“CORRELATION OF SERUM HOMOCYSTEINE IN PATIENTS WITH VENOUS THROMBOEMBOLISM DURING COVID PANDEMIC IN TVMCH”

DISSERTATION SUBMITTED

TO

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

In partial fulfilment of the requirements for the degree of

M.D. BRANCH – I (GENERAL MEDICINE)

Register No:200120104021

DEPARTMENT OF GENERAL MEDICINE

TIRUNELVELI MEDICAL COLLEGE HOSPITAL

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MAY-2023

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I, **Dr. SHINY MISPA MERLIN P,** solemnly declare that the dissertation titled **"CORRELATION OF SERUM HOMOCYSTEINE IN PATIENTS WITH VENOUS THROMBOEMBOLISM DURING COVID PANDEMIC IN TVMCH"** is a bonafide work done by me at Tirunelveli Government Medical College and Hospital from 2021 to 2022 under the guidance and supervision of my unit chief, **Dr. PRINCE PRABHAKARAN A, M.D.,** Professor of General Medicine. This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University towards the partial fulfilment of the requirement of M.D. General Medicine degree examination.

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Last but not the least I would like to thank my patients with gratitude for their cooperation during study and for teaching us the art of medicine and inciting us to learn more.

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PROTOCOL TITLE: CORRELATION OF SERUM HOMOCYSTEINE IN PATINTS WITH VENOUS THROMBOEMBOLISM DURING COVID PANDEMIC IN TVMCH
PRINCIPAL INVESTIGATOR: DR.P. SHINY MISPA MERLIN MBBS
DESIGNATION OF PRINCIPAL INVESTIGATOR: RESIDENT
DEPARTMENT & INSTITUTION: GENERAL MEDICINE, TIRUNELVELI MEDICAL COLLEGE, TIRUNELVELI

Dear DR.P. SHINY MISPA MERLIN MBBS, Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during The IEC meeting held on ***06.07.2021.***

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1. TIREC Application Form ☐
2. Study Protocol ☐
3. Department Research Committee Approval ☐
4. Patient Information Document and Consent Form in English and Vernacular Language ☐
5. Investigator's Brochure ☐
6. Proposed Methods for Patient Accrual Proposed ☐
7. Curriculum Vitae of The Principal Investigator ☐
8. Insurance /Compensation Policy ☐
9. Investigator's Agreement with Sponsor ☐
10. Investigator's Undertaking ☐
11. DCGI/DGFT approval ☐
12. Clinical Trial Agreement (CTA) ☐
13. Memorandum of Understanding (MOU)/ Material Transfer Agreement (MTA) ☐
14. Clinical Trials Registry-India (CTRI) Registration ☐

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1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
3. A written request should be submitted 3weeks before for renewal / extension of the validity
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This is to certify that this dissertation work entitled **"CORRELATION OF SERUM HOMOCYSTEINE IN PATIENTS WITH VENOUS THROMBOEMBOLISM DURING COVID PANDEMIC IN TVMCH"** of the candidate **Dr. SHINY MISPA MERLIN P** with registration Number **200120104021** for the award of **M.D.** Degree in the branch of **GENERAL MEDICINE (I).** I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion page and result shows **0 percentage** of plagiarism in the dissertation.

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INTRODUCTION Venous Thromboembolism is the most common preventable mortality among hospitalized patients. VTE includes both Deep Venous Thrombosis and Pulmonary Embolism. In addition to Sr D dimer, Serum Homocysteine also plays an important role in VTE. There are some research studies which showed positive relationship between Serum homocysteine and VTE.

Homocysteine interacts with lysyl residues of collagen interfering with collagen cross linking, thereby it produces endothelial dysfunction. It also alters the anticoagulant properties of endothelial cells to procoagulant state. So, study of serum homocysteine in VTE plays an important role. IN 2020, COVID 19 causes a global pandemic. The most common clinical feature is a life threatening Acute Respiratory Syndrome requiring prolonged mechanical ventilation and causing a high fatality rate. This viral illness also causes extensive DVT and Pulmonary embolism, even when patients received standard pharmacological prophylaxis as soon as they are hospitalized. At autopsy, about one – fourth of patients have macrovascular and microvascular Pulmonary embolism. The contributing etiologies of this widespread thrombosis are cytokine storm, platelet activation and endothelial dysfunction and stasis. In this study, we are excluding COVID 19 patients with Venous Thromboembolism because this infection itself can cause hyperhomocysteinemia. In addition to survival after Pulmonary embolism, we now focus more attention on the quality of life after Pulmonary embolism. About half of Pulmonary embolism patients report persistent dyspnea, fatigue and reduced exercise capacity and about one-quarter have persistent right ventricular dysfunction on echocardiography following the diagnosis of PE. This constellation of findings is being recognized more frequently and it is called "POST PULMONARY EMBOLISM SYNDROME ". These patients may subsequently develop Chronic Thromboembolic pulmonary hypertension.

Cancer patients have a fourfold increased risk of VTE compared to general population. When unprovoked VTE occurs, there is an increased likelihood that occult cancer will subsequently be detected, especially during the first 6 months after the diagnosis of VTE. Age, prior provoked VTE, cigarette smoking nay help predict the presence of occult cancer in patients with a first unprovoked episode of VTE.

Overall, Pregnancy increases the risk of VTE fivefold, and this risk persists for atleast 12 weeks into the postpartum period.

Our knowledge of genetics in VTE is expanding rapidly. To date, atleast 17 genes have been demonstrated to harbor genetic variation associated with VTE risk. Common polymorphism account for atleast 5% of VTE heritability.

REVIEW OF LITERATURE Venous thromboembolism is a spectrum of disorder in which thrombus originating in a distant venous channel gets dislodged , becomes embolus and commonly occludes a distant venous system. This condition commonly includes

ABSTRACT

TITLE : **CORRELATION OF SERUM HOMOCYSTEINE IN PATIENTS WITH VENOUS THROMBOEMBOLISM DURING COVID PANDEMIC IN TVMCH**

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GUIDE : Dr.A. PRINCE PRABHAKARAN , Professor, Department of General Medicine, Government Tirunelveli Medical College Hospital.

Email ID: shiny031295@gmail.com, Mobile: 9487246435

BACKGROUND :

Venous Thromboembolism is the most common preventable mortality among hospitalized patients. VTE includes both **Deep Venous Thrombosis and Pulmonary Embolism**. In addition to Sr D dimer, Serum Homocysteine also plays an important role in VTE. There are some research studies which showed positive relationship between Serum homocysteine and VTE. Homocysteine interacts with lysyl residues of collagen interfering with collagen cross linking, thereby it produces endothelial dysfunction. It also alters the anticoagulant properties of endothelial cells to procoagulant state. So, study of serum homocysteine in VTE plays an important role. In 2020, COVID 19 causes a global pandemic. The most common clinical feature is a life threatening Acute Respiratory Syndrome requiring prolonged mechanical ventilation and causing a high fatality rate. This viral illness also causes extensive DVT and Pulmonary embolism, even when patients received standard pharmacological prophylaxis as soon as they are hospitalized. At autopsy, about one – fourth of patients have macrovascular and microvascular Pulmonary embolism. The contributing etiologies of this widespread thrombosis are cytokine storm, platelet activation and endothelial dysfunction

and stasis. In this study, we are excluding COVID 19 patients with Venous Thromboembolism because this infection itself can cause hyperhomocysteinemia. Our knowledge of genetics in VTE is expanding rapidly. To date, atleast 17 genes have been demonstrated to harbor genetic variation associated with VTE risk. Common polymorphism account for atleast 5% of VTE heritability.

AIMS AND OBJECTIVE :

- To determine the relationship between Serum homocysteine in patients with VTE.
- To study the risk factors and outcomes among VTE patients

MATERIALS AND METHODS :

This was a hospital based cross – sectional study done in Department of General Medicine and Department of Cardiology in Tirunelveli Medical College Hospital. Participants were recruited into the study based on inclusion and exclusion criteria. Along with routine blood investigation, ECG, CT Chest, CTPA, Sr. Homocysteine and Sr. D Dimer were taken.

RESULTS:

Mean age group of all patients in our study group was 52.4 and majority belongs to 45-60 years of age (66%). Males were more common than female in a ratio of 4.5 :1 among our study group. Most of the patients in our study group were labourers and most common symptomatology prevalent among our population is breathlessness (46 patients). Among the risk factors prevalent in our study group, the most common is smoking (42%). WELL's score predictive of venous thromboembolism was seen in only 30% of population. Hypoxemia was seen in 72% of patients and tachypnoea

being prevalent in 94% of our study population. Hypotension was less common and seen in 40% of our study group. Among laboratory parameters, D-dimer was elevated in 34%, elevated serum creatinine seen in 18% among our study population. Among the signs, tachypnoea was the most prevalent sign. ECG findings suggestive or predictive of pulmonary embolism was seen in 30% of our study group. CT chest /Chest X-ray suggestive of pulmonary embolism was seen in only 10% of our population. Echocardiography was abnormal in 70% of our population. 92% of patients in our study group had positive findings in CT pulmonary angiography. In our study, prevalence of elevated homocysteine level was 88% in VTE patients. Among our study population, DVT was seen in 36% and pulmonary embolism was seen in 88% of population.Mortality is higher accounting to 22% of population.

DISCUSSION :

Elevated homocysteine levels were more prevalent among patients with hypoxemia, tachypnoea and hypotension of VTE group. However, association of elevated homocysteine levels being more common in patients with tachypnoea in VTE showed statistical significance. Similarly, elevated homocysteine were more commonly seen in patients with elevated D-dimer, positive ECG and CTchest/chest x-ray findings. All patients who had echocardiographic findings suggestive of pulmonary embolism had elevated homocysteine levels with statistical significance. Also, around 93.18% of patients with CT pulmonary angiogram positive finding had elevated serum homocysteine levels with statistical significance. Pulmonary embolism patients had more commonly elevated homocysteine levels (93.18%) than DVT patients in our study group.(66.66%) And this association had statistical significance. Importantly, Pulmonary embolism patients had more commonly elevated homocysteine levels (93.18%) than DVT patients in our

study group.(66.66%) And this association had statistical significance. However, elevated serum homocysteine levels does not correlate with the outcome of VTE such as recovery/death. Hence, our study prevalence of serum homocysteine elevation in VTE was very high as 88% and its more commonly elevated in pulmonary embolism than deep vein thrombosis. Also elevated homocysteine levels significantly associated with clinical variable worsening like tachypnoea etc.., correlated well with other diagnostic modalities like ECHO and CT pulmonary angiogram. Hence Elevated homocysteine levels can be considered as a efficient diagnostic tool in diagnosing VTE , particularly pulmonary embolism. However, serum homocysteine levels does not correlate with outcome in our study group.

CONCLUSION :

Venous thromboembolism is often a diagnostic challenge being always complemented by imaging modalities for confirmation of diagnosis. Early diagnostic markers are still needed for avoiding delay in initiation of treatment. Homocysteine can be considered as one such marker which is often found to be elevated in VTE patients in literature. Prevalance of Elevated homocysteine levels among our study population of VTE patients was 88% (much higher than other studies in literature). As estimation of serum homocysteine is easy than many imaging procedures, serum homocysteine can be considered as early screening test for patients suspected to have VTE. Screening by such faster tests helps us in early diagnosis and initiation of treatment which helps immensely in prevention of morbidity and mortality by VTE.

KEYWORDS : Homocysteine, Pulmonary embolism, CT Pulmonary angiogram.

TABLE OF CONTENTS

INTRODUCTION

Venous Thromboembolism is the most common preventable mortality among hospitalized patients. VTE includes both **Deep Venous Thrombosis and Pulmonary Embolism**. In addition to Sr D dimer, Serum Homocysteine also plays an important role in VTE. There are some research studies which showed positive relationship between Serum homocysteine and VTE.

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REVIEW OF LITERATURE

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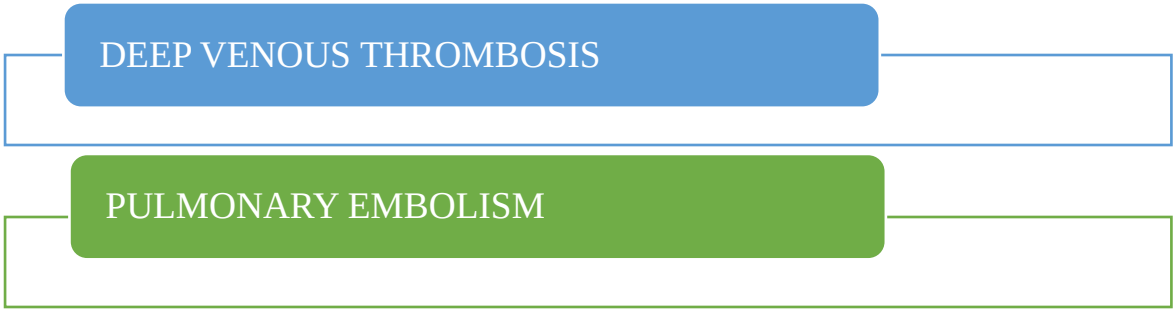


HISTORY OF VENOUS THROMBOEMBOLISM

In literature traced back, description of deep vein thrombosis and pulmonary embolism was seen ancient Indian texts written by Sushrutha Samhita around 600-900 BC approximately. After this , there was a long gap in which mentions of VTE has not been made in literature till 16th century. In his review article" The Chair and venous thrombosis" , Dexter stated that VTE was uncommon and rare during 10th century as because usuage of chair was not common during those period. Following ancient mentionings, recorded evidence of DVT was found to be in 16th century which states that King Henry VIII was affected by post traumatic DVT and treated with pearl dust during

1491 -1547 period in England. Following this , Queen of Scotland Mary was mentioned to develop post partum DVT in literature.

MAJOR CONTRIBUTIONS IN LITERATURE – TO DISCOVERY & UNDERSTANDING OF VTE

1628- William Harvey - Slow flow & Hypercoagulable lymph causes VTE

1793- Hunter - described inflammation in venous walls

1682-1711 - Giovanno Battista Morgagni - described patients with large clots in lungs died suddenly

1781- 1821 - Rene Laennec - described clinial features of PE - named Pulmonary apoplexy

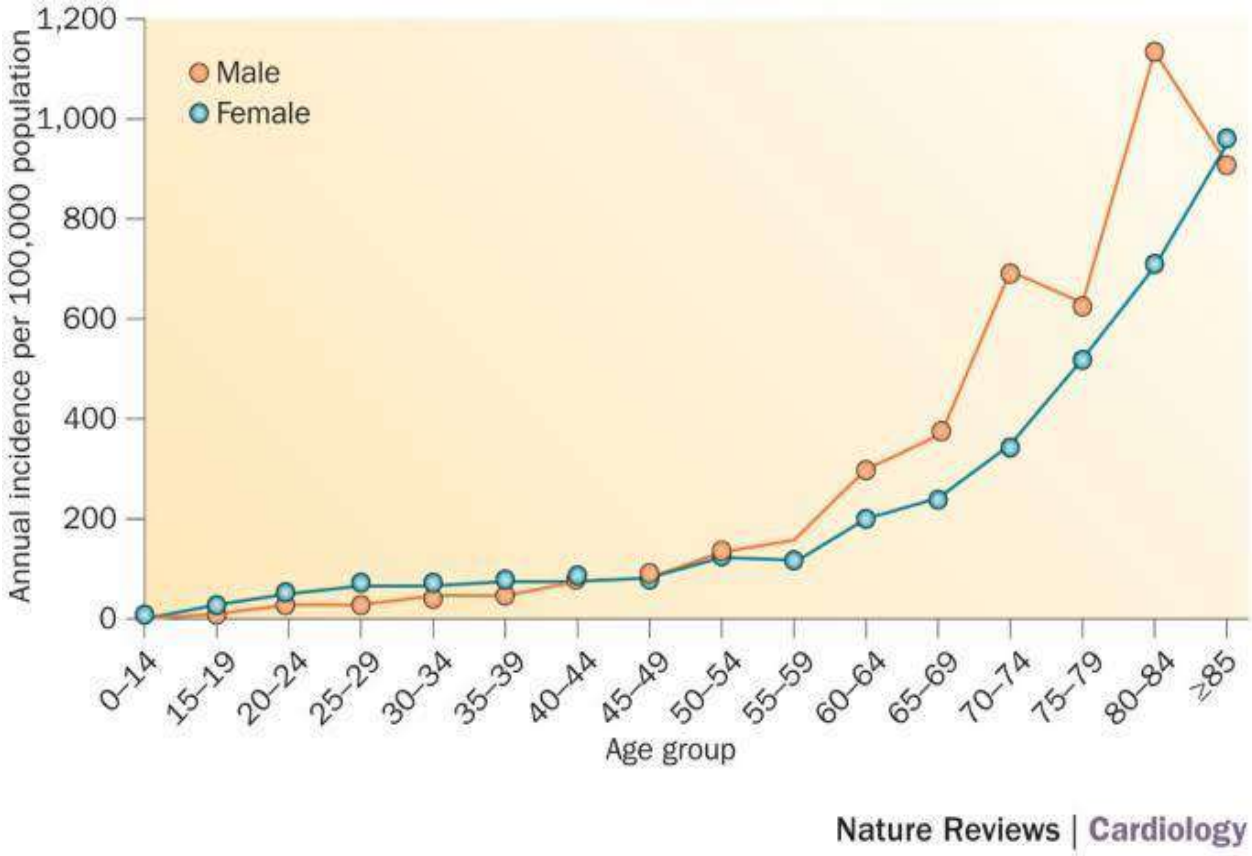
RUDOLF VIRCHOW & VENOUS THROMBOEMBOLISM

Rudolf Virchow was the first person to describe in detail about venous thromboembolism in his literature in 1850. In his medical reviews, he states that " Fragments of softening thrombus.. originated ..upstream of the lung namely veins & right heart " (1). He was the first person to mention that pulmonary thrombus was a embolic material from DVT of lower legs, thereby made a major contribution in understanding of pathophysiology of VTE during 1821-1902 itself.

EPIDEMIOLOGY OF VENOUS THROMBOEMBOLISM

Venous thromboembolism is considered as the third common cardiovascular disease after coronary artery disease and cerebrovascular disease. Estimated 3-year incidence of venous thromboembolism among hospitalised patients was 5.47

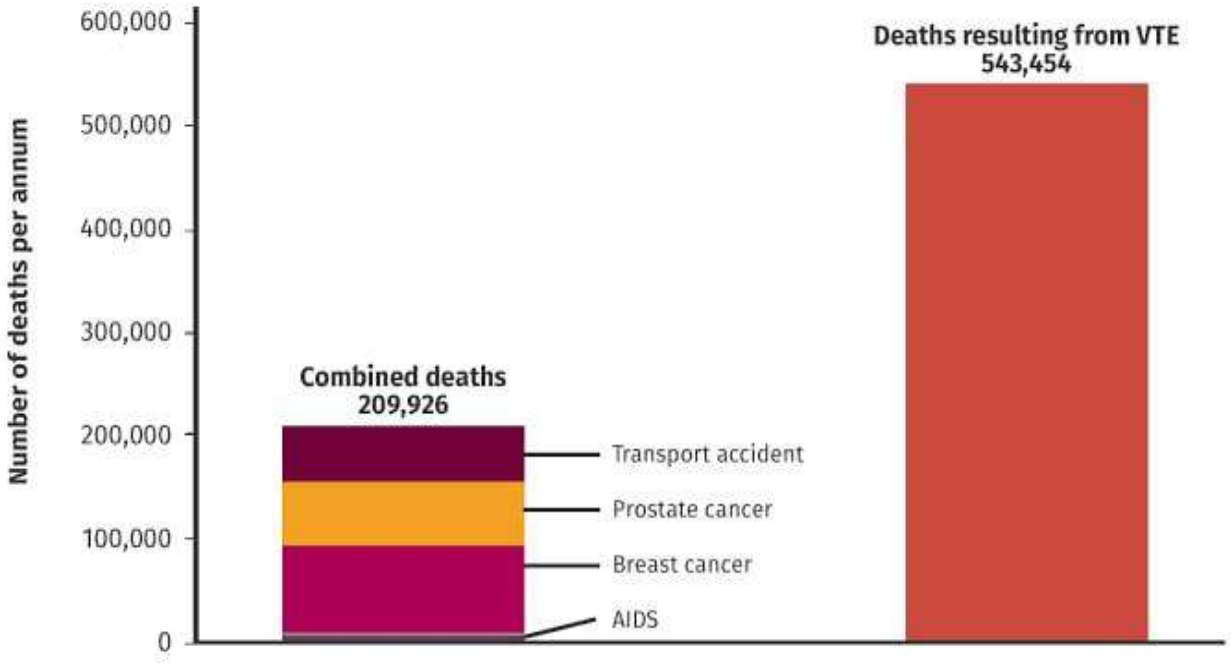
lakhs. (centre for Disease control & prevention report in 2007 –2009). A European union countries report states that annual incidence of DVT was more than 4.65 lakhs and PE was 2.95 lakhs cases which are all nonfatal VTE.



AGE vs VTE INCIDENCE - EPIDEMIOLOGY

Older individuals are more prone to develop VTE and risk of VTE is twice after 40 years of age when compared to younger ones. Pulmonary embolism component of VTE was more prevalent among males than in females. Racial preponderance of African Americans > whites > Asians were seen among incidences of VTE. VTE is a recurrent disease, and especially recurrence is common after first event with a percentage of 7%. Around 30-32% of untreated

DVT will develop pulmonary embolism eventually. Mortality rate among VTE patients are around 6% for cases with DVT diagnosis and 12% for Pulmonary embolism patients.



BURDEN OF VTE MORTALITY PER ANNUM AS COMPARED TO OTHER ETIOLOGY

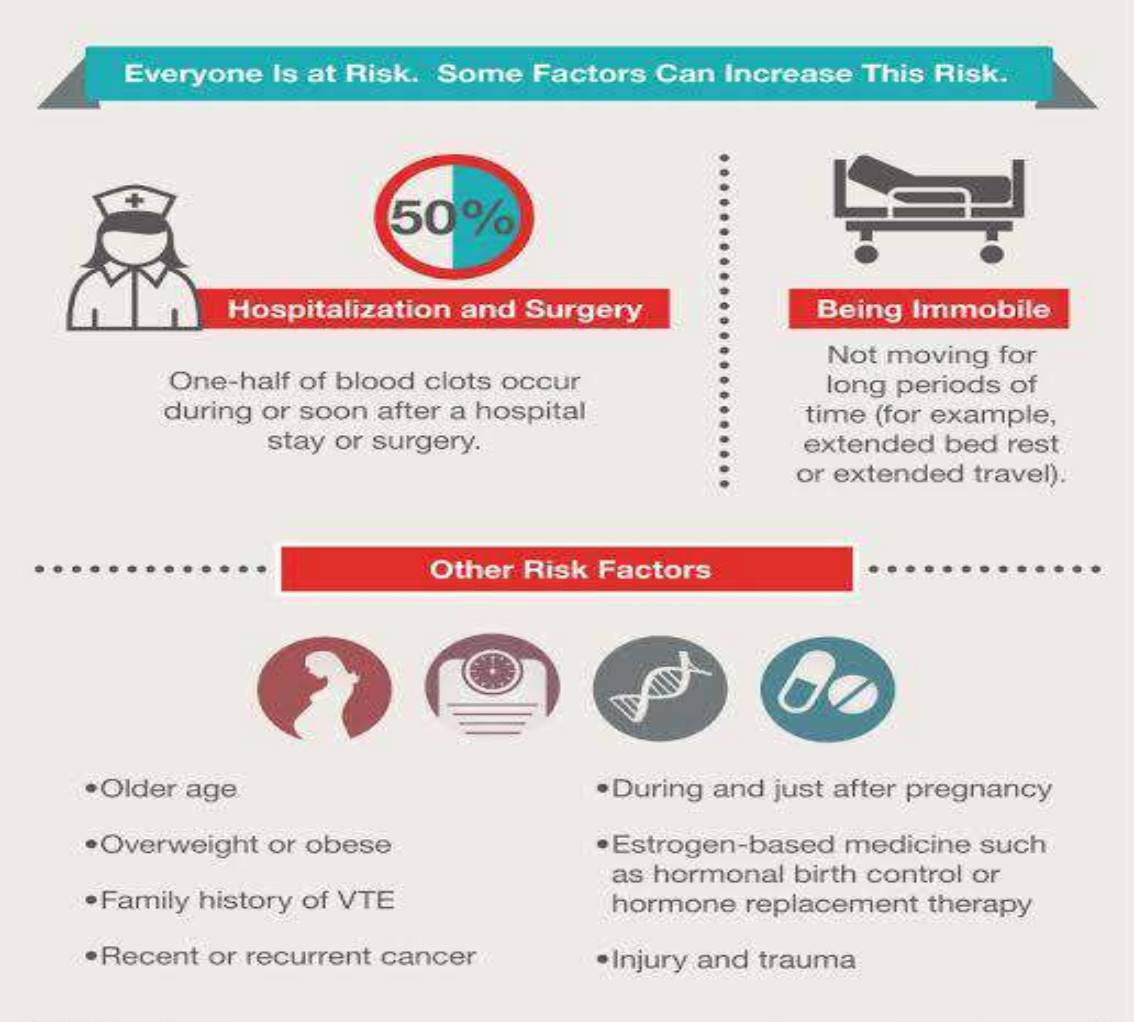
ETIOLOGY – VENOUS THROMBOEMBOLISM

Various conditions , diseases and physiological alterations are associated with higher risk of venous thromboembolism development. Associated etiological factors are broadly divided in four categories as follows

- HOST FACTORS
- MEDICAL DISEASES -RELATED
- SURGICAL /IATROGENIC CAUSES
- HEMATOLOGIC DISORDER

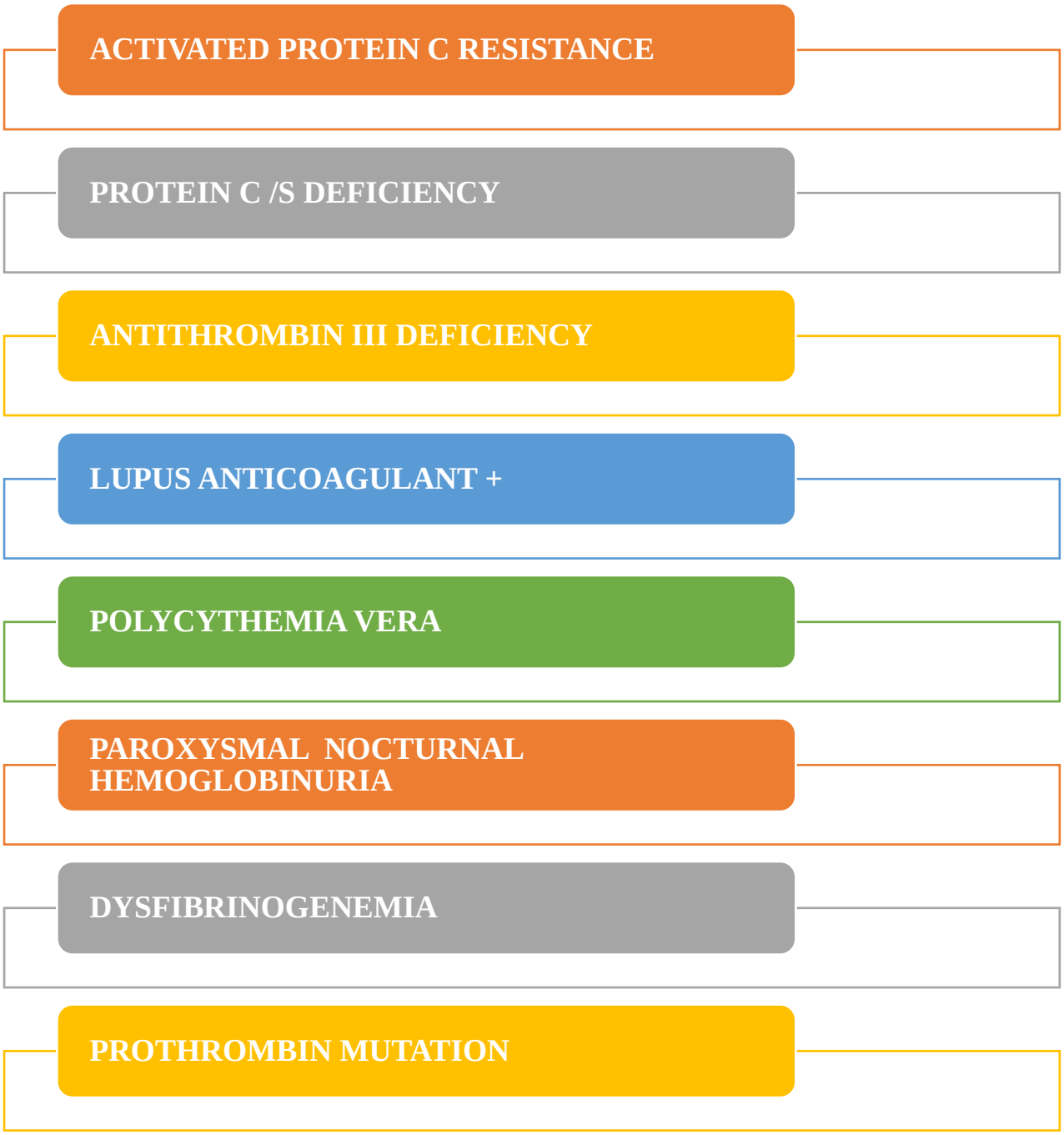
Various conditions in each category carries variable percentage of increased risk in developing venous thromboembolism when compared to normal individuals. These etiological factors associated with VTE also determines the prognosis and outcome and knowledge of these associations are mandatory.

