Section: Cardiology

Original Research Article

THE STUDY OF AETIOLOGY, CLINICAL FEATURES & MANAGEMENT OF DEEP VENOUS THROMBOEMBOLISM

Ravikanth A[1], Ghouse MD[2], MRM.Babu[3]

[1]Associate Professor, Department of Cardiology, Kamineni Academy of Medical Sciences, LB Nagar, Hyderabad, Telangana, India.
[2]Senior Resident, Department of Cardiology, Kamineni Academy of Medical Sciences, LB Nagar, Hyderabad, Telangana, India.
[3]Professor, Department of Cardiology, Kamineni Academy of Medical Sciences, LB Nagar, Hyderabad, Telangana, India.

Received *: 02/06/2024*
Received in revised form *: 07/08/2024*
Accepted *: 22/08/2024*

Corresponding Author:
Dr. Ravikanth A,
Associate Professor, Department of Cardiology, Kamineni Academy of Medical Sciences, LB Nagar, Hyderabad, Telangana, India.
Email: ravimounikaamshala@gmail.com

DOI: 10.70034/ijmedph.2024.3.204

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2024; 14 (3); 1152-1159

ABSTRACT

Background: To study the incidence and risk factors of deep venous thrombosis with or without Pulmonary Embolism in patients presenting to the Kamineni hospital.

Material and Methods: It was a observational study. Patients suspected to suffer from VTE were selected, a detailed history was taken, physical examination was done in all patients and investigated with venous Doppler to diagnose as DVT. Fifty patients were admitted during two-year period.

Results: In an observational study, 50 patients were studied during two years period. There were 29 male and 21 female patients. The ratio of male to female was 1.38. In the study of diagnosed venous thromboembolism found ratio of female/male as 1.6, but found males affected more than female Median duration of presentation after onset of symptoms was 4.2 days (range, 120 days). ECG changes related to VTE were seen 17 (34%) patients in which sinus tachycardia was most common in 14(28%) patients. RBBB was found in 5 (10%). S1Q3T3/S in lead LO and T wave inversion in lead III) in 1(2%) patients, other changes, LVH in 3(6%) and old anterior wall MI in 1 patient (2%) were seen. We studied risk factors in our patients and found Protein S deficiency in 19(38%), Protein C deficiency in 18 (36%), Hyperhomocysteinemia 17(34%). Protein S and Protein C deficiency in 18(36%), Protein S, Protein C deficiency and Hyperhomocysteinemia in 4 (8%), Protein S and Hyperhomocysteinemia in 3 (6%) patients. PTE was proved in nineteen patients (38%) patients on CT pulmonary angiography. Massive and sub massive PE was found in 8 (16%) and minor PE in 11(22%) patients.

Conclusion: The study concluded that VTE and its manifestations, including DVT and PE, are preventable therefore; a greater awareness of VTE and its risk factors is needed. Given the high incidence of DVT, and potentially life-threatening PE, prompt and accurate recognition of risk factors is crucial for implementing effective prophylaxis and reducing the burden of VTE and its manifestations.

Key Words: Pulmonary Embolism, DVT, VTE, Protein C, Hyperhomocysteinemia.

INTRODUCTION

Venous thromboembolism is a multifactorial disease which results from clot in venous circulation. The clinical manifestation of this results, from obstruction of respective vein and/or by embolization of parts of clot into pulmonary circulation.

Venous thrombus formation is due to an imbalance between local and systemic procoagulant-anticoagulant and profibrinolytic antifibrinolytic activity leading to hypercoagulable state.[1] In 1856, Rudolf Virchow postulated that a triad of factors

leads to intravascular coagulation: local trauma to vessel wall, hypercoagulability, and stasis.[2]

The various acquired and genetic factors contribute to venous thromboembolism. Acquired predisposition include obesity, cigarette smoking, oral contraceptives, trauma, pregnancy, postmenopausal hormone replacement, surgery, immobilization and cancer. The inherited conditions like deficiency of protein C. protein S, antithrombinIII mutation of factor V leiden and prothrombin gene, antiphospholipid syndrome and hyperhomocysteinemia may lead to venous thromboembolism.[3]

The largest impact on VTE incidence is age, with incidences of <5/100000 in childhood to > 500/100000 in persons above the age of 80 years. There seems to be no clear gender difference in incidences of VTE. For aetiological constellation, approximately 20% of cases are due to cancer, while surgery, trauma and immobilization account for 50% of cases. The remaining 30% have to be classified as idiopathic or 'unprovoked'.[4,5]

Pulmonary embolism (PE) is a common clinical disorder with an average annual incidence of one case per 1000 population in the western population. It is responsible for about 5-10% of all in-hospital deaths. It is an important diagnosis to consider, given the fact, that 10% of symptomatic PE are fatal in the first hour and that a hospital mortality to untreated PE can be reduced from 30% to nearly 8% if treated appropriately. Most of the deaths occur when the diagnosis is delayed or never made.

