Original Article

Pulmonary Embolism: Clinical Characteristics, Hospital Course and Outcome; Experiences at Lady Reading Hospital Peshawar

Ibrahim Shah,[1] **Shahzeb,**[2] **Mohammad Faheem,**[3] **Kashif Khan,**[4] **Mohammad Hafizullah,**[5] **Mohammad Nisar,**[6] **Rafiullah**[7]

Abstract

Objective: To evaluate the risk factors, clinical characteristics, therapeutic options and outcome of patients with pulmonary embolism (PE).

Materials and Methods: Data were collected retrospectively by reviewing medical records of patients admitted to intensive care unit (ICU) of lady Reading Hospital Peshawar, with primary diagnosis of pulmonary embolism (PE). Demographic, clinical, radiological, treatment and outcome data were collected.

Results: A total of 62 patients; 37 (60%) male and 25 (40%) female, were included in the study. The mean age (± SD) was 49.3 ± 13.14 years. Immobilization 43 (69.4), recent major surgery 25 (40.3), obesity 35 (56.5), active malignancy 16 (25.8) and recent trauma 15 (25.2) were frequent risk factors. Major presenting symptoms were dyspnea 59 (95.2%) and chest pain 28 (45.2%). Vital signs at presentation were; heart rate 117.41 ± 14.36 beats / min, systolic blood pressure 98.36 ± 15.38 mm Hg and respiratory rate 21.06 ± 2.7 breaths/min. Forty (64.5%) patients had hypoxemia and 12 (19.4%) patients were hypotensive on presentation. The chest radiograph was abnormal in 34 (54.8%) patients with cardiomegaly as the commonest finding 22 (35.5%). The most frequent abnormalities on electrocardiogram were sinus tachycardia 46 (74.2%) and $S_1Q_3T_3$ pattern 34 (54.8%). Echocardiography showed evidence of right ventricular dysfunction in 59 (95.16%) patients, right ventricular (RV) hypokinesis in 47 (75.8%) and right ventricular dilatation in 47 (75.8%). On spiral CT chest, embolus was visualized in the main pulmonary artery in 19 (30.6%) patients, right pulmonary artery in 40 (64.6%) and left pulmonary artery in 44 (71%) patients. All patients were anti-coagulated with either intravenous heparin 40

Shah I.[1]
Department of Cardiology, Postgraduate Medical Institute, Lady Reading Hospital, Peshawar

Shahzeb[2]
Resident, Department of Cardiology, Postgraduate Medical Institute, Lady Reading Hospital, Peshawar

Faheem M.[3]
Senior Registrar, Department of Cardiology, Postgraduate Medical Institute, Lady Reading Hospital, Peshawar

Kashif K.[4]
Resident, Department of Cardiology, Punjab Institute of Cardiology, Peshawar

Hafizullah M.[5]
Professor and Head of Department and VC KEMU, Department of Cardiology, Postgraduate Medical Institute, Lady Reading Hospital, Peshawar

Nisar M.[6]
Medical officer, Intensive Care Unit, Postgraduate Medical Institute, Lady Reading Hospital, Peshawar

Rafiullah[7]
Resident, Department of Cardiology, Postgraduate Medical Institute, Lady Reading Hospital, Peshawar

(64.5%) or enoxaparin 22 (35.5). In addition intravenous thrombolytic therapy (streptokinase) was given in 15 (24.2%) patients and 6 (9.7%) patients underwent surgical embolectomy. Warfarin was given in 49 (79%) patients at hospital discharge. During ICU stay various complications developed including cardiogenic shock 12 (19.4%), severe hypoxemia 40 (64.5%), right heart failure 24 (38.3%) and nosocomial infection 12 (19.4%). Ten 16.1% patients died mainly due to cardiogenic shock and refractory hypoxemia.

Conclusion: Pulmonary embolism is a life threatening disease with certain predisposing risk factors and nonspecific clinical presentation. Early diagnosis and aggressive treatment improves the clinical outcome.

