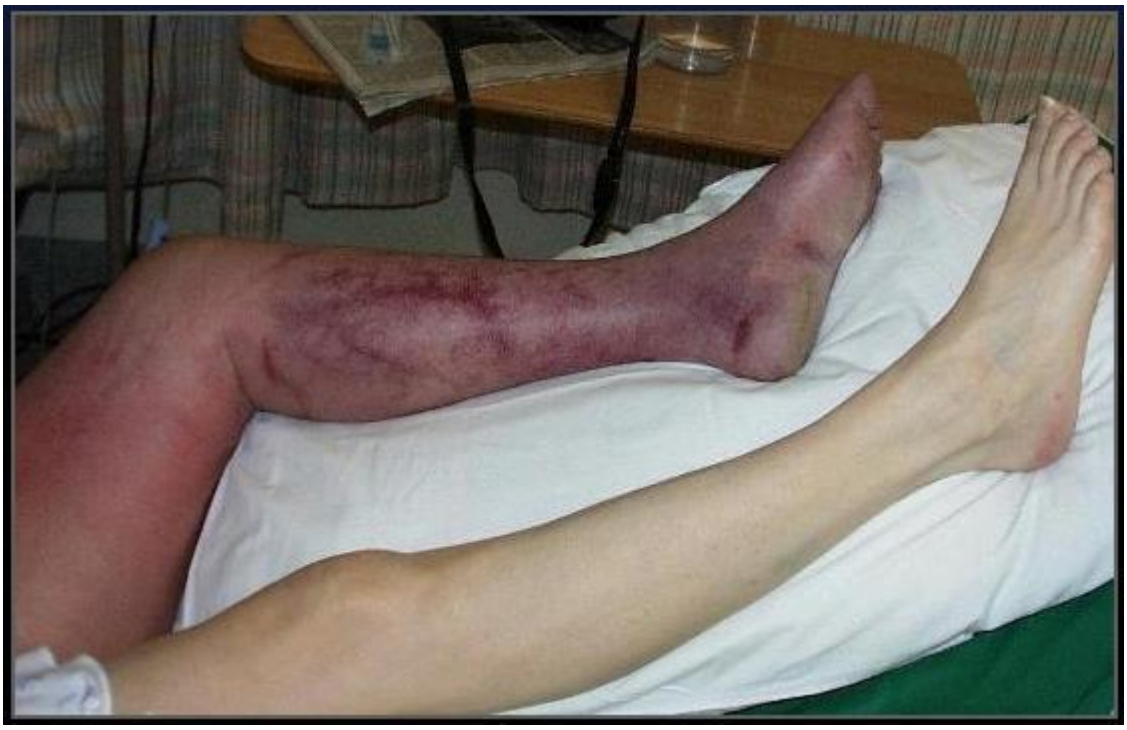


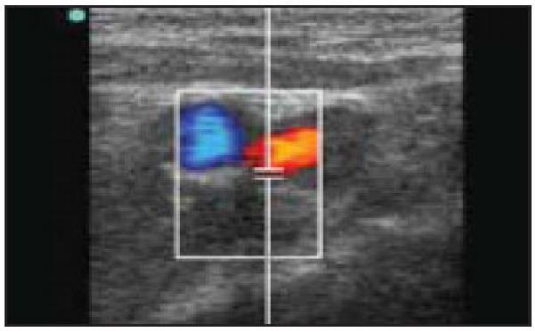
Fig.11

This condition, unless flow is restored, will result in venous gangrene .Phlegmasiaalbadolens is just a variant of cerulean dolens and has associated arterial spasm and the extremity will be pale and cool with diminished pulses.

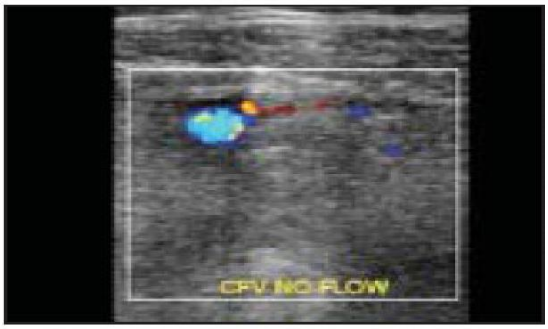
Investigations

Duplex Ultrasound

It is one of the latest diagnostic tool of choice required for diagnosing of Deep Vein Thrombosis. This modality Duplex ultrasound combines real time B-mode ultrasound along with imaging of the color-flow and has thus greatly improved the specificity and sensitivity of ultrasound scanning. Since the advent of color flow duplex imaging a thrombus which partially occludes the lumen does not hinder imaging of the blood flow. On using the probe to compress the vein a normal vein will be easily compressible whereas when a thrombus is present it offers some resistance to compression.



Color-flow Doppler in normal vessels.



Color-flow Doppler in deep vein thrombosis.

Fig.12

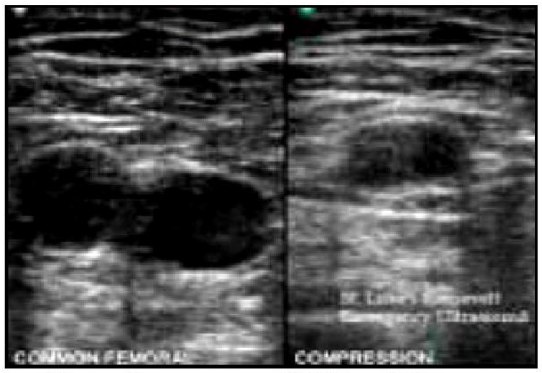
Venous Ultrasonography [4]

This includes examination of the femoral, popliteal, tibial,peroneal, soleal and gastrocnemius muscular veins as routine,plus the iliac veins & IVC where indicated.Patient lies supine – gel on skin

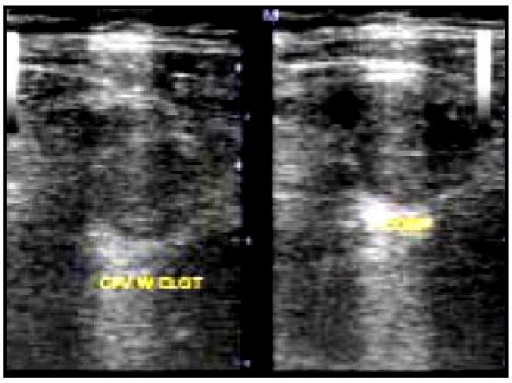
Doppler waveform in groin / common femoral vein (iliacs)

Compression of visualised veins with ultrasound probe

Non compressibility of veins helps to identify Deep vein thrombosis



Normal compressibility of the common femoral vein.



Abnormal compressibility in deep vein thrombosis.

Fig.13

Technical Limitations of Ultrasound

Cellulitis, ulceration, pain

Adductor canal, iliac veins&IVC

Ruptured Baker's Cyst

Large legs, obesity

Cardiac failure

Non-occlusive calf thrombi

Limited mobility eg. post-op

Magnetic Resonance Venography [4]

Along with the major advances in technology used in imaging of veins, magnetic resonance venography is the frontier of imaging used for proximal venous disease.

The cost factor and patient intolerance due to claustrophobia associated with an MRI machine are two factors that limit the its widespread use, but at present with advances in technology this is changing as well.

It is very a useful investigatory method for visualizing the inferior vena cava as well as theiliac veins, both being areas where duplex ultrasound has proven to be inadequate.

.Also various situations like in which ultrasound cannot be used since it requires direct contact with the limb surface especially in plaster casts.

There are also various blind spots in ultrasound such as over the gastrocnemius and profundafemoris. There is no radiation exposure compared to venography.