HOST FACTORS	MEDICAL CONDITIONS - RELATED	SURGICAL/IATROGENIC FACTORS
• Age > 40 years • Obesity • Varicose veins • Use of OCP/HRT • Prolonged Immobility	• Malignancy • Congestive cardiac failure • Nephrotic syndrome • Recent MI • Inflammatory bowel disease • Spinal cord injury • Pelvis, Hip, long bone fracture	• Hip surgery - 50% have proximal DVT • Knee surgery • Pelvic surgery - 40-80% had calf DVT, 10-20% had thigh DVT • CABG • Urologic surgery • Neurosurgery

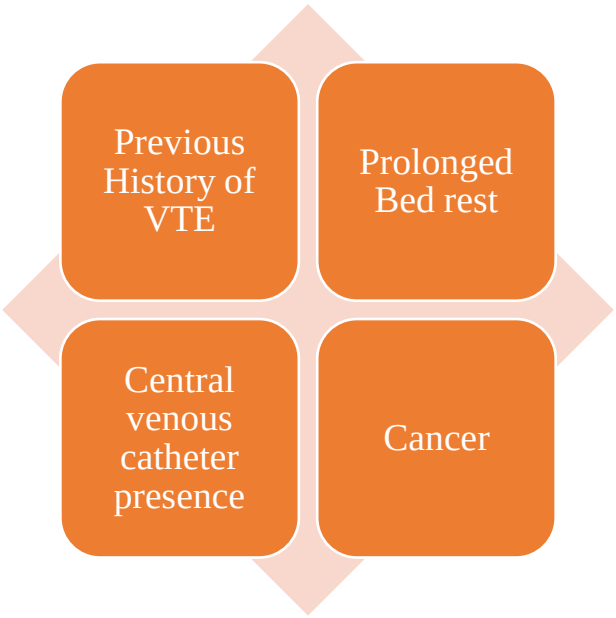


HEMATOLOGIC CONDITIONS – ASSOCIATED WITH VTE

As already described by Rudolf Virchow, blood flow abnormalities and coagulation disorders contribute to development of thrombus and embolism in venous channels. Hematological disorders associated with higher prevalence of VTE are as follows.



For a patient admitted in ward or intensive care unit, risk of developing VTE was assessed using a 4 –point scoring system. Parameters assessed in 4-point risk score are



Presence of any one of these risk elements highly predicts occurance of VTE within 90 days of risk assessment/admission.

In addition to this, risk factors are stratified as low, intermediate , high risk for development for VTE in future. The stratified classification of risk factor are as follows.

TABLE 1. Risk Factors for VTE[10]

Strong	Moderate	Weak
• Fracture of pelvis, hip, or long bones of leg • Hip or knee arthroplasty • Major general surgery • Major trauma • Spinal cord injury	• Arthroscopic knee surgery • Central venous lines • Congestive heart failure • Estrogen therapy • Malignancy • Paralytic stroke • Pregnancy/postpartum • Genetic thrombophilia	• Bed rest >3 days • Prolonged immobility • Age • Laparoscopic surgery • Obesity • Varicose veins

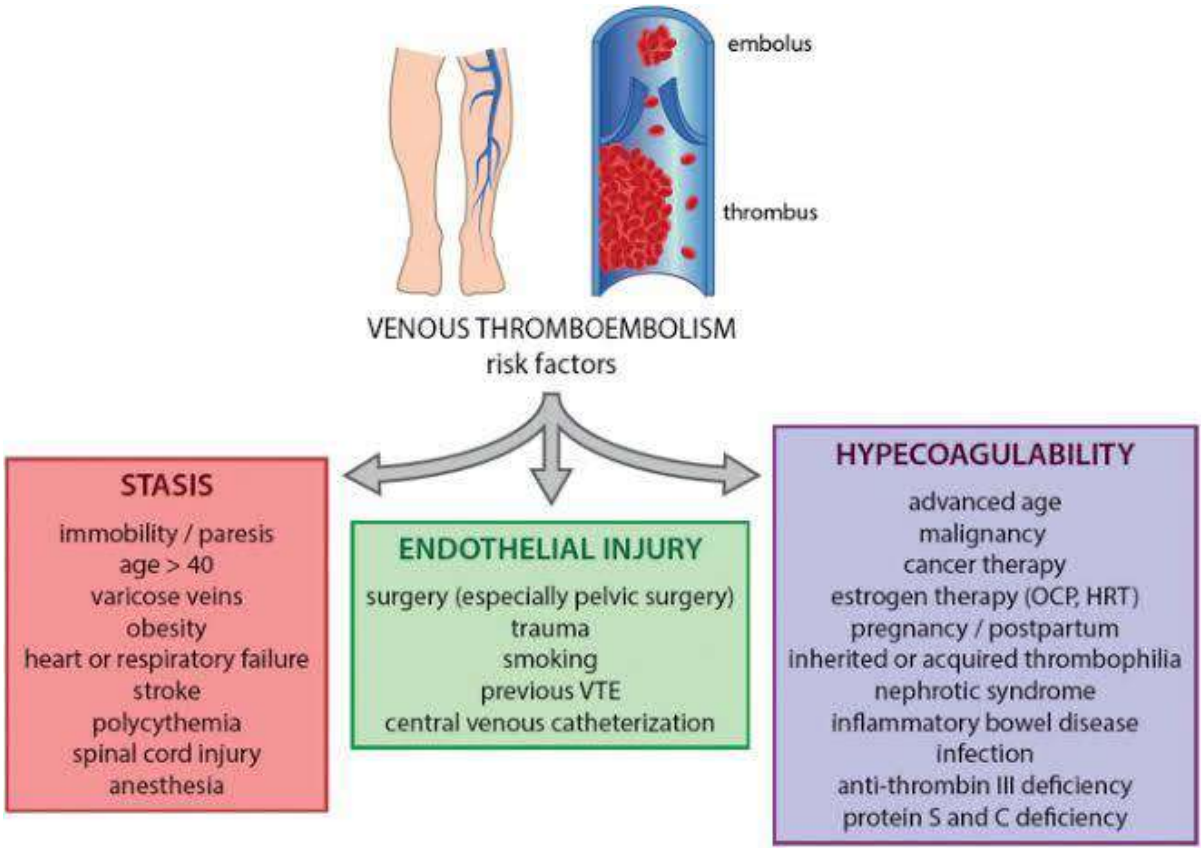
Adapted from Anderson FA Jr, Spencer FA. Risk factors for venous thromboembolism. *Circulation.* 2003;107(23 suppl 1):I9-I16. doi: 10.1161/01.CIR.0000078469.07362.E6.

PATHOPHYSIOLOGY - VENOUS THROMBOEMBOLISM

A thrombus is defined as a platelet and fibrin aggregate plug with few trapped red and white blood cell contents intending to occlude a blood vessel. Formation of thrombus is explained by **Virchow's triad** by Rudolf Virchow. Alterations in one of the three factors leads to formation of thrombus within intact blood vessel. Those three factors are

- BLOOD STASIS
- ENDOTHELIAL INJURY
- ALTERATION OF COAGULATION

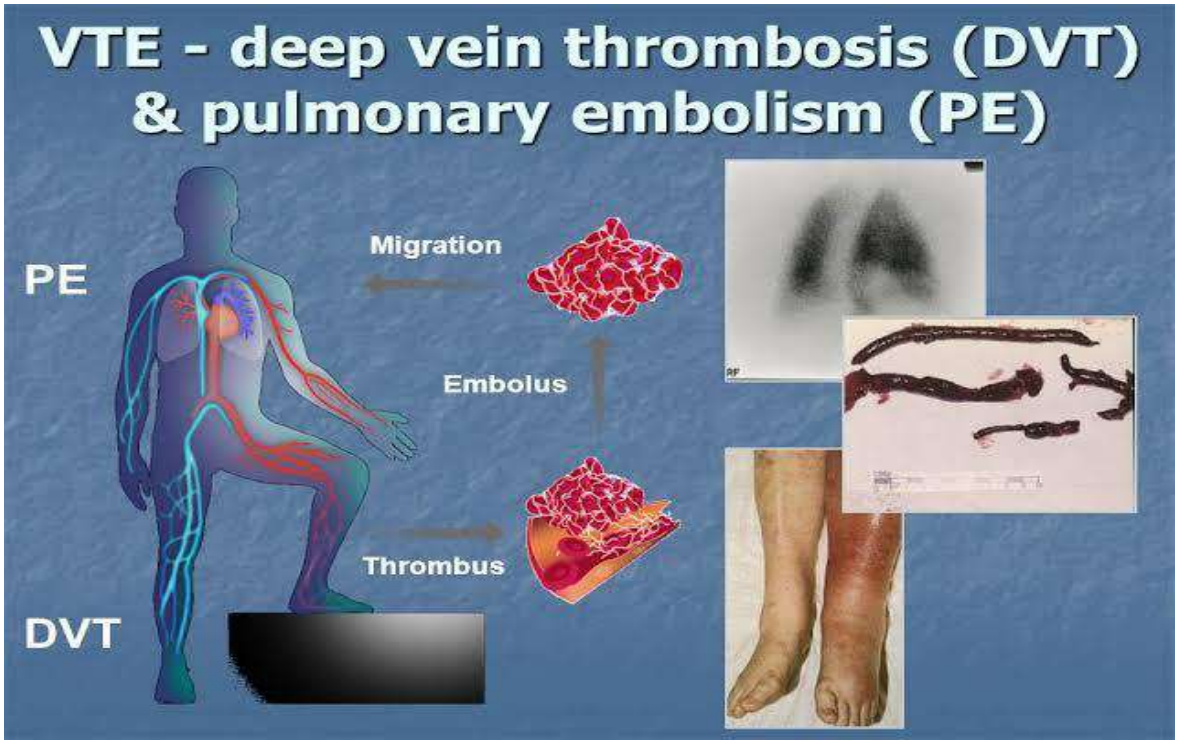
Risk factors related to these three factors alteration causes formation of thrombus in deep venous channels.



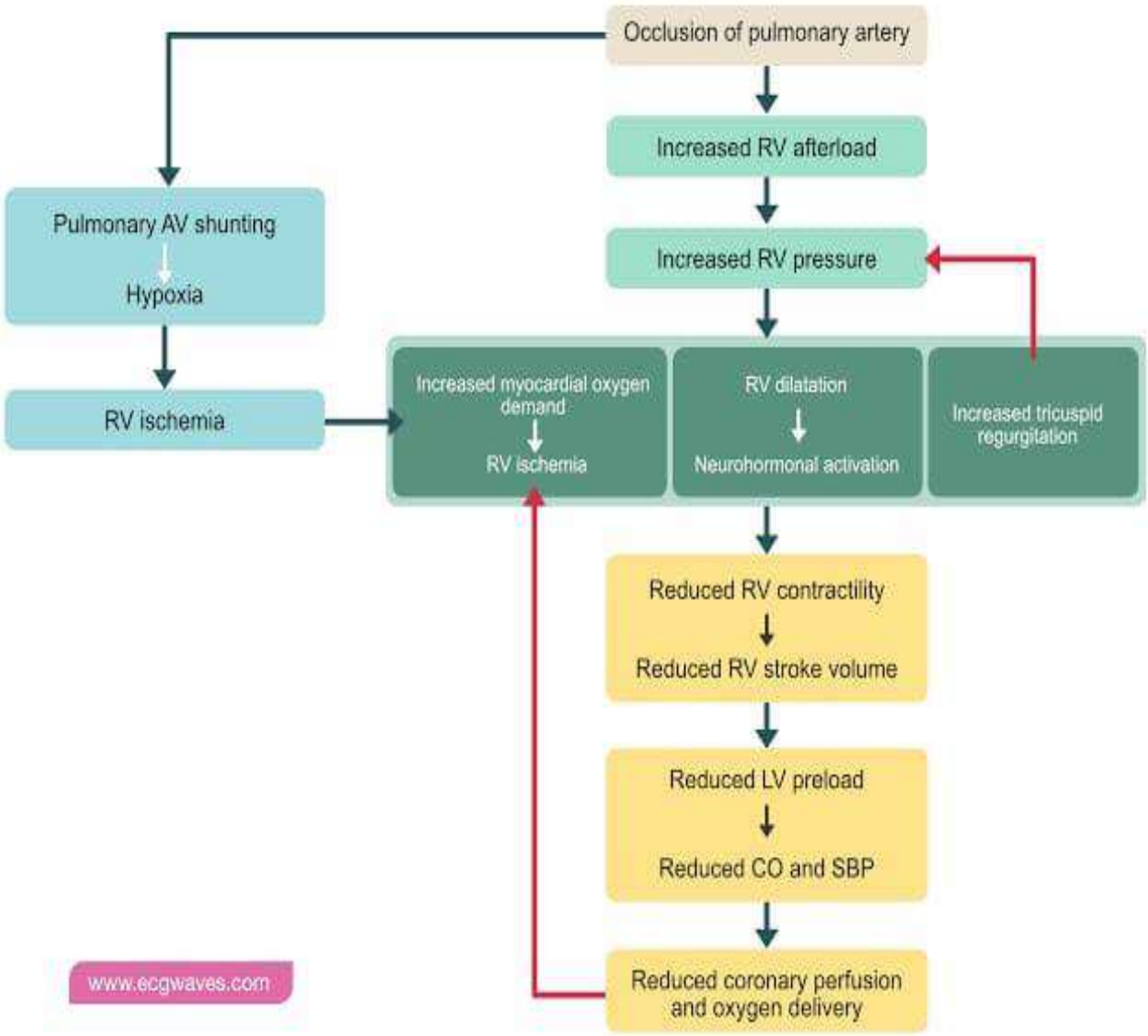
Hence formation of thrombus in deep veins of legs, pelvis and arms are called as DEEP VEIN THROMBOSIS which contributes to 2/3rd of VTE .

PULMONARY THROMBOEMBOLISM – PATHOPHYSIOLOGY

Dislodgement of the propagating clot in deep veins of legs or fragmentation leads to transfer of thrombus as emboli to pulmonary circulation. Obstruction of Pulmonary artery by embolus causes increased pulmonary vascular resistance and arterial hypoxemia resulting in clinical features of pulmonary embolism.



Acute/ Chronic occlusion of pulmonary artery by thrombus or emboli leads to a series of events leading to ventilation – perfusion mismatch among pulmonary parenchyma. In addition, it also increases right heart load and creates eventually right heart dysfunction & failure. Series of events depicted in the following picture in detail

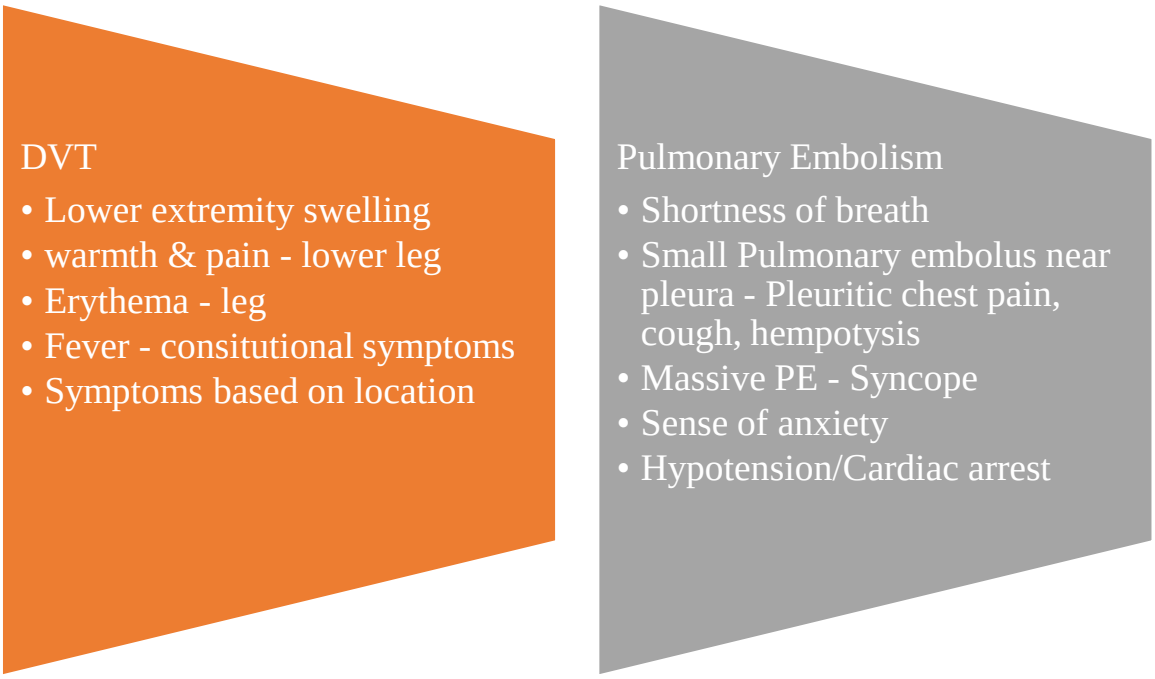


This increase in RV load and pressure is due to reflex pulmonary vasoconstriction due to acute embolus and its released vasoactive substances like serotonin etc.. Also, if the process of pulmonary embolism becomes chronic, chronic thromboembolic pulmonary hypertension will be developed.

CLINICAL FEATURES :

Patients with venous thromboembolism may present with diverse clinical features ranging from mild fever (in DVT) to sudden cardiac arrest (massive Pulmonary embolism).

Symptoms of VTE are described as follows



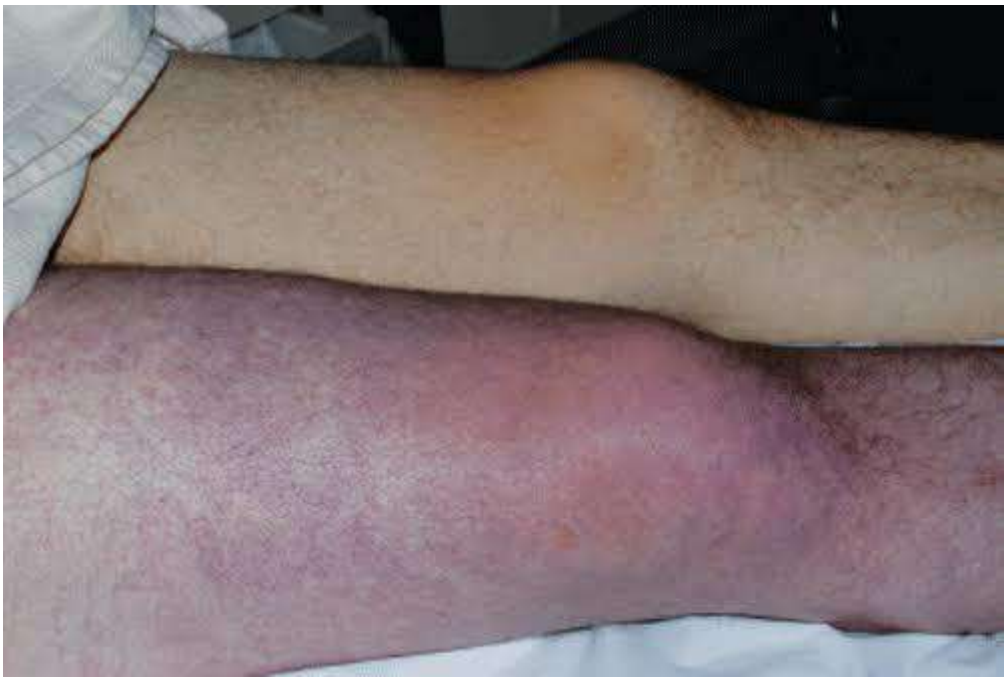
PHYSICAL EXAMINATION

DEEP VEIN THROMBOSIS OF LEG VEINS

As already discussed , localised lower limb swelling as sociated erythema/redness, warmth and tenderness of calf muscles were the usual findings in DVT. However signs include

- HOMAN's SIGN – Forciful dorsiflexion of ankle produces intense pain which signifies the presence of deep vein thrombosis

- MOSES SIGN – Pain elicited during squeezing calf muscles towards tibia
- Phlegmasia alba dolens
- Phlegmasia cerulea dolens



PULMONARY EMBOLISM – SIGNS

Awareness of signs of pulmonary embolism is important as early diagnosis can be made if suspected and looked for. Important signs are as follows.

Table 3

Signs and Symptoms of PE[a]

Symptoms	Signs
Chest pain (pleuritic and nonpleuritic)	Tachypnea (e.g., respirations >16/min)
Dyspnea	Tachycardia (e.g., pulse >100/min)
Apprehension	Hypotension
Cough	↑S_2
Hemoptysis	Gallop
Diaphoresis	Rales
Syncope[b]	Temperature >37.8°C
Cardiopulmonary arrest[b]	Phlebitis
Palpitation	Diaphoresis
Chest tightness	Edema
	Murmur
	Cyanosis[b]

[a] Seen in angiographically proven massive and submassive PE.
[b] Usually seen in massive PE.
PE: pulmonary embolism; S_2: pulmonic component of the second heart sound. Source: References 1, 2, 6, 10.

As already stated the signs and symptoms of Venous thromboembolism are diverse and hence its difficult to localise the diagnosis as VTE with some of these signs alone. Hence various scoring systems which can be used to interpret the likelihood of VTE were formed. One such landmark scoring system is WELL's CRITERIA.

Wells score

Criteria	Points
Clinical signs/symptoms of DVT	3
PE is most likely diagnosis	3
Tachycardia (>100 bpm)	1.5
Immobilization/surgery in previous 4 weeks	1.5
Prior DVT/PE	1.5
Hemoptysis	1
Active malignancy (trt w/in 6 month)	1

Low Risk < 2 points | **Intermediate risk** 2-6 points | **High risk** >6 points

PE unlikely 0-4 points | **PE Likely** >4 points

Modified Well's scoring system is similar with addition of hs-D-Dimer values to the scoring algorithm. Clinically Well's score behaves as a excellent tool in prediction of Venous thromboembolism in high risk patients.

