

Pulmonary Thromboembolism - As a Post Covid Sequele in a Mild Category Case (Case Report)

Dr Pasumarthi Chinmai Sai Aparna¹, Dr Frank Pitchaimuthu², Dr Sonali Trivedi³

¹Post Graduate, Department of Respiratory Medicine, Jawaharlal Nehru Main Hospital and Research Centre, Bhilai, India

²Post Graduate, Department of Respiratory Medicine, Jawaharlal Nehru Main Hospital and Research Centre, Bhilai, India

³Senior Consultant, Department of Respiratory Medicine, Jawaharlal Nehru Main Hospital and Research Centre, Bhilai, India

Abstract: *Global pandemic COVID-19, though primarily presents as a lower respiratory tract infection, increasing data suggest multiorgan involvement. Several reports have been published on the cardiovascular implications of this emerging disease, among which venous thromboembolism appears to be a frequent complication, particularly in patients hospitalised for severe ards. Hereby we report a case who developed pulmonary thromboembolism after first hospitalisation for pneumonia of mild severity despite regular administration of thromboprophylaxis.*

Keywords: COVID-19, SARS COV2, Cytokine Storm, Anticoagulation, Acute Pulmonary Embolism, Virchows Triad, Thromboprophylaxis In Mild Asymptomatic Cases

1. Case Report

A previously healthy 25 years old male, without any comorbidities, tested positive for covid rapid antigen test on 13/4/21

He was in home isolation for 7 days on supportive medication.

Later which on 8th day he presented to emergency with chief complaints of increased intensity of cough, persisting fever.

On evaluation – patient was conscious, coherent and well oriented

Bp- 130/80 mm hg, heart rate-104 bpm

Respiratory rate - 18 /min, spo2 % - 95% at room air

Temperature: 102.8° f

Auscultation - bilateral basal crepitations present

Patient was treated with iv broad spectrum antibiotics [betalactams and aminoglycosides].

Low dose iv steroids [methylprednisolone 40mg iv od].

Anticoagulation [low molecular weight heparin 40mg od]

Antiviral drugs [initially favipiravir was taken by him in home isolation, later it was escalated to remdesivir 200mg iv over 20-30 min followed by 100mg iv from day 2-5 on hospitalisation].

Antipyretic medications were given as and when required.

Supportive medication in the form of zinc, vitamin c, b-complex etc, also was given.

Investigations:

His routine blood investigations, suggested **leucocytosis with selective lymphopenia**, rest all were within normal limits.

Echocardiography, ecg and cardiac enzymes were normal.

Inflammatory markers

Inflammatory Markers	25/4/21	7/5/21	15/5/21	25/5/21 [post anticoagulation on therapy]
D-DIMER	580	3327.33	10,233	2200
CRP	7.2	189	284	102
INTERLEUKIN-6	12.5	37.4	42.33	11.6
Serum Ferritin	448	998	1127	557

Radiological features

HRCT thorax

Multiple diffusely scattered ggo, with mosaic attenuation and linear opacities in peripheral and central zones of both lungs, involving predominantly lower lobes. **corads 6**, ct severity 8/25 [mild].

The patient improved symptomatically and hence treatment plan was altered accordingly in the form of descalation of iv antibiotics, conversion of injectables to orals, and later discharged with tapering dose of steroids and was adviced to continue antiplatelets, aspirin and statins on 30/4/21.

After discharge pateint was on regular follow up during which his d dimer values were continuously escalating

After two weeks of discharge he presented to emergency department with complaints of hemoptysis, sudden onset of chestpain and shortness of breath on 15/5/21.

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On evaluation – patient was conscious, coherent and well Oriented

Bp- 160/100 mm hg, heart rate-152 bpm

Respiratory rate - 38 /min, spo2 % - 88% at room air

Temperature: afebrile.

Auscultation - bilateral basal crepitations present.