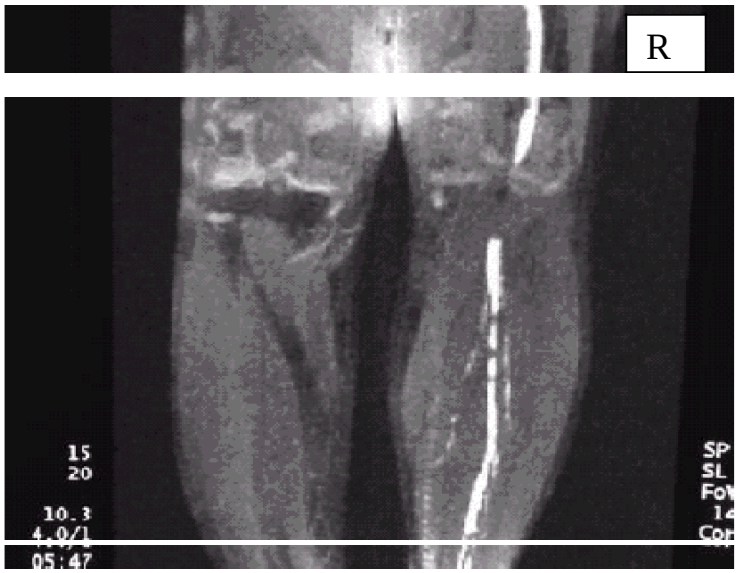


Fig.14

MR venogram showing Unilateral DVT in the right limb

Venography [4,6,7]

It Identifies thrombus in the deep veins especially of the lower limbs, but also neck, upper limb and abdomen/pelvis.thrombus in the superficial veins and also any other related pathology can be identified. Gold standard scans should include the deep calf veins and extend proximally as required. If

abrupt termination of the dye column present it indicates the presence of thrombus or filling defects will be seen in various portions of the venous system.

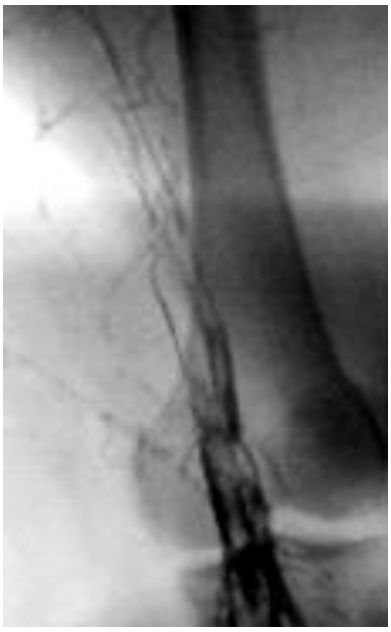


Fig.15

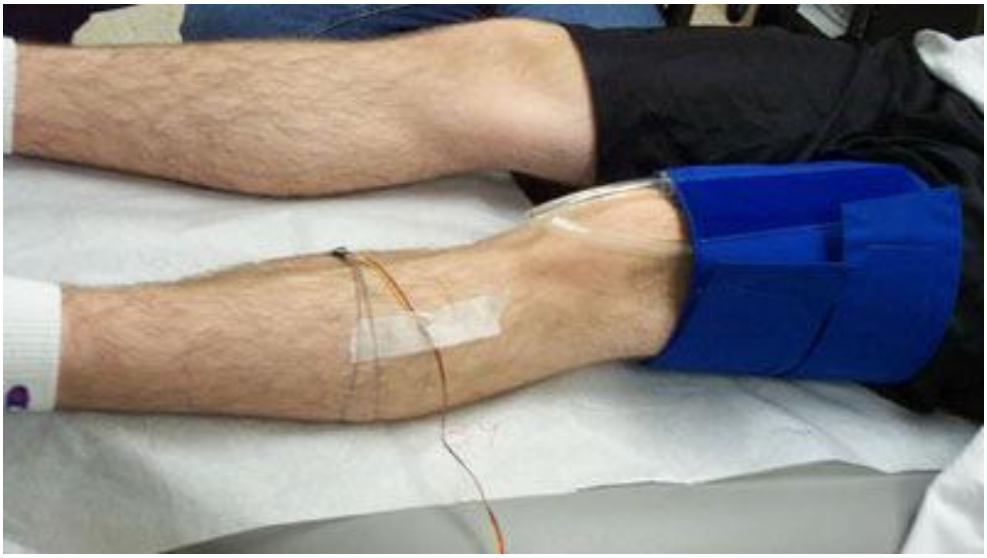
Lower-extremity venogram as shown above with enhancement of contrast depicts acute deep venous thrombosis location being the popliteal vein.

Impedance plethysmography[4]

This is a test which measures volume changes in the extremity. Here changes in electrical resistance of the chest, calf or other regions of the body are measured. But DVT is associated with edematous changes it is helpful in the diagnosis. A BP cuff is placed in the proximal thigh and inflated to a level between arterial and venous pressures. When this is done a previously positioned calf plethysmo graph will record a volume increase.

When the cuff is deflated a reduction in calf volume will occur. The rate at which this volume change occurs reflects the efficiency of venous outflow , thus indirectly indicating the presence or absence of venous thrombosis.

This test offers a good advantage in comparison to venography which is invasive and requires a great amount of skill to conduct it and interpret the results effectively.



Impedance plethysmography Fig.16

Other applications of impedance plethysmography:

- Determination of cardiac stroke volume
- Cerebral blood flow measurement
- Intrathoracic fluid volume measurement
- Determination of body composition

Wells Score [1]

Wells score also known as Wells rule was developed by Wells and his co-workers .This was solely developed with the intention of reducing the time wasted in unnecessary testing. This be given topmost preference ,test has good sensitivity and specificity in secondary care settings and has helped to reduce the cost of unwanted radiological investigations . The Wells rule simplifies diagnostic assessment by combining various aspects of history, clinical examination and investigations and thus improving clinical accuracy.

This suggests whether a patient is at low, moderate or high risk of having suffered a DVT which may guide subsequent investigation and management.

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Wells Clinical Prediction Rule for Deep Venous Thrombosis (DVT)

Clinical feature	Points
Active cancer (treatment within 6 months, or palliation)	1
Paralysis, paresis, or immobilization of lower extremity	1
Bedridden for more than 3 days because of surgery (within 4 weeks)	1
Localized tenderness along distribution of deep veins	1
Entire leg swollen	1
Unilateral calf swelling of greater than 3 cm (below tibial tuberosity)	1
Unilateral pitting edema	1
Collateral superficial veins	1
Alternative diagnosis as likely as or more likely than DVT	-2
Total points	

DVT = deep venous thrombosis.

Risk score interpretation (probability of DVT):

- /=3 points: high risk (75%);
- 1 to 2 points: moderate risk (17%);
- 1 point: low risk (3%).

Wells and coworkers have also put forward a similar scoring system

for diagnosing pulmonary embolism which has been listed below.

Clinical Characteristic	
Clinical signs of deep vein thrombosis	3
Heart rate >100 beats per minute	1.5
Recent surgery or immobilization (within the last 30 d)	1.5
Alternative diagnosis less likely than pulmonary Embolism	3
Hemoptysis	1
Cancer (treated within the last 6 mo)	1
Clinical Probability of Pulmonary Embolism	Score
Low	2
Intermediate	2-6
High	6

Hamilton Score [26]

This is a new score which is developed to overcome the limitations of Wells score. The overlapping redundant features, such as lower limb enlargement, calf enlargement, and pitting edema, render the score less accurate in stratification and more cumbersome to calculate. Important risk factors such as prior history

of deep vein thrombosis or pulmonary embolism, pregnancy, and the use of oral contraceptives were not considered when the Wells score was developed.

Hence a new pretest probability scoring system was devised to overcome all these which was Hamilton score.

Plaster immmobilisation of lower limb	2
Active cancer(within 6 months or current)	2
Strong clinical suspicion of DVT	2
Bed rest >3 days or recent surgery within 4 weeks	1
Male sex	1
Calf circumference > 3 cm on affected side (10 cm below tibial tuberosity)	1
Erythema	1

A score of ≤ 2 represents less likely probability for deep venous thrombosis (DVT); a score of ≥ 3 represents most likely probability for DVT

AIM OF THE STUDY

1)To assess the efficacy of Wells criteria for diagnosing deep vein thrombosis.

2)To bring into light the associated co morbid conditions and to find out the most significant one which has got the highest probability for causing DVT.

STUDY DESIGN:

This is a prospective diagnostic validation study of the Wells Rule for DVT in our setup, ultrasound being the gold standard comparison and will be conducted over a duration starting from 19.11.2017 to 19.09.2019.

Score for each patient will be calculated based on the parameters given in the table below.

Wells Clinical Prediction Rule for Deep Venous Thrombosis (DVT)

Clinical feature	Points
Active cancer (treatment within 6 months, or palliation)	1
Paralysis, paresis, or immobilization of lower extremity	1
Bedridden for more than 3 days because of surgery (within 4 weeks)	1
Localized tenderness along distribution of deep veins	1
Entire leg swollen	1
Unilateral calf swelling of greater than 3 cm (below tibial tuberosity)	1
Unilateral pitting edema	1
Collateral superficial veins	1
Alternative diagnosis as likely as or more likely than DVT	-2
Total points	

DVT = deep venous thrombosis.

