PULMONARY EMBOLISM - AN ENIGMA

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ABSTRACT

Pulmonary embolism is considered a rare disease in India. The diagnosis is likely to be missed as its presentation is often enigmatic. Ten cases of pulmonary embolism are presented. Nine patients presented with acute pulmonary embolism, while one had chronic pulmonary emboli with cor pulmonale. The diagnosis was based on pulmonary angiography (1 case), lung perfusion/ventilation scan (2 cases), autopsy (2 cases) and characteristic ECG, X-ray chest and clinical features (5 cases). The initial diagnosis was often incorrect (7/10 cases). Common presenting clinical features were: sudden breathlessness (10/10), tachypnoea (10/10) and tachycardia (8/10); chest pain (3/10) was uncommon and hemoptysis was not seen in any patient. ECG revealed isolated sinus tachycardia in 2 cases, S₁ Q₃ T₃ pattern in 2, and right axis with ST-T wave changes in 5 patients. X-ray chest showed either an area of oligemia or non-specific opacities in 7 patients and was normal in three cases. Advances in specific diagnostic modalities coupled with heightened awareness should contribute to an early detection of pulmonary embolism, rarity of which may be more apparent than real.

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KEY WORDS : Pulmonary thromboembolism; Acute cor pulmonale.

Introduction

Venous thrombosis and pulmonary embolism (PE) constitute some of the most common cardiopulmonary illnesses in North America and Europe [1]. It is estimated that in the United States 650,000 individuals have PE annually [2]. Only about 15% to 30% of patients with massive PE found at autopsy are identified during their life time [3]. Unfortunately, estimated rates for venous thrombosis and PE in population based studies have been reported in only a few countries. WHO is currently planning studies regarding the frequency of venous thrombosis and PE in different parts of the world [1]. There is no Indian data available, although it is generally felt the PE is a very rare illness in India. This impression may be partly due to the lack of diagnostic facilities available which leads to an underestimation. The diagnosis of PE is important as the mortality rate of undiagnosed and hence, untreated pulmonary em-

bolism is between 18% and 35% [3].

To diagnose PE accurately it is important to suspect the condition clinically and be aware of the limitations and utility of different diagnostic procedures.

Materials and methods

This study analyses the clinical features and investigations in cases of pulmonary embolism seen by the authors. These cases were in tertiary medical centers like Command Hospital (SC) Pune and Army Hospital, Delhi Cantt. The diagnosis of PE was based on one of the following criteria:-

- a. Clinical features suggestive of PE, supported by characteristic ECG and X-ray findings.
- b. Diagnostic pulmonary angiography or radioisotope pulmonary perfusion scan/ventilation scan.
- c. Autopsy findings of PE.

The facilities for pulmonary arteriography,

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radioisotope lung scans and colour doppler echocardiography have become available recently and could not be done in cases seen earlier.

Results

A total of 10 cases were seen comprising of 5 males and 5 females with a mean age of 47.4 years (range 24-73 years). The clinical features are shown in Table 1. The initial diag-

TABLE 1

Signs & symptoms in 10 cases of pulmonary embolism

Symptoms	
Breathlessness (single episode in four and recurrent in six)	10
Pleuritic chest pain	1
Precordial chest pain	2
Hemoptysis	0
Signs	
Heart rate > 100/min	8
Resp. rate > 26/min	10
Hypotension	2
Pleural rub	2
Pleural effusion	1
Calf tenderness	1
Varicose veins	1
Deep vein thrombosis	1

nosis was PE in only 3/10 cases, while in others, the first impression was pneumonia or left ventricular failure. A predisposing factor was seen in all the cases, malignancy being the commonest (Table 2). The classical ECG changes of S₁ Q₃ T₃ pattern were seen only in 2 cases, another 5 cases had features of right ventricular strain or acute right axis deviation and 2 showed sinus tachycardia (Table 2). X-ray findings were often non-specific and localised oligemia was the commonest suggestive finding (Table 2). The diagnosis was confirmed in 2 cases on pulmonary perfusion scan and mismatch with ventilation/X-ray, pulmonary angiography was diagnostic in one, and autopsy revealed the PE in 2 fatal cases. Without these special procedures the diagnosis may have been missed in these 5 cases. Five cases (Table 2. No. 1 to 5) have

been diagnosed since January 1993 and the summaries of 2 cases are given below, to highlight the pitfalls in diagnosis and role of specialised investigations.

