A Dissertation on

ASSESSMENT OF RISK FACTORS FAVOURING DEEP VEIN THROMBOSIS IN PATIENTS UNDERGOING SURGERY

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CERTIFICATE

I, hereby declare that the dissertation entitled "**ASSESSMENT OF RISK FACTORS FAVOURING DEEP VEIN THROMBOSIS IN PATIENTS UNDERGOING SURGERY"** is the bonafide research work done by **Dr.V.JOTHI NARAYANASAMY** and submitted in partial fulfilment of the requirement of the degree of Master of Surgery in General Surgery, Coimbatore Medical College and Hospital, Coimbatore.

Date : **Unit Chief**

Date : **Professor &HoD**
Department of General Surgery

Date : **Dean**
Coimbatore Medical College
Coimbatore – 641014.

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This is to certify that dissertation entitled **"ASSESSMENT OF RISK FACTORS FAVOURING DEEP VEIN THROMBOSIS IN PATIENTS UNDERGOING SURGERY"** of the candidate **DR.V.JOTHI NARAYANASAMY** with registration Number **221611302** for the ward of M.S in the Branch of General Surgery, I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains 81 pages from introduction to conclusion and the result shows 1% (One) percentage of plagiarism in the dissertation.

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This work has been carried out in the Department of General Surgery, Coimbatore Medical College and Hospital, Coimbatore under the guidance of **Dr. ELANGO .V, M.S.,** Professor and HOD of General Surgery, Coimbatore Medical College and Hospital, Coimbatore.

Date : **Dr. V. Jothi Narayanasamy**

Place : Coimbatore

Coimbatore Medical College

COIMBATORE, TAMILNADU, INDIA - 641 014

(Affiliated to The Tamilnadu Dr. MGR Medical University, Chennai)

ETHICS COMMITTEE

CERTIFICATE

Name of the Candidate: Dr. V. Jothi Narayanasamy

Course : MS (General Surgery) Post Graduate

Period of Study : 1 year

College : Coimbatore Medical College & Hospital.

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The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation Proposal is accepted and you are permitted to proceed with the above Study.

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Dr. V. Jothi Narayanasamy

LIST OF ABBREVIATIONS

DVT	**-**	**DEEP VEIN THROMBOSIS**
VTE	**-**	**VENOUS THROMBOEMBOLISM**
PE	**-**	**PULMONARY EMBOLISM**
LMWH	**-**	**LOW MOLECULAR WEIGHT HEPARIN**
HIT	**-**	**HEPARIN INDUCED THROMBOCYTOPENIA**
aPTT	**-**	**ACTIVATED PARTIAL THROMBOPLASTIN TIME**
BMI	**-**	**BODY MASS INDEX**

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INTRODUCTION

DEEP VEIN THROMBOSIS is a clinical challenge for doctors of all disciplines. It can complicate the course of a disease but might be encountered the absence of precipitating disorders. Thrombosis can take place in any section of the venous system , but arises most frequently in deep veins of leg. Long term morbidity due to post thrombotic syndrome was common and may be substantial. The major concern, however, embolization of thrombus to the lung which can be fatal. Deep vein thrombosis is highly prevalent and poses a burden on health economy. The disorders and its sequalae were also among the best examples of the preventable diseases . Relevant data for the frequency for the deep vein thrombosis derive from large community based studies because they mainly reflect the symptomatic rather than the asymptomatic disease. Two- thirds of the first deep vein thrombosis are caused by the risk factors including surgery , cancer , immobilization , or for other reasons. Risks for first deep vein thrombosis seem to be higher in men than in women.

RISK FACTORS FAVOURING DVT

- Age
- Obesity
- Smoking
- Cancer patients
- Previous history of deep vein thrombosis
- Family history of thrombosis
- Previous history of varicose vein
- Chronic medical illness
- History of connective tissue disorder
- Chemo/ radiotheraphy received patients
- Spinal/ general anaesthesia
- Duration of surgery
- Immobilization days

Rudolph virchow is recognized as the first person to link the development of VTE to the presence of atleast 1of 3 conditions:

Venous stasis , vascular injury and hypercoagulability. Some of risk factors can alter the delicate hemostatic balance toward the hypercoagulability and development of the thrombosis. Coleridge-smith et al12 reported in 1990 that venous stasis occur during

general surgery , with veins dilating 22 to 28 % patients undergoing general anaesthesia and surgery and upto 57 % in those received in infusion of 1 litre of saline during the surgery. The investigators suggested that it was intraoperative venous distension that underlies the risk for deep vein thrombosis in patients undergoing surgery. They suggested that the venous distension was the result of loss of muscle tone that was caused by the muscle relaxants used during the surgery. Muscle paralysis resulting from regional anaesthesia also can lead to venous dilatation. These effects can be modified to some extent by the use of graduated compression stockings during surgery. The venous dilatation that occurs during surgery causes cracks in the endothelium , which provides a nidus for the thrombosis as the blood coagulation system is activated. Microscopic vessel damage during surgery precipitating factor for DVT. The third factor in virchow's triad , hypercoagulability was linked to the number of factors , including certain genetic traits. Deficiencies of antithrombin , protein C , protein S OR MUTATIONS OF FACTOR V LEIDEN , or factor II (prothrombin) G 20210A genes lead to the hypercoagulable states.

AIM AND OBJECTIVE OF THE STUDY

It is important to have pre operative discussion with patients and their families regarding relative risks and benefits if particular thromboprophylaxis strategy. This could include realistic evaluation of the risk of serious DVT complications. Individual assessment of thrombosis risk must be done in every patient to minimize morbidity and mortality of DEEP VEIN THROMBOSIS post operatively. And also moderate and high risk group patients definitely need the thromboprophylaxis preoperatively in order to prevent the post operative DVT.

This study shows various risk factors related to DEEP VEIN THROMBOSIS and prophylactic management of DEEP VEIN THROMBOSIS.

REVIEW OF LITRATURE

AGE

Patients >40 years of age are increased risk of venous thromboembolism compared with young patients. With the risk approximately doubling with each decade thereafter. Further evidence for the importance of age as risk factor for deep vein thrombosis was reported by oger in a study of the incidence of DVT in French communititon population. This study found that the incidence of DVT risk increasing with age , reaching >1% inpatients >75 years of age. This study reported that the incidence of PE as a percentage of total VTE also increased with age. A subanalysis of data from the prophylaxis of DVT in medical patients with ENOXaparin (medenox) study confirmed that age >75 years is an independent risk factor for VTE in patients hospitalized with an acute and chronic medical illness. Hence old age patients undergoing surgery greater risk of DVT than young age patients.

OBESITY

Investigations that reported increased risk of DVT caused by obesity are criticized because they failed to include the duration of hospital confinement or other risk factor. High proportion of the

patients with DVT has been found to be obese but the importance of the association was diminished because of the high proportion of obesity in the general population. The Nurses' health study showed that the age related risk ratio for PE women with the body mass index 29 kg/ m2 higher was 3.2 times compared the leanest category of less than 21 kg/ m2. The Framingham heart study showed that metropolitan relative weight was significantly and independently associated with PE among women not men. However, the study of men born in 1913 showed that men in the highest decile of waist circumference >100cm had adjusted relative risk of DVT 3.92 times compared with men with Waist circumference of <100cm. Various abnormalities of haemostasis have been described in obesity , in particular increased plasminogen activator inhibitor-1. Other abnormalities reported include 1) platelet activation 2) increased plasmafibrinogen factor and von willebrand factor , Fibrinogen , factor 7C and PAI-1.

SMOKING

The patients with smokers had higher risk of DVT than non smokers. The potential influence on coagulation offers further potential to contributed to thrombogenesis in all smokers. Plasma

fibrinogen levels elevated in all smokers. Factor XIII , which stabilized fibrin Clots , is increased in smokers. Exposure to nicotine may also increase the plasminogen activator inhibitor -1 (a major regulator of fibrinolysis) , although extend to which nicotin enhances coagulation is unresolved. Hence the deep vein thrombosis is potential and fatal complication of smoking.

VARICOSE VEIN

The importance of varicose veins are independent risk factor for DVT is currently unclear. Varicose veins were identified as an independent risk factor by heit et al with associated risk for venous thrombo embolism decreasing with age. On the basis of the sub analysis of data from MEDENOX study , alikhan et al have reported VTE rate 21.3 percentage. However , it must be noted that these patients also exhibited numerous other risk factors of venous thromboembolism.

CONGENITAL HYPERCOAGULABLE DISORDERS

Antithrombin is serine protese inhibitor of thrombin and also inhibits factors IXa ,Xa ,XIa and XIIa. Thrombin is irreversibly bound by antithrombin and prevents thrombin's action on fibrinogen on factors V ,VIII , XIII and on platelets. The anticoagulant

synthesized in liver and endothelial cells and has a half life of 2.8 days. Anti thrombin deficiency has a prevalence of 1:5000 with more than 100 genetic mutations and an autosomal inheritance pattern. Antithrombin deficiency associated with lower extremity thrombosis as well as mesenteric venous thrombosis. The most common presentation is those with anti thrombin deficiencies are deep vein thrombosis with or without pulmonary embolism.

PROTEIN C AND PROTEIN S DEFICIENCY

Protein C is a vitamin K dependent anticoagulant protein that , once activated by thrombin , will inactivate factor Va and VIIIa thereby inhibiting generation of thrombin. Additionally activated protein C stimulates the release of t-PA. It is produced in the liver and is the dominant endogenous anticoagulant with eighty hour half life. Protein C deficiency has a prevalence of 1in 200 – 300 with more than 150 mutations and an autosomal dominant inheritance.

Protein S is also vitamin k dependent anticoagulant protein that is cofactor to activated protein C. The actions of protein S are regulated by complement C4b finding protein and only the free form of protein S serves as an activated protein C cofactor. Additionally , protein S appears to have independent anti coagulant

function by directly inhibiting procoagulant enzyme complexes. The prevalence of protein S deficiency is about 1 in 500 with autosomal dominant inheritance.

FACTOR V LEIDEN MUTATION AND ACTIVATED PROTEIN C RESISTANCE

Factor V is the glycoprotein synthesized in liver with factor V leiden , a point mutation occurs when arginine is substituted by glutamine at position 506. The activated protein C thus causing the procoagulant state. Clinically, patients may present with deep vein thrombosis in lower extremities , or less commonly in portal vein , cerebral veins or superficial venous system.

PROTHROMBIN G20210 POLYMORPHISM

Prothrombin (factor II) is a zymogen synthesized in liver and dependent on vitamin K. When prothrombin is activated , it forms thrombin (factor IIa). A single mutation where adenine is substituted for guanine occurs at 20210 position . The mechanism for increased thrombotic risk is not well understood , but this individual with this genetic variant have the supranormal level of prothrombin. The mutation is inherited as an autosomal dominant trait and is associated with both arterial and venous thrombosis.

Clinically , patients may present with deep vein thrombosis of lower extremity , cerebral venous thrombosis, as well as arterial thrombosis. The risk of thrombosis increased in the presence of other genetic coagulation defects and with acquired risk factors.

OTHER COAGULATION FACTORS

Elevated levels of several coagulation factors , including factors VIII , IX and XI , has been linked with increased thrombotic risk. In the leiden thromophilia study , 25% of the patients with first episode of DVT had factor VIII levels >150% of normal compared with only 11% of healthy controls. Plasma level of > 150 IU/dL was associated with almost 5 times the risk for an initial event. These findings have been confirmed in the several other small studies. Furthermore , patients with prior DVT and elevated factor VIII levels are at significantly increased risk of recurrent VTE.

Elevations in plasma levels of factors IX and XI also appear to modestly increased thrombotic risk. After excluding subjects with other thrombophilic disorders, patients with factors IX and XI levels were above the 90^{th} percentile in the leiden thrombophilia study had a 2.5 fold and 2.2 fold adjusted risk respectively , for venous

thrombosis compared with those with lower values. As with actor VIII , the molecular basis for an elevated factor IX and XI level was unknown , although the genetic component seems likely.

Further information was needed to define the impact of these conditions on future management of VTE , including duration of theraphy , prophylaxis in high risk clinical situations , and family screening accordingly , the utility of screening for elevations in these factor levels in patients with idiopathic venous thrombosis remains unclear.

HYPERHOMOCYSTEINEMIA

Homocysteine is an aminoacid formed during the metabolism of methionine and may be elevated secondary to Inherited defects in two enzymes that are part of the conversion of homocysteine to cysteine . The two enzymes involved are N5 , N10-methelene tetra hydro folatereductase or cystathione beta synthase. Hyperhomocysteinemia had been shown to increased the risk of atherosclerosis , atherothrombosis and venous thrombosis. Elevated plasma homocysteine levels were leads to various dysfunction of endothelial cells leading to theprothrombotic state.

Hypercoagulable syndromes include inherited and acquired thrombophilia. Acquired thrombophilia include antiphospholipid syndrome, heparin induced thrombocytopenia , acquired dysfibrinogenemia , myeloproliferative disorder and malignancy. Regarding the anti-phospholipid syndrome antiphospholipid antibodies were associated with both arterial and venous thrombosis. The most commonly detected subgroup of anti-phospholipid antibodies were lupus anticoagulant antibodies , anti cardiolipin antibodies , anti b2 glycoprotein I antibodies. DVT the most common manifestation of antiphospholipid syndrome , occurs in 29 to 55 percent of patients with this syndrome , and about half of these patients had pulmonary embolism.

COPD

COPD patients are thought at increased risk of DEEP VEIN THROMBOSIS because of immobilization , heightened inflammation , cigarette smoking , and venous stasis. DVT however , remains under diagnosed because its symptoms mimics a COPD exacerbation . overall the reported prevalence of deep vein thrombosis during the COPD patients undergoing surgery ranges from 5 to 29 % . Hence

COPD patients with smoking history more prone to develop deep vein thrombosis post operatively.

CANCER

CANCER RELATED FACTORS

The frequency of DVT increases 2 to 3 fold in patients undergoing surgery for malignant disease compared with those undergoing surgery for the nonmalignant conditions. Because malignancy commonly associated with other riskfactors , the direct effect of malignancy on risk is uncertain. Advanced cancers are associated with high incidence of DVT , especially cancers of the breast , lung , brain , pelvis , rectum , pancreas and gastrointestinal tract. Administration of chemotheraphy increases the risk. For example , patients newly diagnosed with multiple myeloma receiving thalidomide with multiagent chemotheraph in association with surgery have 3 times risk of DVT compared with women undergo surgery alone.

SITE OF CANCER

In studies looking at pooled groups of patients with different types of malignancy , the rate of venous thromboembolism is consistently

higher with cancer of the stomach , pancreas , brain , kidney , uterus , lung or ovary.

Both large retrospective studies by stein et al and chew et al based on discharge claims databases reported the highest rates of DEEP VEIN THROMBOSIS in patients with pancreatic cancer. Patients with stomach cancer had second and third highest risk of developing DVT in these studies . In patients with testicular and lung cancer those with metastasis to liver and brain were shown to have higher rates of DVT compared with patients with other sites of metastasis.

CANCER STAGE

Multiple studies have shown an increased risk of DVT in patients with advanced stage cancer. In a population based case control study of patients with newly diagnosed DVT including 389 patients with cancer those with distant metastases had a higher risk of DVT. Other studies in ovarian , colorectal , pancreatic , lung and breast cancer support the finding that advanced – stage disease increases the risk of cancer associated DVT.

HISTOLOGY

In certain types of cancer , higher rates of DVT are found in some histological subtypes compared with others. In breast and colon cancer patients , the type of histology does not predict for the incidence of cancer associated DVT , but DVT associated mortality rates are higher in patients with certain histological subtypes.

CHEMOTHERAPHY/RADIOTHERAPHY

Chemotheraphy/radiotheraphy is one of the most important factors in DVT risk stratification of cancer patients. Large population- based studies in groups of pooled cancer patients have demonstrated a significantly increased risk in patients receiving chemotheraphy. Heit et al used a population – based study of patients with a new diagnosis of DVT, 23 % of which had diagnosis of active malignancy , to demonstrate the significantly increased risk of DVT in those an chemotheraphy/radiotheraphy.

Studies in specific type of cancer and with specific anti neoplastic agents have also supported the role of chemotheraphy in predicting the risk of cancer-associated DVT. Two prospective studies of breast cancer patients demonstrated that the risk of DVT

in patients receiving chemotheraphy in addition to tamoxifen or surgery or radiotheraphy increased two to seven -fold. A recent meta analysis of breast cancer patients revealed that use of adjuvant hormonal theraphy was associated with 1.5 – 7 fold increased risk of DEEP VEIN THROMBOSIS.

CHRONIC MEDICAL ILLNESSES

LIVER DISEASE

Patients with chronic liver disease (both with alcoholic and non alchoholic) seems to have higher risk of DVT than patients without liver disease . Chronic liver disease may result in impaired production of vitamin-k dependent procoagulant factors. However decreased production of vitamin –k dependent endogenous anticoagulants such as protein C , protein S and antithrombin III may counter the hypocoagulability in such patients.

HYPOTHYROIDISM

In view of study among the hospitalized patients with a diagnosis of hypothyroidism DVT was diagnosed in 1.36 % of hypothyroid patients.

MYOCARDIAL INFARCTION

Myocardial infarction patients are associated with deep vein thrombosis. The deep vein thrombosis risk of patients hospitalized with acute MI are comparable with that of moderate risk general surgical patient (20% overall and 2% symptomatic) . The number of risk factors are commonly associated with MI including age , bed rest , venous stasis because of congestive heart failure such that MI itself has not been clearly establized as an independent risk factor for DVT.

CONNECTIVE TISSUE DISORDERS

Systemic inflammation may be involved in the pathogenesis of deep vein thrombosis through the vessel wall damage , upregulations of procoagulants , downregulation of anticoagulants , and suppression of fibrinolysis. Autoimmune diseases , which are characterized by an overactive immune response against the body's own tissue , may therefore increased the risk for deep vein thrombosis.

SURGERY

Surgery is a well known risk factor for development of DVT in patients without cancer. The incidence of DVT in cancer patients undergoing general surgery is estimated at 37% compared with 20% in patients without cancer. Factors related to immobility , tissue destruction and venous stasis were likely to be related to the increased risk of DVT after surgery. The risk of DVT after major general surgery has been extensively documented. Although the term major general surgery is imprecise , most investigators apply this term to patients who are undergo abdominal or thoracic operations that require general anaesthesia > 30 minutes.

The recent dramatic increase in endoscopic alternative to open surgery has not been accompanied by the controlled studies of DVT risk although adverse changes in haemostasis after laparoscopy have been reported. Bergqvist and loweconcluded in recent review , that laparoscopic cholecystectomy is low risk procedure such that routine DVT prophylaxis is probably not justified. But the decision regarding prophylaxis for laparoscopy should likely be made in the same manner as for conventional surgery , customized for the particular risk of each patient , taking

into account the length of the operation , amount of time bed bound , and comorbid conditions.

IMMOBILIZATION

Gibbs found that 15% of patients on bed rest for <1 week before death had venous thrombosis at autopsy , whereas incidence rose to 80% in patients in bed for a longer period. The influence of immobility as risk factor was particularly striking in studies of hemiplegia. On the basis of fibrinogen scanning, warlow et al found asymptomatic DVT in 60% of paralysed limbs of stroke patients compared with 7 % in the nonparalyzed limbs. Whereas prolonged bed rest or immobilization alone not provide adequate reason for prescribing prophylactic anticoagulant theraphy. Prolonged immobility combined with other major risk factors increases the likelihood of DEEP VEIN THROMBOSIS.