Risk score interpretation (probability of DVT):

- /=3 points: high risk (75%);
- 1 to 2 points: moderate risk (17%);
- 1 point: low risk (3%).

Fifty patients undergone surgery and post operatively with a clinical diagnosis of Deep Vein Thrombosis will be considered as the sample size.

Inclusion Criteria:

1)Patients undergoing surgery for more than 3 hours

2)Age greater than 18 yrs

3)Onset of symptoms within 7 days

4)Those consenting to the study

Exclusion Criteria

1)Patients with recurrent DVT

2)Patients with bleeding diathesis

MATERIALS AND METHODS

Fifty patients admitted with a diagnosis of deep vein thrombosis in various departments in Stanley Medical College will be studied prospectively between September 2017and September 2019.

All patients with suspected Deep Vein thrombosis will be assigned a Wells score on admission will be followed up by venous Doppler ultrasound to confirm the diagnosis.

The correlation between the Wells score and Doppler will be checked.

Basic blood investigations such as complete blood counts,blood urea, blood sugar,serum creatinine and electrolytesalong with a chest xray and electrolytes will be sent. A urine routine examination and a chest xray will be taken.

SAFETY CONSIDERATIONS:

In every step of the study top most preference given to patient safety . All procedures will be done under strict aseptic precautions and only necessary investigations will be carried out after carefully evaluating the patient.

Any procedure will be done only after getting consent from the patient or the attender.

QUALITY ASSURANCE:

Strict care will be taken to ensure that the study is done in the best quality possible right from patient admissionwhich includes interaction with the patient,history taking ,clinical examination and while subjecting to the necessary investigations.

EXPECTED OUTCOME:

This study is basically intended to test the effectiveness of Wells criteria as a diagnostic tool in DVT.

Wells score application will help the clinician to come for the diagnosis even before confirmation by a VenousDoppler ultrasound and thus saving time in initiating treatment and thereby enabling faster recovery of DVT patients .

RESULTS

The Wells score was applied on a total of 50 cases. Out of the total number of cases it was able to diagnose DVT in 92% of the cases ie 46 out of 50 cases. Thus proving to be a very accurate indicator for DVT .

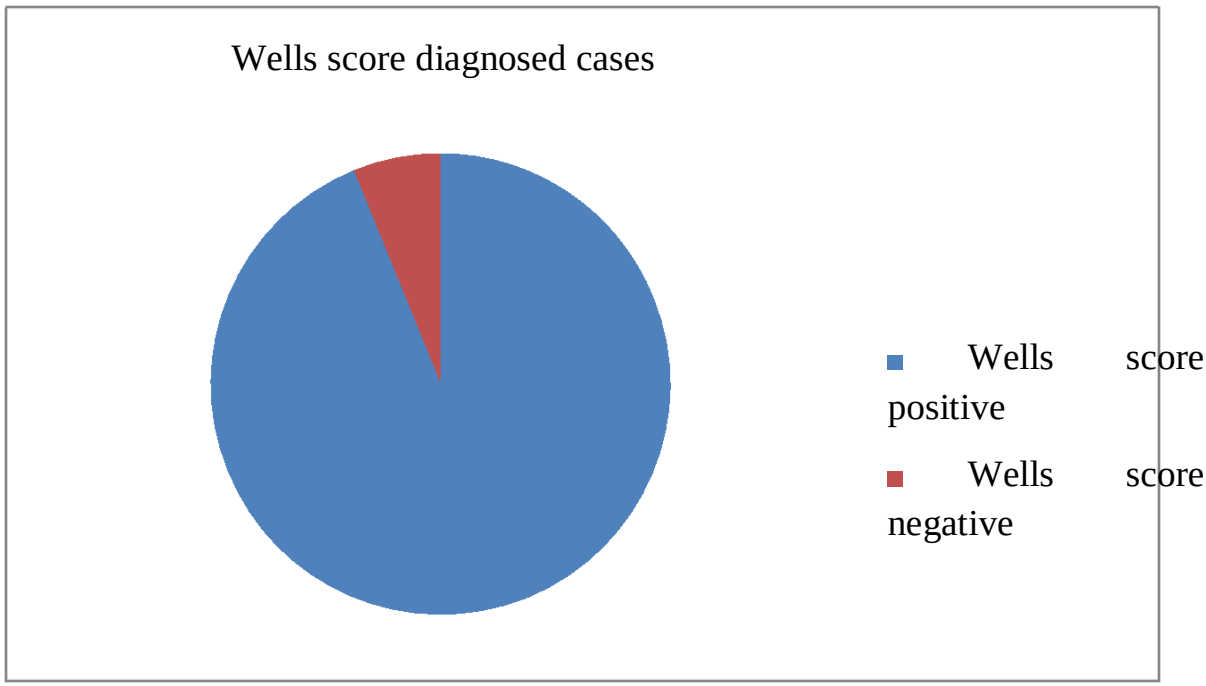

Fig.17

Wells score false positive

On analyzing the Wells score negative cases two of the cases were symptomatic due to acute lymphedema in elderly woman suffering from intra abdominal malignancy and the other two cases were due to early onset cellulitis.

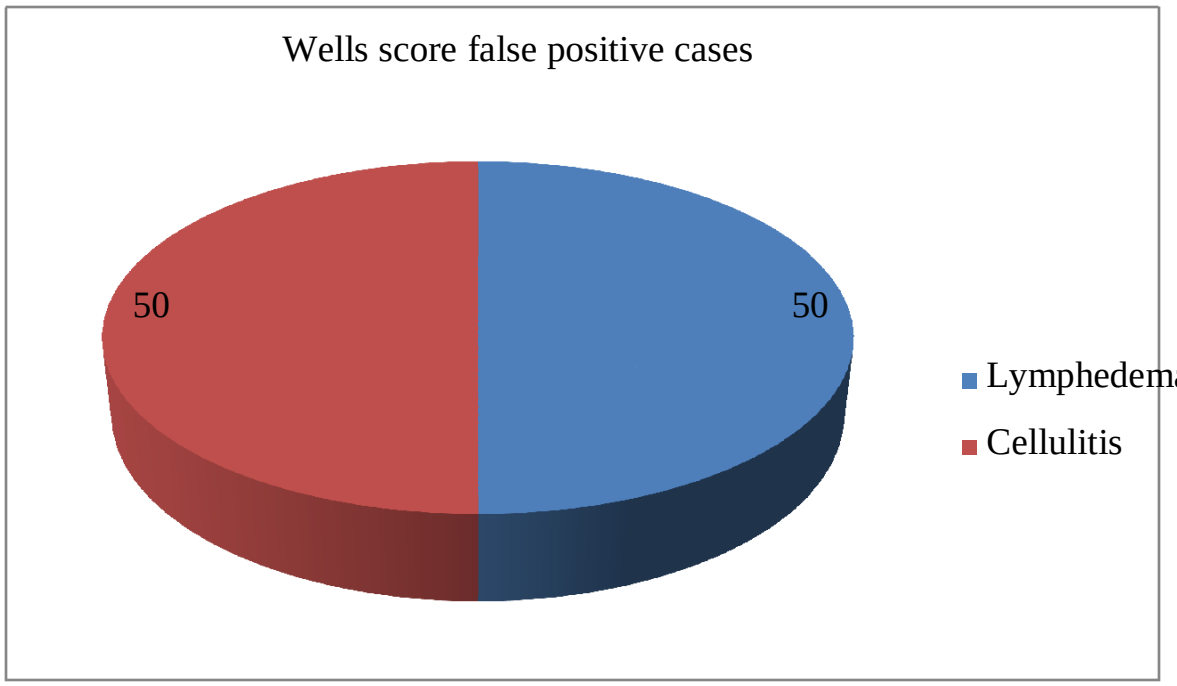

Fig.18

Out of the 4 cases which did not have DVT the highest wells score was 5/8 which included 2 elderly women with intra abdominal malignancy –namely carcinoma of the cervix thus blocking all the lymphatics resulting in tense lymphedema of the leg .The entire leg thus having the appearance of inverted beer bottle appearance similar to acute DVT. Both cases had undergone radiotherapy for cervical cancer a few years ago.

