Original Article

Clinical profile, management and outcome of pulmonary embolism in Shahid Gangalal National Heart Centre, Kathmandu, Nepal

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ABSTRACT

Background and aims: Pulmonary embolism (PE) is associated with a significant mortality and morbidity. We aim to study clinical profile, management and outcome of PE at Shahid Gangalal National heart Centre, Kathmandu, Nepal.

Methods: It was a retrprospective, single centre study, conducted from January 2015 to December 2016. Haemodynamics was used for risk Simplified, PESI score, predisposing factors, symptoms, clinical features at the time of admission, ECG features, echocardiogram, treatment received and the outcome were reviewed.

Results: During the study period 23 cases of PE were admitted. Nine were males and 14 were females. Eleven patients were diagnosed as provoked PE. High risk PE was diagnosed in four patients, Non-high risk in 19 patients. The most common clinical presentation was shortness of breath. The most common finding in ECG is sinus tachycardia followed by ST-T changes in V1-V3. Eight patient had SPO2 less than 90%. Most of the patients had a normal chest radiograph. Echocardiography revealed dilated RA and RV in 20 patients.

Results: All high risk PE patients were thrombolyzed with streptokinase. All patients who were diagnosed as Non-high risk PE were treated with LMWH. All the patients were treated with oral anticoagulants. Mean hospital stay was 9.7 ± 4.9 days. Two patients died during hospital stay. S-PESI score was 1.4 ± 0.9 respectively. Mean warfarin dose at the time of discharge was 5.9 ± 1.6 mg.

Conclusion: PE is an under diagnosed clinical problem world over. Suspicion is the most important part to come to the diagnosis of PE.

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1. Introduction

Pulmonary embolism (PE) is a common clinical problem, often under recognized, under diagnosed and potentially lethal condition. Most patients who succumb to pulmonary embolism do so within the first few hours of the event. Ten percent of PE is fatal in the first hour.[1] PE presents with a wide clinical spectrum, from asymptomatic disease to life threatening massive PE that causes hypotension and cardiogenic shock. Increasing availability of newer imaging modalities are likely to improve its diagnosis. Despite diagnostic advances, delays in PE diagnosis are common and represent an important issue.[2]

Due to the lack in adequate specificity of the clinical presentation and the investigations including electrocardiography, chest radiography, and analysis of arterial blood gases cannot be relied on to confirm or rule out PE.[3] A high index of suspicion is necessary to consider the diagnosis.[1]

In spite of rapid advances in the diagnosis and management of PE, the exact epidemiology of PE in Nepal is largely unknown. In this study, we describe our experience with the diagnosis and management of patients with PE.

2. Methods

All the patients who were admitted in our hospital with the diagnosis of Pulmonary embolism were included in this study. A retrospective study of all patients admitted with the diagnosis acute pulmonary embolism at our center from January 2015 to December 2016 were included in this study. Their clinical presentation, investigation and management were analyzed. Patients were diagnosed to have pulmonary embolism if there is evidence of thrombus in CT pulmonary angiogram. Patients with pulmonary

embolism were classified as High risk if there was evidence of hemodynamic compromise (defined as systolic BP <90 mmHg) and as Non-high risk, if there is no hemodynamic compromise. If patients had predisposing factors the PE id defined as provoked. If there was no predisposing factor it is called as unprovoked. Echocardiogram was done by the cardiologist. Patients who were diagnosed as massive PE were for thrombolysed using streptoki-nase Rest of the patients were anticoagulated with low molecular weight heparin. All patients were treated with warfarin. Ethical approval for the study was obtained from Institutional Review Board of SGNHC. S-PESI[4] score was calculated as the PESI criteria and used for risk stratification as shown in Table 1. Predisposing factors, Symptoms, clinical features at the time of admission, ECG features, echocardiogram, treatment received and the outcome were reviewed.

3. Results

During the study period twenty-three patients were diagnosed as acute PE and were included in the study. Their mean age was 42.1.1 years. Among 23 patients, 9 were males and 14 were females.

Out of 23 patients, High risk PE was diagnosed in 4 patients, Non-high risk in 19 patients. Eleven patients were diagnosed as provoked PE, four patients had a history of surgery with in two weeks, three patients had carcinoma, two patients had EPS and RFA within two weeks and one had undergone PPCI with in two weeks as shown in Table 2.

The most common clinical presentation was shortness of breath followed by chest pain associated with shortness of breath as shown in Table 3.

The most common finding in ECG is sinus tachycardia.

20 patients followed by RV strain pattern 14 patients, S1Q3T3 pattern in 11 patients and RBBB three patients as shown in Table 3. Eight patient had SPO2 less than 90%. Troponin I was positive in all patients.

Eighteen patients had a normal chest radiograph. Of the remaining five patients, one had wedge shaped opacity suggestive of pulmonary infarct, one had lung collapse and three had pleural effusion.

Echocardiography revealed dilated RA and RV in 20 patients.

All high risk PE patients based on hemodynamics were throm-bolyzed with streptokinase. All patients who were survived were

Table 1
Simplified PESI score. *Source:* 2014 ESC Guidelines on the diagnosis and management of acute pulmonary embolism. Eur Heart J. 2014 14;35(43):3033–69.

