DISSERTATION

ON

A PROSPECTIVE STUDY ON INCIDENCES OF DEEP VENOUS THROMBOSIS IN CHOLECYSTECTOMY PATIENTS BETWEEN JULY 2011 TO JUNE 2012

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Dissertation submitted to

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

In partial fulfilment of the regulations

for the award of the degree of

M.S. -GENERAL SURGERY- BRANCH – I

THANJAVUR MEDICAL COLLEGE,

THANJAVUR - 613 004.

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

CHENNAI - 600 032.

APRIL -2013

CERTIFICATE

This is to certify that this dissertation entitled ``**A PROSPECTIVE STUDY ON INCIDENCES OF DEEP VENOUS THROMBOSIS IN CHOLECYSTECTOMY PATIENTS BETWEEN JULY 2011 TO JUNE 2012** '' is the bonafide original work of Dr.S.ELAVARASAN in partial fulfilment of the requirements for M.S. (General Surgery) Examination of the Tamilnadu Dr.M.G.R. Medical University to be held in APRIL - 2013.

The period of the study was from July – 2011 to June -2012.

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DECLARATION

I, **Dr.S.ELAVARASAN**, solemnly declare that the dissertation titled **"A PROSPECTIVE STUDY ON INCIDENCES OF DEEP VENOUS THROMBOSIS IN CHOLECYSTECTOMY PATIENTS BETWEEN JULY 2011 TO JUNE 2012."** is a bonafide work done by me at Thanjavur Medical College, Thanjavur during July 2011 to June 2012 under the guidance and supervision of **Prof.Dr.T.KARUNAHARAN, M.S. FICS.FIAGES,** Professor of general surgery, Thanjavur Medical College, Thanjavur.

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

Place: Thanjavur.

Date: - 12 - 2012. **(Dr.S.ELAVARASAN.)**

ACKNOWLEDGEMENT

I gratefully acknowledge and my sincere thanks to **Prof.Dr.GUNASEKARAN, M.D.DCH,** Dean, Thanjavur Medical College, Thanjavur, for allowing me to do this dissertation and utilize the institutional facilities.

I am extremely grateful to **Prof.Dr.V.BALAKRISHNAN, M.S.,** Professor and Head of the Department, Department of General Surgery, Thanjavur Medical College, for his full-fledged support throughout my study and valuable suggestions and guidance during my study and my post graduate period.

I am greatly indebted to **Prof.Dr.T.Karunaharan, M.S., FICS, FIAGES**, my unit chief , who is my guide in this study, for his timely suggestion , constant encouragement and scholarly guidance in my study and my post graduate period.

I profoundly thank my respected Professors, **Prof.Dr.T.Krishnamurthi,M.S, Prof.Dr.Maragathamani Ilangovan,M.S., Prof.Dr.D.Nagarajan,M.S., Prof.Dr.Elangovan,M.S., Prof.P.Rajagopal,M.S, Prof.Dr.P.Shanthini,M.S.,DGO,** and **Dr.R.Eganathan,M.S.D.A.,** (Registrar), for their advice, guidance and valuable criticism which enabled me to do this work effectively.

I would like to express my gratitude to **Prof.Dr.k.Vijayalakshmi, M.D., DMRD.,** Professor and Head, Department of Radiology, and **Prof.Dr.A.Srinivasan, M.D.,** Professor of Radiology for their immense help in the study which enabled me to complete this work .

My sincere thanks to assistant professors **Dr.A.Michael,M.S.,Mch., Dr.S.Maruthudurai,M.S., Dr.K.Sathyabama,M.S, Dr.K.Anbarasan,M.S. Dr.Vanathi,M.S.**, and **Dr.R.Ashokkumar,M.S.**, , for their motivation, encouragement and support.

I should always thank my senior and junior post graduates, CRRI’s who have helped me in numerable ways for my growth.

A special mention of thanks to all the patients who participated in this study for their kind cooperation.

I would like to thank my colleagues and friends who have been a constant source of encouragement.

CONTENTS

Introduction

Deep venous thrombosis is a condition where there is development of thrombus in the vein of deep venous system, mostly affecting lower limbs.

Deep venous thrombosis is not a rare entity as we think. It is a much more common but often underestimated and under recognised. These are because of the lack of standardisation in all health facilities. That too in general surgery, where it often goes unnoticed because of less suspicion. So to get an idea about DVT in patients undergoing open cholecystectomy, which is one of the common operations done in our hospital, I have done this study.

Background

DVT causes lot of morbidity and mortality. It affects both patient and health faculty, in terms of time, money, materials and often lives in case of pulmonary embolism. To conclude the patient risk for developing DVT, who are undergoing open cholecystectomy in our institution, I have assessed the patients in a prospective manner.

Objective

1. To study the incidence of DVT in patients undergoing open cholecystectomy, who are otherwise free of obvious risk for DVT, for a period of one year from June 2011 to July 2012 in our general surgery department.

2. To form a protocol for whom the pharmacological methods of thrombo prophylaxis are to be started.

Why cholecystectomy in particular?

1. Common major surgery in our hospital.
2. Common risk factors to both like Fatty, fourty etc so Prone for DVT.
3. Reverse Trendelenberg position for Cholecystectomy (as 40% of DVT starts on table itself)
4. The immobilisation following surgery due to upper abdomen incision
5. To prevent pulmonary embolism from its root level, as pulmonary embolism has high mortality.
6. If the guidelines, suggested by this study are accepted it may be used for future protocols.

Materials and Methods

All the patients planned for cholecystectomy were subjected to following investigations

Radiological investigation

- Doppler – for all patients
- X ray chest / CT chest – if suspicion of PE

Bio chemical investigations

- Lipid profile
- Complete hemogram
- PT/INR
- Liver function test
- Renal function test
- ESR

History wise

- h/o DVT/PE
- Family history of connective tissue disorder
- Cancer surgery
- Immobilisation
- OCP intake

- Post partum
- h/o sepsis
- varicose veins

Clinically patient is examined in a symptom oriented manner.

Study cohort

Totally 68 open cholecystectomy done in out department.

30 – Eligible after initial screening

13 – Diabetic patients

8 – Diabetic with hypertension

2 – Septicaemia

8 – Obesity (>30 BMI)

2 – Altered liver function with low protein

1 – Varicose veins

3 – h/o unknown leg swelling, h/o local treatment and drug intake in last 3 months

1 – h/o DVT

Inclusion criteria

The patients who are all undergoing open cholecystectomy, with following indications without any risk factor for DVT

1. Calculus cholecystitis without septicaemia
2. Cholelithiasis
3. Acute cholecystitis with or without calculus without septicaemia
4. CBD stone
5. Mucocele Gallbladder

Exclusion criteria

1. Patients with DVT obvious risk factors
2. Patients with Co morbid conditions known to cause DVT

Key words

- DVT – Deep Vein Thrombosis
- PE – Pulmonary Embolism
- VTE – venous thrombo embolism
- GB – gall bladder
- OC – open cholecystectomy
- OC/CDL – open cholecystectomy with choledocholithotomy

Literature review

DEEP VENOUS THROMBOSIS

One of the most common preventable cause of morbidity and mortality in hospitalised patients is deep venous thrombosis and it s complications like pulmonary embolism and post thrombotic syndromes. So it is essential to know about its incidence, aetiology, pathogenesis, pathology, clinical features, diagnosis, management and its sequalae.

DVT is part of a spectrum of Venous Thrombo Embolism. The remainder being Pulmonary Embolism and post thrombotic sequalae.

Recognition of its risk factors and early management also becomes a important aspect of its management.

Always prevention is better than cure. So every aspect of DVT needs to be understood for its proper management.

Rudolf Virchow's pathophysiologic theory still holds the truth about its development. It is an area where much research is on now and needs still more light on it. A newest area is the response to thrombosis and its amplification response. It is clear now that DVT is a result of multiple factors interplay. And its management needs knowledge about that.

Incidence

The exact data regarding the incidence of DVT in the post operative patients in India is still lacking. In different hospitals it is differing and uniformity is lacking. In USA 1 in 20 is the life time risk of a person developing DVT. Often it is a silent killer. Often the minor occurrence which is the most common thing goes unnoticed.

Populations affected

About 31% of hospital admissions both in medical and surgical wards develop some sort of this DVT. In this population the incidence varies with underlying disease, use of thrombo prophylaxis, the intensity of screening programmes, and the testing modality. For example in spinal injury patients with paraplegia or quadriplegia where the periodical screening is intense shows an 81% incidence. So suspicion on to the occurrence only can diagnose it.In patients of ICU either medical or surgical, the risk is high. In one study it was about 25 to 32%. But in patients receiving thrombo prophylaxis it was about 10 to 18%.

In one interesting study showed that the duration of ICU stay is not related to the DVT incidence. Actually 64% of patients are developing this DVT after discharge only. So duration does not affect the incidence.

Over all the spinal cord injury patients, neurosurgical patients, orthopaedic injury patients are at more risk of developing DVT than simply staying in ICU.

Recurrence

Although being described as an acute event, often it recurs till the cause is completely removed. Often it is not a simple task. And these patients are more at risk of developing PE than just the recurrent DVT alone.

A 7-day case fatality study showed that recurrent PE is more common than recurrent DVT.

Mortality

A 30 year data showed a 26% incidence of PE in one autopsy series. Out of it only 9% were fatal. So it comes around 1% incidence of PE and 0.36 % of death due to PE.

Risk factors

Primary or Idiopathic DVT – no identifiable risk factor

Secondary DVT – in the background of known DVT risk factors

For hospital based studies – availability of objective diagnostic tests, high incidence among these patients, existence of many clinical trials made easy identification of these risk factors.

But for population based studies – less well defined risk factors and criteria's.

In patients (IP) vs. out patients (OP) – different risk factors

OP patient risk factor – malignancy, surgery, trauma within previous 3 months – significant risk factors.

Risk increases as the no of risk factors increases. Three or more risk factors increase significant chances of DVT. The relative risk of developing DVT from 2.4 in those with one risk factor to greater than 20 in subjects with 3 or more risk factors.

Heit and colleagues – older age, male gender, surgery, trauma, hospital or nursing home confinement, malignancy, neurologic disease with extremity paralysis, placing central vein catheter or transvenous pacemaker, history of superficial thrombo phlebitis, and varicose veins.

Among women – pregnancy, OCP use, HRT,

Predictors of recurrent DVT – older age, obesity, malignancy, extremity paresis.

Risk factors in children – local infection and trauma, immobilization, inherited hypercoagulable states, catheters, severe oncologic and infectious diseases and thrombophilia.

Risk Factor for DVT or PE	Odds Ratio
Hospitalization	
With recent surgery	21.72
Without recent surgery	7.98
Trauma	12.69
Malignant neoplasm	
With chemotherapy	6.53
Without chemotherapy	4.05
Previous central venous catheter or pacemaker	5.55
Previous superficial vein thrombosis	4.32
Neurologic disease with extremity paresis	3.04
Varicose veins	
Age 45 yr	4.19
Age 60 yr	1.93
Age 75 yr	0.88
Congestive heart failure	
Thromboembolism not categorized as a cause of death at post-mortem examination	9.64
Thromboembolism categorized as a cause of death at post-morter examination	1.36

Age

Old age – high incidence. A community based study showed increasing incidence of DVT from 0 incidences in childhood to 7.65/1000 in male and 8.22/1000 in female older than 80 years.

Rosendaal study – 0.006/1000 in younger than 14 years, increases to 0.7/1000 in 40 to 54 years old people.

Hansson and colleagues study – objectively documented thrombo embolic events increased from 0.5% at 50 years to 3.8% at 80 years.

Multifactorial influence of age on DVT incidence.

The number of thrombotic risk factors increase with age – 3% DVT incidence in <40 years aged hospitalised patients compared to 30% in >40 years aged patients.

The number of risk factors required for precipitating the thrombotic event is reduced as age advances as the individual factors are getting strengthened.

As the age advances the levels of thrombin activation markers increased like P selectins, tissue factors and procoagulant micro particles.

Soleal veins – anatomically changes are occurring in favour of DVT as age advances.

Low incidences of DVT in hospitalised children are due to early ambulation and early discharge. Also the diagnosis is often not considered in the paediatric age group.

Immobilization

Soleal vein stasis behind the valve cusps is exacerbated by both immobilization and old age. Incidence parallels the duration of immobility. An increase in first3 days and a rapid rise to very high levels after 2 weeks. Preoperative immobilization doubles the chance of post operative DVT risk. Paralysis patients are at increased risk of developing the DVT than non-paralysed. Average is about threefold risk.

Travel

Economy class syndrome – prolonged travel in sitting in a cramped position.

But till now, no rheological or thrombotic changes have been demonstrated.

WHO 2001 consensus

- An association probably exists between air travel and venous thrombosis
- Only a small risk and affects mainly the persons with additional risks
- All forms of travels are associated with risk
- Available evidence does not give the incidence.

History of venous thrombo embolism

Risk is higher in Idiopathic DVT patients where the definitive aetiology could not be established. Fibrous remnants following DVT play the crucial role in DVT recurrence. Other factors like disordered venous hemodynamic, residual damage

to venous walls, abnormalities of coagulation and fibrinolytic mechanisms are also important. However primary hyper coagulability appears to have a significant role in many recurrences.

Simioni and associates reported the recurrence in factor V leiden mutation people was about 40% at 8 years follow up.

Den heijer and collegues reported that 17% of recurrence is due to hyperhomocystinemia.

Malignancy

About 20% of newly diagnosed DVT are associated with malignancy.

Malignancy increases the risk of DVT, a fourfold compared to normal population. 12 to 17% of times the occult malignancy is associated with DVT, where it is considered as idiopathic and often unnoticed. In addition, in patients of recurrent DVT, the incidence of malignancy, in one series is about 17%.

The factors in malignancy responsible for DVT are venous compression by tumour, immobility, thrombocytosis caused by tumour, chemo radiation, central lines, and tumour products like cytokines, procoagulant and inhibitors of fibrinolytic system.

The tumours known to cause DVT are pancreatic malignancies, kidney, ovary, and lung and stomach malignancy. Tissue Factor (TF) is the reason for tumour cell directly activating the coagulation saccade as it binds the VII and VII a

factors and activating the cascade. Cancer Procoagulant (CP) is a other reason for DVT in malignancy, as it directly activates factor X, independent of factor VIIa. CP is mainly indicated in myelogenous leukaemia's, as the reason for DVT. Ib and IIb/III glycoprotein's are also being identified as being released from tumour. It causes platelet activation and aggregation. VEGF, TNF-alpha and IL-1 are also released from tumours which are also the reason for induction of TF on vascular endothelium. Also the IL-1 and TNF down regulate the expression of thrombomodulin on endothelial surface. It also stimulates the production of plasminogen activator inhibitor of fibrinolysis.