There has been recent attention in the popular press about the risks of DVT associated with long duration air travel the so called economy class syndrome. In a study of 231 subjects without prior history of DVT who were embarking in flights for > 8 hours in duration , those randomized to compression stockings had no evidence of DVT on subsequent duplex ultra sonography.

Conversely , 105 of untreated individuals developed asymptomatic DVT. Despite these findings , there is general consensus that clinically important DVT after air travel is rare , and that benefits of providing DVT prophylaxis during long distance flights are doubtful. Case reports suggest that most cases of travel- related thrombosis affected people at risk because previous DVT or other predisposing factors. Thus , until the formal study validates another approach , it makes sense to provide advice to such people regarding hazards and simple precautions , including frequent leg movement and adequate hydration during long flights.

PROPHYLACTIC MEASURES OF DVT

MECHANICAL METHODS

ANTI EMBOLISM STOCKINGS

Anti embolism stockings exert graded circumferential pressure from distal to proximal region of leg confirming to sigel pressure profile. These increase blood velocity , promote venous return ,and have been shown to be effective. Anti-embolism stockings should not be used if the patient had peripheral vascular disease , arteriosclerosis , severe peripheral neuropathy , massive leg edema or pulmonary edema , oedema secondary to congestive heart failure or local skin / soft tissue

diseases , and in patients with acute stroke. Thigh length stockings may be considered preferable as more effective and current evidences are poor. However , If these are not suitable because of compliance or fit , knee length versions must be used.

INTERMITTENT PNEUMATIC COMPRESSION

Intermittent pneumatic compression periodically compresses the calf or thigh muscles with inflation pressure of 35 to 40 mm Hg at a rate of about 10 beats per minute. They mimic muscle pump with effect of walking , promote fibrinolysis , and have been shown to reduce DVT risk.

FOOT IMPULSE DEVICES

Foot impulse devices (foot pumps) increase venous outflow and reduce venous stasis in immobilized patients. They are designed to mimic normal walking by compressing plantar venous plexus producing the pulsatile flow in the veins. These have been shown to be effective after major surgeries in reducing the asymptomatic DEEP VEIN THROMBOSIS.

PHARMACOLOGICAL METHODS

Pharmacological prevention involves the perioperative administration of anti-coagulant drugs. The agents relevant to currently practiced were unfractionated heparin , low molecular weight heparin , oral direct factor Xa inhibitor ,warfarin , aspirin , danaparoid , fondaparinux ,lepirudins and dextrans.

UNFRACTIONATED HEPARIN

Heparin is naturally occurring anticoagulant derived mainly from porcine intestine or bovine lung. It is a large polysaccharide varying size from 5 to 40 Kda. It finds to endogenous antithrombin , producing complex that inhibit activated coagulation factors , including factor Xa and IIa (thrombin). Unfractionated heparin is effective in prevention of DVT. However , subcutaneous administration was less predictable than LMW heparin , it does have an easily measurable mode of action , was reversible , and may be preferred in patients with renal failure. For most patients it is inconvenient to deliver , may induce greater bleeding risk when compared with LMWH , and may cause the rare complication of heparin induced thrombocytopenia. Unfractionated heparin is delivered as twice or three times per day subcutaneous injection ,

the anti coagulant effect of the heparin is measured for using activated partial thromboplastin time. This was not required for prophylactic heparin use. However , , ,because of the risks of HIT , platelets should be monitored intermittently up to day 14 of administration.

LOW MOLECULAR WEIGHT HEPARIN

LMWHs are made from short chain polysaccharides with a molecular weight <8KDa. They have the similar mode of action to unfractionated heparin , but a more predictable dose response with primarily anti factor Xa activity and only limited anti IIa activity. A range of products were obtained by various methods of fractionation or depolymerization of heparin which include enoxaparin ,daltaparin .Tinzaparin ,which give varying chemical , physical and biological properties. LMWHs are more effective at reducing the risk of both DVT and PE with the lower risk of bleeding, HIT, osteoporosis than unfractinated heparin. LMWHs are cleared by the kidneys and should be used caution with severe renal failure. Doses of LMWHs depend on formulation but all are delivered as a once daily sub cutaneous injection. LMWHs require no laboratory monitoring although antiXa assay can be used if drug administration

was suspected in cases of severe renal failure. Platelets should be monitored because of the risks of HIT as with unfractionated heparin.

WARFARIN

Warfarin inhibits production of the vitamin k dependent coagulation factors II ,VII ,IX , and X in the liver. Warfarin must be admininistred at a dose that minimizes the hazards of when comparing its DVT and PE prevention effects with LMWH , although major differences in bleeding risks have not been determined. Warfarin is delivered as single oral daily dose , which is its main advantage , aiming for an INR of 1.3 to 1.5. Disadvantages with its use include an unpredictable effect an coagulation , delay in reaching the therapeutic effect and the potential drug interactions. Monitoring is therefore required every 3 days until stable and then weekly.

DIRECT FACTOR Xa INHIBITORS

The first oral direct acting factor Xa inhibitors , Rivaroxaban and dabigatran , have recently been manufactured offering single daily dosing avoiding the need for the alternative parenteral preparations. They have no effect on prothrombin or platelet function . They are currently licensed in the UK for use in DVT prevention in primary

hip and knee arthroplasty. The anticoagulant effects can be measured using the prothrombin time although routine monitoring was not required. Initial studies have suggested superior thromboprophylaxis to LMWH with similar bleeding risks.

ASPIRIN

Aspirin is a salicylate which irreversibly inhibits cyclooxygenase 1 thereby preventing thromboxane A2 production , which has an important role in platelet aggregation . Aspirin reduces the risk of DVT and PE compared with no prophylaxis though its effect was significantly less than heparin , with the similar increase risk of major bleeding. The optimal dose has not been determined and on its own is insufficient for adequate DVT prophylaxis. Aspirin is used as a single oral daily dose of 75 to 300 mg. However, it is not recommended in any of the major international thromboprophylaxis guidelines.

DANAPAROID

Danaparoid is a heparinoid mixture , which inhibits factor Xa but is chemically distinct from LMWH. It is usually used in patients who had developed HIT. However , there may be some cross reactivity of the antibody to this molecule. It is as effective as LMWH , but may induce the higher risk of major bleeding. These

drugs are administered as a twice daily as subcutaneous injection fo r thromboembolism prophylaxis. Anti – Xa assay levels may be used to monitor the effect. However, this is not required routinely. Platelets monitoring are required for HIT because of potential cross reactivity.

FONDAPARINUX

Fondaparinux is a synthetic polysaccharide having almost identical structures to the high- affinity binding site to antithrombin found on heparin. Its effect appears to be antithrombin as an indirect inhibitor of factor Xa. It had a substantially lower incidence of HIT compared with LMWHs and can be used as a substitute in this situation. Fondaparinux has been demonstrate to be superior to LMWH in prevention of DVT but with some evidence of a higher incidence of major bleeding , although its significance is yet to be established. These drugs are delivered parenterally as a single daily subcutaneous injection. Monitoring can be achieved through an anti-Xa assay. However, this is not required in uncomplicated Treatment situation.

LEPIRUDIN

Lepirudin is a recombinant hirudin is derived from yeast cells and is a direct thrombin inhibitor. It is used as an anticoagulant in

patients who have suspected or confirmed HIT. Despite thrombocytopenia , the risk of thrombosis may be upto 50 % unless an anticoagulant is used. These drugs are delivered as continuous infusion and its activity is measured using the aPTT. Because of the risk of thrombosis , a ratio of 2.0 to 3.0 is the target.

DEXTRANS

Dextran is polysaccharide which , by adhering to red blood cells and platelets, reduces their adhesiveness. Additionally , it enchances the effects of antithrombin. It is available as different molecular weights (e.g.Dextran 10,40,60and 70.) which have additional effects . Volume expanders and which in large volumes provide dilutional antithrombotic effects. Dextrans need to be administered intravenously , with volumes in excess of 1500 ml required to achieving an anticoagulant effect. These drugs used therefore generally confined to the intraoperative period only. The larger molecules are resistant to renal excretion and consequently retain their antithrombosis effects for longer. Side effects include hypersensitivity reactions , fluid overload , ,and interference with the blood group testing because of red cell clumping. Dextran is effective at reducing DVT although this is less

effective than other pharmacological treatments. Hence dextran is no longer recommended for DVT prophylaxis.

REGIONAL ANAESTHESIA

The use of lower limb neuroaxial block , as either a single injection or the continuous infusion , has demonstrated a significant reduction in DVT formation compared with patients receiving general anaesthesia alone. The REGIONAL ANAESTHESIA effects are an improved blood flow through the legs secondary to sympathectomy-induced vasodilatation. This is at a time when patient immobility and hypercoagulable state produced by the surgical stress response create a high risk environment for DVT formation. Consideration must be given to pharmacological dose times and neuroaxial block.

PATIENT INFORMATION

Although an evidence based for this mode of prevention is lacking , it is still recommended that healthcare professionals offer verbal and written information before surgery. This should be as part of obtaining surgical consent , detailing the risks and the effectiveness of prophylaxis . It may be extend to information relevant on discharge including signs and symptoms of DVT and PE , the correct

use of prophylaxis at home , and potential implications of failure to follow this advice .

PHYSIOTHERAPHY AND NURSING

Immobility and lack of exercise are widely accepted as risk factors for developing DVT . When normal lower limb venous pumb function is lost as result of bed rest ,venous stasis manifests itself in two ways . First, there is a decrease in the linear velocity of blood , affecting venous return from the lower extremities. Secondly , this decrease in the mean flow and pulsation of the venous flow is followed by dilatation of the vein delaying the further venous return and leading to venous stasis. Although robust clinical data are lacking , these risks can potentially mediated by mechanical calf and foot venous compression , bed exercise , active or passive and early mobilization.

HYDRATION

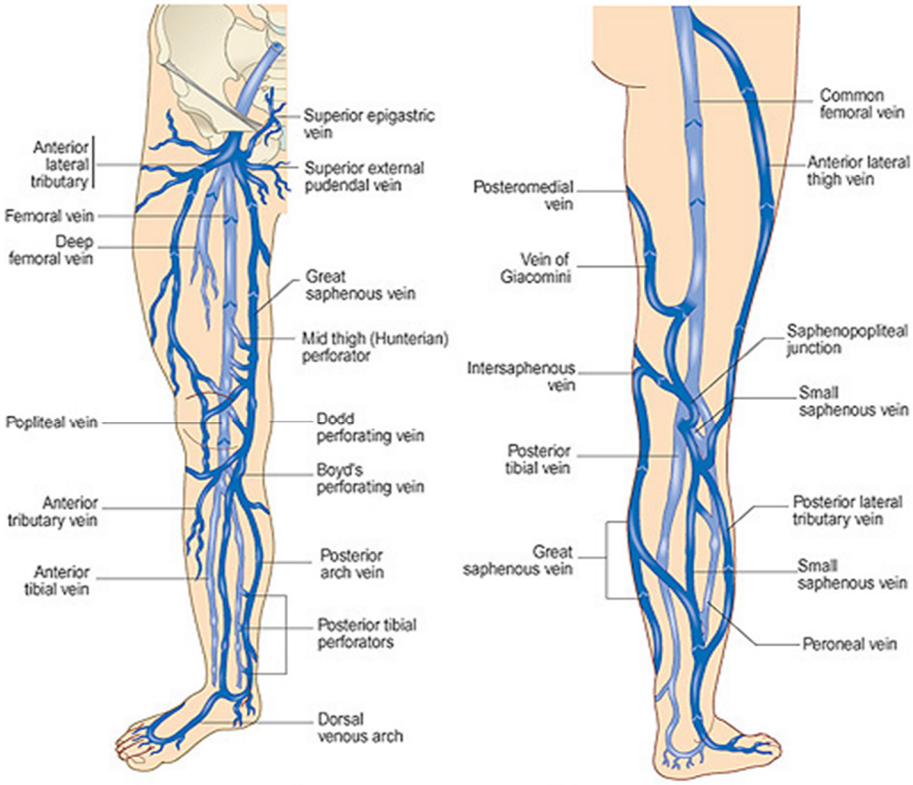
Again clinical evidence was lacking. However avoidance of dehydration in the perioperative period will attenuate the hypercoagulable state produced after surgery and was otherwise good patient management in the majority of clinical situations.

CAVAL FILTERS

Vena caval filters are placed in the inferior or rarely superior vena cava by radiologically controlled techniques. Their purpose is to prevent the embolized thrombous from reaching the pulmonary circulation and can be placed as permanent or temporary retrievable filters. Evidence for their use in the hospitalized patients is limited and therefore they are recommended only for use in patients who have known large proximal DVT , and who have had an embolism (within 1 month) and in whom anticoagulation is contraindicated , they are associated with a higher incidence of recurrent DVTs.

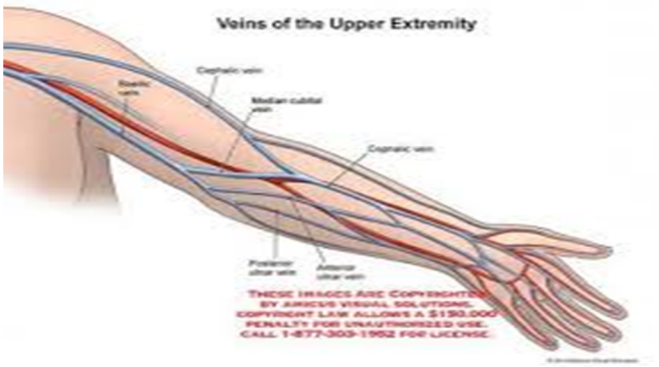
ANATOMY OF VEINS OF UPPER AND LOWERLIMB

FIGURE 1



The Venous Anatomy of the Legs
Deep System - light blue Superficial System - dark blue