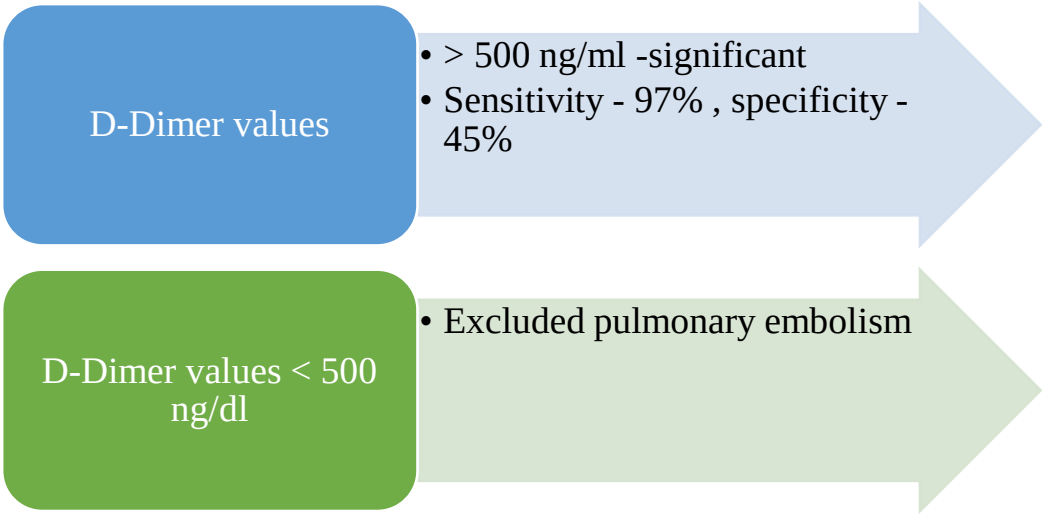
APPROACH TO A PATIENT SUSPECTED TO HAVE VENOUS THROMBOEMBOLISM

If a patient has a risk factor associated with high risk of VTE and greater Well's score, further laboratory investigations are performed for confirmation of the diagnosis. However, therapy should not be delayed and anticoagulants can be

started if the patient was suspicious of VTE according to physical findings & WELL's criteria (>4).

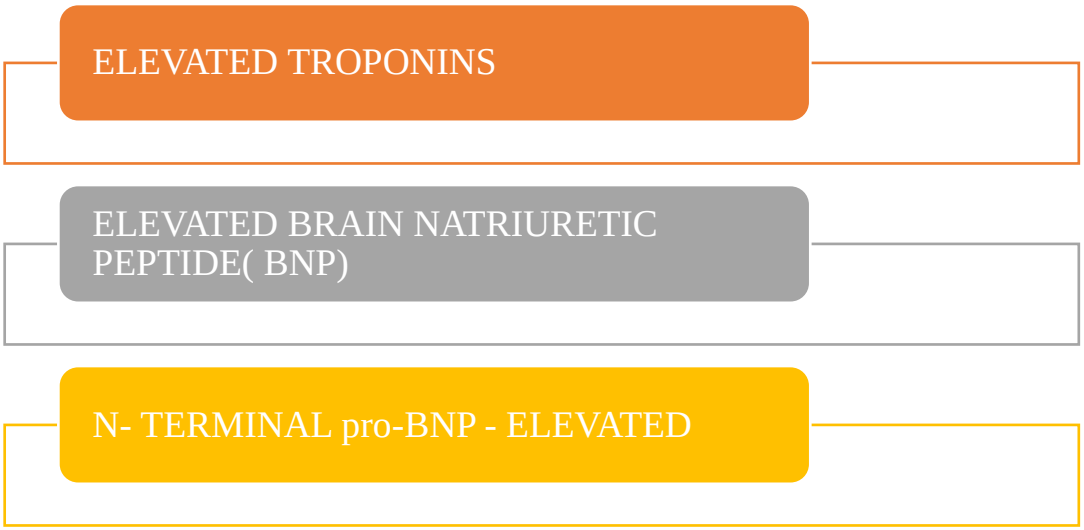
INVESTIGATIONS

- Complete blood count – evidence of underlying risk factor like hemoglobinopathy (anaemia), infection, platelet disorders, sepsis etc.. Higher WBC count are usually prevalent in VTE patients
- Liver Function test – Lower bilirubin levels were seen commonly (described in study by Duman et al)
- C- Reactive protein – may be positive
- Coagulation Profile – abnormalities can be seen
- Plasma D-Dimer levels – elevated which is usually tested by ELISA. D-Dimer is product formed when cross-linked fibrin in thrombus is degraded by fibrinolytic system.



D-dimer levels were included in Modified Well's score for predicting and excluding Venous thromboembolism.

- Arterial Blood gas analysis – Results in VTE/PE include
  - Hypoxemia (PaO2 < 80 mmHg)
  - Hypocapnoea
  - Respiratory Alkalosis
  - Elevated alveolar – arterial oxygen gradient.
- MARKERS OF ADVERSE OUTCOME IN PULMONARY EMBOLISM



These markers in pulmonary embolism predict adverse outcome and mortality accurately than D-dimer levels.

IMAGING – VENOUS THROMBOEMBOLISM

CHEST RADIOGRAPHY

WESTERMARK SIGN

- Decreased pulmonary vascularity

HAMPTON HUMP

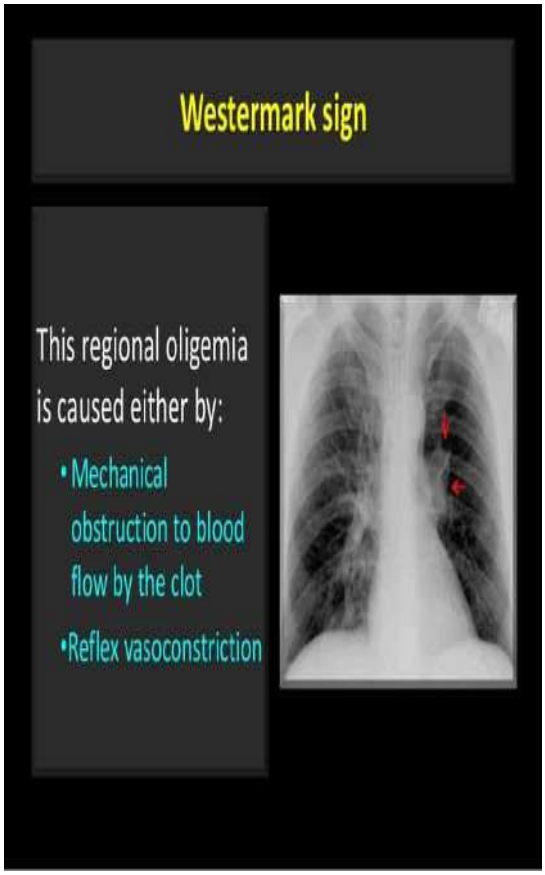
- Elevation of hemidiaphragm

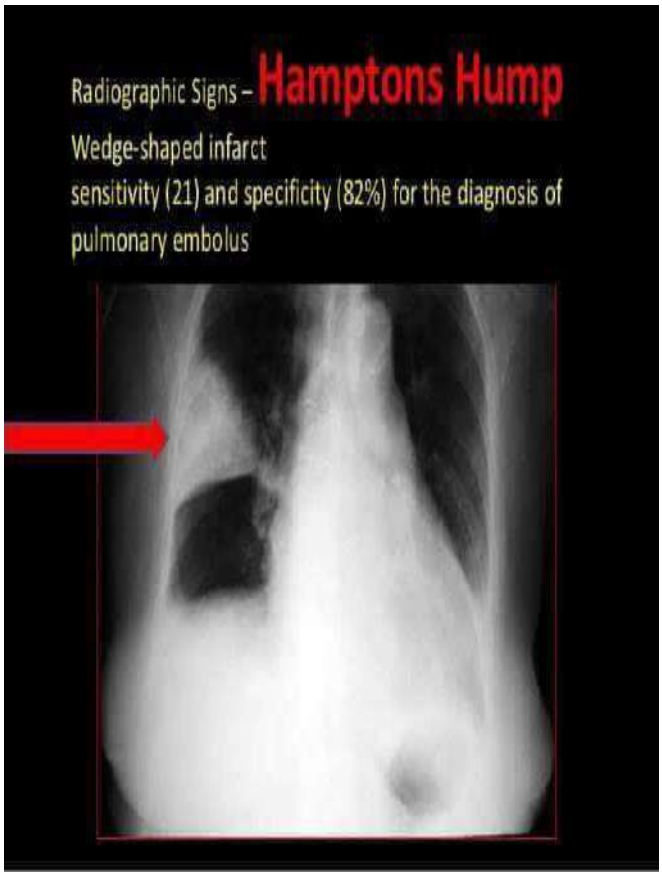
Enlarged Right descending Pulmonary artery

Wedge shaped infiltrate

Pulmonary infarction - Pleural effusion

Can be normal in many patients.


Westermark sign
This regional oligemia is caused either by:
• Mechanical obstruction to blood flow by the clot
• Reflex vasoconstriction


Radiographic Signs – Hamptons Hump
Wedge-shaped infarct
sensitivity (21) and specificity (82%) for the diagnosis of pulmonary embolus

High Resolution Spiral CT chest

Pulmonary embolism is often identified as filling defect either in center or near the wall of lungs. Its sensitivity in detection of Pulmonary embolism is around 93%.

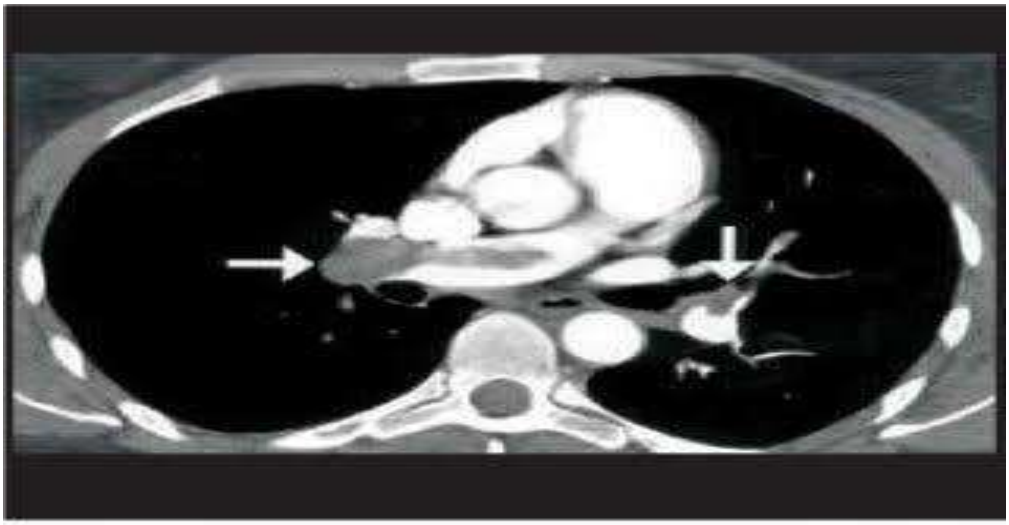


Figure 4- Acute pulmonary thromboembolism in 49-year old woman. Spiral CT image performed on multidetector CT reveals filling defects in the right main and interlobar pulmonary arteries and in the left lower lobe pulmonary artery with extension into the lingular artery (arrows).

LIMITATION

- ✓ Higher dose of radiation exposure
- ✓ Contrast administration needed
- ✓ Oblique or horizontal vessels are less visualized/missed.
- ✓ Inconclusive in 1-10% of PE patients.

VENTILATION –PERFUSION SCANNING

It is a screening tool for diagnosis of pulmonary embolism. This scan estimates defects in perfusion when compared to ventilation and their size, location and total number of areas with low perfusion in lungs.

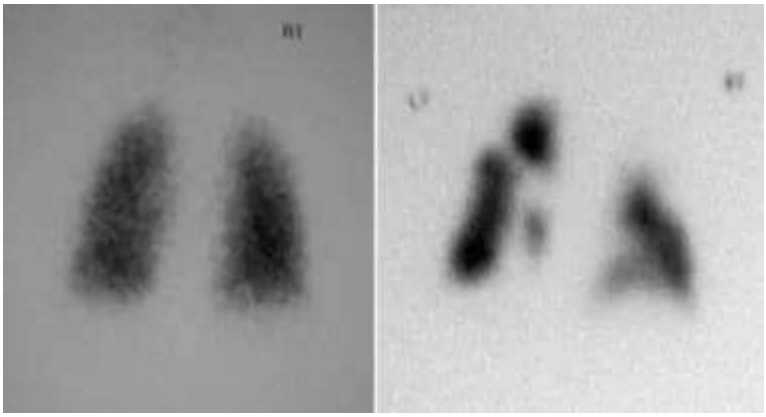


Image shows normal ventilation in right side with reduced perfusion in left upper quadrant in left side image in a patient with ventilation – perfusion mismatch due to pulmonary embolism – VENTILATION-PERFUSION SCAN

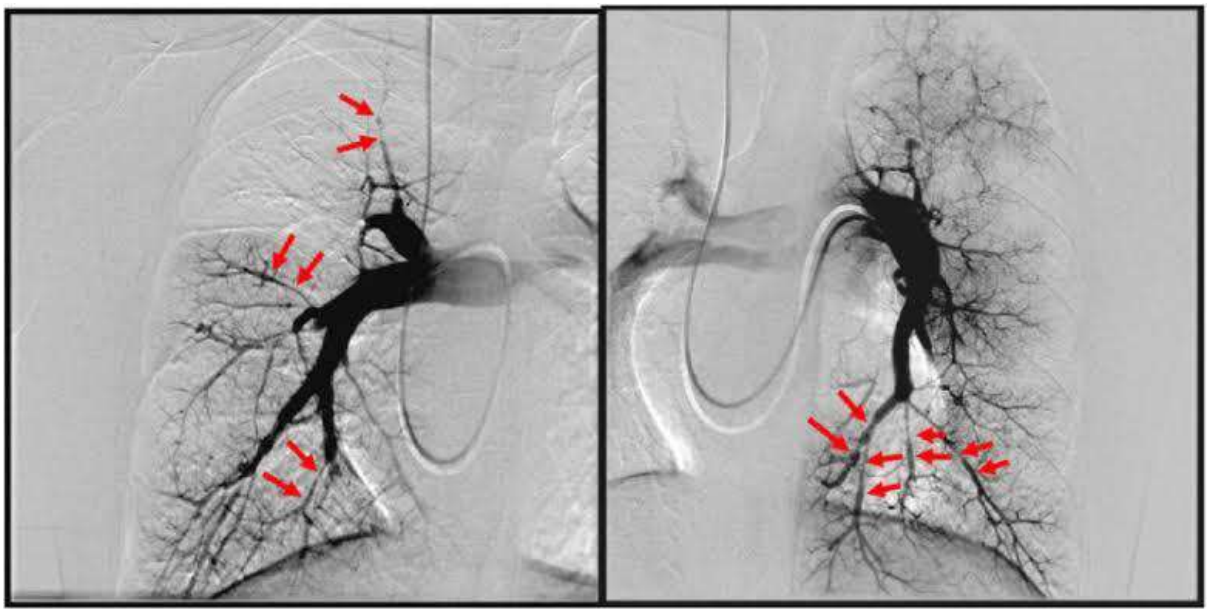
LIMITATIONS:

- Inconclusive in 66% of cases.

PULMONARY ANGIOGRAPHY

Gold standard investigation used in diagnosis of pulmonary embolism. Contrast agent is administerated and the pulmonary vasculature is studied .

CUT –OFF SIGN – Embolus is depicted as cut-off of a vein and reduced flow to distal areas.



CUT-OFF SIGN – PULMONARY EMBOLUS – PULMONARY ANGIOGRAPHY

LIMITATION :

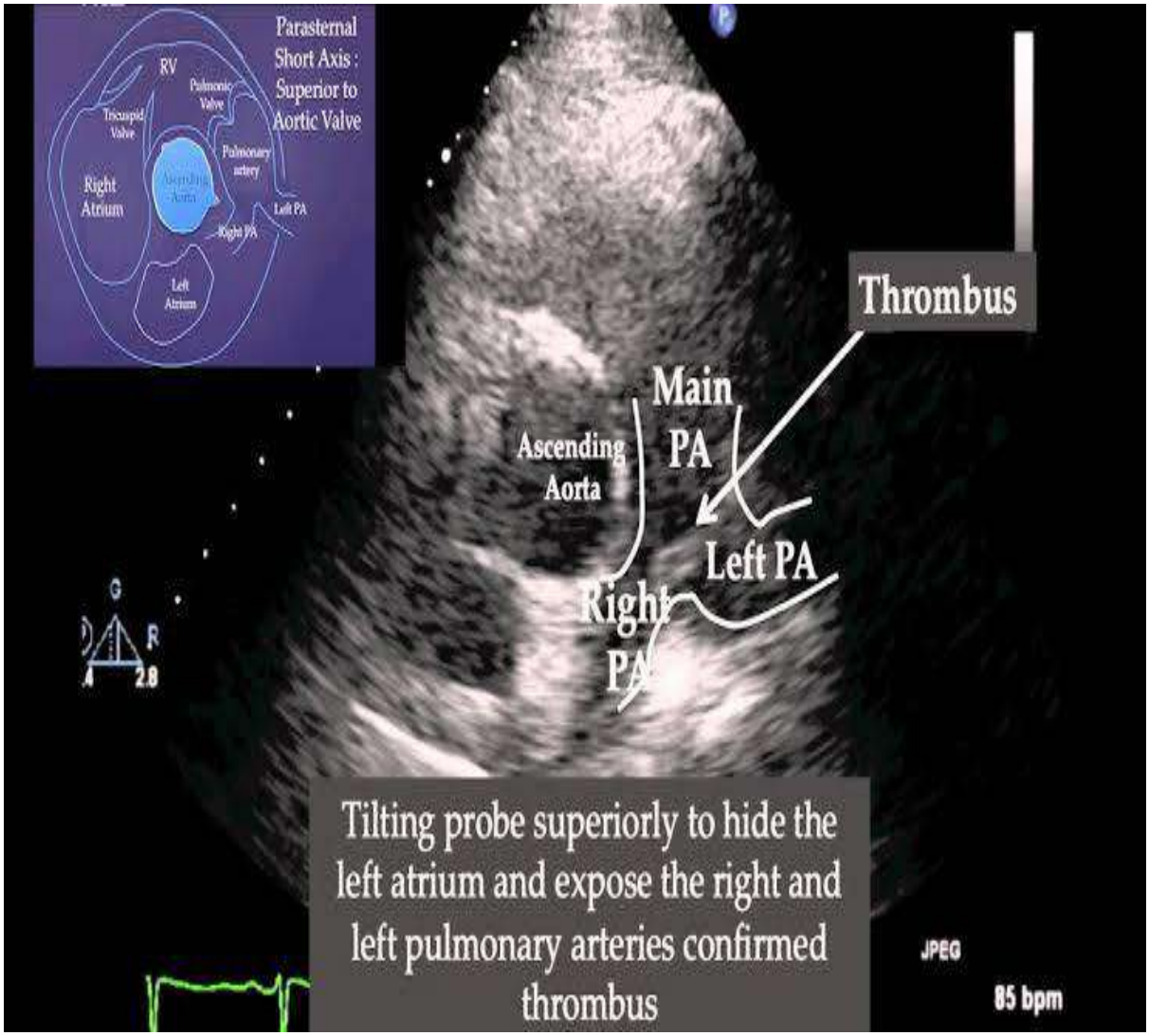
- Invasive procedure
- Contrast related complications can occur
- Expensive
- Complications – Bleeding (2-5%)
- Mortality -`1%

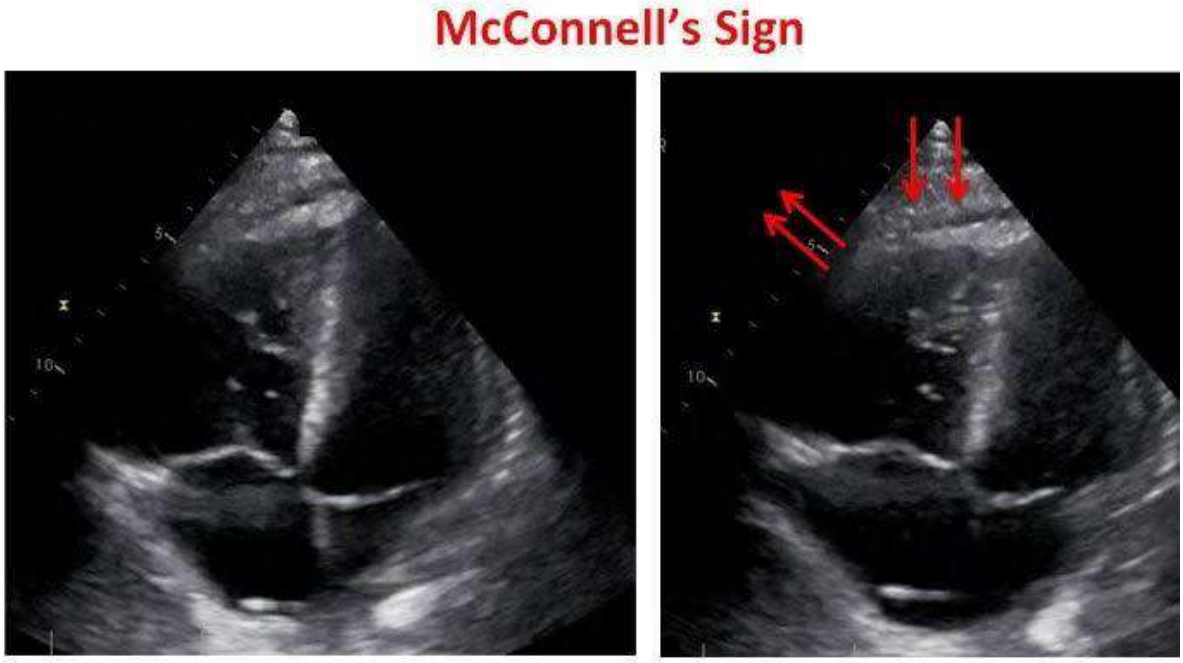
ECHOCARDIOGRAPHY

Features of right heart strain can be visualised in transthoracic echocardiography. Those signs include

- Right ventricular dilatation
- Right ventricular hypokinesia
- Tricuspid regurgitation

- IV septum buldging into left ventricle
- Impending heart failure – regional wall motion abnormality



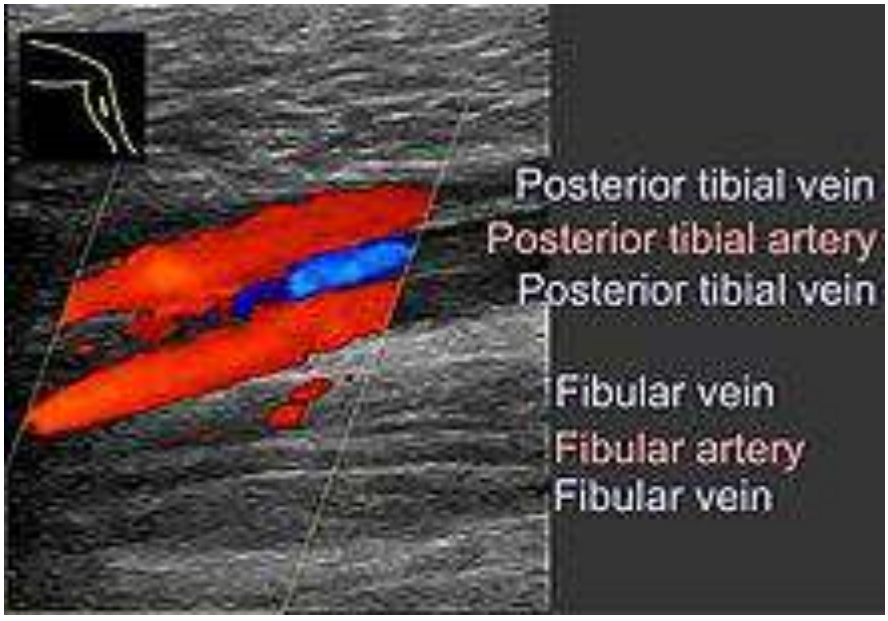


DILATED RIGHT ATRIUM AND RIGHT VENTRICLE WITH McCONNELL's SIGN – Akinetic buldging mid RV free wall with normal RV apex tethered to LV.

DVT – IMAGING

- Doppler Ultrasonagraphy

Doppler/Duplex scanning of lower limb venous system picks up non-compressibility and presence of internal echoes within deep veins of lower limb which is suggestive of Deep vein thrombosis In addition, colour coding helps us to determine the flow velocities and reversal of flow effectively.



LIMITATIONS:

- Sensitivity in picking up distal DVT is lower when compared to proximal DVT

- VENOGRAPHY

Venography employs administration of contrast material and visualisation of filling defects in deep venous system . Less commonly used now due to its invasive nature and complication of allergic reaction produced by extravasation of contrast material.

