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Original Article

Deep Vein Thrombosis(DVT)-Pulmonary Embolism(PE) masquerading as Anterior Wall Myocardial Infarction, late presentation : A case report and review of literature

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Summary

We present an interesting case of young male patient with ECG findings of ST-T changes of late presentation of Anterior wall MI , which turned out to be a case of Deep Vein Thrombosis-Pulmonary Embolism. This presentation of PE is very rare and can easily be misdiagnosed, hence being reported. (Indian J Cardiol 2022;25 (3-4):36-39)

Introduction

Increasing availability of Electrocardiogram (ECG)in rural medical settings has helped in earlier diagnoses of Acute Coronary Syndrome(ACS) and triage of patients based on ST-T changes into ST Elevation Myocardial Infarction (STEMI) and Non STEMI(NSTEMI)/Unstable Angina. Urgent revascularization is recommended for patients with chest pain and STEMI by percutaneous coronary intervention (PCI) or fibrinolysis as per available facilities and time period passed since onset of symptoms and arrival at health care set-up[1,2]. ECG findings of ST elevation can have a variety of causes other than MI and erroneous diagnosis of STEMI can lead not only to delayed appropriate treatment for underlying true cause but also increased risk of mortality. Pulmonary Embolism(PE) rarely can present with ST elevation in ECG[3-5]. Both STEMI and PE patients can present with chest pain, dyspnea, palpitation and syncope. Physical examination usually cannot differentiate satisfactorily between these two entities. ECG alone has low sensitivity and specificity[6-8]. Appropriate diagnosis is critical due to fundamental differences in treatment modalities of arterial pathology in STEMI and venous pathology in PE/ Deep Vein Thrombosis (DVT) and risk of recurrence and fatal outcome in undiagnosed and untreated patients of PE-DVT. We report a case of young male who had ECG changes of anterior wall MI but turned out to be PE/DVT.

Case report

A 45 year old male patient, a referred case from peripheral hospital with history of irregular medication for hypertension since last 3 years and complaint of progressive dyspnea on exertion with mild chest discomfort since last 8-10 days with provisional diagnosis of ACS- Anterior Wall myocardial infarction-late presentation (Fig. 1 ECG), started on dual antiplatelet therapy and antianginal medication already and counseled about need for coronary angiogram. Patient neither had any previous history of Coronary artery disease ,Diabetes, Peripheral Vascular Disease and Stroke; nor family history of similar diseases. He was afebrile with a regular pulse rate of 105 beats/min. His blood pressure was 110/65 mmHg, respiratory rate was 22 breaths/ min, oxygen saturation was 90% at room air and 98% with 2l/min oxygen via nasal prongs. Cardiac

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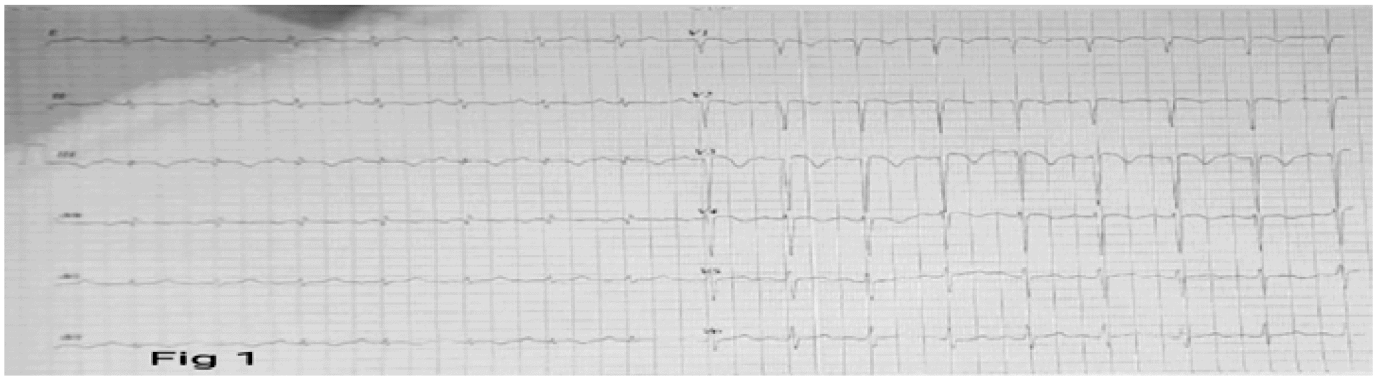