Keywords: Pulmonary embolism, Risk factors, Outcome.

Introduction

Pulmonary embolism (PE) is a relatively common and potentially life threatening condition associated with considerable morbidity and mortality.[1] Infect, an estimated 10 percent of symptomatic patients with PE die within one hour of its onset. The exact incidence of PE remains unknown because the diagnosis is not suspectted in the vast majority.[2,3] The prevalence of PE among hospitalized patients in the United States, according to data collected between 1979 and 1999, was 0.4%.[4,5] Though only 40 – 53 per 100,000 persons were diagnosed with PE per year, the annual incidence in the United States was estimated at 600 000 cases with a related mortality of almost 200,000 deaths.[6]

PE is a difficult diagnosis that may be missed because of non-specific clinical presentation. It is suspected by the presence of common symptoms (including breathing difficulties, chest pain on inspiration and palpitations) and clinical signs including low blood oxygen saturation, tachypnea and tachycardia.[1,7,8] It occludes pulmonary arterial bed and by doing so leads to acute life – threatening condition due to a potentially reversible right ventricular failure. Early diagnosis is fundamental, since immediate treatment is highly effective. Depending on the clinical presentation, initial therapy is primarily aimed either at life – saving restoration of flow through occluded pulmonary arteries (PA) or at the prevention of potentially fatal early recurrences. Both initial treatment and long-term anticoagulation that is required for secondary prevention must be justified in each patient by the results of an appropriately validated diagnostic strategy.[7]

Despite recent advances in prophylactic, diagnostic and therapeutic modalities, PE is still one of the important causes of in – hospital morbidity and mortality.[9,10] This could be partly due to its nonspecific symptoms and lack of specific physical signs. Not infrequently, these patients require admission in the ICU because of hemodynamic instability or severe hypoxemia. In 2002, 101 000 patients with primary diagnosis of PE were admitted to acute care hospitals in the United States with a case fatality rate of 15% at 3 months.[11] Previous reports have described variable outcome of patients with PE. Reported mortality rates ranged from 8.1% in stable patients to 25% in those with cardiogenic shock and 65% post-cardiopulmonary resuscitation.[12]

Pulmonary embolism in Pakistan remains largely unrecognized, under – diagnosed and undertreated clinical problem amongst hospitalized patients due to non-availability of objective tests and lack of awareness among physicians.[13] There are very few studies from Pakistan on this subject. We conducted this study to evaluate the risk factors, clinical features, management strategies and outcome of patients with pulmonary embolism.

Material and Methods

This descriptive study was conducted from January 2007 to Dec 2011 in the intensive care unit (ICU), postgraduate medical institute, Govt. Lady Reading hospital Peshawar. Hospital ethical committee approved study protocol. A retrospective chart review of all hospitalized patients diagnosed with pulmonary embolism was carried out using a computerized database. At our institution, diagnosis of PE is suspected by the presence of dyspnea, pleuritic chest pain, haemptysis and tachypnea and in ventilated patients un-explicated hypoxemia, shock and arterial hypotension. The diagnosis of PE is confirmed by spiral computed tomography (CT) scan showing one or more filling defects or obstruction in the pulmonary artery or its branches. The diagnosis is also done when echocardiography shows a direct visualization of a thrombus in the pulmonary artery. The spiral CT scan is performed after correction of hemodynamic instability (using fluid resuscitation and/or catecholamine) and improvement of hypoxemia (using mechanical ventilation, high fraction of O_2). Massive PE (high risk) was defined as the presence of hemodynamic instability: arterial hypotension and cardiogenic shock whereas submissive PE

(non high risk) was defined as stable hemodynamics in the presence of echocardiograpic right ventricular (RV) dysfunction based on RV dilatation (end diastolic diameter > 30 mm) or hypokinesis or abnormal movement of the inter-ventricular septum with or without tricuspid regurgitation. Arterial hypotension is defined as a systolic arterial pressure < 90 mm Hg or a drop in systolic arterial pressure of at least 40 mm Hg for at least 15 min. Shock was diagnosed when a patient had arterial hypotension, tissue hypoperfusion and hypoxia including an altered level of consciousness, oliguria, or cool, clammy extremities.