The markers of activated coagulation are persistently elevated in malignancy patients. Fibrin peptide A levels reflects the tumour activity. In that it is kept on elevation in malignancy not amenable to heparin, indicating that these DVT are refractory to anticoagulants. The surgical procedure for malignancy and also the chemotherapy for these malignancies are known to risk DVT in these patients. Preoperative elevation of thrombin – thrombomodulin complex is associate with 7.5 fold increase of post operative DVT.

Reported incidence of DVT following treatment is 6% in non Hodgkin lymphoma patients and 17.5% in breast cancer patients and germ cell cancer patients about 11%. The potential mechanism for these DVT include direct endothelial injury, induction of a hyper coagulable state, reduced fibrinolytic activity, tumor cell lysis, and use of central vein catheters. Also some

chemotherapeutic agents are known to activate the coagulation cascade in these patients, which adds further risk.

Surgery

The high incidence in post operative patients, hospital stay, investigations and specialist availability, all these made a better understanding of this condition.

The factors like age of patient, co morbid thrombotic conditions, surgical trauma extent, timing of surgery and post operative immobilization increases the risk of DVT.

The overall incidence of DVT in general surgical patients is 19%.

Based on these factors the patients are categorised as follows

category	Characteristics
Low	Age <40 years, no other risk factors, uncomplicated abdominal / thoracic surgery Age >40 years, no other risk factors, minor elective abdominal/ thoracic surgery <30 minutes,
Modera	Age >40 years, abdominal/thoracic surgery >30 minutes
High	History of recent thromboembolism, abdominal or pelvic procedure malignancy, major lower extremity orthopaedic procedure.

Actually the process of DVT starts in operating table itself and it fully getting established over next 3- 5 days. This process does not stop at discharge and

actually continues beyond also. The re admission after previous discharge actually doubles the risk as the previous risk is getting amplified by this admission. All the components of Virchow's triad are present in post operative patients. Perioperative immobilization leads to venous stasis. Extensive endothelial injury is also present. Because of extensive TF release from surgical trauma, there is a hypercoagulable state in this age group. So there is every chance for DVT to develop waiting the inciting event.

"Post operative fibrinolytic shutdown" increased levels of PAI-I in early post operative period leads to a thrombogenic state. So it leads to a conclusion that levels of PAI-I in early post operative period is a marker of DVT risk.

Trauma

Another group of people, where there is a definite risk of DVT in spite of all measures.

Olmsted county study – 13 fold increased risk of DVT in trauma patients.

Risk factors implicated

- Age
- Blood transfusion
- Surgery
- Bone fracture
- Spinal cord injury

- TF release
- Significant period of immobility
- Paralysis
- Venous injury
- Prolonged hospital stay
- Dehydration chances
- Multiple drugs

Inherited thrombophilia

Prothrombin gene mutation

Guanine to adenine transition at 20210 nucleotide – poort and associates in 1996. 87% of times they have 115% of Prothrombin levels. This mutation is rare in Asian or African population. In heterozygous mutation the risk is threefold and in homozygous it increases further. In 1 to 10% of cases there is co-inheritance of Factor V Leiden mutation.

Factor V Leiden mutation

Replacement of arginine with glutamine at 506 positions is called as this mutation. The mutated factor is resistant cleavage by APC. So more amount of thrombin is generated. Autosomal dominant mode inheritance.In heterozygous states the risk increases by 7fold but in homozygous 80 fold.

Protein C

It is a vitamin k dependant serine protease that inactivates factor X and thrombin.

Deficiency can be both inherited and acquired. **Type I** – heterozygous inheritance – Autosomal dominant inheritance-reduction of both antigenic and functional level. **Type II** – antigen level is normal but the functional level reduced. Acquired deficiency occurs in acute thrombotic events, liver disease, renal disease, disseminated intravascular coagulation, haemolytic uremic syndrome, chemotherapy with asparginase, thrombotic thrombocytopenic purpura and acute infection.

Protein S

It acts as a co factor for protein C action. Autosomal dominant inherited deficiency occurs. More common than protein C deficiency.

Type I – quantitative deficiency

Type II – qualitative

Type III – mutation that increases the affinity of protein S for C4b-binding proteins.

Acquired deficiency – disseminated intravascular deficiency, diabetes mellitus, pregnancy, oral contraceptive use, nephritic syndrome, liver disease and essential thrombocythemia.

Antithrombin

A glycoprotein product of liver, inhibits thrombin and factors Xa,IXa,Xia, and XIIa. Heparin augments the action of this. Deficiency is inherited as Autosomal dominant inheritance. Clinically manifests as thrombosis of ilio femoral veins, upper limb deep veins, mesenteric veins, vena cava, renal veins and retinal veins. Also it may present as cerebral venous thrombosis and budd-chiari syndrome.

Pregnancy

6 to 10 fold increased risk. Risk of thrombosis is 2-3 times more in puerperium compared to pregnancy. Risk is equal throughout all the three trimesters.

Reasons

- Impaired venous flow due to obstruction
- Transient hypercoagulable state
- Increased fibrinogen level
- Acquired functional resistance to APC
- tpa levels reduced. PAI 1&2 increased.

Caesarean increases the risk of DVT compared to normal delivery. Other risk factors for puerperal DVT are maternal age advancement, suppression of lactation, hypertension, and assisted delivery but not with the number of pregnancies.

Oral contraceptives and Hormonal therapy

An independent risk factor. Its OR is 3.8 to 11. 1/4th of idiopathic DVT in young women is due to OCP, which is often unnoticed. The risk is decreased after the withdrawal of OCP. When it is taken by the patients who underwent surgery or associated with inherited disorder of coagulation, the risk increases.

Risk is related to dose of estrogen and type of progesterone. Preparations containing less than <30 to 50 micro g of estrogen carries less risk. Third generation contraceptives containing progestins are associated with two fold risk of DVT. Estrogenic compounds when used for suppression of lactation, cancer prostate, and post menopausal replacement also risk DVT. Hormone replacement therapy has DVT risk, which is more in first year of treatment, and due to adaptation it decreases subsequently. Possible mechanisms are decrease in PAI-I, increase in blood viscosity, fibrinogen, plasma levels of factors VII and X, platelet adhesion and aggregation.

Blood group

A group is associated with 1.41 times more risk of DVT compared to other groups. The safest blood group is O group, due to lower levels of vWF in epithelial surface.

Central venous catheters

They are used mainly for hemodynamic monitoring, infusion lines and pacemakers. They are mainly associated with upper limb DVT. Incidence varies from 28% for subclavian canulation to 12% in femoral vein canulation as it is occurring in cases of trauma. Possible mechanisms are vascular injury and stasis. The catheter material whether Teflon or heparin bonded material is also a deciding factor. Catheter diameter, number of venipuncture attempts, duration of catheter placement and composition of the infusate.

Inflammatory bowel disease

Patients with crohns disease were reported to have 31.4% and 10.3% of DVT and PE. For ulcerative colitis it is 30 and 19.8%. More in patients with active disease. It also depends on the extent of disease, where the extensive disease persons will have repeated DVT episodes. Possible mechanisms are thrombocytosis, elevation of factors V, VIII, and fibrinogen during active episodes, depressed levels of Antithrombin, elevated PAI-I values, and presence of anti-cardiolipin antibodies. And also possibly endotoxin induced monocyte activation.

Systemic lupus erythematosus (SLE)

Multiple arterial and venous thrombosis, recurrent abortion, thrombocytopenia, and neurologic disorders are part of its spectrum. Lupus anticoagulant and

anticardiolipin antibodies may be seen in SLE. Lupus anticoagulant has a six fold and anti cardiolipin antibodies have two fold risk of DVT.

Varicose veins and superficial thrombophlebitis

An independent risk factor. ORs of 3.6 to 6.9. In Young patients with varicosities, it constitutes an important risk factor.

Iliac vein compression

A congenital risk factor.

Compression of left iliac vein by right iliac artery and fifth lumbar artery was identified by May and Thurner.

Left lower extremity venous hypertension with or without iliofemoral DVt is known as May and Thurner syndrome.

Cockett syndrome – acute phase of ilio femoral DVt secondary to compression of the iliac vein with the long term Chronic Venous Insufficiency.

May Thurner syndrome

These syndromes are occurring in young to middle aged women, also after multiple pregnancies. Patients present with left leg pain and swelling. In asymptomatic population CT imaging shows compression of left iliac vein is 35%. Out of it 24% is associated with more than 50% obstruction.

Upper extremity compression

It is more common in those patients who has indwelling central line, canulation and pace makers in situ for to get DVT due to direct injury to vessel wall, and stasis due to canula.

Also in patients of thoracic outlet obstruction where there is risk of vessel wall injury and stasis, post stenotic dilatation also increases the risk.

Paget – schrotter syndrome – primary axillary – subclavian vein thrombosis by repetitive physical activity of upper limb and injury to subclavian vessel and axillary vessel. It is more common in patients like base ball players, swimming, and weight lifting. It will present with arm swelling, cyanotic discoloration, dilated veins of limb, fatigue, heaviness or pain with use.often the patient is young and healthy excepting this.

Popliteal vein entrapment

Anatomic anomalies like medial or lateral head of gastrocnemius is associated with Popliteal vein and sometimes artery also entrapment. Bone tumours and hypertrophied fibrous fascia also are the causes. Venous entrapment is more common in females. The typical patient will be young adult, with signs of chronic venous insufficiency including leg swelling, varicosities, skin changes and occasionally DVT.

Venous aneurysm

These dilatations are associated with high flow states, trauma and additional congenital malformations. Popliteal solitary aneurysms are associated with PE usually as well as venous insufficiency.

Unlike arterial aneurysms, the risk of rupture is very low or negligible.

IVC aneurysms are associated with rupture, embolism, and thrombosis. But thoracic aneurysms are associated with benign course.

Other risk factors

IVC anomalies

Either hypoplasia or absence of IVC, which is usually happening at 6 -8th week of gestation is associated with high risk of DVT. It is postulated to be due to, stasis in collaterals, intimal underdevelopment and associated hyper viscosity. Often the patients are very young to present with. In any adult younger than 30 years with bilateral DVT should be suspected of having these.

Obesity

In post menopausal women with BMI 25 to 30 kg/m2 are associated with higher risk. But in younger age groups it is not an independent risk factor but in presence of other inciting events the risk is high.

Congestive heart failure

Primarily due to venous stasis, when it is associated with other risk factors the risk of DVT increases.

Systemic hyper coagulability

In patients of myocardial infarction and post thrombolytic patients, the hypercoagulability as part of physiologic response is associated with DVT.

Pathophysiology

Virchow's triad

The three components

- abnormalities in blood flow
- abnormalities in blood
- vascular injury

These parameters are subsequently be refined as follows

- flow abnormalities determine the location of DVT
- abnormalities in blood may be either coagulation abnormality or fibrinolytic system abnormality
- biologic injury to endothelium is more important than gross injury

However the origin of DVT is Multifactorial.

Gwendylen Stewart – relation between thrombosis and inflammation. This relationship is more important in the thrombogenic process and evolution of thrombus. Mechanical venous injury plays an important role in direct venous trauma, hip arthroplasty and central venous catheters. Overt endothelial injury is not a significant factor in development of DVT in the absence of other stimuli.

Endothelium and Haemostasis

Normal endothelium provides a vasodilatory and local fibrinolytic environment. It also inhibits coagulation, platelet adhesion, platelet activation, inflammation, and leukocyte activation.

Possible mechanisms are

- thrombomodulin and protein c production
- expression of heparin and dermatan sulphate
- expression of tissue factor pathway inhibitors
- Production of tissue and urokinase type plasminogen activators.
- It also produces NO, prostacyclin, IL-10

During the states of disturbance of endothelium, pro inflammatory and pro thrombotic states occur. PAF and endothelin -1 released by endothelium causes vasoconstriction. vWF, TF, PAI-I and activated factor V cause thrombosis. In inflammation or injury there is also up regulation of P – selectins and E-selectins. This sets the stage for DVT.

Inflammation and Thrombosis

In inflammation following changes are happening

- TF increases
- Platelet activity increased
- Level of fibrinogen increased
- Phosphatidyl serine expression increased
- PAI-I level decreases
- Thrombo modulin and Protein C level and function decreases

Selectins are the first upregulated glycoproteins which increases in inflammation. Venous stasis and ischemia causes upregulation of P selectin and this selectin causes MP expression which leads to prothrombotic stage and eventually DVT.

The receptor for selectin PSGL-1 is highly expressed in leukocytes , platelets and MP's.

P -selectin and receptor interaction -> thrombogenic MP release from leukocyte -> platelets also releases the same-> TF over expression->Phosphatidyl serine rich anionic surface exposed-> coagulation cascade activation.

In DVT, MP's have found to be elevated. These inhibit fibrinolysis. These MP's are co localised in thrombus, they not only prothrombotic but also inhibit fibrinolysis.

Plasminogen system

It is the main fibrinolytic enzyme; with its substrates are fibrin, fibrinogen and other coagulation factors. It also interferes with vWF mediated platelet adhesion. Its activators are being inhibited by PAI-I. PAI-I is synthesized by liver and endothelium. It is stabilised by its binding with vitronectin. Thrombus resolution and vein wall remodelling. Profibrotic growth factors, deposition of collagen, MMP expression and activation – all these leads to resolution. The order of changes happening in thrombus as follows

Neutrophil infiltration of thrombus-> enzymes released from Neutrophil causes fibrinolysis and collagenolysis-> monocyte replacing Neutrophils (levels correlate with MCP-I) -> depletion of CC receptor -2 -> late impairment of thrombus resolution (-> exogenous interferon can fully resolve thrombus.)

The changes in the vein wall are as follows

Elastinolysis -> MMP 2 & 9 elevation-> early vein wall collagenolysis in 7 days-> elevated levels of TGF-B, IL-13and MCP -1. -> Fibrosis with increase in collagen 1 and 3

So to conclude inhibition of inflammation decreases vein wall fibrosis. Inhibition of P-selectin decreases the vein wall fibrosis.

Stasis

Most of thrombosis occur in low velocity flow areas like Soleal veins and post valve areas.

Possible mechanism is the **hypoxia** caused by this inadequate flow causes endothelial damage->cytokine production-> expression of leukocyte adhesion molecule-> adhesion and migration of leukocyte.

Stasis also allows accumulation of activated coagulation factors-> thrombosis

Stasis causes consumption of inhibitors of coagulation -> thrombosis

In large veins, due to low surface to volume, inhibitory effects are less prominent by endothelium. So it promotes thrombosis. But stasis appears to be an inadequate source for thrombosis. And it is a permissive factor only in many studies. It indicates it precipitates much of the existing risk factors.