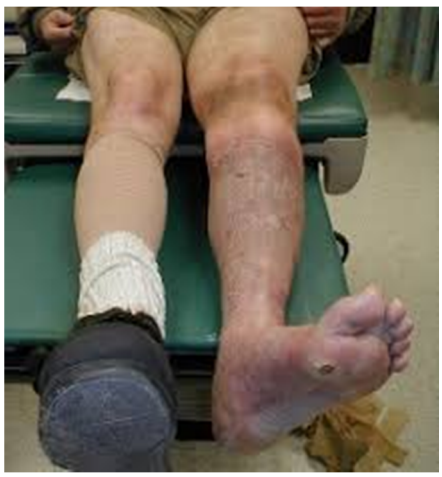
FIGURE 2

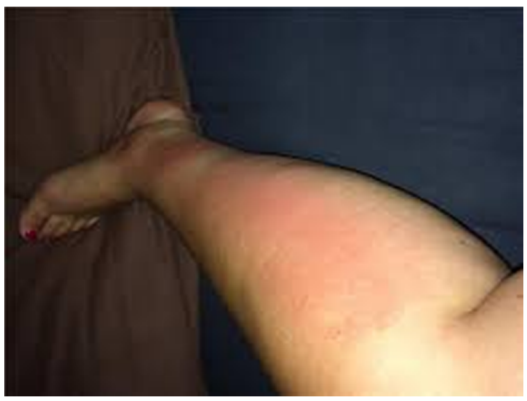


PICTURES OF OUR STUDY POSTOPERATIVE DEEP VEIN THROMBOSIS PATIENTS

FIGURE 3 AND 4

FIGURE 5 AND 6

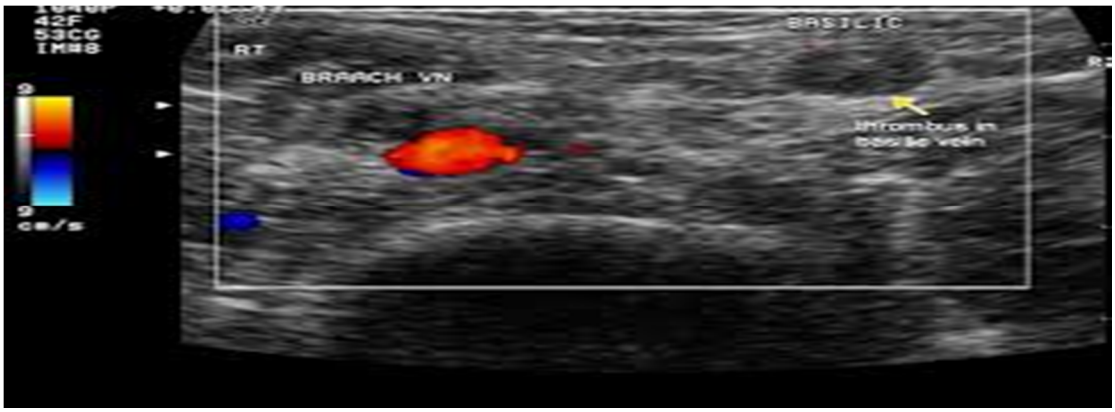


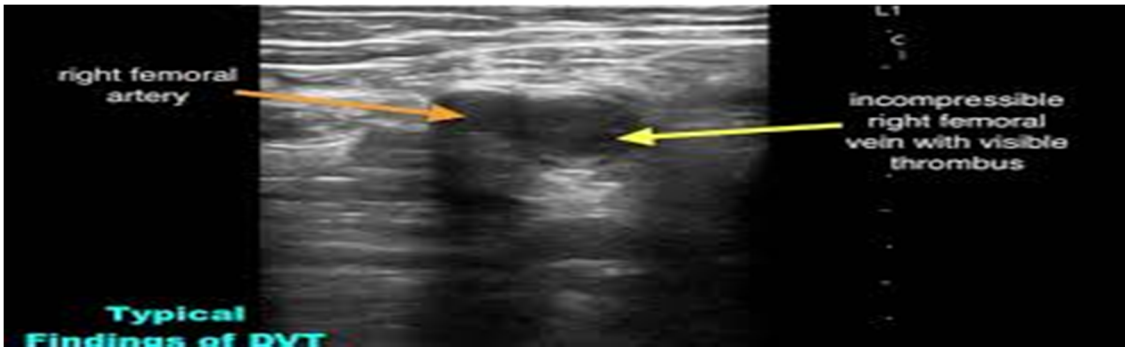


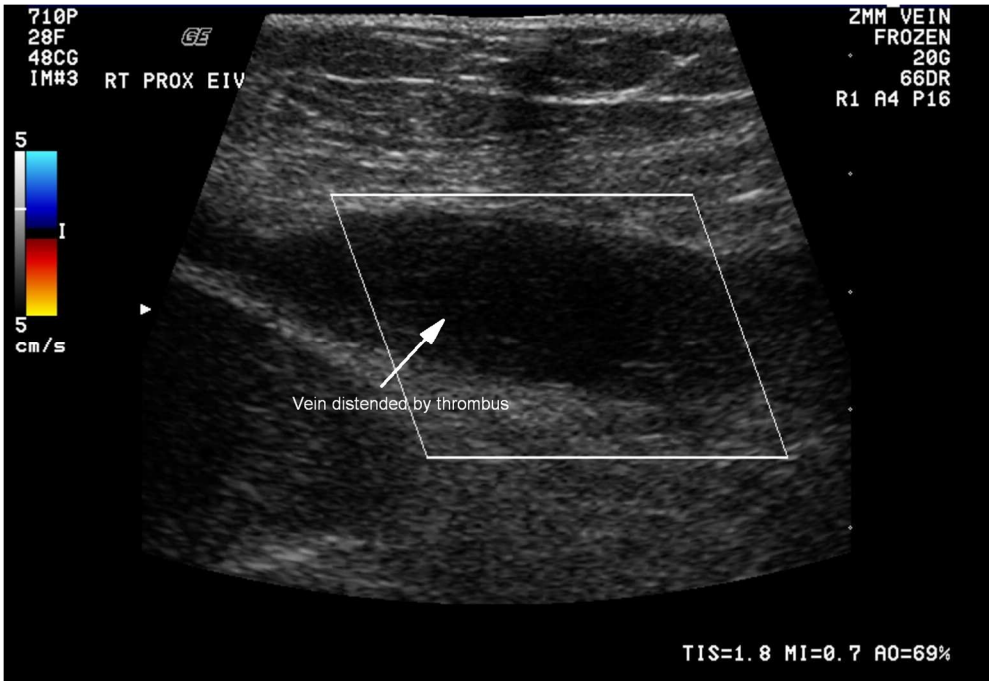
DOPPLER ASSESSMENT PICTURES OF DEEP

VEIN THROMBOSIS

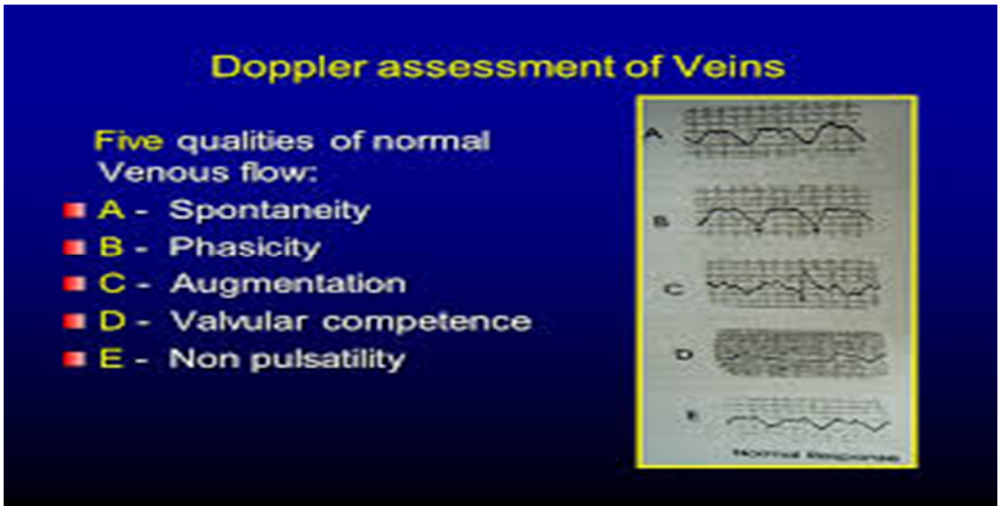
FIGURE 7,8 AND 9







DOPPLER ASSESSMENT OF POSTOPERATIVE DVT



diagnostic criteria for DVT in Duplex

- Among these factors, inability to compress the vein is the most widely used objective criterion for the diagnosis of DVT.
- limitation of compression ultrasound is its lack of accuracy in the evaluation of calf veins
- evaluation of venous flow with color Doppler and spectral Doppler can improve the accuracy of compression ultrasonography

MATERIALS AND METHODS

STUDY DESIGN, METHODOLOGY,TECHNIQUES:

STUDY DESIGN:

CASE CONTROL AND CROSS SECTIONAL STUDY

METHODOLOGY:

In hospital admitted patients undergoing surgery, Doppler assessment of the patients , major abdominal surgeries , individual assessment of risk factors favouring DEEP VEIN THROMBOSIS for the period of one year in COIMBATORE MEDICAL COLLEGE HOSPITAL

SAMPLE SIZE:

N=224 BY 30 PERCENTAGE PREVALENCE RATE.

INCLUSION CRITERIA:

1. All surgical patients in the age group ABOVE 15 YEARS
2. All cancer patients
3. Obesity

EXCLUSION CRITERIA:

- Age below 15 years
- Psychiatric patients
- Pregnancy

RISK FACTOR ASSESSMENT TOOL

POINTS	FACTORS
1	30 to 40 years of age
2	40 to 60 years of age
3	>70 years of age
2	History of smoking
2	Cancer patients underwent surgery
2	Previous history of DVT
2	Family historyof thrombosis
2	History of varicose veins
2	History of chronic medical illness
2	History of connective tissue disorder
2	History of underwent the chemo/radiotheraphy
1	Obesity 25 to 30 BMI
2	Obesity 30 TO 35 BMI
3	Obesity >35 BMI
1	Spinal anaesthesia
2	General anaesthesia
1	30 minutes to 1 hour duration of surgery
2	1 hour to 3 hour duration of surgery

3	> 3 hour duration of surgery
1	<7 days immobilization period
2	7 to 14 days immobilization period
3	> 14 days immobilization period

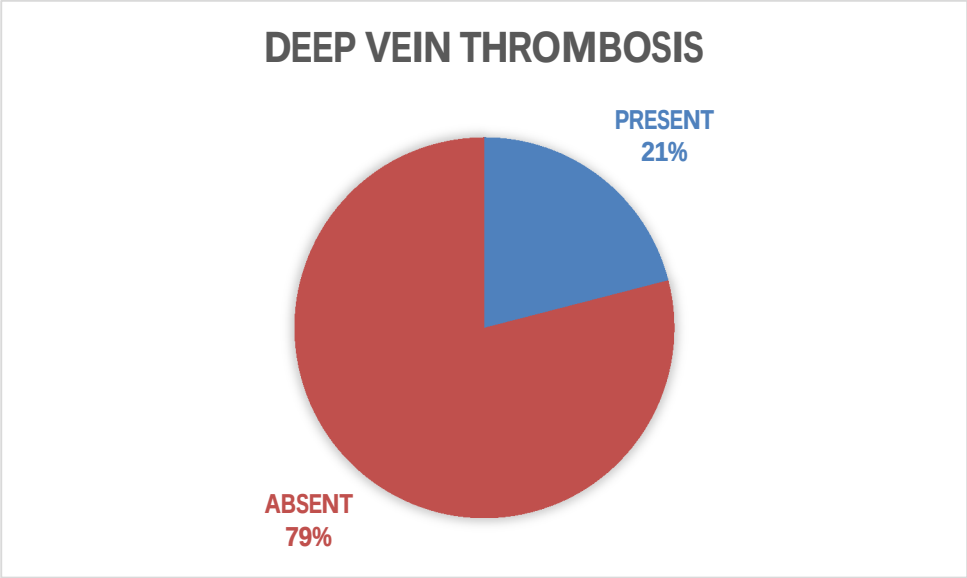
RISK ASSESSMENT SCORE

1 TO 6	LOW RISK GROUPS
7 TO 12	MODERATE RISK GROUPS
>12	HIGH RISK GROUPS

OBSERVATION AND ANALYSIS

TABLE 1 : DVT DISTRIBUTION

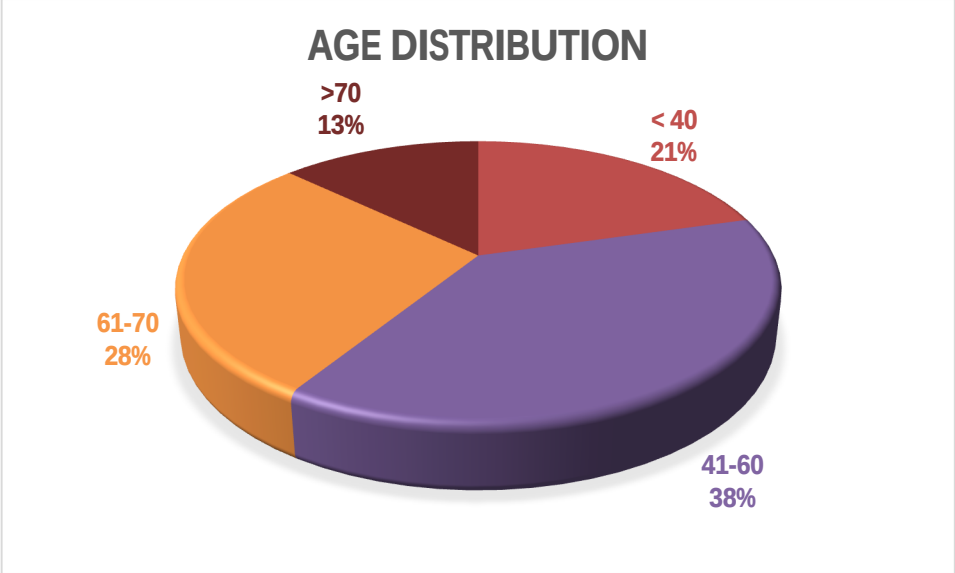
DVT	NO OF PATIENTS	PERCENTAGE
PRESENT	47	21%
ABSENT	177	79%



Total number of patients underwent surgery at CMCH was 224. Among the surgical patients 47 patients developed deep vein thrombosis post operatively around 21 percecnt. And patients not developed Deep vein thrombosis post operatively around 79 percent. Hence in our study 21 percent prevalence risk positive among the surgical patients.

TABLE 2: AGE DISTRIBUTION

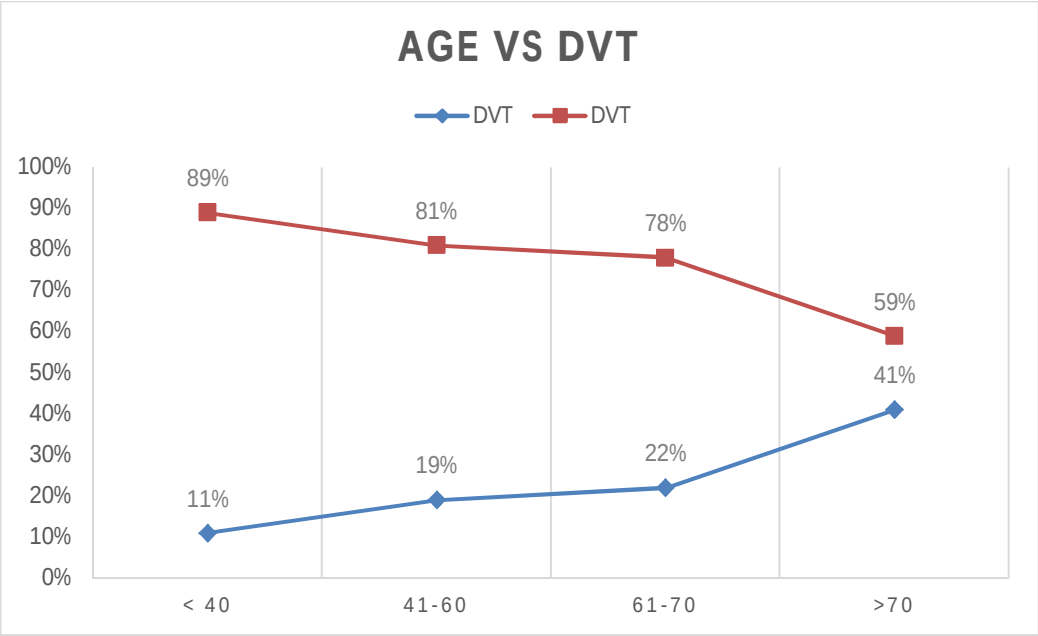
AGE IN YEARS	NO OF PATIENTS	PERCENTAGE
< 40	46	21%
41-60	86	38%
61-70	63	28%
>70	29	13%



In our study 224 patients underwentthe surgery 46 patients comesunder less than 46 years age group with 21 percentage , 86 patients comes under 40 to 60 years age group with 38 percentage , 63 patients comes under 60 to70 years age group with 28 percentage , 29 patients comes under more than 70 years age group with 13 percentage.

TABLE 3: DVT DISTRIBUTION IN AGE GROUPS

AGE IN YEARS	DVT	
	PRESENT	ABSENT
< 40	5	41
41-60	16	70
61-70	14	49
>70	12	17
KRUSKAL WALLIS TEST		
P VALUE - 0.015		
SIGNIFICANT		

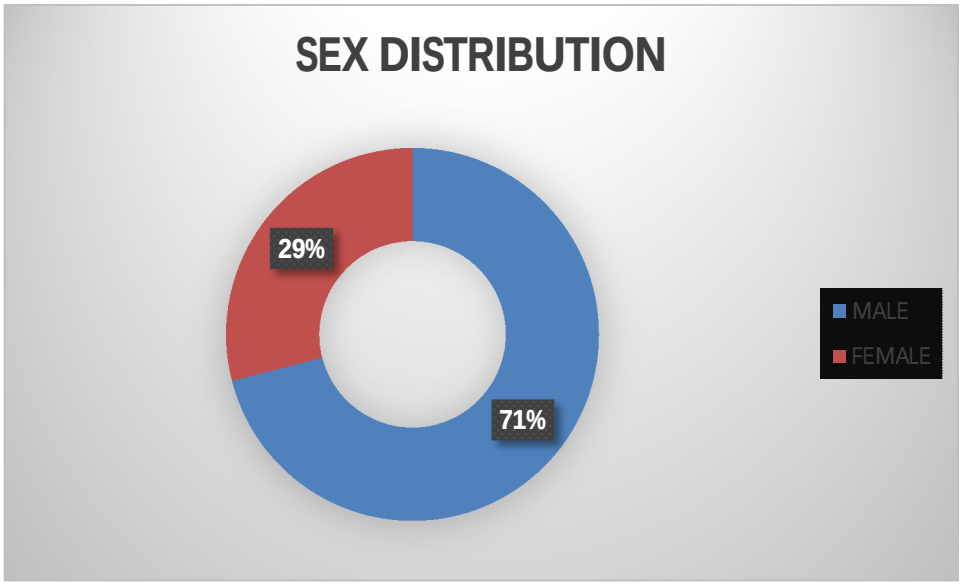


In age group< 40 years patients 5 patients developed DVT post operatively among the 46 patients. In age group 41 to 60 years old patients 16 patients developed DVT postoperatively among the 86 patients. In age group 60 to 70 years patients 14 patients developed DVT post operatively among the 63 patients.

In age group > 70 years old patients 12 patients developed DVT among the 29patients. Finally in our study > 70 years old patients more prone to develop DVT risk patients underwentsurgery with prevalence of 46 percent. KRUSKAL WALLIS test showed P value of 0.015 with significant result.

TABLE 4 : SEX DISTRIBUTION

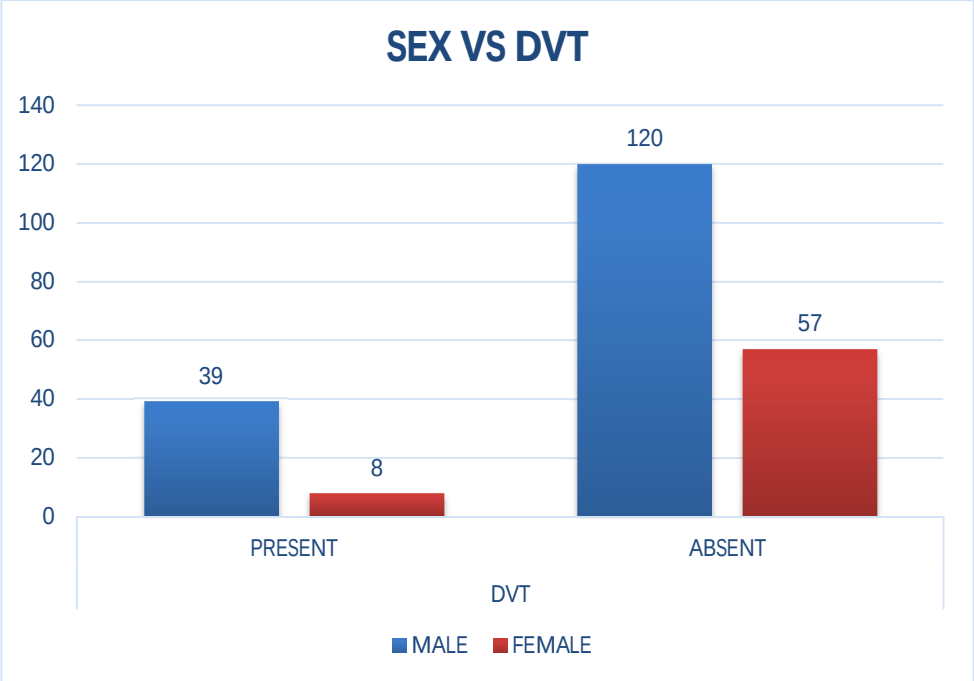
SEX	NO OF PATIENTS	PERCENTAGE
MALE	159	71%
FEMALE	65	29%



In our study 224 patients underwent surgery 159 male patients with 71 percentage and 65 female patients with 29 percentage.

TABLE 5 : DVT DISTRIBUTION AND SEX

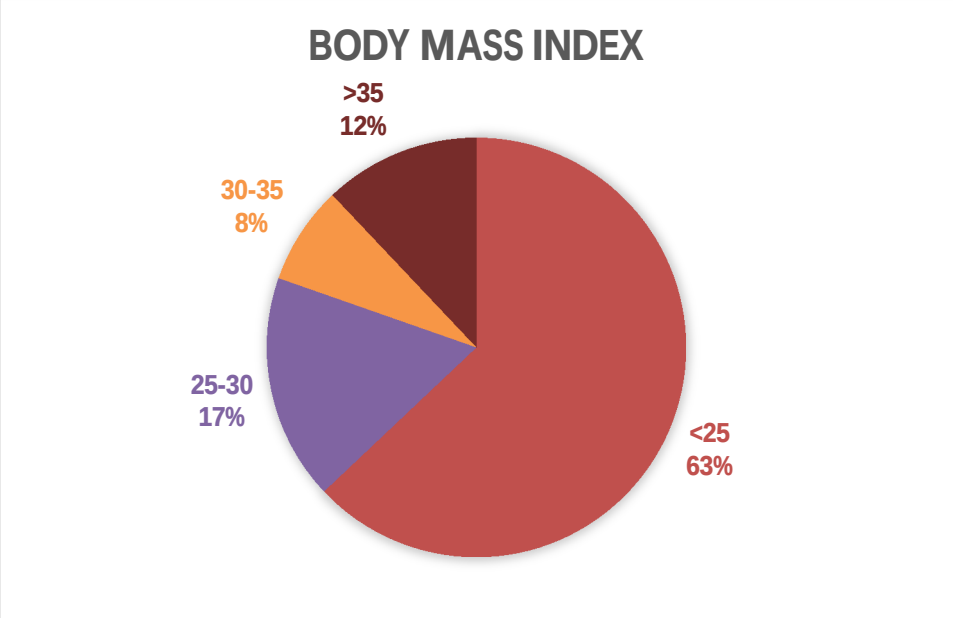
SEX	DVT	
	PRESENT	ABSENT
MALE	39	120
FEMALE	8	57
CHI SQUARE TEST		
P VALUE - 0.041		
SIGNIFICANT		



Totally 39 male patients developed DVT post operatively among the 159 male patients . Totally 8 female patients developed DVT post operatively among the 65 female patients.

