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DECODING SYNCOPE : RECOGNIZING PULMONARY THROMBOEMBOLISM SYMPTOMS

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INTRODUCTION

Pulmonary embolism is an acute emergency that can have fatal consequences, can present initially with a common symptom like syncope, has proven to be a difficult correlation to make a diagnosis as acute pulmonary embolism due to variety of symptoms and signs of presentation. Indian data suggest overall incidence of pulmonary thromboembolism in adults being 15.9% and as a cause for mortality is 80%.[1]In this case we have a 55 year old man with a history of long travel everyday presenting with syncope and dyspnea that was eventually diagnosed as acute pulmonary thromboembolism. It is a uncommon cause of a common condition which when missed could have led to a fatal outcome.

KEY WORDS: Pulmonary thromboembolism, Syncope.

CASE PRESENTATION

A 55 year old male who is a known case of gouty arthritis not on any medication, with no history of recent trauma or fracture and surgery in the past 3 months was admitted to JSS hospital, after he had a syncopal episode at his workplace. Patient was in his usual state of health, until he suddenly collapsed while standing and had a brief loss of consciousness for approximately 5 minute, which he recovered spontaneously, followed by an episode of vomiting and later developed breathlessness and was immediately rushed to the hospital. On arrival to the emergency department, he was tachypneic, diaphoretic and complained of breathlessness on exertion but denied any associated chest pain, palpitations or paroxysmal nocturnal dyspnea. There was no history of involuntary movements, tongue bite, urinary or fecal incontinence, blurring of vision or weakness of limbs.He is a chronic alcoholic and smoker. The patient was a goodsdeliverer by occupation where he would travel 8-9hours a day around 5 times per week. On admission, physical examination revealed heart rate of 114 bpm regular, Blood pressure was 128/80mmHg and his Respiratory rate was 33 cycles/minute. Room air saturation was 88% and shifted to ICU with oxygen support. Arterial blood gas analysis at point of care showed hypoxia with PaO_2–58mmhg. Examination of his head and neck was normal. Lung auscultation appeared normal and there was no focal neurological deficits. Electrocardiogram showed sinus tachycardia with S1Q3T3

and T wave inversions in V1-V4. Serial cardiac markers were within normal limits. Chest 'X' ray showed no radiological abnormality. Levels of serum electrolytes, urea, creatinine, blood glucose, thyroid profile, complete blood counts and PT/INR were within normal limits. Severe dyspnea with clear lungs and classical ECG changes led us to a suspicion of Pulmonary thromboembolism and investigated for D-dimer level which was raised(1.4 microgram/ml). Thrombophilia profile which included antithrombin, protein C, protein S, factor v Leiden mutation was sent before starting low molecular weight heparin based on body weight, which was negative.A transthoracic echocardiogram revealed RA and RV dilatation with depressed Right ventricular function, severe PAH, estimated PASP-65mmHg. A doppler scan of both lower limbs revealed no evidence of deep vein thrombosis. CT pulmonary angiogram showed acute pulmonary thromboembolism of right and left main pulmonary arteries with extension into lobar and segmental branches, dilated main and bilateral pulmonary arteries-pulmonary hypertension. Ultrasound abdomen showed fatty liver. Bilateral upper limb arterial and venous doppler was normal. On the sixth day of admission, patient was shifted to wards with minimal oxygen support. Repeat screening ECHO showed increase of PAP to 75mmHg. Injection LMWX was stopped and switched over to oral anticoagulation Dabigatran 150mg twice a day. 24 hour Holter monitoring report showed sinus rhythm with 1:1 conduction, occasional supraventricular ectopics, no supra ventricular or ventricular arrhythmia seen, no sinus pauses or high grade AV blocks. Patient was discharged after 11 days of hospital stay in good health with no oxygen support, hemodynamically stable with oral anticoagulation. On regular follow up he is continued to be free of symptoms and repeat ECHO showed improvement in RV with PASP of 46mmHg