The clinical syndromes of PE and deep venous thrombosis (DVT) are now considered part of a spectrum of dysregulated haemostasis within the venous system designated as Venous Thromboembolism (VTE). Although rapid advances have taken place in the diagnosis and management of VTE, PE is still an unreported entity form India. Most of the reports are limited to autopsy reports and short case series 10. However, with the advent of spiral computed tomography (especially MDCT), there is now an increased recognition of this entity in India.

This study involves study of risk factors profile for VTE in the patients admitted at a private hospital. The symptoms and clinical signs, when combined with other patient information such as the presence or absence of known factors for DVT, can improve clinical prediction considerably. The most important step in VTE prevention is recognition of predisposing factors and high-risk individuals.

Objective

1. To study the incidence of deep venous thrombosis with or without Pulmonary Embolism in patients presenting to the Kamineni hospital.
2. To study the risk factor profile for venous thromboembolism in our patient population admitted into Kamineni hospital.

MATERIALS AND METHODS

Study Design: Observational study

Settings: Hospital Setting (Kamineni hospital)

Participants

Cases of VTE which includes

i. Patients admitted with VTE in the hospital.
ii. Patient who developed VTE during hospital stay.

Inclusion Criteria

i. Diagnosed cases of Deep vein and/or pulmonary embolism. DVT is clinical features of unilateral or bilateral leg swelling of short duration by warmth, erythema and tenderness and PE by symptoms of dyspnoea, pleuritic pain, cough, haemoptysis, cyanosis, neck vein distension, hypotension.
ii. DVT proven by venous Doppler and Pulmonary embolism by CT Pulmonary Angiography.

Exclusion Criteria:

i. Patients less than 18 years of age.

Methods

Patients suspected to suffer from VTE were selected, a detailed history was taken, physical examination was done in all patients and investigated with venous Doppler to diagnose as DVT. Fifty patients were admitted during two-year period.

Apart from the routine tests, patients with suspected PE underwent spiral computed pulmonary angiography (MDCT) depending upon the clinical condition patients underwent ECG and echocardiography. A patient was diagnosed to have PE if there was a filling defect on MDCT. Patients with PE were classified as ‘massive’ if there was an evidence of hemodynamic compromise (defined as systolic wood <90 mmHg) and as 'sub massive', if there was right ventricular dysfunction on echocardiography with no hemodynamic compromise: patients without any of the features of massive or sub massive PE were classified as 'minor' PE cases.(6) Patients were classified as 'idiopathic', if they had no obvious risk immobilisation, actors, such as, surgery within the last three months, malignancy, pregnancy, air-travel, etc. These patients were investigated for Protein S. Protein C Homocysteine levels.

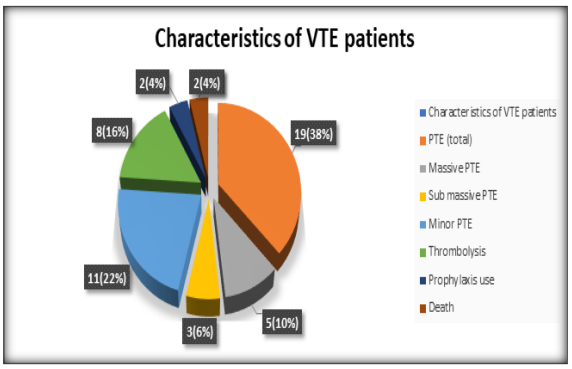
Patients with massive PE and those with sub-massive PE having moderate severe hypoxemia (PaO2<60 mmHg) were thrombolysed. Streptokinase was used for thrombolysis with loading dose of 250000 U over 30 minutes followed by 1 lacU/h over 24 to 36 hours. All patients with VTE received low molecular weight heparin (LMWH), enoxaparin one mg/kg subcutaneously twice daily for seven days followed by oral warfarin. Prothrombin time was regularly monitored to maintain International Normalised Ratio (INR) between two to three. Those who were successfully thrombolysed were discharged with advice for long

term anticoagulation, and others for at least six months. Venous Doppler of lower limb was done in all cases at the time of discharge which revealed at least partial recanalization in all cases.