OTHER ANCILLARY INVESTIGATIONS

- ELECTROCARDIOGRAPHY

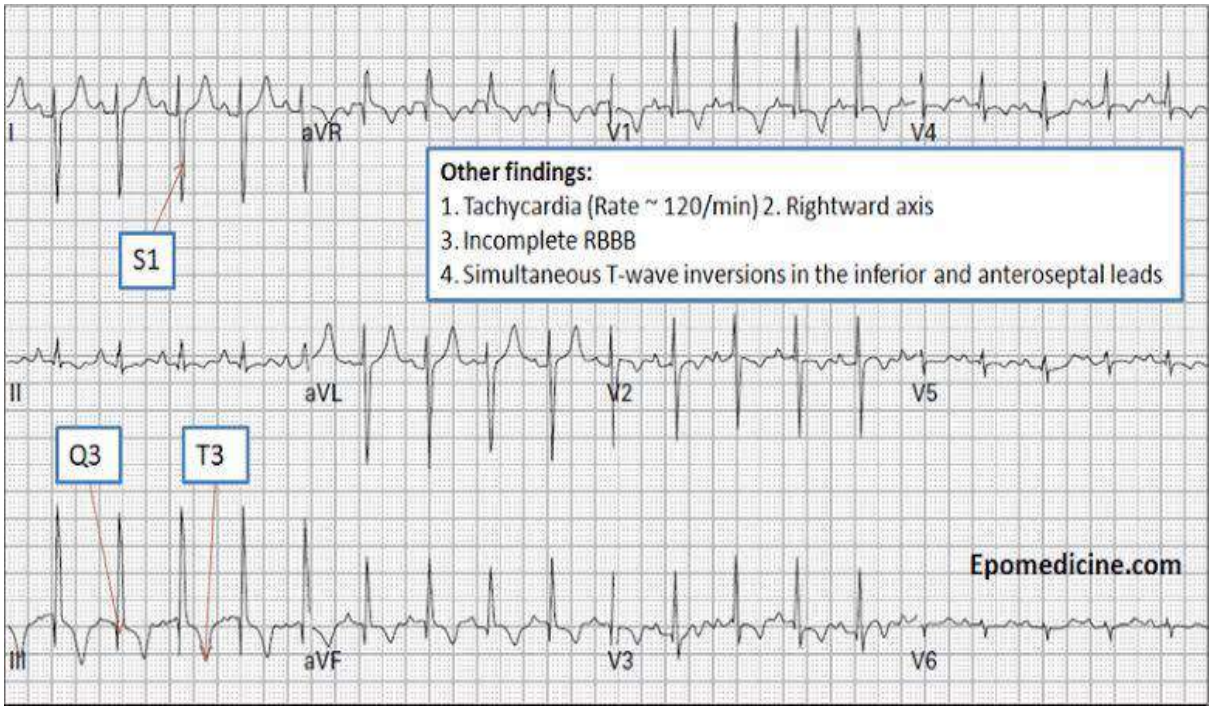
Helps in early identification of patients suspicious of pulmonary embolism. Findings prevalent are

SINUS TACHYCARDIA - most common

RIGHT AXIS DEVIATION, RBBB

S1Q3T3 pattern - most specific

Inverted T waves - V1-V3 leads seen

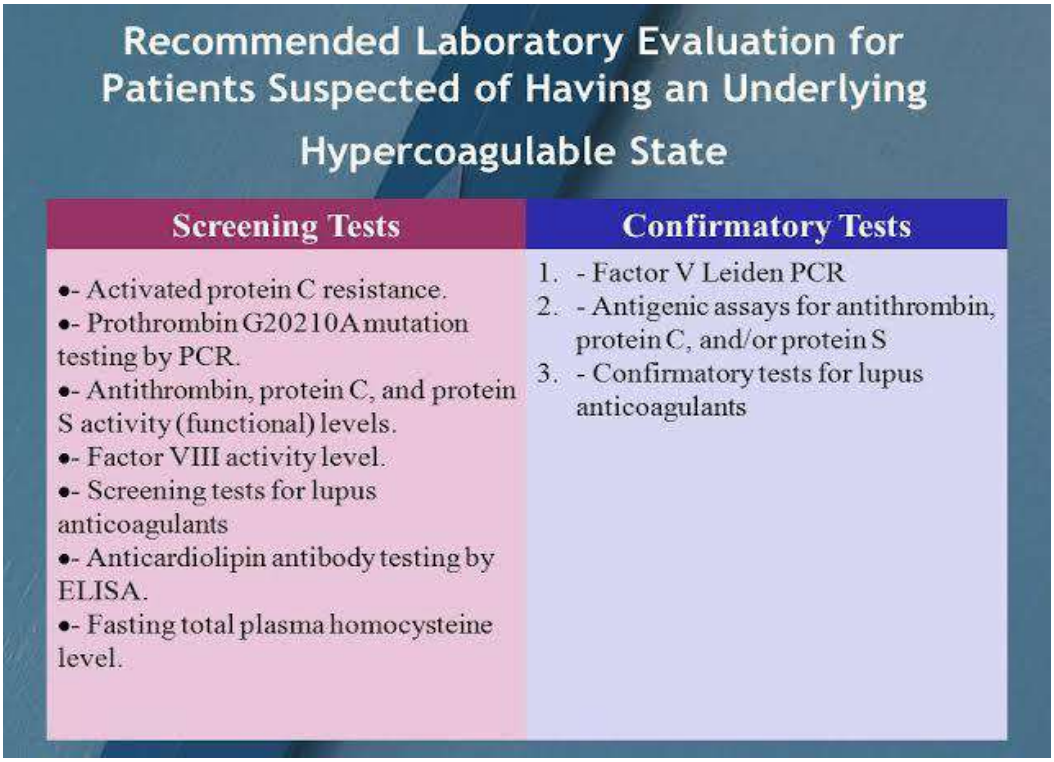


- IMPEDANCE PLETHYSMOGRAPHY

Detects venous emptying in leg which on showing delayed emptying suggests obstruction like DVT.

EVALUATION OF HYPERCOAGULABLE STATES

Various tests are available for evaluation of both inherited and acquired thrombophilia.

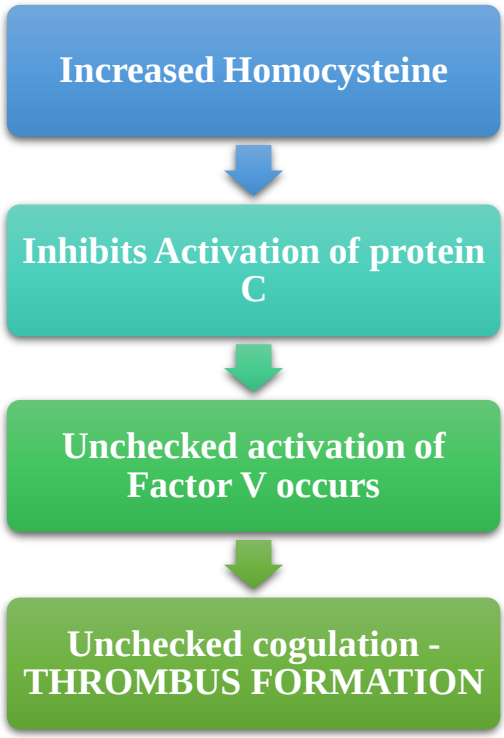


HYPERHOMOCYSTEINEMIA IN VENOUS THROMBOEMBOLISM

Elevated homocysteine levels were commonly associated with development of both arterial and venous thrombosis.

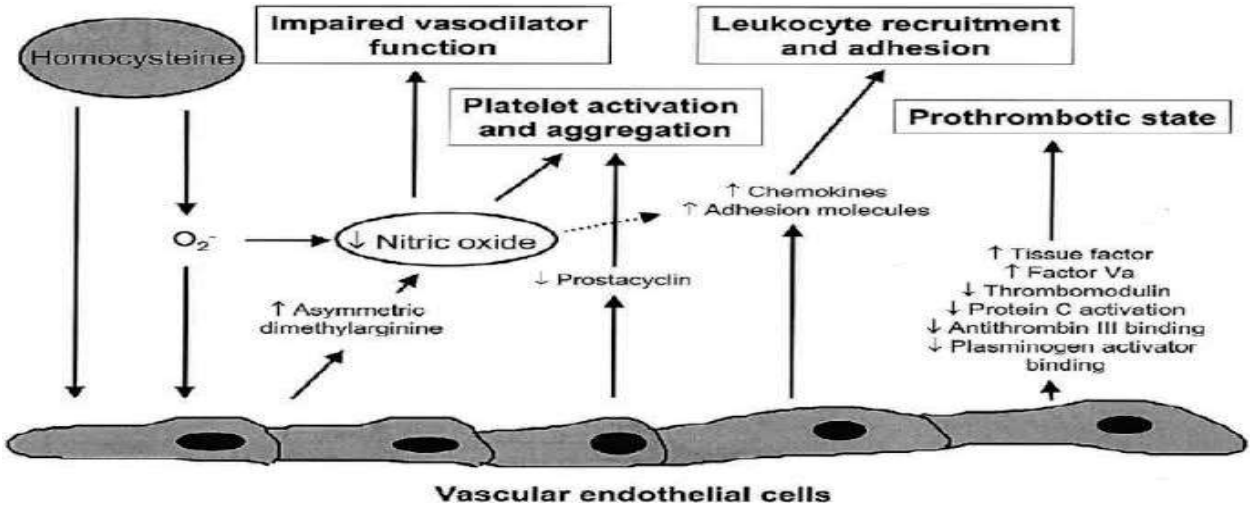
Hyperhomocysteinemia was one of the most common cause of thrombosis in young individuals as evident in studies by Falcon et al. Homocysteine is a aminoacid which plays important role in methionine metabolism

PATHOPHYSIOLOGY OF HYPERHOMOCYSTEINEMIA IN VENOUS THROMBOSIS



OTHER HYPOTHESIS REGARDING PATHOPHYSIOLOGY

TOXIC EFFECT	PROCOAGULANT
• Homocysteinemia has toxic effect on vascular endothelium & clotting cascade	• Homocysteine decrease Antithrombin III binding to endothelial heparan sulphate • Increase affinity between lipoprotein(a) & fibrin • Induction of tissue factor activity in endothelial cells.



THROMBOTIC EFFECTS OF HOMOCYSTEINE

Table 2 – Multiple effects of hyperhomocysteinemia on endothelium and hemostasis [3,4,10,11].

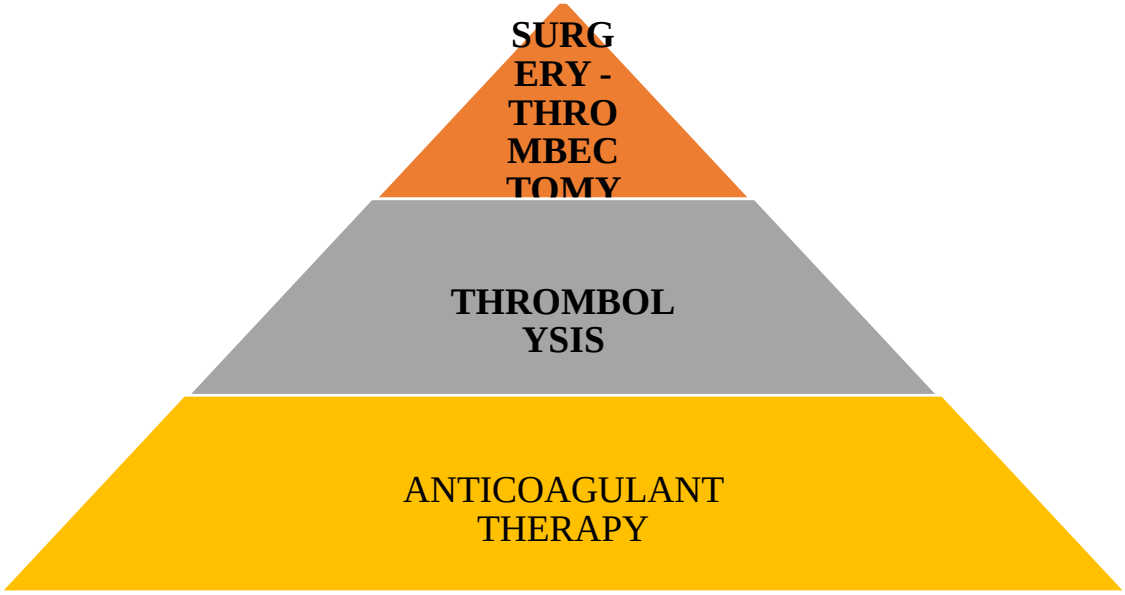
Vascular endothelium	Endothelial dysfunction – Impaired endothelium-dependent vasodilation – Prothrombotic and proinflammatory phenotype of endothelium
Platelets	Increased thromoboxane synthesis Increased platelet reactivity
Fibrinolysis	Impaired fibrinolysis – Decreased binding of tissue plasminogen activator (tPA) – Decreased plasmin generation – Increased level of thrombin activatable fibrinolysis inhibitor (TAFI)
Coagulation factors and natural inhibitors of coagulation	Increased synthesis of tissue factor (TF) Increased activity of factor VII Decreased inactivation of factor Va Increased activation of factor V Decreased activity of antithrombin Increased thrombin generation Fibrinogen modification Inhibition of thrombomodulin activity Inhibition of protein C activation

So, in almost more than 20 studies, hyperhomocysteinemia was strongly associated with venous thromboembolism. In many patients, these elevated homocysteine levels were associated with low cobalamin, folic acid and other various vitamin levels and treatment of the deficient states corrected

homocysteine levels and thereby prevented recurrence of venous thromboembolism.

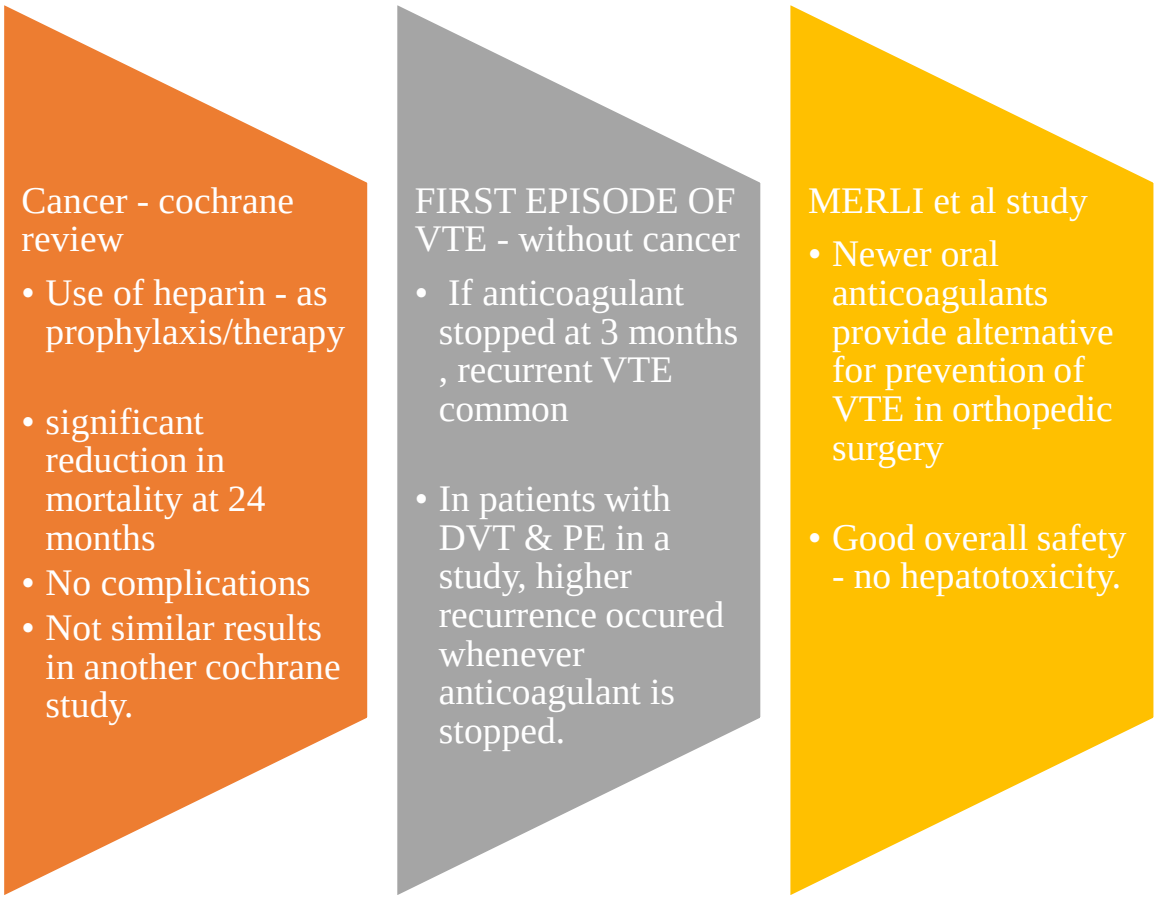
MANAGEMENT

Mainly involves early diagnosis and initiation of low molecular weight heparin (anticoagulants without delay).



- **ANTI-COAGULANT THERAPY**

Prevents further thrombus formation and existing thrombus extension. Duration of anticoagulant therapy is controversial as recurrence is common according to underlying etiology. Hence different duration was studied in various trials in different group of diseased patients.



ANTICOAGULANTS

HEPARIN

- Acute PE - bolus 80mg/kg f/b 18 mg/kg/hr continuous infusion.
- aPTT 1.5-2 times determines adequacy of heparin dose
- Progression or recurrence of thromboembolism is 15 times likely if therapeutic aPTT is not received within first 48 hours.
- Oral anticoagulants started following heparin for 2-3 days

LOW MOLECULAR WEIGHT HEPARIN

- Used in stable PE/DVT, low risk of bleeding, absence of severe renal efficiency, available of monitoring system for LMWH
- Cochrane review - reduced VTE events but not death in cancer pts.

FACTOR Xa INHIBITORS

- Apixaban, edoxaban, rivaroxaban & betrixaban are factor Xa inhibitors
- Apixaban - used for treatment of DVT & PE and prevention of recurrence - AMPLIFY study.
- Edoxaban - for treatment of PE & DVT - 60 mg/day - started after parental anticoagulant for 5-10 days.
- Betrixaban -80-160 mg indicated for prophylaxis of VTE in adults with medical illness causing thromboembolic complications owing to restricted mobility

RIVAROXABAN - FDA INDICATION

- Treatment of DVT & PE
- Reduction in risk of recurrent DVT/PE- 10 mg OD/day -15 mg BD - at least 6 months
- Prophylaxis of DVT following hip/knee replacement surgery
- Prophylaxis of VTE in acute medically ill patients at risk of thromboembolic complications due to restricted mobility

DIRECT THROMBIN INHIBITORS - DABIGATRAN

- Inhibits both free and thrombus bound thrombin formation.
- April 2014 - approved for treatment of DVT & PE - after 5-10 days of parenteral anticoagulation
- RECOVER & RESONATE - noninferior to warfarin

ORAL VITAMIN K ANTAGONIST - WARFARIN

- INR monitoring - target 2-3
- Used after few days of parenteral anticoagulation - as maintanence therapy
- Bleeding risk high

PHASES OF TREATMENT FOR VENOUS THROMBOEMBOLISM

Parenteral	Warfarin (INR 2.0-3.0)	Warfarin (INR 2.0-3.0)
Rivaroxaban 15mg bid	Rivaroxaban 20mg od	Rivaroxaban 20mg od
Apixaban 10mg bid	Apixaban 5mg bid	Apixaban 2.5mg bid
	Dabigatran 150mg bid	Dabigatran 150mg bid
	Edoxaban 60mg od	
		Warfarin (INR 1.5-2.0)*
		Aspirin 100mg od *
		Sulodexide 500LSU bid *

THROMBOLYTIC THERAPY

Acts by activation of plasminogen to plasmin which dissolves fibrin , thus leading to resolution of clot and retrieval of venous flow. Drugs used & approved for FDA in pulmonary embolism are

- rt-PA (recombinant tissue plasminogen activator)
- Tenecteplase
- Alteplase
- Reteplase

INDICATION	CONTRAINDICATION
• Acute PE with hemodynamic instability	• GI bleeding within 6 months • Active/Recent internal bleeding • History of haemorrhagic stroke • Intracranial or intraspinal disease • Recent cranial surgery/Head Trauma • Pregnancy

SURGICAL THERAPY

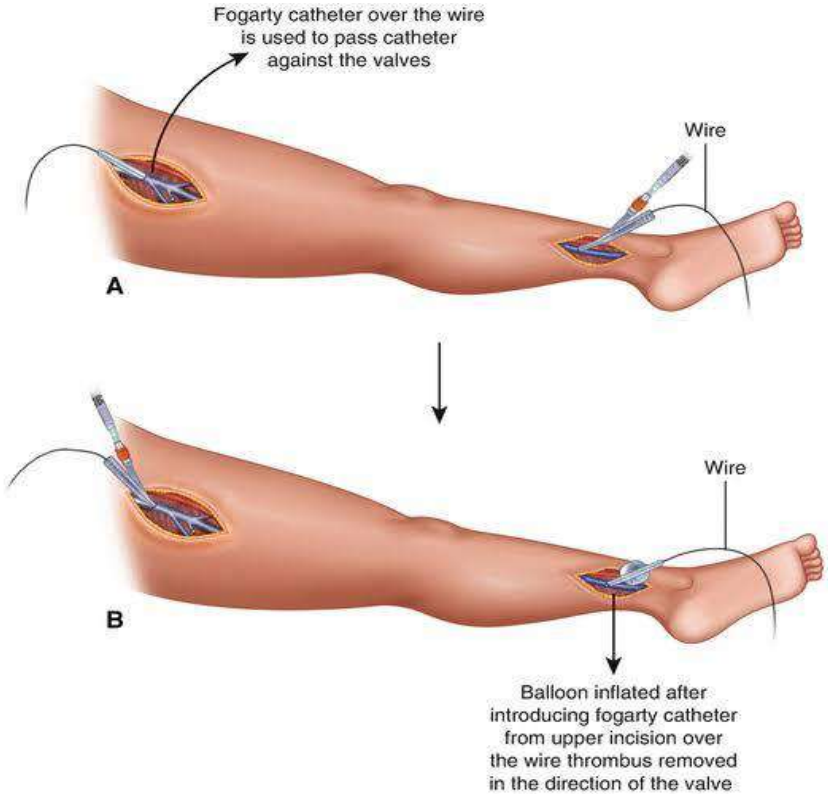
THROMBECTOMY

- Done for venous embolism
- High incidence of re-thrombosis, heparin added usually.

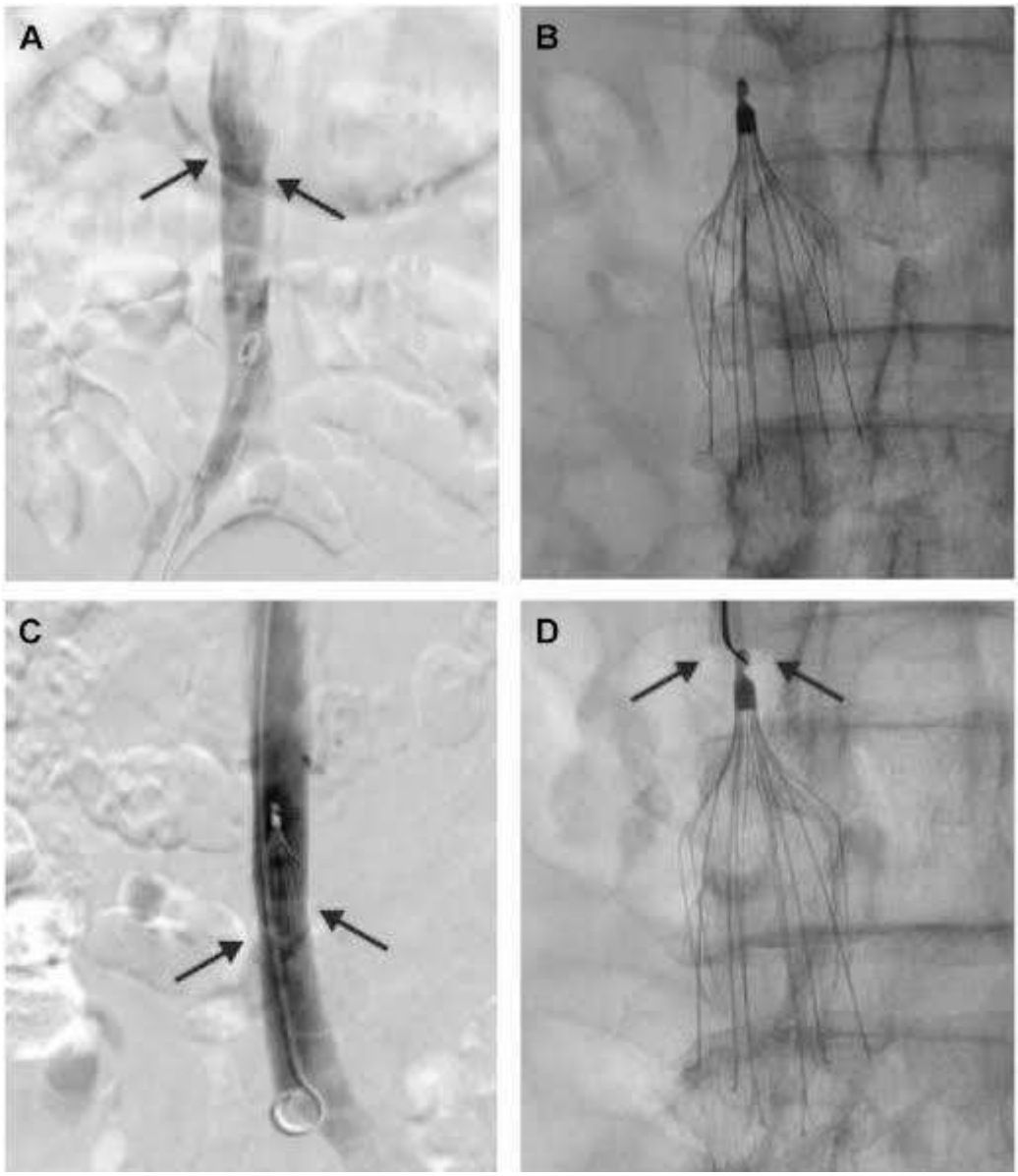
PULMONARY EMBOLECTOMY

- INDICATION - Massive PE with absolute contraindication to thrombolysis
- Effective when clot is in large central vessels
- Mortality very high

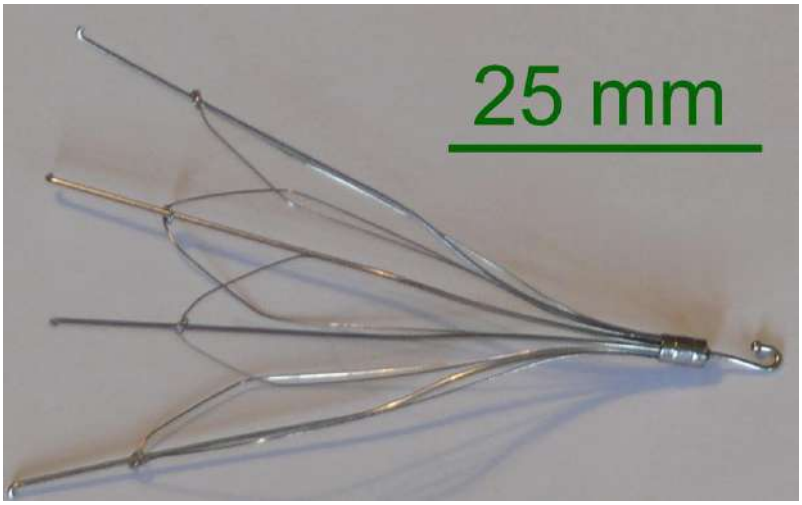
IVC filter



IVC FILTERS	• Traps emboli and maintain patency • Used when anticoagulation is contra-indicated • Used for prevention of recurrence of VTE
LIGATION OF VENOUS TRIBUTARIES	• Rarely practised today • High mortality



PLACEMENT OF IVC FILTER IN IVC

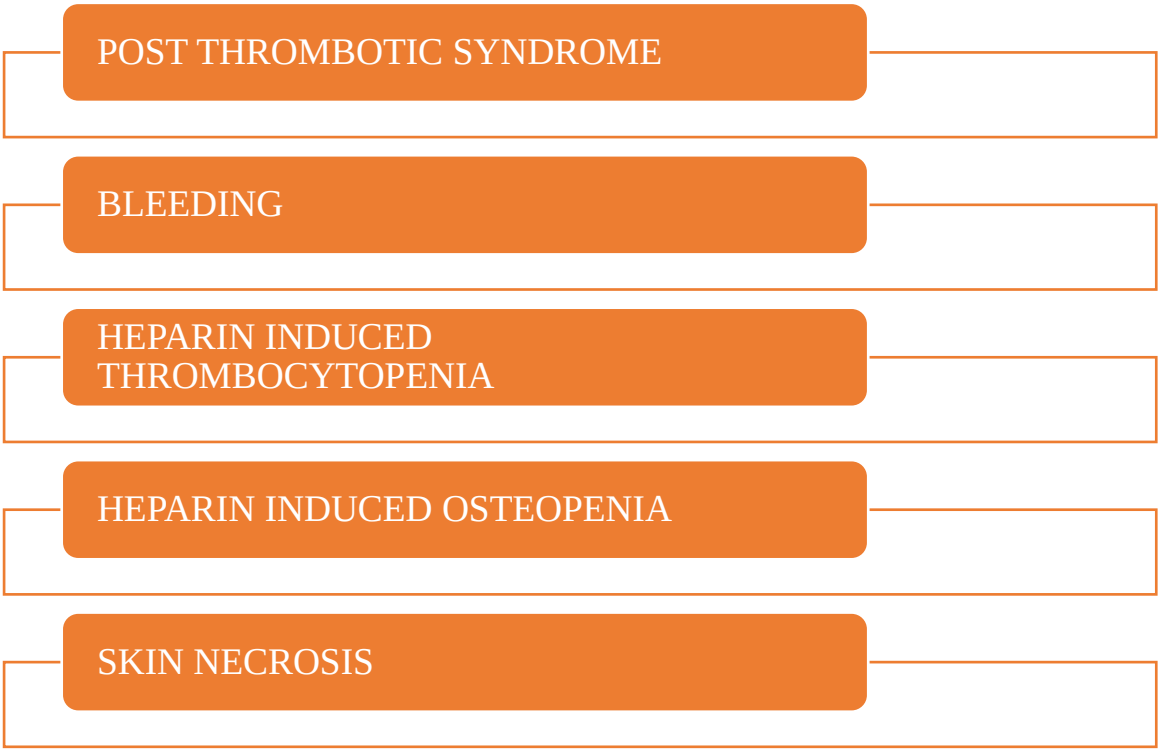


SPECIAL SITUATIONS- VTE IN PREGNANCY

- Heparin is the anticoagulant of choice
- Before delivery , heparin should be stopped and started again after delivery followed by overlapping with warfarin and its continuation in postpartum period
- Pregnant women with history of VTE prior to pregnancy had high chance of recurrence and indicated heparin prophylaxis during pregnancy.