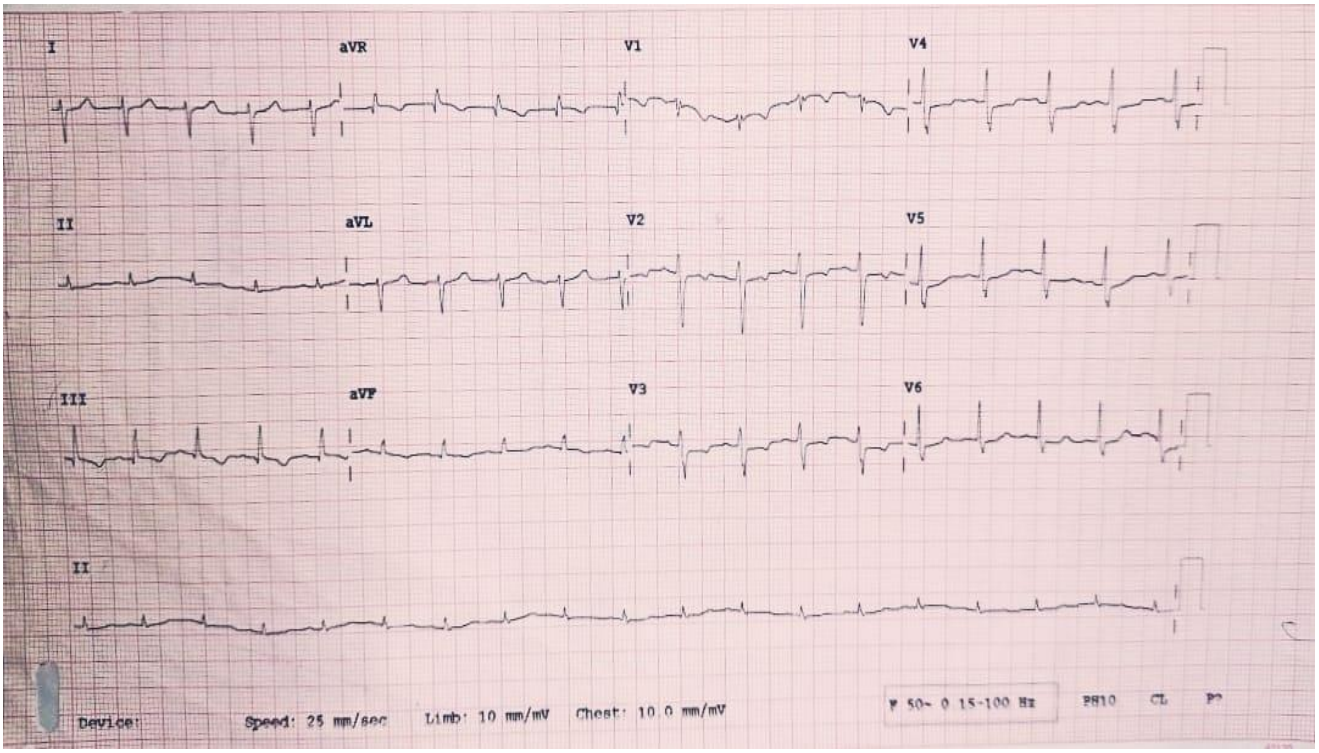

FIG 1 – (ECG SHOWING S1Q3T3 pattern)

