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A Case Report Of A Young Male With Unprovoked Acute Pulmonary Thromboembolism In Absence Of Deep Vein Thrombosis

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ABSTRACT:

Pulmonary thromboembolism is a pulmonary emergency. Isolated Hyperhomocysteinemia causing unprovoked pulmonary thromboembolism is a rare entity. Herein we present a 30-year-old man with multiple thromboembolic events without evidence of deep vein thrombosis. Extensive hypercoagulability workup was done which showed an elevated homocysteine level, with normal vitamin b12 and folate levels.

INTRODUCTION:

Pulmonary thromboembolism is a life-threatening condition which requires immediate intervention. It occurs when arteries to the lungs are blocked by blood clots formed elsewhere in the body usually from deep veins of the legs or pelvis (1). Its causation is attributed to multiple factors. One rare factor among them is hyperhomocysteinemia.

Any alterations in metabolic pathway of homocysteine formation such as genetic abnormalities, vitamin cofactor deficiencies, environmental factors can increase the serum levels of homocysteine in the body (6). The normal levels of homocysteine ranges from 5-15 micromol / L (7). Individuals with increase in serum homocysteine levels are at a higher risk for arterial and venous thromboembolic events (7).

Pulmonary thromboembolism with hyperhomocysteinemia in absence of DVT is a rare condition. Herein we present one such interesting case.

CASE REPORT:

A 30-year-old never smoker, non-alcoholic male presented to our department with complaints of right sided pleuritic chest pain with no inciting factors, progressively worsening dyspnea

and h/o intermitted low grade fever for 5 days. Patient denied any episodes of hemoptysis. Denied any relevant thromboembolic events in the family. Five years prior to current presentation patient gave history of an episode of transient ischemic attack for which he was treated with anticoagulant (warfarin) and discharged with no residual motor or sensory weakness. Patient failed to be on regular follow up and discontinued anticoagulants.

On physical examination – Patient had a Heart rate of 115 bpm, blood pressure of 110/70mmhg, respiratory rate of 24 breaths per minute, and maintained a saturation of 90% at room air on pulse oximetry. Respiratory exam showed absent breath sounds on the right mammary, axillary infra – axillary, lower interscapular and infrascapular areas. and other system examinations were unremarkable.

Initial laboratory examinations including complete blood count, renal and liver function parameters were with in normal limits. ECG – Sinus tachycardia, Chest xray - right moderate pleural effusion, wedge shaped opacity in right MZ and LZ. A Well's score of 4 was calculated suggesting moderate risk of Pulmonary thromboembolism after which a D-dimer of 712 was obtained. Coagulation profile was normal. Echocardiography was normal. Bilateral lower limb venous doppler done showed normal waveforms. Patient was taken up for Pulmonary angiogram which showed emboli in right basal segmental pulmonary branches with pulmonary infarcts.

Extensive Hypercoagulation work up was done, which showed beta 2 glycoprotein IgM, IgG, Antithormbin, Protein C and protein S, cardiolipin antibody, lupus anticoagulant levels to be within normal limits except elevated levels of homocysteine >50.00. Vitamin b12 and folate levels were under normal limits. Patient was treated with systemic unfractionated heparin infusion, then switched to newer oral anticoagulants, discharged in stable condition with vitamin b12, folic acid supplements. Patient is on regular follow up.

DISCUSSION:

Homocysteine is a sulphur containing amino acid which is obtained as a demethylated by product of methionine during metabolism of methionine to cysteine (3,4). Elevated homocysteine levels contribute to endothelial dysfunction and promote the formation of blood clot and thus predisposing to development of thromboembolic events. It is necessary to monitor a patient with hyperhomocysteinemia for such events.

The classification of hyperhomocysteinemia is as follows: (1) moderate risk, 15 to 30 µmol/L; (2) intermediate risk, 30 to 100 µmol/L; (3) severe risk, >100 µmol/L (7). Although increase homocysteine levels in the body is a risk factor for thromboembolic events, when combined with other risk factors like vitamin B12 or vitamin B6 deficiency, chronic renal insufficiency, lifestyle factors (smoking, chronic alcohol, high coffee intake), end-stage diabetes, hypothyroidism, systemic lupus erythematosus, hyperproliferative disorder and medications (methotrexate, sulphonamides, or antacid) the risk increases multiple folds.

Clinical features of pulmonary thromboembolism may include consolidations and pleural effusion which can be often mistaken for pneumonia (5). Our patient presented with pleural effusion and coagulation profile was within normal limits but had high levels of homocysteine which predisposed him to multiple thromboembolic events.