TABLE 6 : BODY MASS INDEX

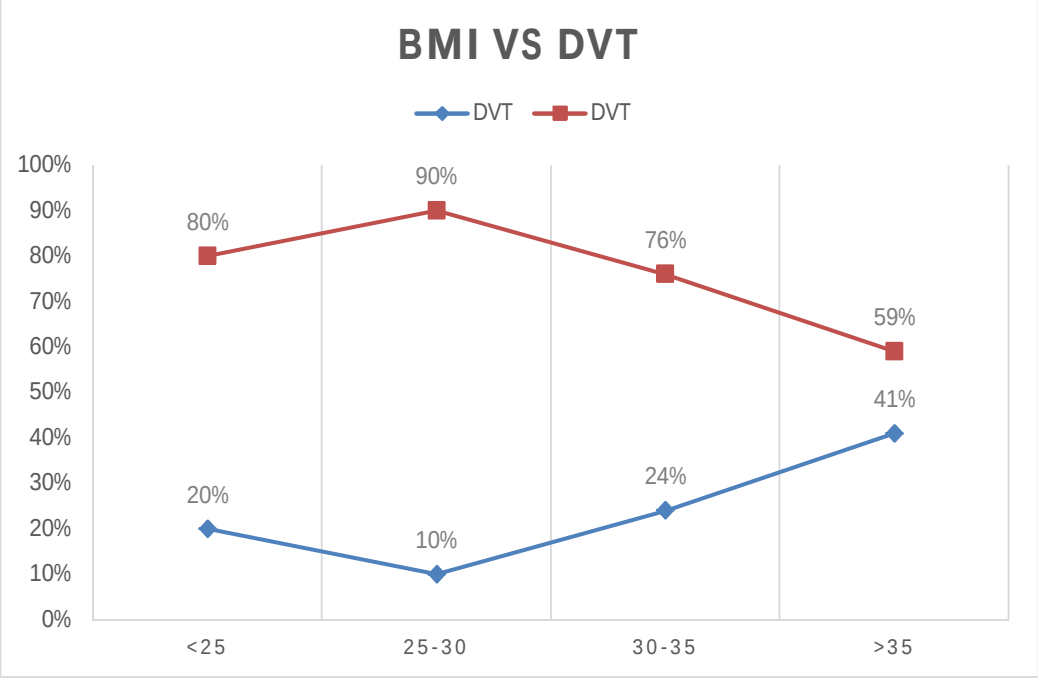
BMI	NO OF PATIENTS	PERCENTAGE
<25	141	63%
25-30	39	17%
30-35	17	8%
>35	27	12%



In our study 224 patients underwent surgery 141 patients comes under BMI of less than 25 with 63 percentage , 39 patients comes under BMI of 25 to 30 with 17 percentage , 17 patients comes under BMI of 30 to 35 with 8 percentage , 27 patients comes under BMI of more than 35 with 12 percentage.

TABLE 7 : DVT DISTRIBUTION ACCORDING TO BMI

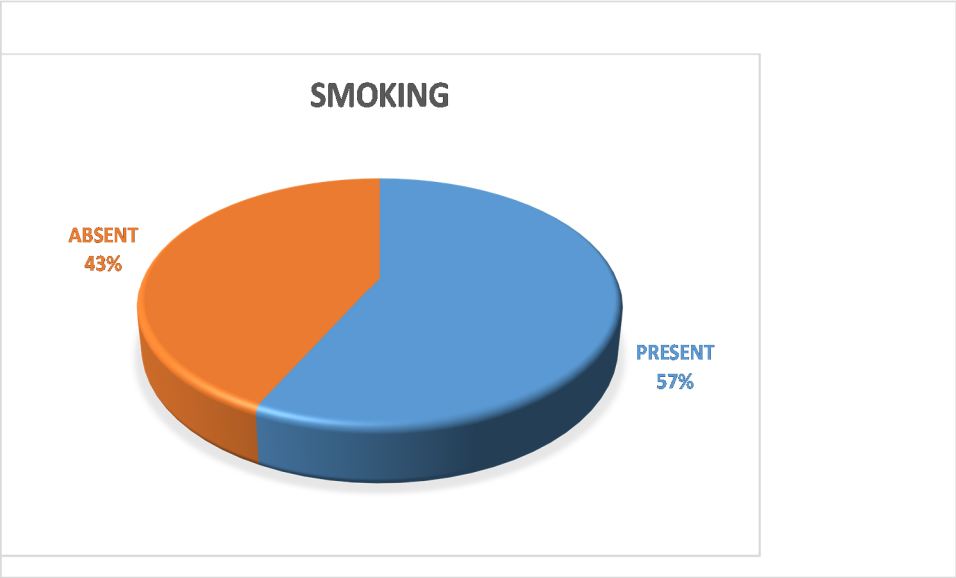
BMI	DVT	
	PRESENT	ABSENT
<25	28	113
25-30	4	35
30-35	4	13
>35	11	16
KRUSKAL WALLIS TEST		
P VALUE - 0.026		
SIGNIFICANT		



Patients with BMI < 25 around 28 patients developed DVT post operatively among the 141 patients. Patients with BMI 25 to 30 around 4 patients developed DVT post operatively among the 39 patients. Patients with BMI 30 to 35 around 4 patients developed DVT post operatively among the 27 patients. Patients with BMI >35 around 11patients developed DVT post operatively among the 17 patients with the prevalence of 41 percent. Compared to other BMI , morbid obese patients undergoing surgery more prone for DVT risk post operatively.

TABLE 8 : SMOKING PATIENTS

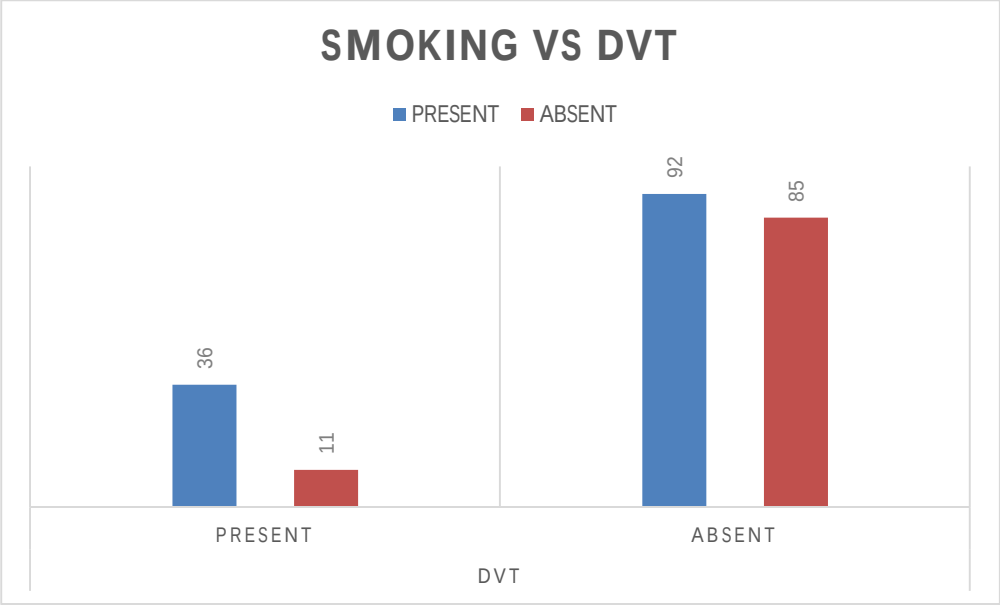
SMOKING	NO OF PATIENTS	PERCENTAGE
PRESENT	128	57%
ABSENT	96	43%



In our study 224 patients underwent surgery 128 patients were smokers with 57 percentage , 96 patients were non smokers with 43 percentage.

TABLE 9 : DVT DISTRIBUTION IN SMOKERS

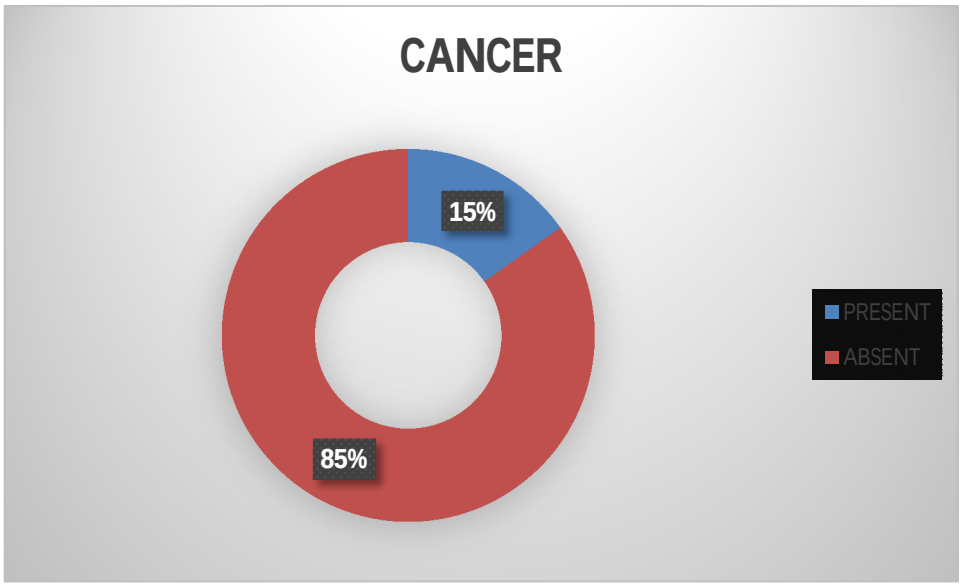
SMOKING	DVT	
	PRESENT	ABSENT
PRESENT	36	92
ABSENT	11	85
CHI SQUARE TEST		
P VALUE - 0.001		
SIGNIFICANT		



Among the 128 patients with smoking history underwent surgery 36 patients developed deep vein thrombosis . Chi square test showed P value of 0.001 with significant result . Hence in our study patients underwent surgery with smoking history more prone to develop the DVT post operatively.

TABLE 10 : CANCER PATIENTS

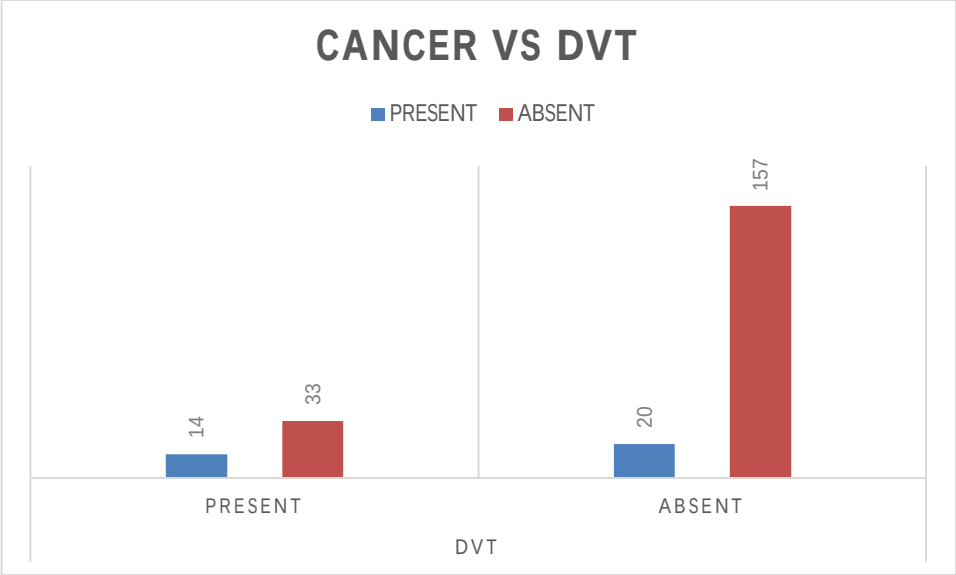
CANCER	NO OF PATIENTS	PERCENTAGE
PRESENT	34	15%
ABSENT	190	85%



Totally 224 patients underwent surgery 3 4 cancer patients with 15 percentage . 190 non cancer patients with 8 5 percentage.

TABLE 11 : DVT DISTRIBUTION AMONG CANCER PATIENTS

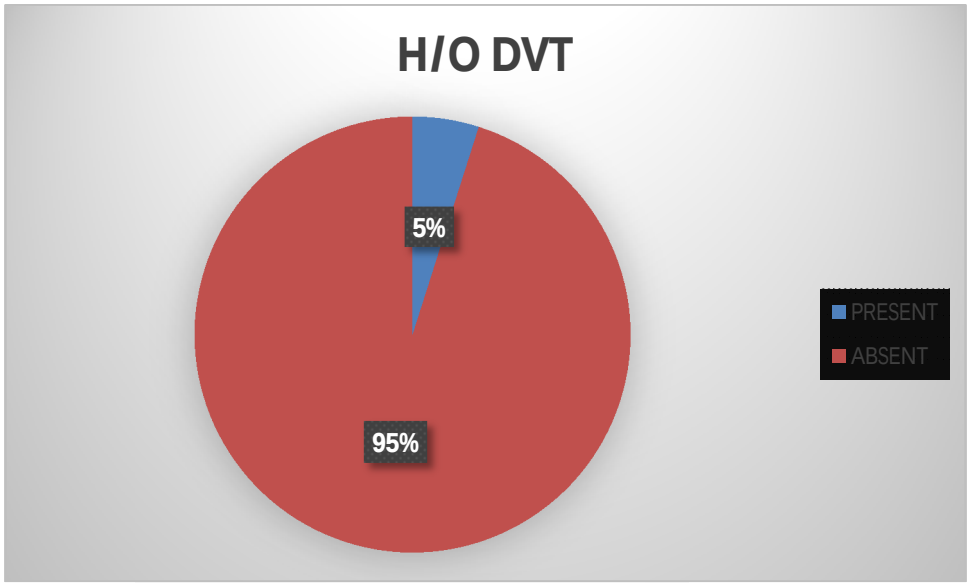
CANCER	DVT	
	PRESENT	ABSENT
PRESENT	14	20
ABSENT	33	157
CHI SQUARE TEST		
P VALUE - 0.002		
SIGNIFICANT		



Total number of 14 patients developed DVT among the cancer patients underwent the surgery with the prevalence of 41 percent. Hence in our study cancer patients underwent surgery more prone to develop DVT risk . Chi square test showed P value of 0.002 with significant result.

TABLE 12 : PREVIOUS HISTORY OF DVT PATIENTS

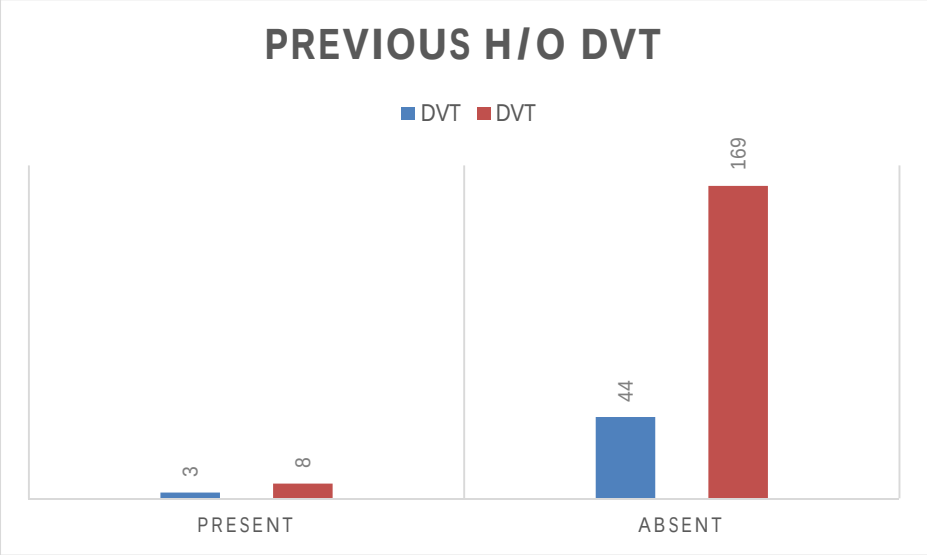
H/O DVT	NO OF PATIENTS	PERCENTAGE
PRESENT	11	5%
ABSENT	213	95%



Among the 224 patients underwent surgery 11 patients had history of deep vein thrombosis previously.

TABLE 13 : DVT DISTRIBUTION AMONG PREVIOUS HISTORY OF DVT PATIENTS

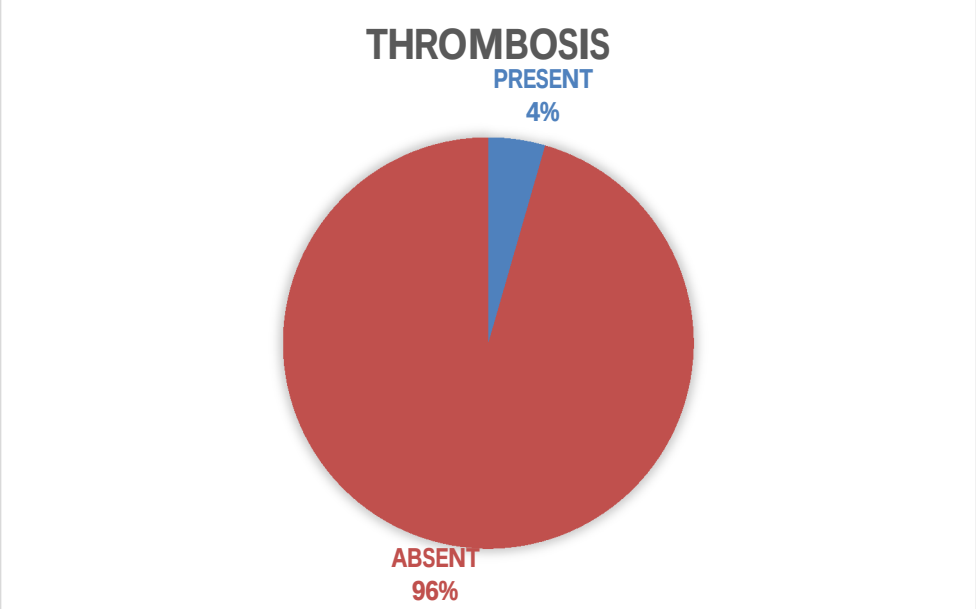
H/O DVT	DVT	
	PRESENT	ABSENT
PRESENT	3	8
ABSENT	44	169
CHI SQUARE TEST		
P VALUE - 0.599		
NON SIGNIFICANT		



In our study 224 patients underwent surgery 3 patients developed post operative deep vein thrombosis had previous history of deep vein thrombosis. In our study chi square test showed p value of 0.599 with non-significant result. In our study patient had previous history of deep vein thrombosis less prone for post-operative DVT. These 3 patients had other risk factors along with previous history of deep vein thrombosis developed post operative DVT.

TABLE 14 : FAMILY HISTORY OF THROMBOSIS PATIENTS

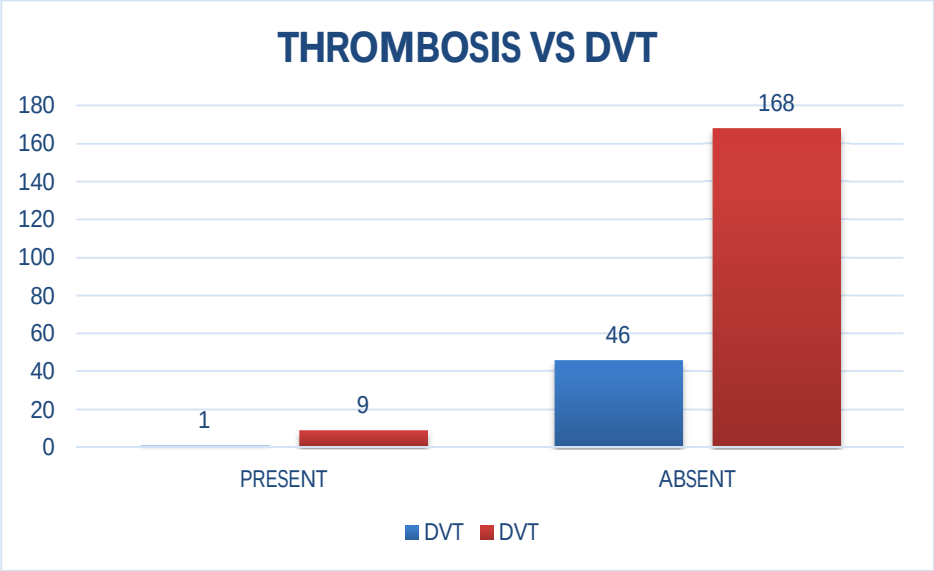
THROMOBOSIS	NO OF PATIENTS	PERCENTAGE
PRESENT	10	5%
ABSENT	214	95%



In our study 10 patients had previous history of thrombosis among the 224 patients underwent surgery.