COMPLICATIONS OF VENOUS THROMBOEMBOLISM

TREATEMENT



THROMOBOPROPHYLAXIS

Usually given for patients undergoing surgery 12 hours before and continued for 7-10 days after surgery. Drugs used are

- Unfractionated Heparin Subcutaneous – BD/TDS
- Low Molecular Weight Heparin – Enoxaparin
- Apixaban
- Danaparoid – used in HIT also
- Warfarin - INR monitoring needed 2-3
- Dalteparin

- Rivaroxaban 10 mg/day
- Aspirin
- Dabigatran – 2015 approval for prophylaxis of DVT and PE after hip replacement surgery.

NON-PHARMACOLOGIC THROMBOPROPHYLAXIS

Apart from medical treatment, various non –pharmacological mechanical devices were used and are as follows.

VTE prevention

Venous thromboembolism (VTE) prevention requires nurses to stay current with VTE prevention guidelines and to participate in related quality-improvement projects. Assess your patients for VTE risk factors and take these monitoring and patient education steps:

Increase mobility
- Assist patient with ambulation at least three or four times per day
- Teach patient how to perform lower-extremity range-of-motion exercises and verify completion

Avoid constrictive clothing or devices
- Ensure that any socks or stockings aren't tight around the patient's leg; remeasure for compression stockings if edema develops
- If wrapping the extremity, extend the wrap over a larger area to avoid multiple layers within a small area
- If a leg strap is used to secure urinary catheter tubing or a leg bag, make sure it's not too tight

Promote adequate hydration
- Ask patient for preferred beverage and keep it within reach at the bedside
- Encourage fluid intake throughout the day (unless contraindicated)

Provide mechanical prophylaxis as ordered
- Intermittent pneumatic compression and foot impulse devices
  - Ensure proper fit
  - Encourage consistent use
- Graduated compression stockings (14 mmHg to 15 mmHg)
  - Explain contraindications: arterial disease, significant skin issues, heart failure, unusual leg size or deformity
  - Ensure proper fit and remeasure as indicated
  - Instruct patient to remove daily for skin care and inspection

Monitor closely for any signs of VTE and report immediately
- Deep vein thrombosis—edema, pain, erythema, warmth, or tenderness in an extremity
- Pulmonary embolism—sudden onset of shortness of breath, pleuritic chest pain, cough, hemoptysis or frothy sputum, tachycardia, or lightheadedness

AIMS AND OBJECTIVE

- To determine the relationship between Serum homocysteine in patients with VTE.
- To study the risk fators and outcomes among VTE patients

MATERIALS AND METHODS

Study Design:

Hospital based Cross sectional study

Source of study:

1. Patients attending Cardiology OPD and admitted in Cardiology ward,
2. Patients attending USG Doppler OPD in Radiology Department.

Duration of study:

After approval of ethical committee, approximately 18 months

Study Period : 2021 - 2022

Sample size :

Depends on the case load coming during the study period.

Inclusion Criteria :

1. Adult patients attending USG venous Doppler OPD with clinical features suggestive of DVT.

2. Adult patients attending Cardiology OPD and subsequently admitted in
3. Cardiology ward with suspicious of Pulmonary Thromboembolism.
4. Call over given to Cardiology department from other department with suspicious of PTE.
5. Call over given to radiology department from other department with suspicious of DVT.

EXCLUSION CRITERIA :

1. Age ≤ 12 yrs
2. Patients attending Cardiology OPD with signs and symptoms of Valvular heart disease, Acute Coronary Syndrome, Cardiac arrhythmia, CCF, Pericarditis, Costochondritis and Pulmonary oedema
3. Respiratory causes of chest pain like Pleural effusion, Pneumothorax
4. Non Thrombotic Pulmonary embolism like Fat embolism, Amniotic fluid embolism, Tumor embolism, septic embolism, Air embolism
5. Arterial Doppler study in Radiology OPD.

Investigations :

1. Venous Doppler study of the suspected site
2. ECG in 12 leads
3. Chest X Ray
4. D dimer

5. 2 D ECHO
6. CT Pulmonary Angiography
7. Sr. Homocysteine

STATISTICAL ANALYSIS :

Collected data were verified prior to computerized data entry. The Statistical Package for Social sciences was used for statistical analysis of data. Descriptive studies (eg. Frequency, mean, standard deivation) were applied. Chi square test and test of significance were calculated.

RESULTS

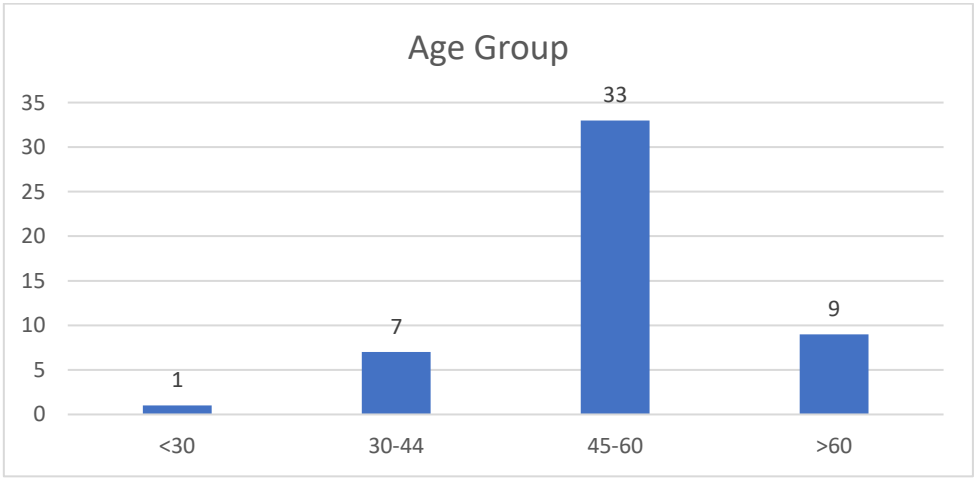
In our study we recruited 50 patients who were diagnosed to have venous thromboembolism in our medical college hospital and serum homocysteine levels were estimated.

Serum Homocysteine levels were compared with other diagnostic modalities of venous thromboembolism like D-dimer, clinical variables, imaging features , outcome and results were analysed.

DESCRIPTIVE STATISTICS

I DISTRIBUTION OF AGE AMONG OUR STUDY GROUP

Age Group	No of cases	Percentage
<30	1	2.00%
30-44	7	14.00%
45-60	33	66.00%
>60	9	18.00%
Grand Total	**50**	**100.00%**
Mean	**52.4**	
SD	**10.999**	

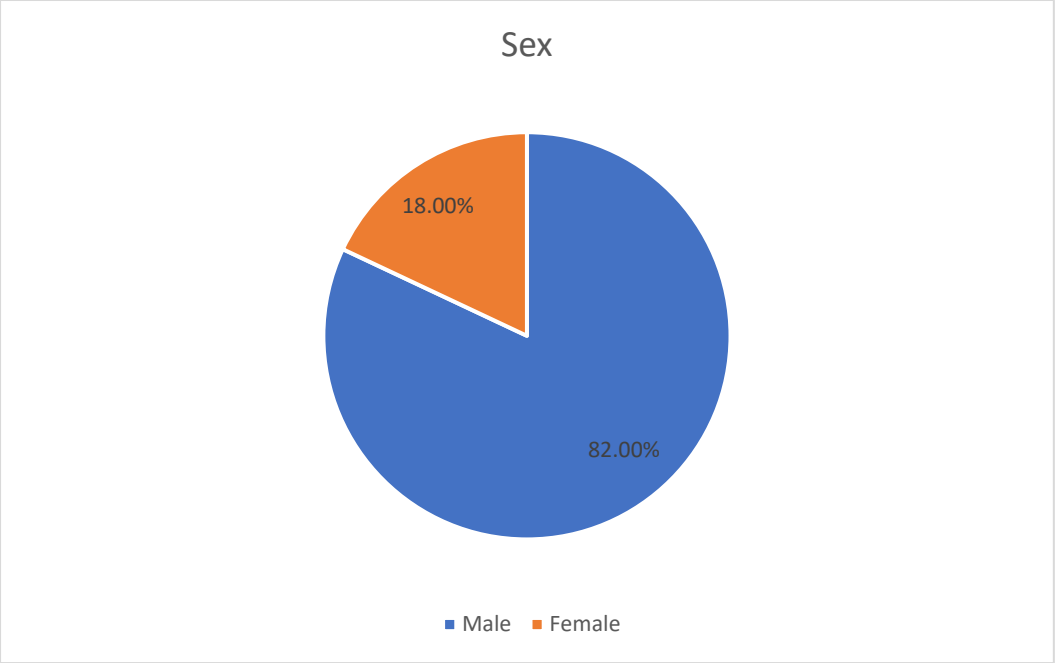


In our study, 66% - majority of patients belong to 45-60 years age group category. 18% of patients were > 60 years of age . Only 2% of patients were less than 30 years of age (young.)

Mean age of all patients in our study group was 52.4 years with a standard deviation of 10.99

II DISTRIBUTION OF GENDER AMONG OUR STUDY GROUP

Sex	No of cases	Percentage
Male	41	82.00%
Female	9	18.00%
Grand Total	**50**	**100.00%**

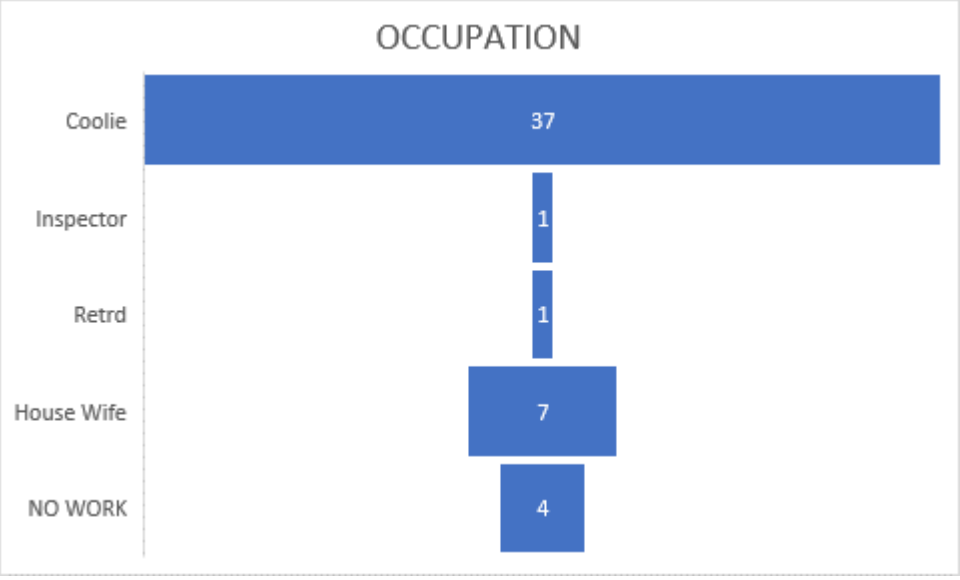


Among our study group, 82% of patients were males whereas only 18% were females.

Male : female ratio in our study group was 4.5:1

III DISTRIBUTION OF OCCUPATION AMONG OUR STUDY GROUP

OCCUPATION	No of cases	Percentage
Daily wage Labourers	37	74.00%
Inspector	1	2.00%
Retrd	1	2.00%
House Wife	7	14.00%
NO WORK	4	8.00%
Grand Total	**50**	**100.00%**

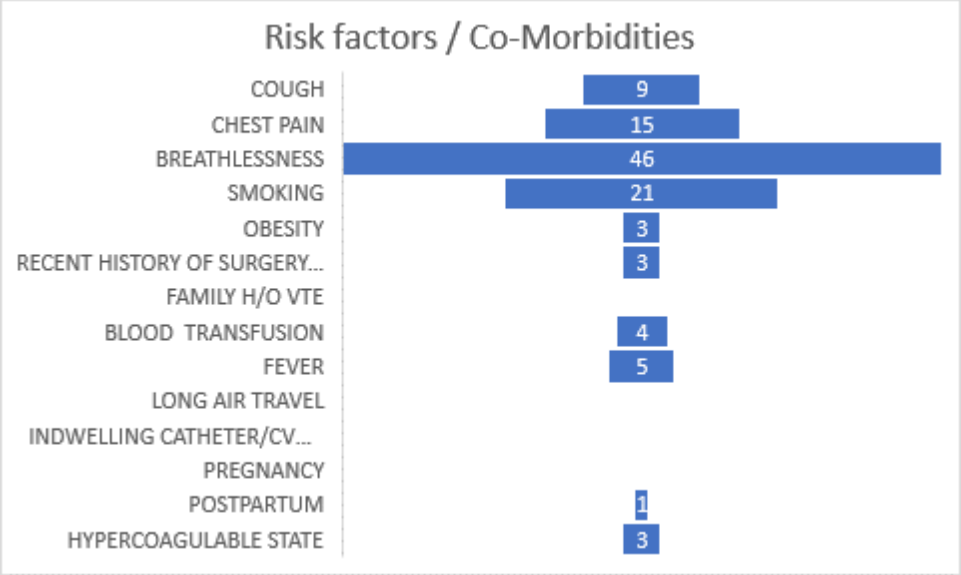


Among our study group, majority of patients were daily wage labourers (74%).

2% of patients were police officers, 2% were retired persons and 14% were home makers respectively.

IV DISTRIBUTION OF CO-MORBIDITIES/RISK FACTORS AMONG OUR STUDY GROUP

Risk factors / Co-Morbidities	No of cases
COUGH	9
CHEST PAIN	15
BREATHLESSNESS	46
SMOKING	21
OBESITY	3
RECENT HISTORY OF SURGERY/IMMOBILISATION	3
FAMILY H/O VTE	0
BLOOD TRANSFUSION	4
FEVER	5
LONG AIR TRAVEL	0
INDWELLING CATHETER/CV LINE	0
PREGNANCY	0
POSTPARTUM	1
HYPERCOAGULABLE STATE	3



Among our study population, Smoking was the most common co-morbidity (21 patients) present.

History of prolonged fever was present in 5 patients as co-morbidity among our study group.

History of blood transfusion was seen among 4 patients of our study group. Obesity, recent history of surgey and immobilization, Hypercoagulable states were seen as risk factor among 3 patients of our study group respectively. Postpartum was seen as risk factor in 1 patient respectively.

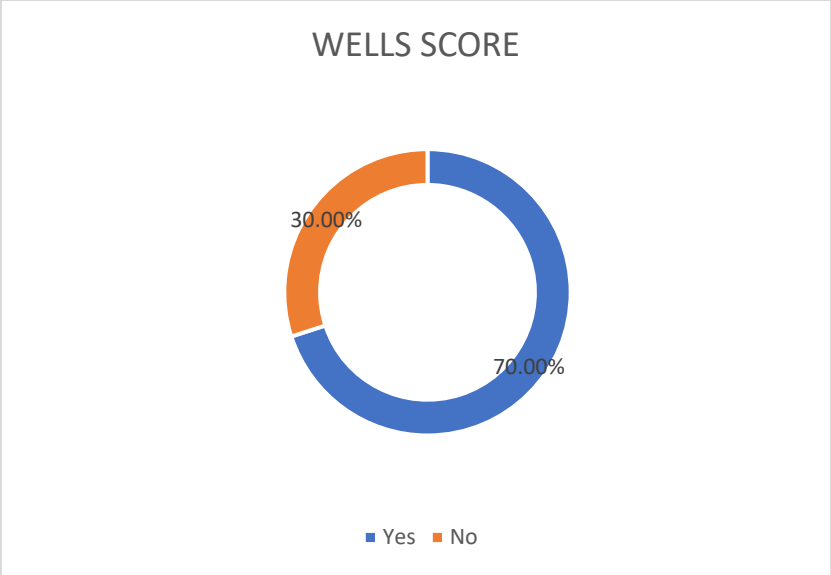
V DISTRIBUTION OF SYMPTOMS AMONG OUR STUDY GROUP

Breathlessness was the most common symptom prevalent among our study group (46%) .

Chest pain was seen in 15% and cough in 9% of patients of our study group.

VI DISTRIBUTION OF WELL'S SCORE AMONG OUR STUDY GROUP

WELLS SCORE	No of cases	Percentage
Yes	35	70.00%
No	15	30.00%
Grand Total	**50**	**100.00%**

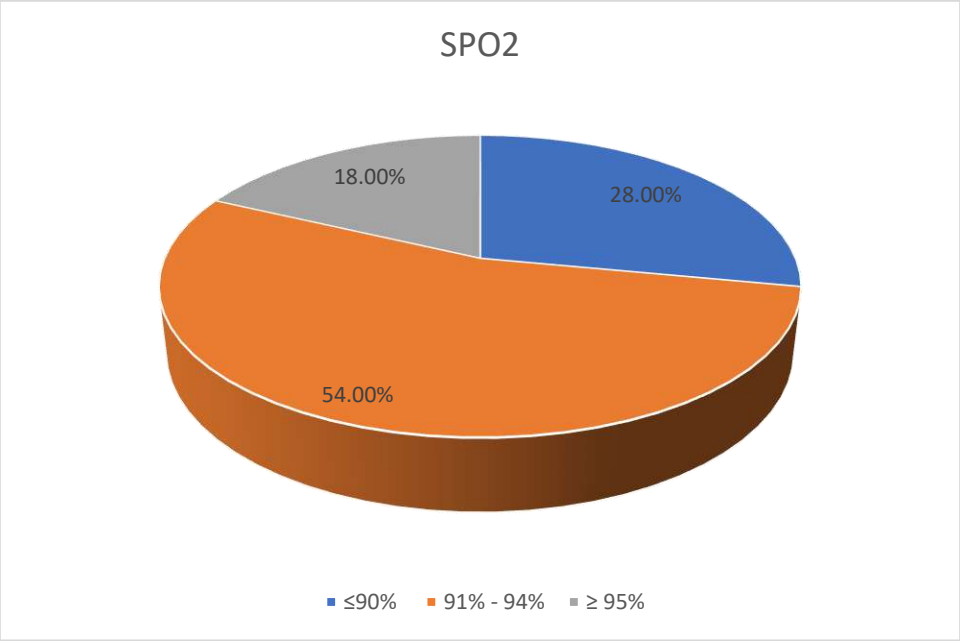


Among our study group, WELLS score is suggestive of VTE among 70% of patients.

Whereas WELL's score is not predictive of VTE among 30% of patients.

VII DISTRIBUTION OF HYPOXEMIA AMONG OUR STUDY GROUP

SPO2	No of cases	Percentage
≤90%	14	28.00%
91% - 94%	27	54.00%
≥ 95%	9	18.00%
Grand Total	**50**	**100.00%**



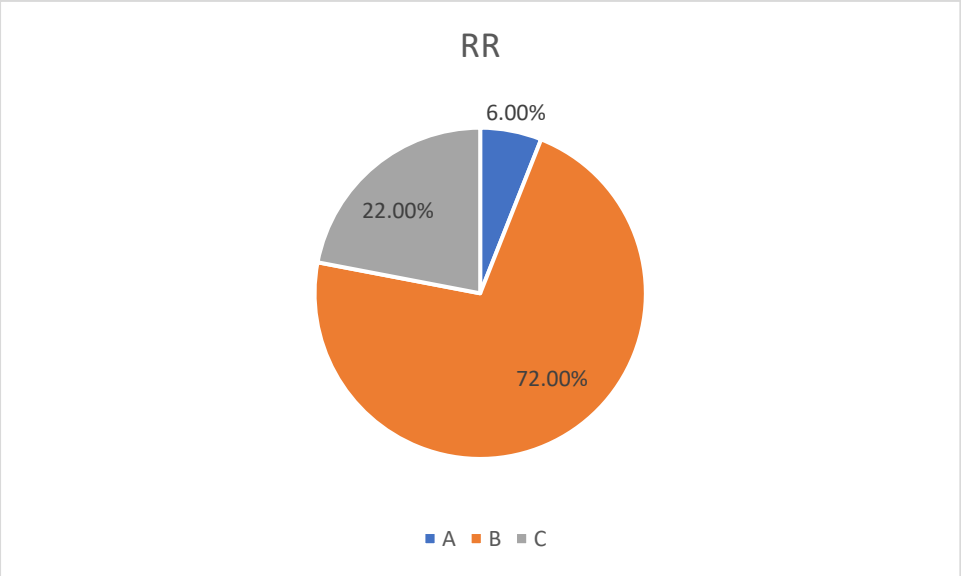
Among our study group, hypoxemia < 90% were seen among 28% of our patients.

Hypoxemia with Spo2 of 91-94% was more common , being seen in 54% of our population

Normal oxygen saturation of spo2 of >95% were seen in 18% of our study group.

VIII DISTRIBUTION OF RESPIRATORY RATE AMONG OUR STUDY GROUP

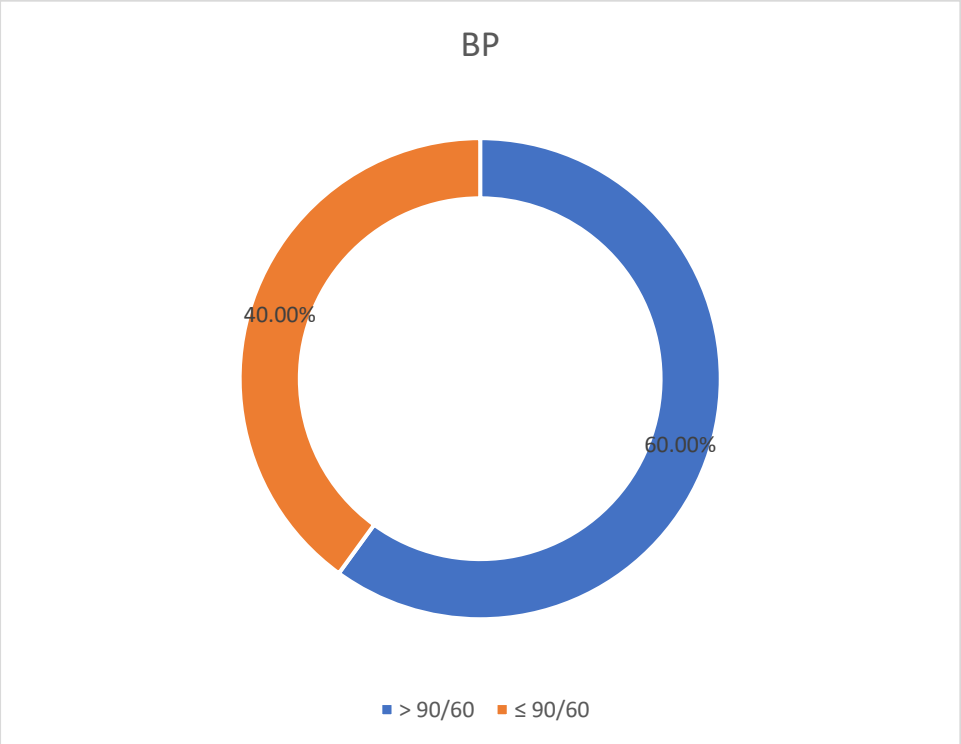
RR	No of cases	Percentage
A	3	6.00%
B	36	72.00%
C	11	22.00%
Grand Total	**50**	**100.00%**



Tachypnoea was seen in 94% of patients , with 72% having respiratory rate in range of 22-30/min, whereas 22% of patients had respiratory rate > 30/min. 6% of patients had normal respiratory rate < 22/min in our study group.

