Pulmonary Embolism in COVID-19: A Case Report and Narrative Review of the Literature

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ABSTRACT

Coronavirus Disease 2019 (COVID-19), caused by SARS-CoV-2, is associated with a prothrombotic state characterized by endothelial dysfunction, hypercoagulability, and systemic inflammation. Pulmonary embolism (PE) represents a serious and potentially fatal thromboembolic complication in COVID-19 patients, with reported incidences of 25–27% in hospitalized and intensive care unit populations. We report a case of a patient presenting to the emergency department with dyspnea and chest pain, in whom COVID-19 and concomitant massive pulmonary embolism were diagnosed, necessitating thrombolytic therapy. This case is contextualized within a narrative review of the literature on the pathophysiology of COVID-19-associated coagulopathy, the incidence and risk factors for PE in COVID-19, current diagnostic approaches, and evidence-based antithrombotic and thrombolytic strategies. Early recognition of thromboembolic risk and timely intervention are critical to reducing mortality in this patient population.

Keywords: COVID-19; SARS-CoV-2; pulmonary embolism; coagulopathy; thrombolysis; venous thromboembolism

INTRODUCTION

Coronavirus Disease 2019 (COVID-19), caused by the Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), emerged in Wuhan, China in December 2019 and rapidly evolved into a global pandemic, infecting over 219 million individuals and resulting in more than 4.5 million deaths within the first two years. Beyond its primary manifestation as a respiratory illness, COVID-19 is increasingly recognized as a systemic disease with profound effects on the coagulation system, vascular endothelium, and immune response [1].

Thromboembolic complications represent a major source of morbidity and mortality in COVID-19. Venous thromboembolism (VTE), including deep vein thrombosis (DVT) and pulmonary embolism (PE), occurs at substantially higher rates than in non-COVID-19 patients,

even in those receiving standard pharmacological thromboprophylaxis. The incidence of VTE in COVID-19 patients has been reported between 25% and 27% in some cohorts [2,3]. Pulmonary embolism, in particular, carries significant risk of hemodynamic compromise and respiratory failure, compounding the already severe respiratory pathology of COVID-19 pneumonia.

We present a case of a COVID-19 PCR-positive patient who presented to the emergency department with dyspnea and chest pain, in whom imaging confirmed pulmonary embolism requiring thrombolytic therapy. This case is accompanied by a narrative review of the current literature on COVID-19-associated coagulopathy, PE incidence, risk stratification, and treatment.

PATHOPHYSIOLOGY OF COVID-19-ASSOCIATED COAGULOPATHY

The coagulopathy of COVID-19 is mechanistically distinct from classic disseminated intravascular coagulation (DIC) and is best characterized as a hypercoagulable, prothrombotic state. SARS-CoV-2 enters host cells via the angiotensin-converting enzyme 2 (ACE2) receptor, which is expressed on pulmonary alveolar cells as well as vascular endothelial cells. Direct endothelial infection and the ensuing inflammatory response result in endothelial activation, denudation, and dysfunction—a central driver of COVID-19-associated thrombosis [4].

Laboratory hallmarks of COVID-19 coagulopathy include markedly elevated D-dimer levels, elevated fibrinogen, prolonged prothrombin time, and thrombocytopenia in severe cases. Elevated D-dimer has emerged as one of the strongest predictors of in-hospital mortality and disease severity in COVID-19 [5]. In contrast to sepsis-associated DIC, platelet counts and fibrinogen are often preserved or even elevated in early COVID-19 coagulopathy, reflecting an acute phase reaction rather than consumptive coagulopathy.

Additional mechanisms contributing to thrombosis include complement system activation, neutrophil extracellular trap (NET) formation, hypoxia-induced vasoconstriction, and prolonged immobilization. The combination of endothelial injury, stasis, and hypercoagulability—Virchow's triad—is thus comprehensively fulfilled in severe COVID-19, creating a high-risk environment for both venous and arterial thrombotic events [4,5].

CASE REPORT

A patient presented to our emergency department with complaints of progressive dyspnea and pleuritic chest pain. A nasopharyngeal swab was positive for SARS-CoV-2 by RT-PCR. On

examination, the patient was tachycardic and hypoxic. Chest computed tomography pulmonary angiography (CTPA) confirmed bilateral pulmonary emboli with evidence of right heart strain. Electrocardiography demonstrated the S1Q3T3 pattern. Echocardiography showed right ventricular dilatation and interventricular septal flattening consistent with acute cor pulmonale.

Given the hemodynamic compromise and imaging findings consistent with high-risk (massive) PE, systemic thrombolytic therapy was initiated. The patient received intravenous alteplase according to standard dosing protocols. Clinical and hemodynamic improvement was observed within hours of thrombolysis. The patient was subsequently anticoagulated and monitored in the intensive care unit before being transferred to the ward. Recovery was uneventful, and the patient was discharged on therapeutic anticoagulation.

