1. Introduction

Coronavirus disease-2019 (COVID-19) caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is associated with a hypercoagulable state leading to increased incidence of thromboembolism. However, it is exceedingly rare to see presence of both arterial and venous thromboembolism simultaneously. Herein, we report an unusual presentation of a 39-year-old male with recently diagnosed COVID-19 who initially had acute myocardial infarction secondary to thrombotic occlusion of right coronary artery followed by acute pulmonary embolism. Health care providers should be aware of this uncommon yet possible co-existence of two life-threatening manifestations in order to prevent fatal consequences.

Patient returned after 5 days with acute onset left-sided, pressure like chest pain. EKG showed ST changes consistent with early repolarization in anterior leads and diffuse T wave depression in multiple leads. (Figure 1) Further workup showed the patient had maximum elevated troponin of 34 ng/L. D-Dimer was mildly elevated at 1256 ng/mL. Other laboratory investigations were unrewardable. Patient underwent coronary computed tomography angiography (CTA) for evaluation of his coronaries which showed a filling defect at the right coronary sinus at the level of the right coronary artery ostium suggesting thrombus in occluding the proximal right coronary artery (RCA). (Figure 2) Subsequent left heart catheterization revealed RCA had subtotal occlusion with filling defect at proximal segment consistent with acute thrombus. (Figure 3) The lesion was treated with balloon angioplasty and stenting with excellent results. Patient did not have additional risk factor for coronary artery disease including smoking, family history, intravenous drug abuse, or HIV. Lab work revealed low-density lipoprotein of 73 mg/dl and Hemoglobin A1c of 5.3. Patient was discharged on dual-antiplateletas, high-intensity statin and beta-blockers. Patient, however, returned 2 days later complaining of pelvic pain and shortness of breath. CTA of lungs showed moderate-sized filling defects in the left lower lobe consistent with pulmonary embolism. (Figure 4) D-dimer in this admission was 6,880 ng/mL, which was markedly elevated compared to his previous admission. He was started on heparin drip and was eventually discharged home on...
ticagrelor and apixaban. Clinical follow-up after 1 week showed improvement in his symptoms.

3. Discussion

The COVID-19 pandemic has been a challenge around the world, in part attributed to the fast nature of the transmission and high number of cases. COVID-19 has affected over 30 million patients and caused over 900 thousand deaths to date. COVID-19 mainly presents as a respiratory illness, but studies have shown a significant association of thrombotic events and the predisposition to worsen underlying cardiovascular disease (CVD). Furthermore, patients with comorbidities have higher mortality and the presence of CVD is an imminent threat.

To our best of knowledge, there is only one reported case of COVID-19 with simultaneous acute myocardial infarction (MI) and acute pulmonary embolism (PE) [1]. However, in that case the patient had significant comorbidities that could predispose to MI or PE. In contrast, our
COVID-19, seen in up to one-third of patients in the intensive care unit, even when prophylactic anticoagulation is used [6]. Studies have noted the preponderance of males with a high prevalence of obesity and other chronic medical comorbidities, especially cardiovascular disease, hypertension, and diabetes mellitus [7].

Empiric full-dose anticoagulation for individuals with COVID−19 who do not have VTE remains controversial, since data demonstrating improved outcomes are lacking, and some of the risk factors for VTE are also risk factors for increased risk of bleeding [8,9]. However, there is consensus that all hospitalized patients with COVID−19 should receive pharmacologic thromboprophylaxis with low molecular weight heparin (LMWH) or fondaparinux unless bleeding risk and full therapeutic-intensity anticoagulation in the appropriate clinical scenario, including documented or strongly suspected VTE and clotting of vascular access devices [10,11]. Appropriate testing to document suspected thromboembolism is also advised if feasible.

However, it is exceedingly rare to see both arterial and venous thromboembolic manifestations in that same patient in the same clinical setting. Treatment in such scenarios requires multidisciplinary expertise to address the multifaceted clinical manifestations of this viral disease. Our case also highlights that in patients with COVID−19, even without predisposing risk factors for thromboembolism can have higher risk of MI and VTE. Since both, the conditions are acute life threatening if not treated emergently once must be extra vigilant in identifying thromboembolic events associated with this globally pandemic disease.

4. Conclusion

Our case and review of literature reveals that health care providers should be aware of this uncommon yet possible co-existence of two life-threatening manifestations: MI and PE in a patient with underlying COVID 19. Appropriate measures need to be taken in a timely manner in these situations in order to prevent fatal consequences.

Disclosure statement
No potential conflict of interest.

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Figure 4. CTA of lungs showed moderate-sized filling defects in the left lower lobe consistent with pulmonary embolism (asterisk *).
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