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Internal Medicine

ASYMPTOMATIC PULMONARY THROMBOEMBOLISM IN A PATIENT WITH POST-COVID INFECTION

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ABSTRACT Very minimal data is available regarding the clinical sequelae of COVID-19 and the prevalence of thromboembolic disease in asymptomatic patients. Surely, there is increased prevalence of thromboembolic disease and pulmonary embolism (PE) in critically ill patients with COVID-19. Hence the administration of thromboprophylaxis to all the hospitalized patients with COVID-19 was suggested now. However, the administration of regular thromboprophylaxis in asymptomatic outpatients is an entirely different matter. Through this case report, we present the case of insidious PE development in an otherwise asymptomatic patient without any predisposing factors.

KEYWORDS :

INTRODUCTION:

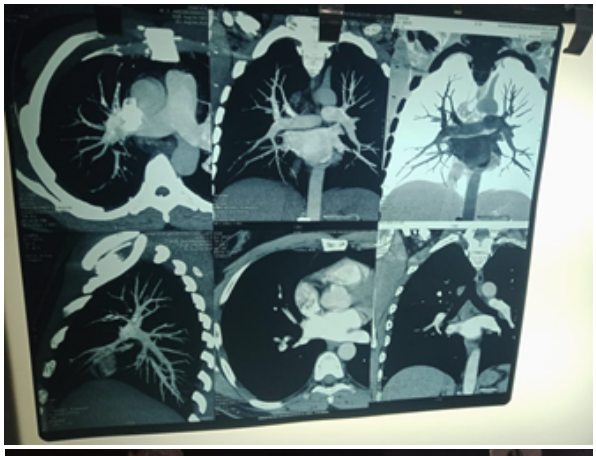
The novel corona virus-induced severe acute respiratory syndrome (COVID-19) outbreak was first reported in December 2019. Three months later, the Director-General of the World Health Organization, declared the COVID-19 a global pandemic. Accumulated research and evidence reveals that a thrombotic coagulation disorder is often seen in COVID-19, and the incidence is higher in severe cases.

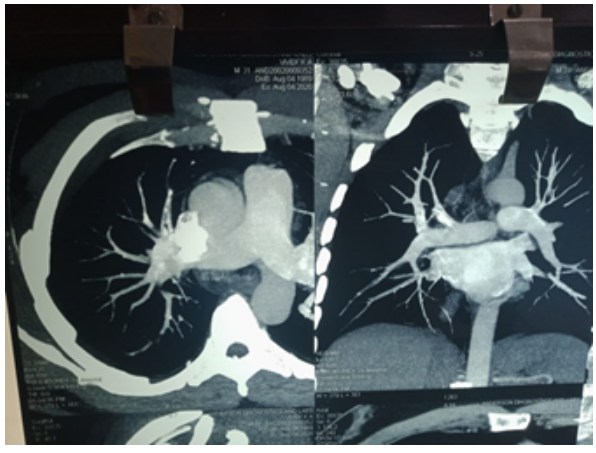
Case Report:

A previously healthy 31 year old gentleman, who is a doctor by profession, came to Apollo Main Hospital, Chennai with report of elevated D-dimer levels. He was asymptomatic at the time of presentation. On eliciting history, he had intermittent fever spikes for 2 -3 days associated with myalgia and generalized fatigue before 2 weeks. He didn't have cough, sore throat, breathlessness or chest pain. He was tested thrice for COVID-19 through RT-PCR technique immediately after onset of symptoms but all the three samples collected were negative. His Complete Blood Counts, CRP, ferritin, LDH and D-dimer levels were done 2 weeks back and they were in normal range. His fever settled down after 3 days and he was absolutely normal after that episode except that he had persistent fatigue. After 14 days, he experienced mild chest discomfort and dyspnoea on exertion which relieved on taking rest. CRP, ferritin and D-dimer levels were repeated which showed mildly elevated CRP (22mg/L), normal ferritin levels and elevated D-dimer (4.8).

On examination, he was tachycardic with 112 bpm, normotensive, respiratory rate was normal and saturation was 98% in room air. Echocardiogram showed grade 1 LV diastolic dysfunction, normal ejection fraction, no RWMA, Right atrium and ventricles were normal in size and function and no evidence of PAH and PE. Contrast enhanced CT chest with PE protocol was done in view of elevated levels of D-dimer and it showed segmental and sub-segmental right pulmonary artery thromboembolism. Bilateral lower limb venous Doppler was done and it was normal without any signs of DVT. He was tested for antibody to SARS-CoV-2 and it was positive.

His PT/INR and APTT were in normal range. Lupus anticoagulant, anti-cardiolipin antibody, IgM and IgG anti-phosphatidyl serine antibodies, IgM and IgG anti beta-2 glycoprotein antibodies were negative. He was evaluated for prothrombin gene and factor V Leiden gene mutation but both mutations were negative. He was started on therapeutic doses of enoxaparin for PTE and was changed to rivaroxaban 15mg twice daily during discharge





DISCUSSION:

After comprehensive evaluation and ruling out all the other causes of pulmonary thromboembolism, as antibody to SARS-CoV-2 was positive, we considered this segmental PTE as sequelae of post-COVID infection. The clinical sequelae of COVID-19 and the prevalence of thromboembolic disease in asymptomatic patients remain partially studied. Our patient was asymptomatic on presentation and had no predisposing factors for thromboembolic disease. He presented with elevated CRP and increased levels of D-

dimer. SARS-CoV-2 can bind via its spike protein to the angiotensin-converting enzyme 2 receptor resulting in endothelial inflammation. Evaluation of the data available till now suggests that the thrombotic events with COVID-19 are a result of the endothelial inflammatory response to SARS-CoV-2 infection. As per the available data, coagulopathy in COVID-19 manifests with significant elevation of D-dimer levels, elevated fibrinogen levels and minimal changes in PT, APTT, and platelet counts. This case report describes the insidious onset of PE in asymptomatic COVID-19 patients who did not have any other risk factors for thromboembolic disease.

CONCLUSION:

Asymptomatic COVID-19 patients may exhibit a variety of underlying pathophysiology, which could promote the insidious onset of thromboembolic phenomena related to their COVID-19 status. Future large prospective studies are clearly required to confirm the present findings.

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