IX DISTRIBUTION OF BLOOD PRESSURE AMONG OUR STUDY GROUP

BP	No of cases	Percentage
> 90/60	30	60.00%
≤ 90/60	20	40.00%
Grand Total	**50**	**100.00%**

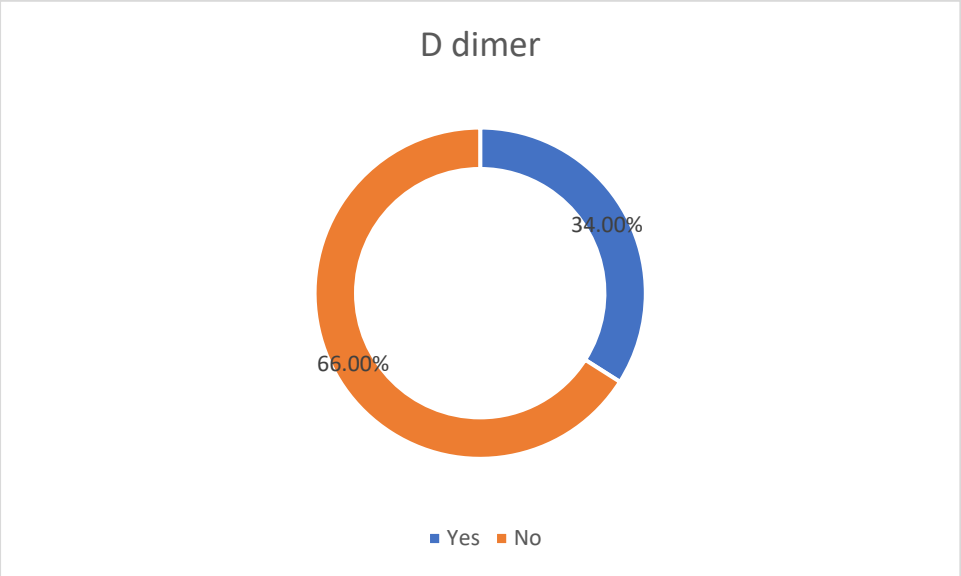


Hypotension was prevalent among 40% of patients in our study group (<90/60 mmHg)

Normal Blood pressure was seen among 60% of patients among our study group.

X DISTRIBUTION OF D-DIMER AMONG OUR STUDY GROUP

D dimer	No of cases	Percentage
Yes	17	34.00%
No	33	66.00%
Grand Total	**50**	**100.00%**

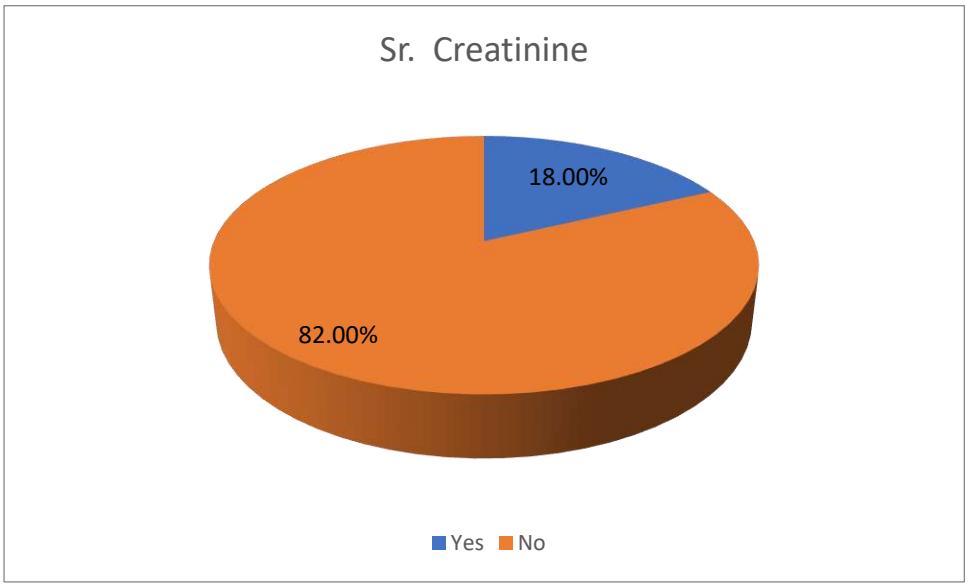


Among our study population, elevated D-dimer values were seen among 34% of patients.

Whereas normal D-dimer values are seen among 66% of our population.

XI DISTRIBUTION OF CREATININE VALUES AMONG OUR STUDY GROUP

Sr. Creatinine	No of cases	Percentage
Yes	9	18.00%
No	41	82.00%
Grand Total	**50**	**100.00%**

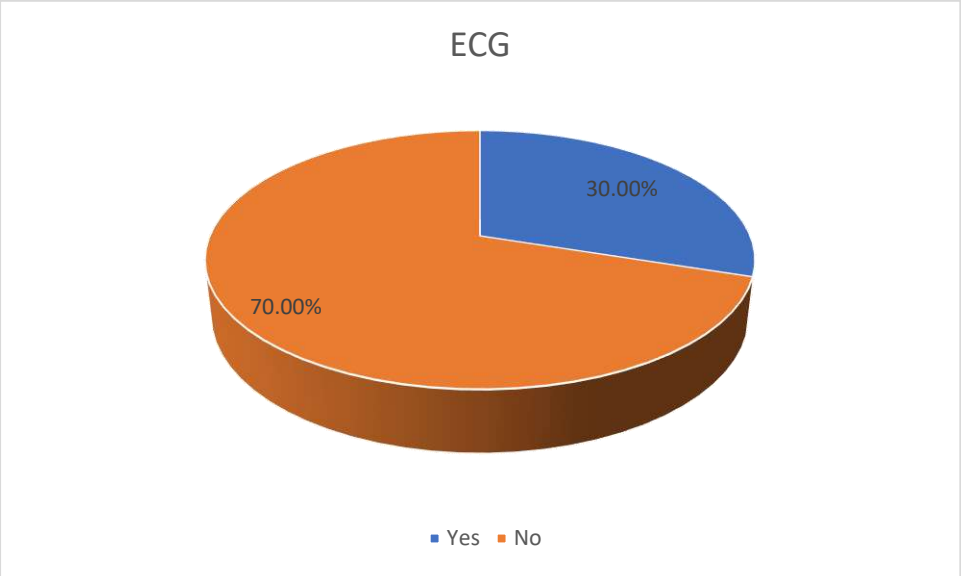


Elevated creatinine values were seen among 18% of our study population only.

82% of patients displayed normal serum creatinine levels among our study population.

XII DISTRIBUTION OF ECG FINDINGS AMONG OUR STUDY GROUP

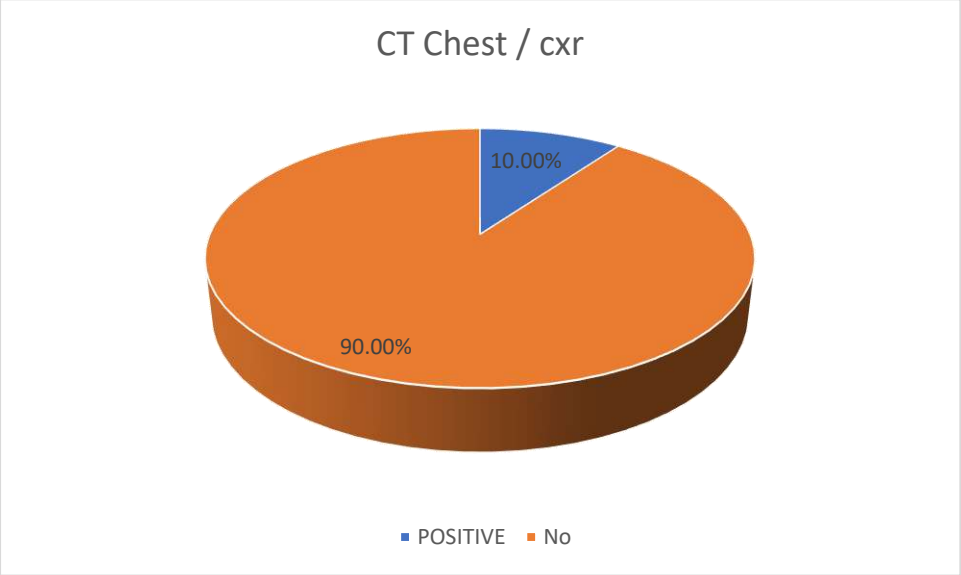
ECG	No of cases	Percentage
Yes	15	30.00%
No	35	70.00%
Grand Total	**50**	**100.00%**



Significant ECG findings which were prevalent/supportive of diagnosis of pulmonary embolism were seen among 30% of our population Normal ECG was seen in 70% of our population.

XIII DISTRIBUTION OF X-RAY/CT CHEST FINDINGS AMONG OUR STUDY GROUP

CT Chest / cxr	No of cases	Percentage
POSITIVE	5	10.00%
No	45	90.00%
Grand Total	**50**	**100.00%**

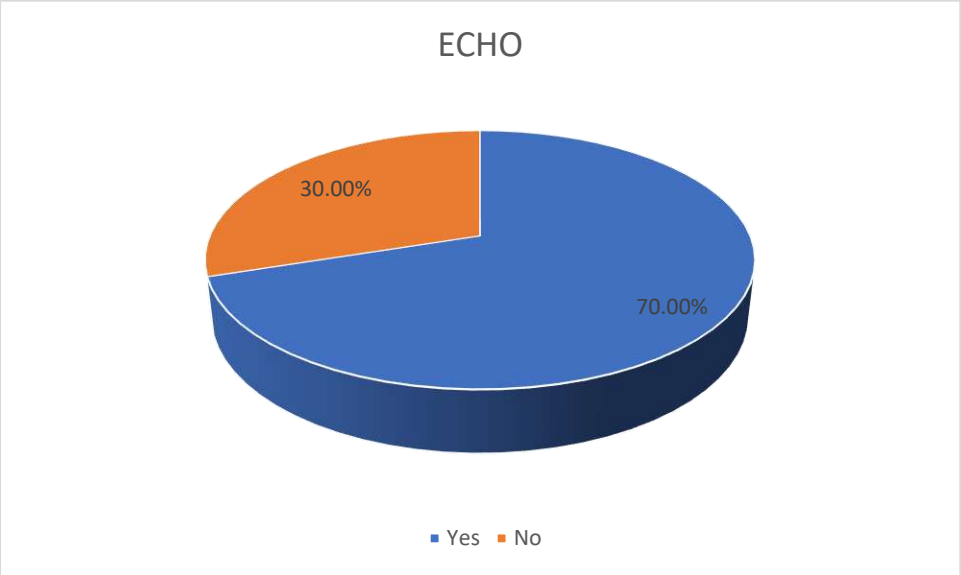


Positive findings suggestive of Pulmonary embolism were seen in Chest X-ray or CT chest among 10% of our study population only.

90% of our study population exhibited normal Chest X-ray and CT chest.

XIV DISTRIBUTION OF ECHOCARDIOGRAM FINDINGS AMONG OUR STUDY GROUP

ECHO	No of cases	Percentage
Yes	35	70.00%
No	15	30.00%
Grand Total	**50**	**100.00%**

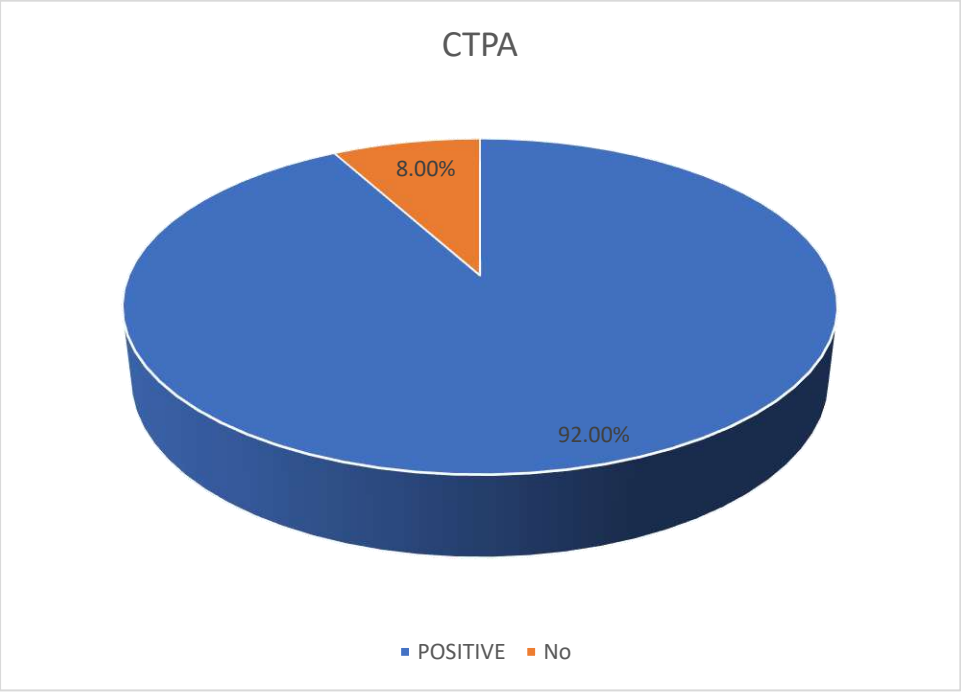


Positive findings suggestive of Pulmonary embolism were seen among 70% of patients in echocardiogram among our study population

Whereas Normal Echocardiogram was seen in 30% of our population

XV DISTRIBUTION OF CT PULMONARY ANGIOGRAM AMONG OUR STUDY POPULATION

CTPA	No of cases	Percentage
POSITIVE	46	92.00%
No	4	8.00%
Grand Total	**50**	**100.00%**

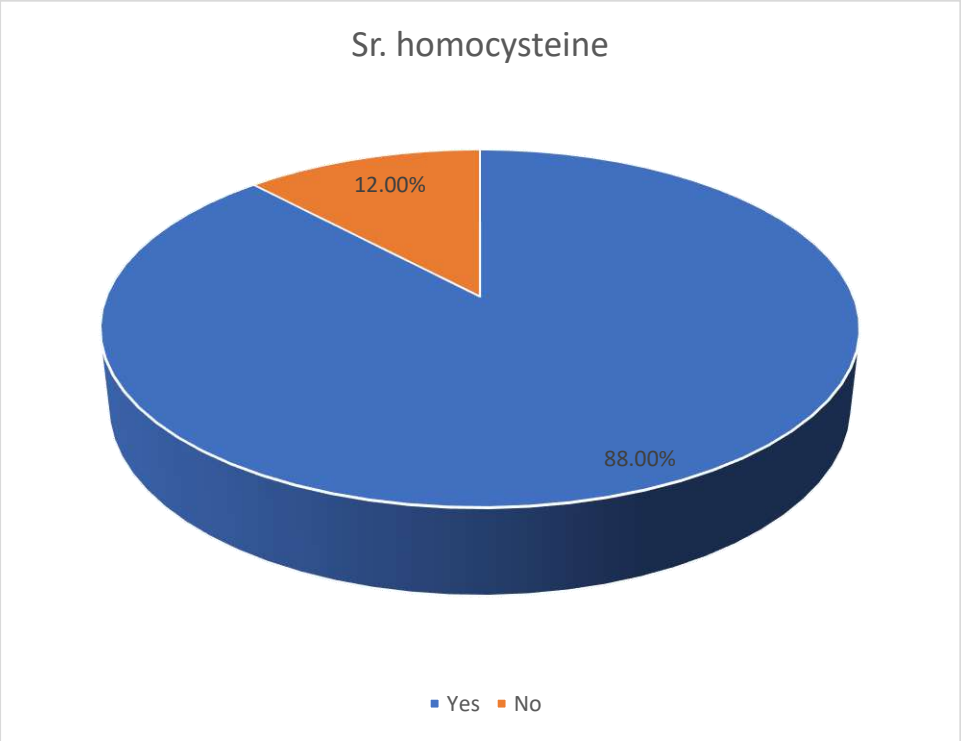


Positive findings suggestive of Pulmonary embolism were seen in 92% of patients in CT pulmonary angiogram among our study population.

Normal CT pulmonary angiogram was seen in only 8% of patients of our study group.

XVI DISTRIBUTION OF SERUM HOMOCYSTEINE LEVELS AMONG OUR STUDY GROUP

Sr. homocysteine	No of cases	Percentage
Yes	44	88.00%
No	6	12.00%
Grand Total	**50**	**100.00%**

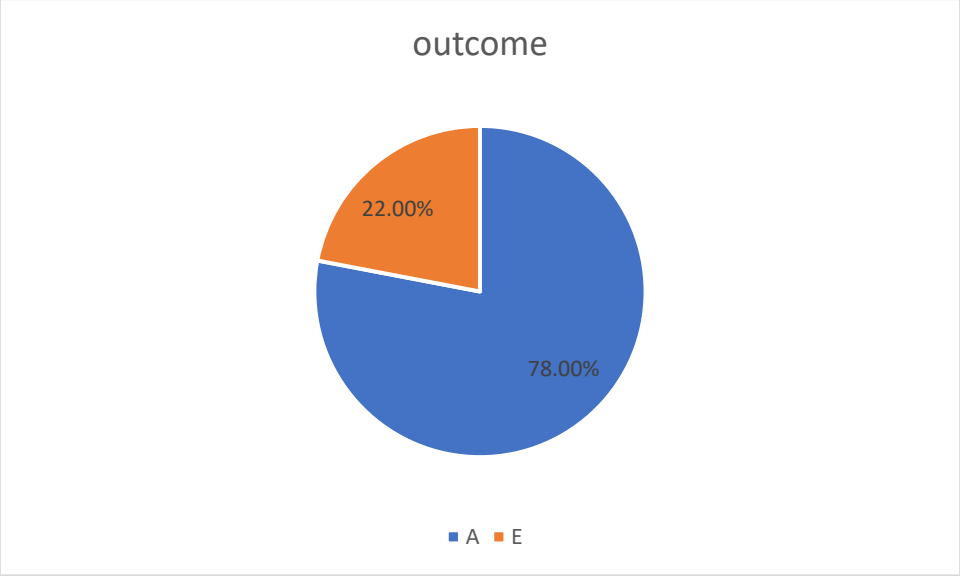


Elevated serum homocysteine levels were seen among 88% of our population.

Whereas normal homocysteine levels were seen in 12% of our study population.

XVII DISTRIBUTION OF OUTCOME AMONG OUR STUDY GROUP

outcome	No of cases	Percentage
A	39	78.00%
E	11	22.00%
Grand Total	**50**	**100.00%**

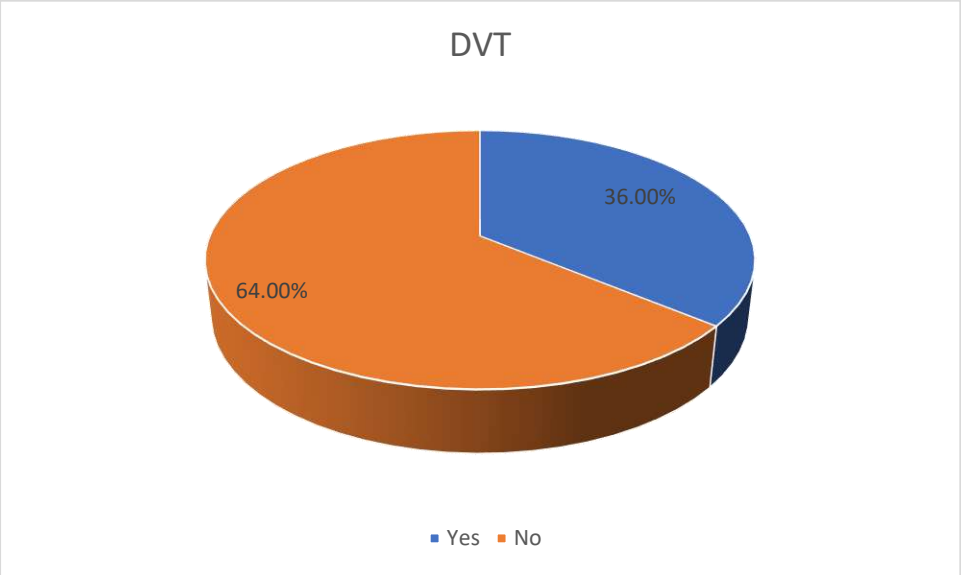


78% of our study population recovered and got discharged.

Whereas 22% of patients had worst outcome of death among our study population.

XVIII DISTRIBUTION OF DEEP VEIN THROMBOSIS AMONG OUR STUDY GROUP

DVT	No of cases	Percentage
Yes	18	36.00%
No	32	64.00%
Grand Total	**50**	**100.00%**

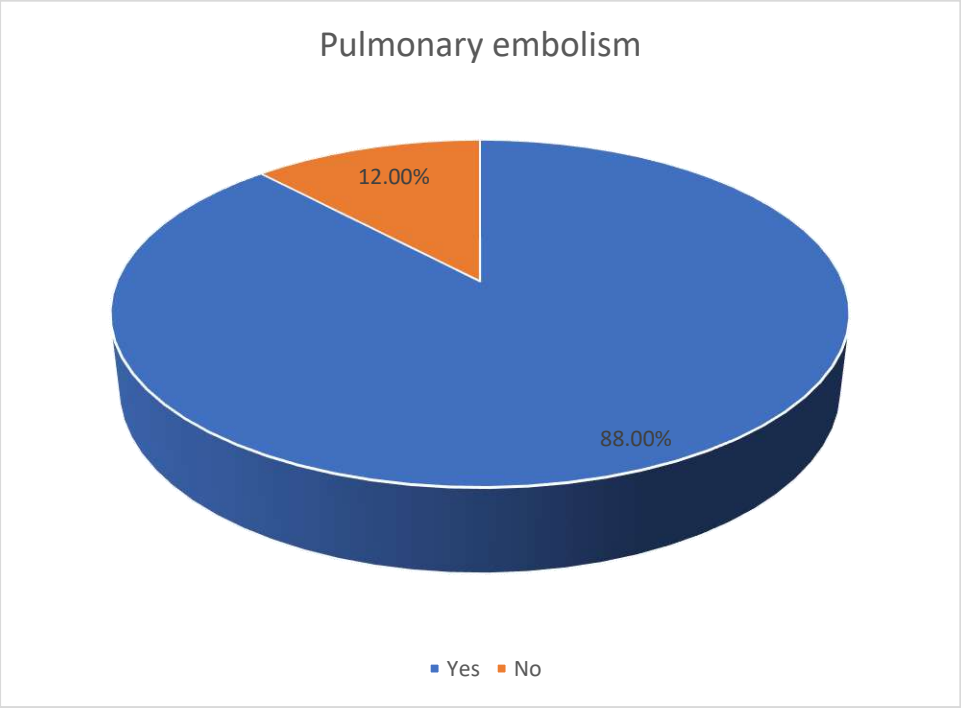


Among our study population, deep vein thrombosis was seen in 36% of our study population.

Prevalance of DVT among our study group is therefore 36%

XIX DISTRIBUTION OF PULMONARY EMBOLISM AMONG OUR STUDY GROUP

Pulmonary embolism	No of cases	Percentage
Yes	44	88.00%
No	6	12.00%
Grand Total	**50**	**100.00%**

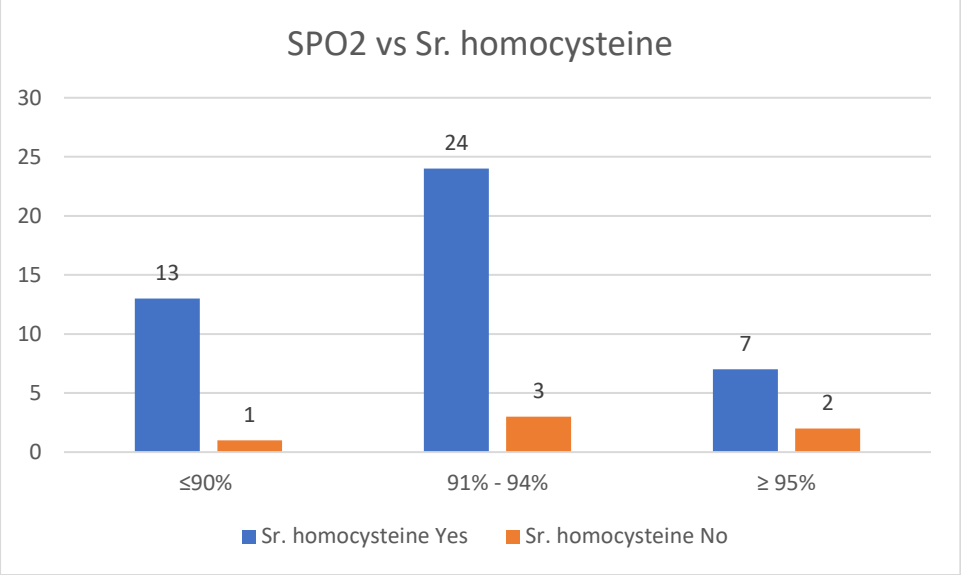


Pulmonary embolism was seen as diagnosis in 88% of our study population, hence prevalence of Pulmonary embolism among our study group was 88%.

