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Case Report

An Interesting Case Of Bronchial Asthma

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Abstract

Acute pulmonary embolism is a component of venous thromboembolism (VTE), which may prove fatal if not suspected and subsequently treated. Here we present a 82 year female patient with Bronchial asthma who deteriorated and developed pulmonary thromboembolism during the hospital stay.

Acute pulmonary embolism in elderly asthmatics is not very uncommon. Asthmatic presenting with acute onset of dyspnea, pulmonary embolism should be kept as a possibility. most of the time PE is not considered in differential diagnosis while evaluating such patients in emergency. Pulmonary embolism requires high clinical suspicion, based on validated scores and requires further multi-modality investigation to confirm or rule out disease.

Key words: Acute Pulmonary Embolism, Bronchial Asthma, High Clinical Suspicion

Introduction

Acute pulmonary embolism is a component of venous thromboembolism (VTE), which may prove fatal if not suspected and subsequently treated. Incidence of pulmonary embolism is around 0.5–1 case per 1000.[1] This has been estimated that 70% of proven post mortem cases of pulmonary embolism are not even suspected during the course of treatment.[2] On the contrary, only 25–30% of suspected cases turned out to be cases of pulmonary embolism in post mortem studies.[3]

Here we present a case of Bronchial asthma patient, who deteriorated and developed pulmonary thromboembolism during the hospital stay.

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Case Report

A 82 year old female was brought to Emergency medicine department with history of fever since 4 days. Breathlessness since 1 day.

-Fever of 4 days duration, moderate grade, on and off, not associated with chills and rigors.

-Breathlessness of 1 day duration, sudden onset, grade 3 Modified Medical Research council , not associated with orthopnea or PND. Wheeze present.

NO h/o chest pain, palpitations, pain abdomen, No h/o cough/ expectoration/ haemoptysis/sore throat/ ear ache/ headache. Bowel and bladder habits are normal

No h/o bleeding manifestations. No h/o weight loss.

Past History

Patient is k/c/o HTN since past 7 years on regular medication.(Tab. Telma 40mg 1-0-0)

k/c/o Bronchial asthma since past 20 years on regular medication.(inhaler therapy)

Patient has h/o similar complaints in the past and has repeated history of hospital admissions in the past.

Not k/c/o T2DM, TB, Epilepsy, Hypothyroid

She is a post menopausal women, with no history of postmenopausal bleeding.

No ill habits.

Patient is moderately built and nourished.
No pallor/icterus/cyanosis/clubbing/ lymphadenopathy/ pedal edema.
BP: 124/80mm Hg. PR: 96/min, SPO2: 80% at room air, 98% with4litres oxygen, Respiratory Rate: 30/minute
CVS: JVP normal, S1S2 +, NO murmurs
R/S: Trachea central, wheeze present, B/L air entry was present, B/L rhonchi present.
B/L fine crackles heard in Infra scapular area.
P/A: soft, non tender. NS: conscious, oriented. No focal neurological deficits.
Patient was diagnosed as Acute infective exacerbation of Bronchial asthma , HTN.
Patient was started on oxygen therapy, bronchodilator therapy, antibiotics and supportive management. Patient condition improved symptomatically in EMD, and was admitted for the same.

Admission day reports

Hb: 9.0gm/dl , RBC: 4.4 mil/mm3, WBC: 11.8T/mm3, Platelets: 263thousands/mm3
BU: 40mg/dl, Serum.Creatinine: 1.1mg/dl , Na+: 132meq/dl, K+:4.7meq/dl
ECG: Sinus Rhythm, no significant ST-T changes.
On day 2 of admission:
At around 2:00am patient developed sudden onset breathlessness with orthopnoea.
Patient BP: 70/50mm Hg, PR: 130/min, Spo2: 90% with6litres Oxygen, Respiratory Rate: 36/min

ECG: S1Q3T3 changes+
Cardiac markers: **D-Dimer: 4010ng/ml, Brain natriuretic Peptide:662pg/ml,**
2D-echo: Dilated RA & RV, no Regional Wall Motion Abnormality, mild MR/AR, severe TR with severe PAH (70MMHg), no clot, preserved LV systolic function, LV EF- 50%.