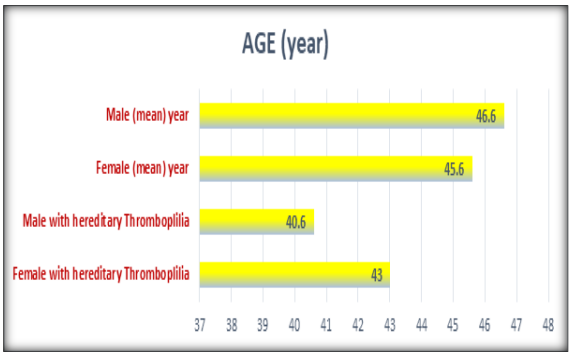
Optimal duration of anticoagulation

Clinical Setting	Recommendation
First provoked PE/proximal leg DVT	6 months
Fist provoked upper extremity DVT or isolated calf DVT	3 months
Second provoked VTE	12 months or indefinite
Third VTE	Indefinite
Cancer	6 month or indefinite
Unprovoked (Hereditary thrombophilias)	Consider Indefinite duration

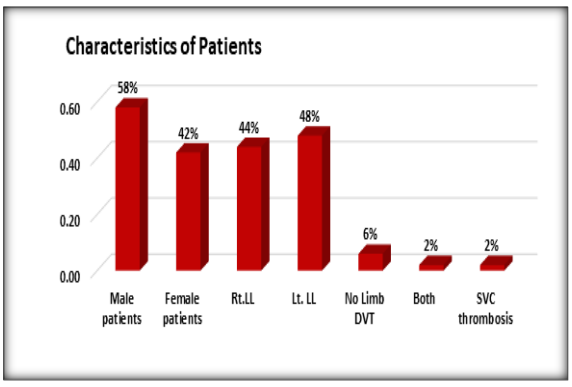
RESULTS



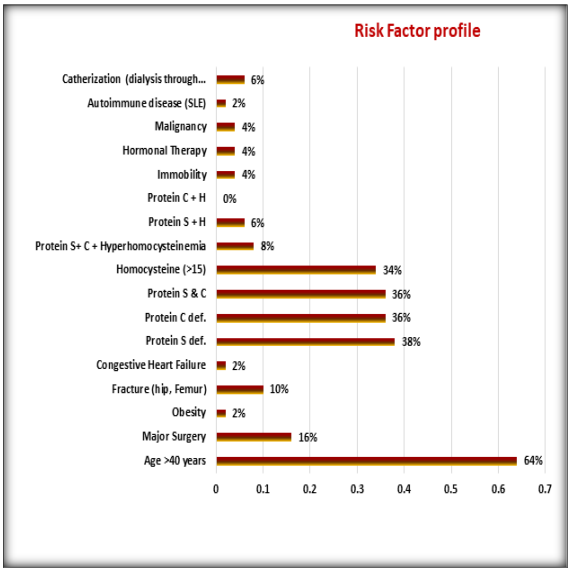
Graph 1: Showing Characteristics of VTE patients



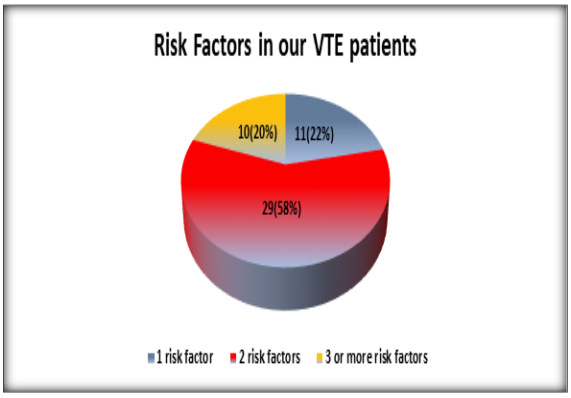
Graph 2: Showing Age characteristics of VTE patients



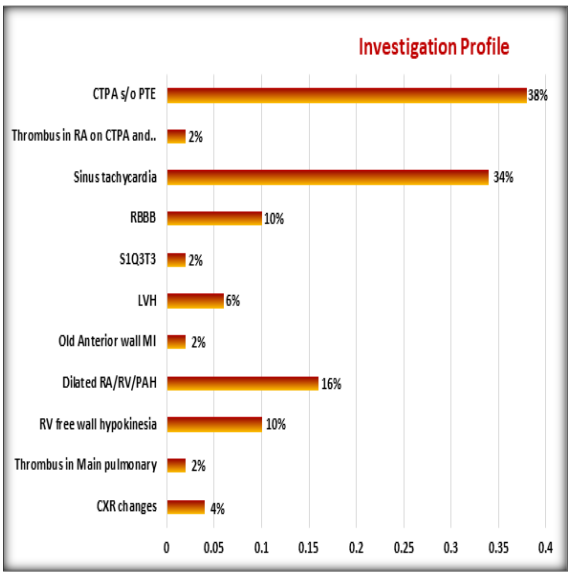
Graph 3: Showing various characteristics of VTE patients