TABLE 15 : DVT DISTRIBUTION AMONG THE FAMILY HISTORY OF THROMBOSIS PATIENTS

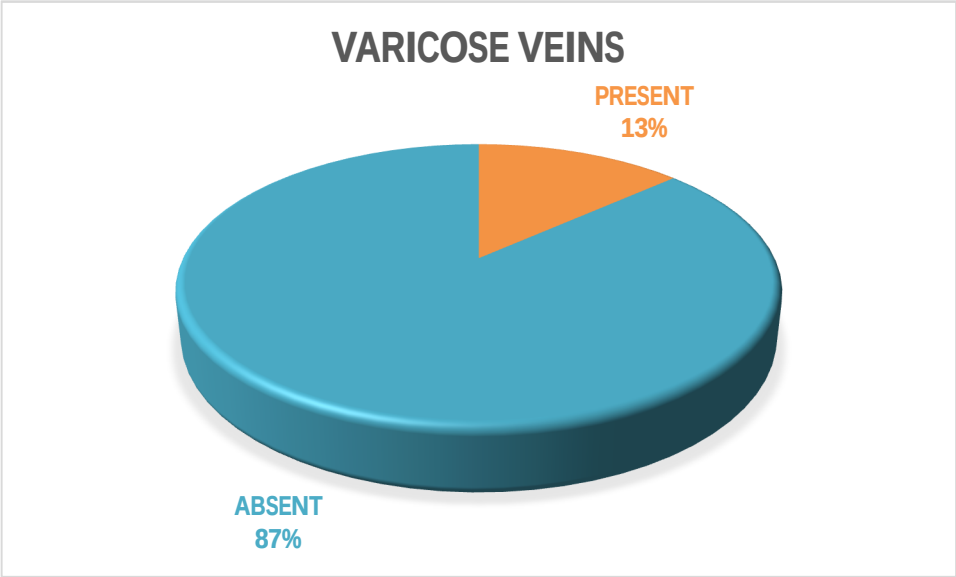
THROMBOSIS	DVT	
	PRESENT	ABSENT
PRESENT	1	9
ABSENT	46	168
CHI SQUARE TEST		
P VALUE - 0.634		
NON SIGNIFICANT		



In our study 10 patients had the previous history of deep vein thrombosis 1 patient developed DVT post operatively. In our study chi square test showed non significant result. Hence in our hospital this study showed previous history of thrombosis patients less likely to develop the DVT compared to other risk factors.

TABLE 16 : VARICOSE VEINS PATIENTS

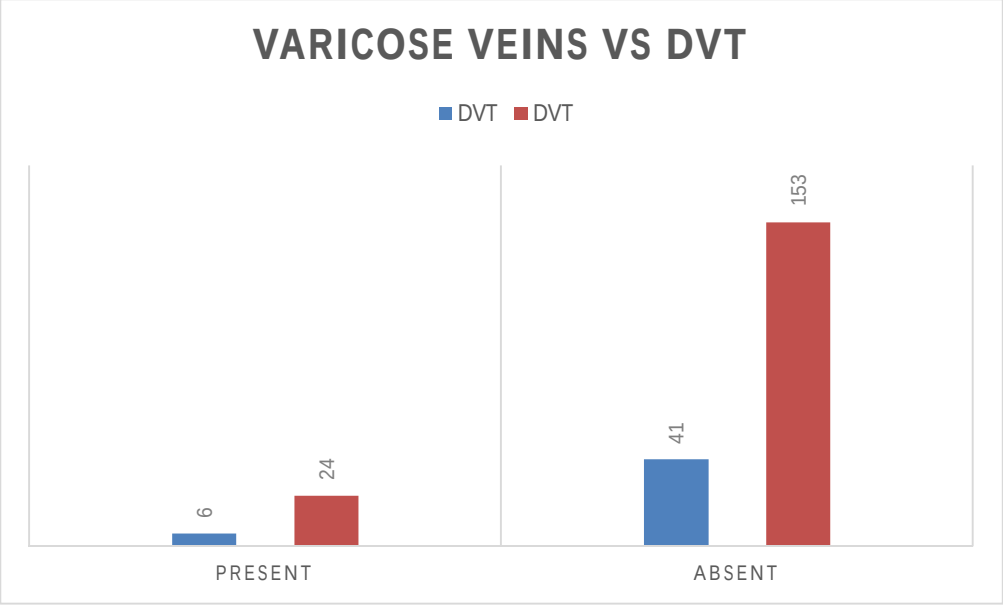
VARICOSE VEINS	NO OF PATIENTS	PERCENTAGE
PRESENT	30	14%
ABSENT	194	86%



30 patients had previous history of varicose vein among the 24 patients underwent surgery.

TABLE 17 : DVT DISTRIBUTION AMONG THE VARICOSE VEIN PATIENTS

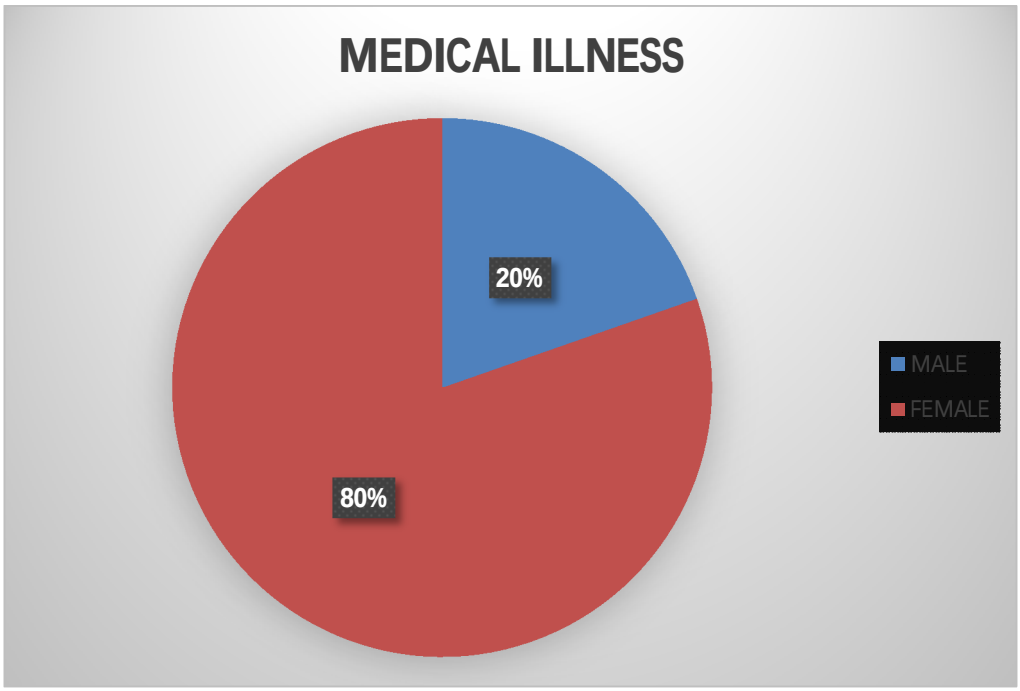
VARICOSE VEINS	DVT	
	PRESENT	ABSENT
PRESENT	6	24
ABSENT	41	153
CHI SQUARE TEST		
P VALUE - 0.865		
NON SIGNIFICANT		



In our study 30 patients had previous history of varicose vein underwent surgery 6 patients developed deep vein thrombosis post operatively. In our study chi square tests showed non significant result. Hence in our hospital this study showed patients with previous history of varicose vein underwent surgery less likely to develop DVT risk compared to other risk factors.

TABLE 18 : MEDICAL ILLNESS PATIENTS

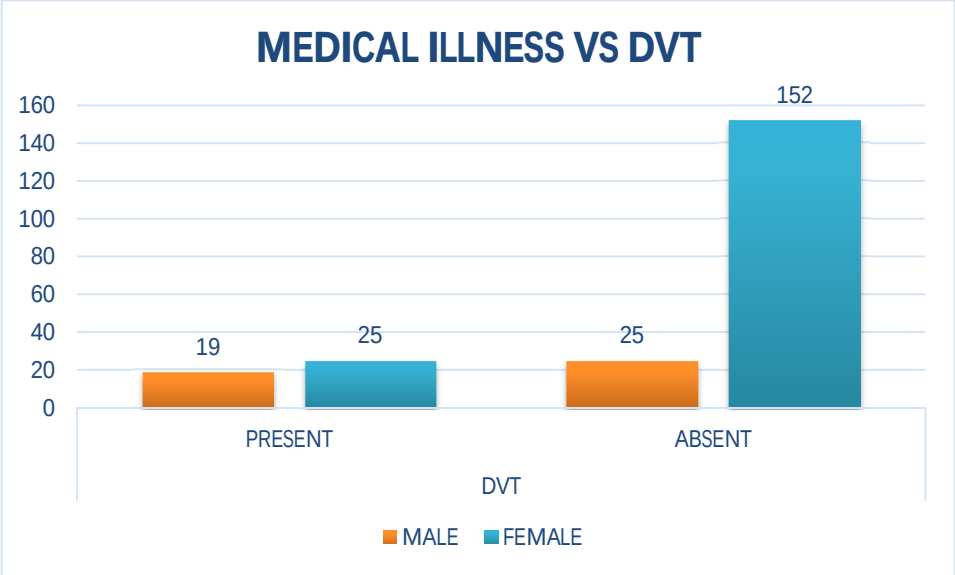
MEDICAL ILLNESS	NO OF PATIENTS	PERCENTAGE
PRESENT	44	20%
ABSENT	180	80%



Totally 44 patients had history of chronic medical illness among the 224 patients underwent surgery.

TABLE 19 : DVT DISTRIBUTION AMONG CHRONIC MEDICAL ILLNESS PATIENTS

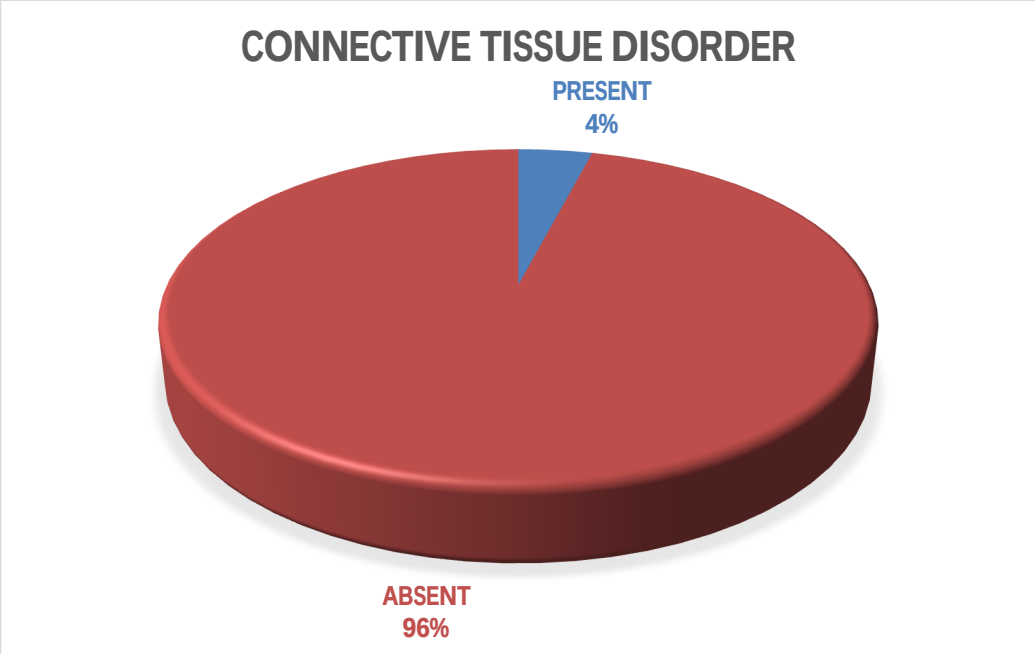
MEDICAL ILLNESS	DVT	
	PRESENT	ABSENT
PRESENT	19	25
ABSENT	25	152
CHI SQUARE TEST		
P VALUE - 0.001		
SIGNIFICANT		



In chronic medical illness patients underwent surgery 19 patients developed deep vein thrombosis among the 44 patients. Chi square test showed P value of 0.001 with significant result. Hence in our study chronic medical illness patients underwent surgery more prone to develop the deep vein thrombosis risk with prevalence of 43 percentage.

TABLE 20 : CONNECTIVE TISSUE DISORDER PATIENTS

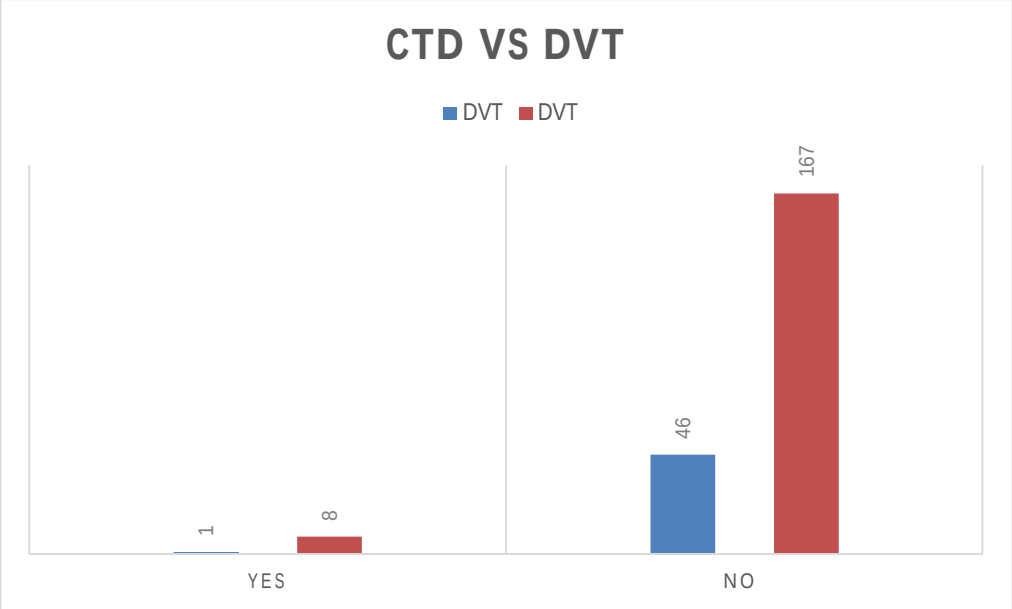
CTD	NO OF PATIENTS	PERCENTAGE
PRESENT	9	4%
ABSENT	215	96%



9 patients had the connective tissue disorder history among the 224 patients underwent surgery.

TABLE 21 : DVT DISTRIBUTION AMONG CONNECTIVE TISSUE DISORDER PATIENTS

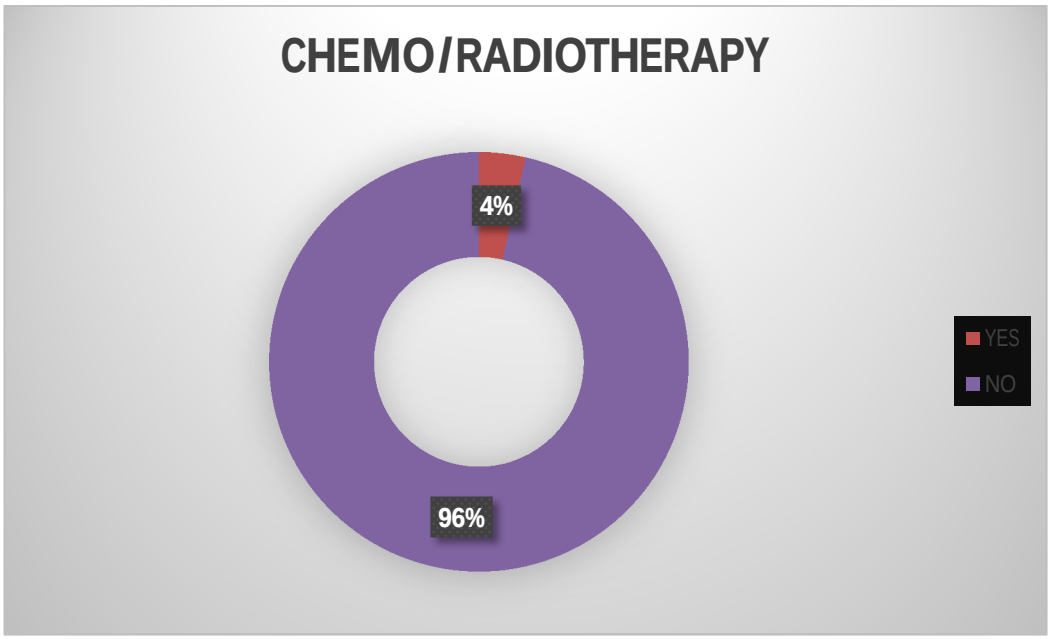
CTD	DVT	
	PRESENT	ABSENT
YES	1	8
NO	46	167
CHI SQUARE TEST		
P VALUE - 0.661		
NON SIGNIFICANT		



Among the connective tissue disorder patients underwent the surgery 1 patient developed DVT post operatively. Chi square test showed non significant result. Hence in our study connective tissue disorder patients underwent surgery less likely to develop deep vein thrombosis risk post operatively.

TABLE 22 : CHEMO/RADIOTHERAPY RECEIVED PATIENTS

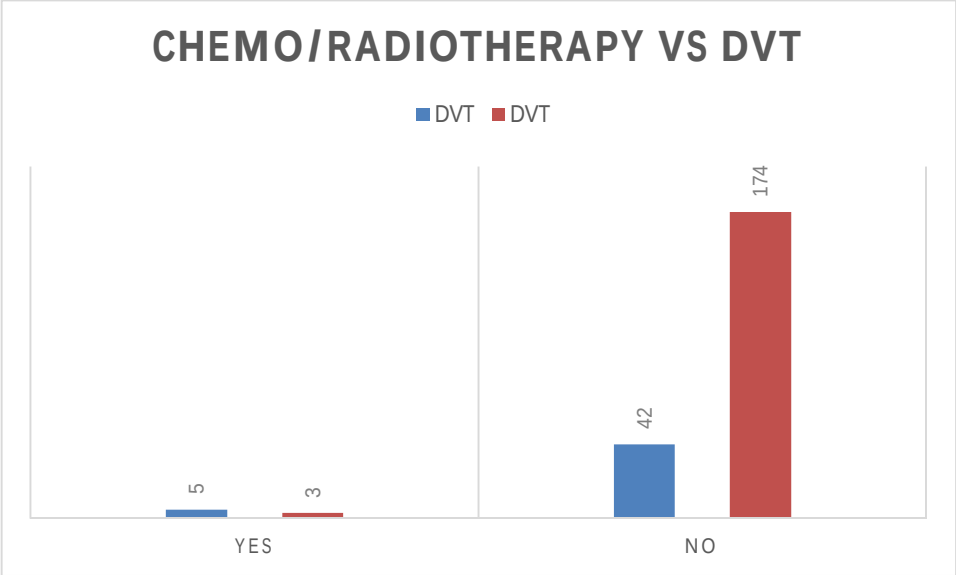
CHEMO/RADIOTHERAPY	NO OF PATIENTS	PERCENTAGE
YES	8	4%
NO	216	96%



Among the 224 patients underwent surgery 8 patients received chemotheraphy /rdaiotheraphy with 4 percentage .