Wells score distribution

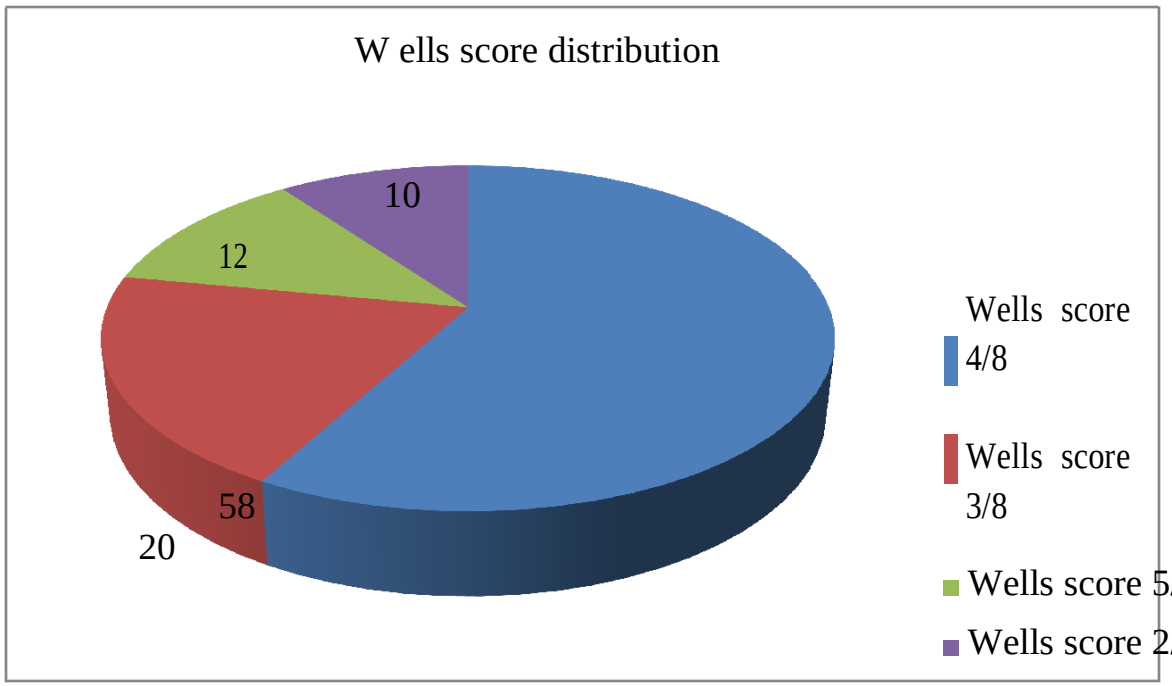

Fig.19

Most of the cases diagnosed with DVT had a Wells score of 4/8 which being above 3 thus indicates a 7 5% risk of having deep venous thromb osis.29 of the cases had a Wells score of 4/8. Followed by 10 cases which were also in the same risk category with a score of 3/8.

Six cases had the highest score of 5/8 which were also in the highest risk category out of which 2 did not have DVT and were having lymphedema

The other 2 cases were ha ving the lowest score of 2/8 which put them in the moderate risk category for having DVT which was a risk of 17% .

Blood group distribution in DVT cases

On observing the distribution of blood groups in the entire case list of DVT confirmed cases 22 outof the 46 confirmed cases of DVT ie 47.82 % had A positiveblood group. This was followed by 19 cases which amounted to 41.30 % which had B positiveblood group, the rest of the cases were AB positive ie 3 amounting to 6.52 % and finally O positive which consisted of 2 cases amounting to 4.34 % of the cases.

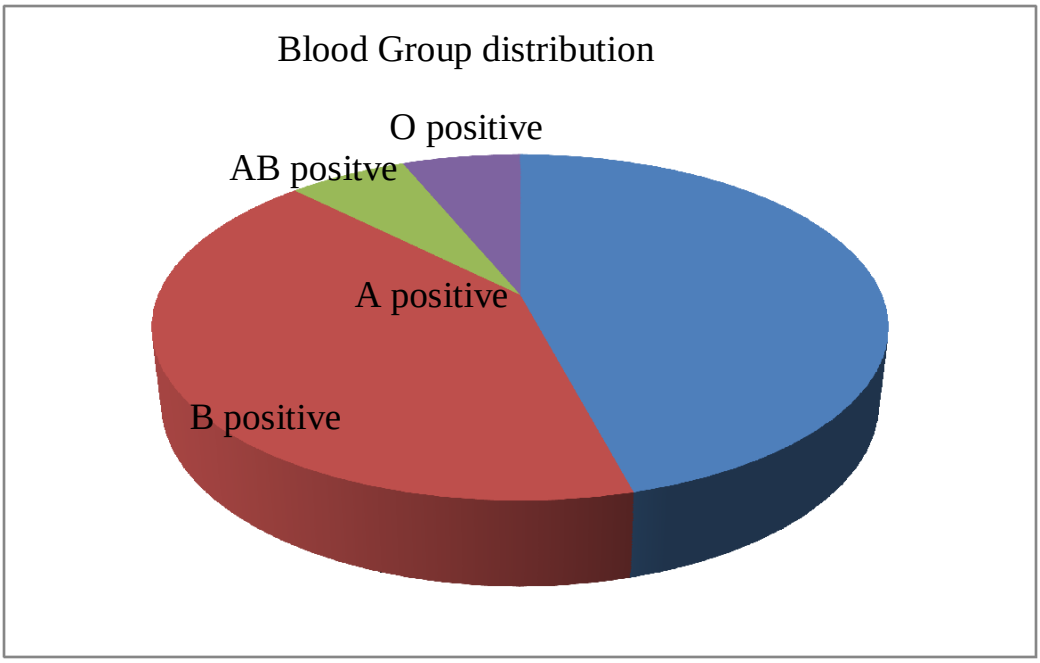

Fig.20

This in turn supports various studies which suggest a higher rate of deep vein thrombosis in individuals with blood group A .

Sex Distribution of DVT cases

On taking into account the sex distribution of cases of deep vein thrombosis .it can be seen that the number of DVT cases are more common in women in our hospital due to the high incidence of DVT in post partum females.

This is almost double the ratio of males affected by DVT . 29 females out of the total of 46 were affected. And this constituted 63% of the total number confirmed cases of deep vein thrombosis. From this ratio we canclearly understand that hormones namely estrogen and progesterone do have an important role in the causation of DVT as evident from the high number of females affected with DVT. The risk of Deep Venous Thrombosis also increased in young females who is taking oralcontraceptives and also inpost menopausal women who are on hormonereplacement therapy. (4)

