Analysis of Clinical Characteristics and Outcome of Pulmonary Thromboembolism in a Tertiary Care Hospital

P. Aarthi[1], N. Babu[2], Arun Kumar Bathena[3], Radha Ganesan[4], Sindhupriya[5]

ABSTRACT

Introduction: Pulmonary embolism is a potentially lethal condition that is frequently underdiagnosed. The severity and clinical presentation of PE are variable, hence clinching the correct diagnosis is difficult. Prompt diagnosis of PE is essential to reduce morbidity and mortality. Current research aimed to study the clinical characteristics, risk factors and clinical outcomes of patients diagnosed with pulmonary thromboembolism.

Material and Methods: In this retrospective study data were collected from baseline clinical characteristics, presenting signs and symptoms, results of echocardiography and imaging studies, therapeutic modality and in-hospital course.

Results: 52 cases of Pulmonary embolism were reported with an incidence of 0.33%. Male predominance (58%) was noted. Associated comorbid conditions like HT was present in 24 patients and DM was present in15 patients. The most common symptom at presentation was dyspnea (31 patients). The overall mortality rate was 3.8% (2 patients).

Conclusion: The outcome of PE is improving; however, it remains an important risk factor for mortality in hospitalized patients.

Keywords: Outcome, Pulmonary Embolism, Risk Factor

INTRODUCTION

Pulmonary thromboembolism (PE) is an acute, potentially fatal condition in which the thrombus or the embolic material originating from the deep veins of the legs or pelvis blocks one or more of the pulmonary arteries causing impairment in blood flow and increased pressure to the right cardiac ventricle.[1] PE and deep vein thrombosis (DVT) are the two manifestations of the same condition with DVT being the third most common cardiovascular disorder.[2] The non-specific symptoms and a wide variety of clinical presentation of patients with suspected PE make diagnosis difficult. The incidence of PE increases proportionately with age with a reported 500- 600 cases per 1,00,000 individuals older than 75 years of age.[3] PE has a slight inclination towards women of reproductive age due to the association of the disease with pregnancy and increased use of oral contraceptives.[4] However in older age the incidence is higher in men than in women.

PE-related mortality can be high around 25% if left untreated and with appropriate anticoagulant therapy the mortality rate decreases to 2 – 8% in the 3 months follow-up. The actual mortality rates might be even higher because a solid number of patients die before diagnosis.[5,6] In the first month following diagnosis, i.e. in the acute phase of PE the mortality rate is influenced by hemodynamic instability, immobility and comorbidities whereas post 1 year the underlying comorbidities like congestive cardiac failure, malignancies, chronic kidney and lung disease became the major influential factors and the mortality rate reaches 24-27%.[7] PE can occur without any predisposing risk factors and it is usually multicausal.

Current research aimed to study the clinical characteristics, risk factors and clinical outcomes of patients diagnosed with pulmonary thromboembolism.

MATERIAL AND METHODS

This retrospective study was conducted in a tertiary care hospital during a one year period between, January 2017 to December 2017.

Inclusion criteria: All patients admitted and diagnosed with PE based on CT pulmonary angiogram.

The following data were recorded: Demographic features (age, sex), venous thromboembolism (VTE) risk factors (obesity, immobility, recent surgery, etc.), co-morbid diseases (chronic lung disease, cancer, congestive heart failure, central nervous system disorders, history of previous deep vein thrombosis [DVT]), presenting symptoms and signs (shortness of breath, chest pain, hemoptysis, heart rate, respiratory rate [RR], etc.), echocardiographic findings, treatment given and clinical outcome until hospital discharge. The primary outcome was in-hospital mortality rate, clinical deterioration and length of hospital stay. Clinical deterioration was defined as the development of hypotension (blood pressure <90/60) need for endotracheal

[1]Junior Consultant, Department of Critical Care, Vijaya Hospital, Chennai – 26, Tamilnadu, [2]Senior Consultant / Head of Department, Department of Critical Care, Vijaya Hospital, Chennai – 26, Tamilnadu, [3]Junior consultant, Department of Critical Care, Vijaya Hospital, Chennai – 26, Tamilnadu, [4]Medical Officer, Department of Critical Care, Vijaya Hospital, Chennai – 26, Tamilnadu, [5]Junior Registrar, Department of Critical Care, Vijaya Hospital, Chennai – 26, Tamilnadu, India

Corresponding author: Dr. N. Babu, Senior Consultant / Head of Department, Department of Critical Care, Vijaya Hospital, Chennai – 26, Tamilnadu, India

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intubation, vasopressors, cardiopulmonary resuscitation and rescue thrombolytics.