Activation of coagulation

It is the critical step in the pathogenesis of DVT. The balance between the pro and anti factors only maintains the equilibrium. When the homeostasis is disturbed to coagulation side DVT occurs. Individual factor assay is not much helpful. But the following factors are found to be help full in assaying

- Prothrombin fragment F1+2, it is generated by factor Xa in the cleavage of Prothrombin to thrombin

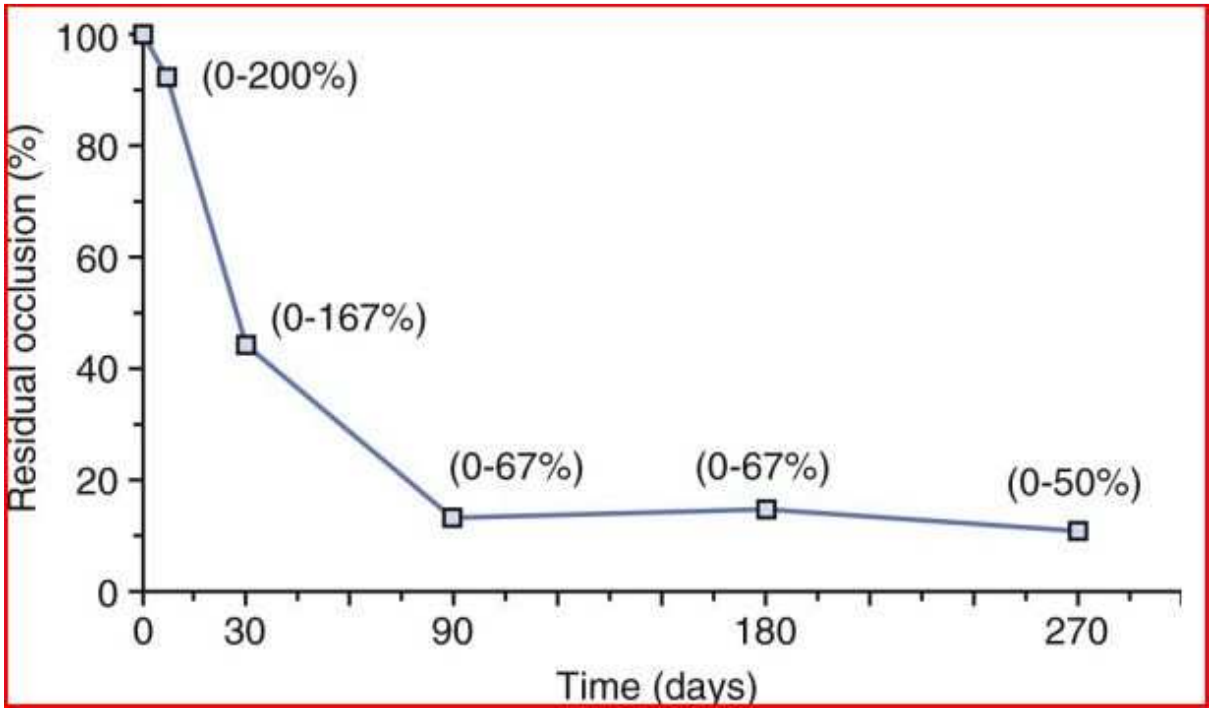
- Fibrinopeptide A , formed in the conversion of fibrinogen to fibrin
- Thrombin – anti thrombin complex
- Fibrin degradation products like fragment D and E. But clinically only the D fraction is being used. D dimer assay used in these settings predict the ongoing risk for recurrent thromboembolism.

Even though only activated coagulation system cannot cause DVT. In the presence of the endothelial injury and stasis only it can cause the thrombosis. Otherwise it is insufficient to cause DVT. So it is a multi factorial causation than a simple factor.

Natural history

Thrombus evolution as Determined by Non-invasive studies. The balance between organization, thrombolysis, propagation, and re-thrombosis determines the outcome after thrombosis. The most important outcome after thrombosis is recanalization and recurrent thrombosis. Impedence plethysmography, a non invasive test detected the improvement after thrombosis. But it cannot differentiate between recanalization and collateral development. Venous duplex ultrasonography can be used for the same purpose and it can able to differentiate between these two. Van Ramshorst and associates – exponential decrease in thrombus load over the first 6 months after bypass surgeries. That too the process occurs in first 6 weeks.

Recanalization begins early after an acute episode of acute DVT that is within 6 weeks, but often it completes within 3 months of the events. Complete thrombus resolution occurs in 56% of times in 9 months. Recurrent thrombotic events often compete with recanalization. So there is often both processes are undergoing. More proximal the DVT more chances of getting PE in these patients. Proximal propagation also occurs in this group of patients. Propagation to new segments in the ipsilateral limb tends to occur at the earliest within 40 days of diagnosis, but contralateral limb extension occurred only rarely.



Complications

Pulmonary embolism

The most devastating complication of acute DVT is PE.

But it is usually silent, but when it floridly occurring it is fatal. Asymptomatic presentation can be up to 80%. Approximately 90% of PE is caused by proximal lower extremity venous thrombosis. Some autopsy series states that the combination of DVT and PE is 1.8 times more common than DVT alone. Modern investigations increased the detection of asymptomatic PE when it is done for some other indication. Symptomatic presentation of PE is not only dependant on amount of vessel occlusion, but also the cardio respiratory reserve of the patient. The risk of death in symptomatic PE is 18 fold higher than DVT patient alone. But in occasional cases the first presentation may be sudden death also.

Pulmonary hypertension

It is also called as Chronic Thrombo Embolic Pulmonary Hypertension (CTEPH). 0.1to 0.5% of individuals who recovered acute episode of PE develop this complication. Its presentations can vary from progressive exertional dyspnoea, Right Ventricular Failure features, edema, chest pain, light headedness, and syncope. But many patients can present with this without any history of PE. So it is appropriate to try the surgical treatment when it is

indicated than medical management which is associated with poor outcomes. So either pulmonary thromboembolectomy or pulmonary endarterectomy can be a appropriate option.

Post thrombotic syndrome

A less common complication than PE, but a significant complication.

It may present as pain, edema, heaviness or hyper pigmentation but severe manifestation occurs only in 7 to 23%. In 4 to 6 % of patients, ulcers may develop. But often the stasis changes are more prevalent.

One of the severe complications is ambulatory venous hypertension. It is associated with valvular reflux, persistent venous obstruction and the anatomic distribution of these abnormalities.

Valvular reflux is an important complication of DVT. About 33% of initially thrombosed segments show evidence of reflux on duplex ultrasonography one year after the event.

A natural mechanism which causes the thrombus organization will not to affect the valve cusp. It is separated from cusp by a clear zone of fibrinolysis caused by cusp endothelium to protect it from process. But it fails in 10% of individuals.

There are several factors to influence the outcome after thrombosis like rate of recanalization, extent of reflux, anatomic distribution of reflux and obstruction and recurrent thrombotic event.

Also a high body mass index is also a risk factor for post thrombotic syndrome.

Reflux is associated with long time for recanalization, and recurrent thrombo embolism. The incidence of venous stasis syndrome continues to rise for 20 years after VTE. It is shown in so many studies.

Prandoni and colleagues – post thrombotic syndrome 22.8% after 2 years, 28% after 5 years and 29.1% after 8 years.

Mohr and associates- venous stasis syndrome is three time more common in proximal DVT than distal type.

Mortality

Usually DVT is associated with co morbid conditions. So the patient is so fragile and cannot withstand this stress.

Kniffen and associates – 21% mortality in first year

Early mortality is very high in upper extremity DVT patients. These patients tend to be more fragile due to usually metastatic malignancy. Six month mortality is 48% in upper extremity DVT compared to 13% in the lower extremity DVT patients.

Calf vein thrombosis

It is a condition where isolated occurrence of thrombosis in calf muscles. This thrombus recanalize early. 50 % reduction occurs in 1 month and complete

recanalization in 1 year. Incidence of reflux is also low. One third to one fourth patients may develop proximal propagation. 10 % of patients may develop PE. Post thrombotic syndromes also can develop in these patients like pain, swelling and recurrent thrombosis. These muscular vein thromboses should be treated with anticoagulation for at least 15 to 30 days and longer than this in high risk patients.

DEEP VEIN THROMBOSIS: CLINICAL AND DIAGNOSTIC EVALUATION

DVT should be diagnosed timely and accurately as the consequences of late diagnosis may be fatal. It may result in pulmonary embolism, post thrombotic syndrome and even death. In many cases the clinical presentation of DVT may not be typical. Newer diagnostic techniques now make the diagnosis of DVT earlier and easier.

CLINICAL FINDINGS

Findings commonly seen are pain, fever, edema, tenderness, pain on dorsiflexion of foot (Homan’s sign) and peripheral cyanosis. 50% patients may not have the typical signs and symptoms.

RISK FACTORs

Risk factors will be present in most cases. They include age, prolonged bed rest, malignancy, trauma, pregnancy, and other factors.80% of patients will have at least one risk factor.

Wells Prediction Rules

Table 1. Wells prediction rules for diagnosing DVT [1,2]

Clinical characteristic	Score (if yes)
Active cancer (treatment ongoing, within previous 6 months, or palliative)	+1
Paralysis, paresis, or recent plaster immobilization of the lower extremities	+1
Recently bedridden > 3 days or major surgery within 12 weeks requiring general or regional anesthesia	+1
Localized tenderness along the distribution of the deep venous system	+1
Entire leg swollen	+1
Calf swelling 3 cm larger than the asymptomatic side (measured 10 cm below tibial tuberosity)	+1
Pitting edema confined to the symptomatic leg	+1
Collateral superficial veins (non-varicose)	+1
Alternative diagnosis at least as likely as deep venous thrombosis [3]	-2

[1] Wells et al 1997. The Wells criteria are available online at: http://www.mdcalc.com/wells-criteria-for-dvt.

[2] In patients with symptoms in both legs, use the more symptomatic leg.

[3] An alternative diagnosis may include: superficial phlebitis, postphlebitic syndrome, cellulitis, muscle strain or tear, leg swelling in paralyzed limb, venous insufficiency, edema due to systemic cause such as CHF or cirrhosis, external venous obstruction (e.g., due to tumor), lymphangitis or lymphedema, popliteal (Baker's) cyst, hematoma, pseudoaneurysm, or knee abnormality.

Table 2. Interpreting Wells prediction rules for DVT

Test	Results	Interpretation
Wells prediction rule for diagnosing DVT	≥ 3	High pretest probability of DVT
	1–2	Intermediate pretest probability of DVT
	≤ 0	Low pretest probability of DVT

Table 3. Recommended test for evaluating suspected DVT in patients with intermediate or high pretest probability (Wells score ≥ 1)

Test	Results	Next steps
Ultrasound [1,2]	Positive	Initiate treatment. (See Table 12 for treatment recommendations.)
	Negative	Consider a D-dimer (if not already done) if there is high degree of clinical suspicion. If D-dimer is positive, order a second ultrasound in 3–7 days. If still suspected, order an MR venogram.

1 Ultrasound is less sensitive for diagnosing DVT limited to the calf than it is for diagnosing DVT in proximal veins.

2 Contrast venography is considered the definitive test to rule out the diagnosis of DVT; however, use contrast venography sparingly due to the morbidity associated with the procedure.

Table 4. Recommended test for evaluating suspected DVT in patients with low pretest probability (Wells score < 1)

Test	Interpretation	Result / facility Reference ranges vary by specific test and location			Next steps
		AMB, BVU, CH OLY, SIL, TCS (GH labs)	EVT (Providence Hospital lab)	All other locations	
D-dimer [1–3]	Positive	> 400 ng/mL	> 0.49 ug/mL	Consult the facility's reference range.	Perform duplex ultrasound with compression.
	Negative	≤ 400 ng/mL	≤ 0.49 ug/mL	Consult the facility's reference range.	Consider alternative diagnosis.

1 This test should be performed on a STAT ("patient waiting") basis.

2 For patients with reliable follow-up, primary care clinicians may consider ordering a routine D-dimer test if the primary care physician can review the results the same day **and** refer the patient to urgent care for further evaluation if indicated. The PCP should contact the urgent care provider to discuss the patient and disposition plan. This step is important for patient safety and quality of care.

3 False positive D-dimer results may be found in the following circumstances:

- Age (> 50 years)
- Heart disease
- Liver disease
- Pregnant women
- Immobility
- Recent surgery
- Active malignancy
- Trauma
- Prolonged/permanent immobility
- Infection
- Sickle cell disease
- Disseminated intravascular coagulation (DIC)

DIAGNOSTIC TESTS

VENOGRAPHY

It was historically the gold standard for the diagnosis of acute DVT, but it has been replaced by newer less invasive techniques. It use has been limited as it involves risk of phlebitis, nephrotoxicity, allergic reactions, cost and need for an adequate intravenous access.

DUPLEX ULTRASONOGRAPHY

It has almost completely replaced venography as the diagnostic test of choice for the detection of DVT. Its advantage is that it is non invasive, lack of radiation, portability and cost effectiveness.

DIAGNOSTIC CRITERIA

It includes assessment of venous compressibility and intra luminal echoes using the B-scan image, luminal colour filling using colour Doppler, venous flow characteristic using Doppler. Of these venous incompressibility is the most widely used objective criterion. Incompressibility has excellent sensitivity and specificity for the detection of proximal DVT. The use of colour flow imaging has also improved the accuracy of detecting isolated thrombi below the level of knee.

REPEAT ULTRASOUND STUDIES

Studies have shown that repeat duplex ultrasound help to distinguish high risk patients who may benefit from vena caval filter placement. The consensus from different studies has been to repeat ultra sound studies when clinical symptoms persist despite a negative initial examination.

D-DIMER

D-dimer is the degradation product of cross linked fibrin by plasmin. Tests are based on the development of monoclonal antibodies capable of differentiating

the degradation products of fibrin and fibrinogen. The presence of D-dimer reflects the presence of intravascular fibrin and it is sensitive for the diagnosis of thromboembolism. The sensitivity for the diagnosis of DVT is as high as 97%, but the specificities are as low as 35%. Elevated D-dimer values are also seen in disseminated intravascular coagulation, malignancy, post operative states, infection, preeclampsia and recent trauma. The specificity of D-dimer measurements diminishes with age.

ASSAYS

Four methods of measuring D-dimer levels in blood are currently available:

Conventional membrane ELISA, Micro plate ELISA, Latex agglutination and whole blood agglutination assays.

ELISAs may have higher sensitivity but agglutination assays may have higher specificity. Several rapid semi-quantitative assay results can be reported in 1hour, with sensitivities equivalent to those of ELISA. These rapid quantitative assays appear to have the best combination of convenience, sensitivity and specificity.

