Case Report

Large Pulmonary Embolism - Wind Down The Ambugity

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Abstract

Pulmonary embolism (PE) accounts for hundreds of hospitalizations annually in our country. The death rate among them is high approximately 15 %. Although D Dimer testing for exclusion of PE and chest computed tomography (CT) for imaging PE have revolutionized the diagnostic approach, in our country where CT scan is either not available in most of the hospital or financially it is beyond reach for many patients. In this study we have diagnosed and managed a moderate sized pulmonary embolism with the help of Electrocardiography, Chest X Ray, D Dimer assay and Echocardiography. In this study, a patient who presented with sign and symptoms which were highly suggestive of Pulmonary embolism and we diagnosed a case of PE with the help of Chest Xray, Electrocardiography, D Dimer assay and Echocardiography. The reason to highlight this study is because in our country, in absence of High frequency multi detector 64 slice CT scan in most of the hospital and financial constrain of we can still diagnose and treat effectively most of the pulmonary embolism.

Keywords: Acute Pulmonary embolism, Large PE, Pulmonary embolism, PE

INTRODUCTION

Pulmonary embolism accounts for millions of hospitalizations annually worldwide. Although D-Dimer testing for exclusion of PE and chest computed tomography (CT), for imaging PE have revolutionized the diagnostic approach, PE remain difficult to detect unless high index of clinical suspicion in kept in management of critically ill patients.

Our understanding of the precipitants of PE has improved especially the role of hyper coaguable states and potentially modifiable risk factors such as long–haul air travel and obesity.

Doctors in critical care and cardiologist must provide expertise in the treatment of hemodynamically compromised patients with PE as well as those with right ventricular failure who maintain a stable blood pressure and heart rate. This requires rapid and accurate risk stratification, often with echocardiography, elevation of troponin, brain natriuretic peptide (BNP) levels, so that those patient with adverse prognosis will be identified and treated with thrombolysis or embolectomy.[1,2]

CASE REPORT

An elderly male aged 68 years who came to Narmada trauma centre on 10 March 2013 at 7.40 pm with history of fall from motorcycle, presented with sudden onset breathlessness, restlessness and perspiration. He sustained mild abrasion on face, scalp and bruise on the chest without any major obvious external injury and internal organ injury. There was no evident fracture of hip, femur or spine. He had blunt injury of chest.

On examination patient was conscious, restless, tachyponeic, with respiratory rate of 38/minute, diaphoresis, mild cyanosis with Spo2 of 70 %. His pulse rate was 130 per minute, blood pressure was 90/60 mm hg and was afebrile. He had a drop of blood pressure and saturation (70 systoloic with oxygen saturation of 68%).

His investigations were as follows:

Hemogram

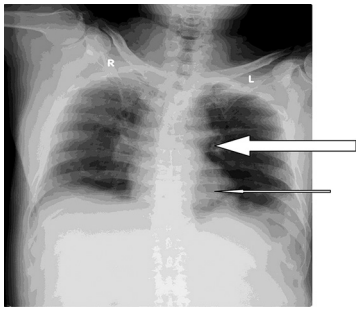
Hemoglobin	11.8 Gm%
Total erythrocyte count	4.08
Erythrocyte sedimentation rate	17 mmfhr
Total leucocyte count	12,300/cumm
Diferential leucocyte count	
Neutrophiles	81%
Lymphocytes	18%
Monocytes	01%
Eosinophiles	%
Basophiles	%
Platelet count	1.95 Lac/cumm

D Dimer Assay (Elisa) was Positive by Elisa Method

Biochemistry

RBG	224 mg/dl
Urea	64 mg/dl
Creatinine	1.5 mg/dl
Calcium	9.0 mg/dl
Total Billirubin	0.91
Direct	0.38
Indirect	0.53
AST	50
ALT	40
Total protein	6.8
Albumin	3.6
Globulin	3.2
CK-MB	05
Troponin negative	
Amylase	53
Lipase	80

Chest X-Ray



Chest X ray of patient showing wedge shape opacity (thin arrow) at left base and prominent left pulmonary vascular markings (thick and big arrow) respectively

Electrocardiography

The patient ECG was done in which he had sinus tachycardia with heart rate of 156 per minute, axis was +80 and he had S1Q3T3.

Echocardiography

There was evidence of enlarged right ventricle with size of 3.10 cm, and reduced right ventricular free wall movement. Left ventricle function was normal and there was no regional wall motion abnormality.Pericardium was normal. No evidence of RA/RV/LA/LAA/LV clot, thrombus or vegetation. Valve were normal.There was grade-2 tricuspid regurgitation with peak TR gradient of 45 mm Hg. There was mild pulmonary artery hypertension.