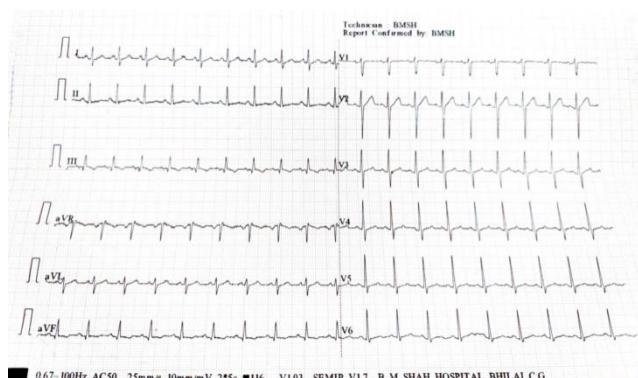
He was treated with external oxygen support, iv antibiotics, anticoagulation [low molecular weight heparin 40mg od], and antihypertensives.

Investigations:

His routine blood investigations were within normal limits.

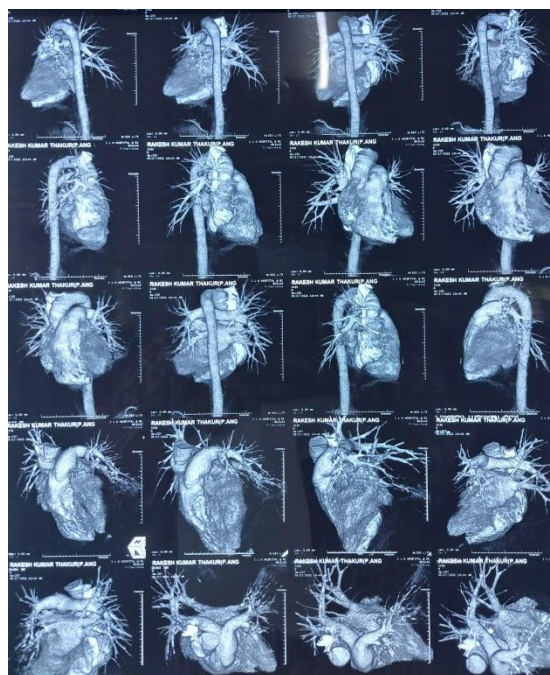
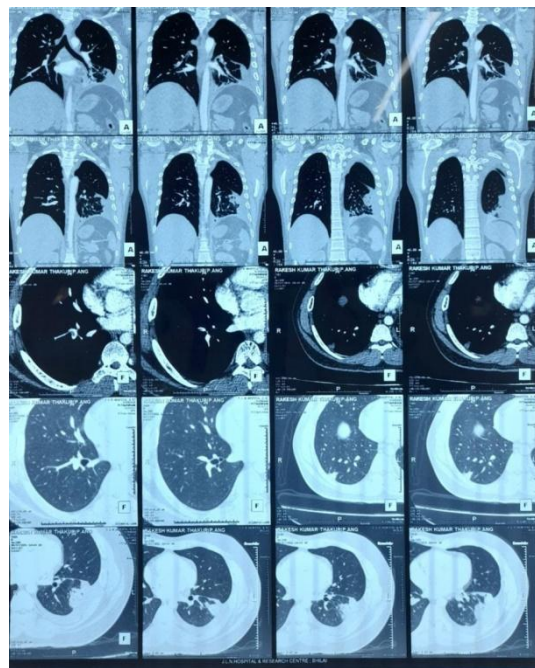
His inflammatory markers were drastically elevated.

His electrocardiography was suggestive of sinus tachycardia and s1q3t3 pattern.