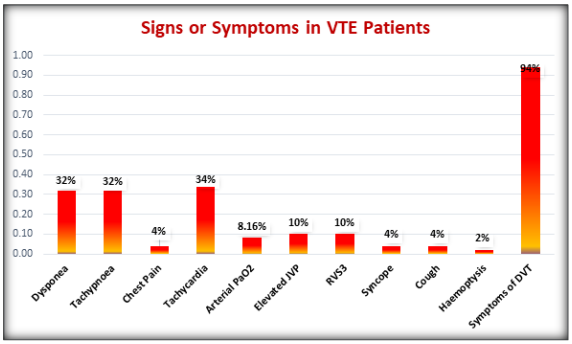
Graph 4: Showing risk factor profile in our VTE patients



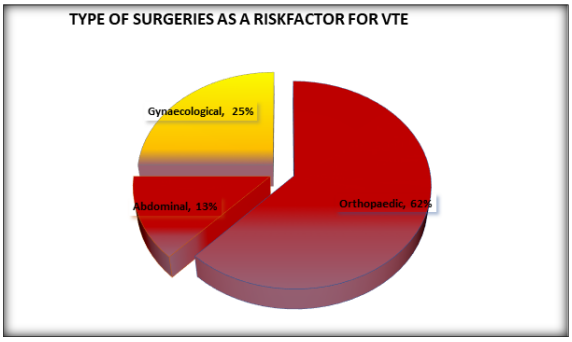
Graph 5: Showing no. of risk factors in relation to VTE



Graph 6: Showing investigation profile in our VTE patients



Graph 7: Showing Signs and Symptoms in VTE patients



Graph 8: Showing types of surgeries predisposing to VTE

Table 1: Characteristics of 50 VTE patients

Characteristics	No.	Percent
Male patients	29	58.0%
Female patients	21	42%
Right Lower Limb	22	44%
Left Lower Limb	24	48%
No Limb DVT with PTE	03	6%
Both Lower Limb DVT	01	2%
SVC thrombosis	01	2%
PE (total)	19	38%
Massive PE	05	10%
Sub massive PE	03	06%
Minor PE	11	22%
Thrombolysis	08	16%
Patients who were on Prophylaxis use developing DVT	02	04%
Death	02	04%

Table 2: Mean age in VTE patients

Characteristics	AGE (year)
Male (mean) year	46.6
Female (mean) year	45.6
Male with Hereditary Thrombophilias	40.6
Female with Hereditary Thrombophilias	43
Mean Age of male with PE	38.8
Mean Age of female with PE	33

Table 3: Showing various risk factors in our patients

Age >40 years	32	64%
Minor surgery	08	16%
Obesity	01	02%
Fracture (hip, femur) {Orthopedic Surgery}	05	10%
Congestive Heart Failure	01	02%
Protein S def.	19	38%
Protein C def.	18	36%
Protein S & C	18	36%
Homocysteine (>15)	17	34%
Protein S + C + Hyperhomocysteinemia (H)	04	8%
Protein S + H	03	6%
Protein C + H	00	0%
Immobility	02	4%
Hormonal Therapy	02	4%
Malignancy	02	4%
Autoimmune disease (SLE)	01	02%
Catheterization (dialysis through femoral vein)	03	06%
Gynecological Surgery	02	04%
Abdominal Surgery	01	02%
1 risk factor for VTE	11	22%
2 risk factors for VTE	29	58%
3 or more risk factors for VTE	10	20%
PE with 1 risk factor	04	02%
PE with 2 risk factors	07	14%
PE with 3 risk factors	08	16%

Table 4: ECG, 2DECHO, and CT pulmonary angiography changes (CTPA)

Investigation	No. of patients	Percent
Spinal CT pulmonary angiography (MSCT)		
CTPA s/o PTE	**19**	**38%**
Thrombus in RA	**01**	**02%**
ECG Changes		
Sinus tachycardia	**17**	**34%**
RBBB	**05**	**10%**
$S_1Q_3T_3$	**01**	**02%**
LVH	**03**	**06%**
Old anterior wall MI	**01**	**02%**
2DECHO		
Dilated RA/RV/PAH	**08**	**16%**
RV free wall hypokinesia	**05**	**10%**
Thrombus in Main pulmonary artery	**01**	**02%**
CXR changes showing lower lobe collapse, consolidation and pleural effusion	**02**	**04%**
Elevated D-dimer	**32**	**64%**
Venous Doppler showing DVT	**47**	**94%**