For all included patients, a data entry form was designed to collect demographic, clinical, radiological and outcome data on admission and during ICU stay. Following data were collected: age, gender, heart rate, respiratory rate before mechanical ventilation, blood pressure, use of inotropic drugs, the presence of shock and cardiac arrest. Moreover, risk factors (immobility, recent surgery within 1 week, comorbid medical conditions, congestive heart failure, chronic obstructive pulmonary disease (COPD), cancer etc.) were also collected. The use of prophylactic anticoagulant agents and the clinical manifestations associated with the PE were also recorded for each patient. Biochemical parameters measured on admission and during the ICU stay including arterial blood gases, D-diemer level, urea and creatinine were recorded.

In our study, the presence of arterial hypoxemia was defined by arterial oxygen saturation in room air ≤ 92%. In patients receiving mechanical ventilation, arterial hypoxemia was defined as a PaO_2 / FiO_2 ratio < 300.

Findings on the reports of chest X-rays, ECGs, echocardiography, duplex ultrasound of lower limbs and spiral CT chest were recorded. Chest X-rays were analysed by a radiologist who is blinded to the patient's diagnosis. Therapeutic agents given including un-fractionated heparin, low molecular weight heparin, thrombolytic agent and warfarin were noted. Use of surgical embolectomy and caval filters implantation were noted. During the ICU stay, complications including respiratory failure, RV failure, nosocomial infections, thrombocytopenia, gastrointestinal bleeding and cerebral hemorrhage were recorded. Patients who died in the ICU and in the hospital were recorded.

The Statistical Package for Social Science SPSS version 19 was used for data analysis. Results were expressed as mean ± standard deviation for numerical variables and frequencies (percentages) for categorical variables in the forms of tables.

Results

A total of 62 patients were included in the study. Among these 37 (60%) were male and 25 (40%) were female. The mean age (± SD) was 49.3 ± 13.14 years (range 29 – 75 years). Risk factors for thromboembolism were present in 49 (83%) patients. Among these, immobilization 43 (69.4), recent major surgery 25 (40.3), obesity 35 (56.5), active malignancy 16 (25.8) and recent trauma 15 (25.2) were more frequent. Other important risk factors included previous history of deep vein thrombosis (DVT) 13 (21), current DVT 15 (24.2), lake of prophylaxis against DVT and hypercoagulable state 6 (9.7). These demographics and risk factors are presented in Table 1.

All patients were symptomatic at presentation. Major presenting symptoms were dyspnea 59 (95.2%), chest pain 28 (45.2%) and cough with haemptysis 9 (14.5%). Vital signs at presentation were; heart rate 117.41 ± 14.36 beats / min, systolic blood pressure 98.36 ± 15.38 mm Hg and respiratory rate 21.06 ± 2.7 breaths / min. Eighteen (29.03%) patients had lung crackles on auscultation and 15 (24.2%) had leg swelling due to deep venous thrombosis. Forty (64.5%) patients had hypoxia and 12 (19.4%) patients were hypotensive on presentation. The clinical features of the study group on admission are shown in Table 2.