CT Scan

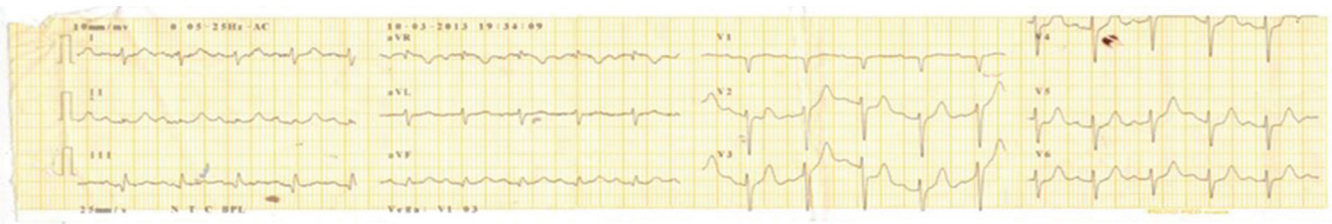
CT scan brain was normal. No abnormalities we detected.

Management

Patient was started on Low molecular weight heparin (Enoxaparin)with a dose of 1 mg/kg body weight twice a day, IV fluids, antibiotic. His oxygen saturation by next 6-8 hours had dropped to 68% and developed hypotension with blood pressure of 80/60 mm Hg. Naso- Trachea intubation was done and patient was put on assisted ventilation CMV-ACMV mode with tidal volume of 6 litres/kg, PEEP of 7-10, respiratory rate of 12/minute, Fi02 of 70 which was subsequently reduced to 30 over next 10-12 hour.He was also started on Dopamine at rate of 15 microdrop/kg per minute and noradrenaline at the rate of 0.1 mg/kg/minute. Low molecular weight heparin was continued for 10 days, during this period patient was on assisted ventilation and inotropic support which were gradually tapered and stopped by tenth day,and ventilator was gradually weaned to pressure support by eight day and weaned off completely by 9th day. During the second post admission day, patient was started on oral anticoaugulation (Warfarin-5 mg once a day) and continued to keep INR between 2-2.5. Patient made complete recovery by 15th day and was discharged on 28 march 2013.

DISCUSSION & CONCLUSION

There are various precipitating factors for venous thrombosis which in turn can cause PE.



A-inherited factors

Hypercoagulable states

1. Mutation in factor v gene (factor v laden)
2. Resistance to activated protein C
3. Prothrombin gene mutation
4. Mutation in protein C gene
5. Protein S deficiency
6. Antithrombin 3 deficiency
7. Hyperhomocysteinemia
8. Antipphospholipid antibody

B-acquired conditions

Acquired conditions may precipitate venous thrombosis

1. Long-haul air travel
2. Surgery/immobilization/trauma
3. Hospitalization with medical illness such as pneumonia or congestive heart failure, stay in medical or surgical intensive care unit.
4. Obesity
5. Increasing age
6. Cigarette smoking
7. Systemic arterial hypertension
8. Diabetes mellitus
9. Use of oral contraceptives/pregnancy/postpartum state
10. Cancer and cancer chemotherapy
11. Stroke/spinal cord injury
12. Indwelling central venous catheter , pacemakers and internal cardiac defibrillators

In critical care and trauma units there is paucity of time for an complete investigational approach in rapidly deteriorating patients where clinical explanation of hemodynamic compromise is not acceptable. A high index of suspicion of pulmonary embolism can resolve the issue in certain patients and lead to a definitive diagnosis of pulmonary embolism. Therapy of pulmonary embolism is tailored according to the patient's clinical presentation, the anatomical extent of the embolous, presence of underlying cardiopulmonary disease, cardiac biomarkers such as troponin, D-Dimer and detection of right side heart dysfunction by physical examination,electrocardiogram and echocardiogram. High risk patients warrant thrombolysis or embolectomy as primary therapy to dissolve or remove the embolous,in addition to anticoagulation to prevent recurrent venous thromboembolism. In low risk patients, anticoagulation should suffice. The patient in our study had a large embolous; along with positive D-Dimer, ECG changes as sinus tachycardia and S1Q3T3. His chest X ray had prominent pulmonary vascular markings and echocardiography was highly suggestive of pulmonary embolism; with features like enlarged right ventricle, hypokinetic free right ventricular wall and grade-2 tricuspid regurgitation. He was managed by anticoagulation, inotropes and assisted ventilation. Patient made complete recovery and was discharged home with graduated compression stocking for six month.

The important aspect of this study is that we know there are less than 2% of hospital in our country who has in-house multidetector 64 slice latest CT scan (3) which is the gold standard for diagnosis of PE. Therefore it is important for critical care specialist and cardiologist to detect and recognize massive PE so that they can timely investigate aggressively with multidetecter CT and manage by thrombolysis or embolectomy/thrombectomy (1). On the other hand we can diagnose mild to moderate grades of pulmonary embolism with the help of D Dimer assay, Echocardiography, Chest X Ray, Electrocardiography, lower limb venous Doppler (5) and in turn manage other grades of PE with Anticoagulation and supportive treatment, without CT scan and thrombolysis effectively as we did in this study and can save a large number of patients who form the majority of ambiguous PE.[2-6]

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