RESULTS

The study duration was one year from January 2017 to December 2017 and the total number of reported cases is 52 with an incidence of 0.33% PE. The mean age at presentation was 34.5 years with a male predominance (58%). (fig 1) The mean hospital stay of patients affected with PE is 8.90±4.19 days and the mean ICU stay was 3.85±2.14 days. Associated comorbid conditions like HT was present in 24 patients and DM was present in15 patients. CAD, CVA, TB and COPD are the other associated comorbidities. (fig 2) PE is multicausal, however the chief risk factor is observed to be

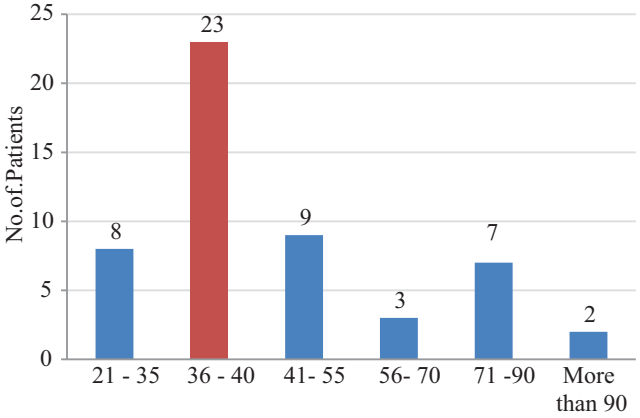

Figure-1: Age Distribution

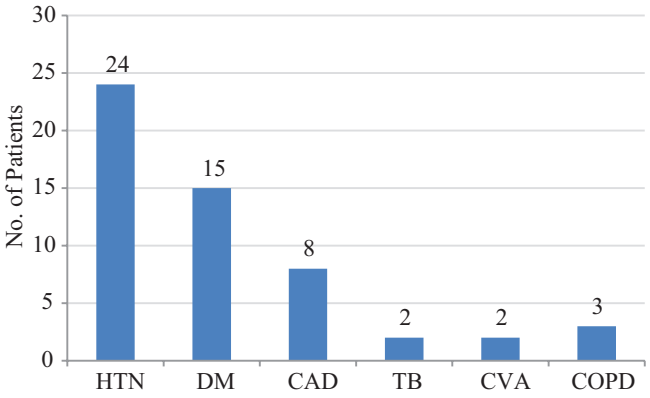

Figure-2: Co-Morbidities

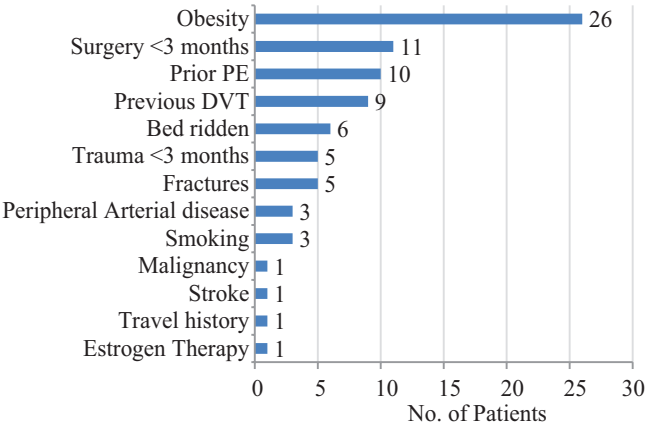

Figure-3: Risk Factors

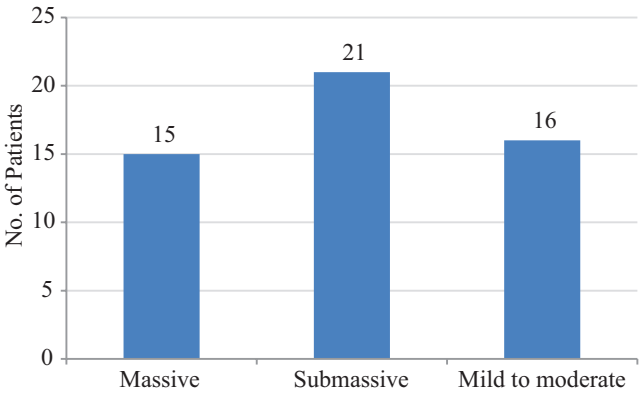

Figure-4: Pulmonary Embolism

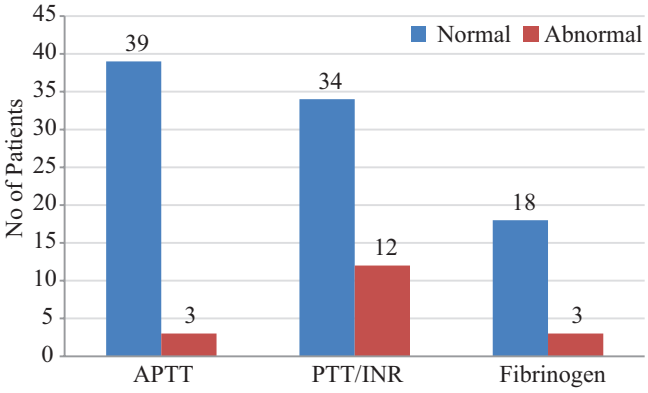

Figure-5: Coagulation Parameters

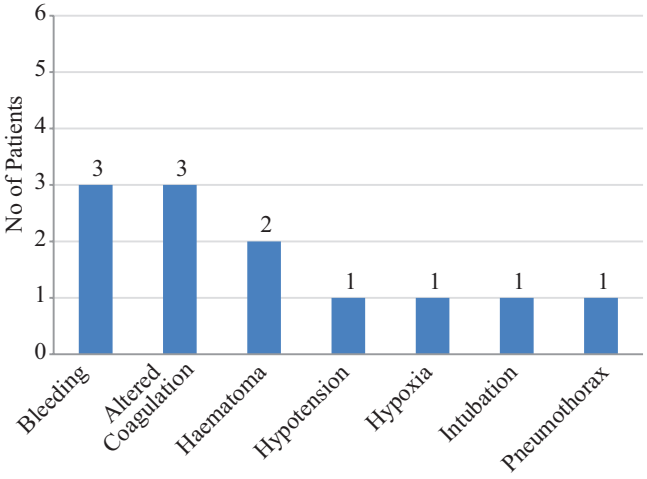

Figure-6: Complications

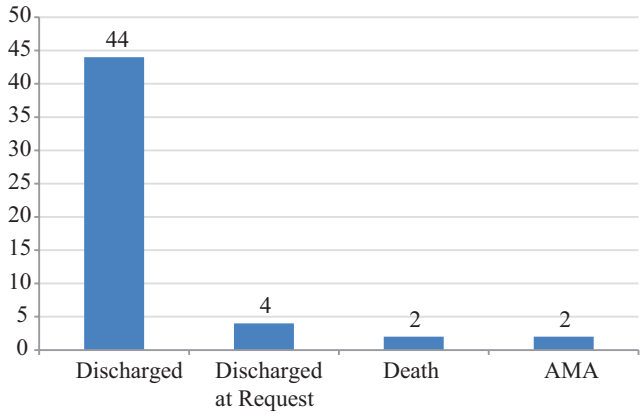

Figure-7: Outcome

obesity with the other risk factors being prior surgeries done < 3months, previous H/O DVT, bedridden patients, trauma, fractures, smoking, malignancy and oestrogen therapy. (fig 3) The most common symptom at presentation was dyspnoea(31 patients) followed by pedal edema, calf pain, chest pain, sweating, haemoptysis and syncope. There was a significant rise in heart rate and respiratory rate, however the temperature was normal in most of the patients. Massive PE was diagnosed in 15 patients, submassive PE in 21 patients and 16 patients were affected with mild to moderate PE. (fig 4)

ECHO analysis demonstrated pulmonary arterial hypertension(PAH) in 40% of the cases, followed by RV dysfunction(31%) and RV dilation(19%). PO_2 > 60 in 17 patients and PCO_2 30-40 was observed in 10 patients. Other specific investigations like CT chest, CTPA, lower limb Doppler and V/Q scan was done of which CT pulmonary angiogram(CTPA) was more helpful in diagnosing in PE. Abnormal protein C and protein S levels were noted in some patients when tested for inherited thrombophilia. Other investigations like lupus anticoagulant, jack 2 mutation, ANA immunoblot, anti-CCP were negative. All the coagulation parameters (APTT, PTT/INR and fibrinogen) were normal in a majority of patients. (fig 5) Upon treatment with heparin therapy 14 patients showed improvement in the first 24 hours and 9 patients responded to LMWH within 24 hours and 7 patients were thrombolysed. The chief complications associated with PE are bleeding and altered coagulation followed by hematoma, hypotension, hypoxia, intubation and pneumothorax (fig 6). IVC filter was placed in 9 patients and antiplatelets were administered to 56% of the patients. The overall mortality rate was 3.8% (2 patients) and both these patients had massive PE. 84% (44 patients) were discharged after cure.

