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# A Rare Complication Following SARS-Cov-2 Infection: ST-Elevation Myocardial Infarction and Bilateral Pulmonary Embolism.

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Coronavirus disease-2019; acute pulmonary embolism; acute myocardial infarction; thromboembolism.

## 1. Abstract

Sars CoV-2 infection has been associated with a hypercoagulability leading to increased incidence of thromboembolism. However, it is exceedingly rare to see presence of both venous and arterial thromboembolism simultaneously. Herein, we report an unusual presentation of a 69-year-old male with COVID -19 who initially had acute inferior myocardial infarction secondary to thrombotic occlusion of right coronary artery followed by bilateral pulmonary embolism. Healthcare providers need to be aware of this unusual but potential coexistence of two life-threatening events in order to avoid fatal consequences.

## 2. Clinical Presentation

A 69-year-old male with a history of arterial hypertension presented to the emergency department reporting a 4-day history of severe fatigue, dyspnoea and symptoms of upper respiratory tract infection. The patient's nasopharyngeal swab tested positive for 2019-nCoV by real-time reverse-transcriptase–polymerase-chain-reaction assay. At presentation, physical examination revealed severe respiratory distress, with respiratory rate of 36/min, 74% oxygen saturation on pulse oximetry and arterial blood pressure of 90/60 mmHg. The patient was supported with oxygen via non-rebreather mask. Blood gas analysis subsequently showed pH 7.47, pCO2 33.6mmHg, pO2 41.5mmHg, HCO3 25.4mmol/L. Due to persisting refractory hypoxemia despite oxygen escalation therapy, the patient was intubated. A standard approach of lung protective ventilation was used with low tidal volume of 480 ml, positive end-expiratory pressure of 10 cmH2O, and FiO2 100%.

After the patient's intubation, the electrocardiogram (ECG) revealed sinus tachycardia with ST-elevation (STE) of 2-3mm in leads II, III, aVF and 1mm in lead V4R, reciprocal ST depression in leads I and aVL and also in the precordial leads (Figure 1).

An abnormal D-Dimer finding (>10 μg/ml) prompted computed tomography angiography to be performed to investigate for acute aortic syndrome or pulmonary embolism. This excluded acute aortic syndrome but revealed extensive ground glass opacities in both lungs. Filling defects were found in the lobar branches of the right and left upper lobes, and in multiple segmental and subsegmental branches of both lungs (Figure 2 a,b).

Transthoracic echocardiography revealed normal left ventricular (LV) dimensions with a mildly increased wall thickness (interventricular septum 12mm, posterior wall 11mm), impaired LV systolic function with akinesia of the basal inferoseptal, mid-inferoseptal

segments, and hypokinesia of the basal inferior and mid inferior segments, with an estimated LV ejection fraction of 40%. Mild mitral and tricuspid regurgitation were also observed. The left ventricular diastolic function was mildly impaired (E<A) and E/e' ratio was less than 8 with no signs of elevated filling pressures of the LV. The left atrium was dilated with a diameter of 43mm and calculated volume of 55ml. The diameter of the right ventricle was 46mm with normal systolic function (SRx 11-12 cm/sec). In the subcostal view, flattening of the interventricular septum was observed. The diameter of the inferior vena cava was 25mm without inspiratory collapse (Figure 3).

Due to the diagnosis of inferior STEMI the patient was urgently admitted to the catheterization laboratory for primary percutaneous coronary intervention (Figure 4).

Coronary angiography revealed a mild stenosis [50%] of the proximal left anterior descending (LAD) coronary artery, a severe stenosis [80%] of the LAD distal to the point of origin of the first diagonal branch, which had a severe proximal stenosis [70%] and proximal stenosis of 50-70% of the first and second obtuse marginal arteries. There was acute total occlusion of the right coronary artery (RCA II) [TIMI 0 – Rentrop I-II (through a reticulated network from LCA)].

After cannulating the RCA via a trans-femoral approach using a Judkins Right 4 6Fr guide catheter, the lesion was crossed using a ChoICE polymer-tip (PT) guidewire (Boston Scientific Corporation, MN). Shortly after balloon dilation of the mid RCA, TIMI 3 antegrade flow was recovered. 4.0mm × 38mm and 4. mm × 1 mm Promus PremierTM drug eluting stents (Boston Scientific Corporation, MN) were then deployed to the mid-segment of the RCA achieving a good angiographic result and maintaining TIMI 3 antegrade flow.

