Volume-4, Issue-7, July-2015 • ISSN No 2277 - 8160

Research Paper | Medical Science

Unprovoked massive pulmonary embolism in a young male

Dr Vishwa B Unadkat | MD Internal Medicine, Assistant Professor, PDU Medical college and Hospital, Rajkot

ABSTRACT

Massive pulmonary embolism (PE), specially unprovoked can be easily under looked as differential diagnosis of patients present with acute onset of breathlessness. If promptly diagnosed, such patients respond well to the conventional medical therapy. This paper describes a case of massive pulmonary embolism in a young male, who couldn't be found to be having risk factors for venous thromboembolism (VTE), so called idiopathic or unprovoked embolism. Missing to rule out such a massive unprovoked PE can be rapidly fatal.

KEYWORDS : Pulmonary embolism (PE), Deep vein thrombosis (DVT), venous thromboembolism (VTE), Dyspnea, Breathlessness, Unprovoked PE, Thrombolysis, Anticoagulation, International flight.

Introduction:

PE is traditionally been considered as "the Great Masquerader", making diagnosis difficult [1]. Venous thromboembolism (VTE), which encompasses deep venous thrombosis (DVT) and pulmonary embolism (PE), is one of the three major cardiovascular causes of death, along with myocardial infarction and stroke. Literature mention many provocative conditions for development of VTE, including some genetic mutations in naturally occurring anticoagulants as well as some clinical conditions (vide infra). VTE that occurs during long-haul air travel is considered unprovoked. Diagnosing massive unprovoked PE requires great amount of suspiciousness on the part of clinician and treatment includes medical management in the form of anticoagulation plus thrombolysis and sometimes embolectomy (catheter/surgical) as primary therapy plus secondary prevention.

Case Report:

A 25 year male (BMI 24), a businessman, presented to our ward with sudden onset of breathlessness, chest pain, ghabharaman and history of syncope. Her mother noticed his bluish face just prior to fall down which recovered within seconds (suggestive of transient cyanosis). He was completely healthy prior to this and was not having addiction. He landed at his hometown after a long (7 hours) international flight 5 days prior to development of his symptoms.

On examination, he was very anxious and looking pale. Striking signs, BP 80/60 mmHg, raised JVP and pulse rate 120/min, regular and respiratory rate 26/min. Rhonchi were present and rest other systems including CVS was unremarkable. His EKG was showing classical S1Q3T3 [figure 1] pattern with tachycardia and partial RBBB.

Investigations: His routine blood analysis and chest X-ray was normal.

His MDCT PULMONARY ANGIOGRAPHY revealed evidence of filling defect in bifurcation of main pulmonary artery with extension in to right and left pulmonary arteries and their segmental branches, suggestive of pulmonary thromboembolism with normal lung parenchyma and pleura. Main pulmonary artery (PA) 25mm, right PA 19mm, left PA 18mm [figure 2].

2 D Echo and color Doppler study of heart revealed moderate PAH with mild TR, which was done on third day of admission after thrombolysis.

His blood was tested negative for hypercoaguable (thrombophilic) states namely factor V Leiden mutation, protein C, protein S and anti-thrombin III deficiency, anti phospholipids antibody and connective tissue disease profile. Cardiac enzymes were normal.

Discussion:

Differential diagnosis of acute dyspnea, chest pain and ghabharaman in an otherwise healthy young male includes CVS causes like acute coronary syndrome, congestive heart failure, tachyarrhythmia, pericarditis and Respiratory causes like pneumonia, asthma, COPD, pleurisy. Other causes include costochondritis, musculoskeletal discomfort, anxiety etc [2]. Here in our case history of hypotention and transient cyanosis clinches attention to rule out PE as dyspnea, syncope, hypotension or cyanosis indicates massive PE, whereas pleuritic pain, cough, hemoptysis often suggests a small embolism situated distally near the pleura [1].

Table [2]:-High clinical likelihood of PE if score exceeds 4;

Clinical Variable	Score
Signs and symptoms of DVT	3.0
Alternative diagnosis less likely than PE	3.0
Heart rate > 100	1.5
Immobilization >3 days; surgery within 4 weeks	1.5
Prior PE or DVT	1.5
Hemoptysis	1.0
Cancer	1.0

In our case score exceeds 4, as we could rule out other causes with examination and investigation (3) and his heart rate was >100(1.5) and hence score is 4.5.

We can directly go on to order CT pulmonary angio rather than to wait for D-dimer level which takes additional time to come, once we got the high likelihood of PE. Advantages of CT scan includes <= 1mm resolution during a short breath hold, images small peripheral emboli, sixth order branches can be visualized with resolution superior to that of conventional invasive contrast pulmonary angio, size of RV, imaging of lung parenchyma and pleura in case of absent PE. Because of this it has replaced conventional invasive pulmonary angio as investigation of choice. Central pulmonary artery is embolised in all patients of massive PE .[3]

Thorough work up to rule out DVT done in the form of extending CT scanning to rule out pelvis and upper thigh DVT and venous ultrasonography and color doppler study of all the limbs, which was found negative.

S1Q3T3 pattern on EKG is relatively specific but poorly sensitive as its present only in 50% of cases [4]. Other changes seen frequently are T wave inversions in leads V1 to V4, sinus tachycardia, acute onset of RBBB.

Other corroborative tests include serum troponins and cardiac fatty acid binding protein, which increases in case of right ventricular micro infarction. It wasn't the case here.

Non invasive imaging study includes chest X-ray, echocardiography, lung scanning and MRI contrast. The best known sign of McConnell

on echo denotes RV free wall hypokinesia with normal motion of RV apex, is very important from the treatment point of view. Though it wasn't there in our case probably we were late for that.

It was clear in our case as hypotension (massive PE) on presentation is an indication of thrombolysis; the drug of choice is 100mg recombinant tissue plasminogen activator, IV slowly over two hours [2.] Because of its unavailability this patient was thrombolysed with streptokinase (SK), 2.5 lac unit IV slowly over 1 hour followed by 1 lac unit per hour for whole day, total 25.5 lac SK is required, under thorough monitoring for bleeding tendencies [5.] Other alternative includes catheter directed thrombolysis, embolectomy surgical or catheter based.

Patients with normotension and normal RV can be managed with anticoagulation alone but individualization of therapy needed when patient has normotenion plus RV hypokinesia.

All the patients, with unprovoked PE have to be put on anticoagulation; we have given heparin (target aPTT 2- times normal, monitor every 6 hours) for 5 days with institution of oral anticoagulation, Warfarin from day 3 (target INR between 2 and 3 for at least 6 months, followed by lifelong Warfarin (target INR 1.5 to 2) as they are prone to recurrence. Enoxaparin and fondaperinux are the better alternative of heparin available. Anticoagulation for 6 months would suffice for a provoked PE in conditions like cancer, systemic arterial hypertension, COPD, obesity, cigarette smoking, oral contraception, postmenopausal hormone replacement, surgery, trauma etc.

REFERENCES

1] Harrison's principle of Internal Medicine, 18th edition, chap. 262. | 2] Braunwald's Heart Disease, 8th edition, chap. 72. | 3] Findik S, Erkan L, Light RW, Uzun O, Atia AG, Akan H. Respiration, 2008:76(4):403- 12 doi 10.11591. 000147765. Epub 2008, Jul 22. | 4] Chan T, Vilke GM et al, EKG manifestation: Pulmonary Embolism, J Emerg Med. 200; 21(3):263-70. | 5] Essentials of Medical Pharmacology by K. D. Tripathi; chap. 44.