ACCURACY

Due to the high sensitivity of this test, it is possible to exclude a diagnosis of DVT on the basis of negative result. But due to the low specificity a positive D-dimer assay will need another confirmatory testing.

LIMITATONS

D-dimer levels are falsely elevated in malignancy, pregnancy, recent post operative state and total bilirubin level more than 2mg/dL.

Despite its limitations, D-dimer is a useful tool to rule out DVT as long as the threshold is kept low so as to keep the sensitivity high.

MAGNETIC RESONANCE VENOGRAPHY (MRV)

MRV has been developed for the imaging of venous circulation. It takes advantage of the ability of magnetic resonance to distinguish stationary from moving signals and it has gained momentum in recent years for the detection of DVT.

It is less costly than contrast venography an also overcomes the limitations of contrast – enhanced ultrasound and Impedence plethysmography. Since MRV directly visualizes the thrombus, even non-flow- limiting thrombi can be detectable.MRV can also be able to detect thrombi proximal to the inguinal ligament, an area that has been problematic for duplex ultrasound in the past. Also in contrast to contrast ultrasound, MRV results are independent of technician availability and experience.

Different types of MRV protocols have been used for the detection of DVT, including non- contrast-enhanced MRV techniques like time of flight (TOF) and phase contrast as well as contrast enhanced MRV (CEMRV). TOF MRV and

phase contrast MRV rely on low- related enhancement and velocity shifts to provide signals from flowing blood. For magnetic resonance arteriography, CEMRV provides rapid three- dimensional sequences after timed injection of a contrast agent and post processing algorithms to remove the arterial signal. Post processing techniques allow the creation of three – dimensional images analogous to those obtained with conventional venography.

ACCURACY

Studies have shown that the results of MRV when compared to contrast venography were almost similar.CEMRV has shown to be more reliable than venography in predicting proximal extent of DVT. CEMRV has been shown to have a sensitivity of 100% for iliac and femoral vein DVT with corresponding specificities of 100% and 97%.

DISADVANTAGES

There are few disadvantages that limit its utility as a screening test. One disadvantage is the cost which is higher than most other DVT tests. Other disadvantages involve patient characteristics, methodology, and safety concerns. Patients with certain implants may not be able to undergo testing. MRV has limitations related to imaging of calf veins. The paired nature of tibial veins and their proximity to arteries create significant artefact during post processing.

Methodological disadvantages include slow acquisition times and susceptibility to flow artefacts in parallel vessels,(common iliac veins).CEMRV overcomes these disadvantages through the use of three- dimensional acquisition sequencing.

Lastly, concern has been raised about the safety of gadolinium in patients with renal insufficiency, as there are some evidences which suggest that gadolinium is associated with nephrogenic systemic fibrosis. Additionally the inflammatory changes associated with DVT can cause gadolinium to accumulate within the vein wall and perivenous tissue. This characteristic enhancement pattern fades with thrombus organization, thus allowing differentiation between acute and chronic DVT.

ADVANTAGES

MRV complements duplex ultrasound in some cases, in that the increased ability to detect proximal extent of venous thrombi may advantageously affect clinical decision making.

The accuracy of MRV in imaging the iliac and central venous circulation makes it more useful for evaluation of pelvic or central vein thrombosis.

MRV has also shown to superior in detecting hypo gastric vein thrombosis. In future the ability to combine pulmonary magnetic resonance angiography and MRV of the lower extremities may provide a valuable diagnostic tool.

COMPUTED TOMOGRAPHIC VENOGRAPHY (CTV)

This is widely used for the diagnosis of pulmonary embolism. With the introduction of (CTV) a single scan may become a practical a practical test for both DVT and pulmonary embolism.

Studies have shown that CTV is specific but not very sensitive for the detection of DVT. Also CTV has not been investigated for the detection of isolated calf vein thrombosis.

IMPEDENCE PLETHYSMOGRAPHY (IPG)

IPG is based on the principle that the Impedence between two points on the skin of an extremity will decrease as the volume of blood contained in the extremity increases. IPG measures the rate of venous outflow from the calf after an inflated proximal blood pressure cuff is deflated, thus deducing the presence or absence of venous outflow obstruction. The presence of DVT in the major vessels of the lower extremity, including popliteal vein and proximally, should reduce the rate of venous outflow and subsequently affect the tracing.

ISOTOPE SCINTIGRAPHY

New radio pharmaceuticals have been approved for the imaging of DVT. Tc-apcitide is a complex of the radionuclide technetium 99m and apcitide is a small synthetic peptide that binds the glycoprotein 2b/3a receptor on activated platelets and is specific for acute thrombosis. Imaging is done 10 to 60 minutes after

administering the radionuclide. Venous segments above and below the knee are targeted. This technique has the advantage of being functional rather than depending on anatomical structural changes in the vein. Thus it may help in avoiding some problems associated with venography and ultrasonography.

Prevention of DVT

Primary versus secondary thrombosis prophylaxis

Primary – given prophylactically in pre operative and early post operative patients.

Secondary – after the onset of thrombosis to prevent further propagation of thrombus.

As many a times the thrombosis events start primarily in operation table as it is shown in many studies, primary prophylaxis is gaining importance. When compared to olden times, the duration of stay in hospital is much shorter nowadays. And patients are being mobilised earlier.

In view of all these facts the patients are being advised to continue the prophylaxis after discharge also.

Points to start post discharge prophylaxis are age older than 60 years, previous VTE, advanced cancer, anaesthesia lasting longer than 2 hours and bed rest for more than 3 days. In this group of patients it is wise to start post discharge comfortable prophylaxis in order to prevent DVT.

Evidence based versus Real clinical practice

Over the last few years there are so many evidence based guidelines provide optimal DVT prophylaxis. But the clinicians at work point are not often adhering to these guidelines. Either over treatment for sub optimal problems or under treatment for florid problems is happening. Recent ACCP guidelines say that with adequate literature knowledge and clinical judgements, each patient should be viewed. The decisions should be made accordingly. In such times the patients risk factors for DVT, potential advantage and disadvantage of prophylaxis regimen has to be weighed and accordingly to be started. Local available options mechanical, pharmacological or combined has to be decided.

Risk for venous thromboembolism

It is the single most important factor to find out the risk factor in time. Timingly intervention prevents the evolution of risk factor to frank DVT.

Risk for Venous Thromboembolism in Surgical Patients without Thromboprophylaxis

	DVT (%	95% CI (%	Symptomatic PE (%)	95% CI (%
General surgery	24	23-26	1.6	1-2
Urologic surgery	10	6-15	9	3-19
Gynecologic surger	16	13-19	1	0-3
Total hip replaceme	44	42-47	3	2-5
Total knee replacem	27	22-32	—	
Hip fracture surgery	37	35-40	6	4-7

Risk assessment models

Appropriate prophylaxis is to be given for individual patients according to his risk level.

Low risk patient – no need for routine prophylaxis

Moderate risk – physical methods are given with a close observation to detect DVT

High risk – individualised form of prophylaxis should be given

If the patient is diagnosed to have multiple risk factors then some form of prophylaxis tailored to the patient must be given. As the number of risk factors increases the need for prophylaxis also increases.

ACCP guidelines do not advice prophylaxis in cases of foot and ankle surgery, vascular surgery, and laparoscopic procedures unless the patient has some other risk.

Usually the patients are assessed by simple questionnaire listing all the risk factors. And the patient has to make option in the list which will be given marks and all the factor will be summed to give the risk.

VTE is the leading cause of mortality in post operative patients. So measures to prevent it and treating it should be prioritised.

Like giving importance for bed sore, each patient should be given importance for DVT.

Each risk factor is given highest score of 5, which indicate the incidence of DVT 40 to 80% in this group of patients.

In abdomen surgery done for cancer, if it is done for cancer the risk is 36%, and if it is done for benign indication the risk is 20% only.

As the age increases the risk also increases. So the higher the scores are given in aging populations.

Standard guideline models are ACCP guidelines, Caprini score and International Union of Angiology consensus statement.

Despite these guidelines adequate prophylaxis is often not given.

Methods of prophylaxis

As per virchow's triad 3 components are present.

Hyper coagulability is common in following situations

- post operative period
- trauma
- sepsis
- cancer
- hereditary thromphophilia

Likewise venous stasis is playing major role as it causes failure of clearance of activated coagulation factors and reduces the chance of binding with their inhibitors.

Stasis also alters the laminar flow, which causes the platelets and other blood cells to come in contact with endothelium and adherence.

Vascular damage, as it occurs in dilatational trauma of endothelium while surgery, causes the sub endothelial exposure to blood stream which causes the pro coagulable state.

Methods available for DVT preventions

Mechanical – improves venous flow and reduces the stasis

Pharmacological – mainly acts by reducing the hypercoagulability

Combined – often both the methods are combined for better results

General measures

Adequate hydration

Adequate analgesia

Early ambulation

Active flexion and extension exercises if patient is bed ridden

Assistance of nurses and auxiliary personnel is also important to carry out aforementioned things.

Leg elevation has dual effects. First it reduces the swelling thereby increase the venous outflow. Secondly it reduces the venous pressure by its gravitational effect. So it is better to be on bed with leg elevated than sitting in chair with legs hanging.

Mechanical method of prophylaxis

These have not been studied extensively. It is not given as single treatment modality rather given to patients to whom anti coagulants are contra indicated.

These modalities can be either passive compression as in the case of graduated compression stockings (GCS) or active as in the case of intermittent pneumatic compression (IPC) or foot pumps.

Apart from stasis reduction these methods also activate fibrinolytic system of endothelium.

Elastic compression stockings

They reduce the cross sectional area of veins and also increases the venous flow. The pressure created in this is gradient.

At ankle level – 18 mmHg, at upper portion of thigh – 8 mmHg, is created. It increases the venous flow velocity up to 75%.

It can be either thigh length or calf length. It does not make any outcome difference. But calf length ECS is easy to apply, less expensive, and better accepted by patients.

The incidence of DVT is reduced from 29% in control group to 15% in study group favouring ECS.

For post operative patients it showed a still better response by reducing about 69% of risk compared to control group.

It can be used in moderate risk group patients as a single modality option.

Limitations – lack of international standardization, physical problems like application, poor patient compliance, cannot be used in presence of occlusive arterial diseases, absent foot pulse, ABI< 0.8

Contraindicated in massive leg edema, Congestive cardiac failure patients, dermatitis like conditions.

Advantages- safe, no bleeding risk, easy to use, inexpensive, possibility to combine with other modalities.

Intermittent pneumatic compression

Its application in lower limbs is extensively studied.

It consists of pneumatic boot or garments – wrapped around foot, electrical compressor to intermittently compress the foot.

Hemodynamic studies showed IPC enhances venous return by intermittent compression.

IPC avoids venous stasis by intermittent pumping of leg veins.

Various commonly used parameters

- maximum pressure use 35 to 55 mmHg
- inflation time 10 to 35 seconds
- deflation period 1 minute

Device was designed to detect the functioning of this equipment to detect change in venous volume.

IPC – improves venous hemodynamic, increases the fibrinolytic activity by increasing prostacyclin and tPA levels in blood, and it also increases the levels of tissue factor pathway inhibitors.

In various studies, it is an important method of reducing the DVT in post operative DVT in various surgical specialities. It reduces the risk of DVT by about 60%.

Of the existing prophylactic methods, IPC is the best in orthopaedic surgeries. When it is used in combination with warfarin, their efficacy improves to the highest level in orthopaedic population.

IPC started immediately before surgery in operation theatre and continued in post operative period until patient resumes normal mobility.

As like GCS, whether to use above knee type or below knee type is still controversial.

IPC is used in following conditions

- patients at high risk of bleeding complications
- when pharmacologic agents are contraindicated

In one study, it is shown that up to 9% of individuals have contraindication to use of pharmacologic prophylaxis. In this subset, only mechanical methods can be used.

Adverse effects

- skin irritation
- peroneal nerve palsy
- skin pressure necrosis
- improper sleeve placements leading to under working of machine

Contra indications

- Skin infections
- Cellulitis
- Erysipelas

- Massive edema – will lead to increase in preload
- Established DVT
- Peripheral arterial diseases

Limitations

- Low compliance by Para medical persons
- Wearing the IPC sleeves
- Need for continuous monitoring and education of patients, attendees and nurses

Foot compression devices

It consists of inelastic slippers or boots with an air bladder.

Inflation up to 200 mmHg over a period of 3 seconds period for every 20 seconds is used.

So the plantar compression by this pressure causes stasis reduction and increases the venous outflow.

Foot pumps reduce the incidence of DVT by about 77% compared to placebo in many studies.

But is sole use in prevention of DVT in limited. So it is used when there is contraindication exist for pharmacologic methods present. And also in conditions when IPC cannot be used as the mechanical method.

Contraindications and Precautions for the Use of Anticoagulant Agents the Prophylaxis of Venous Thromboembolism

CONTRAINDICATIONS

- Potential bleeding disorders
- Hemophilias
- Thrombocytopenia (<70,000 platelets/mm^3)
- Active or recent bleeding
  - Esophageal varices
  - Active peptic ulcer
  - Intracranial or gastrointestinal bleeding in the past 3 mo
  - Intracranial aneurysm or angioma
- Allergy to heparin
- History of heparin-induced thrombocytopenia
- Acute bacterial endocarditis

PRECAUTIONS

- Liver failure
- Renal failure
- Multiple trauma and head trauma
- Recent intracranial, spinal, or ophthalmologic surg
- Neuraxial anesthesia

Avoid heparin, low-molecular-weight heparin, or fondaparinux injections cl
to possible surgical incisions, drains, and laparoscopic trocar sites.

Pharmacologic methods of prophylaxis

As mentioned earlier, this method counteracts, hyper coagulabity component of Virchow's triad. Various components are sub cutaneous Unfractioned Heparin (UFH), Low Molecular Weight Heparin (LMWH), dextrans, and oral Vitamin K

Antagonists (VKA). More recently more promising molecules are also coming up.

Unfractioned Heparin (UFH)

It is the first method used. 5000 units were given 2 hours before surgery followed by 10000 to 15000 units / day subcutaneously.

Kakkar and associates – low dose heparin significantly reduces the DVT and PE similar way higher doses do.

UFH is associated with minor bleeding complications when used post operatively in 50 % of times. So it created a panic in those with less literature update. UFH reduced the DVT incidence by about 56% and PE incidence by 30% compared to placebo. UFH is twice a day in moderate risk patients and thrice a day in high risk patients, subcutaneously.