Chest X-ray was performed in all patients and it was normal in 28 (45.2%) patients. The chest radiograph was abnormal in 34 (54.8%) patients with cardiomegaly as the commonest finding 22 (35.5%). Other features included focal oligaemia13 (21%), pleural effusion 6 (9.7%), atelectasis 6 (9.7%), elevated hemidiaphragm 3 (4.8%) and wedge shaped opacity 3 (4.8%). An electrocardiogram was performed in all patients. The most frequent abnormalities recorded were sinus tachycardia 46 (74.2%), $S_1Q_3T_3$ pattern 34 (54.8%), complete or incomplete right bundle branch block 15 (24.2%), T-wave inversion over the right precordial leads (T-wave inversion in leads $V_2 – V_3$) 19 (30.6%) and atrial fibrillation 6 (9.7%). Echocardiography was performed in all patients with evidence of right ventricular dysfunction in 59 (95.16%). Right ventricular (RV) hypokinesis was found in 47 (75.8%) patients while right ventricular dilatation in 47 (75.8%), tricuspid regurgitation in 56 (90.3%), severe pulmonary hypertension in 34 (54.8%) and paradoxical interventricular septal shift in 16 (25.8%) patients. All patients underwent spiral CT chest. The embolus was visualized in the main pulmonary artery in 19 (30.6%) patients, right pulmonary artery in 40

Table 1: Risk factors and co-morbid conditions present in patients with pulmonary embolism.

Feature	No.	Percentage (%)	Mean ± SD
Mean age, years (SD)			49.3 ± 13.41
Female sex	25	40.3	
Obesity	35	56.5	
Immobility	43	69.4	
Recent surgery	25	40.3	
Current DVT	15	24.2	
Previous history of DVT	13	21	
History of PE	6	9.7	
Recent significant trauma	15	24.2	
Neurologic paralysis	6	9.7	
Malignancy, active	16	25.8	
History of heart failure	10	16.1	
COPD	6	9.7	
Current smoker	19	30.6	
Hypertension	15	24.2	
Diabetes Mellitus	12	19.4	
Oral contraceptives	6	9.7	
Known hypercoagulable state	6	9.7	
Pharmacological prevention of venous thromboembolism	6	9.7	

Table 2: Signs and symptoms at presentation in patients with pulmonary embolism.

Feature	No.	Percentage	Mean ± SD
Symptoms			
Dyspnea	59	95.2	
Pleuritic chest pain	28	45.2	
Cough with haemptysis	9	14.5	
Altered mental status	6	9.7	
Fever (> 38)	12	19.4	
Vital signs at presentation			
Heart rate, beats / min			117.41 ± 14.36
Respiratory rate, breaths / min			21.06 ± 2.7
Systolic blood pressure, mm Hg			98.36 ± 15.38
Diastolic blood pressure, mm Hg			50.48 ± 7.55
Others Findings			
Extremity swelling suggestive of DVT	15	24.2	
Respiratory crackles	18	29.03	

Table 3: Findings on Investigations in patients with pulmonary embolism

Feature	No	Percentage (%)
Chest Radiograph		
Normal	28	45.2
Cardiomegaly	22	35.5
Westermark sign	3	4.8
Hampton hump	3	4.8
Atelectasis	6	9.7
Oligemia	13	21
Pleural effusion	6	9.7
Elevated hemidiaphragm	3	4.8
Wedge-shaped opacity	3	4.8
Electrocardiogram		
Sinus tachycardia	46	74.2
$S_1Q_3T_3$ pattern	34	54.8
Complete or incomplete RBB	15	24.2
RV strain pattern	19	30.6
Atrial fibrillation	6	9.7
Echocardiography		
RV dysfunction	59	95.16
Right ventricular dilatation	47	75.8
Right ventricular (RV) hypokinesis	47	75.8
Tricuspid regurgitation	56	90.3
Severe pulmonary hypertension	34	54.8
paradoxical interventricular septal shift	16	25.8
CTA		
Main pulmonary trunk	19	30.6
Right pulmonary artery	40	64.4
Left pulmonary artery	44	71
Pulmonary infarction	10	16.1
Duplex Ultrasonograpy showing thrombus in the deep veins of the extremities.	25	40.3

(64.6%) and left pulmonary artery in 44 (71%) patients. Ten (16.1%) patients were noted to have pulmonary infarction. Twenty – five (40.3%) patients had thrombus in the deep veins of the lower extremities on duplex ultrasonography. Arterial blood gases (ABG) were done in all patients. Respiratory alkalosis (pH > 7.45, $PaCO_2$ < 35 mmHg) was the commonest finding, present in 50 (86%) patients. D-dime assay was tested in all patients, being positive (value > 500 ng/l) in 38 patients (61.3%). Mean blood urea and creatinine were 36.3 ± 10.5 and 1.3 ± 0.2 respectively. Findings on investigations are summarized in tables 3 and 4.