COMPARATIVE STATISTICS

XX COMPARISON OF SERUM HOMOCYSTEINE LEVELS vs HYPOXEMIA AMONG OUR STUDY GROUP

SPO2	Sr. Homocysteine		Grand Total
	Yes	**No**	**Grand Total**
≤90%	13	1	14
91% - 94%	24	3	27
≥ 95%	7	2	9
Grand Total	**44**	**6**	**50**
Chi-Square = 1.22354			
Degrees of Freedom = 2			
p = 0.5423			



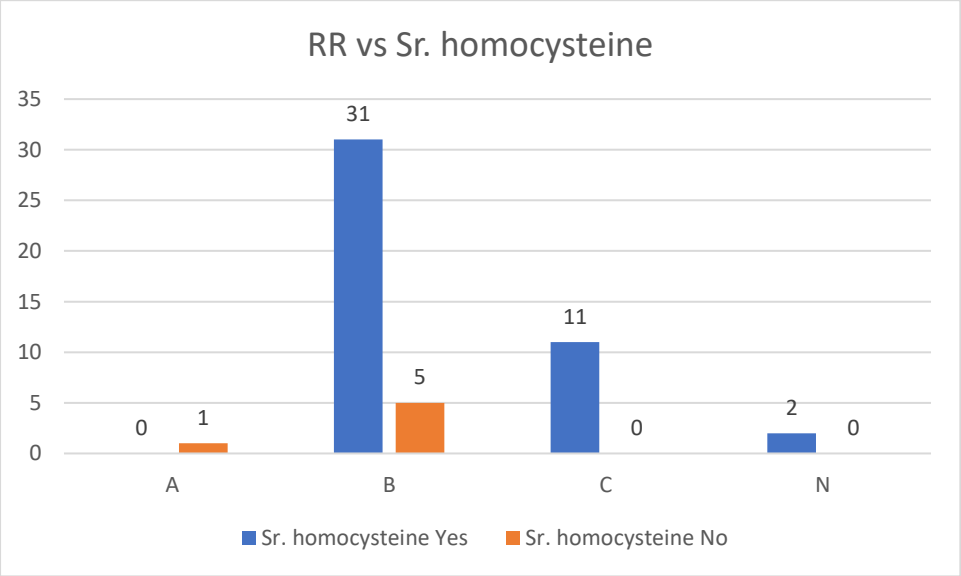
Among our study group, 90.2% (37 out of 41) of patients with hypoxemia had elevated homocysteine levels .

Whereas only 77.77% of patients with normal saturation levels had elevated homocysteine levels (7 out of 9)

However , this association of hypoxemia with elevated homocysteine levels does not have statistical correlation (p value 0.54).

XXI COMPARISON OF TACHYPNOEA vs SERUM HOMOCYSTEINE LEVELS AMONG OUR STUDY GROUP

RR	Sr. Homocysteine		Grand Total
	Yes	**No**	**Grand Total**
A	2	1	3
B	31	5	36
C	11	0	11
Grand Total	**44**	**6**	**50**
Chi-Square = 9.22769			
Degrees of Freedom = 3			
p = 0.02641			



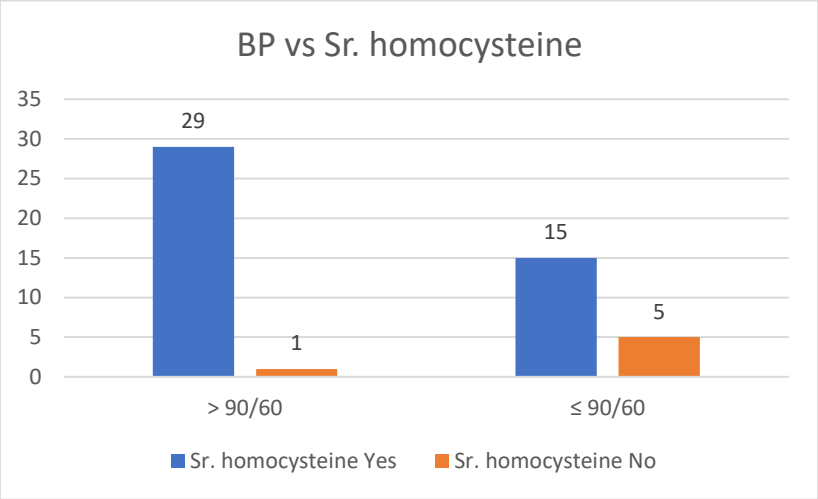
Among our study population, 89.3% (42 out of 47) of patients with tachypnoea had elevated serum homocysteine levels.

However, only 66.6% (2 out 3) of patients with normal respiratory rate had elevated serum homocysteine levels.

And this association of elevated serum homocysteine levels with tachypnoea carries statistical significance with a p value of 0.026 .

XXII COMPARISON OF HYPOTENSION vs ELEVATED SERUM HOMOCYSTEINE LEVELS AMONG OUR STUDY POPULATION

BP	Sr. Homocysteine		Grand Total
	Yes	**No**	**Grand Total**
> 90/60	29	1	30
≤ 90/60	15	5	20
Grand Total	**44**	**6**	**50**
Chi-Square = 5.3346			
Degrees of Freedom = 1			
p = 0.0209			

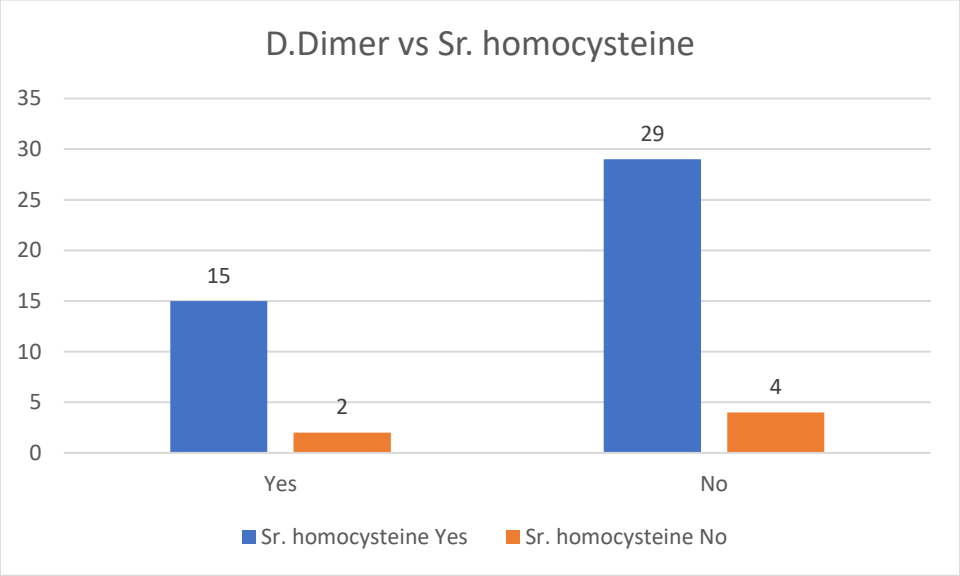


Among our study population , 75% (15 out of 20) of patients with hypotension had elevated serum homocysteine levels.

However, 96.6% (29 out of 30) of patients with normal pressure also showed elevated serum homocysteine levels.

XXIII COMPARISON OF SERUM HOMOCYSTEINE LEVELS vs D-DIMER LEVELS AMONG OUR STUDY GROUP

D Dimer	Sr. Homocysteine		Grand Total
	Yes	**No**	**Grand Total**
Yes	15	2	17
No	29	4	33
Grand Total	**44**	**6**	**50**
Chi-Square = 0.0013504			
Degrees of Freedom = 1			
p = 0.970			



Among our study group, 88% of patients (15 out of 17) with elevated D-Dimer levels had elevated serum homocysteine levels also.

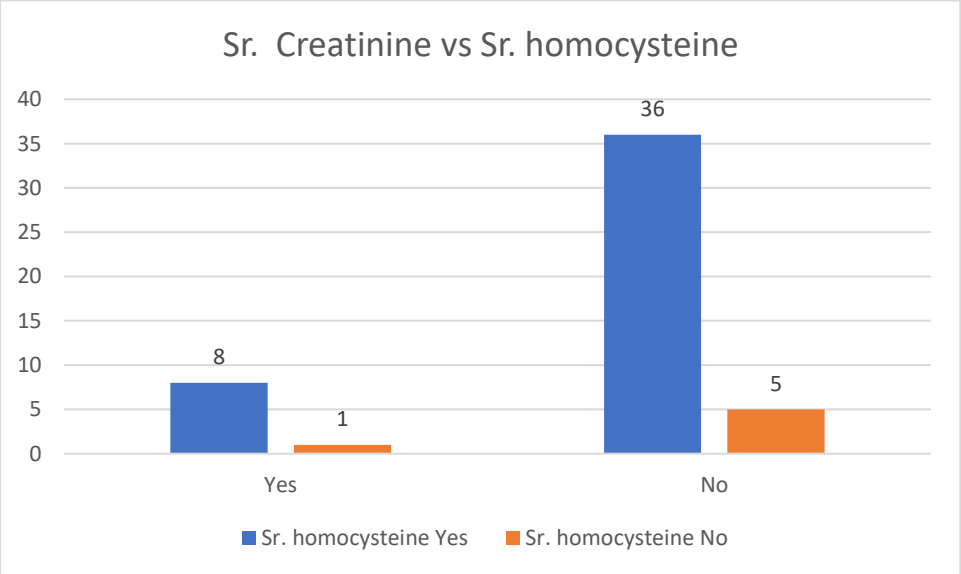
Also, 87.8% of patients (29 out of 33) had elevated serum homocysteine levels in the absence of elevation of D-dimer levels.

This mild association of elevated D-dimer and elevated serum homocysteine levels does not show any statistical correlation (p value 0.970).

Prevalance of both D-dimer and homocysteine elevation among our study population was 30%.

XXIV COMPARISON OF SERUM HOMOCYSTEINE LEVELS vs SERUM CREATININE LEVELS AMONG OUR STUDY GROUP

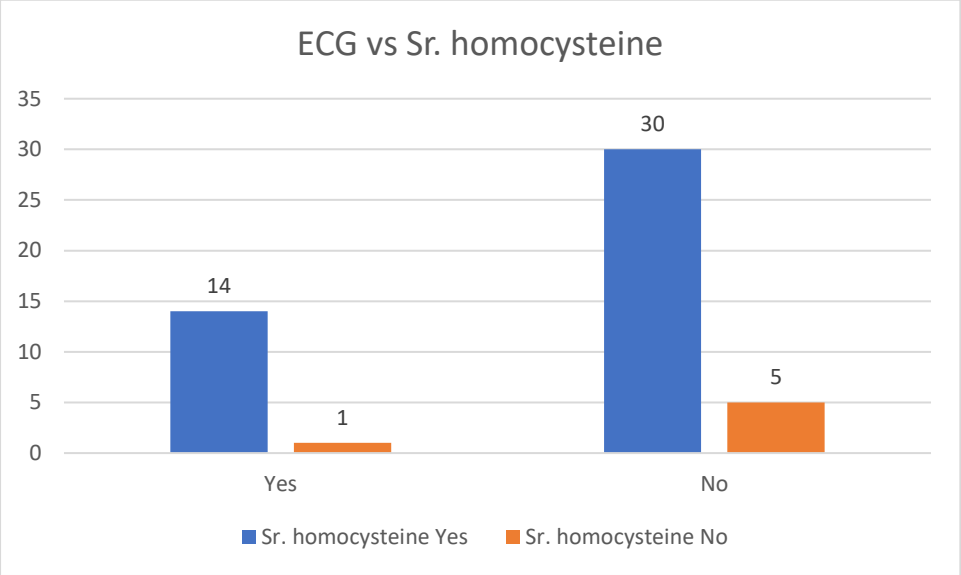
Sr. Creatinine	Sr. Homocysteine		Grand Total
	Yes	**No**	**Grand Total**
Yes	8	1	9
No	36	5	41
Grand Total	**44**	**6**	**50**
Chi-Square = 0.0082122			
Degrees of Freedom = 1			
p = 0.927			



Correlation of elevated homocysteine levels with elevated serum creatinine levels does not show any statistical correlation.

XXV COMPARISON OF ECG FINDINGS vs SERUM HOMOCYSTEINE LEVELS AMONG OUR STUDY GROUP

ECG	Sr. homocysteine		Grand Total
	Yes	**No**	**Grand Total**
Yes	14	1	15
No	30	5	35
Grand Total	**44**	**6**	**50**
Chi-Square = 0.577201			
Degrees of Freedom = 1			
p = 0.447			



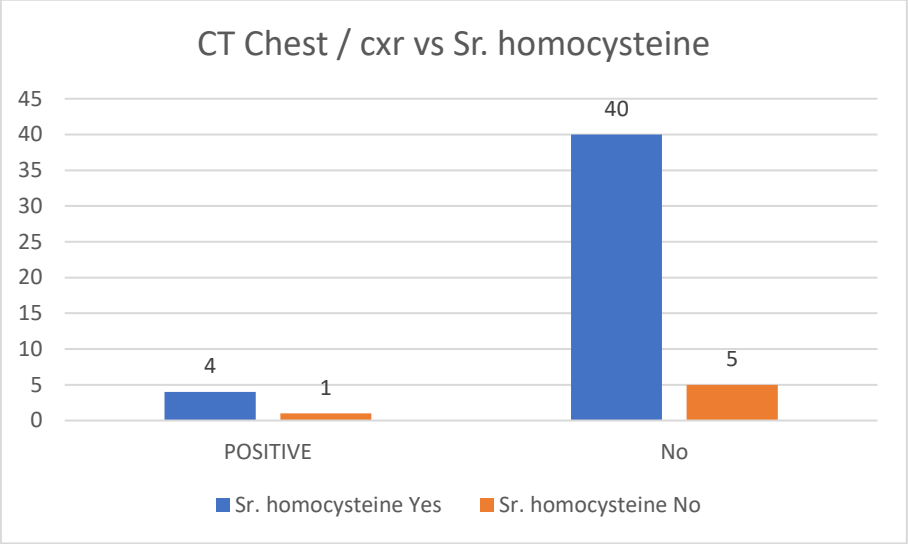
Among patients with positive ECG findings, elevated serum homocysteine levels were seen in 93.3% patients.

85.7% of patients with normal ECG also show elevated serum homocysteine levels among our study population.

However, this association does not has statistical significance. (p value 0.447)

XXVI COMPARISON OF CT CHEST vs SERUM HOMOCYSTEINE LEVELS AMONG OUR STUDY GROUP

CT Chest / cxr	Sr. Homocysteine		Grand Total
	Yes	**No**	**Grand Total**
POSITIVE	4	1	5
No	40	5	45
Grand Total	**44**	**6**	**50**
Chi-Square = 0.3367			
Degrees of Freedom = 1			
p = 0.561			



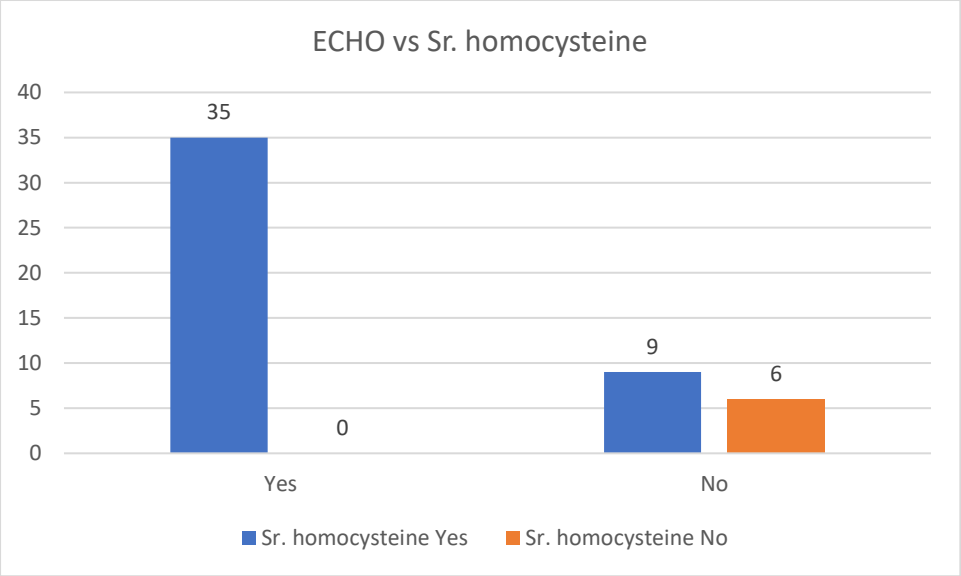
80% of patients (4 out of 5) with positive CT chest findings had elevated serum homocysteine levels.

88% of patients with normal CT chest had elevated serum homocysteine levels.

There is no statistical correlation between CT chest findings and elevated serum homocysteine levels.

XXVII COMPARISON OF ECHO FINDINGS vs SERUM HOMOCYSTEINE LEVELS AMONG OUR STUDY GROUP

ECHO	Sr. Homocysteine		Grand Total
	Yes	**No**	**Grand Total**
Yes	35	0	35
No	9	6	15
Grand Total	**44**	**6**	**50**
Chi-Square = 15.9091			
Degrees of Freedom = 1			
p = 0.00006			



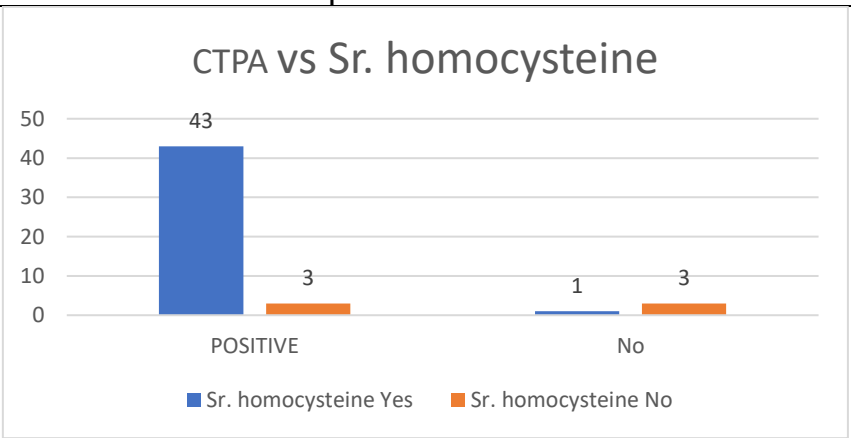
100% of patients who had positive echocardiographic features suggestive of pulmonary embolism had elevated serum homocysteine levels.

Only 60% (9 out of 15) of patients with normal echocardiogram had elevated serum homocysteine levels among our study population.

And this association of positive echocardiographic findings and elevated serum homocysteine levels had strong statistical correlation (p value 0.00006).

XXVIII COMPARISON OF CT PULMONARY ANGIOGRAM vs SERUM HOMOCYSTEINE LEVELS AMONG OUR STUDY GROUP

CTPA	Sr. Homocysteine		Grand Total
	Yes	**No**	**Grand Total**
POSITIVE	43	3	46
No	1	3	4
Grand Total	**44**	**6**	**50**
Chi-Square = 16.3414			
Degrees of Freedom = 1			
p = 0.000052			



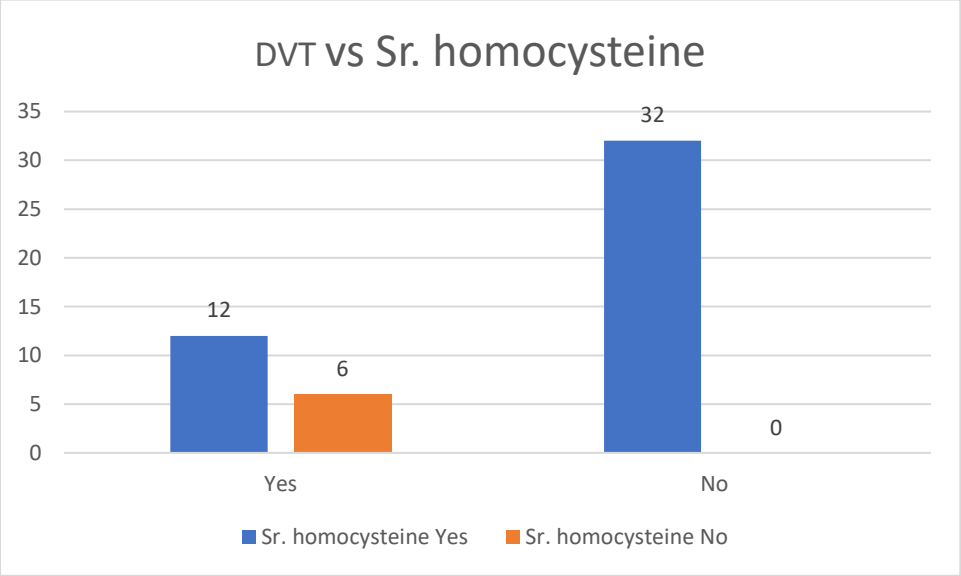
Among patients having CT pulmonary angiogram findings suggestive of pulmonary embolism, 93.47% of patients (43 out of 46) had elevated serum homocysteine levels.

Whereas, only 25% of patients with normal CT pulmonary angiogram had elevated serum homocysteine levels among our study population.

And this association of elevated serum homocysteine levels and positive CT pulmonary angiogram for pulmonary embolism had strong statistical correlation with significance (p value 0.000052).

XXIX COMPARISON OF DEEP VEIN THROMBOSIS vs SERUM HOMOCYSTEINE LEVELS AMONG OUR STUDY GROUP

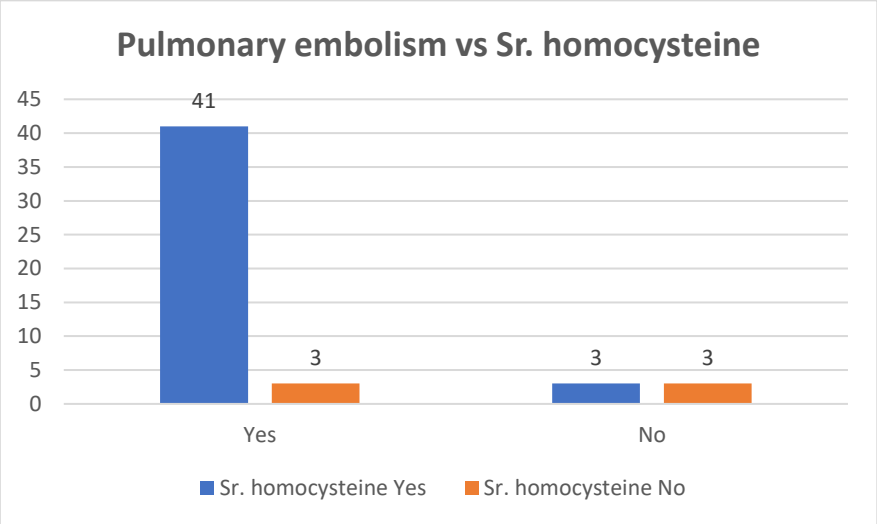
DVT	Sr. Homocysteine		Grand Total
	Yes	**No**	**Grand Total**
Yes	12	6	18
No	32	0	32
Grand Total	**44**	**6**	**50**
Chi-Square = 12.1212			
Degrees of Freedom = 1			
p = 0.00049			



Among our DVT patients , 66.66% (12 out of 18) had elevated homocysteine levels.

XXX COMPARISON OF PULMONARY EMBOLISM vs SERUM HOMOCYSTEINE LEVELS

Pulmonary embolism	Sr. Homocysteine		Grand Total
	Yes	**No**	**Grand Total**
Yes	41	3	44
No	3	3	6
Grand Total	**44**	**6**	**50**
Chi-Square = 9.32335			
Degrees of Freedom = 1			
p = 0.0022			

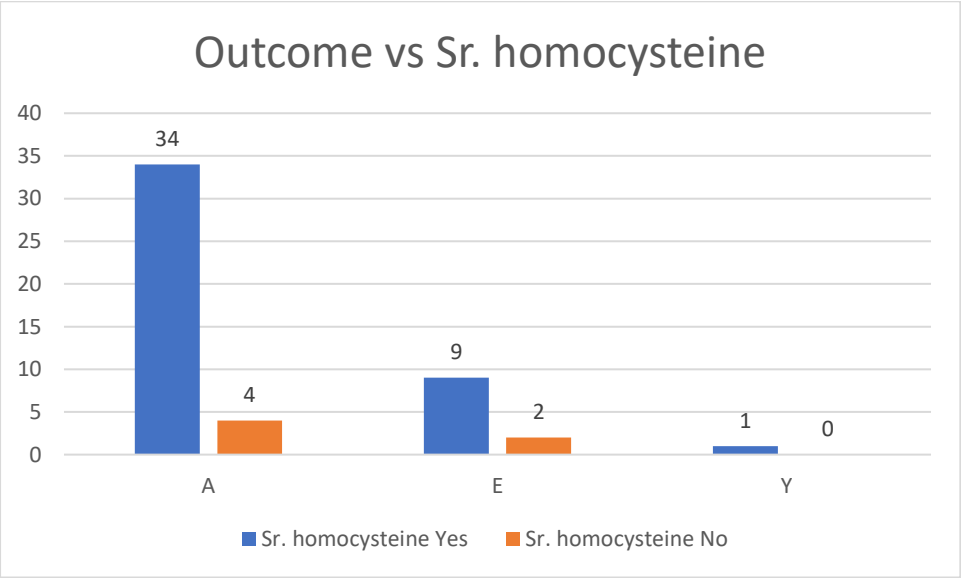


Among our pulmonary embolism patients, 93.18% of patients had elevated serum homocysteine levels.

And this association has strong statistical significance with a p value of 0.0022.

XXXI COMPARISON OF SERUM HOMOCYSTEINE LEVELS vs OUTCOME AMONG OUR STUDY GROUP

outcome	Sr. homocysteine		Grand Total
	Yes	**No**	**Grand Total**
A	34	4	38
E	9	2	11
Y	1	0	1
Grand Total	**44**	**6**	**50**
Chi-Square = 0.612585			
Degrees of Freedom = 2			
p = 0.736			



Among patients who recovered from venous thromboembolism, 35 out of 39(89.7%) had elevated serum homocysteine levels.

However, among patients who died due to venous thromboembolism, 9 out of 11(81.8%) had elevated serum homocysteine levels.

And there is no statistical correlation (p value 0.736).

DISCUSSION

Venous thromboembolism particularly pulmonary embolism is considered as a medical emergency among ICU patients. However, clinical manifestations of VTE are diverse and nonspecific making the diagnosis challenging. More promising markers are needed for early diagnosis. Serum homocysteine plays important role in pathogenesis of venous thromboembolism. However its role in diagnosis and assessment of prognosis in VTE , particularly pulmonary embolism needs large scale and in detail studies. Our study analysis the prevalence of elevated homocysteine levels among our venous thromboembolism patients and compares with other diagnostic parameters and outcome.

DESCRIPTION OF PATIENT CHARACTERISTICS

Mean age group of all patients in our study group was 52.4 and majority belongs to 45-60 years of age (66%). This age distribution was similar to study by Kokturk et al which has mean age of all patients as 54 years. Males were more common than female in a ratio of 4.5 :1 among our study group, whereas in study by kokturk et al VTE is more common in females (60%). Most of the patients in our study group were labourers and most common symptomatology prevalent among our population is breathlessness (46 patients). Similarly in studies by kokturk et al also, breathlessness is the most common presenting symptom accounting to be seen in 60% of patients.