In view of high clinical susceptibility with patient being hemodynamically unstable, patient was shifted to ICU and started on Low molecular weight Heparin in therapeutic dose.

CTPA was done which was suggestive of acute pulmonary thromboembolism with right ventricular strain and borderline PAH, Multiple filling defects in right atrium and right atrial appendages.

Diagnosis

Acute massive thrombo-embolism with severe TR with severe PAH with RV dysfunction, Acute exacerbation of bronchial asthma, Hypertension.

Patient was advised for thrombolysis and started on Injection Streptokinase @ 2.5lakh units over 30mins and followed by 1lakh units infusion over 12hours. Patient has improved gradually over a period of 1 week with normal pulse and respiratory rate and maintaining oxygen saturation on room air. ECG improved with normal sinus rhythm.

Figure 1:Chest Xray

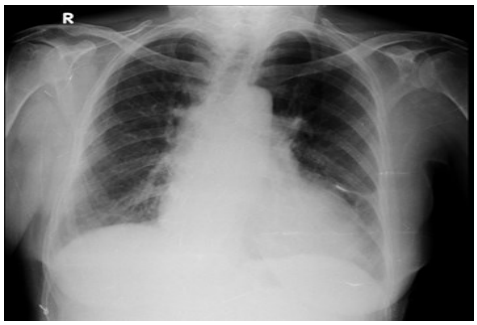
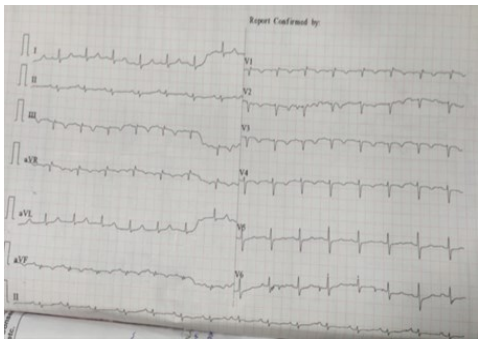


Figure 2:ECG showing S1Q3T3 pattern



Figures 3: CTPA

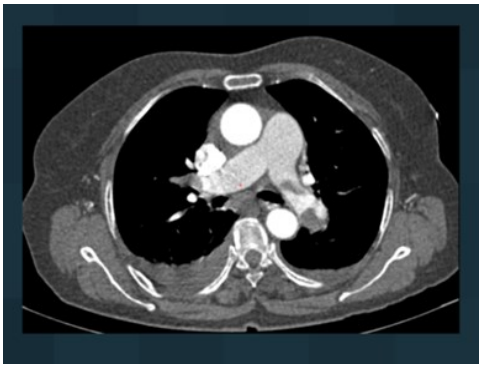
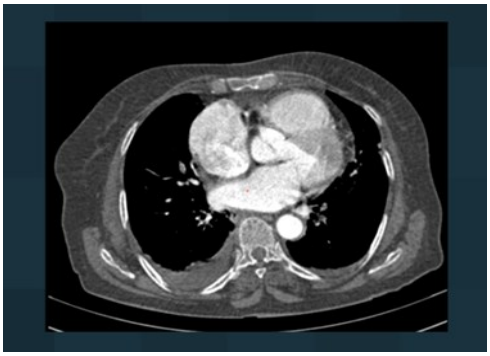


Figure : 4



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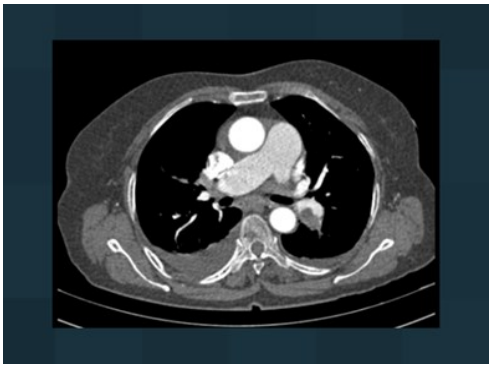