In one study, the wound hematoma rate was 3.8% to 6.2% when used in post operative patients.Nowadays it is mainly used in general, gynaecologic and urologic surgeries than any other specialities. UFH has been associated with Heparin Induced Thrombocytopenia in up to5% of individuals. In this condition, there is formation of antibodies against platelets. So it causes precipitous fall in platelet count, often below 10000. In 20% of patients it causes heparin related thrombocytopenia. UFH has a half life of about 0.5 to 2 hours. This short half life is taken as a advantage in renal failure.Main advantage of using this is the

availability of protamine sulphate, in case where there is a need for rapid reversal.

Low Molecular Weight Heparin

Its average molecular weight varies between 4000 to 8000 Daltons. These fractions have greater activity against factor Xa. They are able to prevent thrombosis without increasing bleeding risk. They have better bio availability and longer half life than UFH. They have low binding ability to endothelial cells, macrophages, and circulating proteins. So it has a predictable action than UFH. It is good for single daily dosing. They don't need laboratory monitoring. It is the Ideal drug for outpatient monitoring.

In one study, it shows there is no difference in their efficacy between UFH and LMWH, without increasing bleeding complication.

Mismetti and colleagues analysis-

- LMWH are better for reduction of both symptomatic and asymptomatic DVT compared to placebo.
- LMWH compared to UFH also shows trend towards LMWH
- Below 3400 anti Xa units daily showed to be effective and safe compared to UFH

Bergqvist and colleagues – higher doses of LMWH is better for cancer patients, because of their hyper coagulability.

In total hip replacement, UFH is better than LMWH. It is shown in many studies conducted in last 20 years. But when compared to warfarin, heparin either in toto or its fractions shows a better result.

In summary, heparin either regular or fractionated by fractionation and depolymerisation technique is better. LMWH is the stay nowadays in both medical and surgical wards. Even though they are collectively called, each has different quality and actions. So should not be given in exchange.

Oral Vitamin K Antagonists

Coumarin derivatives are the agents that are commonly used. Synthesis of active clotting factors II, VII, IX and X needs carboxylation of glutamic acid residues. Warfarin is the most commonly used drug .It is administered orally. Dosage – in fixed dose it does not need laboratory monitoring as they are well studied before. In adjusted doses it is necessary to monitor the treatment regularly. For monitoring purpose PT and INR are being used. In high risk patients INR to be maintained between 2 and 3.Most of these drugs need 3 to 4 days to reach the therapeutic plasma concentration. So on the evening of surgery these drugs are started, so that the required concentration comes around 4^{th} post operative day.

Advantages – oral administration,

Disadvantages – risk of bleeding, laboratory monitoring, drug interactions, diet related absorption changes. The effect can be neutralised with Vitamin K,

Prothrombin concentrates and fresh frozen plasma. Even then they are inferior to LMWH. But it is commonly used by orthopaedic surgeons for prophylaxis.

Aspirin

Aspirin irreversibly inhibits cyclooxygenase – 1 enzyme and further production of thromboxane A2.Due to its irreversible action, the action lasts for about 7 -10 days , the platelet half life. It is mostly used in arterial conditions requiring anticoagulation. They don't have any difference between low and high doses either in anticoagulation or bleeding risk. But it is often inferior to other methods like Heparin.In hip fracture and total hip replacement, aspirin is found to be better than other methods.

New Antithrombotic Anticoagulants

Limitations with currently available anti coagulants

- Insufficient efficacy in special situations
- Subcutaneous administration
- Risk of thrombocytopenia
- Need for laboratory monitoring
- Narrow therapeutic margins

So, in order to overcome all these, major effects are being taken to develop drugs that will inhibit single enzyme and avoiding all the complications. Subsequently these drugs will replace UFN and LMWH.

Factor Xa Inhibitors (Indirect: Fondaparinux and Idraparinux)

Fondaparinux

A synthetic pentasaacharide. Inhibits factor Xa by making conformational change in Antithrombin molecule. High bio availability.Subcutaneous injection.17 hours half life.It does not cause thrombocytopenia and osteoporosis.It should be started 6 to 8 hours after the end of surgery. As long as the drug is continued there is substantial reduction of DVT risk, as shown in 7 and 30 days DVT incidence studies.

FDA has approved for the following indications

- Total hip replacement
- Total knee replacement
- Hip fracture surgery

PEGASUS study – high risk abdominal surgery

- 2.5 mg post operatively studied versus dalteparin
- No difference of bleeding complications

Idraparinux

130 hours half life.Once a week administration

Disadvantage – long half life without antidote

Direct Factor Xa inhibitors – Rivaroxiban

Direct selective reversible inhibitor of factor Xa.Oral drug.Absorbed rapidly.Maximum plasma concentration after 2 to 4 hours.Half life between 5 and 9 hours.Initial dose given 6 to 8 hours after surgery.Acceptable safety limits and no need for laboratory monitoring.10 mg daily is now accepted as standard dose.Yet much result about the drug is awaited.

Direct Thrombin Inhibitors (DTI)

Normally thrombin converts fibrinogen to fibrin. Activates factor V, VIII, and XI. Stimulates platelets, stabilises fibrin by activating XIII. It directly binds to its catalytic site. Heparin inhibits only the free thrombin. But clot bound thrombin causes further propagation of thrombus.On the contrary, DTI's inhibit both free and clot bound thrombin. They do not bind with plasma proteins and will not interact with platelet factor. It will not cause thrombocytopenia.

Hirudin

Leeches saliva derivative. First DTI used. Desirudin is a recombinant product of Hirudin. Subcutaneous injection. Mainly used for hip arthroplasty post operative patients to prevent VTE.Bleeding complication is high with this drug.Hirudin and argatroban, a new DTI, have got approval for Heparin Induced Thrombocytopenia (HIT)

Dabigatran Etexilate

Orally administered DTI. High affinity and specificity for thrombin. Peak plasma concentration occurs in 2 hours. Half life after single dose is 8 to 10 hours. But after 14 to 17 hours, it increases up to 14 to 17 hours. Usually started after 1 to 4 hours after the end of surgery.Advantages – orally administered, no need for routine monitoring, fixed doe regimens are possible.Combination of mechanical and Pharmacological methods

As the result of combining both mechanical and pharmacological methods, two of three risk factors of Virchow's triad are being addressed.Both are complementary to each other not antagonist.Combining stockings of any type or IPC with either heparin or others results in better outcomes and protection.

In Cochrane collaboration review, it is shown about 80% increased reduction achievement when combined modalities are being used.

Apollo study – IPC and Fondaparinux vs Fontaparinux alone – results showed combined modality is better option than either of these alone.

Recommendations for Prophylaxis of Venous Thromboembolism in Surgical patients.

The type and duration of surgery and presence of risk factors only ultimately decide the need.In patients of low risk – early ambulation and observation – no need for pharmacological prophylaxis.In moderate risk patients – UFH 5000

units twice daily, LMWH < 3400 units once a day- some groups say only mechanical methods. In high risk patients – active cancer, previous VTE, multiple risk factors – combined modality treatment. Duration of prophylaxis – usually until discharge. For abdomen cancer surgery – LMWH for 3 to 4 weeks. If the patient has active bleeding or high risk for bleeding complication – mechanical methods should be used. Once the complication settles then it is changed to pharmacologic method.

Agent	Dose	Initial dose
Unfractionated heparin	5000 U every 8-12 hr	1-2 hr before surgery
Vitamin K antagonists	5-10 mg (warfarin) (INR 2.5)	Day of surgery or 1st postoperat day
Fondaparinux	2.5 mg	6 hr after end of surgery
Low-molecular-weight heparins		
Moderate risk		
Dalteparin	2500 U daily	1-2 hr before surgery
Enoxaparin	20 mg (2000 U) daily	1-2 hr before surgery
Nadroparin	2850 U daily	2-4 hr before surgery
Tinzaparin	3500 U daily	2 hr before surgery
Bemiparin	2500 U daily	2 hr before or 6 hr after surgery
High or highest ris		
Dalteparin	5000 U daily	8-12 hr before surgery
Enoxaparin	40 mg (4000 U) daily	10-12 hr before surgery
Nadroparin	≥3800 U daily	2-4 hr before surgery
Tinzaparin	50 U/kg daily	2 hr before surgery
Bemiparin	3500 U daily	2 hr before or 6 hr after surgery

Risk Le	Prophylaxis Regimen
Low risl	Early and aggressive mobilization
Modera risk	LMWH at low doses (<3400 U daily)
	Unfractionated heparin (5000 U twice daily)
	Elastic stockings (18-23 mm Hg) or IPC, especially in patients in wh anticoagulants are contraindicated
High ris	High-dose LMWH (>3400 U daily)
	Unfractionated heparin (5000 U every 8 hours)
	Elastic stockings (18-23 mm Hg) or IPC, especially in patients in wh anticoagulants are contraindicated
	Combination of high-dose heparin or LMWH with stockings or IPC
Highest risk	High-dose LMWH (>3400 U daily)
	Unfractionated heparin (5000 U every 8 hours)
	Elastic stockings (18-23 mm Hg at the ankle) or IPC, especially in patients in whom anticoagulants are contraindicated
	Combination of high-dose heparin or LMWH with stockings or IPC
	Warfarin for patients undergoing hip or knee arthroplasty (target INF 2-3)
	Fondaparinux (2.5 mg daily)
	In patients undergoing major orthopedic or abdominal cancer surgery consider extending prophylaxis with LMWH for 28-35 days

Timing of prophylaxis

1 or 12 hours before surgery. In one study fondaparinux and dalteparin compared, no advantage of either drug in 6 or 8 hours after the end of surgery has been concluded.

Vascular surgery

Patients undergoing vascular surgeries receive several antithrombotic agents, anti platelets and anti coagulants. So the incidence of DVT is low. Risk factors in these patients are advanced age, limb ischemia, long duration of surgery, and intra operative local trauma including venous trauma. Latest consensus says that no special prophylaxis is needed in addition to all those. Based on individual risk factors only type of prophylaxis, duration and starting time are to be started.

Multiple trauma

Spinal injuries are associated with highest incidence of VTE and PE. PE is the third leading cause of death. All trauma patients should receive either physical or pharmacological method of prophylaxis. The greatest risk is present in the first three months. Routine duplex scan is advised. IVC filters role is still controversial.

Laparoscopic surgery

Theoretically it is a less thrombogenic surgery. Reasons – less tissue trauma, early ambulation.

But in surgeries like cholecystectomy and hiatal hernia – reverse Trendelenberg position, 12 – 14 mmHg pneumoperitoneum – impaired venous return, venous stasis.

In laparoscopic cholecystectomy, the incidence of symptomatic DVT, PE, and fatal PE are 0.02%, 0.03% and 0.02%.

A recent prospective study revealed 16% of DVT incidence in OC compared to 7% incidence of DVT in LC.

The recommendations of Society of American Gastrointestinal and Endoscopic Surgeons are given in the following guidelines.

Procedure	Risk factor	Recommendation
Laparoscopic cholecystectomy	0 or 1	None, IPC, LMWH, or UFH
	≥2	IPC, LMWH, or UFH
Laparoscopic appendectomy	0 or 1	None, IPC, LMWH, or UFH
	≥2	IPC, LMWH, or UFH
Laparoscopic Nissen procedure	0 or 1≥2	IPC, LMWH, or UFHIPC and UFH or LMWH
Laparoscopic splenectomy	0 or 1≥2	IPC, LMWH, or UFHIPC and UFH or LMWH
Other major procedures	0 or more	IPC and UFH or LMWH

Bariatric surgery

Patients with >30kg/m2 BMI are at increased risk of DVT.

In one mayo clinic study, about 50% of all fatal PE occurred in obese.

All obese patients should receive prophylaxis.

Doses of drugs also need to be increased about 25%.

Patients with 35 to 40 kg/ m2 BMI are at increased risk of prolonged surgery and restricted post operative mobility.

0.1 To 2% of bariatric surgery patients who received prophylaxis also developed VTE.

Risk factor in bariatric surgery patients

- severe chronic venous insufficiency of legs
- BMI greater than 60kg/m2
- Abdominal obesity
- Sleep apnoea syndrome
- History of DVT
- Smoking
- Anastomotic leak
- >50 years of age

Recommended prophylactic methods

- Heparin SC at 5000 to 7500 units every 8 hours
- Intravenous infusion at 400 U/Hr
- Heparin dose adjusted to plasma anti Xa activity between 0.11 and 0.25 U/hr.
- Enoxaparin 40 to 80 mg/ day
- Nadroparin 5700 to 9500 U/day
- Dalteparin 5000 U/day

Mechanical methods are not used as the sole method. When used with pharmacologic methods it definitely augments other methods and reduces the incidence of DVT. It is shown in many studies.

Duration of prophylaxis is not yet concluded. Studies are still on these areas.

Cancer surgery

Normally this group of patients have a fourfold risk of VTE.

When chemotherapy is instituted it further increases the risk.

In addition these patients are at increased risk of recurrence.

The rate of VTE is high in cancers of lung, pancreas, colon, stomach, kidney and prostate. .

When these cancer patients undergo surgery, the risk further increases twofold in post operative period.

In one study DVT incidence in general surgery, with cancer patients was about 37% and 20% without cancer.

In cancer patients, there is platelet activation and aggregation and hypercoagulability secondary to procoagulant released by tumours as well as increased release of tissue factors.

In addition vessel wall damage during radical surgeries, central lines, chemotherapy and radiotherapy causes further risk.

Vessel compression by tumours causes venous stasis also in these patients.

The patients undergoing these surgeries should be given 5000 units, 8 hourly regularly.

Due to hypercoagulable states these patients are given higher doses of drugs to compensate it.

The recommended LMWH are 40 mg of enoxaparin and 5000 units of dalteparin. For other LMWHs, >3400 units daily are to be given.

In abdomen surgeries fondaparinux 2.5mg daily is recommended.

Regular duration of prophylaxis is at least 7 days. But for 4-5 weeks is given in abdomino pelvic cancer surgery, residual malignancy patients, obesity and history of DVT.

20 to 25% of all patients with symptomatic VTE have cancer in some form.

Orthopaedic surgery

Total hip/knee replacement surgeries and fractured hip definitely need prophylaxis.

VKAs are most commonly used. Because it is taken orally and associated with less post operative bleeding.

Fondaparinux has shown the best venographic clearance rate compared to other drugs with no bleeding complication risk.

Prophylaxis in spinal surgery is not well studied, as this group of patients routinely given prophylaxis.

Neurosurgery

These are the patients at very high risk of bleeding, as they cannot be given pharmacologic prophylaxis, because of catastrophic bleeding risk. All these patients are given routine IPC or GCS along with UFH or LMWH appropriately.

Inferior vena cava filters are also tried with varied success rates.

Timing and duration of prophylaxis

Timing

Fibrinogen scan data – about half of DVT, process starts in operating table itself.