The initial treatment of the patient included iv Propofol 25cc/h, iv Remifentanil 10cc/h, iv Cisatracurium 10cc/h, iv Hydrocortisone 10cc/h, iv Furosemide 10cc/h, iv Esomeprazole 40mg bd, tb qds, sc Enoxaparine 60mg bd, tb Atorvastatin 40mg qds.

Antimicrobial therapy primarily included iv Colistin 4.500.000 bd, iv Sultamicillin 4gr q4h, iv Fosfomycin 4gr q4h and was modified according to antibiotics sensitivities on the 13th day of hospitalisation due to blood infection with Klebsiella pneumoniae.

During the first days of his hospitalization, the patient remained hypotensive and required inotropic support, maintaining mean blood pressure of about 80mmHg. Ionotropic demand increased further during the next 72 hours. On the 2nd day, due to acute kidney injury the patient required continuous renal replacement therapy with veno-venous hemodiafiltration. After 31 days in total of hospitalisation the patient died due to multi-organ failure.

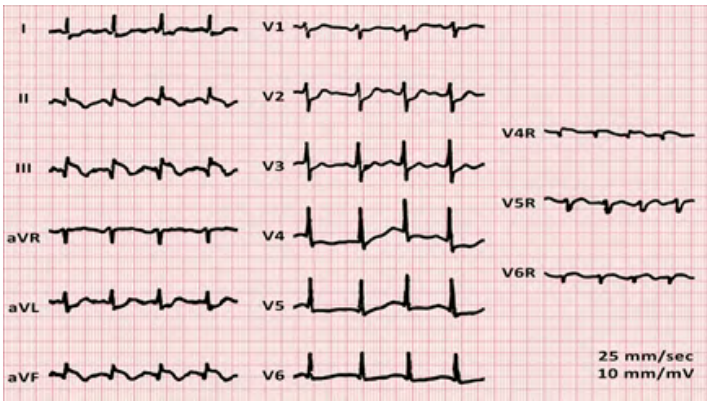


**Figure 1:** ECG of the patient after admission.

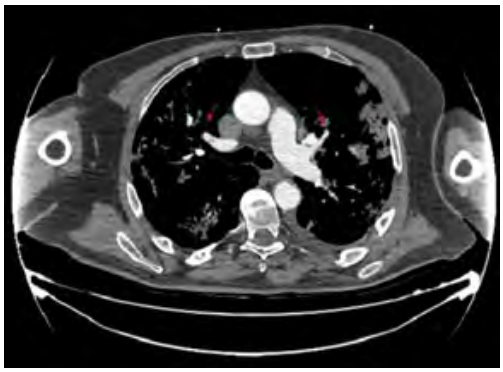

**Figure 2a:** Filling defects in the lobar branches of the right upper and left upper lobes.

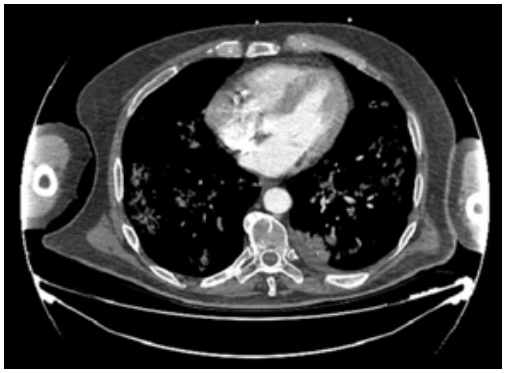

**Figure 2b:** Filling defects in multiple segmental and subsegmental branches of the right and left lung.

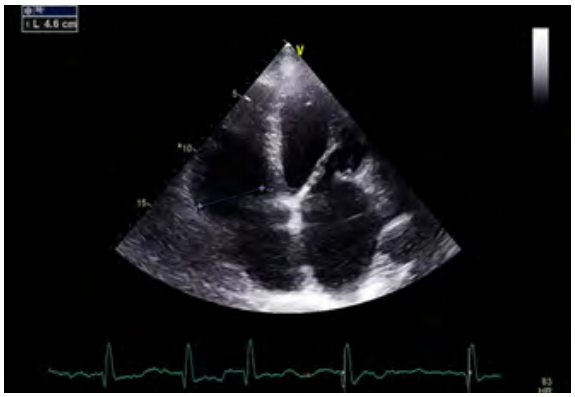

**Figure 3:** Transthoracic echocardiogram on admission

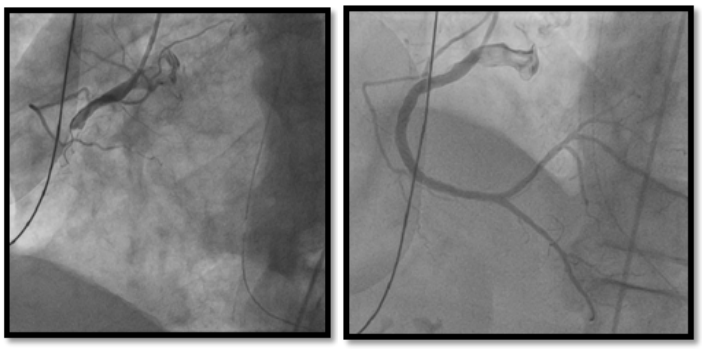

**Figure 4:**

## 3. Discussion

Sars-CoV-2 infection has been associated with serious cardiac manifestations including acute myocardial infraction, pulmonary embolism, myocarditis, heart failure, and Takotsubo myocardiopathy [1] in as many as 20.6-25% of patients [2,3]. One possible mechanism driving these poor outcomes is hypercoagulability, with microvascular or macrovascular thrombi affecting multiple organ systems [6,2,3].

Cases reported in the literature describe different aspects of the vascular manifestations of COVID-19.6 However, all the existing studies so far are limited in size, have not reported all thrombotic events, and were focused on patients with severe disease hospitalized in intensive care units (ICUs).

Studies from both China and the USA suggest markers such as D-dimer may be associated with increased mortality in hospitalized patients with COVID-19, with follow-up studies showing complications related to thrombosis in the lungs and brain as well as clotting of renal replacement and extracorporeal membrane oxygenation circuits [2-4]. COVID-19 has significantly disrupted the management of acute cardiovascular disease. Data from China has shown that an elevation in troponin, with or without previous underlying cardiovascular conditions, was associated with increased mortality [5, 6]. Primary cardiac manifestations of COVID-19 were also examined in an Italian study in which 85% of patients presenting with STEMI were eventually found to be COVID-19 positive. Interestingly, up to 40% of patients had no culprit lesion identified on the angiogram [7].