Among the risk factors prevalent among our study group, most common is smoking (42%). However, in studies by Kokturk et al, smoking was seen only in 28% of patients. Other risk factors seen in our study group and its comparison with Kokturk study is as follows.

RISK FACTOR	OUR STUDY	KOKTURK et al
Smoking	**42%**	**28%**
Obesity	**6%**	**30%**
Recent surgery/Immobilisation	**6%**	**31%**
Family History of VTE	**0**	**12%**
Blood Transfusion	**8%**	**0**
Fever	**10%**	**0**
Long air Travel	**0**	**19%**
Central line	**0**	**0**
Pregnancy	**0**	**1%**
Postpartum	**2%**	**2%**
Hypercoagulable state	**6%**	**0**

- **DESCRIPTION OF CLINICAL VARIABLES & LABORATORY PARAMETERS**

WELL's score predictive of venous thromboembolism was seen in only 30% of population. Hypoxemia was seen in 72% of patients and tachypnoea being prevalent in 94% of our study population. Hypotension was less common and seen in 40% of our study group.

Among laboratory parameters, D-dimer was elevated in 34%, elevated creatinine seen in 18% among our study population.

PARAMETERS	PREVALANCE AMONG VTE PATIENTS – OUR STUDY
WELL's score	**30%**
Hypoxemia	**72%**
Tachypnoea	**94%**
Hypotension	**40%**
D-dimer	**34%**
Serum creatinine	**18%**

Among these, tachypnoea was the most prevalent and elevated serum creatinine levels were least prevalent among our patients of VTE . D-dimer levels were elevated in 34% of our VTE patients which is much lower proportion when compared to study by Kokturk et al in which D-dimer elevation prevalence is 80% among VTE patients.

ECG findings suggestive or predictive of pulmonary embolism was seen in 30% of our study group. This is in contrast to studies by Kokturk et al in which 51% of VTE patients had positive ECG findings.

- **DESCRIPTION OF DIAGNOSTIC IMAGING MODALITIES AMONG VTE**

CTchest /Chest X-ray suggestive of pulmonary embolism was seen in only 10% of our population. This yield is much lower than study by kokturk et al in which some abnormal findings were seen in CT /X-ray among 60% of VTE patients.

Echocardiography was abnormal in 70% of our population. Echocardiogram was mostly not used /added in many studies. CT pulmonary angiogram was used as gold standard diagnostic modality in our study group whereas in study by Kokturk et al perfusion scintigraphy was used as gold standard diagnostic modality. 92% of patients in our study group had positive findings in CT pulmonary angiography, whereas 84% of patients yielded high probability results in perfusion scintigraphy in study by kokturk et al

PARAMETER	OUR STUDY - PREVALANCE	Kokturk et al – PREVALANCE
D-dimer	**34%**	**80%**
ECG	**30%**	**51%**
CTchest/Chest X-ray	**10%**	**60%**
Echocardiography	**70%**	**-**
CT pulmonary angiography/ Perfusion scintigraphy	**92%**	**84%**

- **SERUM HOMOCYSTEINE vs VTE**

In our study, prevalence of elevated homocysteine level was 88%. This is much higher than study by kokturk et al in which prevalence of elevated omocysteine levels were 63%.

STUDY	ELEVATED HOMOCYSTEINE LEVELS – PREVALANCE
Our study	88%
Kokturk et al	63%
Eichinger et al	7.9%
Ducros et al	25%

Our results of elevated homocysteine levels in VTE patients (88%) were much higher than other studies.Among our study population, DVT was seen in 36% and pulmonary embolism was seen in 88% of population. In our study, mortality is higher accounting to 22% of population.

COMPARATIVE STATISTICS - DESCRIPTION

We compared prevalence of elevated serum homocysteine levels in VTE patients with clinical variables, laboratory findings and imaging modalities, anxillary tests, and with the outcome. Association of these variables and prevalence of elevated homocysteine were analysed. Elevated homocysteine levels were more

prevalent among patients with hypoxemia, tachypnoea and hypotension of VTE group. However, association of elevated homocysteine levels being more common patients with tachypnoea in VTE showed statistical significance.

Similarly, elevated homocysteine were more commonly seen in patients with elevated D-dimer, positive ECG and CTchest/chest x-ray findings. All patients who had echocardiographic findings suggestive of pulmonary embolism had elevated homocysteine levels with statistical significance. Also, around 93.18% of patients with CT pulmonary angiogram positive finding had elevated serum homocysteine levels with statistical significance.

PARAMETERS	ELEVATED HOMOCYSTEINE – PREVALANCE – COMPARISON PERCENTAGE – OUR STUDY	SIGNIFICANCE – OUR STUDY	P value – OUR STUDY
Hypoxemia	90.2%	Insignificant	0.54
Tachypnoea	89.3%	Significant	0.02
Hypotension	75%	Insignificant	0.02

D-Dimer	88%	Insignificant	0.97
ECG	93.3%	InSignificant	0.44
CT/Chest X-ray	80%	Insignificant	0.56
ECHO	100%	Significant	0.00006
CT pulmonary angiogram	93.47%	Significant	0.000052

Importantly, Pulmonary embolism patients had more commonly elevated homocysteine levels (93.18%) than DVT patients in our study group.(66.66%) And this association had statistical significance. This is also comparable with studies by kokturk et al which also homocysteine elevation more commonly seen in PE than DVT. This is contrast to studies by Okumus et al, in which no difference exists between DVT and PE regarding homocysteine level elevation in serum.

However, elevated serum homocysteine levels does not correlate with the outcome of VTE such as recovery/death.

PARAMETERS	ELEVATED HOMOCYSTEINE – PREVALANCE – COMPARISON PERCENTAGE – OUR STUDY	SIGNIFICANCE – OUR STUDY	P value – our study
DVT	66.66%	0.00049	Significant
Pulmonary embolism	93.18%	0.0022	Significant
Outcome	89.7%	0.736	Insignificant

Hence, our study prevalence of serum homocysteine elevation in VTE was very high as 88% and its more commonly elevated in pulmonary embolism than deep vein thrombosis. Also elevated homocysteine levels significantly associated with clinical variable worsening like tachypnoea etc.., correlated well with other diagnostic modalities like ECHO and CT pulmonary angiogram.

Hence Elevated homocysteine levels can be considered as a efficient diagnostic tool in diagnosing VTE , particularly pulmonary embolism. However, serum homocysteine levels does not correlate with outcome in our study group.

CONCLUSION

Venous thromboembolism is often a diagnostic challenge , being always complemented by imaging modalities for confirmation of diagnosis. Early diagnostic markers are still needed for avoiding delay in initiation of treatment. Homocysteine can be considered as one such marker which is often found to be elevated in VTE patients in literature. Our study analysis its association with VTE and concludes the result as follows

- Prevalance of Elevated homocysteine levels among our study population of VTE patients was 88% (much higher than other studies in literature)
- Particularly, elevated homocysteine levels were more prevalent among pulmonary embolism patients (93.18%) than Deep vein thrombosis patients (66.66%) with strong statistical significance. Hence it can be considered as a more definite marker for Pulmonary embolism.
- Elevated Homocysteine levels correlate more commonly with tachypnoea than with hypoxemia and hypotension among clinical variables.
- Both D-dimer and Homocysteine were elevated in 30% of our VTE Patients
- Homocysteine were more commonly elevated than D-dimer among VTE patients in our study group, thus considering Homocysteine as a more sensitive marker to detect VTE

- Among imaging modalities, elevated homocysteine levels correlate well with positive echocardiographic findings and CT pulmonary angiogram than with ECG and CTc hest.
- However, Homocysteine levels does not correlate with outcome of VTE in our study group.

Hence, elevated homocysteine levels were considered as a more sensitive and efficacious marker for VTE, particularly for Pulmonary embolism than DVT. It is more sensitive marker than D-dimer. It complements and as efficacious as imaging modalities in picking up pulmonary embolism among VTE.

However, as it does not correlate with outcome , elevated homocysteine levels is not considered as a good prognostic marker among VTE patients. As estimation of serum homocysteine is easy than many imaging procedures,serum homocysteine can be considered as early screening test for patients suspected to have VTE. Screening by such faster tests helps us in early diagnosis and initiation of treatment which helps immensely in prevention of morbidity and mortality by VTE.

LIMITATIONS

- It is a single centered study conducted in a tertiary care centre with a small study population.
- False positive hyperhomocysteinemia due to vitamin deficiency and drug induced had not been excluded.

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PROFORMA

S.no

Name : Age: Sex:

Address:

Occupation:

Contact number:

IP No :

Symptoms :

1. Cough

2. Chest pain
3. Breathlessness
4. Leg pain
5. Diarrhoea/vomiting
6. Fever

Past History :

1. Recent H/O surgery
2. Recent H/O Immobilisation
3. H/O blood transfusion
4. Previous H/O Dvt or Pulmonary embolism
5. H/O Pacemaker/ indwelling central venous catheter

Personal H/O :

H/O smoking if present , duration and no of cigareetes/beedi per day

Travel History :

H/O Long haul air travel

Treatment H/O :

H/O OC pills/ HRT

General Examination :

GCS :

Systemic Examination :

VITALS

BP :

Pulse rate :

Respiratory rate :

SpO2:

ANTHROPOMETRY :

BMI

INVESTIGATIONS :

1. ECG
2. Sr. D Dimer
3. Sr. Hoocysteine
4. 2 D ECHO
5. CXR
6. CT CHEST
7. Venous doppler of Lower Limb.....

CONSENT FORM

Format for Informed Consent Form for Parent / Guardian of the Subjects

Informed Consent form to participate in a research study

Study Title:

Study Number: _____

Subject's Initials: _____ **Subject's Name:** _____

Date of Birth / Age: _____

(i) I confirm that I have read and understood the information sheet dated _____ for the above study and have had the opportunity to ask questions. [ ]

(ii) I understand that my son / daughter's participation in the study is voluntary and that he/she is free to withdraw at any time, without giving any reason, without his/her medical care or legal rights being affected. [ ]

(iii) I understand that the Ethics Committee and the regulatory authorities will not need my permission to look at my son / daughter's health records both in respect of the current study and any further research that may be conducted in relation to it, even if he/she withdraws from the trial. I agree to this access. However, I understand that my son / daughter identity will not be revealed in any information released to third parties or published. [ ]

(iv) I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s). [ ]

(v) I agree for the participation of my son/daughter in the above study. [ ]

Signature (or Thumb impression) of the Subject's parent /Legally Acceptable Guardian
Date: _____/_____/_____
Signatory's Name: _____
Signature: _____
Or
Representative: _____
Date: _____/_____/_____
Signatory's Name: _____

Signature or thumb impression of the Witness: _____
Date: _____/_____/_____
Name & Address of the Witness: _____

நோயாளிகளுக்கு அறிவிப்பு மற்றும் ஒப்புதல் படிவம்
(மருத்துவ ஆய்வில் பங்கேற்பதற்கு)

ஆய்வு செய்யப்படும் தலைப்பு:
பங்கு பெறுவரின் பெயர்:
பங்கு பெறுவரின் வயது:

		பங்கு பெறுவர் இதனை ✓ குறிக்கவும்
1.	நான் மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்களை படித்து புரிந்து கொண்டேன். என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டுள்ளது என அறிந்து கொண்டேன்.	☐
2.	நான் இவ்வாய்வில் தன்னிச்சையாக தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும், எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.	☐
3.	இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு மேற்காள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.	☐
4.	இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக் கொள்ள மறுக்க மாட்டேன்.	☐
5.	இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன் எனக்கு கொடுக்கப்பட்டஅறிவுரைகளின் படி நடந்து கொள்வதுடன், ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ, அல்லது எதிர்பாராத, வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ உடனே இதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்றேன்.	☐

பங்கேற்பவரின் கையொப்பம் / ..... இடம் .....
கட்டைவிரல் ரேகை
பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....
ஆய்வாளரின் கையொப்பம் /..... இடம் .....
ஆய்வாளரின் பெயர் .....
மையம் .....
கல்வியறிவு இல்லாதவற்கு (கைரேகை வைத்தவர்களுக்கு) இது அவசியம் தேவை
சாட்சியின் கையொப்பம் /..... இடம் .....
பெயர் மற்றும் விலாசம் ....

SI NO	NAME	IP NO	AGE	SEX	OCCUPATION	COUGH	CHEST PAIN	BREATHLESSNESS	SMOKING	OBESITY	RECENT HISTORY OF SURGERY/IMMOBILISATION	FAMILY H/O VTE	BLOOD TRANSFUSION	FEVER	LONG AIR TRAVEL	INDWELLING CATHETER/CV LINE	PREGNANCY	POSTPARTUM	HYPERCOAGULABLE STATE	WELLS SCORE	SPO2	RR	BP	D dimer	CPK MB	Sr. Creatinine	ECG	CT Chest / cxr	ECHO	CTPA	Sr. homocysteine	Outcome	DVT	Pulmonary embolism
1	Arumugak	55651	42	F	House Wife	No	No	Yes	No	No	No	No	No	No	No	No	No	Yes	No	Yes	91% - 94%	N	> 90/60	Yes	No	No	Yes	POSITIVE	Yes	POSITIVE	Yes	A	Yes	Yes
2	Esakimani	51859	30	M	Coolie	No	No	Yes	Yes	No	No	No	No	No	No	No	No	Yes	No	No	Yes	≤90%	B	≤ 90/60	No	No	No	No	No	Yes	POSITIVE	Yes	E	Yes	Yes
3	Vanamoort	51761	43	M	Coolie	No	No	Yes	Yes	No	No	No	No	No	No	No	No	Yes	No	No	Yes	91% - 94%	B	> 90/60	Yes	No	Yes	No	No	Yes	POSITIVE	Yes	A	No	Yes
4	Esakiamma	24313	72	F	House Wife	No	No	Yes	No	No	No	No	No	No	No	No	No	Yes	No	No	No	≥ 95%	C	> 90/60	No	No	No	Yes	No	Yes	POSITIVE	Yes	E	No	Yes
5	Sivakumar	11640	35	M	Coolie	No	No	Yes	Yes	Yes	Yes	No	No	No	No	No	Yes	No	No	Yes	≤90%	B	≤ 90/60	Yes	No	No	No	No	No	Yes	POSITIVE	Yes	A	Yes	Yes
6	Subbuthai	87594	48	F	House Wife	No	No	Yes	No	No	No	No	No	No	No	No	No	Yes	No	No	No	≥ 95%	B	> 90/60	No	No	No	No	No	Yes	POSITIVE	Yes	A	No	Yes
7	Petchiappa	11731	66	M	Coolie	No	No	Yes	Yes	No	No	No	No	Yes	No	No	No	Yes	No	No	Yes	91% - 94%	B	≤ 90/60	No	No	No	No	No	No	POSITIVE	No	E	Yes	Yes
8	Srirenga na	64178	53	M	Coolie	Yes	No	Yes	Yes	No	No	No	No	No	No	No	No	Yes	No	No	No	≥ 95%	B	> 90/60	No	No	No	Yes	No	Yes	POSITIVE	Yes	A	No	Yes
9	Annamuth	67563	72	F	Coolie	No	No	No	No	No	No	No	No	No	No	No	No	Yes	No	No	No	≥ 95%	B	≤ 90/60	Yes	No	No	No	No	No	No	No	A	Yes	No
10	Samsudeer	64603	76	M	NO WORK	No	No	Yes	No	No	No	No	No	Yes	No	No	No	Yes	No	No	No	≤90%	B	≤ 90/60	Yes	No	No	No	No	Yes	POSITIVE	Yes	A	No	Yes
11	Vijaya	69850	19	F	NO WORK	No	No	Yes	No	No	No	No	Yes	Yes	No	No	No	No	No	No	Yes	≤90%	C	> 90/60	Yes	No	No	Yes	No	Yes	POSITIVE	Yes	E	Yes	No
12	Petchimuth	69987	44	M	Coolie	No	No	Yes	No	No	No	No	No	No	No	No	No	Yes	No	No	Yes	≤90%	B	≤ 90/60	Yes	No	No	Yes	No	Yes	POSITIVE	Yes	A	Yes	Yes
13	Muthusam	71289	50	M	Coolie	No	Yes	Yes	Yes	Yes	No	No	No	No	No	No	No	Yes	No	No	Yes	91% - 94%	C	≤ 90/60	No	No	No	No	No	No	POSITIVE	Yes	E	No	Yes
14	Ravi	76869	48	M	Coolie	No	Yes	Yes	No	No	No	No	No	No	No	No	No	Yes	No	No	Yes	91% - 94%	B	> 90/60	No	No	No	No	No	No	POSITIVE	Yes	A	Yes	Yes
15	Durairaj	79760	60	M	Coolie	No	Yes	Yes	Yes	No	No	No	No	No	No	No	No	Yes	No	No	Yes	≥ 95%	B	> 90/60	No	No	No	No	No	No	POSITIVE	Yes	A	Yes	No
16	Ponniah	81361	52	M	Coolie	Yes	Yes	Yes	Yes	No	No	No	No	No	No	No	No	Yes	No	No	Yes	≤90%	C	≤ 90/60	No	No	No	No	No	Yes	POSITIVE	Yes	E	No	Yes
17	Selvamani	87798	46	M	Coolie	No	No	Yes	No	No	No	No	No	No	No	No	No	Yes	No	No	No	91% - 94%	B	> 90/60	No	No	No	Yes	POSITIVE	Yes	POSITIVE	Yes	A	No	Yes
18	Devi	89978	38	F	House Wife	No	No	Yes	No	No	No	No	No	No	No	No	No	Yes	No	Yes (AP	Yes	≤90%	C	> 90/60	No	No	No	Yes	No	Yes	POSITIVE	Yes	A	No	Yes
19	Arjunan	91224	57	M	coolie	No	No	Yes	Yes	No	No	No	No	No	No	No	No	Yes	No	No	No	91% - 94%	B	> 90/60	No	No	No	No	No	No	POSITIVE	Yes	A	Yes	Yes
20	Dasan	95438	62	M	NO WORK	No	Yes	No	No	No	No	No	Yes	No	No	No	No	Yes	No	No	Yes	≤90%	C	≤ 90/60	No	No	Yes	Yes	No	Yes	POSITIVE	Yes	A	No	Yes
21	Krishnan	98749	47	M	Coolie	No	No	Yes	Yes	No	Yes	No	Yes	No	No	No	No	Yes	No	No	Yes	91% - 94%	B	> 90/60	Yes	No	No	Yes	No	Yes	POSITIVE	Yes	A	No	Yes
22	Vadivu	99760	57	F	House Wife	No	No	Yes	No	No	No	No	No	No	No	No	No	Yes	No	No	No	91% - 94%	B	> 90/60	No	No	No	No	No	Yes	POSITIVE	Yes	A	No	Yes
23	Subbiah	10873	61	M	NO WORK	No	No	Yes	Yes	No	No	No	No	No	No	No	No	Yes	No	No	No	91% - 94%	B	> 90/60	No	No	No	No	No	No	POSITIVE	Yes	A	No	Yes
24	Yovan	13248	52	M	Coolie	No	No	Yes	Yes	No	No	No	No	Yes	No	No	No	Yes	No	No	Yes	91% - 94%	B	≤ 90/60	No	No	No	No	No	No	POSITIVE	No	E	Yes	Yes
25	Thangiah	13897	48	M	coolie	No	No	Yes	No	No	No	No	No	No	No	No	No	Yes	No	No	Yes	91% - 94%	B	> 90/60	Yes	No	Yes	No	No	Yes	POSITIVE	Yes	A	No	Yes
26	Sankar	13973	53	M	coolie	Yes	Yes	Yes	Yes	No	No	No	No	No	No	No	No	Yes	No	No	Yes	≤90%	C	≤ 90/60	No	No	No	No	No	Yes	POSITIVE	Yes	E	No	Yes
27	Sivan	14358	46	M	Coolie	No	No	Yes	Yes	Yes	No	No	No	No	No	No	No	Yes	No	No	Yes	91% - 94%	C	≤ 90/60	No	No	No	No	No	No	POSITIVE	Yes	A	No	Yes
28	Marisamy	14472	42	M	Coolie	No	Yes	Yes	No	No	Yes	No	No	No	No	No	No	Yes	No	No	Yes	91% - 94%	B	> 90/60	Yes	No	No	Yes	No	Yes	POSITIVE	Yes	A	No	Yes
29	Veldurai	14623	55	M	Coolie	No	No	Yes	Yes	No	No	No	No	No	No	No	No	Yes	No	No	No	91% - 94%	B	> 90/60	No	No	No	No	No	No	POSITIVE	Yes	A	Yes	Yes
30	kannan	14850	49	M	Coolie	No	Yes	Yes	No	No	No	No	No	No	No	No	No	Yes	No	No	Yes	91% - 94%	B	> 90/60	Yes	No	Yes	No	No	Yes	POSITIVE	Yes	A	No	Yes
31	Muniasamy	14957	47	M	Coolie	Yes	Yes	Yes	No	No	No	No	No	No	No	No	No	Yes	No	No	No	91% - 94%	B	> 90/60	No	No	No	No	No	Yes	POSITIVE	Yes	Y	No	Yes
32	Paraman	15237	52	M	Coolie	No	No	No	No	No	No	No	No	No	No	No	No	Yes	No	No	No	≥ 95%	B	≤ 90/60	Yes	No	No	No	No	No	No	No	A	Yes	No
33	Mookiah	15297	49	M	Coolie	No	No	Yes	Yes	No	No	No	No	No	No	No	No	Yes	No	No	Yes	91% - 94%	B	> 90/60	Yes	No	Yes	No	No	Yes	POSITIVE	Yes	A	No	Yes
34	Sivabalan	15389	59	M	Coolie	Yes	No	Yes	Yes	No	No	No	No	No	No	No	No	Yes	No	No	No	≥ 95%	B	> 90/60	No	No	No	Yes	No	Yes	POSITIVE	Yes	A	No	Yes
35	Jeyaraj	15609	49	M	Coolie	No	Yes	No	No	No	No	No	Yes	No	No	No	No	Yes	No	No	Yes	≤90%	C	≤ 90/60	No	No	Yes	Yes	No	Yes	POSITIVE	Yes	A	No	Yes
36	Kennedy	15710	52	M	Inspector	No	Yes	Yes	No	No	No	No	No	No	No	No	No	Yes	No	No	Yes	91% - 94%	B	≤ 90/60	No	No	No	No	No	No	POSITIVE	Yes	A	No	Yes
37	Pavithra	15891	48	F	House Wife	No	No	Yes	No	No	No	No	No	No	No	No	No	Yes	No	Yes (Re	No	≥ 95%	B	> 90/60	No	No	No	No	No	No	POSITIVE	Yes	A	No	Yes
38	Sudalai	15902	53	M	Coolie	No	No	Yes	Yes	No	No	No	No	No	No	No	No	Yes	No	No	Yes	≤90%	B	≤ 90/60	No	No	No	No	No	Yes	POSITIVE	Yes	E	Yes	Yes
39	Kupusamy	15821	49	M	Coolie	No	No	Yes	Yes	No	No	No	No	No	No	No	No	Yes	No	No	Yes	91% - 94%	B	> 90/60	Yes	No	Yes	No	No	Yes	POSITIVE	Yes	A	No	Yes
40	Mani	15999	67	M	Coolie	Yes	Yes	Yes	Yes	No	No	No	No	Yes	No	No	No	Yes	No	Yes (Ca	Yes	91% - 94%	B	≤ 90/60	Yes	No	No	No	POSITIVE	Yes	POSITIVE	Yes	A	No	Yes
41	Ramiah	16402	49	M	Coolie	No	Yes	Yes	No	No	No	No	No	No	No	No	No	Yes	No	No	Yes	91% - 94%	B	> 90/60	No	No	No	Yes	No	Yes	POSITIVE	Yes	A	No	Yes
42	Kannan	16319	52	M	Retrd	Yes	No	Yes	Yes	No	No	No	No	No	No	No	No	Yes	No	No	Yes	≤90%	C	≤ 90/60	No	No	No	No	No	No	POSITIVE	Yes	E	No	Yes
43	kumar	16519	59	M	Coolie	No	No	Yes	No	No	No	No	No	No	No	No	No	Yes	No	No	Yes	91% - 94%	N	> 90/60	Yes	No	No	Yes	POSITIVE	Yes	POSITIVE	Yes	A	Yes	Yes
44	Samy	16789	69	M	Coolie	No	No	Yes	No	No	No	No	No	No	No	No	No	Yes	No	No	Yes	91% - 94%	B	> 90/60	Yes	No	Yes	No	No	Yes	POSITIVE	Yes	A	No	Yes
45	Durga	16951	52	F	House Wife	No	Yes	Yes	No	No	No	No	No	No	No	No	No	Yes	No	No	No	≥ 95%	B	> 90/60	No	No	No	No	No	No	POSITIVE	Yes	A	No	Yes
46	Karupasmy	17328	59	M	Coolie	No	No	Yes	No	No	No	No	No	No	No	No	No	Yes	No	No	Yes	≤90%	C	≤ 90/60	No	No	No	No	No	No	POSITIVE	Yes	E	No	Yes
47	veerandi	17493	50	M	Coolie	No	No	Yes	No	No	No	No	No	No	No	No	No	Yes	No	No	Yes	91% - 94%	A	> 90/60	No	No	No	Yes	POSITIVE	No	POSITIVE	No	A	Yes	Yes
48	prakash	17982	48	M	Coolie	No	Yes	Yes	No	No	No	No	No	No	No	No	No	Yes	No	No	Yes	91% - 94%	B	> 90/60	No	No	No	No	No	No	POSITIVE	Yes	A	No	Yes
49	Susai	18321	55	M	Coolie	Yes	No	Yes	No	No	No	No	No	No	No	No	No	Yes	No	No	Yes	≤90%	B	≤ 90/60	No	No	Yes	No	No	No	No	No	A	Yes	No
50	Kannapan	19536	79	M	Coolie	Yes	No	Yes	No	No	No	No	No	No	No	No	No	Yes	No	No	Yes	91% - 94%	B	> 90/60	No	No	No	No	No	No	No	Yes	A	Yes	No