Table 5: Signs or Symptoms of VTE

Symptoms or Sign	No. of patients
Dyspnoea	16 (32%)
Tachypnea	16 (32%)
Chest Pain	02 (4%)
Tachycardia	17 (34%)
Arterial PaO_2<70mm of Hg	08 (16%)
Elevated JVP	05 (10%)
RVS_3	05 (10%)
Syncope	02 (4%)
Cough	02 (4%)
Haemoptysis	01 (2%)
Symptoms of DVT	47 (94%)

Table 6: Type of Surgery in Patients with VTE

Operation	No. of patients	Percent
Orthopaedic	5	10%
Gynaecologic	2	4%
Abdominal	1	2%

DISCUSSION

Pulmonary embolism presents with a wide clinical spectrum, from disease to life threatening massive PE that causes hypotension and shock. The clinical presentation and the investigations including electrocardiography. asymptomatic chest radiography, and analysis of arterial blood gases cannot be relied on to confirm or rule out PE because of lack of adequate specificity. The presence of one or more risk factors may lower the threshold for diagnostic evaluation. D-dimer testing has been reported to have a sensitivity ranging from almost 80-100 percent.

Since the origin of the thrombus is mostly from deep veins of the legs, compression ultrasound of lower limb veins is a useful investigation in the diagnosis of PE. It is, however, reported to be positive only in 10-20% of patients without leg signs who undergo evaluation and in approximately 50% of patients with proven PE. Thus, PE cannot be ruled out on the basis of negative results on ultrasound. Compression ultrasound has its value in situations where there is a highclinical probability of PE and the patient has no past history of VTE.[1] We found relatively younger patients coming to us with signs and symptoms of VTE. So, we selected this VTE patient group to study the risk factors profile in thesepatients.

In an observational study, 50 patients were studied during two years period. The annual incidence of DVT was 75.6 per 1, 00,000 inpatients and annual incidence of PTE was 35 per 1,00,000 inpatients. Anderson FA in his The Worcester DVT study found the annual incidence of DVT as 48 per 1,00,000 and annual incidence of PTE as 23 per 1,00,000 inpatients.[7] Hasson Po et al in their the study found population based incidence of DVT and PE as 182 and 205 per 1, 00,000 respectively.[8]

There were 29 male and 21 female patients. The ratio of male to female was 1.38. Enayat Safavi et al in the study of diagnosed venous thromboembolism found ratio of female/male as 1.6, but found males affected more than female.[9] In studies conducted in Worcester, Mass, and Olmsted County, Minn, the incidence of VTE in both studies, VTE was in men."[10] Median duration of presentation after onset of symptoms was 4.2 days (range, 120 days). R. Agrawal et al in the clinical profile, diagnosis and management of thepatients presenting with symptomatic pulmonary embolism found median duration of presentation after onset of symptoms was 4.5 days with range of 1-60 days.[11]

Mean age of the male patient was 46.6 years and mean age of female patient was 45.6 years. Enayat Safavi et al in the study of diagnosed venous found mean age of male and female patient 48 and 35 years respectively.[9] The mean age of male patient with hereditary thrombophilias was 40 wars and mean age of female with hereditary thrombophilias was 43 years. The mean age of male patient with PE was 38.8 years and mean age of female patient with PE was 33 years. It is seen in various studied that patients with PE are older than DVT but we found younger patients presenting with PE which cannot be explained.

Sign and symptoms of DVT (94%) like limb swelling, warmth, erythema and tenderness, and dyspnoea (32%) were the most common presenting complaints (Table 5). Agarwal et al in the clinical profile, diagnosis and management plaints patients presenting with symptomatic pulmonary embolism found dyspnoea as most common presenting complaint in PE patients.[11] Haemoptysis and chest pain were lessoften seen. Two (4%) patients presented with syncope and hypotension. Enayat Safavi et al in the study of diagnosed venous thromboembolism found that 10% of cases presented with syncope.[9] The right lower limb DVT was found in 22 (44%) and left lower limb DVT was found in 24 (48%) patients. Three (6%) patients were presented as VTE without DVT. DVT was detected in 94% cases by venous Doppler. One patient with cancer had initial involvement of right lower limb followed by involvement of left lower limb. PE was confirmed in all 19 (38%) cases by MDCT pulmonary angiography.