Death from fatal PE is reduced by 50% in last 30 years, as shown in many studies. In these studies, anticoagulants were given 2 hours pre operatively and they were also associated with less bleeding rates. In one recent study, eliminating the pre operative dose reduced the bleeding complications and associated with no change in efficacy.

Duration - One recent study showed 40% of DVT is occurring in more than 21 days after surgery. So it is necessary to extend the period of prophylaxis till this period of risk.

In one study comparing 7 days versus 30 days LMWH, showed that 30 days heparin is associated with less incidence of DVT. Individual patient should be considered for duration deciding.Bleeding complications in surgical patients receiving anticoagulants6% of general surgical patients developed post operative bleeding after prophylaxis with UFH. Similarly 2.8% of major haemorrhage and 3.8% wound hematoma are associated with LMWH prophylaxis.0.2% of GI bleeding and 0.1% of retro peritoneal bleeding were reported. In major orthopaedic injury 2% of times major bleeding is associated. The timing of administration is a important deciding factor in orthopaedic surgeries. The current recommendation is to wait 8 – 12 hours post operatively for initiating LMWH and 6 hours minimum for fondaparinux.

Neuraxial anaesthesia in prophylaxis patients

American Society of Regional Anaesthesia

- Regional anaesthesia avoided to the possible extent
- Minimum time gap between LMWH to spinal injection should be 10 to 12 hours
- If hemorrhagic aspirate is present initially, then procedure should be abandoned
- Single shot spinal is advised than epidural
- Minimum of 2 hours should be allowed from LMWH to spinal needle placement

All these patients are given some form of mechanical prophylaxis.

Physician attitudes and practices

Recently published data showed that only 55% of physicians are adhering to the guidelines for treatment.

They use LMWH lesser time than expected.

Lacking areas are inadequate dosage, inadequate bridging of heparin to warfarin and failure to give post operative prophylaxis.

Ultimately the already treated patients are 50% of times are at risk of developing thrombosis subsequently.

Hospitals should have individual policy for these regimens and adherence should be evaluated periodically.

Prophylaxis for medical patients

50 to 75% of all cases occur in medical background than surgical back ground.

GCS alone or 5000 units of UFH, twice daily was the reason for all these failures.

Anticoagulant prophylaxis reduced the incidence of clinical and fatal PE. Both UFH and LMWH are used in these patients. But co morbid should be considered before giving these drugs.

Treatment

Objectives – prevent thrombus extension, early recurrence, and death from PE. Also to prevent late recurrence and PTS and chronic pulmonary hypertension.

Mobilisation and Leg elevation

Early ambulation should be done. This causes pain and swelling to come down early. Compression stocking are to be done at the earliest.

Anticoagulation

As soon as to be started. High clinical suspicion or objective evidence is needed. Either IV or SC UFH or LMWH is to be given along with VKA. This overlap for about 5-10 days is necessary, with discontinuing the UFH or LMWH after the INR reaching 2 to 3 for two consecutive days.

aPTT (sec)	Heparin Dose
Initial dose	80-U/kg bolus, then 18-U/kg/hr IV infusion
<35	80-U/kg bolus, then increase 4-U/kg/hr infusion
35-45	40-U/kg bolus, then increase 2-U/kg/hr infusion
46-70	No change (corresponds to 0.3-0.7 anti-Xa U/mL)
71-90	Decrease infusion rate by 2 U/kg/hr
>90	Stop infusion for 1 hour, then decrease infusion by 3 U/kg/hr

Unfractionated Heparin

IV regimen – 80 IU/kg bolus followed by 18IU/kg/hr continuous infusion. The aPTT is done 6 hours after first bolus dose and titrated accordingly.

SC regimen – 5000 units SC, followed by 17500 IU twice daily to achieve the aPTT of 1.5 to 2.5.

Low Molecular Weight Heparins

100 anti Xa U/kg every 12 hours or 150 anti Xa U/kg every 24 hours for enoxaparin. 100 anti Xa U/kg twice daily or 200 anti Xa U/kg every 24 hours for dalteparin. 175 anti Xa U/kg every 24 hours for tinzaparin.

Fondaparinux

Subcutaneous injection, once daily, 5, 7.5, 10 mg OD, according to INR

Thrombolysis

It is indicated for painful, swollen limb, skin changes, ulceration and functional disability. Catheter directed therapy is best. Access is via popliteal vein. Also we can stent the area in case of anatomic abnormality.

ACUTE DEEP VENOUS THROMBOSIS: SURGICAL AND INTERVENTIONAL TREATMENT

Acute DVT represents a disease spectrum which ranges from asymptomatic calf vein thrombosis to PHLEGMASIA CERULEA DOLENS. Till recent times, guidelines only recommended anti coagulation. However 8th ACCP conference suggests interventional strategies may be developed to reduce PTS.

UNDERSTANDING POST THROMBOTIC VENOUS INSUFFICIENCY

Recent ACCP guidelines recommend major differences from earlier guidelines. Extensive venous thrombosis supports use of venous thrombectomy grade 2b and catheter directed thrombolysis grade 2b, thrombolytic therapy used when these techniques are not available. Thrombolysis is most effective when plasminogen activator is delivered directly into the thrombus. Pharmacological techniques have found to reduce treatment time and dose of plasminogen activator

MORBIDITY OF PTS

Early elimination of thrombus prevents morbidity. There is significant reduction in quality of life which is predicted by the severity of the disease. In ilio-femoral DVT treated by anti-coagulation alone, 90% have ambulatory venous hypertension, 40% venous claudication, 15% venous ulceration within 5 years. This shows that thrombus removal is the best management for best long term outcome

RATIONALE OF THROMBUS REMOVAL

Ambulatory venous hypertension is the cause of microcirculatory changes which is the Pathophysiology of PTS. There is venous valvular incompetence and luminal obstruction. No single radiological technique exists to assess the impact of venous obstruction on severity of PTS. Neither ascending phlebography nor maximal venous outflow test can identify abnormalities causing venous obstruction. Luminal venous obstruction causes severe forms of venous obstruction. Thus early detection and intervention in acute stage significantly reduces incidence of PTS. Acute successful thrombolysis preserves endothelial function and valvular competence

STRATEGIES FOR THROMBUS REMOVAL

Poor randomised trials and lack of international guidelines coupled with age old data are major roadblocks. CDT benefit has been enunciated recently. In the absence of large randomised trials clinical data is to be trusted which suggests CDT is superior to anticoagulation

CURRENT ACCP GUIDELINES

Current guidelines recommend operative venous thrombectomy to be preferred in low risk of bleeding and contemporary venous thrombectomy when CDT is indisposed of. Anti coagulation therapy of same intensity and duration is to be coupled for better outcome.

TREATMENT OPTIONS FOR THROMBUS REMOVAL

CDT is the preferred treatment to store unobstructed venous return to the vena cava. Preoperative knowledge and intra operative imaging of extent of thrombus is crucial for success.

THROMBOLYTIC THERAPY

Anticoagulation therapy was initially systemic and insufficient for clearing thrombus to return venous patency and valve function. Initial lytic therapy had significant venous valve competency but benefits were not as apparent and bleeding tendencies were more

INTRA THROMBUS CDT

Pathophysiology of thrombus includes creating binding sites for plasminogen activators and production of plasmin. Thus intra thrombotic delivery of plasminogen activators is better than systemic. Bleeding disorders is a rarity localised to venous access site. Pulse spray technique of four sessions had 50-70% lysis and little bleeding manifestations. There was continued lysis after treatment sessions

OUTCOMES

CDT was associated with better QOL than anticoagulation alone. But large randomised studies are required. Two such studies are underway

PHARMACO-MECHANICAL THROMBOLYSIS

CDT provides good results but at the cost of longer duration approximately 71 hours. Endovascular mechanical thrombectomy

ENDOVASCULAR MECHANICAL THROMBECTOMY

Mechanical thrombectomy was found to be less effective than when combined with pharmacological thrombolysis by Vedhantham and associates and Kasirajan and associates.

ULTRASOUND ACCELERATED THROMBOLYSIS

Treatment time and dosage of lytic agent was found to be less with ultrasound accelerated thrombolysis with an infusion time of 22 hours and only 4 % people suffering major complications such as puncture site hematomas

ISOLATED SEGMENTAL PHARMACO-MECHANICAL THROMBOLYSIS

This is done using Trellis catheter. A double balloon catheter is inserted into the thrombus and one balloon is inflated above the level of the thrombus while the other is inflated below the level of the thrombus trapping the thrombus within. Plasminogen activator is released into the thrombus. The intervening segment of the catheter assumes a spiral configuration and rotates at the rate of 1500 rpm for 15-20 minutes. The thrombus is fragmented and dissolved within and is aspirated. Phlebography demonstrates the success of the procedure. ISPBT was

found to have better success rate than CDT alone but the bleeding complications were equivocal

OPERATIVE VENOUS THROMBECTOMY

This offers by far the best short term and long term outcomes with comparatively few complications. But this depends upon technical skill and prevention of recurrent thrombosis

It involves identification of cause, definition of full extent, preventing pulmonary embolism, thrombectomy, to ensure unobstructed venous inflow and outflow and preventing recurrent thrombosis by AV fistula, extended oral anti coagulation, catheter directed post operative anti coagulation

PREOPERATIVE CARE

This involves use of unfractionated heparin, identification of full extent of thrombus and vena caval filtering. It is done under fluoroscopic guidance and the entire pelvic and abdominal venous system should be in the field

OPERATIVE CARE

General anaesthesia is recommended for these patients. Longitudinal venotomy is made in the common femoral vein ensuring access to the origin of the saphenous and profunda femoris branches. In case of an infra inguinal thrombus leg is elevated and compressed with a tight bandage and the foot dorsiflexed, calf and thigh are squeezed. If the infra inguinal thrombus persists a curt down has to

be performed to expose the distal posterior tibial vein and a No 3Fogaty catheter is introduced and advanced up to the common femoral venotomy. The silastic stem is amputated from its hub and inserted half way onto the balloon catheter and out of the common femoral venotomy. Now a NO 4 Fogarty balloon catheter is placed at the other end of the silastic sheath and guided distally through the thrombosed valves and clotted veins up to the posterior tibial venotomy. The infrainguinal venous thrombectomy is performed. The infrainguinal venous system is flushed with a large red rubber catheter in the proximal posterior tibial vein with a heparin saline solution to hydraulically force residual thrombus from the deep venous system. After adequate clearance of infrainguinal venous system, vascular clamp is applied below the femoral venotomy and dilute plasminogen activator solution consisting of 4-6 mg of rtPA in 200 ml of saline is filled in the infrainguinal venous system. The plasminogen activator solution remains there for the rest of the procedure. The local rtPA binds to fibrin bound plasminogen in residual thrombus and promotes further clot dissolution while not causing a systemic lytic response. If the infrainguinal venous thrombectomy is not successful the femoral vein is ligated and divided below the profunda femoris. Iliofemoral venous thrombectomy is now performed with a No 8 or 10 venous thrombectomy balloon catheter. It is passed partially into the iliac vein to remove the bulk of the thrombus before advancing the catheter into the vena cava. The proximal thrombectomy is performed under fluoroscopic guidance. During this part of the procedure PEEP is given to reduce the risk of pulmonary

embolism. If a clot is present in the vena cava caval thrombectomy can be performed as an alternative to vena caval filtration. The nature of the venous drainage into the vena cava is assessed using intraoperative phlebography or fluoroscopy. Intravascular ultrasound is better for detecting iliac vein stenosis. Once the venotomy is closed and end to side AV fistula is constructed to increase venous velocity but not pressure. A piece of polytetrafluroethylene or silastic is kept around the saphenous AVF and permanent monofilament suture looped to serve as a guide for future dissection. Closed suction drain is placed in the wound to evacuate any sero-sanguinous fluid that accumulates post operatively and the wound is closed with multilayered running absorbable sutures for better haemostatic and lymphostatic wound closure and elimination of dead space. Paediatric feeding tube is inserted and fixed to the proximal posterior tibial vein for post operative anticoagulation while the distal posterior tibial vein is ligated.

POSTOPERATIVE CARE

Therapeutic anticoagulation with UFH through posterior tibial vein catheter is given. Before removal of the catheter an ascending phlebogram is done. Intermittent pneumatic compression garments are used when the patient is not ambulatory. 30-40 mm Hg ankle grading below knee compression stockings can also be used with the benefit of 50% reduction in post thrombotic morbidity. Venous duplex and other venous duplex function studies are performed for evaluating vein valve function and ultrasonic patency.

RISK ASSESSMENT. Patients at highest risk for procedure related PE are the ones with non obstructive venous thrombus. Systemic complications are reduced with modern techniques such as direct intrathrombus infusion, pharmaco mechanical techniques, etc. reduction in the dose of lytic agent, duration of therapy, circulating antiplasmins and Plasminogen activator inhibitors serve to neutralise the systemic effect of lytic therapy.

PRE TREATMENT EVALUATION

Etiological factors such as trauma, underlying thrombophilia, occult cancer etc have to be evaluated before choosing the treatment options.

IMPORTANCE OF IMAGING

Imaging of the vena cava is important to assess the degree of venacaval involvement. Characterisation of the proximal and distal end of the thrombus is very important. A CT of chest, abdomen and pelvis is an important part where an asymptomatic PE may be found. This helps distinguish pre-treatment PE from treatment failure/complication of lytic therapy or venous thrombectomy.

INTERVENTIONAL TREATMENT OF PE

Most cases of acute PE anticoagulation alone are sufficient. However large PE causing hemodynamic instability and/or PHT require restoration of normal outflow from RV thereby avoiding chronic thromboembolic PHT. Systemic

thrombolytic therapy includes RV and pulmonary function decrease chronic PHT and recurrent venous thromboembolism.

When contraindicated, catheter based techniques can be used. Modified rotating pigtailed catheter is used as a part of catheter based thrombolysis. 13mg of rtPA is injected after embolic disruption and manual clot aspiration done.

Rheolytic thrombectomy combined with infusion of plasminogen activator solution seems to be a better technique.

PATIENT EVALUATION

All patients with PE should be evaluated with cardiac ECHO to assess pulmonary artery pressure and RV function. Significant morbidities which is right heart abnormalities- RV dilatation, Tricuspid insufficiency and PHT warrant catheter based fragmentation with thrombolysis or systemic thrombolysis.

VENA CAVAL INTERRUPTION

PE is the significant cause of mortality. It is managed with anticoagulants which decrease recurrence and risk of bleeding complications.

When this anticoagulation fails or is contraindicated, vena caval interruption with a filter device is appropriate. Initially femoral vein ligation, IVC ligation & partial interruption of IVC were used. But these turned out less effective in preventing the problems. Following this technique of Trans venous delivery of

intra vascular vena caval devices. At first these filters were fitted in the operation room but now it is placed bedside under transabdominal or intravascular ultrasound guidance.

