INTERNATIONAL JOURNAL
Research in
Biological Science
www.gphjournal.org || editor@gphjournal.org

Case Report | Vol. 04 Issue 09 Sept - 2021 | Manuscript ID: #0484

UNDERDIAGNOSIS OF ACUTE PULMONARY EMBOLISM IN PHYSICALLY ACTIVE MIDDLE AGE MAN WITH SYNCOPE

***Keshav Budhathoki, Shyam raj Regmi, Sudhir Regmi,**
Bishnu Mani Dhital, Ananda GC, Sabina Sedhai
Department of Cardiology, Chitwan Medical College, Chitwan, Nepal.

Corresponding author: *Keshav Budhathoki
Email : keshavbudhathoki@gmail.com

ABSTRACT

Acute pulmonary embolism is a component of venous thromboembolism which may prove fatal if not suspected and subsequently treated. So, it is a disease that needs high clinical suspicion to prevent mortality and morbidity. Suspicion is very low in young healthy individuals in comparison to old age individuals with multiple co-morbid conditions. A physically active individual without genetic predisposition is considered to be at low risk for deep vein thrombosis and pulmonary embolism. Here, we present one overweight case who had 3 episodes of syncope in 4 days interval as well as breathlessness who came to our hospital and was diagnosed and treated with acute pulmonary thromboembolism.

KEYWORDS

Pulmonary embolism, physically active, overweight, STK, NOACs

INTRODUCTION

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] Young patients are more likely to be mismanaged as clinical suspicion in healthier physically active patient is very low and in spite of various diagnostic modalities, high clinical suspicion remains the key for diagnosis. The most common risk factors for DVT and pulmonary embolism include prolonged immobility, older age, history of smoking, inherited clotting factors and post-operative condition.[4-7] without genetic predisposition in physically active healthy individual, it is considered low risk for DVT and pulmonary embolism.

Case report

A 49 years middle aged, physically active but overweight patient with no known co-morbidity, presented in our Cardiology OPD 4 days after with the history of first syncope while going for a morning walk then he presented to emergency department of some hospital where some simple blood test were done and found to be normal and discharged from emergency department. After returning back home, he feels breathlessness somehow but he stayed at his residence for 2 days. As the breathlessness keeps on increasing, on the 3rd day, he planned to visit the hospital again where he was consulted first. From that hospital, he was referred to other hospital for echocardiography where found only concentric LVH but then he was asked to go to other hospital having the facility of coronary angiography. Besides coming to hospital with CATH-LAB facility, he went back home. He feels breathlessness keeps on increasing even in rest. So, he again appeared to the hospital where echocardiography was done. On the way to hospital, he had syncope inside the vehicle two times lasting few seconds. Again from that hospital, he was referred to our Cardiology OPD for further checkup. He looks clinically dyspnoeic but physical examination was normal besides his RR being 22 per minute and tachycardia. He weighs 100kg with height 5.8 feet and BMI 32.4.

We did echocardiography immediately where we found dilated RA and RV with moderate PAH without McConnels sign. We repeated ECG which reveals sinus tachycardia @108bpm with S1Q3T3. We immediately asked patient and patient party to undergo CT Pulmonary angiogram thinking that it could be pulmonary embolism. Immediately CT pulmonary embolism report showed saddle embolus in the pulmonary bifurcation along with embolism involving the bilateral main, lobar and segmental arteries. He immediately got admitted to the CCU from where all the blood tests were sent for analysis. After this, we thrombolysed the patient with loading and maintenance dose of streptokinase and then continued LMWH. 48 hours after thrombolysis, we added warfarin despite continuation of LMWH. His blood test for hypercoaguable states showed gene mutation for MTHFR C677T otherwise normal. CT pulmonary angiogram got repeated on fifth day which showed clear pulmonary artery and its branches. As patient improved a lot from second day, on sixth day, his medication warfarin changed to dabigatran and discharged on seventh day and is under follow up doing good since one and half years with NOACs.