D-dimer was elevated in total 32 (64%) out of 50 patients and all the 19 PE patients. This blood screening test relies on the principle that most patients with PE have ongoing fibrinolysis that is not effective enough to prevent PE but does break down some of fibrin clot to D-dimer. This test is not generally useful for acutely ill hospitalised patients because their D-dimer levels are usually elevated. The Sensitivity of the ELISA D-dimer assay for acute PE was 96.4% and negative Predictive value was 99.6%. A normal D-dimer assay appears to be as diagnostically as normal lung scan to exclude PE.[2]

Eight(16%) patients had hypoxemia with PaO2, <70mmHg. In the Prospective ligation of Pulmonary Embolism Diagnosis (PIOPED), there was no difference between average Pa_2 (70 mmHg) among those with and without PE (72mmHg) at pulmonary angiography. Therefore, arterial blood gas of routine diagnostic strategy when investigating suspected PE.[12]

ECG changes related to VTE were seen 17 (34%) patients in which sinus tachycardia was most common in 14(28%) patients. RBBB was found in 5 (10%). S1Q3T3/S in lead LO and T wave inversion in lead III) in 1(2%) patients, other changes, LVH in 3(6%) and old anterior wall MI in 1 patient (2%) were seen. Enayat Safavi et al in the study of diagnosed venous thromboembolism found that sinus tachycardia was most common finding (68%) and RBBB in 10% of cases.[9] ECG helps to exclude acute myocardial infarction and may help to raise suspicion or confirm the diagnosis of PE.[2]

Chest X-ray showed right lower lobe consolidation with pleural effusion in one (2%) and left lower lobo consolidation in one (2%) patient in suggestive of plum in one infarction in two cases of minor PE.

Echocardiography is a useful tool in identifying high risk patients such as those with right ventricular dysfunction, patent foramen ovale, free floating thrombus and persistent pulmonary hypertension. It is a rapid, practical and sensitive technique for detection of right ventricular overload among patients with established and large PTE.[2] In our study, 2DECHO dilated RA/RV and PAH in 8(16%) patients who had PE proven by MDCT. One patient showed large thrombus in main pulmonary artery on 2DECHO. Five (10%) patients showed RV free wall hypokinesia and dysfunction.

We studied risk factors in our patients and found Protein S deficiency in 19(38%), Protein C deficiency in 18 (36%), Hyperhomocysteinemia 17(34%). Protein S and Protein C deficiency in 18(36%), Protein S, Protein C deficiency and Hyperhomocysteinemia in 4 (8%), Protein S and Hyperhomocysteinemia in 3 (6%) patients. None of the patient had combined Protein C deficiency and Hyperhomocysteinemia. The first case-control study looking at protein C deficiency was conducted by Heijboer et al in 1990 who did a study on 277 Dutch patients and 138 controls. The overall prevalence of protein C deficiency in the patients with venous thrombosis was 8.3 percent (23 of 277 patients) (95% CI 5.4-12.4), as compared with 2.2 percent in the controls. Both the work done by Heijboer et al and a subsequent study by Tait et al estimated the population prevalence of heterozygous protein C deficiency at 0.2%.[11] There is marked variability in risk among families with protein C deficiency that cannot be explained by the genetic defect itself. In severely affected families, as many as 75 percent of protein C deficient individuals experience one or more thrombotic events, in other families, the thrombosis rate is much lower.[13]

In 1987, Engesser and colleagues conducted a study on 12 Swedish families with 136 members and found 71 of them to be heterozygous for Type I protein S deficiency. 55% of those who carried the defect were found to have had a thrombotic event and 77% of those were recurrent. About half of the cases were precipitated by another condition. Overall, both family and cohort studies demonstrate that like other thrombophilic disorders, heterozygous protein S deficiency usually manifests in adulthood with a thromboembolic event.[14]