Fig. 1 : ECG at presentation

auscultation and neck vein examination were grossly unremarkable. An initial ECG showed a sinus tachycardia, qs with t wave inversion in V1-V3, poor progression of R wave till V5, large S wave in v6, t inversion in leads ii, iii & avF, q wave in lead iii (Fig 1).Trans-thorasic echocardiography (TTE) revealed right ventricular dilatation, right atrial dilatation, diastolic flattening of inter-ventricular septum, Pulmonary artery dilated, decreased TAPSE (11.1 mm), low pressure severe Tricuspid regurgitation with RVSP ~ 35+15=50 mmHg and PAT < 60 mS, all echo findings highly suggestive of Acute Cor Pulmonale. (Fig 2A). D-dimer assessment was done and values was significantly raised, 2705 ng/ml (normal value < 500 ng/ml). Patient was taken for Computed Tomography Pulmonary Angiography(CTPA), which showed hypodense thrombus in bilateral main pulmonary arteries and inferior segmental branches (Figure 2B). Venous Doppler study of bilateral lower limbs showed hypoechoic thrombus in left Poplitial veins and calf veins compatible with diagnosis of deep venous thrombosis.. An etiological investigation of thromboembolic disease demonstrated the presence of high Homo-cysteine levels , 23.68 micromol/L (normal reference range 3.75-15 micromol/L).Protein C & S, factor V Leiden and APLA assessment were refused by patient. In view of significant RV dysfunction and low SPO2 levels, patient was planned for systemic thrombolysis by Streptokinase infusion for 24 hours along with subcutaneous enoxaparin, 1mg/kg body weight in BD doses. The patient responded well to treatment ; his symptoms subsided significantly in 24 hours with maintenance of SPO2 above 95% at room air.ECG changes resolved partially. (Fig 3A). Transthoracic Echo post thrombolysis showed resolution of RA/RV dilatation, improvement of TAPSE upto absolute value of 16.1 mm, PAT ~90 mS, mild TR with RVSP ~15+5 =20 mmHg.(Fig 3B). oral

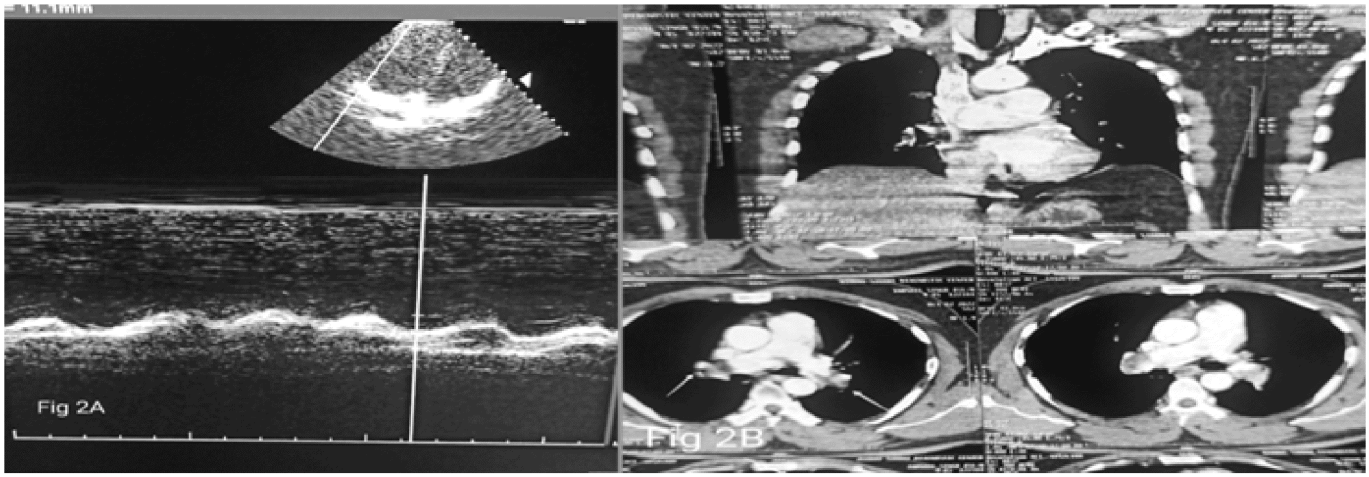

Fig. 2 : Signicantly deranged TAPSE on echoradiographic examination (2A) and CT pulmonary angiogram showing hypodense thrombus denoted by arrows(2B).

anticoagulation was initiated during hospitalization only in form of Rivaroxaban 15 mg BD.Patient was discharged with advise of regular follow up.Coronary angiography performed later showed normal coronary arteries withoutany significant stenosis