Recently, a case series of STEMI patients with COVID-19 from New York City reported by Bilaloglu et al. revealed that all of these patients also presented with an elevated D-dimer, with 27% requiring percutaneous coronary intervention [8]. This data suggests that the increased thrombotic risk of COVID-19 could manifest as acute coronary thrombosis and STEMI. Management of these patients initially presented logistical challenges with respect to prompt intervention, although this has improved as protocols and procedures have evolved [9]. For this reason, when assessing a COVID-19 infected patient with ST segment elevation, clinicians should be aware of the possibility of PE, AMI and the association between them [5].

Ackermann et al. first reported the presence of pulmonary intussusceptive angiogenesis and other pulmonary vascular features in the lungs of seven patients who died from COVID-19 [7]. Prior studies varied regarding the precise incidence of thrombosis; however, all suggested an elevated thrombosis risk in patients with COVID-19 [3, 9]. This analysis found a variation by clinical setting and type of thrombosis event. While thrombosis is also observed in other acute infections10 ( 5.9% prevalence during the 2009 influenza pandemic) [11]. the thrombotic risk appears higher in COVID-19 cases. Various mechanisms are implicated in COVID-19 induced thrombosis, including vascular and systemic inflammation caused by the SARS-CoV-2-mediated cytokine storm, antiphospholipid antibody syndrome, macrophage activation syndrome, the complement cascade, and RAS dysregulation [4].

In our case, the patient presented with acute STEMI during his hospitalization in the ICU. The patient received dual antiplatelet therapy, high-dose statin, heparin infusion and prompt percutaneous coronary intervention, as per clinical guidelines for management of acute coronary syndrome. Postintervention management remains an active area of clinical research, as the potential interactions of antiviral and immunomodulating medications used to treat systemic COVID-19 may interfere with common antiplatelet therapies and anticoagulation [10].

To the best of our knowledge, we are the first to report a case of a critically-ill COVID-19 patient with bilateral pulmonary embolism in combination with myocardial infraction, requiring both primary angioplasty and anticoagulation therapy.

As there is still debate over the most effective anti-coagulant therapy for COVID-19 patients, the need to develop an algorithm to determine the optimal antithrombotic therapy for these patients is crucial. Although the administration of prophylactic doses of low molecular weight heparin has been recommended by the International Society on Thrombosis and Haemostasis (ISTH) and the American Society of Hematology (ASH), the most effective dosage remains undefined. Without doubt the treatment of COVID-19 requires multidisciplinary expertise to address its multifaceted clinical manifestations. Moreover, attention must be paid to the interactions of antiviral and other pharmaceutical agents including oral and intravenous anticoagulants, with an aim to minimize the risk of bleeding and thrombo-embolic complications.

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