CASE NO.4

A 40 year old obese female, a treated case of carcinoma cervix, was admitted to CH(SC) with a diagnosis of pneumonia left side. History revealed a sudden onset of breathlessness of 3 days duration, mild dry cough and palpitations. Examination: pulse 130/min, respiration 44/min, no fever, few crepts left mammary and axillary region. Hb 12g/dl, TLC 6300/cmm, P₆₃ L₂₂ E₃ M₂. X-ray chest - nonhomogenous opacities left upper and mid-zone. (Fig 1). ECG - tachycardia, non-specific T inversion V₁ to V₆ with no right axis deviation or specific pattern to suggest PE. Arterial blood gases showed pO₂ 67.3 mmHg, pCO₂ 17.6 mmHg and pH 7.53. In view of a normal temperature, no leucocytosis, sudden onset of tachypnoea and tachycardia without an obvious cause, a diagnosis of PE was considered and heparin was started. 2D Echo doppler done the next day showed a large right ventricle (RV), paradoxical interventricular septum (IVS) motion with normal valves and left ventricle, compatible with PE. Pulmonary catheterisation and arteriography done the next day showed elevated pressures in the right atrium (11 mmHg), right ventricle (75/10 mmHg) and pulmonary artery (76/20 mmHg) with delayed appearance of contrast in the left pulmonary artery and persistent lucent defect in its upper branch (Fig.2).

CASE NO.1

A 45 year old civilian male, being treated for congestive cardiac failure in civil hospital was admitted with breathlessness of fourteen months duration accompanied by swelling feet. History revealed that he was in perfect health when he suddenly developed breathlessness at rest which lasted for 48 hours, accompanied by precordial non-pleuritic chest pain for 2 hrs. This was followed by gradually progressive dyspnoea on effort, unaccompanied by cough, wheezing or palpitations. This was exacerbated by acute transient attacks of breath-

Fig. 1 Non-homogenous opacities left upper and mid zone.

TABLE 2

Associated conditions & special investigations in 10 cases of pulmonary embolism

Case No.	Age	Sex	Initial Diagnosis	Associated Conditions	ECG Changes	X-Ray Chest	Special Tests/Autopsy
1	45	M	CCF ?RHD ? IHD	Varicose veins	RVH, Rt axis	RVH, Lt lung oligemia	ECHO-RVH, ventilation perfusion mismatch
2	70	F	LVF	Ca ovary malignant Pleural effusion	Tachycardia	Rt-NAD, Lt-effusion	Perfusion scan - Multiple defects right lung
3	73	M	LVF, Ac.MI	#Neck femur	ST $\uparrow$ T $\downarrow$ in V ₁ V ₂ V ₃	Rt. lung oligemia	S. bilirubin transiently raised - DIED
4	40	F	Pneumonitis	Old Ca cervix	Tachycardia	Lt. lung opacities	Pulm angio-Filling defect Lt. pulm. artery? ECHO-RV pressure $\uparrow$
5	63	F	PE	#Neck femur	S ₁ Q ₃ T ₃	Rt lung oligemia	Recovered with normal ECG and X-Ray
6	40	F	PE	Ca breast	S ₁ Q ₃ T ₃	Dilated Rt. pulm. art.	Recovered with normal ECG and X-ray
7	34	M	PE	Diss. Ca	Tachycardia	Ca lung	Autopsy - Pulm. embolism
8	33	F	Pneumonitis	RHD	*Acute Rt. axis	RHD, Lungs clear	DIED
9	52	M	Rt. ventricular M.I.	Ac.MI	Acute Rt. axis	Rt. lung opacities	Autopsy - Massive pulmonary embolism S. bilirubin transiently raised
10	24	M	Pneumonitis ? PE	Adeno Ca lung	Tachycardia	Rt. Pl. effusion	Inferior vena cava thrombosis Hemorrhagic pleural effusion