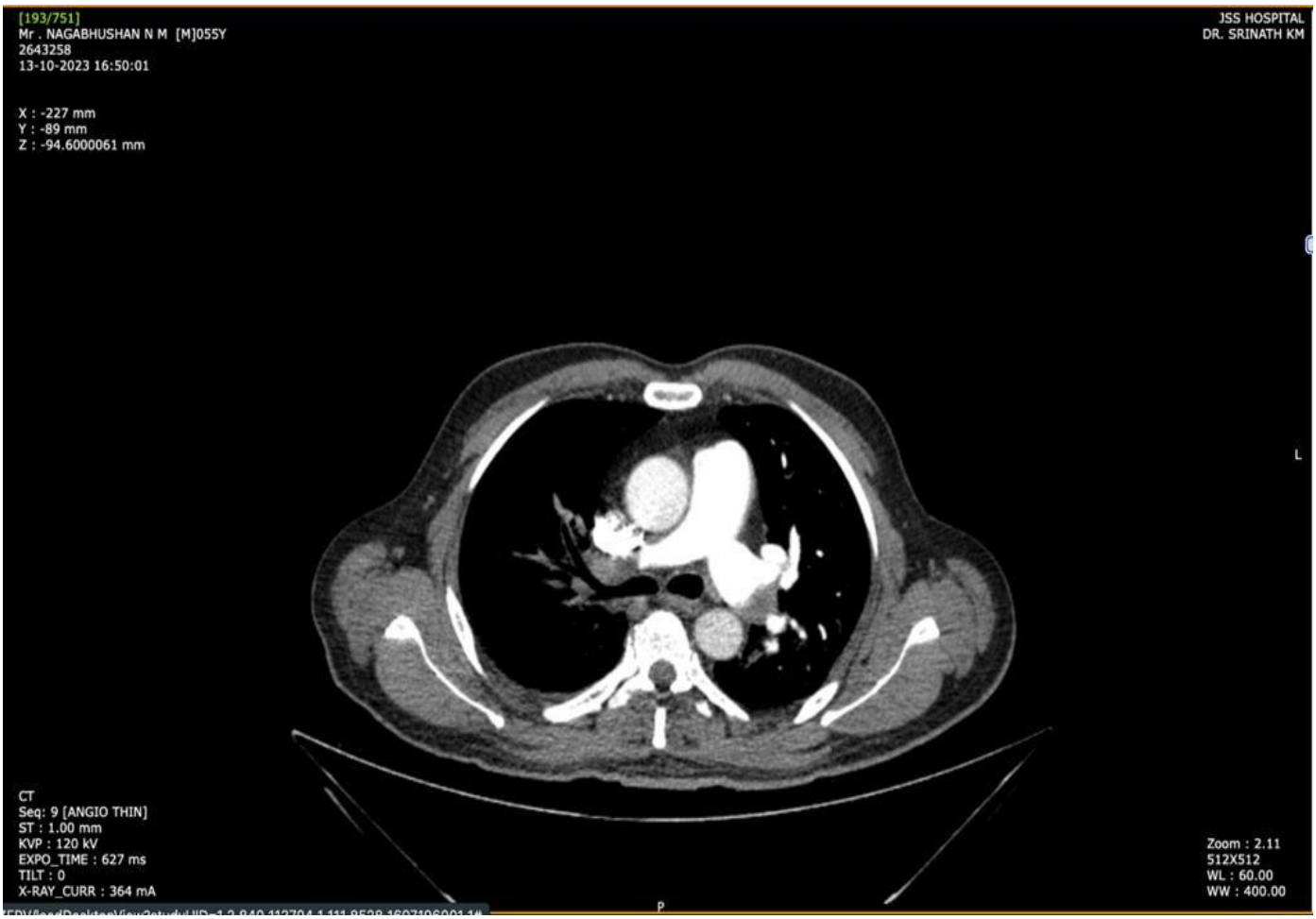

FIG 2 – (CT PULMONARY ANGIOGRAM SHOWING ACUTE PULMONARY THROMBOEMBOLISM OF RIGHT AND LEFT MAIN PULMONARY ARTERIES, DILATED MAIN AND BILATERAL PULMONARY ARTERIES- PULMONARY HYPERTENSION)