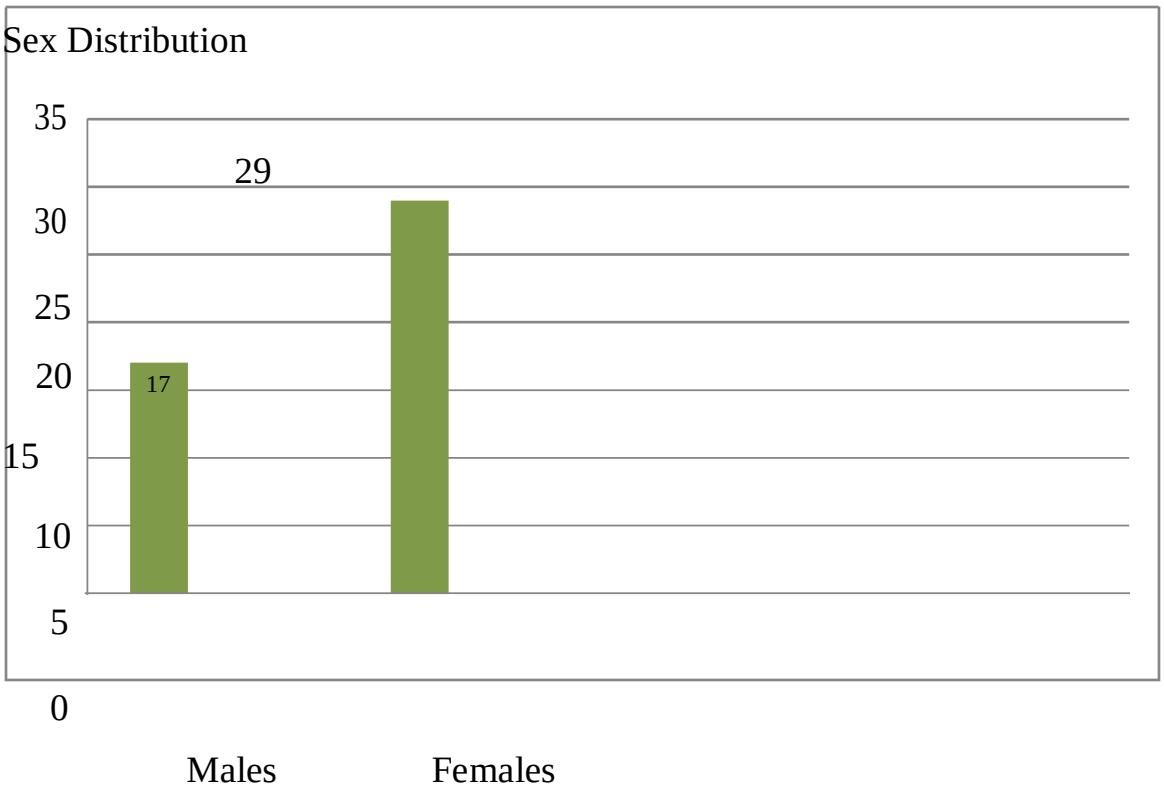

Fig.21

DVT in LSCS vs normal delivery

There was a higher incidence of cases of DVT in females who underwent caesarean section comparing with those who underwent normal delivery. This is most probably due to the prolonged periods of immobilization and pregnancy whichitself is a prothrombotic state. Out of the total of 26 pregnant females with DVT 18 patients had undergone caesarean section and 8 patients sunderwent normal delivery.

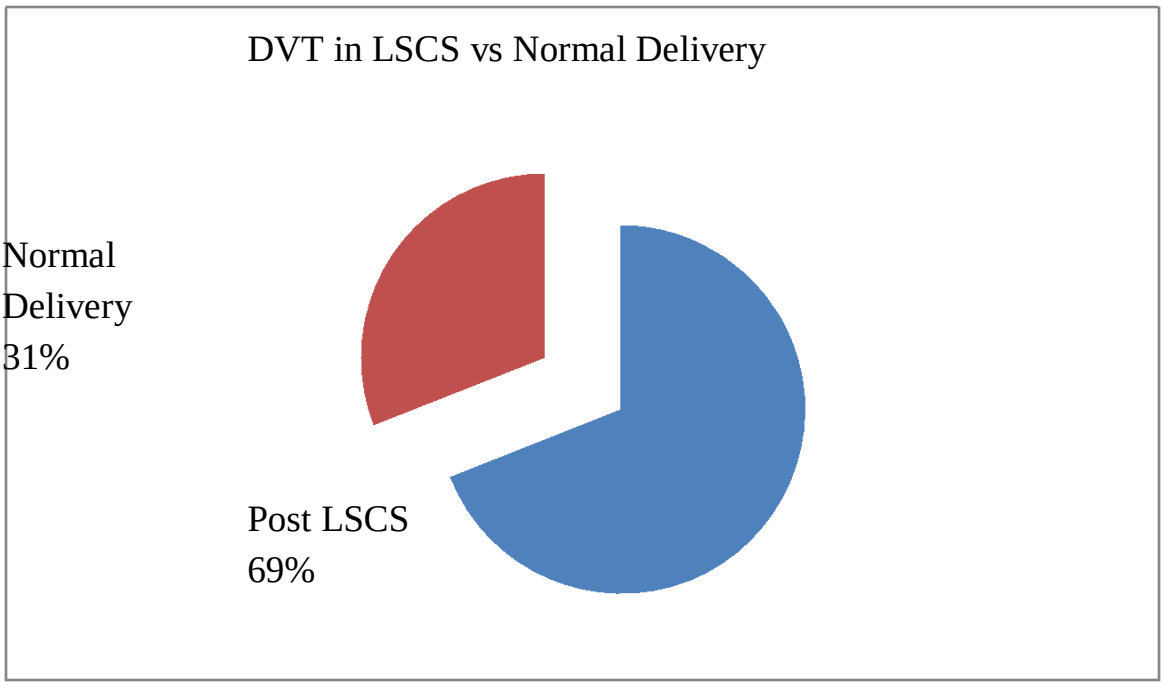

Fig.22

Age distribution of cases

The average age of male cases with DVT was 42 years whereas in cases of pregnant females it was 27 years and in non- pregnant females it was 57 years.

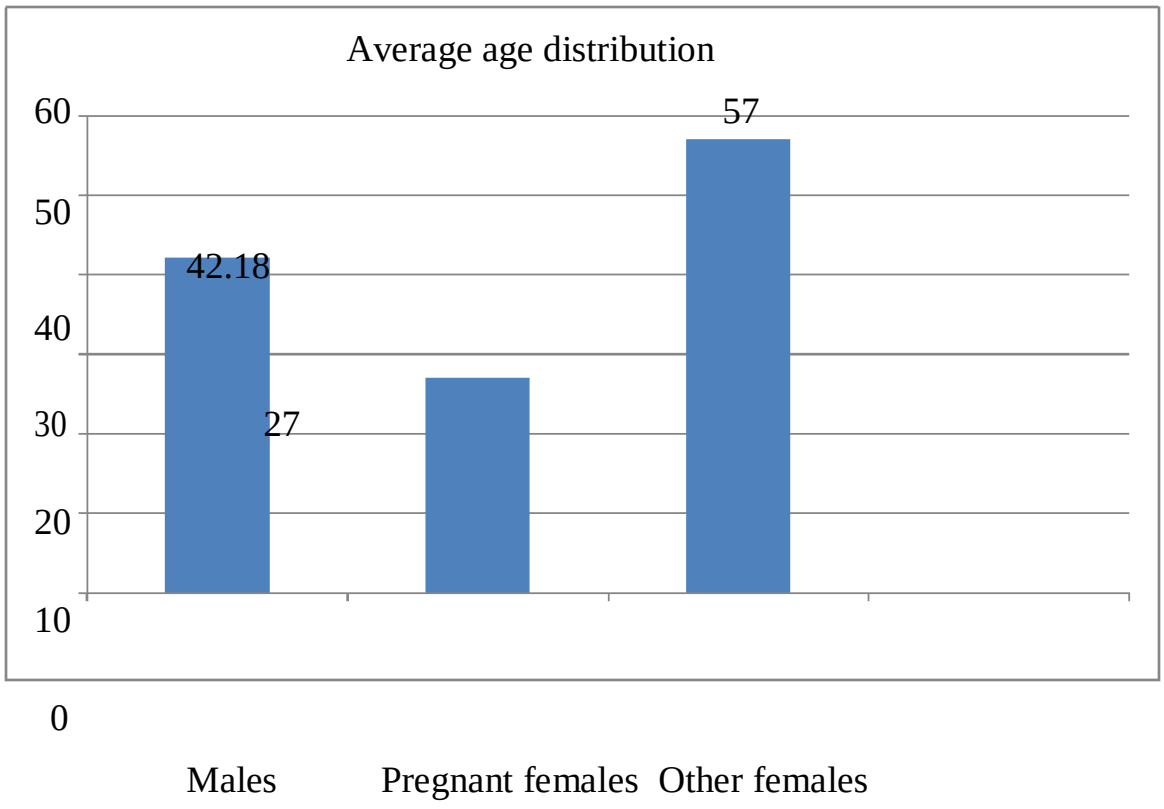

Fig.24

DVT in post op cases

Out of the total number of post op cases with DVT, 90% of the cases with DVT were post caesarean section followed by orthopaedics . 2post operative cases in general surgery were reported to have DVT. This may be possibly due to early mobilization of the post operative cases and passive flexion exercises started early in the post operativewards .