TABLE 23 : DVT DISTRIBUTION AMONG CHEMO / RADIOTHERAPHY RECEIVED PATIENTS

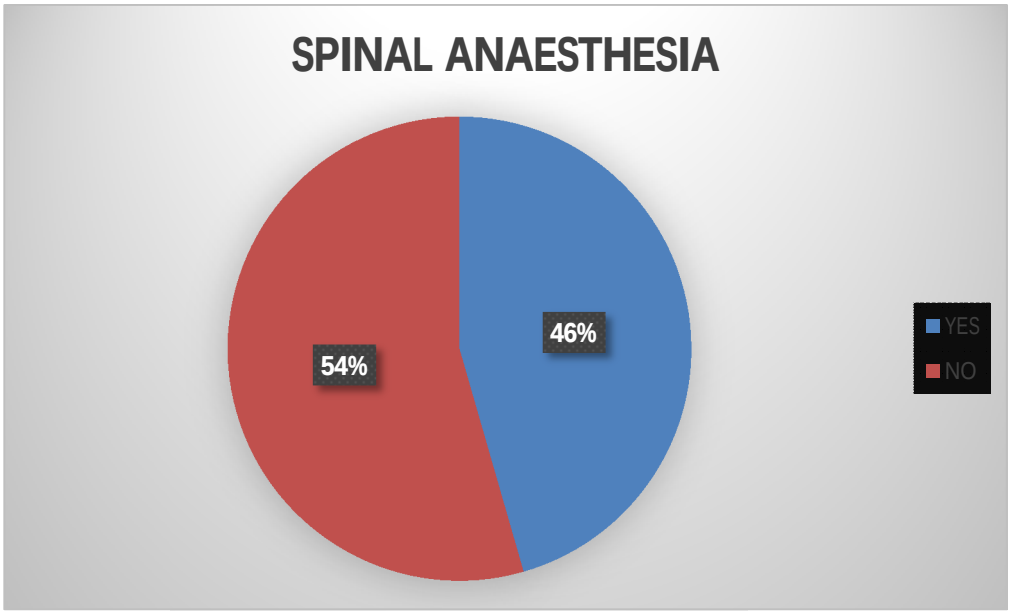
CHEMO/RADIOTHERAPY	DVT	
	PRESENT	ABSENT
YES	5	3
NO	42	174
CHI SQUARE TEST		
P VALUE - 0.003		
SIGNIFICANT		



Among the chemotheraphy/radiotheraphy received patients 5 patients developed DVT Post Operatively. Chi square test showedp value of 0.003 with significant result. Hence in our study chemotheraphy/ radiotheraphy received patients significantly associated with the post operative **DVT risk.**

TABLE 24 : SPINAL ANAESTHESIA

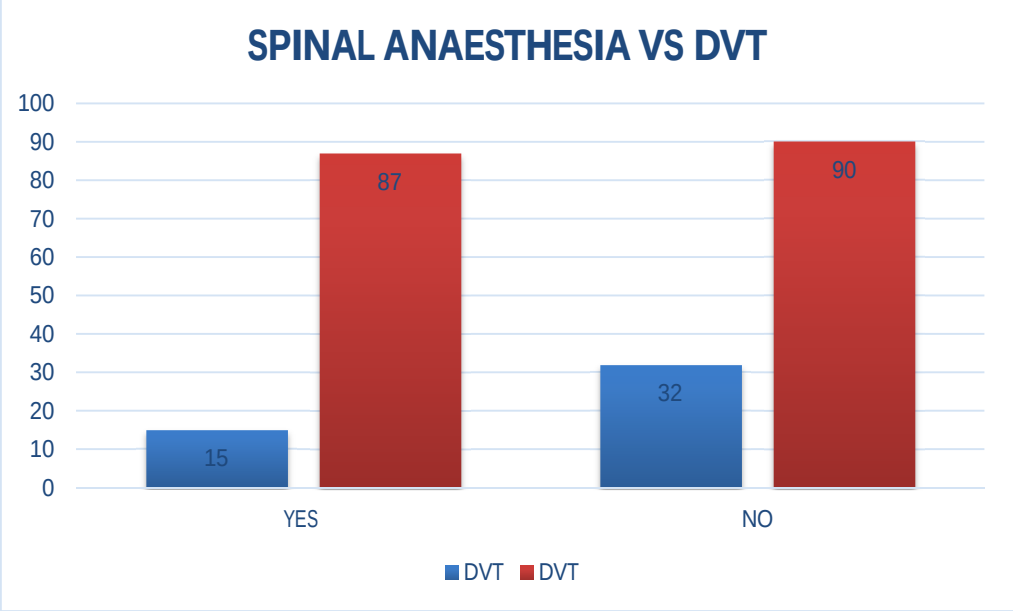
SPINAL ANAESTHESIA	NO OF PATIENTS	PERCENTAGE
YES	102	46%
NO	122	54%



Among the 224 patients 102 patients underwent surgery with spinal anaesthesia.

TABLE 25 : DVT DISTRIBUTION AMONG PATIENTS UNDERWENT SURGERY WITH SPINAL ANAES THESIA

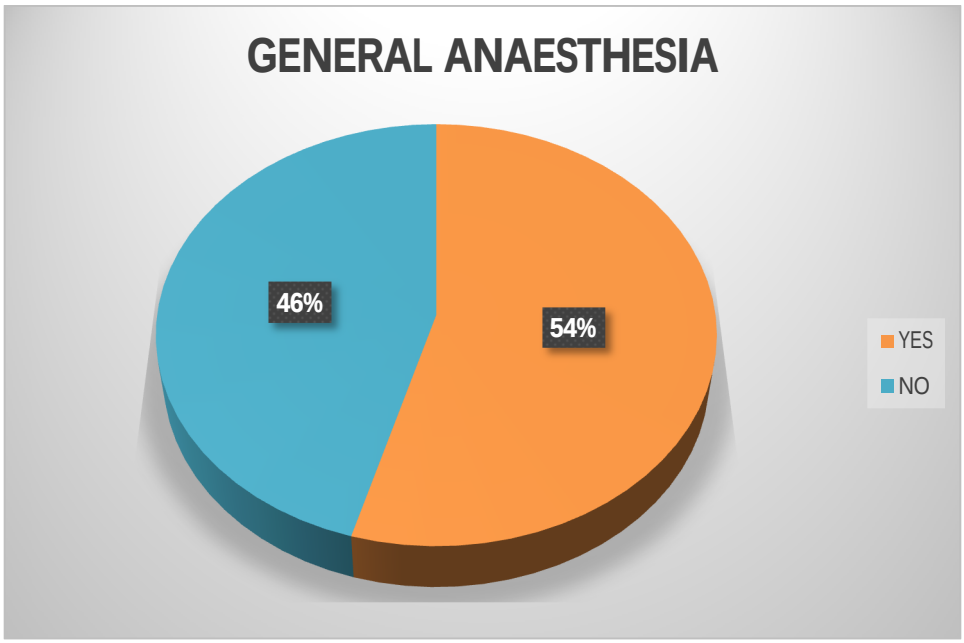
SPINAL ANAESTHESIA	DVT	
	PRESENT	ABSENT
YES	15	87
NO	32	90
CHI SQUARE TEST		
P VALUE - 0.035		
SIGNIFICANT		



In our study 15 patients developed deep vein thrombosis post operatively among the 102 patient underwent surgery with spinal anaesthesia . Chi square test showed P value of 0.035 with significant result. Hence in our study patients underwent the surgery with spinal anaesthesia more prone to develop deep vein thrombosis post operatively.

TABLE 26 : GENERAL ANAESTHESIA

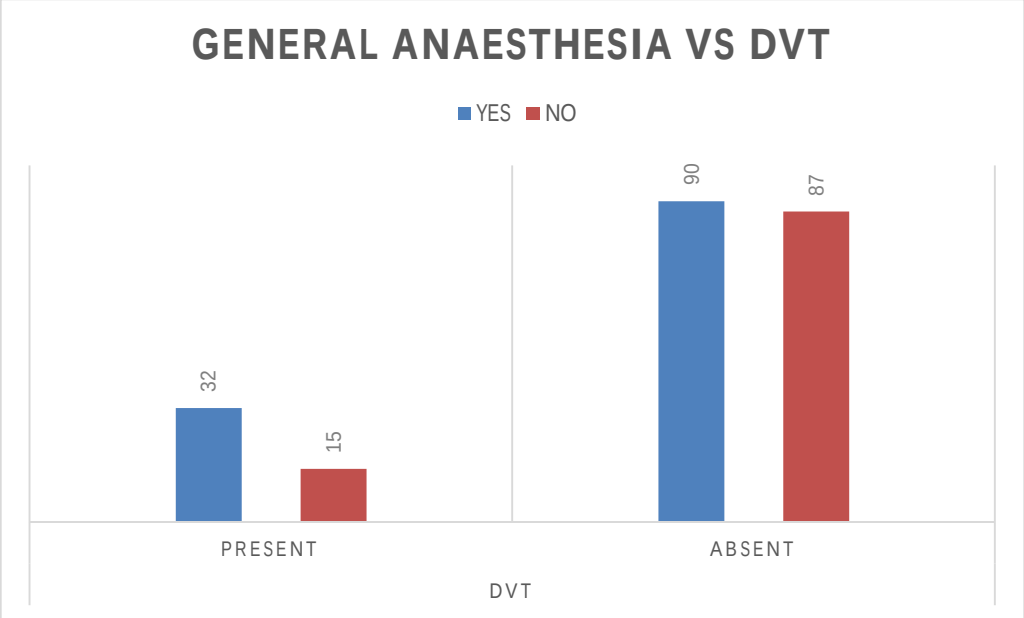
GENERAL ANAESTHESIA	NO OF PATIENTS	PERCENTAGE
YES	122	5%
NO	102	46%



Among 224 patients underwent the surgery 122 patients underwent surgery with general anaesthesia with 54 percentage.

TABLE 27 : DVT DISTRIBUTION AMONG PATIENTS UNDERWENT SURGERY WITH GENERAL ANAES THESIA

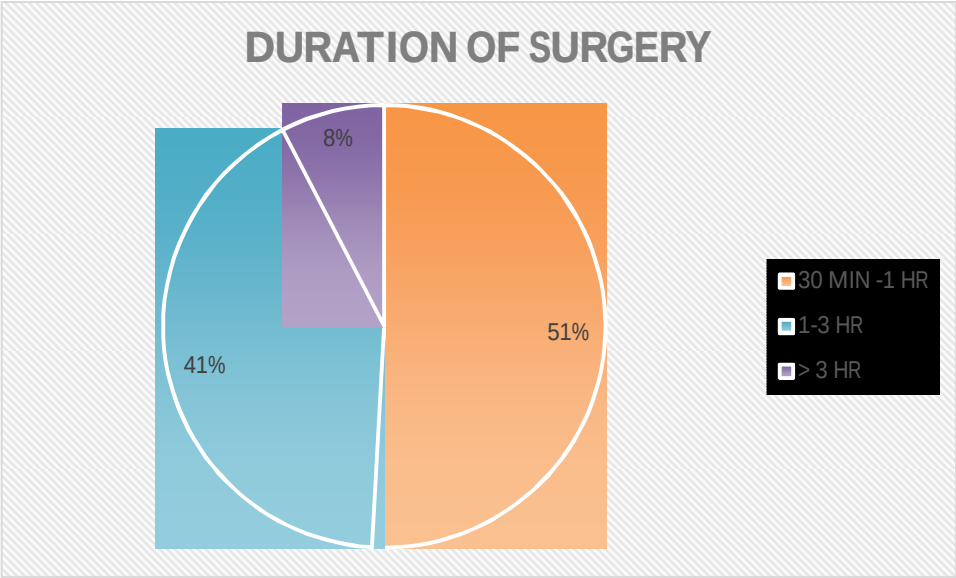
GENERAL ANAESTHESIA	DVT	
	PRESENT	ABSENT
YES	32	90
NO	15	87
CHI SQUARE TEST		
P VALUE - 0.034		
SIGNIFICANT		



In our study 32 patients developed deep vein thrombosis post operatively among the 122 patient underwent surgery with general anaesthesia . Chi square test showed P value of 0.034 with significant result. Hence in our study patients underwent the surgery with general anaesthesia more prone to develop deep vein thrombosis post operatively.

TABLE 28 : DURATION OF SURGERY

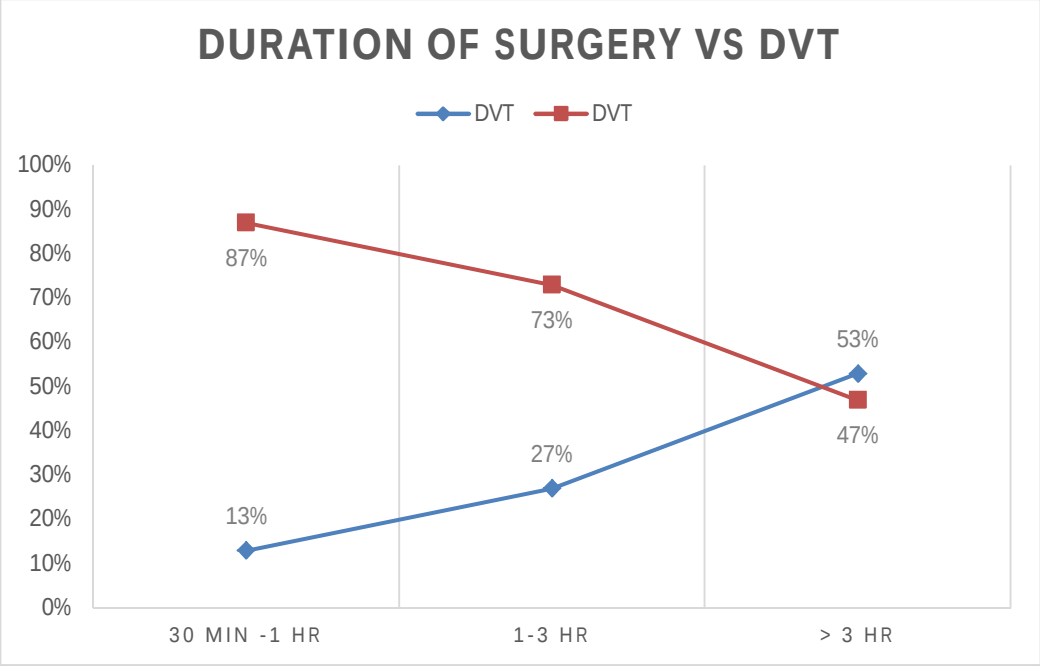
DURATION OF SURGERY	NO OF PATIENTS	PERCENTAGE
30 MIN -1 HR	114	51%
1-3 HR	93	41%
> 3 HR	17	8%



Among the 224 patients underwent surgery 114 patients underwent surgery with duration of 30 minutes to1 hour. 93 patients underwent surgery with duration of 1hour to 3 hour. 17 patients underwent surgery with duration of more than 3 hours.

TABLE 29 : DVT DISTRIBUTION AND DURATION OF SURGERY

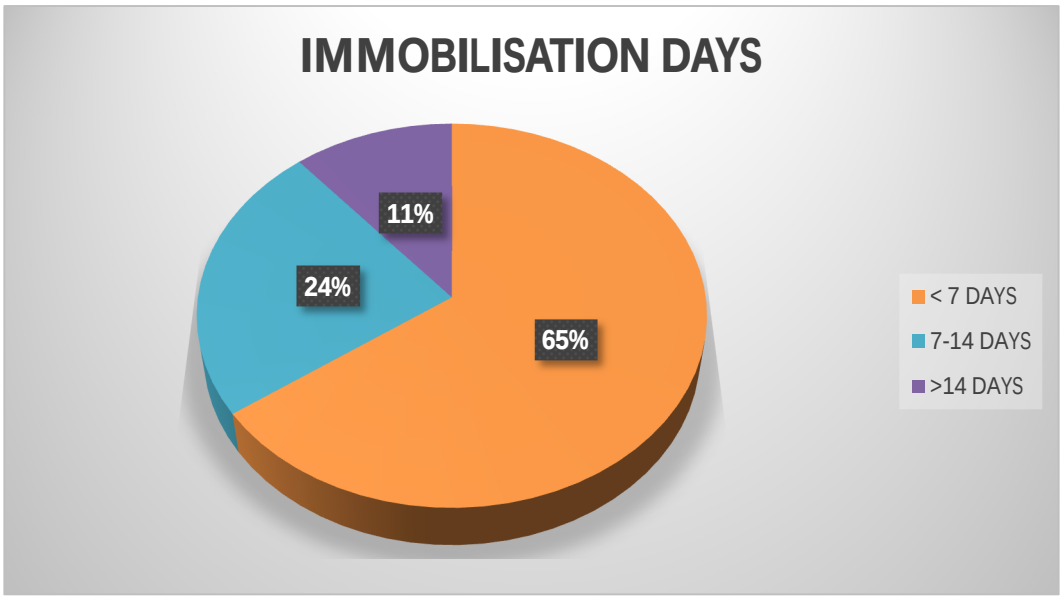
DURATION OF SURGERY	DVT	
	PRESENT	ABSENT
30 MIN -1 HR	13	101
1-3 HR	25	68
> 3 HR	9	8
KRUSKAL WALLIS TEST		
P VALUE - 0.001		
SIGNIFICANT		



13 patients developed deep vein thrombosis with the duration of surgery 30 minutes to 1 hour among the 114 patients . 25 patients developed deep vein thrombosis with the duration of surgery 1 hour to 3 hour among the 93 patients. 9 patients developed deep vein thrombosis with duration of surgery more than 3 hours among the 17 patients . KRUSKAL WALLIS test showed p value of 0.001 with significant result. Hence in our study patients underwent surgery with more than 3 hours duration significantly associated with post operative deep vein thrombosis risk with prevalence of 53 percentage.