Heijer Den et al have demonstrated that hyperhomocysteinemia mild is an independent risk factor for venous thromboembolism. They found a marked increase in the risk of venous thrombosis at the highest plasma homocysteine concentrations. In

their case-control study of 269 patients with a first, objectively diagnosed episode of deep vein thrombosis and 269 healthy controls, 28 had plasma homocysteine levels above the 95th percentile for the controls, as compared with 13 of the controls with a matched odds ratio of 2.5; 95% Ck: 1.2-5.2.72 To investigate if homocysteine is a modifiable risk factor, Bonna studied the effect of supplementation on homocysteine levels as part of the Heart Outcomes Prevention Evaluation (HOPE 2) investigation. The study showed that supplements combining folic acid and vitamins B6 and B12 did not reduce the risk of major cardiovascular events in patients with vascular disease. Bonna also suggested that homocysteine could be a marker, but not a cause, of vascular disease.[15] At the same time, Loscalzo pointed out that while folic acid, vitamin B12, and vitamin B6 change the homocysteine levels there is likely a complex metabolic interaction involved that will need to be explored further before any conclusions can be drawn regarding homocysteine and the risk of thrombosis.[16]

While there is evidence that hyperhomocysteinemia is also a risk factor for Venous thromboembolism (VTE), there are conflicting data as to whether the risk of VTE is markedly increased in patients when hyperhomocysteinemia is combined with an inherited thrombophilias.

As thrombophilias in VTE, we found higher percentage of prevalence in our hospital which we are unable to explain.

Among the acquired risk factors immobility was in 2(4%), hormonal therapy in 2(4%), malignancy 2(4%), post orthopaedic surgery 5(10%), gynaecological surgery 2(4%), abdominal surgery in 1(2%) patient. Enayat Safavi et al in the study of diagnosed venous thromboembolism found that immobility, malignancy and major surgery as risk factors in 29%, 5% and 16% cases respectively.

Gibbs found that 15% of the patients on bed rest for 1 week before death had venous thromund that autopsy, whereas incidence rose to 80% in patients in bed for longer period.[17]

Advanced cancers are associated with high incidence of VTE especially cancers of the breast, lung, brain, pelvis, rectum, pancreas and gastrointestinal tract. The most common types of surgeries predisposing to VTE are orthopaedic abdominal, obstetrics and gynaecological. Frederic A. Anderson et al in the prevalence of risk factors for venous thromboembolism among hospital patients showed 23% of orthopaedic and abdominal surgeries each, obstetric 13% and gynaecological surgery 7% responsible for VTE.[3])

A recent case control study by Lidegaard concluded that the incidence of VTE in young women is between 1 and 3 per 10,000 per year. Pregnancy increases this risk by 5 times, low dose third generation oral contraceptives 4 times and low dose second generation oral pills 3 times.[18]

We found 58% of our patients had two risk factors, 22% had one risk factor and 20% had more than three risk factors. Risk factors in relation to PTE showed 4 patients with one risk factor and 15 patients with more than two risk factors. Arthur Wheeler in Venous Thromboembolism in Medically III Patients: Identifying Risk and Strategies for Prevention found that in a typical hospital population, 78% of patients have risk factors for VTE, and ~20% of patients had at least three risk factors.[19]

PTE was proved in nineteen patients (38%) patients on CT pulmonary angiography. Massive and sub massive PE was found in 8 (16%) and minor PE in 11(22%) patients. Patients with massive and sub massive PE were thrombolysed streptokinase. Dose of streptokinase → 250000 U over 30 min as loading dose and 100000 U/h for 24 36 hours was used. 7 out of 8 patients improved with thrombolytic therapy. Improvement in dyspnoea, tachycardia, Pao2, hypotension syncope and sign of RV failure 2DECHO showed improvement in RV functionease in PAH and tricuspid regurgitation. One patient showed reversal of RBBB ECG to normal pattern after thrombolytic therapy.

MAPPET-3 the largest randomised controlled trial of thrombolysis versus heparin alone, studied patients with sub massive PE. Tissue plasminogen activator with placebo. It showed, the frequency of escalation of the therapy- defined as the need for pressors, mechanical ventilation, cardiopulmonary resuscitation or open labelled thrombolysis was halved in cases of PE treated with thrombolysis as compared with placebo and did not increase major bleeding.

A meta-analysis of 748 patients in 11 prior randomized thrombolysis trials found that in subset of trials that included major PE. the mortality rate was halved and the major bleedingrate doubled in thrombolysis treated group.[2]

In-hospital mortality rate was 4% (2 patients). One patient who had large thrombus in main pulmonary artery died in spite of thrombolytic therapy and another developed DVT after discharge from hospital, was readmitted but developed PE in spite of on prophylaxis for DVT. All patients received LMWH and oral anticoagulants. They were followed and INR was monitored to maintain between 2 to There was no major bleeding in any of the thrombolysed patient.

Three Patients came back with recurrent DVT 6 months after stopping anticoagulant therapy. One of them developed DVT of same lower limb and two Developed DVT of other limb. Long term anticoagulation was advised to those patients.