FILTER DESIGN

The filters are designed with the aim of trapping the venous thromboembolism in the vena cava. The material used should be non thrombogenic, biocompatible, non Ferro magnetic, durable & implantable.

IVC FILTERS

Considering the anatomy of inferior vena cava and a note on the variations before filter placement.

The variations include IVC transposition, duplication, IVC agenesis, renal vein anomalies.

1. IVC TRANSPOSITION:

   Generally the left sided IVC drains into left renal vein, crosses to right and continues in cephalic direction. Filters can be placed but the location of renal veins should be accurately defined& suprarenal vena cava placement may be preferred.

2. IVC DUPLICATION:

   It is present in 0.2-0.3%. The right iliac and right renal vein typically drains into right IVC whereas the left sided IVC drains left iliac vein, joins the left renal

vein, where it crosses over into the right sided vena cava. Thus with this anomaly, placing a filter in the right IVC may not adequately prevent PE in a patient with left sided DVT. In this case, placing a filter in each vena cava or in the suprarenal vein after crossing the left renal vein is required.

3. RENAL VEIN ANOMALIES:

The common anomalies are retro or circumaortic left renal vein or multiple bilateral renal veins. Of these the circumaortic renal veins have separate drainage, so the tip of the filter is positioned inferior to the lowest portion of the circumaortic vein

4. IVC AGENESIS:

There can be abnormal IVC with azygos drainage or infrahepatic interruption of IVC with azygos continuation.

TECHNICAL CONSIDERATIONS

To place the filters with accurate assessment of venous anatomical landmarks &under fluoroscopic guidance VENOGRAPHY is the best technique used. Still complications such as hematoma formation, arterial puncture, air embolism, malpositioning, pneumothorax & AV fistulas occur. Bird's nest filter has a higher mortality rate compared to other filters probably patient's co morbidity adding to it. A thrombus in the IVC or a malpositioned infrarenal filter, or duplicate IVC necessitates the placement of a SUPRARENAL FILTER.

To prevent the complications during the transport of the patient for filter placement, bedside placement under transabdominal duplex ultrasound or

intravascular ultrasound guidance and it is even cost effective. If the transabdominal ultrasound cannot image adequately bedside intravascular ultrasound was performed. The major risk with bedside insertion is malpositioned filters. It can be managed by repositioning or insertion of a second filter or percutaneous retrieval is possible. Bedside filter techniques have more advantages than contrast enhanced venography.

VENOGRAPHY GUIDED FILTER PLACEMENT

On the basis of filter type and the clinical situation access can be femoral, jugular or antecubital. The fluoroscopy can either be in a operating room setting with C arm fluoroscope or in endovascular or radiological suite with fixed arm imaging. Guide wire and catheter access is useful in initial non selective venography. The images are obtained to confirm landmarks, correlate bony landmarks, presence of thrombus at the filter placement level. This correlation has lead to the study of different positions of the renal vein in relation to lumbar vertebra and disc spaces. It corresponded to L1 in 17.3%, L1 L2 disc space in 36.5%, L2 in 34.6%, L2L3 disc space in 7.7%.Similarly in common iliac vein. For a suprarenal filter placement, venographic imaging of the suprarenal vena cava is needed to confirm the level of renal veins. Normally the suprarenal vena cava is larger than the IVC. Thus accurate diameter measurements should be made to confirm the diameter. The filter leg attachment point should be above the most cephalic renal vein else the filter may tilt.

TRANSABDOMINAL DUPLEX ULTRASOUND GUIDED FILTER PLACEMENT

It is done to assess if the visualization of IVC at the renal vein level is adequate in both transverse and longitudinal axis. The lowermost renal vein that is the right renal vein is to be identified with the help of the right renal artery. A venous access is obtained and a guide wire is passed under Duplex ultrasound visualization. After visualizing the right renal vein- IVC junction, filter delivery catheter is advanced inside to the renal vein level. Ensuring its correct position, the sheath ids pulled back slowly such that the filter delivery catheter tip is at the renal vein level. With direct visualization under duplex ultrasound the filter is deployed. Post deployment duplex imaging ensures proper position of the filter in IVC.

INTRAVASCULAR ULTRASOUND GUIDED FILTER PLACEMENT.

A percutaneous femoral vein access is obtained under local anaesthesia. The intravascular ultrasound probe is inserted into the sheath and directed up to the level of right atrium of the heart. The venous anatomical landmarks such as right atrium, hepatic vein, renal vein, & confluence of the iliac vein are identified by pull back technique. The ultrasound probe is directed just below the level of the lower most renal vein and the IVC diameter is recorded before proceeding with deployment of filter. The filter delivery is advanced to the mark in case of predetermined marks by aligning the tip of the filter with the end of the sheath. In case of systems without predetermined marks, the sheath is first pulled to a

distance equivalent to the filter delivery catheter extends beyond the sheath. This enables the tip of the filter to align with the lowermost renal vein on deployment. When a single venous access is not possible, dual venous access is adapted. Separate venous access in contralateral femoral vein, thus avoids double large sheaths in the same femoral vein. Ipsilateral femoral vein probe is used in case of contralateral venous thrombosis. The filter is deployed through pull back technique. To confirm the filter position post procedure plain X RAY Abdomen are taken.

FILTER RETRIEVAL

A retrieval time period is to be determined when the risk of PE is low, or when anticoagulation can be restarted. Ensure radio graphically that there are no filter related problems which hinder their retrieval. Detect the presence of any thrombus within the filter using venography in which case filter is left in place. On the basis of filter types different approaches and techniques are used. If there is difficulty in releasing the filter from the IVC attachment, retrieval is aborted. For the “difficult to remove filters” or malpositioned filters, special techniques such as bronchoscopic forceps can be used.

SVC FILTERS

The most important causes for upper extremity DVT is the use of central venous access, thoracic outlet related venous occlusion, implantable defibrillators, sepsis, malignancy, thrombophilia. When anticoagulants are contraindicated superior vena cava filter is used for upper extremity DVT. Percutaneous Jugular

or femoral venous access is obtained. If the situation arises where the superior and inferior vena cava are to be placed simultaneously, the most distal filter is to be placed first. Remove any previous central venous catheters. Superior vena cava anatomy is studied detail under venography and any anomalies must be appreciated. Venography also determines the filter leg attachment which is just at the confluence of innominate veins. The filter leg length should be determined in such a way that it does not enter into the right atrium. With correct position and size the filter is passed through the sheath and the filter is deployed. Complications such as filter malpositioning, superior vena cava perforation, pneumothorax, and guide wire entrapment, erosion into thoracic aorta.

SPECIFIC PATIENT GROUPS

TRAUMA PATIENTS

The risk of developing DVT and PE is as high as 50% & 30%. The risk factors include advancing age, multiple transfusions, high injury severity score, head injury, spinal cord or vertebral injury, long bone fracture, multiple transfusions. Contributing to this are prolonged immobility, venous stasis & proinflammatory hypercoagulable state. Thromboprophylaxis is preferred for trauma patients with at least one risk factor. Mechanical thromboprophylaxis is preferred in patients who cannot receive anticoagulants. Trauma patients at high risk are suggested routine prophylactic filter placement. Use of retrievable filters in the high risk trauma patients has repoted success. Use of IVC filters is indicated in patients with proven DVT and a contraindication to therapeutic coagulation.

BARIATRIC PATIENTS

Obesity poses a strong risk for developing venous thromboembolism. The most common cause of Perioperative death in bariatric patients is PE.The mortality is due to improper dosing of the anticoagulation thromboprophylaxis the anticoagulation has many drawbacks even with standard heparin protocols. Thus vena cava filter placements have become increasingly common. Several indications such as BMI >60, hypoventilation syndrome, truncal obesity were considered for prophylactic filter placement. The optional / retrievable filters are used in the highest risk period following subsequent removal. A study was conducted where filters were placed before surgery and removed 4 weeks postoperatively. There was no fatal PE or deaths.

ORTHOPEDIC PATIENTS

Orthopaedic surgeries such as total hip arthroplasty, total knee arthroplasty, lower extremity fracture repair. The incidence of PE in patients not receiving prophylaxis was 60 to 70%. The filters were placed perioperatively. Optional filters are used in high risk period and are retrieved when the risk for thromboembolism is less. Morbidity was minimal in filter prophylaxis. The retrievable filter placement was suggested an adjunct to the standard prophylaxis in high risk orthopaedic patients. Thus the retrievable filters are preserved in patients where anticoagulation prophylaxis is contraindicated.

CANCER PATIENTS

Malignancy poses a great risk for venous thromboembolism development. Even with anticoagulant prophylaxis the risk was found to be high. In patients with a previous history of thromboembolism, there is increased chance of recurrence after the development of new metastasis or neutropenia. Cancer patients are also at increased risk of bleeding despite anticoagulation. Vena cava filters is an alternative to long term anticoagulant in a subgroup of patients with increased risk of recurrence. The filter placement is based on the survival. In brain tumour patients filter placements are not cost effective. But in breast cancer patients vena cava filter placement is more cost effective than anticoagulation. In patients with advanced stages of malignancy, filter placement is of little clinical significance. The filters should be reserved for patients with documented venous thromboembolism because of the potential for increased thrombotic events.

HIGH RISK GENERAL SURGICAL PATIENTS

The risk of venous thromboembolism is high in patients with increased risk of malignancy, obesity, varicose veins and oestrogen use, type of anaesthesia (high risk with general anaesthesia). Perioperative factors such as degree of mobilization, fluid status, and transfusion practices. Shorter duration of prophylaxis may increase the risk of venous thromboembolism. After major general surgery DVT's develop within 1 to 2 weeks. Thus postoperative anticoagulants are given up to 28 days in high risk patients. Uses of prophylactic filters are an attractive option. These filters have an advantage of decreased

bleeding risk. Vena cava filter use is limited to standard indication for documented venous thromboembolism.

Results

One patient out of 30 had asymptomatic DVT on 7th POD on left leg. The patient is 25 yrs old female, her operation time 1-2 hours, and mobilised early < 24 hrs with no obvious risk factors.

She was further evaluated.

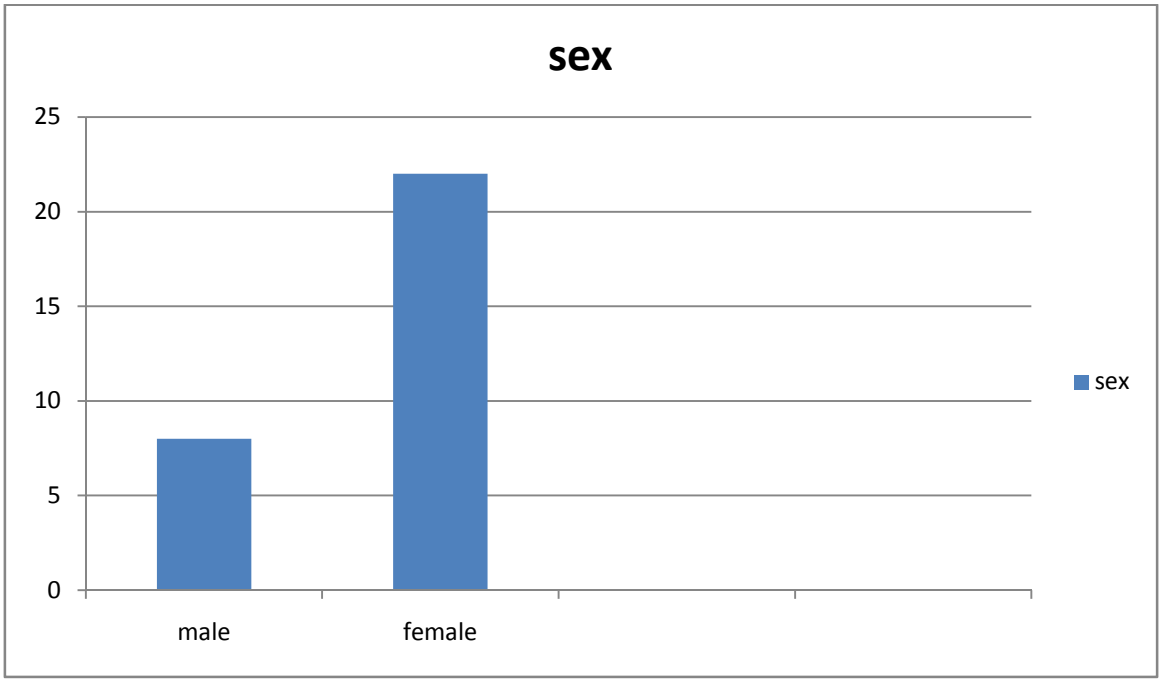
Repeat LFT – normal, PT/INR – normal, complete hemogram with ESR – normal, BT/CT – normal, Rheumatoid Factor – negative,

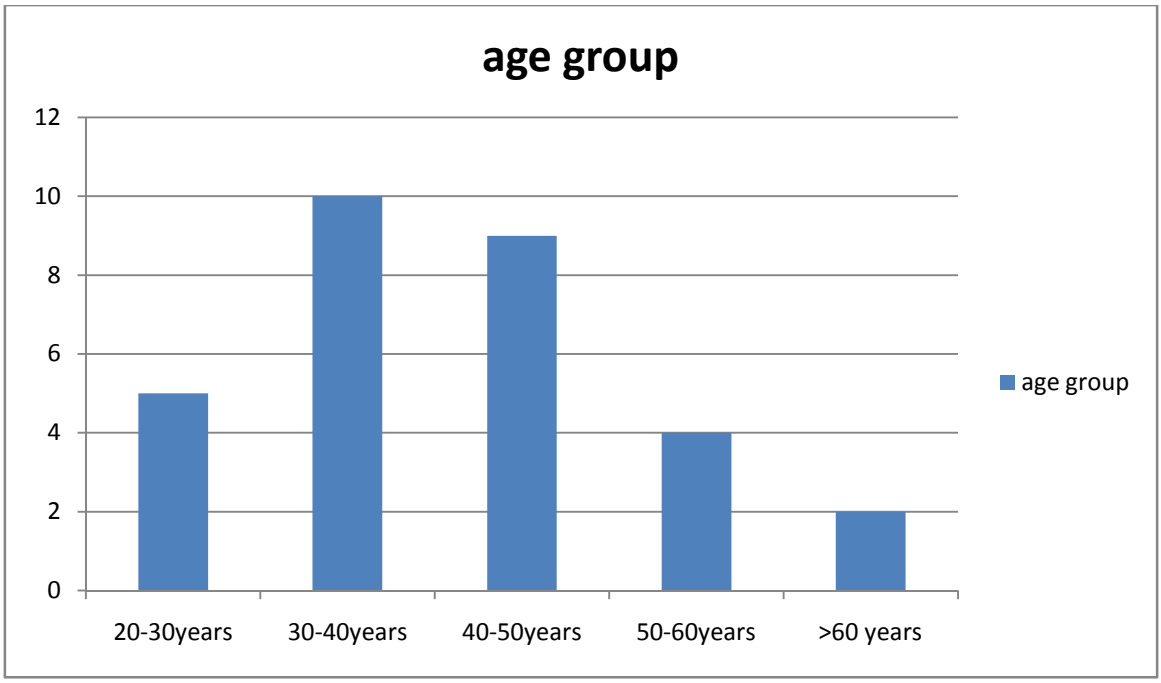
She was started on LMWH along with warfarin. Her INR was maintained around 1-2. Again she underwent Doppler on 12th POD and on 17th POD and found to be normal.

