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ACUTE PULMONARY EMBOLISM IN YOUNG

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ABSTRACT Pulmonary embolism remains a disease which needs high clinical suspicion to prevent mortality and morbidity. More so in young healthy individuals, suspicion is very low as compared to old age individuals with multiple co-morbid conditions. Pulmonary embolism carries high mortality if not suspected and treatment initiated as early as possible. There are two case reports of young male individuals who presented as acute onset of breathlessness and later diagnosed and treated as a case of pulmonary thromboembolism.

KEY WORDS : Acute Pulmonary, streaky haemoptysis, pulmonary thromboembolism, hypercoaglability

CASE REPORT

A 24-year-old young male patient, smoker, no known comorbidity, presented to emergency department with history of cough with streaky haemoptysis for 7 days, fever and breathlessness on exertion for 2 days. Initial examination: Respiratory rate – 28/min, pulse – 120/min, blood pressure – 98/68mm of Hg, SpO2–88% on room air, temp. – 99.4°F, BMI – 28 kg/m2 and scattered wheeze on chest examination. No other significant finding was noted, and he was managed as a case of bronchial asthma and started on intravenous antibiotics, nebulisation and steroid. The patient was shifted to intensive care unit (ICU) for further management.

Investigations: Haemoglobin (Hb) – 13.2 g%, total leukocyte count (TLC) – 12400/cumm, differential leukocyte count (DLC) – P: 75%, L: 23%, E: 2%, biochemistry was normal. Chest X-ray (CXR) PA view – normal [Figure 1], ECG (electrocardiogram) – sinus tachycardia with right axis deviation and right ventricular (RV)strain pattern [Figure 2].Cardiac markers were normal. The patient was initially continued on same management and CXR, ECG was repeated next morning, revealing similar findings. In the view of persistent breathlessness, unremarkable chest finding on next day and persistent CXR, ECG findings, pulmonary embolism were suspected. He was started on low molecular weight heparin (LMWH) in therapeutic dosage and further evaluated. D-dimer came within normal limits, but in the view of strong suspicion of pulmonary embolism, computed tomography (CT) angiography of thorax was done, which revealed pulmonary embolism in bilateral main pulmonary artery (MPA) involving right MPA more than left and few segmental arteries involvement [Figure 3]. The patient had a history of crushing injury to left upper limb few years back. Ultrasound of bilateral lower and upper limb was normal. 2-D ECHO revealed moderate pulmonary artery hypertension (PAH), dilated RV with McConnel's sign [decreased movement of RV free wall as compared to apex]. The patient was continued on LMWH and supportive care. 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. Patient was evaluated at higher centre for genetic and acquired factors but no cause could be ascertained. Patient was put on oral coagulant and lost follow-up after 2 months on treatment.

DISCUSSION

VTE encompasses pulmonary embolism, deep vein thrombosis and superficial thrombophlebitis. Thrombosis is caused by interaction of three Virchow factors, that is, hypercoaglability, trauma and stasis. There are acquired and genetic risk factors responsible for thromboembolism. Recent surgery, trauma, immobilisation, 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 haematological, 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 hospitalised 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 LeidenV mutation, etc. One patient had history of smoking and both of them were obese. Smoking and obesity are independent acquired risk factors for PE. Cough, haemoptysis, breathlessness and chest pain were common symptoms, which are common but not specific for PE as well as sinus tachycardia and tachyapnea and low blood pressure were found in both patients. Classical triad of chest pain, haemoptysis and dyspnoea is present less than 20% of cases. Modified Wells score is a validated clinical tool to guide for further evaluation of suspected patient of PE[5] and both patients had more than four score on initial presentation. Initial chest examination revealed no significant abnormality. CXR is useful to rule out other causes of dyspnoea like Pneumothorax, etc. Hampton's hump (subpleural wedge shape opacity), Westermarck's sign (areas of hyperlucency), raised hemi diaphragm, pleural effusion and normal chest radiograph are other findings on CXR.[6] ECG findings in above cases are common in PE but again not specific. S1Q3T3 is seen in around 50% of cases, which denotes RV damage, whereas sinus tachycardia is present in around 40% of cases. Most common finding in pulmonary embolism is T wave inversion.[7] D dimer was positive in one patient only but in high probability cases D dimer may not be helpful. D dimer assay based on enzyme linked immunosorbent assay (ELISA) can rule out pulmonary embolism in low clinical probability with sensitivity of 95%.[8]

CT angiography of thorax has diagnosed PE in both cases. 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).[9] 2-D ECHO revealed PAH and McConnel's sign in above presented cases. McConnel's sign has high positive predictive value for pulmonary embolism even in presence of other co-morbid conditions.[10] RV dilatation, presence of thrombi in RV, is other important finding in 2-D ECHO. Compression ultrasound (CUS) of

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lower limb shows DVT in 30–50%of cases, and presence of thrombus in lower limb in suspected case is good enough to start anticoagulation. Both patients were treated with LMWH and supportive care.

Before heparin, surgery was the only option with 100% mortality. LMWH anticoagulants such as Enoxaparin, Dalteparin, Tinzaparin and Fondaparinux are preferred over Unfractioned heparin (UFH), as chances of bleeding and Heparin-Induced Thrombocytopenia (HIT) are very low, and monitoring is not required except in special circumstances such as pregnancy, in which anti Factor Xa activity is monitored.[11] UFH is preferred in patients with renal impairment, obese (>150 kg), low weight (<30 kg) as monitoring with activated partial thromboplastin time (aPTT) is easy and reversal with protamine is available. Though 2-DECHOrevealedRVchanges but cardiacmarkers were normal and no fibrinolytic therapy was administered in above cases. It has been seen that thrombolytic therapy causes early resolution of thrombus and early recovery of RV and PAH, but difference is no longer identified after 1 week of treatment as compared to heparin.[12] Exact cause ofpulmonary embolism could not be ascertained in first case, and second case had raised level of homocysteine and APLA antibodies. Pulmonary embolismmay remain undiagnosed in 29% of cases. Both the patients improved gradually and had no evidence of chronic sequel.

CONCLUSION

Acute pulmonary embolism in young adults is not very uncommon. A young adult presenting with acute onset of dyspnoea, pulmonary embolism should be kept a possibility, as most of the time PE is not considered in differential diagnosis while evaluating such patients in emergency. Most of young patients reported in literature are young females, either pregnant or on oral contraceptives. 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 investigations reflect a disease process immediately. Hence young male presenting with acute onset of dyspnoea should be evaluated for common cause of dyspnoea but pulmonary embolism should be kept in mind, so as not to miss this fatal disease.

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