International Journal of Science and Research (IJSR)
ISSN: 2319-7064
SJIF (2022): 7.942

Severe Young Pulmonary Thrombosis in a Mild COVID-19

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Abstract: ***21 year old second M.B.B.S. medical student admitted in Government College and Hospital with complaints of productive cough since 9 days followed by sudden onset breathlessness since 7 days. Patient received Second dose of Covishield vaccine one month prior to admission. He did not give history of COVID 19 infection in the past but he had exposure to his infected friends. There were no known risk factors. On admission pulse rate was 140/min and blood pressure was 120/70mmhg and Saturation was 90% on room air for which patient started on Oxygen therapy. ECG was suggestive of Sinus tachycardia with S1T3 pattern. D-Dimer was raised markedly i.e. 3360 mg/lit. Bilateral lower limb Doppler study was normal. CT with pulmonary angiography done which suggests Massive pulmonary thromboembolism. 2D-Echo was suggestive of RV dysfunction. Patient thrombolysed with tenectaplase 40mg and is started on heparin and oral anticoagulant therapy and discharged after stabilisation.***

Keywords: COVID-19, thromboembolism, Covishield

1. Introduction

There is COVID 19 pandemic which apart from causing deaths affected every aspect of human life. The severity of lung involvement ranges from asymptomatic to critical disease. Hypercoagulabilty is common in COVID 19 patients. The mechanisms contributing to increased thrombosis in COVID 19 involve extensive cross talk between hemostasis and immune system [(10).] Venous thromboembolism encompasses deep venous thrombosis DVT and pulmonary embolism (PE) and causes cardiovascular death and disability. PE is known as the great Masquerader. It is the most common preventable cause of death among hospitalized patients.

Idiopathic or unprovoked VTE is defined as any VTE in the absence of an identifiable predisposing factor.

2. Case Presentation

21 year old second M. B. B. S. medical student admitted with complaints of productive cough since 9 days, breathlessness since 7days. He had history of cough with expectoration approximately 10 cc per 24 hrs since 9 days. He developed sudden onset breathlessness 7 days back after 5 minutes of cycling then he stopped cycling.

He had no history of fever, no retrosternal, pleuritic chest pain, palpitations, No vomiting, sweating, No history of trauma or injury, prolonged immobilisation, air travelling, no calf pain.

At that time he visited military hospital, Aurangabad where he received 5 days antibiotics and cough syrup but his breathlessness and cough was not relieved. Hence ECG was done which showed sinus tachycardia with S wave in lead 1 & T inversion in lead 3 so he was referred to tertiary care centre.

Six months back he experienced sudden onset of breathlessness for 15 minutes while trekking that was not associated with cough. This breathlessness was relieved after 30 minutes rest.

Personal history- He used to do regular exercise of 6 km jogging per day. Normal appetite, sleep, bowel & bladder habits. No addiction.

Family history- Not suggestive of any coagulopathy disorder. Father is an army man, mother is housewife. Non consanguineous marriage.

No H/O COVID 19 positive in family.

At the time of admission he was conscious, oriented. His pulse rate was 140/min regular, and blood pressure was - 120/70mmhg. His O2 saturation was 90% on room air, 98% on O2 @ 4liters per minute and respiratory rate - 30/minutes. His BMI is 23.3 kg per m^2.

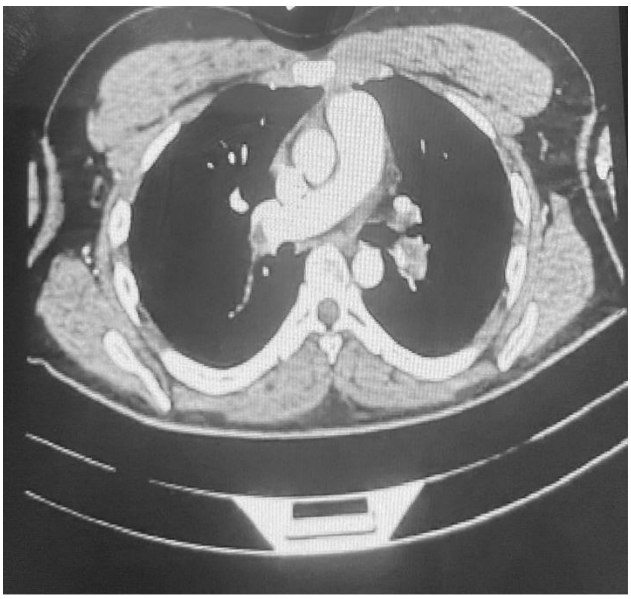
Jugular venous pressure raised (4 cm of water) and bilateral fine basal crepitations. No pedal edema. No murmur or gallop. Abdomen was soft, nontender, no organomegaly.

His ECG on admission showed heart rate 140per minute, with S wave in Lead 1 and T inversion in lead 3. Other leads normal.

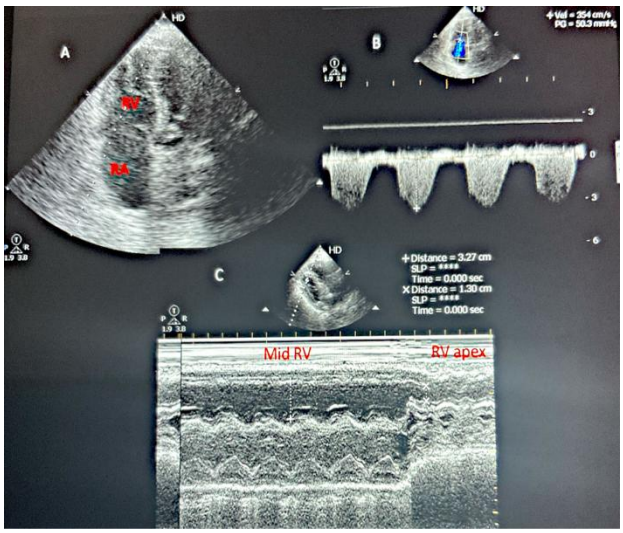
D dimer was 3360mg/L.