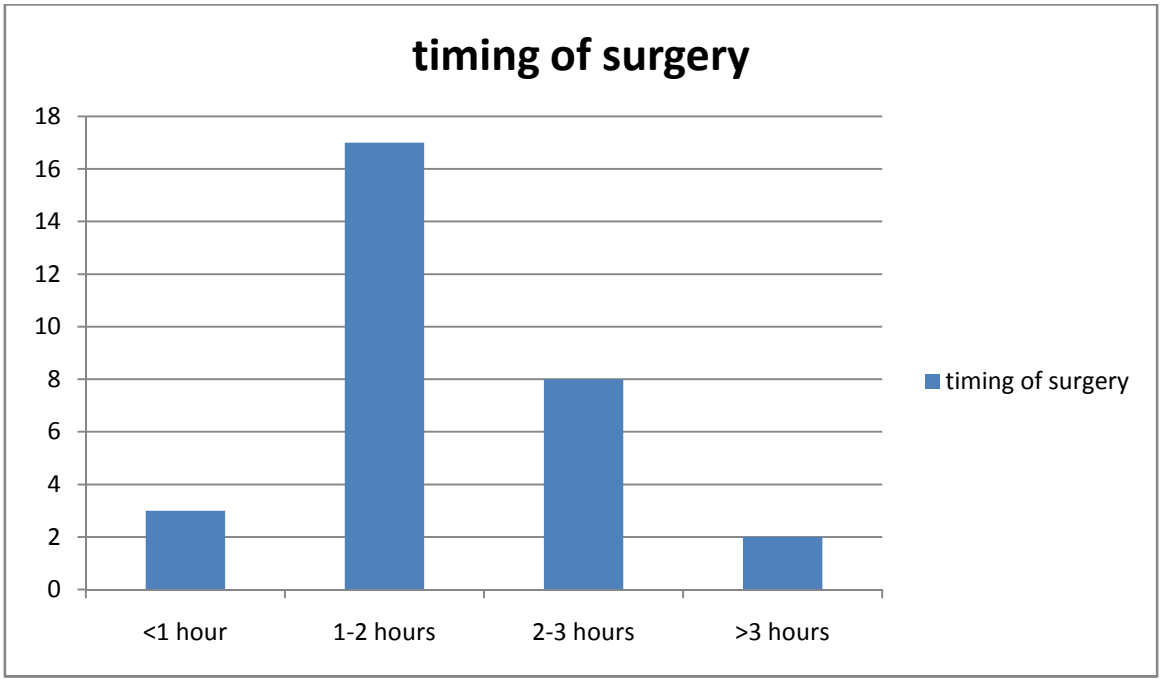
The patient was referred to Madras Medical College, Vascular surgery department for further work up and management.

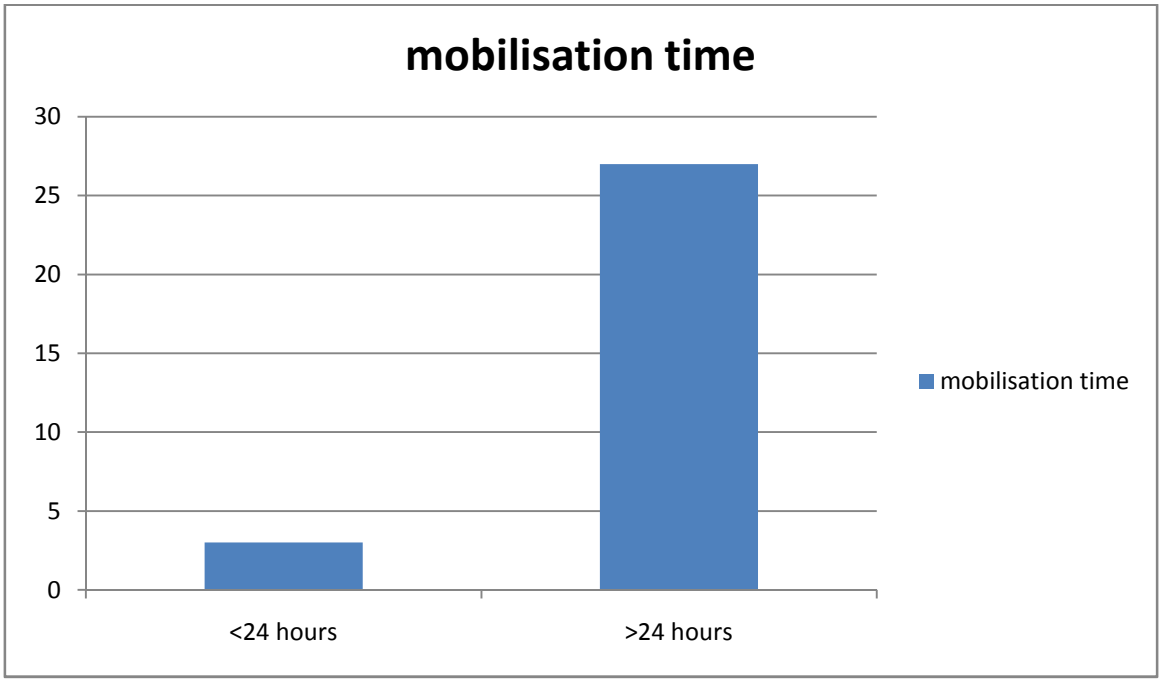
She came for follow up after a month time to our OP. she was evaluated there at vascular surgery and found every thing normal. Now she is doing well.

Statistical analysis






timing of surgery
18
16
14
12
10
8
6
4
2
0
<1 hour
1-2 hours
2-3 hours
>3 hours
timing of surgery


mobilisation time
30
25
20
15
10
5
0
<24 hours
>24 hours
mobilisation time

Conclusion

The patients who had indications for cholecystectomy, and had undergone surgery, who otherwise had no risk factors for DVT, developed DVT with the incidence of 1/30 in our hospital.

In my study, I have excluded all the DVT risk patients.

But the sample size and its representation about various population groups are not adequate. So with this experience further studies are to be done.

So it is better to start some form of mechanical method of prophylaxis to all patients undergoing cholecystectomy, as the immobilisation is expected from pain due to upper abdomen incision, obesity, females and other risk factors, then according to post operative clinical findings and investigations if any is positive for DVT, further management is to be done.

Because cholecystectomy is done for patients are also found to have risk factors for DVT as it is shared by both.

In the presence of some risk factors for DVT, the patient should be started on pharmacological method of prophylaxis.

But ultimately, all the patients undergoing cholecystectomy should be given the basic measures like adequate hydration, adequate analgesia, and identification of risk factors, early ambulation, both active and passive flexion exercises and proper information to patients on the clinical presentation of DVT.

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MASTER CHART

		1	2	3	4	5	6	7	8	9	10	11	12
NAME		SAMUTHRAM	MALA	PERAMAIYA	SENTAMIL SELVI	MARIYA	SHANTHI	SEVU	BASKAR	KOKILA	PAPPU	MUTHUKUMAR	RENGAMMAL
AGE		48	32	70	29	25	38	55	53	45	40	60	40
SEX		F	F	M	F	F	F	M	M	F	F	M	F
BMI													
IP NO		1346628	1363524	1379884	1374930	1391647	1351427	1351417	1369375	1364646	1343370	1391578	1390354
DIAGNOSIS		CHOLILITHIASIS	CHOLILITHIASIS WITH R SUPRARENAL CYST	CHOLILITHIASIS WITH HIATUS HERNIA	CALCULOUS CHOLECYSTITIS WITH CBD CALCULUS	CHOLELITHIASIS	CHOLELITHIASIS	CHOLELITHIASIS	CHOLELITHIASIS	CALCULOUS CHOLECYSTITIS	CALCULOUS CHOLECYSTITIS	CHOLELITHIASIS	CHOLELITHIASIS
TREATMENT PLAN		OC	OC	OC	OC/CDL	OC	OC	OC	OC	OC	OC	OC	OC
RISK FACTORS													
AGE A	20-30 Y				Y	Y							
B	30-40 Y		Y				Y				Y		Y
C	30-40 Y	Y								Y			
D	30-40 Y							Y	Y			Y	
E	30-40 Y			Y									
MALIGNANCY		NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
RECENT MAJOR SURGERY IN THE LAST SIX MONTHS		NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
IMMOBILISATION >72 HOURS IN LAST 15 DAYS		NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
OBESITY >30 BMI													
H/O DVT		NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
OC PILLS IN LAST SIX ONTHS		NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
SEPSIS IN THIS EPISODE OF ILLNESS		NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
POST PARTUM		NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
VARICOSE VEINS		NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
PRE OP HOMAN SIGN		NEG	NEG	NEG	NEG	NEG	NEG	NEG	NEG	NEG	NEG	NEG	NEG
PRE OP DOPPLER FINDING		NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR
SURGERY		OC	OC	OC	OCC	OC	OC	OC	OC	OC	OC	OC	OC
TIMING OF SURGERY	< 1 H			Y							Y		
	1-2 H		Y			Y			Y	Y		Y	Y
	2-3 H						Y	Y					
	>3 H	Y			Y								
MOBILISATION TIME	<24 H					Y			Y			Y	
	>24 H	Y	Y	Y	Y		Y	Y		Y	Y		Y
POST OP DOPPLER FINDING	3RD D	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR
	7TH D	NOR	NOR	NOR	NOR	pos	NOR	NOR	NOR	NOR	NOR	NOR	NOR
DISCHARGE		10TH	8TH	8TH	10TH	17TH	11TH	9TH	8TH	10TH	13TH	8TH	13TH

MASTER CHART

13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30
CHINNASI	POORANAM	MANJULA	NATARAJAN	JAYALAXMI	GNANAMANI	UMAYAL	SELVI	ARUNA	BHUVANESWARA	JOTHI	RANJITHAM	FEMINA FLORENCE	PATHARNISHA BEEVI	SELVARAJ	ADAIKALAM	MANIMEGALAI	VEERAIYAN
47	40	35	62	42	55	38	35	22	42	45	32	21	42	50	23	40	60
F	F	F	M	F	F	F	F	F	M	F	F	F	F	M	F	F	M
1346847	1390379	1346970	1406446	1349398	1364081	1347789	1366574	1390387	1338217	1372761	1369997	1383829	1389807	1384524	1357259	1387701	1376598
CHOLELITHIASIS	CHOLELITHIASIS	ACUTE CALCULUS CHOLECYSTITIS	CHOLEDOCHOLITHIASIS	CHOLELITHIASIS	CHOLILITHIASIS	CHOLILITHIASIS	CALCULOUS CHOLECYSTITIS	CHOLILITHIASIS	ACUTE CALCULUS CHOLECYSTITIS	CALCULUS CHOLECYSYTITIS	CALCULUS CHOLECYSYTITIS	CALCULUS CHOLECYSYTITIS	CHOLELITHIASIS	MUCOCELE GALL BLADDER	CHOLILITHIASIS	CHOLILITHIASIS	CALCULOUS CHOLECYSTITIS
OC	OC	OC	OC/ CDL	OC	OC	OC	OC	OC	OC	OC	OC	OC	OC	OC	OC	OC	OC
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			Y														
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NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
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NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
NEG	NEG	NEG	NEG	NEG	NEG	NEG	NEG	NEG	NEG	NEG	NEG	NEG	NEG	NEG	NEG	NEG	NEG
NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR
OC	OC	OC	OC	OC	OC	OC	OC	OC	OC	OC	OC	OC	OC	OC	OC	OC	OC
	Y																
Y		Y		Y		Y		Y	Y	Y	Y		Y			Y	Y
			Y		Y		Y					Y		Y	Y		
Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR
NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR	NOR
7TH	9TH	8TH	10TH	13TH	8TH	13TH	7TH	9TH	15TH	15TH	9TH	12TH	11TH	12TH	5TH	7TH	8TH

A PROSPECTIVE STUDY ON INCIDENCES OF DEEP VENOUS THROMBOSIS IN CHOLECYSTECTOMY PATIENTS BETWEEN JULY 2011 TO JUNE 2012- study proforma

NAME .` AGE

SEX M/F

BMI ;

IP NO .`

DIAGNOSIS .`

TREATMENT PLAN ;

RISK FACTORS

AGE	A	20-30 Y	
	B	30-40 Y	
	C	30-40 Y	
	D	30-40 Y	
	E	30-40 Y	
MALIGNANCY		;	Y/N
RECENT MAJOR SURGERY IN THE LAST SIX MONTHS		;	Y/N
IMMOBILISATION >72 HOURS IN LAST 15 DAYS		;	Y/N
OBESITY >30 BMI		;	
H/O DVT		;	Y/N
OC PILLS IN LAST SIX ONTHS		;	Y/N
SEPSIS IN THIS EPISODE OF ILLNESS		;	Y/N
POST PARTUM		;	Y/N
VARICOSE VEINS		;	Y/N
PRE OP HOMAN SIGN		;	Y/N
PRE OP DOPPLER FINDING		;	
SURGERY		;	
TIMING OF SURGERY		< 1 H	
		1-2 H	
		2-3 H	
		>3 H	
MOBILISATION TIME		<24 H	
		>24 H	
POST OP DOPPLER FINDING		3RD D	
		7TH D	
DISCHARGE		;	

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Author	Elavarasan 22101172 M.S. General Surgery
E-mail	elavarasan.selvanathan@gmail.com
Submission time	19-Dec-2012 09:35PM
Total words	18041

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A prospective study on Incidences of Deep Venous Thrombosis in Cholecystectomy patients between July 2011 to June 2012 Introduction Deep venous thrombosis is a condition where there is development of thrombus in the vein of deep venous system, mostly affecting lower limbs. Deep venous thrombosis is not a rare entity as we think. It is a much more common but often underestimated and under recognised. These are because of the lack of standardisation in all health facilities. That too in general surgery it often goes unnoticed because of less suspicion. So to get an idea about DVT in patients undergoing open cholecystectomy, which is one of the common operations done in our hospital, we have...