INCIDENCE AND RISK FACTORS FOR PE IN COVID-19

Multiple observational studies have documented a substantially elevated incidence of PE in hospitalized COVID-19 patients. Llitjos et al. reported a VTE incidence of 69% in anticoagulated COVID-19 ICU patients, with PE occurring in 56% [2]. Lodigiani et al., in a large Italian cohort, reported an overall VTE rate of 7.7%, rising to 27.6% in ICU-admitted patients [3]. The true incidence may be even higher given the autopsy-based evidence of microvascular thrombosis not captured by conventional imaging.

Risk factors for COVID-19-associated PE include disease severity (ICU admission, mechanical ventilation), obesity, elevated D-dimer (>1 μg/mL on admission), elevated C-reactive protein, pre-existing hypercoagulable states, and prolonged immobilization. Notably, PE in COVID-19 patients may occur despite standard prophylactic anticoagulation, suggesting that conventional thromboprophylaxis doses may be insufficient in high-risk patients [5].

DIAGNOSIS

The clinical diagnosis of PE in COVID-19 is challenging, as symptoms of PE—dyspnea, hypoxemia, tachycardia, and chest pain—overlap substantially with those of COVID-19 pneumonia itself. A high index of suspicion is therefore required, particularly in patients with disproportionate hypoxemia, unexplained hemodynamic instability, or sudden clinical deterioration [1].

D-dimer, while highly sensitive for PE, has limited specificity in COVID-19 due to universally elevated levels in active infection. Clinical prediction scores such as the Wells score and the Geneva score retain utility in pre-test probability stratification, though their calibration in

COVID-19 populations has not been fully validated. CTPA remains the gold standard for PE diagnosis when patient stability and contrast tolerance permit. Bedside echocardiography and lower limb venous compression ultrasonography are valuable adjuncts in critically ill patients who cannot undergo CTPA [2,3].

TREATMENT

Anticoagulation is the cornerstone of VTE treatment in COVID-19. Low molecular weight heparin (LMWH) is preferred due to its predictable pharmacokinetics, subcutaneous administration, and absence of the drug interactions associated with direct oral anticoagulants in patients on antiviral regimens. For patients with confirmed PE and hemodynamic stability, therapeutic anticoagulation is initiated immediately and continued for at least 3–6 months [5].

For high-risk (massive) PE with hemodynamic compromise, systemic thrombolysis with alteplase represents the treatment of choice when no absolute contraindications exist. Catheter-directed thrombolysis is an alternative in centres with appropriate expertise, particularly in intermediate-high-risk patients or those with relative contraindications to systemic thrombolysis. Surgical embolectomy remains an option in refractory cases [2].

The optimal anticoagulation strategy in COVID-19 patients without confirmed VTE—prophylactic, intermediate, or therapeutic—has been the subject of several randomized trials. Current evidence, including the ATTACC, ACTIV-4a, and REMAP-CAP platform trials, suggests that therapeutic anticoagulation may reduce the need for organ support in moderately ill hospitalized patients, but does not provide benefit and may increase bleeding risk in critically ill ICU patients [5]. An individualized approach guided by D-dimer levels, clinical severity, and bleeding risk is therefore recommended.

DISCUSSION

This case illustrates the convergence of COVID-19 infection and acute PE—a combination that poses substantial diagnostic and therapeutic challenges. The clinical presentation of dyspnea and chest pain in a COVID-19-positive patient is common and nonspecific; however, the severity of hemodynamic compromise and imaging findings mandated urgent thrombolytic intervention.

The case highlights the importance of maintaining a high clinical suspicion for PE in COVID-19 patients, even in those receiving thromboprophylaxis. Routine monitoring of D-dimer and early use of CTPA or bedside echocardiography in deteriorating COVID-19 patients are

warranted. Furthermore, the selection of appropriate anticoagulation strategies—balancing thrombotic and hemorrhagic risk—is a critical clinical decision that must be individualized.

From an anesthetic and critical care perspective, management of COVID-19-associated PE in the ICU setting requires integrated expertise in respiratory support, hemodynamic monitoring, anticoagulation management, and timely recognition of contraindications to thrombolysis. Multidisciplinary collaboration between emergency physicians, intensivists, pulmonologists, and hematologists is essential for optimal outcomes.

CONCLUSION

COVID-19 is associated with a prothrombotic state that substantially increases the risk of pulmonary embolism. Clinicians must maintain a high index of suspicion for PE in COVID-19 patients presenting with hemodynamic instability or disproportionate hypoxemia. CTPA remains the diagnostic gold standard, and systemic thrombolysis is indicated for hemodynamically significant PE. Individualized anticoagulation strategies guided by clinical severity and D-dimer levels are recommended. Early recognition and prompt treatment are critical to reducing COVID-19-associated PE mortality.

AUTHOR CONTRIBUTIONS

Concept and design: H.K. Data collection: H.K. Data analysis and interpretation: H.K. Manuscript writing: H.K. Critical revision: H.K. Final approval: H.K.

CONFLICT OF INTEREST

The author declares no conflict of interest.

ETHICS STATEMENT

Written informed consent was obtained from the patient for publication of this case report.

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