TABLE 30 : IMMOBILISATION DAYS

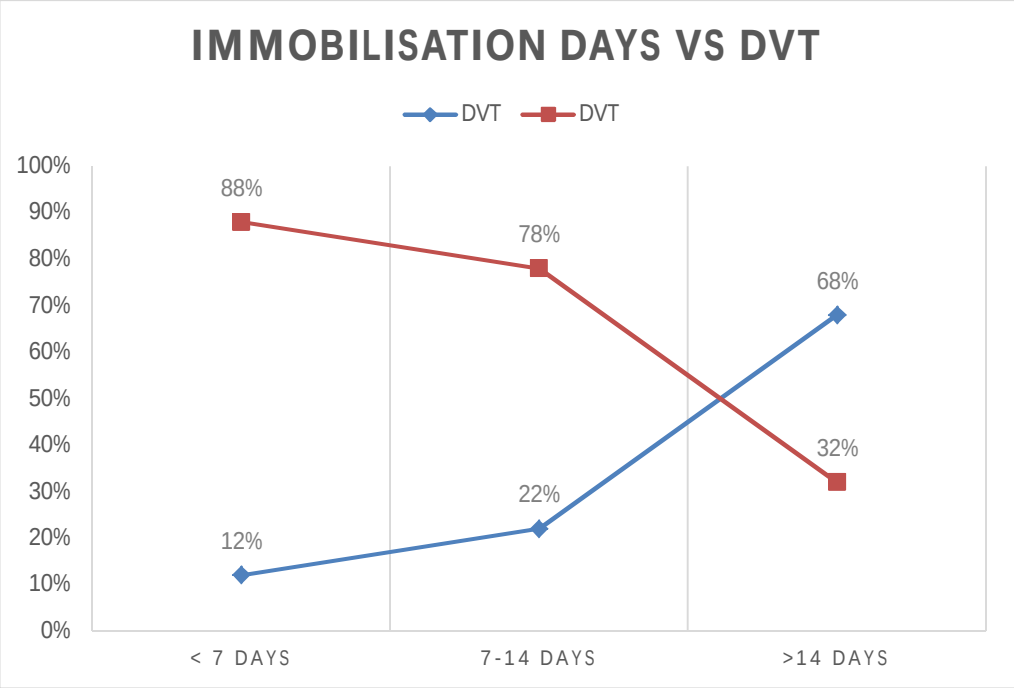
IMMOBILISATION DAYS	NO OF PATIENTS	PERCENTAGE
< 7 DAYS	146	65%
7-14 DAYS	53	24%
>14 DAYS	25	11%



Among the 224 patients underwent surgery 146 patients had less than 7 days immobilization. 53 patients had 7 to 14 days immobilization. 25 patients hadmore than 14 days immobilization.

TABLE 31 : DVT DISTRIBUTION AND IMMOBILIZATION DAYS

IMMOBILISATION DAYS	DVT	
	PRESENT	ABSENT
< 7 DAYS	18	128
7-14 DAYS	12	41
>14 DAYS	17	8
KRUSKAL WALLIS TEST		
P VALUE - 0.001		
SIGNIFICANT		



18 patients developed DVT post operatively patients had less than 7 days immobilization among 146 patients. 5 patients had 7 to 14 days immobilization post operatively developed DVT among 53 patients. 17 patients had immobilization more than 14 days post operatively developed DVT among the 25 patients. KRUSKAL WALLIS test showed p value of 0.001 with significant result. Hence in our study showed patients underwent surgery with more than 14 days immobilization significantly associated with post operative deep vein thrombosis risk with prevalence of 68 percentage.

TABLE 32 : DVT RISK SCORE

RISK SCORE	NO OF PATIENTS	PERCENTAGE
LOW RISK (1-6)	90	40%
MODERATE RISK (7-12)	75	33%
HIGH RISK (>12)	59	27%

Among the 224 patients underwent surgery 90 patients under the low risk group. 75 patients under the moderate risk group. 59 patients under the high risk group.

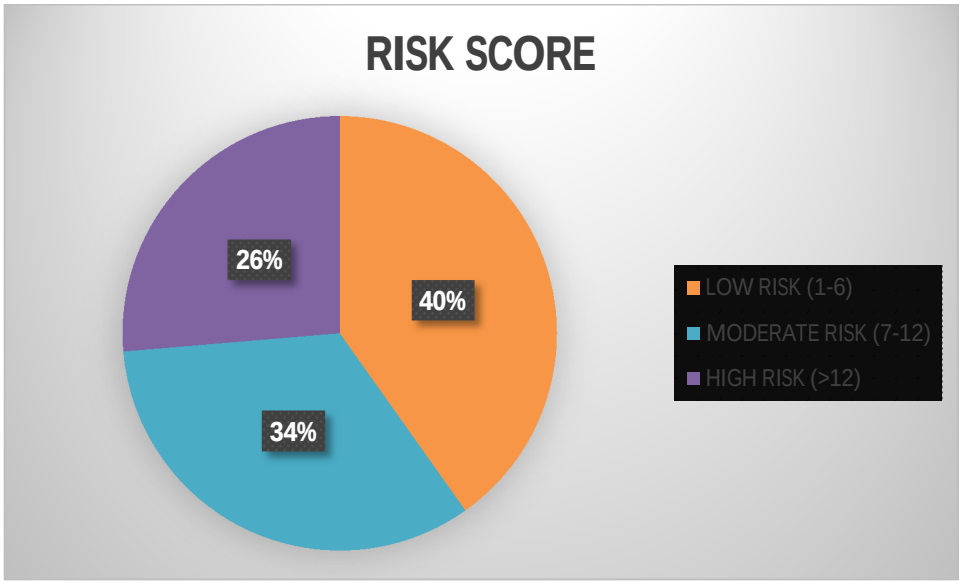
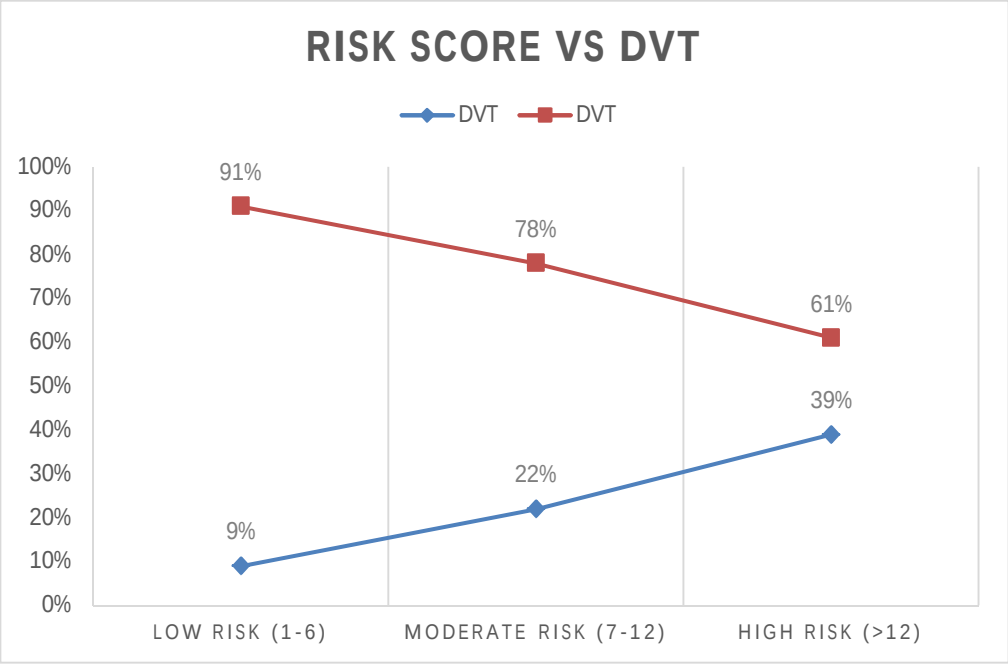


TABLE 33 : DVT DISTRIBUTION AMONG LOW , MODERATE , HIGH RISK GROUP PATIENTS

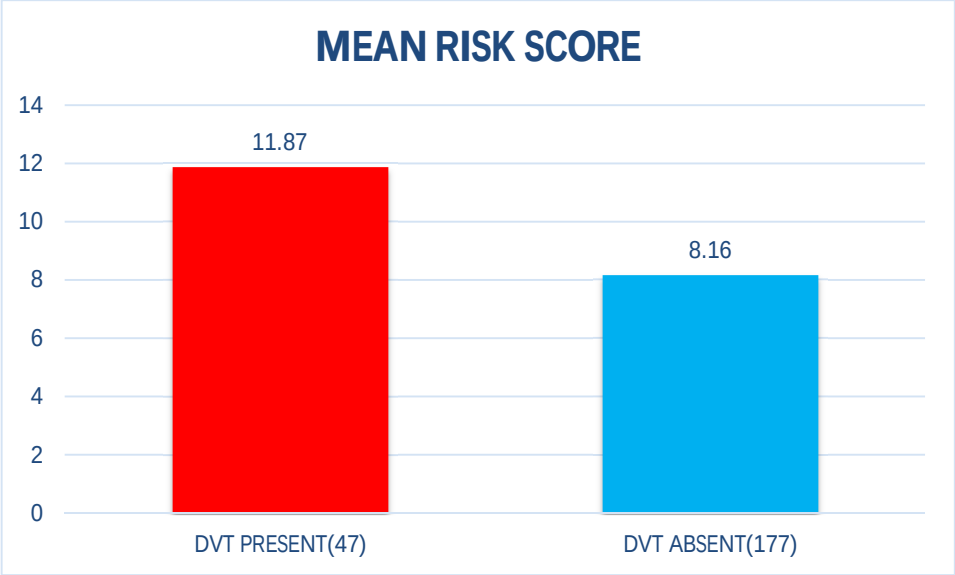
RISK SCORE	***DVT***	
	PRESENT	**ABSENT**
LOW RISK (1-6)	8	82
MODERATE RISK (7-12)	16	59
HIGH RISK (>12)	23	36
KRUSKAL WALLIS TEST		
P VALUE - 0.001		
SIGNIFICANT		



In patients underwent surgery with low risk group 8 patients developed deep vein thrombosis among 90 patients. Moderate risk group, 16 patients developed deep vein thrombosis among 65 patients. High risk group, patients 23 patients developed deep vein thrombosis among 59 patients. UN PAIRED T TEST showed p value of 0.001 with significant result. Hence in our study high risk group patients underwent surgery significantly associated with post operative risk of DVT with the prevalence of 39 percentage.

TABLE 34 : MEAN RISK SCORE VS DVT

DEEP VEIN THROMBOSIS	RISK SCORE	
	MEAN	**SD**
PRESENT(47)	11.87	3.75
ABSENT(177)	8.16	3.64
UNPAIRED T TEST		
P VALUE - 0.001		
SIGNIFICANT		



In our study totally 224 patients underwent surgery 47 patients developed the post operative DEEP VEIN THROMBOSIS with overall 21 percentage.

DISCUSSION

DVT is a common cause of preventable hospital mortality. DVT occurring after general surgery is a common pathology in the absence of specific prophylaxis with significant morbidity and mortality and high socioeconomic costs. This study assessed the potential of several parameters to be risk factors contributing to the occurrence of DEEP VEIN THROMBOSIS of the lower limbs after general surgery for the patients without preoperative DVT prophylaxis.

Approximately 50 % of all cases of calf deep vein thrombosis are self-limiting and resolve within the first 72 hours after surgery , 40 % remain in calf , and 10 % progress to proximal vein thrombosis , which greatly increases the risk of pulmonary embolism. The risk of DEEP VEIN THROMBOSIS is different depending on the type of surgery performed. Surgicalinterventions such as laparoscopic cholecystectomy , appendectomy , transurethral prostatectomy or mastectomy have the lower risk of developing DVT. Various previous studies revealed general surgery had a 15 to 30 % risk of developing DVT in the absence of anticoagulant or mechanical prophylaxis.

In our study totally 47 patients developed DEEP VEIN THROMBOSIS post operatively among the 224 patients underwent surgery without thromboembolism prophylaxis with prevalence of 21 percentage . Various risk factors analysed and needed for pre-operative thromboembolism prophylaxis documented below.

In our review of literature showed high risk group patients with risk factors of previous history of cancer ,chronic medical illness, previous history of varicose vein ,obesity , smoking , prolonged duration of surgery , chronic immobilization days were developed DEEP VEIN THROMBOSIS with 39 percentage . Moderate risk group patients with risk factors of age , smoking , obesity , cancer patients , duration of surgery and immobilization days were developed DEEP VEIN THROMBOSIS with 21 percentage. Low risk group patients were developed DEEP VEIN THROMBOSIS with 8 percentage .

According to our study old age more than 70 year patients underwent surgery more prone to develop DVT after surgery . Obesity and smoking major predisposing factors for DVT post operatively. Morbid obese patients and chronic smoking history patients significantly associated with the development of DVT postoperatively. In our study cancer patients , chronic medical illness patients , post operatively

prolonged immobilization patients were significantly associated with development of DEEP VEIN THROMBOSIS post operatively.Hence these patients , moderate and high risk group patients definitely require the preoperative thromboembolism prophylaxis in order to prevention of post operative DEEP VEIN THROMBOSIS.

The ACCP guidelines highlight 2 approaches to decision making withregard to thromboembolism prophylaxis. The first is to consider the risk of DVT in each patients based on their individual predisposing factors and the risk associated with their current illness or procedure , with the use of prophylaxis based on the composite risk estimate. A second approach , favoured by the ACCP , is to implement group specific prophylaxis for all patients who belong to one of several major target groups (eg , type of surgery , age , and presence of additional risk factors).

A simplified approach to estimating an individual patient's risk , based on the presence of both predisposing and exposing (in hospital) risk factors , might provide a suitable way to ascertain a patient's risk of DEEP VEIN THROMBOSIS. Such a system would allocate the patients with a reasonable measure of low , moderate , or high risk groups so that prophylaxis could be tailored to the element of risk that the patient is

facing. Highly safe and effective types of DVT prophylaxis include low molecular weight heparin ,warfarin , intermittent pneumatic compression and low dose unfractionated heparin. If patients contraindication for DEEP VEIN THEOMBOSIS prophylaxis (active bleeding , history of heparin induced thrombocytopenia , a platelet count less than 1,00,000 or platelet inhibitors , or abnormal creatinine clearance rate) intermittent pneumatic compression should be considered.

Various studies showed use of pre-operative thromboembolism prophylaxis significantly reduced mortality and morbidity of post-operative deep vein thrombosis.According to our study showed any patient underwent the surgery needed for evaluation of individual risk factors and usage of pre-operative thromboembolism prophylaxis in order to prevent the post operative DEEP VEIN THROMBOSIS.

CONCLUSION

Pulmonary embolism is the most common preventable cause of death among hospital patients. DVT is often overlooked as major public health problem and viewed as complication of hospitalization for another illness rather than a specific diseases entity.

The prevalence and prevention of DEEP VEIN THROMBOSIS has historically been poorly recognized and lacked clinical status. This is changing with greater public awareness , clinician and nursing education , in high profile publication of risk assessments , and national guidelines and preventive measures. Evidence is now emerging that DVT risks extend 6 to 12 weeks post surgery in the presence of certain risk factors. This evidence may impact further on recommended duration of prophylaxis for targeted group in future DVT guidelines.

The potential public health benefit of DVT is substantial:data from randomized trials involving general surgical patients suggest that adequate prophylaxis in high risk patients can prevent DVT in 1 of 10 patients and save the life of 1 of 200 patients. In our study also showed moderate and high risk group patients underwent surgery definitely needed thromboprophylaxis pre operatively and 6 to 12 weeks of postoperative period for prevention of postoperative DVT.

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PROFORMA

NAME : UNIT NO :

I.P.NO:

AGE /SEX:

OCCUPATION : DATE OF ADMISSION :

ADDRESS: DATE OF DISCHARGE:

CONTACT NO:

DIAGNOSIS:

PROCEDURE DONE :

PARAMETERS

- AGE
- OBESITY
- SMOKING
- CANCER PATIENTS
- PREVIOUS HISTORY OF DEEP VEIN THROMBOSIS
- FAMILY HISTORY OF THROMBOSIS

- PREVIOUS HISTORY OF VARICOSE VEIN
- CHRONIC MEDICAL ILLNESS
- HISTORY OF CONNECTIVE TISSUE DISORDER
- CHEMO/ RADIOTHERAPHY RECEIVED PATIENTS
- SPINAL/ GENERAL ANAESTHESIA
- DURATION OF SURGERY
- IMMOBILIZATION DAYS

TOTAL RISK SCORE :

POST OPERATIVE FOLLOW UP :

OUTCOME:

ஒப்புதல் படிவம்

பெயர் –

பாலினம் –

முகவரி – வயது –

அரசு கோவை மருத்துவக் கல்லூரியில் பொது அறுவை சிகிச்சை துறையில், பட்டமேற்படிப்பு பயிலும் மாணவர் வே. ஜோதி நாராயணசாமி அவர்கள் மேற்கொள்ளும் **ASSESSMENT OF RISK FACTORS FAVOURING DEEP VEIN THROMBOSIS IN PATIENTS UNDERGOING SURGERY** என்ற சோதனையின் செய்முறை மற்றும் அனைத்து விபரங்களையும் கேட்டுக் கொண்டதுடன், எனது அனைத்து சந்தேகங்களையும் தெளிவுபடுத்திக் கொண்டேன் என்பதை தெரிவித்துக் கொள்கிறேன்.

நான் இந்த ஆய்வில் முழு சம்மதத்துடனும், சுய சிந்தனையுடனும் கலந்து கொள்ள சம்மதிக்கிறேன்.

இந்த ஆய்வில் என்னுடைய அனைத்து விபரங்களும் பாதுகாக்கப்படுவதுடன் இதன் முடிவுகள் ஆய்விதழில் வெளியிடப்படுவதில் எனக்கு எந்த ஆட்சேபனையும் இல்லை என்பதை தெரிவித்துக் கொள்கிறேன். எந்த நேரத்திலும் இந்த ஆய்வில் இருந்து விலகிக் கொள்ள எனக்கு உரிமை உண்டு என்பதையும் அறிவேன்.

