Pulmonary embolism and coronavirus disease 2019: persistent pulmonary hypertension?

Alejandro Cruz-Utrilla[1], Miguel Calderón-Flores[2], María P. Escribano-Subias[1,3]

1 Pulmonary Hypertension Unit, Cardiology Department, Hospital Universitario 12 de Octubre, Madrid, Spain
2 Cardiology Department, Hospital Universitario 12 de Octubre, Madrid, Spain
3 CIBER – Center for Biomedical Research Network, Madrid, Spain

A 47-year-old man with an unremarkable medical history was admitted to the emergency department because of ageusia, fever, cough, and dyspnea. The symptoms started 10 days before presentation. At that time, the patient showed acute hypoxemic respiratory failure and fever of up to 40 °C. Lymphopenia with a lymphocyte count of 0.6×10^3/µl, a C-reactive protein level of 25.7 mg/dl, and a D-dimer level of 354 µg/l were noted. On electrocardiography, the patient presented sinus tachycardia at 110 bpm with left anterior bundle-branch block. Chest X-ray showed peripheral, bilateral infiltrates (FIGURE 1A). In the context of the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) pandemic, the patient was diagnosed with bilateral coronavirus disease 2019 (COVID-19) pneumonia and ceftriaxone, azithromycin, and hydroxychloroquine were initiated. Eventually, a real-time polymerase chain reaction test for SARS-CoV-2, performed on a nasopharyngeal swab, yielded a positive result.

Two days later, the patient suddenly deteriorated and suffered from pleuritic pain. D-dimer levels peaked up to 52 958 µg/l. Computed tomography angiography was carried out and demonstrated the main pulmonary artery of 35 mm in diameter (FIGURE 1B), which was suggestive of significant pulmonary hypertension (PH), and bilateral pulmonary embolism (PE) in segmental arteries (FIGURE 1C and 1D). Extensive pulmonary parenchymal involvement was also observed. Despite the patient's hemodynamic stability at that time, he was admitted to the intensive care unit, requiring only mild supplemental oxygen supply. On transthoracic echocardiography performed during his stay in the intensive care unit, his systolic pulmonary artery pressure rose to 70 mm Hg. The examination was repeated 2 days before the patient's discharge and showed similar systolic pulmonary artery pressure, which confirmed significant PH.

The patient was discharged on day 35. Due to mild hypoxemia and dyspnea on exertion, he was receiving hospital-at-home care for 2 weeks. During that time, oxygen therapy was finally withdrawn.

It is too early to assert what consequences will appear in the lungs or pulmonary arteries of the patient, but persistent PH could be one of them. Acute respiratory distress syndrome, which is often present in patients with COVID-19, could cause PH due to hypoxic vasoconstriction.[1] Also, an increased prevalence of PE[2] as a consequence of a high thrombotic risk has also been observed, which has been related to coagulation abnormalities and an enhanced inflammatory response against the virus. The presence of elevated levels of D-dimer and parameters such as interleukin 6 or ferritin have been associated with those reactions and have shown prognostic relevance.[3] In this case, PH could be related to the acute episode of PE in addition to respiratory failure, but further assessment in a PH-dedicated outpatient clinic is required to confirm this hypothesis. Additionally, not only persistent PH due to incomplete resolution of PE may cause chronic thromboembolic disease (group IV PH) but also PH due to lung parenchymal damage (group III) or pulmonary artery hypertension (group I) secondary to endothelial dysfunction could all be plausible causes of persistent PH. These 3 hypotheses, however, need to be confirmed in further studies. A high suspicion index of PH is necessary in survivors with chronic dyspnea after SARS-CoV-2 infection–related pneumonia, acute respiratory distress syndrome, or PE.