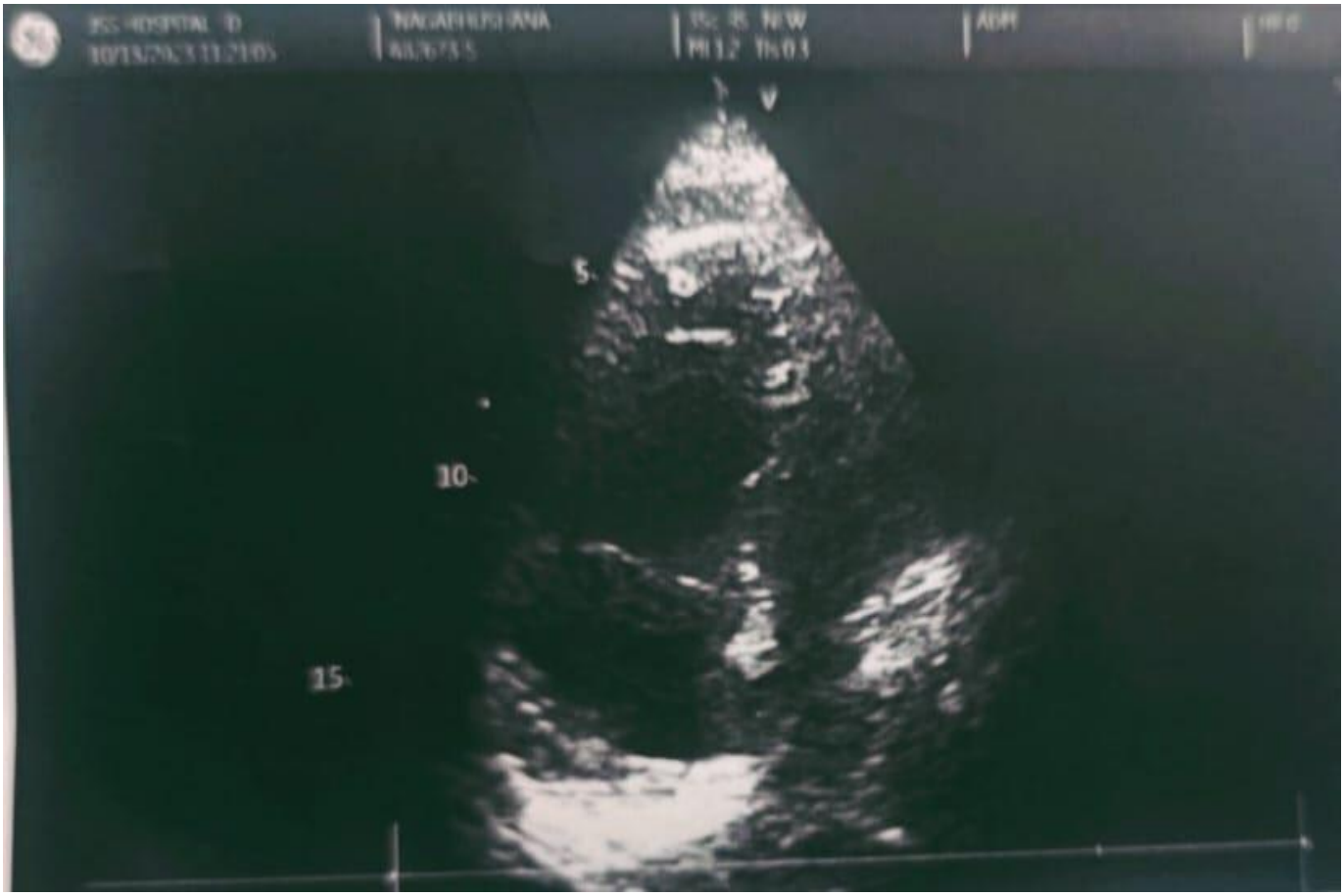

FIG 3: (2D ECHOCARDIOGRAPHY SHOWING RA AND RV DILATATION)

DISCUSSION

Pulmonary thromboembolism has a wide range of clinical presentations and is a frequent cause of death, hence timely diagnosis and apt management is of utmost importance to reduce mortality. The classic triad of pleuritic chest pain, dyspnea and hemoptysis is common however many patients with large pulmonary thromboembolism have mild or nonspecific symptoms or are asymptomatic. Syncope is a possible but littleknown presenting symptom of acute pulmonary thromboembolism. Its presence causes difficulty in making a diagnosis.[3]Syncope in pulmonary thromboembolism could be due to acute right ventricular failure due to occlusion of pulmonary vasculature causing reduced left ventricular filling leading to reduced cardiac output. Other mechanism could be vasovagal reflex causing neurogenic syncope or development of complete bundle brunch block in presence of pre-existing left bundle branch block.[4] Syncope as initial presentation in acute pulmonary thromboembolism occurs in 10% of patients, one study showed that syncope as a presentation of pulmonary thromboembolism could be due to massive, main pulmonary artery embolism, prognosis of these cases depends on timely diagnosis and management.[3]The clinician should seek the following clues to the diagnosis of pulmonary thromboembolism in a patient presenting with syncope – Hypotension, tachycardia, Hypoxemia and features of acute corpulmonale as they are at risk of shock or sudden cardiac death.[7]The presence of these signs in the absence of other common causes of syncope should lead to further workup like arterial

blood gas analysis, D-dimer, lower limb venous doppler, 2D Echocardiography, CT pulmonary angiogram.[8]Well's score can be calculated for clinical probability of pulmonary thromboembolism. High probability of pulmonary thromboembolism warrants for more definitive testing like CT pulmonary angiogram. Based on the risk stratification, acute pulmonary thromboembolism can be classified into low risk, sub-massive and massive pulmonary thromboembolism.[6]ECG is a simple and easy tool available readily in most hospitals. Most common ECG finding is sinus tachycardia, however S1Q3T3 pattern has low sensitivity and specificity.[2] , [5]Management with anticoagulants alone is typically sufficient for low-risk patients, more aggressive treatments such as thrombolysis, embolectomy, and inferior vena cava (IVC) filters are recommended for higher-risk patients. Thrombolytic therapy should be considered in all patients with massive pulmonary thromboembolism and hypotension associated with deep vein thrombosis in the popliteal area or higher.

CONCLUSION

Pulmonary embolism must be considered and excluded in patients presenting with syncope. Awareness should be raised regarding the diagnosis and clinical presentation of pulmonary embolism for timely management.

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