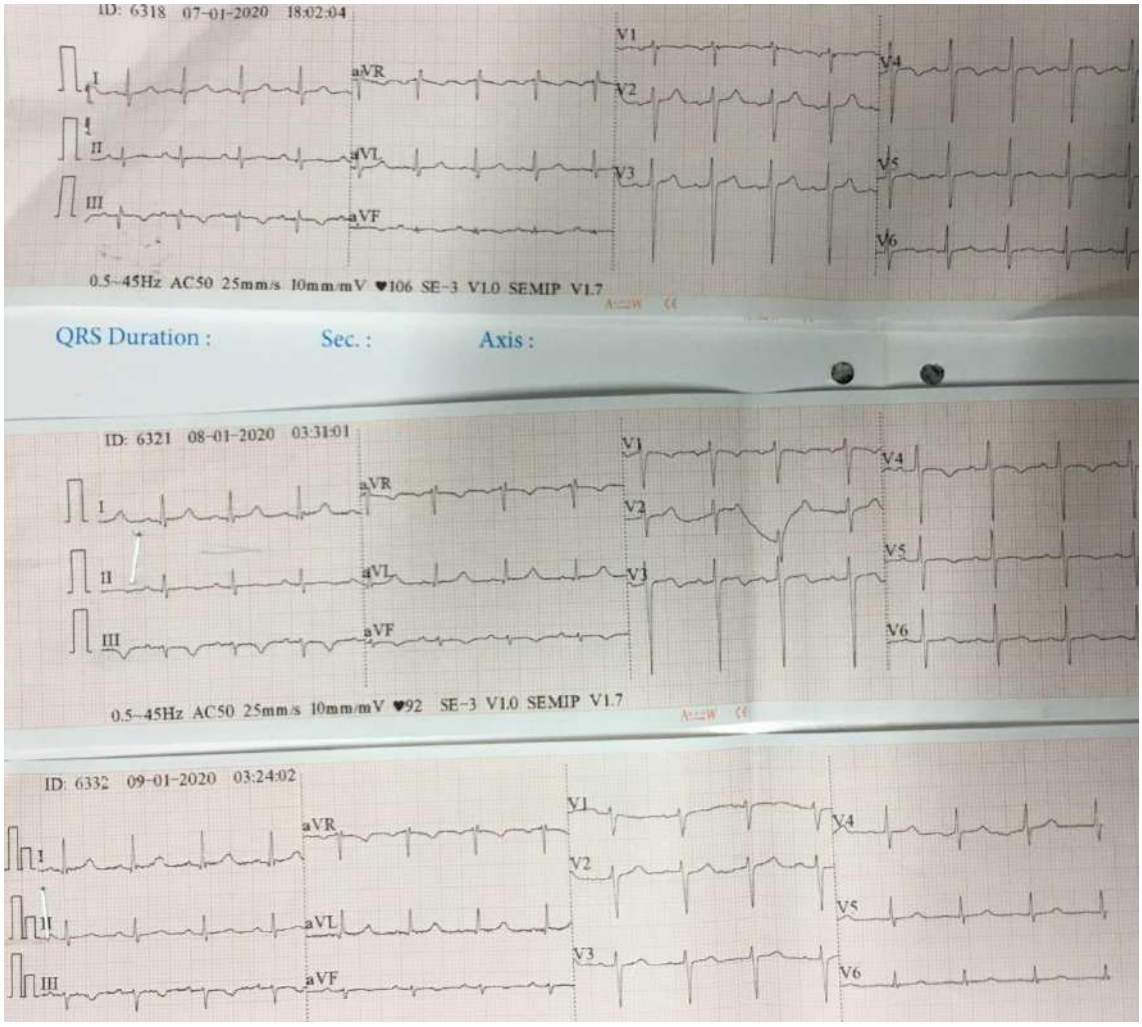

Fig 1: Three days serial ECGs from day of an admission.

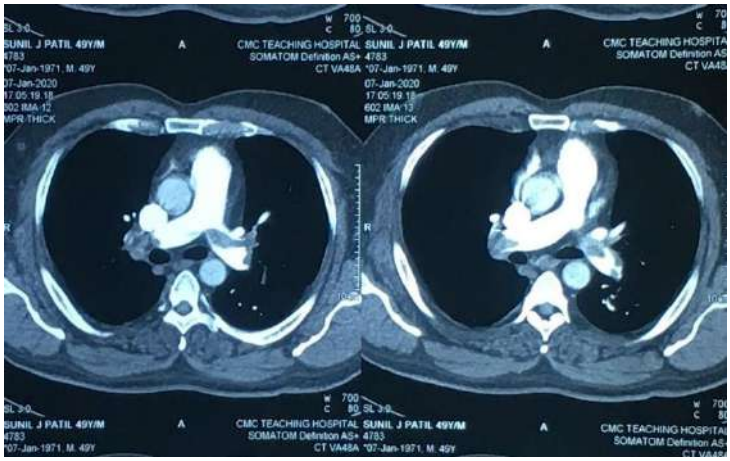

Fig 2: CTPA showing filling defect in the right and left MPA including bifurcation.