Correspondence to:
Alejandro Cruz Utrilla, MD, Pulmonary Hypertension Unit, Cardiology Department, Hospital Universitario 12 de Octubre, Avenida de Córdoba S/N, 28041 Madrid, Spain, phone: +34 635765340, email: alejandro.cruz@salud.madrid.org, Twitter: @AlexCruzUtrilla
Received: April 27, 2020.
Revision accepted: June 8, 2020.
Published online: June 18, 2020.
Kardiol Pol. 2020; 78 (9): 937-938
doi:10.33963/KP.15436

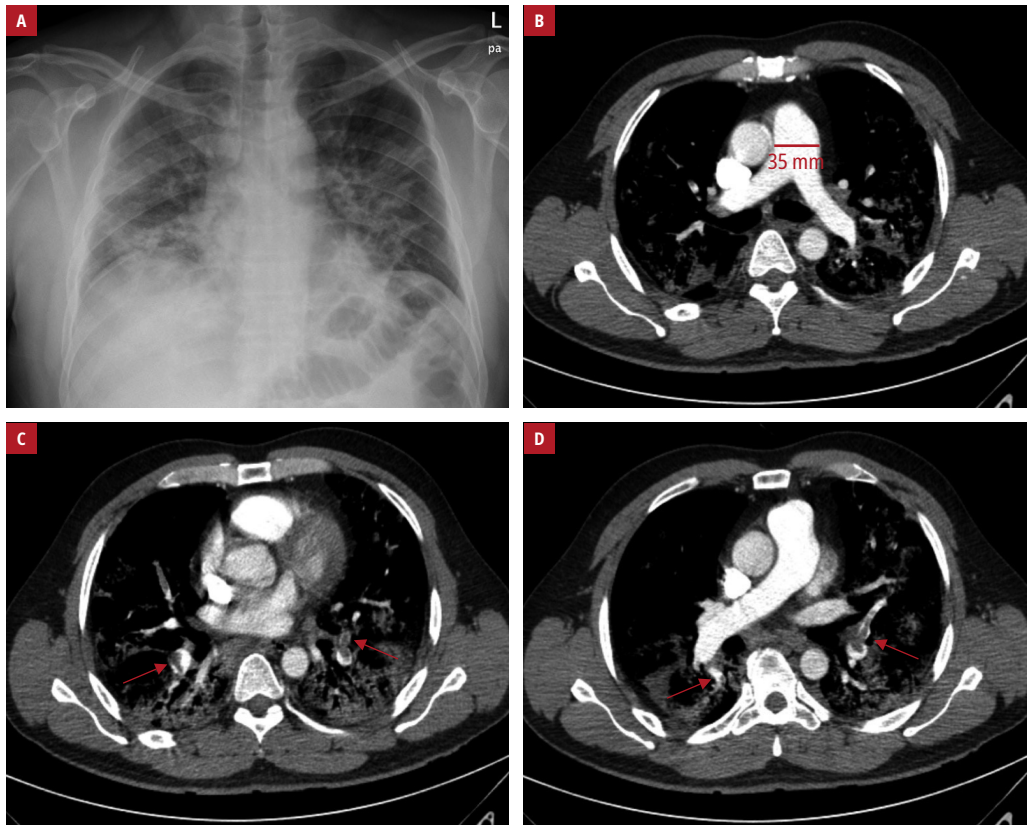

FIGURE 1 **A** – chest X-ray showing bilateral interstitial pneumonia; **B** – computed tomography angiography of the main pulmonary artery and the main branches. The main pulmonary artery is up to 35 mm in diameter, which is suggestive of significant pulmonary hypertension. **C**, **D** – computed tomography angiography demonstrating a thrombus in the bilateral segmental arteries (arrows)

ARTICLE INFORMATION

CONFLICT OF INTEREST None declared.

HOW TO CITE Cruz-Utrilla A, Calderón-Flores M, Escribano-Subias MP. Pulmonary embolism and coronavirus disease 2019: persistent pulmonary hypertension? Kardiol Pol. 2020; 78: 937-938. doi:10.33963/KP.15436

REFERENCES

1 Bikdeli B, Madhavan MV, Jimenez D, et al. COVID-19 and thrombotic or thromboembolic disease: implications for prevention, antithrombotic therapy, and follow-up. J Am Coll Cardiol. 2020; 75: 2950-2973.

2 Rotzinger DC, Beigelman-Aubry C, von Garnier C, Qanadli SD. Pulmonary embolism in patients with COVID-19: time to change the paradigm of computed tomography. Thromb Res. 2020; 190: 58-59.

3 Kollias A, Kyriakoulis KG, Dimakakos E, et al. Thromboembolic risk and anticoagulant therapy in COVID-19 patients: emerging evidence and call for action. Br J Haematol. 2020; 189: 846-847.