AnnalsofClinicalandMedical Case Reports

CaseReport ISSN2639-8109Volume10

ARareComplicationFollowingSARS-Cov-2Infection:ST-ElevationMyocardial Infarction and Bilateral Pulmonary Embolism.

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Received: 19 Nov 2022
Accepted: 26 Dec 2022
Published: 03 Jan 2023
JShort Name:ACMCR

Citation:

Saplaouras A, A Rare Complication Following SARS-Cov-2 Infection: ST-Elevation Myocardial Infarction andBilateralPulmonaryEmbolism..AnnClinMedCase Rep. 2022; V10(10): 1-4

Keywords:

Coronavirusdisease-2019;acutepulmonaryembolism;acutemyocardialinfarction;thromboembolism.

1. Abstract

SarsCoV-2infectionhasbeenassociatedwithahypercoagulabilityleadingtoincreasedincidenceofthromboembolism.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 occlusionofrightcoronaryarteryfollowedbybilateralpulmonary embolism. Healthcare providers need to be aware of this unusual butpotentialcoexistenceoftwolife-threateningeventsinorderto avoid fatal consequences.

2. Clinical Presentation

A69-year-oldmalewithahistoryofarterialhypertensionpresentedtotheemergencydepartmentreportinga4-dayhistoryofsevere 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.Thepatientwassupportedwithoxygenvianon-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.Astandard 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 leadsII, III, aVFand 1mm in leadV4R, reciprocalSTdepression 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 aorticsyndromeorpulmonaryembolism.Thisexcludedacuteaorticsyndromebutrevealedextensivegroundglassopacitiesinboth lungs.Fillingdefectswerefoundinthelobarbranchesoftheright 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)dimensionswithamildlyincreasedwallthickness(interventricularseptum12mm,posteriorwall11mm),impairedLVsystolic functionwithakinesiaofthebasalinferoseptal,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 a 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 46Fr 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×1mm 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 4grq4h, iv Fosfomycin 4grq4h 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.

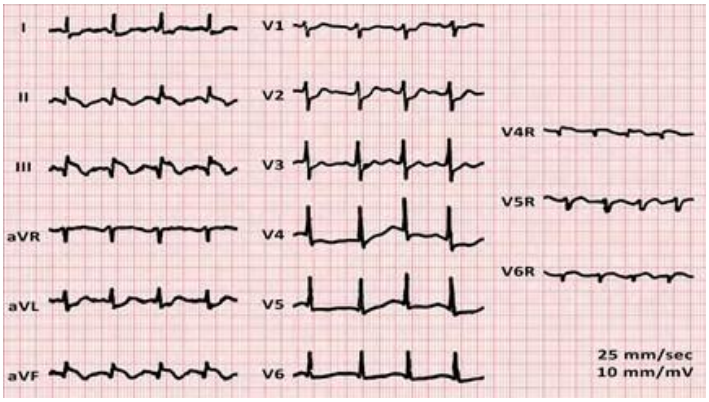

Figure1: ECG of the patient after admission.

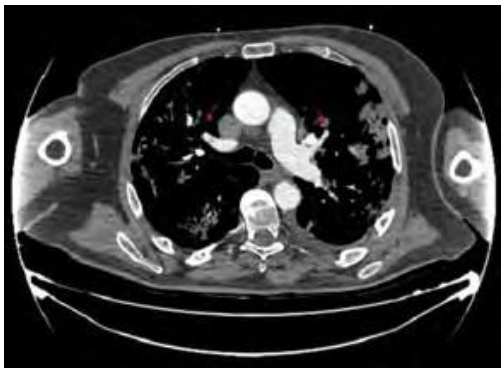

Figure2a: Filling defects in the lobar branches of the right upper and left upper lobes.

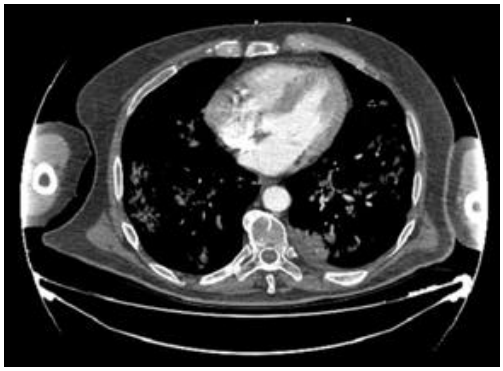

Figure2b: Filling defects in multiple segmental and subsegmental branches of the right and left lung.

