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Original Research Paper

General Medicine

A 22 YEAR OLD WITH ACUTE PTE – UNPROVOKED.

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ABSTRACT Pulmonary embolism accounts to one -third causes of cardiovascular mortality. It is responsible for 1,00,000 cardiovascular disease related deaths.Only 7% of the patients with PTE who die are correctly diagnosed before death. There are acquired and genetic risk factors responsible for thromboembolism. Recent surgery,trauma,immobilisation, pregnancy, OCPs are commonest acquired factors which are most of the time temporary in nature.Other factors such as smoking, atherosclerosis, obesity, malignancies pose greatest risk for PTE. Pulmonary embolism in young adults is not very uncommon.Most of the young patients reported in the literature are young females either pregnant or on OCPs. Here we present a case of 21 yr old male with acute PTE diagnosed to be protein C deficient.

KEYWORDS :

INTRODUCTION:

pulmonary thromboembolism is an acute emergency encountered in clinical practice Most patients die within first hour of presentation.Untreated cases carry high mortality(30%).Therefore it is essential to undergo a comprehensive evaluation for the cause of PTE.

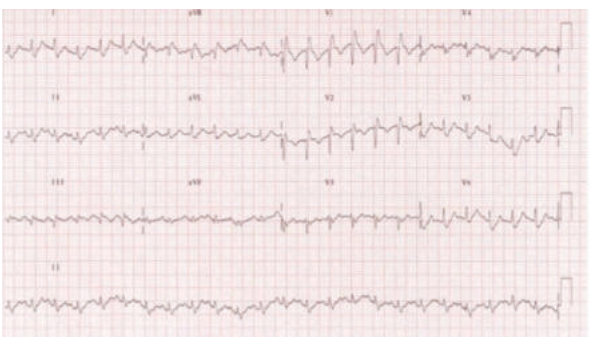
Here is a case of 22 year old medical student presented with acute shortness of breath with no underlying risk factors who got diagnosed with acute PTE evaluated for its cause.

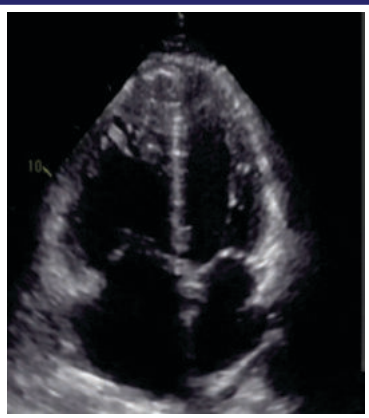
Case Report:

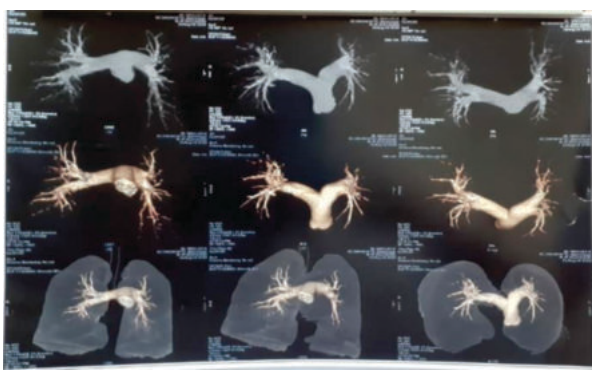
A 22 yr old male with no comorbidities presented to ER with chest pain and shortness of breath since four hours. patient was tacypneic with PR:125/min BP 90/60mmhg with saturation of 88% on room air.

On examination there is raised JVP.ECG showed sinus tachycardia with RBBB. Echocardiography showed dilated RA,RV and IVC plethora with EF of 56%.His Dimers were 3800.CT pulmonary angiography showed acute PTE involving posterobasal and lateral basal segmental arteries of right lung and lateral basal segments of left lung.He was thrombolysed with third generation thrombolytic agent. Patient was managed in ICU and treated with anticoagulants and other supportive medications.

Repeat CTPA was done as patient had streaky hemoptysis.It showed complete resolution of Patient was discharged in a stable condition with NOACS and vitamin B12 and folate supplements and asked for review with coagulation profile after two weeks







During follow up patient was found to have hyper homocysteinemia(>50micromol/lit);Activated protein C/ factor 5 - low normalised ratio suggestive of APC resistance. Other investigations: protein C :90.7IU; AT 3 - 0.25g/L.

DISCUSSION:

PTE is the condition in which thrombus or multiple thrombi migrate from systemic circulation to pulmonary vasculature Major risk factors for PTE include surgery,active cancer,immobility,trauma or fracture,pregnancy ,estrogen therapy and hereditary protein C mutations.

Protein C deficiency incidence is 1 in 200 - 1in 500.However many individuals are asymptomatic.The cardinal manifestation is venous thromboembolism. Protein C is

activated to activated protein C via proteolytic cleavage by thrombin bound to thrombomodulin,an endothelial cell surface membrane protein.APC downregulates the procoagulant system by inactivating 5α and 8α.APC resistance is characterised by α reduced anticoagulant response of patient plasma after adding α standard amount of APC

CONCLUSION:

Protein C deficiency (autosomal recessive) and its coinheritance with deficiency of factor 5 leiden(FVL) in heterozygous carriers results in high degrees of penetrance. The inherited heterozygous state and its deficiency are most frequently associated with DVT and PTE .Majority are asymptomatic.Two types of protein C are described.Type 1 is associated with quantitative deficiency and Type 2 associated with molecular structure.Recently APC's systemic anticoagulant activity and its beneficial anti inflammatory effects were more closely revisited for its ability to down regulate thrombin.

Protein C is α VIT K dependant glycoprotein synthesised in the liver and is circulated as an inactive zymogen.Activation of protein C(APC), α serine protease enzyme is catalysed by thrombin. APC exerts its anticoagulant activity primarily through inactivation of factors 5 and 8. APC is enhanced by vit K dependant cofactor protein S. So,it's deficiency disturbs the delicate balance between the procoagulant and anticoagulant proteins and causing α prothrombotic state.

Classic manifestation of protein C deficiency is venous thromboembolism.Arterial strokes and MI can also occur but their association with APC remains controversial.

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