Age	1 point
Cancer	1 point
Chronic heart failure	
COPD	1 point
Pulse rate $\geq$110 bpm	1 point
Systolic BP <100 mmHg	1 point
Arterial oxyhaemoglobin saturation <90%	1 point
Risk stratification	
0 points = 30-day mortality risk1.0% (95% CI 0.0%–2.1%)	
$\geq$1 point(s) = 30-day mortality risk 10.9% (95% CI 8.5%–13.2%)	

Table 2
Predisposing factor.

Predisposing factor	No. of patients n = 23
Surgery	4
Post primary PCI	1
Post EPS + RFA	2
Carcinoma	3
Air travel	1

Table 3
Clinical features and diagnostic methods in patients with PE.

Clinical features and diagnostic method	
Chest pain	12
Shortness of breath	11
Chest pain and shortness of breath	10
ECG	
Sinus tachycardia	20
RV strain	14
S1Q3T3	11
RBBB	3
Chest X-ray	
Normal	18
Wedge shaped opacity	1
Lobar collapse	1
Pleural effusion	3
Echocardiogram	
Dilated RA and RV	20

diagnosed as Non-high risk PE were treated with LMWH. All the patients were treated with oral anticoagulants.

Mean hospital stay was 9.7 ± 4.9 days. Two patients died during hospital stay. S-PESI score was 1.4 ± 0.9 respectively. Six patients had S-PESI score of zero, Six patients had S-PESI score of one, nine patients had two and two had S-PESI score of three. Mean warfarin dose at the time of discharge was 5.9 ± 1.5 mg.

Two patients who died were of unprovoked PE. One had S-PESI score was zero, another patient S-PESI score was one and presented in shock.

4. Discussion

Suspicion is the most important part to come to the diagnosis of PE. PE is a cardiovascular emergency with non-specific sign and symptom.[5] Clinical suspicion of this disease is of paramount importance in guiding diagnostic testing. Diagnosis of PE is more difficult than treatment.[1]

Risk factors for venous thromboembolic disease and pulmonary embolism is well known. These risk factors are present in almost 96% of patients with confirmed venous thromboembolic disease.[6] In our study 47.8% patients had provoked PE. In an Indian[1] series only 34.2% of patients had a definite risk factor. But the Interna-tional Cooperative Pulmonary Embolism Registry (ICOPER) states that about 20% patients were idiopathic or unprovoked PE.[7] In a Pakistani[8] Study risk factors for thromboembolism were identified in 22 patients (73%), immobilization and recent surgery as the commonest recognized factors.

Sign and symptoms of PE are non-specific. Unexplained dysp-nea and chest pain are the most frequent symptoms, and sudden onset dyspnea and pleuritic chest pain are the most typical. In our study most of the patients presented with shortness of breath and chest pain. In an Indian study shortness of breath was the most common followed by syncope and chest pain.[1]

The ESC guidelines recommend thrombolytic therapy be used in high risk PE patients, except in the presence of major contraindica-tions due to bleeding risks. Same guideline recommends LMWH over UFH for patients with acute non-high risk PE. We follow the same guideline and were able to thrombolyse all our high risk patients.

The ECG in addition to clinical acumen, can help to direct the physician towards the diagnosis. No isolated ECG abnormality is definitively associated with PE. Several of the more frequently described associations include: normal ECG, sinus tachycardia, complete and incomplete RBBB, axis changes, transition zone shift, low voltage complexes, ST segment and T-wave changes, S1Q3T3 pattern, P-pulmonale and atrial arrhythmias.[10,11] In our cases Sinus

tachycardia was the most common followed by ST_T changes in V1-V3 and S1Q3T3. Our finding was similar to the Indian study.[1]

Stein et al.[9] found that the most common chest X-ray finding was atelectasis or parenchymal abnormality. It is a fact, however, that one cannot depend on chest X-ray for the diagnosis of pulmonary embolism. Although the chest radiograph cannot be used to diagnose or exclude PE, it contributes to the non-invasive diagnostic assessment of PE through the exclusion of disease processes that may mimic PE. In our study most of the patients had normal chest X-ray like the study from India and Pakistan.[8]

The common echocardiographic feature of PE is dilated pulmonary artery, dilated right atrium, right ventricular hypokinesis, right ventricular enlargement, reduced left ventricular size, McConnell's sign, Flattening of the intraventricular septum or paradoxical septal motion, direct visualization of thrombus in the right heart or pulmonary artery and distention of the inferior vena cava with loss of normal respiratory variation.[11] Echocardiography is a useful tool in identifying high risk patients such as those with right ventricular dysfunction, free floating thrombus and persistent pulmonary hypertension. Most of our patients had dilated RA and RV in echocardiogram. Right ventricular dilatation is found in at least 25% of patients with PE, and its detection, either by echocardiography or CT, is useful in risk stratification.[12] In an Indian study 88.5% had evidence of RV dysfunction and 85.7% had pulmonary arterial hypertension.[1] In a study done in Pakistan only 73% patients had echocardiography done and all of them had signs of right ventricular dysfunction (100% sensitivity). But in our study RV function was not reported.

Retrospective, single center study without long term follow up were the limitations of our study.

5. Conclusion

PE is an under diagnosed clinical problem world over. Diagnosis of PE is tricky because of non-specific sign and symptom. Suspicion is the most important part to come to the diagnosis of PE.

Conflict of interest

The authors declared that there is no conflict of interest.

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