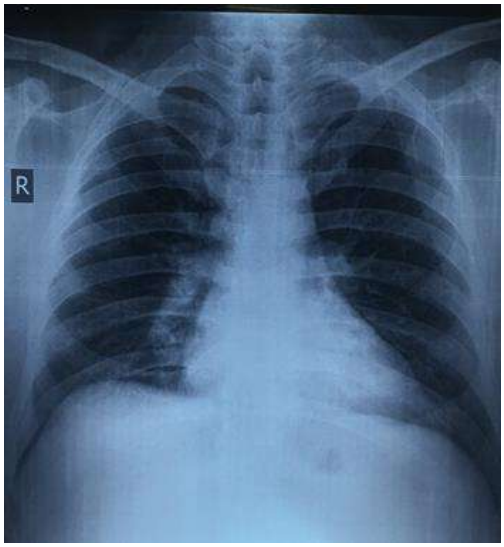

Fig 3: Normal CXR PA view

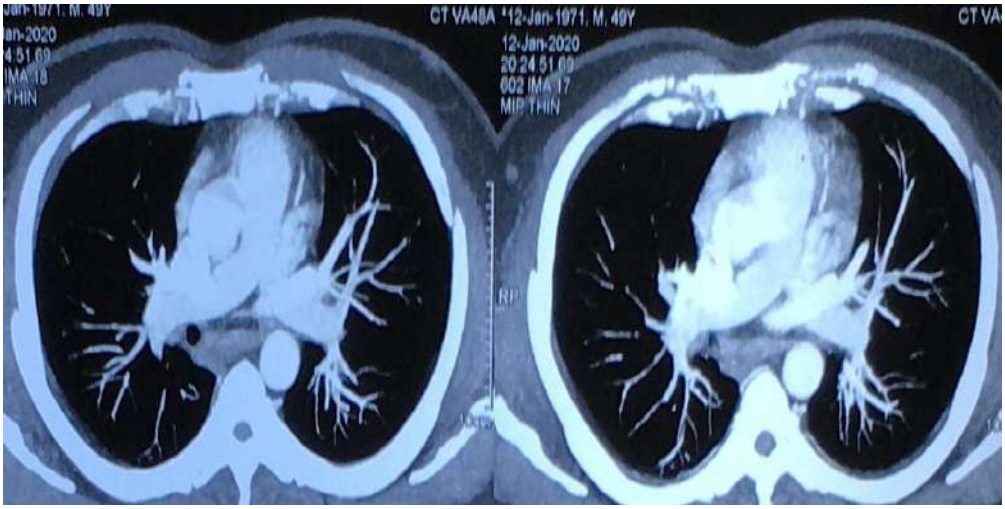

Fig 4: Five days after Thrombolysis with STK.

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 the commonest acquired factors which are most of the time temporary in nature.[8] Other factors such as malignancies, for example hematological, lungs, pancreatic and brain cancer pose the greatest risk for PE, and cancer association is the predictor of increased mortality. Smoking, obesity, atherosclerosis, hypertension and infection in hospitalized patient are also common cause of PE. Deep vein thrombosis present in 79% of cases of PE and 40-50% of DVT is complicated by pulmonary embolism. Inherited risk factor includes antithrombin III deficiency, protein C, protein S, factor leiden V mutation etc. Smoking and obesity are independent acquired risk factors for PE.