Table 4: Findings on Investigations in patients with pulmonary embolism.

Feature	Mean ± SD	Frequency / Percentage (%)
Arterial Blood Gases		
PH	7.48 ± 0.09	
PaO2 (mm Hg)	58.48 ± 2.67	
PaC02 (mm Hg)	33.33 ± 2.27	
SaO2%	89.98 ± 5.65	
Respiratory alkalosis		50 (80.6)
Others investigations		
D-diemer		38 (61.3)
Blood Urea	36.3 ± 10.5	
Creatinine	1.3 ± 0.2	

All patients were anti-coagulated with either intravenous heparin 40 (64.5%) or subcutaneous low molecular weight heparin (LMWH), enoxaparin 22 (35.5). In addition intravenous thrombolytic therapy (streptokinase) was given in 15 (24.2%) patients and 6 (9.7%) patients underwent surgical embolectomy. Fifteen (24.2%) patients required mechanical ventilation for respiratory failure. Warfarin was given in 49 (79%) patients at hospital discharge while 3 (4.8%) patients underwent caval filter implantation due to contra-indication to anticoagulant. The main therapeutics used in the treatment of these patients are given in table 5.

Table 5: Main therapeutics used in treatments of patients with pulmonary embolism

Therapeutics	No.	Percentage (%)
Un-fractionated heparin	40	64.5
Low molecular weight heparin	22	35.5
Streptokinase	15	24.2
Warfarin	49	79
Dopamine	24	38.3
Dobutamine	24	38.3
Mechanical ventilation	15	24.2
Surgical embolectomy	6	9.7
Caval filter	3	4.8

Mean stay in intensive care unit (ICU) and hospital were 10.9 ± 2.17 and 14.9 ± 1.9 respectively. During ICU stay various complications developed including cardiogenic shock 12 (19.4%), severe hypoxia 40 (64.5%), right heart failure 24 (38.3%) and nosocomial infection 12 (19.4%). Fifteen (24.5%) patients developed respiratory failure and needed ventilator support. Due to anticoagulant therapy, 4 (6.5%) patients have a bleeding complication, including one patient (1.6%), who has an intracranial hemorrhage. Moreover, 2 (3.2%) patients developed thrombocytopenia. Ten 16.1% patients died mainly due to cardiogenic shock and refractory hypoxemia. Major complications developed during ICU stay and mortality is given in table 6.

Discussion

Although PE can occur in patients without any identifiable predisposing factors, one or more of these factors are usually identified. The proportion of patients with idiopathic or unprovoked PE was about 20% in the International Cooperative Pulmonary Embolism Registry (ICOPER).[12]

Venous thromboembolism (VTE) is currently regarded as the result of the interaction between patient-related and setting – related risk factors.[1,15,16] These risk factors includes advanced age, prolonged immobility, recent major surgery, recent trauma, malignancy, obstructive lung disease, heart failure, hypercoagulable states and infrequent use of prophylactic

measures for venous thromboembolism.[13] The incidence of VTE increases exponentially with age and this is the case for both idiopathic and secondary PE.[17,18] The mean age of patients with acute PE is 62 years; about 65% of patients are aged 60 years or older. Eight – fold higher rates are observed in patients over 80 compared with those younger than 50.[19] Identification of the presence and estimation of the relative significance of predisposing factors may be helpful both in the assessment of clinical probability for diagnostic purposes and for decisions regarding primary prevention. However, according to a recent survey performed in 358 hospitals across 32 countries, only 58.5% and 39.5% patients at risk of VTE due to medical or surgical causes, respectively, received adequate prophylaxis.[20] In our study mean age was 49.3 ± 13.41, obesity 35 (56.5%) and recent surgery was 25(40.3%). Other major risk factors present in our patients were prolonged immobility due some bone fractures or neurological paralysis, malignancies and heart failure. Moreover, we found in the current study that those patients who do not receive pharmacological prevention of venous thromboembolism developed PE more frequently.