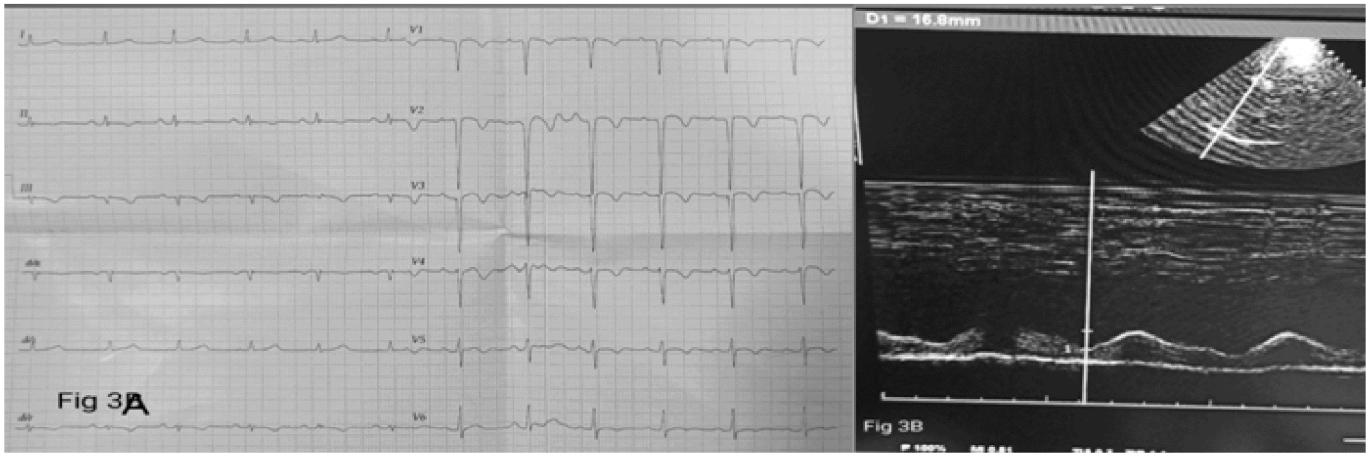

Fig. 3 : Post thrombolysis ECG (3A) and significant improvement inTAPSE on Echocardioghic examination(3B)

Discussion

The American College of Cardiology/American Heart Association (ACC/AHA) recommendsimmediate reperfusion therapy for patients with STEMI[2]. Problem arises with the fact that significant proportion of patient with ECG changes suggestive of STEMIcan havefinal diagnosis something else other than AMI[9]. The differential diagnosis of STEMI can include conditions associated with myocardium,pericardium or even extracardiaccauses[10,11].These causes include but not limited to acute pericarditis, aortic dissection, left ventricular aneurysm, Brugada syndrome , Prinzmetal's angina, hyperkalemia, hypothermia, Early Repolarization Syndrome, blunt Trauma to chest and rarely Pulmonary Embolism[10,12]. ST changes in reciprocal leads can be a clue for differentiating ischemic from nonischemic etiologies[13]. Typical ECG findings of PE are summarized as S1Q3T3 pattern; other findings aretransientincomplete or complete right bundlebranch block (RBBB), right axis deviation and negative T waves in the right precordial leads, anterior and inferior leads,deepS waves in I and aVL, Q waves in III and aVF and low QRS voltage in extremity leads[14,15]. ST elevation is a rare ECG finding in PE[3,16-20]. ECG findings suggestive of Anterior wall STEMI is very rarely associated with PE. Probable etiology of these ST-T changes is thought to be a sub massive or massive PE resulting in elevated right ventricular pressure and strain[21]. Normalization of these ECG abnormalities in a 6 weeks period has been documented[22]. Trans Thoracic Echocardiogram can differentiate between fatal causes of STEMI i.e, MI, PE and Aortic dissection and guide to initiation of appropriate targeted treatment[1]. Judicious use of biochemistry (D -Dimer and extended Coagulation Profile), radiological investigation(CT pulmonary angiogram, Aortogram, HRCT chest and Duplex studies of limbs) is often fruitful. While patients of Aortic dissection often need urgent cardio-thoracic surgery services and fibrinolysis may prove fatal in this condition, patients of both STEMI and PE may benefit from fibrinolysis done in the window period. Both STEMI and PE will warrant prolonged, often life long medical therapy targeted to underlying baseline pathology and comorbid conditions. If a patient of pulmonary embolism with ECG changes suggestive of STEMI is thrombolysed initially and undergoes coronary angiography later, he will carry probably a diagnosis of "STEMI with normal/recanalized coronary arteries" for rest of his life and high probability of recurrent PE/DVT event poses a threat to these patients.

Conclusion

While confronting ECG findings of ST elevation, a clinician must combine not only other ECG findings such as reciprocal changes and s1q3t3 but also give attention to typical nature of chest pain, physical

examination to rule out other potential causes and use of trans esophageal Echo if possible, rather than jumping to reflex mediated diagnosis of acute MI at once. This approach can increase chances of more accurate cause specific treatment while minimizing chances of unwanted and potentially lethal treatment modalities.

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