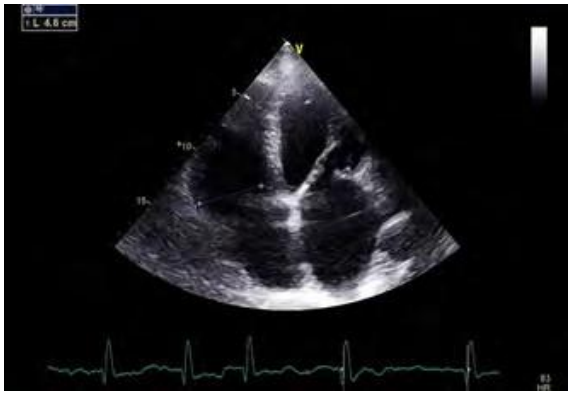

Figure3: Transthoracic echocardiogram on admission

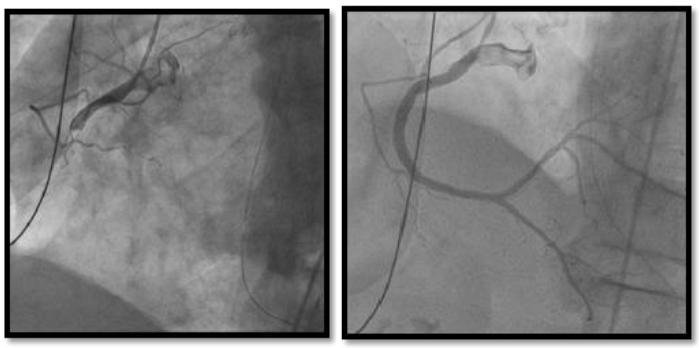

Figure4:

3. Discussion

Sars-CoV-2 infection has been associated with serious cardiac manifestations including acute myocardial infraction, pulmonary embolism,myocarditis,heartfailure,andTakotsubomyocardiopathy [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 vascularmanifestationsofCOVID-19.6However,alltheexisting studies so far are limited in size, have not reported all thrombotic events,andwerefocusedonpatientswithseverediseasehospital- ized in intensive care units (ICUs).

Studies from both China and the USA suggest markers such asD-dimer may be associated with increased mortality in hospitalized patients with COVID-19, with follow-up studies showing complicationsrelatedtothrombosisinthelungsandbrainaswell asclottingofrenalreplacementandextracorporealmembraneoxygenationcircuits[2-4].COVID-19hassignificantlydisruptedthe managementofacutecardiovasculardisease.DatafromChinahas shown that an elevation in troponin, with or without previous underlyingcardiovascularconditions,wasassociatedwithincreased 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 patientshad no culpritlesion 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 suggeststhattheincreasedthromboticriskofCOVID-19couldman- ifest as acute coronary thrombosis and STEMI. Management of thesepatientsinitiallypresentedlogisticalchallengeswithrespect to prompt intervention, although this has improved as protocols andprocedureshaveevolved[9].Forthisreason,whenassessinga COVID-19 infected patient with STsegment elevation, clinicians shouldbeawareofthepossibilityofPE,AMIandtheassociation between them [5].

Ackermannetal.firstreportedthepresenceofpulmonaryintussusceptiveangiogenesisandotherpulmonaryvascularfeaturesinthe lungsofsevenpatientswhodiedfromCOVID-19[7].Priorstudies variedregardingtheprecise incidenceofthrombosis;however,all suggestedanelevatedthrombosisriskinpatientswithCOVID-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,includingvascularandsystemicinflammationcaused 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 treatsystemicCOVID-19mayinterferewithcommonantiplatelet therapies and anticoagulation [10].

To the best of our knowledge, we are the first to report a case ofa critically-ill COVID-19 patient with bilateral pulmonary embolismincombinationwithmyocardialinfraction,requiringbothprimary angioplasty and anticoagulation therapy.

Asthereisstilldebateoverthemosteffectiveanti-coagulantther- apy 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 dosageremainsundefined.WithoutdoubtthetreatmentofCOVID-19 requires multidisciplinary expertise to address its multifaceted clinical manifestations. Moreover, attention must be paid to the interactionsofantiviralandotherpharmaceuticalagentsincluding oral and intravenous anticoagulants, with an aim to minimize the risk of bleeding and thrombo-embolic complications.

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