இடம் –

தேதி –

கையொப்பம், ரேகை

MASTER CHART

S.No	Name	Gender	Age	BMI	Smoking	Cancer	DVT	Thrombosis	Varicose vein	Medical illness	CTD	Chemo or RT	Spinal	General	Duration of surgery	Immobilization days	Risk score
1	Subbiah	M	0	1	2	0	0	0	0	0	0	0	1	0	1	1	6
2	Velmurugan	M	1	1	2	0	0	0	0	2	0	0	1	0	1	1	9
3	Saraswathi	F	0	1	0	0	0	0	0	0	0	0	1	0	1	1	4
4	Arumugam	M	0	1	2	0	0	0	0	0	0	0	1	0	1	1	6
5	Subramani	M	1	2	2	0	0	0	0	2	0	0	0	2	2	2	13
6	Aadhi	M	0	1	2	0	0	0	0	0	0	0	1	0	1	1	6
7	Kannagi	F	0	1	0	0	0	0	0	0	0	0	0	2	2	1	6
8	Manimozhi	F	0	1	0	0	0	0	0	0	0	0	1	0	1	1	4
9	Velan	M	1	1	0	0	0	0	0	0	0	0	1	0	1	1	5
10	Kandhasamy	M	2	1	2	0	0	0	0	2	0	0	0	2	2	2	13
11	Palani	M	1	1	2	0	0	0	2	0	0	0	1	0	1	1	9
12	Kathir	M	1	0	2	0	0	0	0	0	0	0	0	2	2	1	10
13	Mani	M	1	0	2	0	0	0	0	0	0	0	1	0	1	1	6
14	Karthi	M	0	0	0	0	0	0	0	0	0	0	1	0	0	1	2
15	Balan	M	1	0	2	0	0	0	0	0	0	2	0	2	2	2	11
16	Surya	M	1	0	2	2	0	0	0	0	0	2	0	2	2	2	13
17	Kumaran	M	1	0	2	0	0	0	0	2	0	0	0	2	2	2	11
18	Baiamurugan	M	2	0	2	0	0	0	0	2	0	0	1	0	1	1	9
19	Muniyandi	M	1	3	2	0	0	0	0	2	0	0	1	0	1	1	11
20	Alagammal	F	1	0	0	0	0	0	0	0	0	0	0	2	2	1	6
21	Kabilan	M	0	2	2	0	0	0	0	0	0	0	1	0	1	1	7
22	Ayusha	F	0	0	0	0	0	0	0	0	0	0	0	2	2	1	5
23	Kathiravan	M	1	2	0	0	0	0	0	0	0	0	1	0	1	1	6
24	Rhagavan	M	1	0	2	0	0	0	0	0	0	0	1	0	1	1	6
25	Vellaisamy	M	2	2	2	2	0	0	0	0	0	0	0	2	2	3	15

26	Saminathan	M	0	0	0	0	0	0	0	0	0	0	1	0	0	1	2
27	Guru	M	2	3	2	0	0	0	0	0	0	0	0	2	2	1	12
28	Selvam	M	2	0	2	0	0	0	0	0	0	0	1		1	1	7
29	Nalini	F	2	0	0	0	0	0	0	0	0	0	0	2	1	1	6
30	Subban	M	2	0	2	2	0	0	0	0	0	0	0	2	2	3	13
31	Mukundan	M	2	1	2	2	0	0	0	0	0	0	0	2	2	2	13
32	Kannan	M	3	0	2	2	0	0	0	0	0	0	0	2	3	2	14
33	Velan	M	2	1	2	0	0	0	2	2	0	0	1	0	1	2	13
34	Arasan	M	2	0	2	0	2	0	0	0	0	0	0	2	2	1	11
35	Sundari	F	2	1	0	0	0	2	0	0	0	0	1	0	1	1	8
36	Dhivyalakshmi	F	3	0	0	0	0	0	0	0	2	0	0	2	2	2	11
37	Senthil	M	0	0	2	0	0	0	0	0	0	0	0	2	2	2	8
38	Kala	F	1	1	0	0	0	0	0	0	0	0	1	0	1	1	5
39	Arun	M	2	3	2	0	0	0	0	0	0	0	1	0	1	1	10
40	Kumar	M	1	1	0	0	0	0	0	0	0	0	0	2	2	1	7
41	lakshmi	F	1	0	0	0	0	0	0	0	0	0	0	2	2	1	6
42	Vinoth	M	3	0	2	0	0	0	0	0	0	0	0	2	2	3	12
43	Nallathambi	M	1	0	0	0	0	0	0	0	0	0	0	2	2	1	6
44	Durai	M	0	0	2	0	0	0	0	0	0	0	0	2	2	1	7
45	Kuppan	M	2	0	0	0	0	0	0	0	0	0	1	0	1	1	5
46	Manoharan	M	1	0	2	0	0	0	0	0	0	0	1	0	1	1	6
47	Priya	F	1	1	0	0	0	0	0	0	0	0	0	2	2	1	7
48	Dhandapani	M	2	0	2	2	0	0	0	0	0	0	0	2	3	2	13
49	Dharshini	F	1	0	0	0	0	0	0	0	0	0	1	0	1	1	4
50	Chinnasamy	M	2	0	2	0	0	0	2	0	0	0	0	2	2	1	11
51	Lavanya	F	0	0	0	0	0	0	0	0	0	0	1	0	0	1	2
52	`Andavar`	M	3	0	2	0	0	0	0	2	0	0	0	2	2	3	14

53	Chandran	M	2	0	2	2	0	0	0	0	0	0	0	2	3	2	13
54	Muthu	M	2	0	2	2	0	0	0	2	0	0	0	2	3	2	15
55	Rajan	M	1	1	2	0	0	0	0	0	0	0	1	0	1	1	7
56	Jothimuruga	M	1	0	2	0	0	0	0	2	0	0	0	2	2	2	11
57	Devi	F	1	1	0	0	0	0	0	0	0	0	1	0	1	1	5
58	Ramu	M	0	1	2	0	0	0	0	0	0	0	1	0	1	1	6
59	kesavan	M	1	0	0	0	0	0	0	0	0	0	0	2	2	1	6
60	Shagundala	F	2	2	0	0	0	0	0	2	0	0	0	2	2	2	12
61	Babu	M	0	0	2	0	0	0	0	0	0	0	0	2	2	2	8
62	Duraimurugan	M	1	0	2	2	0	0	0	0	0	2	0	2	3	3	15
63	Duraisamy	M	3	0	2	0	0	0	2	2	0	0	0	2	2	2	15
64	Vimal	M	2	3	2	0	0	0	0	2	0	0	1	0	1	1	12
65	velathal	F	1	0	0	0	0	0	2	0	0	0	0	2	2	1	8
66	Kamal	M	1	0	0	0	0	0	0	0	0	0	0	2	2	1	6
67	Kannadhasan	M	1	0	0	0	0	0	2	0	0	0	1	0	1	1	6
68	Parimala	F	0	0	0	2	0	0	0	0	0	0	0	2	2	1	7
69	Venu	M	0	0	0	0	0	0	0	0	0	0	0	2	2	1	5
70	Gopal	M	1	0	2	0	0	2	0	0	0	0	1	0	1	1	8
71	Malar devi	F	0	0	0	0	0	0	0	0	0	0	0	2	0	1	3
72	Eswari	F	1	1	0	0	0	0	0	0	0	0	1	0	1	1	5
73	Arjun	M	0	0	2	0	0	0	0	0	0	0	1	0	1	1	5
74	Ankit	M	1	0	2	2	0	0	0	2	0	0	1	0	0	1	9
75	Gonath	M	0	0	2	2	0	0	0	0	0	0	0	2	2	2	10
76	Angathal	F	1	3	0	0	0	0	0	2	0	0	1	0	1	1	9
77	Amir	M	1	0	2	2	0	0	0	2	0	0	0	2	3	2	14
78	Chokkan	M	2	1	2	0	0	0	2	0	0	0	0	2	2	2	13
79	Saran	M	2	0	2	0	0	0	2	0	0	0	0	2	2	2	12

80	Raj	M	1	3	2	0	2	0	0	0	0	0	1	0	1	1	11
81	Dhina	M	1	3	2	0		0	2	0	0	0	1	0	1	3	13
82	Raman	M	1	0	2	0	0	0	0	0	0	0	1	0	1	1	6
83	Selvi	F	0	0	0	0	0	0	0	0	0	0	0	2	1	1	4
84	Moorthi	M	2	0	0	0	0	0	0	0	0	0	0	2	2	1	7
85	Karthikeyan	M	1	0	2	2	0	0	0	2	0	0	0	2	2	3	14
86	Perumal	M	3	1	2	0	0	0	0	0	0	0	0	2	2	1	11
87	Nandu	M	1	1	2	0	0	0	0	0	0	0	1	0	0	1	6
88	Kalan	M	0	0	0	0	0	0	0	0	0	0	0	2	2	1	5
89	Balamurugan	M	1	0	2	0	0	2	0	0	0	0	1	0	1	1	8
90	Ramaya	F	3	0	2	2	0	0	0	2	0	0	1	0	2	2	14
91	Leela	F	1	2	0	0	2	0	0	0	0	0	0	2	2	2	11
92	Mariyammal	F	2	0	0	0	0	0	0	0	0	0	1	0	1	1	5
93	Sekar	M	1	0	0	0	0	0	0	0	0	0	0	2	2	2	7
94	Vijay	M	0	2	2	0	2	0	0	0	0	0	0	2	3	2	13
95	Nagasamy	M	3	0	2	0	0	0	0	2	0	0	1	0	1	1	10
96	Praveen	M	2	0	2	0	0	0	0	0	0	0	1	0	0	1	6
97	Gayathri	F	2	0	0	0	0	0	0	0	0	0	1	0	1	1	5
98	Velu	M	1	3	2	0	0	0	0	2	0	0	0	2	2	3	15
99	Niranjan	M	1	0	2	2	0	0	0	0	0	2	0	2	2	2	13
100	Ashwin	M	0	0	2	0	0	0	2	0	0	0	1	0	1	1	7
101	Velusamy	M	3	0	2	0	0	0	0	2	0	0	1	0	2	3	13
102	Palanivelu	M	2	0	2	2	0	0	0	2	0	0	0	2	3	3	16
103	Malar	F	0	0	0	0	0	0	0	0	0	0	1	0	1	1	3
104	Vinay	M	1	1	2	0	2	0	0	0	0	0	0	2	2	2	12
105	Arul	M	1	0	0	0	0	0	0	0	0	0	0	2	1	1	5
106	Velappan	M	3	0	2	0	0	0	0	2	0	0	0	2	2	2	13

107	Sheela	F	3	0	0	2	0	0	0	0	0	0	1	0	1	1	8
108	Rangasamy	M	1	0	0	0	0	0	2	0	0	0	1	0	1	1	6
109	Elango	M	1	3	2	0	0	0	2	0	0	0	0	2	2	3	15
110	Devaki	F	0	0	0	0	2	0	0	0	0	0	1	0	1	1	5
111	Sundhar	M	1	2	0	0	0	0	0	0	0	0	1	0	1	1	6
112	Ayyammal	F	0	0	0	0	0	0	0	0	0	0	0	2	2	2	6
113	Michael	M	0	3	2	0	0	0	2	2	0	0	1	0	2	2	14
114	Saral	F	0	1	0	0	0	0	0	0	0	0	0	2	2	1	6
115	Surumoorthi	M	0	2	2	0	0	0	2	0	0	0	1	0	1	1	9
116	Kabilnath	M	1	1	0	0	0	0	0	0	0	0	1	0	1	1	5
117	Kalyani	F	2	0	0	0	0	2	0	0	0	0	0	2	2	1	9
118	Janaki	F	2	1	0	0	0	0	0	0	0	0	1	0	1	1	6
119	Mahalakshmi	F	3	0	0	2	0	0	0	0	0	2	0	2	2	3	14
120	Raguvaran	M	1	0	2	0	0	0	0	0	2	0	1	0	1	1	8
121	Muniyammal	F	3	0	0	0	0	0	0	0	0	0	1	0	0	1	5
122	Appasamy	M	1	0	2	0	0	0	0	0	0	0	0	2	3	3	11
123	Palaniyammal	F	2	0	0	0	0	0	0	0	0	0	0	2	1	1	6
124	Ramanan	M	1	0	0	0	0	0	0	0	0	0	0	2	2	2	7
125	Lalitha	F	3	0	0	0	0	0	0	0	0	0	1	0	1	1	6
126	vellaiyappan	M	3	0	2	0	0	0	2	0	0	0	0	2	3	3	15
127	Chenniyappan	M	2	2	2	2	0	0	0	2	0	0	0	2	2	2	16
128	Manivasakan	M	1	3	2	0	0	0	2	0	0	0	0	2	2	2	14
129	Krishna	M	1	2	2	0	0	0	0	0	0	0	1	0	1	1	8
130	Monisha	F	0	0	0	0	0	0	0	0	0	0	1	0	0	1	2
131	Neelakandan	M	1	0	2	0	0	0	0	0	0	0	1	0	1	1	6
132	Rajarajan	M	3	0	2	2	0	0	2	0	0	0	0	2	3	2	16
133	Jegan	M	1	3	2	0	0	0	2	0	0	0	1	0	1	1	11

134	Parvathi	F	3	0	0	0	0	0	0	0	0	0	1	0	1	1	6
135	Gugan	M	1	3	2	0	0	0	0	0	2	0	0	2	2	1	13
136	Mohan	M	2	0	2	2	0	0	0	0	0	2	0	2	3	3	16
137	Surveshvaran	M	1	0	2	0	2	0	0	0	0	0	0	2	2	2	11
138	Chandra	F	1	0	0	0	0	0	0	0	0	0	1	0	1	1	4
139	Akilan	M	0	0	2	0	0	0	0	0	0	0	0	2	1	1	6
140	Kathiresh	M	1	2	2	0	0	0	0	0	2	0	0	2	2	1	12
141	Palanal	F	2	0	0	0	0	0	0	0	0	0	1	0	1	1	5
142	Shanmugam	M	2	3	2	0	0	0	0	2	0	0	0	2	2	2	` 15
143	Abishek	M	1	3	2	0	0	0	2	0	0	0	1	0	2	2	13
144	Valliyammal	F	2	1	0	0	2	0	0	0	0	0	0	2	2	1	10
145	Noorjahan	F	2	1	0	0	0	0	0	0	0	0	1	0	1	1	6
146	Narmatha	F	0	2	0	0	0	2	0	0	0	0	1	0	1	1	7
147	Suresh	M	1	0	0	0	0	0	0	0	0	0	1	0	1	1	4
148	Ravi	M	2	3	2	0	0	0	0	2	0	0	0	2	2	3	16
149	Gunalan	M	1	2	2	0	0	0	0	0	2	0	1	0	1	1	10
150	Bhuvana	F	3	0	0	0	0	0	0	0	0	0	1	0	1	1	6
151	Maheshwaran	M	0	0	2	0	0	0	0	0		0	0	2	2	1	7
152	Kali	M	1	0	0	0	0	0	0	0	0	0	1	0	1	1	4
153	Shilba	F	0	0	0	0	0	0	0	0	0	0	0	2	2	1	5
154	Jeyakumar	M	2	3	2	0	0	0	0	0	0	0	0	2	2	2	13
155	Murugavel	M	2	3	2	0	0	0	0	0	0	0	0	2	2	1	12
156	Kuppusamy	M	1	0	2	0	0	0	2	0	0	0	1	0	1	1	8
157	Bose	M	2	0	2	0	0	0	0	2	0	0	1	0	1	1	9
158	Ilakkiyan	M	0	0	2	0	0	0	0	0	0	0	1	0	1	1	5
159	Selvaraj	M	1	3	2	0	0	0	2	2	0	0	0	2	2	2	16
160	Venkatachalam	M	2	0	2	0	0	0	2	0	0	0	0	2	3	3	14

161	Faritha	F	1	0	0	0	0	0	0	0	0	0	0	2	1	1	5
162	Shankar	M	3	0	2	2	0	0	0	2	0	0	0	2	2	3	16
163	Giri	M	3	0	2	2	0	0	0	2	2	0	0	2	3	3	19
164	Fathima	F	1	0	0	0	0	0	0	0	0	0	1	0	1	1	4
165	Karunaiyappan	M	2	3	2	0	0	0	2	2	0	0	0	2	2	2	17
166	Nikil	M	2	0	2	0	0	0	0	0	0	0	0	2	2	1	9
167	Moolan	M	1	0	2	2	0	0	0	2	0	0	1	0	1	1	10
168	Aadhi	M	1	3	2	0	0	0	2	0	0	0	0	2	2	2	14
169	Poomika	F	2	0	0	0	0	0	0	0	0	0	0	2	1	1	6
170	Saravanan	M	1	0	0	0	0	0	0	0	0	0	1	0	0	1	3
171	Bhuvaneshwari	F	2	0	0	2	0	0	0	0	0	0	0	2	3	3	12
172	Kumaravel	M	0	0	0	0	0	0	0	0	0	0	1	0	1	1	3
173	Shreedevi	F	1	0	0	0	0	0	0	0	0	0	0	2	1	1	5
174	Pandi	M	3	0	2	2	0	0	0	2	0	0	0	2	2	2	15
175	Vignesh	M	2	1	2	0	2	0	0	0	0	0	0	2	2	2	13
176	Santhosh	M	2	1	2	0	0	0	0	0	2	0	1	0	1	1	10
177	Sanjay	M	1	0	2	0	0	0	0	0	0	0	1	0	1	1	6
178	Lakshmanan	M	2	0	0	0	0	0	0	0	0	0	0	2	1	1	6
179	Prakash	M	1	1	2	0	0	0	0	0	0	0	0	2	2	1	9
180	Neela	F	1	1	0	0	0	0	0	0	0	0	1	0	1	1	5
181	Ramamoorthi	M	2	3	0	0	0	2	0	0	0	0	0	2	2	2	13
182	Poorvika	F	0	0	0	0	0	2	0	0	0	0	0	2	1	1	6
183	Shreekala	F	1	0	0	0	0	0	0	2	0	0	1	0	1	1	6
184	Ramasamy	M	3	0	2	2	0	0	0	0	0	0	0	2	2	2	13
185	Senthinathan	M	2	3	2	0	0	0	0	2	0	0	1	0	1	1	12
186	Kulanthaiyappan	M	2	2	2	0	0	0	0	2	0	0	1	0	1	1	11
187	Ragav	M	2	0	0	0	0	0	0	0	0	0	1	0	1	1	5

188	Nayaki	F	0	0	0	0	2	0	0	0	0	0	1	0	1	1	5
189	Thiyagarajan	M	2	1	2	2	0	0	0	0	0	2	0	2	2	2	15
190	Alwin	M	2	0	0	0	0	0	0	0	0	0	1	0	0	1	4
191	Ayyanar	M	3	1	2	0	0	0	2	2	0	0	0	2	2	3	17
192	Revathi	F	2	0	0	0	0	0	0	0	2	0	0	2	2	2	10
193	Renukadevi	F	3	0	0	0	0	0	0	0	0	0	1	0	1	1	6
194	Prasath	M	1	3	2	0	0	0	2	2	0	0	0	2	2	2	16
195	Rangal	F	2	0	0	0	0	0	0	2	0	0	0	2	2	2	10
196	Mangai	F	1	0	0	0	0	0	0	0	0	0	1	0	1	1	4
197	Sathasivam	M	3	0	2	0	0	0	0	0	0	0	1	0	1	1	8
198	Sivaraj	M	2	0	2	2	0	0	0	0	0	0	0	2	3	3	14
199	Selvan	M	0	3	2	0	0	0	0	0	0	0	1	0	2	1	9
200	Murali	M	1	0	2	0	0	0	0	0	0	0	1	0	1	1	6
201	Srinivasan	M	3	0	2	2	0	0	0	0	0	0	0	2	3	3	15
202	Dhenna	M	0	0	0	0	0	2	0	0	0	0	1	0	1	1	5
203	Yamini	F	0	0	0	0	0	0	2	0	0	0	1	0	0	1	4
204	Muneeshwaran	M	0	0	0	0	0	0	2	0	0	0	0	2	1	1	6
205	Valarmathi	F	0	0	0	0	2	0	0	0	0	0	0	2	2	1	7
206	Thangavel	M	3	0	2	2	0	0	0	2	0	2	0	2	2	3	18
207	Jeyaprakash	M	2	2	2	2	0	0	0	0	0	0	0	2	2	2	14
208	Ayyasamy	M	3	1	2	0	0	0	0	0	0	0	0	2	1	1	10
209	Senniyammal	F	3	0	0	0	0	0	0	0	2	0	0	2	1	1	9
210	Vaisali	F	2	0	0	0	0	0	0	0	0	0	1	0	1	1	5
211	Pratheep	M	1	2	2	0	0	0	2	2	0	0	0	2	1	1	13
212	Dhinesh	M	1	1	2	0	0	0	0	0	0	0	1	0	0	1	6
213	Dhinakar	M	2	0	2	0	0	0	0	0	0	0	0	2	1	1	8
214	Jeyabalan	M	2	0	2	2	0	0	0	2	0	0	0	2	2	2	14

215	Manikam	M	1	3	2	0	0	0	2	0	0	0	0	2	2	3	15
216	Guruvan	M	0	0	2	0	0	0	0	0	0	0	0	2	2	1	7
217	Tamilan	M	0	0	2	0	0	0	0	0	0	0	0	2	2	2	8
218	Arunraj	M	1	3	2	0	0	0	0	2	0	0	0	2	2	2	14
219	Devikala	F	2	1	0	0	0	0	0	0	0	0	0	2	2	1	8
220	Valli	F	1	0	0	0	0	0	0	0	0	0	1	0	0	1	3
221	Sudhakar	M	2	0	0	0	0	0	0	0	0	0	1	0	1	1	5
222	Suryadevi	F	1	0	0	0	0	0	0	0	0	0	0	2	1	1	5
223	Velan	M	1	1	1	0	0	0	0	0	0	0	1	0	1	1	6
224	Sukumar	M	2	0	0	0	0	0	0	0	0	0	1	0	1	1	5