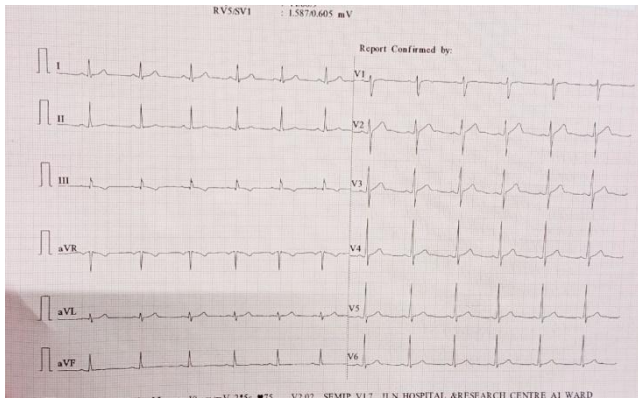
Hence he was advised for ct pulmonary angiography to ruleout anythromboembolic plaques.

CTPA [17/5/21]: thrombotic pulmonary embolism seen in pulmonary artery branches supplying right anterior basal right posterior basal, right lateral basal left lateral basal and left posterior basal segments with thrombus in left interlobular artery. **Right heart strain** and **pulmonary artery hypertension** is observed. **pulmonary infarct** in superior segment of left lower lobe and posterior basal segment of right lower lobe.



He was improved symptomatically hence he was shifted to ward and was later discharged on oral anticoagulants [apixaban 2.5mg 1 tab twice daily], and oral antihypertensives.

His follow up **ECG [4/6/21]** after 20 days of anticoagulation



2D ECHO findings [5/6/21 after 20 days of anticoagulation]

No mitral and tricuspid valve regurgitation, no pulmonary artery hypertension, left ventricular ejection fraction 60%, no rwm.

2. Discussion

Several reports have been published on the cardiovascular implications of COVID 19, among which venous thromboembolism is most common implication. **Pathophysiology of COVID-19 associated venous thromboembolism:**

- SARS-CoV-2 downregulates ace-2 expression and subsequent protective signalling pathways in endothelial cells of blood vessels.
- Diffuse endothelial injury and endothelitis from direct invasion of sars-cov-2 or due to aggravated host inflammatory response.
- Severe inflammation, hypoxia, endothelial dysfunction, platelet activation and stasis [virchow's triad], particularly in intensive care patients, lead to prothrombotic state.
- Inflammation and stress resulted pulmonary plaque rupture leading to lung infarction.

At readmission majority of ground glass opacities seen in prior imaging were resolved.

The main intention of this case report is to modify the guidelines regarding thromboprophylaxis of patients in covid-19, in which usually thrombolysis is recommended in persistent haemodynamic compromise and refractory hypoxia, and also to highlight potential readmission of patient after active infection.

Patients affected with covid-19 demonstrate abnormal activation of clotting cascade. Markers like d-dimer elevation is associated with deleterious patient trajectory and increased incidence of mortality.

Implementing extended courses of thromboprophylaxis in patients with covid-19 with other risks factors for VTE for example, reduced mobility, pre-existing comorbidity or malignancy, d-dimer levels, greater than two times the upper limit of normal at point of discharge, have also been

suggested as a guide for the initiation of prolonged anticoagulation treatment.

3. Conclusion

- Despite being eclipsed by respiratory failure due to ards in covid 19 outbreak, acute pulmonary embolism are still major cause of morbidity and mortality worldwide.
- **Thromboprophylaxis** with anticoagulants seems to play the major role in treatment of covid-19 patients even in patients **without any comorbidities**.
- Patients with high inflammatory markers like elevated serum ferritin and serum LDH, increased il-6 levels are more prone for occlusion of vessels due to hypercoagulation and stasis, thereby resulting venous thromboembolism.
- Early detection of possible thromboembolism with serial monitoring of d-dimer values prevent possibility of subsequent thromboembolism and infarction.
- **Reduced ambulation** due to home isolation may also precipitate possible development of deep vein thrombosis and pulmonary embolism.
- Several risk factors were identified, including the presence of a d-dimer level twice the upper limit of normal at the time of potential discharge.
- The role and duration of oral anticoagulant has to be studied in mild asymptomatic cases to prevent potential risk of **vte**.

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