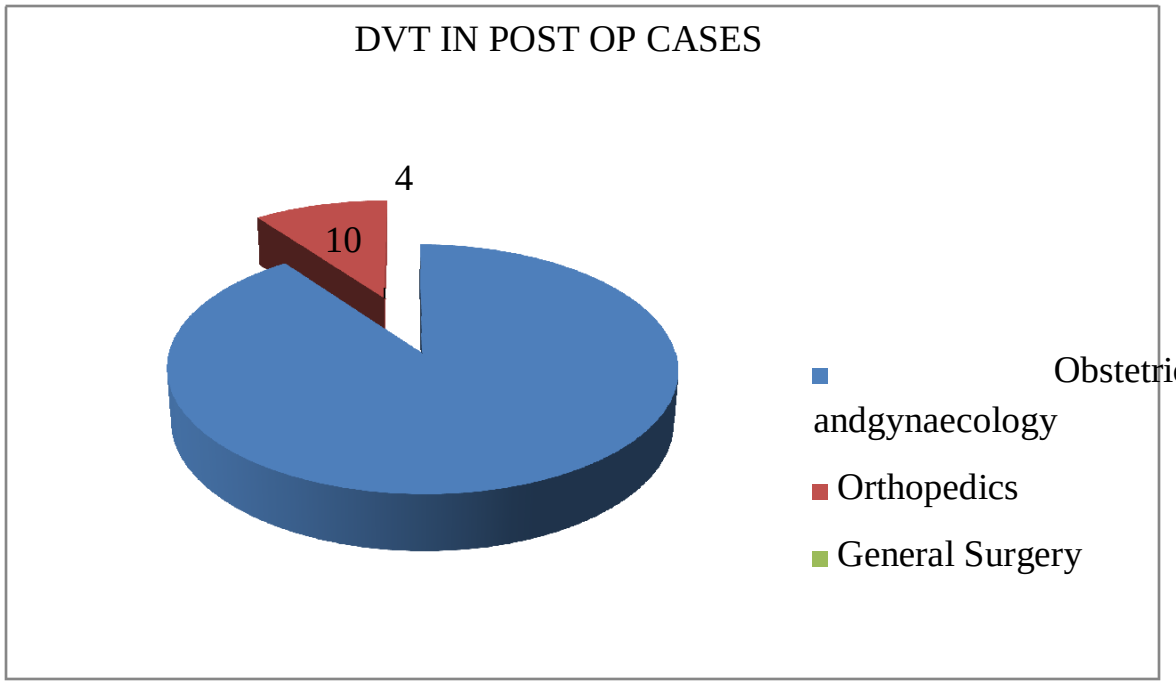

Fig.25

Total post op cases with DVT-20

LSCS cases -16

Orhtopedics cases- 2

General Surgery -2

Complications seen in the study group-

Out of the 46 cases of DVT 3 cases went in for complications. Out of the three cases 2 were female who had just delivered and had gone in for cerebral venous thrombosis. This was resolved by standard dose of heparin titrated according to

PT/INR values .One male patient died as he went in for cardiac complications due to pulmonary thromboembolism.

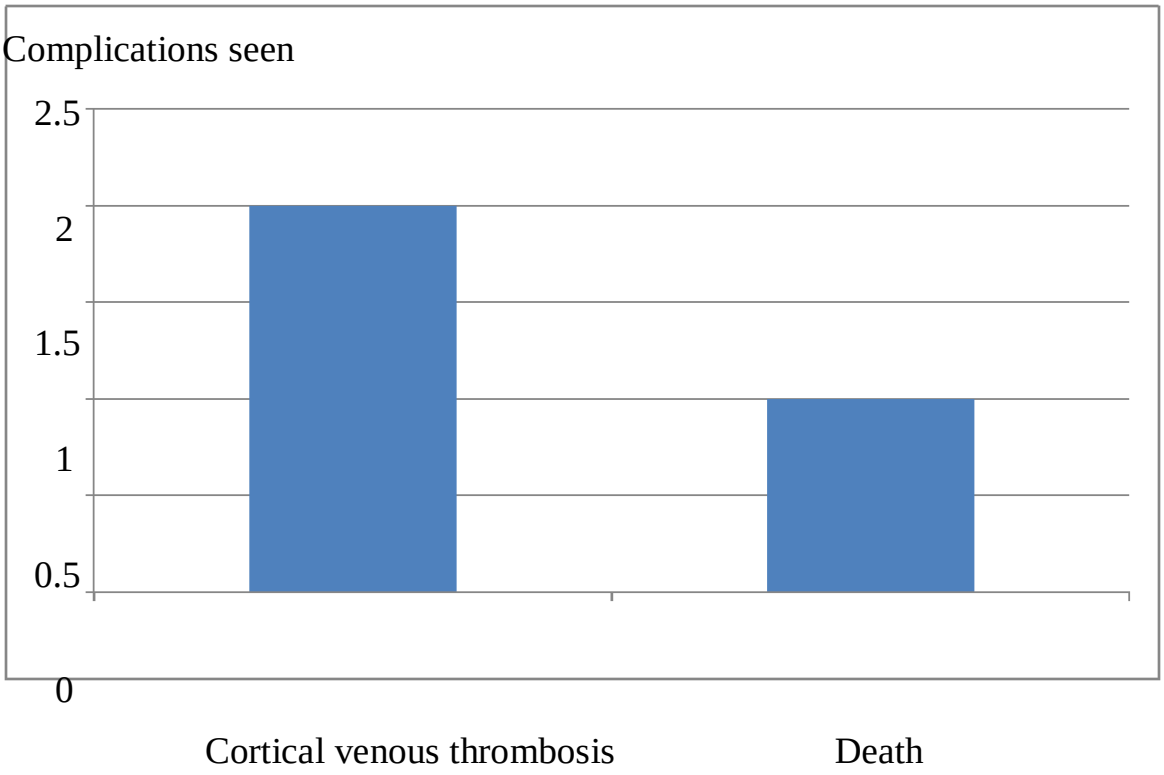

Fig.26

Analysis of co morbidities-

On analyzing the associated co morbidities in the study group it is clear that surgery was the most important contributing factor that result in DVT. In our study group most of the surgical cases were post caesarean section. Other co morbid condition causing DVT in descending order are smoking, dyslipidemia ,obesity ,immobilization, cancer, myocardial infarction, renal failure andcancer. And finally a single case of IV drug abuse was also reported to have DVT.

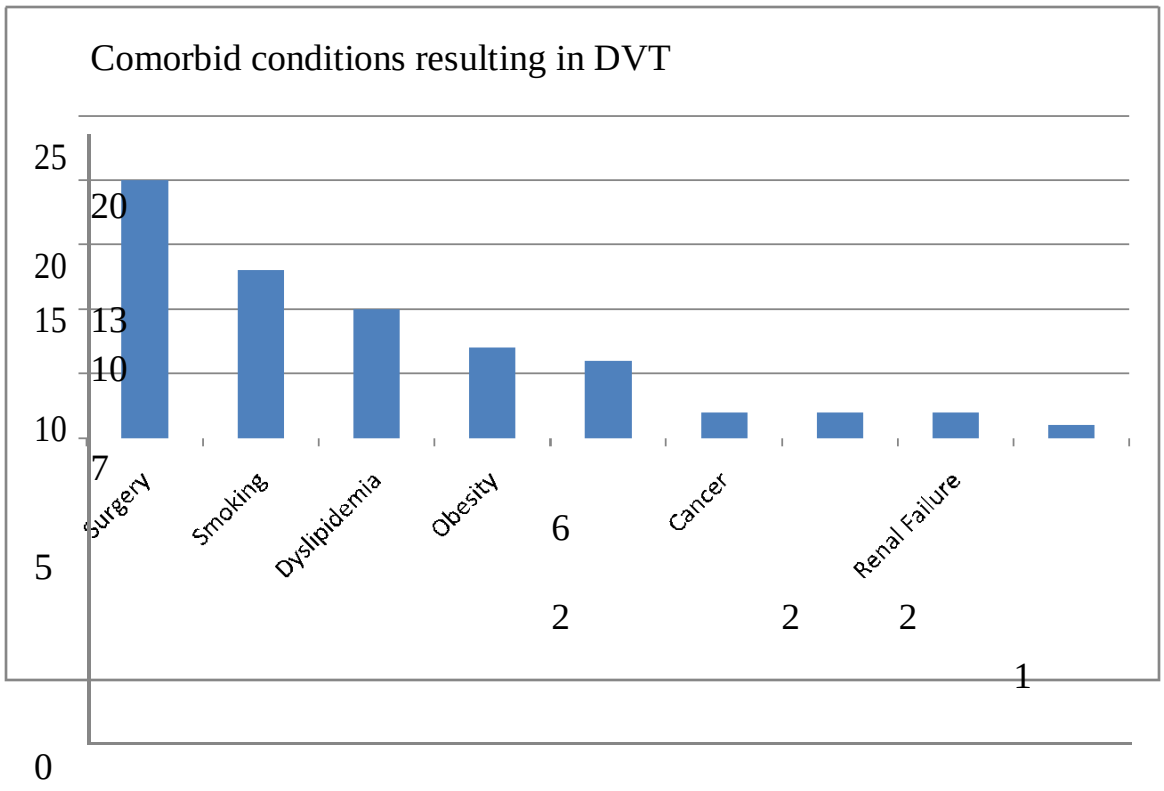
Comorbid conditions resulting in DVT
25
20
15
10
5
0
20
13
10
7
6
2
2
2
1
Surgery
Smoking
Dyslipidemia
Obesity
Cancer
Renal Failure

Fig.27

DISCUSSION

Deep vein thrombosis is a condition which occurs in any location within the venous circulation. Majority of deep venous thrombi are known to occur within the deep veins of the leg , followed by the pelvis.