Cough, hemoptysis, breathlessness and chest pain were common symptoms which are common but not specific for PE as well as sinus tachycardia and tachypnea and low blood pressure. In our patient, he had only tachypnea and breathlessness. Classical triad of chest pain, hemoptysis and dyspnea 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 [9] and our patient had more than four score on initial presentation. CXR is useful to rule out other causes of dyspnea like pneumothorax. Hampton's hump (subpleural edge shaped opacity), Westermarck's sign (areas of hyperlucency), raised hemidiaphragm, pleural effusion and normal chest radiograph are other findings on CXR.[10] 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 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%. [11]

CT pulmonary angiography 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.[12] 2D ECHO revealed moderate PAH without McConnel's sign in our case. Mcconnel's sign has high positive predictive value for pulmonary embolism even in presence of other co-morbid conditions. [13]RV dilatation, presence of thrombi in RV is other important finding in 2D ECHO. Compression ultrasound of 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.

In our case, we treated our patient with streptokinase later with anticoagulation and supportive care. It is 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.

CONCLUSION

Acute pulmonary embolism in a physical active middle aged man is not very uncommon. Gene mutations proved to play an important role in the development of pulmonary embolism that might be accompanied by other thromboses elsewhere or the patients might be at risk of recurrence of PE attacks. This necessitates testing for the gene mutations, especially that for the FVL in young patients who present with venous thrombo-embolism or recurrent thrombo-embolic manifestations. Even though middle aged physical active man with or without co-morbidity when presented with syncope, pulmonary embolism should be considered as possibility as 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 the disease. Unlike infectious disease, in cardiac disorder or blood disorder, no specific symptoms, signs or investigations reflects a disease process immediately. Hence, middle aged male even with or without co-morbidity presenting with syncope should be evaluated for pulmonary embolism so as not to miss this fatal disease.

References

1. Heit JA. The epidemiology of venous thromboembolism in the community. ArterosclerThrombVascBiol 2008; 28:370-2.
2. Goldhaber SZ, Hennekens CH, Evans DA, Newton EC, Goldleski JJ. Factors associated with correct antemortem diagnosis of major pulmonary embolism. Am J Med 1982; 73: 822-6
3. Rubeinstein I, Murray D, Hoffstein V. Fatal pulmonary emboli in hospitalised patients: An autopsy study. Arch Intern Med 1988;148: 1425-6.
4. Heit JA, Silverstein MD, Mohr DN, Petterson TM, O'Fallon WM, Melton LJ, 3rd. Risk factors for deep vein thrombosis and pulmonary embolism: a population-based case-control study. Arch Intern Med. 2000;160: 809-15.
5. Silverstein MD, Heit JA, Mohr DN, Petterson TM, O'Fallon WM, Melton LJ, 3rd. Trends in the incidence of deep vein thrombosis and pulmonary embolism: a 25-year population-based study. Arch Intern Med. 1998; 158: 585-93.
6. 6. Inman WH, Vessey MP, Westerholm B, Engelund A. Thromboembolic disease and the steroidal content of oral contraceptives. A report to the Committee on Safety of Drugs. BMJ. 1970;2: 203-9.
7. Realini JP, Goldzieher JW. Oral contraceptives and cardiovascular disease: a critique of the epidemiologic studies. Am J Obstet Gynecol. 1985;152: 729- 98
8. Anderson FA Jr, Spencer FA. Risk factors for venous thromboembolism. Circulation 2003; 107: I9-16.
9. Wells PS, Ginsberg JS, Anderson DR, Kearon C, Gent M, Turpie AG et al. Use of clinical model for safe management of patients with suspected pulmonary embolism. Ann Intern Med 1998; 129: 997-1005.
10. Elliot CG, Goldhaber SZ, Visani L, De Rosa M. Chest radiography in acute pulmonary embolism. Results from the International Cooperative Pulmonary Embolism Registry. Chest 2000; 118: 33-8.
11. Ferrari E, Imbert A, Chevalier T, Mihoubi A, Morand P, Baudouy M. The ECG in pulmonary embolism. Predictive value of negative T waves in precordial leads − 80 case reports. Chest 1997; 111: 537-43.
12. Wells PS, Anderson DR, Rodger M, Steill I, Dreyer JF, Barnes D et al. Excluding pulmonary embolism at the bedside without diagnostic imaging: Management of patients with suspected pulmonary embolism presenting to the emergency department by using a simple clinical model and d-dimer. Ann Intern Med 2001; 135: 98-107.
13. Stein PD, Fowler SD, Goodman LR, Gottschalk A, Hales A, Hull RD et al. Multi detector computed tomography for acute pulmonary embolism. N Engl J Med 2006; 354:2317-27.