DISCUSSION

This is an observational study reporting the clinical features, treatment modalities and outcomes of PE. Pulmonary embolism occurs when a deep vein thrombus detaches itself and embolises the pulmonary circulation, which in turn leads to pulmonary vascular occlusion and impaired gas exchange. The lower lobes of the lungs are more frequently affected than the upper lobes. Smaller emboli wedge the peripheral arteries while the larger emboli occlude the main pulmonary artery. When the systolic pressure in the pulmonary artery increases the RV afterload increases which lead to RV failure. The RV failure progresses to impaired LV filling which rapidly progresses to myocardial ischemia secondary to inadequate coronary artery filling. This leads to hypotension, electromechanical dissociation, syncope and sudden death.[8]

Prompt diagnosis of PE is important to limit the associated mortality and morbidity because most PE goes underdiagnosed. Evidence suggests that about 30% of the undiagnosed and untreated patients die.[9] Most of the PEs are asymptomatic, in this study the primary symptom was dyspnoea followed by pedal edema and calf pain. Other non-specific signs like tachycardia, hypoxia, and shock may be present which is also common in MI, CHF and Pneumonia which leads to misdiagnosis of PE.

The main risk factor of PE was observed to be obesity in this study followed by the history of recent surgeries. The other inherited risk factors include low levels of anticoagulant proteins and high levels of coagulant proteins. Deficiencies in natural coagulation inhibitors like protein C, protein S and antithrombin are also strong risk factors as observed in this study. Factor II (prothrombin) G20210A and factor V Leiden are also other genetic variants associated with PE. The acquired risk factors are surgery, cancer, prolonged hospitalization and immobility, infections and previous episodes of thromboembolism.[10-12] The literature states that concomitant cancer was diagnosed in about 4- 12% of the PE patients and this incidence of cancer in the early months following diagnosis of PE seems to be alarming and patients must be screened for any underlying malignancy.[13] The risk of PE also increases five-fold during pregnancy.[14] Hormonal replacement therapy is also known to increase the risk of PE. The clinical suspicion should guide the choice of initial diagnostic test used.[15] PE cannot be diagnosed clinically as the presenting symptoms are nonspecific. Fibrin D-dimer is a degradation product of cross-linked fibrin, and its levels are elevated in PE due to the simultaneous activation of coagulation and fibrinolysis.[16] In this study ECG, ECHO,ABG and D- Dimer tests were done and more specific investigations like CT chest, CTPA, lower and upper limb Doppler were also done out of which CTPA seemed to be more promising in the diagnosis of PE. Haemodynamic stabilization and reversal of hypoxemia are the two main therapeutic goals to be achieved in the treatment of PE. In the absence of contraindications, parenteral unfractionated heparin(UFH), low molecular weight heparin (LMWH) and fondaparinux can be administered. Most of the patients in this study responded well to unfractionated heparin and LMWH with additional oxygen therapy.

The main complications observed were bleeding, altered coagulation, hematoma, hypotension and hypoxia. Intubation was also required in one patient leading to a prolonged hospital stay and associated risks. However most of the patients (84%) were discharged after recovery and 2 deaths have been documented. The limitations are the small sample size and the randomly distributed samples. By correlating patient presentation, clinical suspicion and scoring systems diagnosis can be streamlined and unnecessary treatment can be avoided.

CONCLUSION

PE is associated with an overall mortality rate of 3.8% in this study mainly due to massive disease. A high index of suspicion is warranted if the patients presented with symptoms like dysponea or edema. Bleeding complications are also high and obesity is found to be a major risk factor of PE in patients of age 30 -35 years. CTPA proves to be a promising test for diagnosis of PE and subsequent thrombolysis is achieved with parenteral administration of LMWH. Future studies should focus on developing a proper

diagnostic and treatment strategy for PE.

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