Occasionally deep vein thrombosis can also occur in the cerebral venous sinuses which in our study was seen in two of the post partum cases.

The Wells score was applied to 50 cases out of which 46 cases have deep venous thrombosis proven by Venous Doppler ultrasound.

Out of the proven cases of DVT there was a female had high predominance of cases in a ratio of 1.7:1 . Out of the total number of cases 63.04% was female and the remaining 36.96 were male

This is similar result with a study done in New Zealand by MaelenTagelagi and co-workers where 62% were women.[1]

However, Wells Score was not so effective in their setup according to the New Zealand study the unlike the high predictive rate I.e 46 out of 50 suspected cases seen in our study.

Among the false positive cases two were due to malignancy of the cervix who had underwent radiotherapy, the other 2 were due to early onset cellulitis.

By various mechanisms lymphedema known to develop cancer. Primary cause is mainly due toextensive lymph nodal dissection following surgery .Radiotherapy, following surgery for cancers also causes scarring of the lymph nodes and lymph vessels.

Also lymph vessels are blocked by tumour embolus. As mentioned by Ruud Oudega and coworkers [10], Any cancer is a predisposing factor for unprovoked DVT Lymphedema may also develop in cellulitis due to stagnation of protein rich lymphatic fluid which acts to be a rich medium for bacteria to grow thus creating a vicious cycle.On analysis of the average Wells score seen in the DVT positive cases out of the 46 cases 29 cases had a Wells score of 4 out of 8 which indicates a 75% or greaterchance of having DVT. [1]The average age for DVT in male cases in our study group was 42 years ,and in pregnant females it is 27 followed by a sudden rise in age which were mostly post menopausal women with cancer. Pregnant women have a five times higher chance ofhaving DVT compared to non pregnant women.[3,4,5

This again correlates with the statistics given by Office of the Surgeon General (US) and the National Heart, Lung, and Blood Institute (US). [5]It was also seen that non O blood grouppatientshad a higher incidence of DVT . In our study majority of the cases of DVT were having a positive blood group thus indicates a higher level of plasma Von Willebrand factor, possibly a mutated variant. This correlated with the studiesby Massimo Franchini and Mike Makris. [4,8]Among the cases of DVT which had occurred in the post operative period, thehighest incidence of DVT was following caesarean section, followed by orthopedics department in polytrauma cases. There was 2 case of DVT in the General surgery post operative ward in our study group. This reduction could have been due to the early mobilization of cases in post op wards and advocation of passive flexion exercises.There is also evidence in literature as written by Victor et al supporting that smoking ,by increasing platelet activation, also creates a procoagulant state , decreasing fibrinolysis and various other mechanisms which puts smoking as a very high risk factor for DVT. [10]

CONCLUSION

- Among the 50 cases suspected DVT , the Wells score was able to predict DVT in 46 of the cases thus proving to be a very efficient diagnostic indicator.
- The average Wells score among the various cases was 4/8.
- Female cases of DVT higher compared to males in a ratio of 1.7:1.
- Considering the age distribution of cases males were mostly in the 4^{th} decade followed by pregnant females in 2^{nd} decade and other females in the 5^{th} decade.
- The most important co morbid state for DVT is surgery especially caesarean section as seen in our study group as the patient is immobile as well as there is aprothrombotic state.
- Among the cases of DVT post delivery, majority of them had developed DVT within the first 2 weeks following delivery whether via labournaturalis or LSCS, thus it is very important to promote early mobilization in the post partum period or even prophylactic anticoagulants with caution given the scale of the problem to decrease the morbidity in the post partum period.

- Complications noted in 2 cases in the study group were of cortical vein thrombosis in the post partum period which fully recovered.
- Mortality rate in the study group is 4.3% in which a single case of diagnosed pulmonary thromboembolism died of heart failure.
- To sum up we can conclude that the Wells score is indeed a very good predictive criteria for deep venous thrombosis and can be applied with ease as it required only clinical assessment and thus avoids unnecessary delays in waiting for scans thereby allowing us to start anticoagulants as early as possible.

APPENDIX I

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7)Schwartz principles of Surgery -9th edition

8) Non-O blood group: an important genetic risk factor for venous thromboembolism

Massimo Franchini1 and Mike Makris

9)The role of dyslipidemia and statins in venous thromboembolism-Joel G Ray and Frits R Rosendaal

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12) Deep vein thrombosis in primary care: possible malignancy? -Ruud Oudega, MD, PhD, GP and Senior

Researcher and Karel GM Moons, MSc, PhD, Professor of Clinical Epidemiology

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16)Does This Patient Have Deep Vein Thrombosis? Sonia S. Anand, MD et al

17) Lensing AWA, Prandoni P, Brandjes DPM, et al. Detection of deep-vein thrombosis by real-time B-mode ultrasonograph*N Engl J Med* 1989; 320: 342–45.

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23)Cogo A, Lensing AWA, Koopman MMW, et al. Compression ultrasound for the diagnostic management of patients with clinically suspected deep-vein thrombosis: a prospective cohort study. *BMJ* 1997;

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24) Huisman MV, Buller HR, ten Cate JW, et al. Management of clinically suspected acute venous thrombosis in outpatients with serial impedance plethysmography in a community hospital setting. *Arch Intern Med* 1989;

149: 511–15.

25) Prandoni P, Lensing AWA, Buller HR, Vigo M, CogoA, ten Cate JW.Failure of computerized impedance plethysmography in the diagnostic management of patients with clinically suspected deep-vein thrombosis.

26) Importance of Pretest Probability Score and D-Dimer Assay Before Sonography for Lower Limb Deep Venous Thrombosis

Rathan M. Subramaniam[1 2 3], Tina Chou[1], Rebekah Heath[1] and Robin Allen[4]

27) Ganong's review of medical physiology

APPENDIX II

PROFORMA

Patient particulars

- Name
- Age
- Sex
- IP No
- Address
- DOA
- DOS
- DOD

History

- Complaints
- History of present illness
- History of Chronic Diseases

General Physical Examination

- Pulse
- Blood Pressure
- Temperature
- Hydration
- GCS

Examination of Abdomen

- Inspection
- Palpation
- Percussion
- Auscultation
- PR examination

Systemic Examination

- Respiratory System
- Cardiovascular System
- Central Nervous System

Investigations

- WBC Counts
- Blood grouping
- Serum Sodium
- Serum Potassium
- Serum Bicarbonate
- Blood Urea
- Serum Cholesterol
- X-Ray chest
- Ultrasound venous doppler

Wells Clinical Prediction Rule for Deep Venous Thrombosis (DVT)

Clinical feature	Points
Active cancer (treatment within 6 months, or palliation)	1
Paralysis, paresis, or immobilization of lower extremity	1
Bedridden for more than 3 days because of surgery (within 4 weeks)	1
Localized tenderness along distribution of deep veins	1
Entire leg swollen	1
Unilateral calf swelling of greater than 3 cm (below tibial tuberosity)	1
Unilateral pitting edema	1
Collateral superficial veins	1
Alternative diagnosis as likely as or more likely than DVT	-2
Total points	

DVT = deep venous thrombosis.

Risk score interpretation (probability of DVT):

- /=3 points: high risk (75%);
- 1 to 2 points: moderate risk (17%);
- 1 point: low risk (3%).