Table 6: Main complications observed in patients with pulmonary embolism.

Complication	No.	Percentage (%)	Mean ± SD
Hypoxemia	40	64.5	
Shock	12	19.4	
Respiratory failure	15	24.2	
Right heart failure	24	38.3	
Cardiac arrest			
Nosocomial infections	12	19.4	
Hemorrhage	4	6.5	
Thrombocytopenia	2	3.2	
Mean ICU stay			10.9 ± 2.17
Mean hospital stay			14.9 ± 1.9
Mortality	10	16.1	

Current guidelines suggest replacing potentially misleading terms such as 'massive', 'sub-massive' and 'non-massive' with the estimated level of the risk of PE – related early death. PE can be stratified into several levels of risk of early death (understood as in – hospital or 30 – day mortality) based on the presence of risk markers. For practical purposes, risk markers useful for risk stratification in PE can be classified into three groups. Clinical markers include shock and hypotension. Markers of RV dysfunction includes RV dilatation, hypokinesis or pressure overload on echocardiography, RV dilatation on spiral computed tomography, BNP or NT – pro BNP elevation and elevated right heart pressure at RHC. Markers of myocardial injury include cardiac troponin T or I. Immediate bedside clinical assessment for the presence or absence of clinical markers allows stratification into high-risk and non-high – risk PE.[1,21,22] High-risk PE is a life – threatening emergency requiring specific diagnostic and therapeutic strategy (short – term mortality 15%).[23,14] Non-high – risk PE can be further stratified according to the presence of markers of RVD and/or myocardial injury into intermediate – and low – risk PE. Intermediate – risk PE is diagnosed if at least one RVD or one myocardial injury marker is positive. Low – risk PE is diagnosed when all checked RVD and myocardial injury markers are found negative (short – term PE – related mortality, 1%).[1] Our cohort included twenty – four (38.3%) patients with hypotension on presentation (High risk PE), the rest (62%) were haemodynamically stable (non-high risk PE).

Most of our patients presented with dyspnea (90%) and chest pain (45.2%) which is consistent with other studies.[23] PE is also suspected when unexplicated hypoxemia, shock and arterial hypotension are observed. Despite the vast array of physiological abnormalities associated with PE, hypoxemia is not a uniform finding.[24,25] In our study hypoxemia was documented in 64.5% of patients. Part of the reason could be that most patients who present with breathlessness and tachycardia are generally placed on supplemental oxygen before checking their pulse oximetry or arterial blood gases. When PE is suspected, diagnostic confirmation is needed. In fact, prompt and accurate diagnosis of PE greatly influences patient outcome.[1,8,9] In our study, the diagnosis of PE was made by spiral CT in all patients.

Spiral CT scan of chest has been considered a very good diagnostic tool due to its availability, non-invasiveness, rapidity and accurate results.[26-28] Studies have also suggested that this modality should replace pulmonary angiogram as a gold standard for the diag-

nosis of PE, particularly due to the invasiveness and the morbidity of the latter.[29] In our institution the spiral CT has replaced the pulmonary angiogram as the diagnostic procedure of choice for PE. Trans-thoracic echocardiography is of tremendous value in the setting of major pulmonary emboli, due to its ability to diagnose the severity and impact of the embolism and response to therapy.[30] Echocardiograpic findings in patients with major PE are signs of right ventricular pressure overload which include right ventricular dilatation, pulmonary artery dilatation, paradoxical septal motion, right ventricular hypokinesis and tricuspid regurgitation.[31] All our patients had echocardiography and 95.5% of them had signs of right ventricular dysfunction. In our study, common x-rays findings included cardiomegaly, lung oligemia and atelectasis which are consistent with other studies. Common findings on ECG included sinus tachycardia, $S_1Q_3T_3$ and RV overload which is consistent with other studies.[32] On ABG analysis 80.6% of patients had respiratory alkalosis ($pH > 7.45$ and $PaCO_2 < 35$ mmHg) which is due to the tachypnea resulting from the sudden occlusion of the pulmonary arterioles by the thromboembolism.[33]