Ca = Carcinoma, # = Fracture
LVF = Left ventricular failure

RVH = Right ventricular hypertrophy
PE = Pulmonary embolism

lessness at rest. Exam: pulse 84/min, RR-20/min, JVP raised till angle of jaw and varicose veins in both lower limbs. There was evidence of gross pulmonary artery hypertension, right ventricular hypertrophy and tricuspid regurgitation. ECG - right axis + 140°, R wave 8 mm in V₁, T inversion on V₁ V₂ V₃. X-ray chest - prominent pulmonary conus, mild cardiomegaly, and oligemia of left lung field (Fig. 3). Echodoppler: large RV, abnormal IVS motion, severe tricuspid regurgitation and

pulmonary arterial systolic pressure of 137 mmHg with normal left ventricle and left atrium. Arterial gases - pO₂ 68 mmHg, pCO₂ 30 mmHg, pH 7.43 Pulmonary function tests showed mild restrictive defect. Ventilation perfusion scan showed no perfusion on the left lung and multiple perfusion defects in right lung with normal ventilation (Fig. 4). The final diagnosis was chronic pulmonary embolism leading to cor pulmonale. The likely source was the varicose veins.

Fig. 2 Pulmonary angiography: Delayed appearance of contrast in left pulmonary artery, a lucent defect in left upper branch.

Discussion

The clinical suspicion of pulmonary embolism (PE) is a prerequisite for its diagnosis [1-4]. Unfortunately the symptoms of PE are often subtle and nonspecific [2,5]. Even when suspected, the diagnosis is difficult to confirm in India, as the facilities for pulmonary angiography and pulmonary scans are not easily available.

Our experience confirms that PE does occur in our setting. The condition is, however, often misdiagnosed as is obvious from the initial diagnosis in our patients. The most common clinical features at presenta-

Fig. 3 X-ray chest : Left pulmonary oligemia, prominent pulmonary conus.

tion were unexplained dyspnoea and tachycardia, and being non-specific, were often misinterpreted. It is significant that 5 cases were diagnosed since January 1993 when special investigations became available, whereas the other five were seen over a period of five years.

Fig. 4 Ventilation perfusion mismatch. Normal ventilation scan with absent perfusion left lung, and small defects right lung.

Most pulmonary emboli lyse completely, but occasionally the clots fail to resolve appropriately, and lead to a syndrome of chronic thromboembolic pulmonary hypertension [6]. The exact incidence of cases characterised by failure of clot resolution is not known, but it is postulated that this occurs in 0.01% to 0.1% cases of pulmonary embolism [6,7]. In our small series, there was one case who presented with chronic pulmonary hypertension and cor pulmonale. If Western statistics can be extrapolated to our data, then this single case would signify the existence of a much larger number of patients of acute PE, the majority of whom remain undiagnosed.

The initial investigations ordered on suspicion of PE are an ECG and X-ray chest. However experience from large centres suggests that a normal X-ray chest is the most common finding in embolic disease [7], and aside from sinus tachycardia, the ECG is normal in most cases [7,8]. The main utility of ECG and X-ray is to exclude other conditions with a similar presentation like myocardial infarction, valvular heart disease, pneumonia and pneumothorax. ECG changes suggestive of pulmonary hypertension, right axis deviation and right ventricular strain are infrequent and occur mainly in severe PE. In our series 7 cases showed some abnormality in ECG apart from tachycardia, while X-ray chest showed some suggestive or non-specific changes in 7 cases. This implies that our cases suffered from moderate to severe PE.

Echocardiography reveals an enlarged right ventricle, abnormal movement of interventricular septum and enlarged pulmonary artery [3]. Moreover ECHO can also help in ruling out other conditions mistaken for acute PE. In two of our cases ECHO strengthened the suspicion of PE and provided sufficient grounds to initiate anticoagulants prior to definite confirmation which requires pulmonary arteriography or ventilation/perfusion scans [7,9].

Our experience is primarily confined to

"medical" cases but it is well known that patients suffering from surgical and gynaecological disorders are also at high risk of PE [1,8,10]. It is hoped that this study will increase the awareness of PE among specialists of other clinical disciplines.

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