Discussion

VTE encompasses pulmonary embolism, deep vein thrombosis and superficial thrombophlebitis. Thrombosis is caused by interaction of three Virchow factors, that is, hypercoagulability, trauma and stasis. There are acquired and genetic risk factors responsible for thromboembolism. Recent surgery, trauma, immobilization, pregnancy and oral contraceptives are commonest acquired factors which are most of the time, temporary in nature.[4] Other factors such as malignancies, for example hematological, lung, and pancreatic and brain cancer pose greatest risk for PTE, and cancer association is a predictor of increased mortality. Smoking, obesity, atherosclerosis, hypertension and infection in hospitalized patients are also common causes for PTE. Deep vein thrombosis (DVT) is present in 79% of cases of PE and 40–50% of DVT is complicated by pulmonary embolism. Inherited risk factors include antithrombin III deficiency, protein C, protein S deficiency, factor V Leiden mutation, etc.

Modified Wells score and revised Geneva classification are validated clinical tool to guide for further evaluation of suspected patient of PE [5] and our patient had intermediate risk in both the scores.

The clinical signs and symptoms of acute PE are non-specific. In most cases, PE is suspected in a patient with dyspnea, chest pain, presyncope or syncope, or hemoptysis.

Hemodynamic instability is a rare but important form of clinical presentation, as it indicates central or extensive PE with severely reduced hemodynamic reserve. Dyspnea may be acute and severe in central PE; in small peripheral PE, it is often mild and may be transient. In patients with pre-existing heart failure or pulmonary disease, worsening dyspnea may be the only symptom indicative of PE. In central PE, chest pain may have a typical angina character, possibly reflecting RV ischemia, and requiring differential diagnosis from an acute coronary syndrome or aortic dissection. Electrocardiographic changes indicative of RV strain-such as inversion of T waves in leads V1V4, a QR pattern in V1, a S1Q3T3 pattern, and incomplete or complete right bundle branch block-are usually found in more severe cases of PE.[5] Our patient has S1Q3T3 changes. D dimer assay based on enzyme-linked immunosorbent assay (ELISA) can rule out pulmonary embolism in low clinical probability with sensitivity of 95%. [6]

D-dimer was found to be very high in our patient during the attack. 2D-Echo revealed dilated RA,RV with strain pattern which is important indicator for PTE. CT angiography of thorax has diagnosed PE in our case. Prospective investigation of pulmonary embolism diagnosis (PIOPED) II has observed sensitivity of 83% and specificity of 96% in (four detector) multi-detector computed tomography (MDCT) [7]. In view of patient being hemodynamically unstable and CT showing massive pulmonary thrombus patient was started on thrombolytic therapy. Exact cause of pulmonary embolism could not be ascertained our case, though patient being chronic asthmatic might have acted as risk factor. PESI severity scoring of our patient was >1 with 30 day mortality risk standing at 10.9%. Pulmonary embolism may remain undiagnosed in 29% of cases. Patient has improved gradually over time.

Conclusions

Acute pulmonary embolism in elderly asthmatics is not very uncommon. Asthmatic presenting with acute onset of dyspnea, pulmonary embolism should be kept as a possibility, most of the time PE is not considered in differential diagnosis while evaluating such patients in emergency. Pulmonary embolism remains a disease which requires high clinical suspicion, based on validated scores and requires further multi-modality investigation to confirm or rule out disease. Unlike infectious diseases, cardiac disorder or blood disorder, no specific symptoms, signs or in-

vestigations reflect a disease process immediately. Hence elderly patient presenting with acute onset of dyspnea should be evaluated for common cause of dyspnea but pulmonary embolism should be kept in mind, so as not to miss this fatal disease.

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