At last, without a non-PE, non-DVT control group with exact histories, we are unable to comment on the relative risk conferred by various risk factors and we could tot study Factor V Leiden, prothrombin G20210A mutation and antithrombin III deficiency.

CONCLUSION

Because there are few signs and symptoms, VTE often goes unrecognized. VTE and its manifestations, including DVT and PE, are preventable therefore; a greater awareness of VTE and its risk factors is needed. Given the high incidence of DVT, and potentially life-threatening PE, prompt and accurate recognition of risk factors is crucial for implementing effective prophylaxis and reducing the burden of VTE and its manifestations.

Conflict of Interest: None
Funding Support: Nil.

REFERENCES

1. Schellong SM, Bounameaux H, Büller HR. Venous Thromboembolism. In: Camm AJ, Lüscher TF, Serruys PW, editors. The European Society Textbook of Cardiovascular Medicine. Oxford University Press; 2009. p. 1335–58.
2. Z. Golghaber, editor. Pulmonary Embolism. Braunwald's Heart Disease, a Cardivascular Medicine 6th edition. 6th edition. Philadelphia London New York: W.B. Saunders Company, a Harcourt Health Sciences Company; 2001. 1886–1907 p.
3. Anderson FA, Spencer FA. Risk factors for venous thromboembolism. Circulation. 2003;107(23 Suppl 1):I9-16.
4. White RH. The epidemiology of venous thromboembolism. Circulation. 2003 Jun 17;107(23 Suppl 1):I4-8.
5. Seligsohn U, Lubetsky A. Genetic susceptibility to venous thrombosis. N Engl J Med. 2001 Apr 19;344(16):1222–31.
6. Goldhaber Samuel Z. Pulmonary Embolism. New England Journal of Medicine. 1998;339(2):93–104.
7. Anderson FA Jr, Wheeler HB, Goldberg RJ, Hosmer DW, Patwardhan NA, Jovanovic B, et al. A Population-Based Perspective of the Hospital Incidence and Case-Fatality Rates of Deep Vein Thrombosis and Pulmonary Embolism: The Worcester DVT Study. Archives of Internal Medicine. 1991 May 1;151(5):933–8.
8. Hansson PO, Welin L, Tibblin G, Eriksson H. Deep Vein Thrombosis and Pulmonary Embolism in the General Population: "The Study of Men Born in 1913." Archives of Internal Medicine. 1997 Aug 11;157(15):1665–70.
9. Safavi E, Zahedpour Anaraki MR, Firoozbakhsh S, Fard MN. The Study of Diagnosed Venous Thromboembolism. TANAFFOS (Respiration). 2003 Jan 1;2(1(winter)):15–22.
10. Goldhaber SZ. Pulmonary embolism. Lancet. 2004 Apr 17;363(9417):1295–305.
11. R A, M G, Br M, Sk J. Clinical profile, diagnosis and management of patients presenting with symptomatic pulmonary embolism. The Indian journal of chest diseases & allied sciences. 2006 Jun;48(2).
12. Value of the ventilation/perfusion scan in acute pulmonary embolism. Results of the prospective investigation of pulmonary embolism diagnosis (PIOPED). JAMA. 1990 May;263(20).
13. Broekmans André W., Veltkamp Jan J., Bertina Rogier M. Congenital Protein C Deficiency and Venous Thromboembolism. New England Journal of Medicine. 1983 Aug 11;309(6):340–4.
14. Engesser L, Broekmans AW, Briët E, Brommer EJ, Bertina RM. Hereditary protein S deficiency: clinical manifestations. Ann Intern Med. 1987 May;106(5):677–82.
15. Bønaa KH, Njølstad I, Ueland PM, Schirmer H, Tverdal A, Steigen T, et al. Homocysteine lowering and cardiovascular events after acute myocardial infarction. N Engl J Med. 2006 Apr 13;354(15):1578–88.
16. Loscalzo Joseph. Homocysteine Trials — Clear Outcomes for Complex Reasons. New England Journal of Medicine. 2006;354(15):1629–32.
17. Gibbs NM. Venous thrombosis of the lower limbs with particular reference to bed-rest. Br J Surg. 1957 Nov;45(191):209–36.
18. Anderson FA, Wheeler HB, Goldberg RJ, Hosmer DW, Forcier A. The prevalence of risk factors for venous thromboembolism among hospital patients. Arch Intern Med. 1992 Aug;152(8):1660–4.
19. Wheeler A. Venous thromboembolism in medically ill patients: identifying risk and strategies for prevention. Clin Cornerstone. 2005;7(4):23–31.