Haemodynamic and respiratory support is necessary in patients with suspected or confirmed PE presenting with shock or hypotension. A modest fluid challenge may help increase cardiac index in patients with PE, low cardiac index and normal blood pressure. Isoproterenol, norepinephrine, dopamine and dobutamine can be used as inotropic agents.[1] In our study, all patients received saline infusion. Oxygen supplement, inotropic agents, intubation and mechanical ventilation are instituted when necessary for respiratory and haemodynamic support.

Anticoagulant treatment plays a pivotal role in the management of patients with PE. Heparin, low molecular weight heparins are administered initially. Considering the high mortality rate in untreated patients, anticoagulant treatment should be considered in patients with suspected PE while waiting definitive diagnostic confirmation. Anticoagulation therapy with heparin reduces mortality rates from 30% to less than 10%.[1,8,9] Thrombolytic therapy for MPE has been studied extensively. It produces more rapid clot lysis as compared to heparin therapy alone.[34,35] It is uniformly acknowledged as the treatment of choice for haemodynamically unstable patients without absolute contraindications. In case of sub massive PE the data is less clear. Recently, two studies of sub-massive pulmonary embolism have shown a mortality advantage and reduced incidence of recurrent venous thromboembolism with intravenous thrombolytics.[36,37] Among the fifteen patients that received thrombolytics, two patients died and there was one bleeding complication.

Surgical embolectomy is generally reserved for patients with refractory shock despite aggressive medical therapy, ongoing cardiac arrest, or patients with contraindications to thrombolytics.[38-40] Recently a single center experience with massive PE without shock had a 86% survival at one year.[40] All six patients who underwent surgical embolectomy survived till discharge. Catheter thrombectomy is especially useful in the presence of an increased bleeding risk or if surgical embolectomy is not an option.[41-43] At hospital discharge warfarin should be given at doses adjusted to maintain a target INR of 2.5 (range 2.0 – 3.0) .On the other hand, venous filters may be used when there are absolute contraindications to anticoagulation and a high risk of VTE recurrence.[1] In our cohort, all patients were started on treatment once the diagnosis was suspected. Intravenous un-fractionated heparin was used in 40 (64.5) cases and low molecular weight heparins were used in 22 cases (35.5%). Fifteen 24.2% of patient received thrombolysis with streptokinase. Warfarin was used in 49(79%) patients and 4 patients underwent caval filters implantation due to warfarin contraindication. The outcome of patients with PE is quite variable depending primarily on the hemodynamic status and embolus size.[44] The overall in – hospital mortality rate for the 62 patients studied in our center was 16.1% which is in accordance with the published literature.[13]

Conclusion

Pulmonary embolism is life threatening but preventable disease. We must maintain a high index of suspicion for its diagnosis in patients presenting with shock, refractory hypoxia unexplained tachypnea and tachycardia. Echocardiography is a good diagnostic and prognosticating tool for it with an excellent sensitivity. Once diagnosed, early and aggressive management can result in improved survival.

Limitations

The limitations of the study were the retrospective charts review so the complete data of all patients were not available. The rationale for giving thrombolytics was not uniform and was dependent on physician discretion.

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Amjid Khan

Statistical Officer CPSP (Peshawar Branch).

Alamger Khan

Assist Professor, Department of Statistics, University of Peshawar.

Waris Khan

Statistical Officer, Quid-e-Azam College of commerce and business administration, University of Peshawar

Anayat Ullah

Statistical Officer, Department of Statistics, University of Peshawar.

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