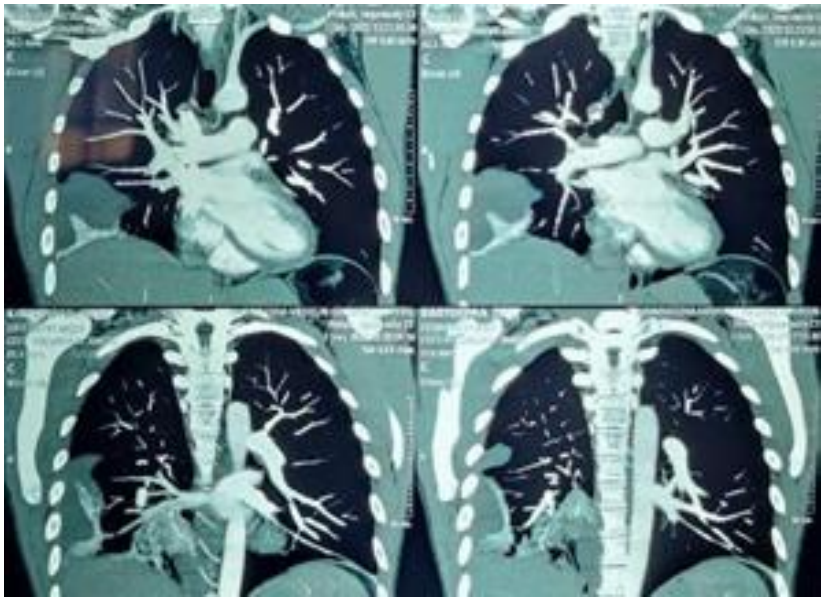
A case-control study by Falcon et al. found that hyperhomocysteinemia was a risk factor for thrombosis in people younger than 40 years [9]. In order to fully link hyperhomocysteinemia to unprovoked thromboembolic events further studies with larger samples are required.

CONCLUSION:

Hyperhomocysteinemia is a rare autosomal disease and serum levels with hypercoagulability profile must be checked in any young patient presenting with thrombotic events despite with adequate coagulation to prevent further recurrence.

In high suspicious cases with hyperhomocysteinemia with normal 2DECHO and in absence of Deep vein thrombosis, it is advised to proceed with workup of Pulmonary thromboembolism as early recognition can not only reverse but can also prevent pulmonary thromboembolism.

PULMONARY ANGIOGRAM WHICH SHOWED EMBOLI IN RIGHT BASAL SEGMENTAL PULMONARY BRANCHES WITH PULMONARY INFARCTS:



LUNG WINDOW SHOWING RIGHT LOWER LOBE CONSOLIDATION.

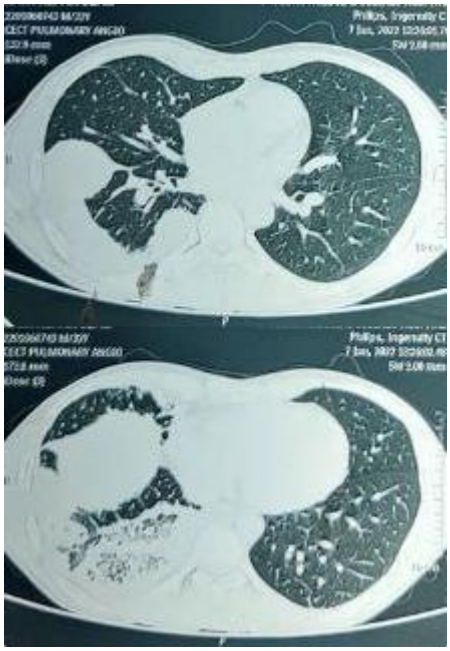
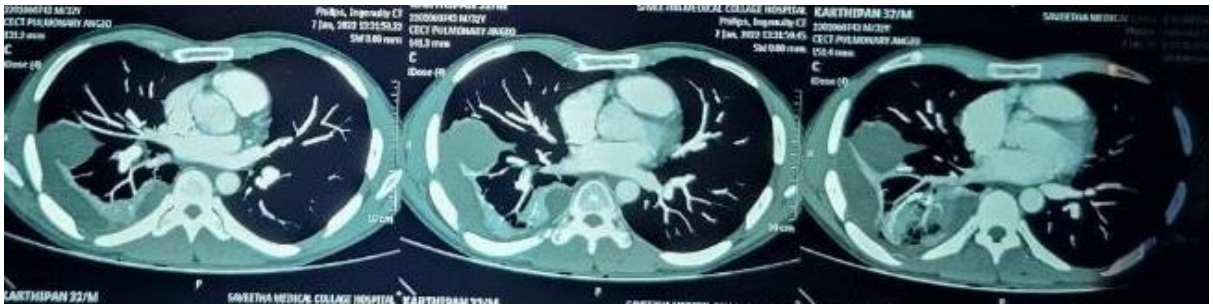


IMAGE 2



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KEYWORDS: Pulmonary thromboembolism, hyperhomocysteinemia, deep vein thrombosis