APPENDIX III

MASTER CHART

s.no	Name	Age/sex	Ip.no	D.O.A	I.M	S.X	C.A	Smoking	M.I	R.F	T.A	B.G	I.V	O.B	DYS	PREG	W.S	DVT
1	samsudhin	31/m	1965215	26/9/19	No	No	No	Yes	No	No	No	A+	No	No	No	N.A	4/8	Yes
2	Elumalai	45/m	1966295	13/9/19	No	No	No	Yes	No	No	No	B+	No	No	Yes	N.A	3/8	Yes
3	Arumugam	33/m	1967892	23/8/19	Yes	No	No	Yes	No	No	No	B+	No	No	No	N.A	4/8	Yes
4	Chandran	52/m	1968253	2/9/19	Yes	no	No	Yes	No	No	No	AB+	No	No	No	N.A	2/8	Yes
5	latha	70/f	1970107	26/9/19	Yes	No	Yes	No	No	No	No	B+	No	No	No	N.A	5/8	Yes
6	elangovan	35/m	1871225	14/11/18	No	No	No	No	No	No	No	A+	No	No	No	N.A	4/8	Yes
7	malliga	35/f	1872449	27/11/18	Yes	Yes	No	No	No	No	No	B+	No	No	No	Yes	4/8	Yes
8	shanthi	38/f	1875814	7/12/18	No	No	No	No	No	No	No	A+	No	No	No	N.A	4/8	Yes
9	Palaniammal	35/f	1879854	15/12/18	No	No	No	No	No	No	No	B+	No	No	No	Yes	3/8	Yes
10	Mari	26/f	1878855	19/12/18	No	No	No	No	No	No	No	B+`	No	No	No	Yes	4/8	Yes
11	Janeth	27/f	181923	1/1/18	No	Yes	No	No	No	No	No	A+	No	No	No	Yes	3/8	Yes
12	Jamilaa	23/f	181021	17/1/18	No	No	No	No	No	No	No	B+	No	Yes	No	Yes	4/8	Yes
13	Arusaamy	42/m	181572	18/1/19	No	No	No	Yes	Yes	No	No	AB+	No	No	Yes	N.A	2/8	Yes
14	Banupriya	21/f	181645	4/2/19	No	Yes	No	No	No	No	No	A+	No	No	No	Yes	4/8	Yes
15	Shanthaa	45/f	182378	15/2/19	No	No	Yes	No	No	No	No	B+	No	No	No	N.A	5/8	Yes
16	Ramanan	47/m	183376	18/2/18	No	No	No	Yes	No	No	No	B+	No	No	Yes	N.A	3/8	Yes
17	Maragathamal	41/f	184998	3/3/18	Yes	No	No	No	No	No	No	A+	No	No	No	N.A	3/8	Yes
18	Srinivaasan	45/m	185279	15/3/18	No	No	No	Yes	No	No	No	A+	No	No	Yes	N.A	2/8	Yes
19	Jaya	23/f	185663	21/3/18	No	Yes	No	No	No	No	No	B+	No	No	No	Yes	4/8	Yes
20	Eswari	25/f	186491	28/3/18	No	Yes	No	No	No	No	No	O+	No	No	No	Yes	4/8	Yes
21	Natarajan	52/m	187975	7/4/18	No	No	No	No	No	No	No	A+	No	No	Yes	N.A	3/8	No
22	selvam	45/m	188331	9/4/18	No	No	No	No	No	No	No	A+	No	Yes	Yes	N.A	4/8	Yes
23	Lakshmipriya	21/f	189241	16/4/18	No	No	No	No	No	No	No	O+	No	No	No	Yes	3/8	No
24	Durgadevi	25/f	1810214	29/4/18	No	Yes	No	No	No	No	No	B+	No	No	No	Yes	4/8	Yes

25	Sumatra	30/f	1815476	30/4/18	No	No	No	No	No	No	No	B+	No	Yes	No	Yes	4/8	Yes
26	Jayanth	32/m	1817582	2/5/18	Yes	Yes	No	Yes	No	No	Yes	A+	No	Yes	No	N.A	5/8	Yes
27	Nallammal	31/f	1821884	11/5/18	No	No	No	No	No	No	No	A+	No	No	No	Yes	4/8	Yes
28	Karthikeyan	32/m	1824662	12/5/18	No	No	No	Yes	No	Yes	No	B+	No	No	Yes	N.A	5/8	Yes
29	Perumalsamy	45/m	1827492	27/5/18	No	No	No	Yes	Yes	No	No	B+	No	No	Yes	N.A	4/8	Yes
30	Chinnaponnu	56/f	1831226	5/6/18	No	No	Yes	No	No	No	No	B+	No	Yes	Yes	N.A	5/8	No
S.No	Name	Age/sex	Ip .no	D.O.A	I.M	S.X	C.A	Smoking	M.I	R.F	T.A	B.G	I.V	O.B	DYS	PREG	W.S	DVT
31	Dhandapaani	54/m	1834887	8/6/18	No	no	No	No	No	Yes	No	A+	No	No	Yes	N.A	4/8	Yes
32	Suseela	67/f	1837922	9/6/18	Yes	No	Yes	No	No	No	No	A+	No	Yes	Yes	N.A	5/8	No
33	Ravi	45/m	1838655	15/6/18	No	No	No	Yes	No	Yes	No	A+	No	No	Yes	N.A	4/8	Yes
34	Arumugam	70/m	1839179	19/6/13	No	No	No	Yes	No	No	No	B+	No	No	Yes	N.A	4/8	Yes
35	Nataraj	55/m	1847283	21/6/18	No	Yes	No	No	No	No	No	A+	No	No	No	N.A	4/8	Yes
36	Krishnan	34/m	1847349	26/6/18	No	No	No	Yes	No	No	Yes	B+	No	No	No	N.A	2/8	Yes

37	Rangamma	23/f	1848927	4/7/18	No	Yes	No	No	No	No	No	B+	No	Yes	No	Yes	4/8	Yes
38	Dharshini	27/f	1850821	11/7/18	No	Yes	No	No	No	No	No	AB+	No	No	No	Yes	3/8	Yes
39	Shanthalakshmi	25/f	1851173	16/7/18	No	Yes	No	No	No	No	No	B+	No	No	No	Yes	4/8	Yes
39	ranjini	30/f	1852746	7/8/18	No	No	No	No	No	No	No	O+	No	No	No	Yes	4/8	Yes
40	Sivakaami	26/f	1852739	12/8/18	No	Yes	No	No	No	No	No	A+	No	No	No	Yes	4/8	Yes
41	Sanmugamal	24/f	1852882	18/8/18	No	No	No	No	No	No	No	A+	No	No	No	Yes	4/8	Yes
42	Ranjitham	28/f	1854661	23/8/18	No	Yes	No	No	No	No	No	A+	No	No	No	Yes	3/8	Yes
43	Eswari	29/f	1855721	29/8/18	No	Yes	No	No	No	No	No	O+	No	No	No	Yes	3/8	Yes
44	Janaki	22/f	1856982	1/9/18	No	Yes	No	No	No	No	No	A+	No	No	No	Yes	4/8	Yes
45	Vani	26/f	1861883	7/9/18	No	Yes	No	No	No	No	No	A+	No	No	No	Yes	4/8	Yes
46	Lakshmi	25/f	1862017	12/9/18	No	Yes	No	No	No	No	No	A+	No	No	No	Yes	4/8	Yes
47	Ramasamy	52/m	1863715	23/9/18	No	No	No	No	Yes	No	No	B+	No	No	No	No	2/8	Yes
48	Subathra	31/f	1863740	2/10/18	No	Yes	No	No	No	No	No	A+	No	No	No	Yes	4/8	Yes
49	malliga	28/f	1866752	17/10/18	No	Yes	No	No	No	no	No	A+	no	No	No	Yes	3/8	Yes
50	banumathy	25/f	1867931	19/10/18	No	Yes	No	No	No	No	No	A+	No	No	No	Yes	4/8	Yes

LEGENDS

DVT-deep vein thrombosis

D.O.A –date of admission

I.M-immobilization

S.X- Surgery done

C.A- cancer diagnosed case

M.I-myocardial infarction

R.F –renal failure

T.A –Trauma case

B.G –blood group

DYS-dyslipidemia

I.V –intravenous drug abuser

PREG- pregnant female

W.S-Wells Score

N.A- not applicable