CT Pulmonary Angiography was suggestive of wedge shaped partial filling defect in the left main pulmonary artery, proximally extending into the main pulmonary trunk and distally into the left upper lobe segmental and subsegmental

branches, left interlobar artery and left lower lobe segmental branches of left pulmonary artery; wedge shaped partial filling defect in the distal part of right main pulmonary artery features suggestive of pulmonary thromboembolism, also CT severity score 5/25, CORADS 4 that is infective changes in lungs.



CTPA suggestive of massive pulmonary thrombosis



2 d echo showing RV dysfunction, RA, RV dilated that is also known as McConnell's sign - an early and specific indicator of acute pulmonary embolism

On 2d echo RV dysfunction that is RA, RV dilated and good LV systolic function.

Patient was thrombolysed with tenectaplase 40 mg i. v. over 30 seconds and heparin was started.

His laboratories revealed: normal complete blood count, normal renal and liver function tests and serum electrolytes; normal thrombophilia profile; normal lipid profile; normal non invasive lower extremity Doppler studies. His COVID 19 antibody titre was done which was negative for SARS CoV2 nucleocapsid antibody.

It was a case of massive pulmonary thromboembolism but the etiology remains debatable. Probably it is due to COVID coagulopathy which was remained undiagnosed.

Investigations	Reference range	Values at admission
Hb	12 - 16	16.7gm%
TLC	4000 - 11000	7700
DLC		81/13/2/4.
Platelet	1.5 - 4.5	3.03
Urea	5 - 20 mg/dl	24.7.
Creatinine	0.9 - 1.3 mg/ dl	1.23
Bilirubin	0 - 1.2	0.7
SGOT/PT	15 - 41/10 - 35	25/23
Sodium	135 - 145	135
Potassium	3.5 - 5	4.1
Chlorides	96 - 106	108
D Dimer	<500	3360mg/l
CRP	<6	6.2
Lupus anticoagulant gulant	28 - 45sec	38 sec
Antithrombin 3	17 - 30 mg/dl	22mg/dl
Cardiolipin IgG antibody	<0.8	0.43
Cardiolipin IgM	<0.8	0.52
Homocysteine	<15umol/L	12.10
Protein C	70 - 150	88.9
Protein S	70 - 140	93.7
Sr. cholesterol	125 - 200mg/dl	167mg/dl
Sr. LDL	<100mg/dl	159 mg/ dl
Sr. Triglycerides	<150 mg/dl	130 mg/dl
HDL	60 mg/dl and above	34mg/dl
VLDL	2 - 30 mg/dl	26 mg/ dl
SARS COV 2 total Antibody	>=1.0 COI	0.485 COI

His hospital course was remarkable for clinical improvement with the use of anticoagulants and thrombolysis. After thrombolysis the thrombus in the main pulmonary trunk resolved significantly in the review CT pulmonary angiography - Decrease in size and extent of pulmonary thromboembolism (previously seen thrombus in main pulmonary trunk not seen in repeat scan. Mild decrease in the thrombus load right and left pulmonary arteries). Near complete resolution of the infective changes in lung parenchyma. Near complete resolution of pulmonary infarct in right middle lobe.

3. Discussion

Hypercoaguability in COVID 19 manifests as a spectrum ranging from venous thrombosis involving pulmonary vessels, central venous sinus thrombosis (CVST) and areterial micro vascular thrombosis to large arterial thrombosis with limb ischemia, myocardial infarction, stroke and other complications like clotting of intravascular catheters [8]. A meta - analysis of 42 studies involving 8271 patients showed that overall rate of VTE is 21%.

On admission, the Wells score for PE was 4.5 with a moderate probability of having a PE for the present patient

based on the history and clinical features. [2] The well's score for DVT was 0, as there was no history of cancer, surgery, immobilization, calf swelling, superficial veins, lower limb swelling, tenderness, paralysis or previous history of DVT. Hence high clinical suspicion remains the key in diagnosing PE, especially in the absence of DVT [2].

The patient is a young male with no obvious comorbidities or risk factor other than exposure to his infected friends. He received 2 doses of Covishield vaccine and 2nd dose was received 1 month before onset of symptoms; there is a possibility of vaccine induced thromboembolic event.

Vaccine induced thrombosis and thrombocytopenia [9] (VITT) were identified in Europe after administration of Vaxzevria (Covishield in India), the COVID-19 vaccine developed by AstraZeneca and Oxford group that is also an adenovirus vector vaccine with Chimpanzee Adenovirus (ChAdOx1) as a vector encoding the spike protein antigen of the SARS - CoV - 2 [1].

On March 7, 2021, the Austrian National competent authority suspended use of one batch of Vaxzevria after reports of thromboembolic events following vaccination. But as antibody titre is negative for SARS CoV2 nucleocapsid antibody, so this possibility is ruled out.

Patient was thrombolysed with tenectaplase [4]. Massive PE is caused by obstruction of pulmonary arterial tree, leading to an acute and subsequently severe cardiopulmonary failure due to right ventricular overload [5].

Literature suggests that the risk of early death among patients with symptomatic PE is 18 fold higher when compared with the patients having DVT alone [6]. Furthermore the incidence rate of VTE is about 1.5 per 1000 person years while DVT appearance is twice common. [7].

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