Abstract

English:
COVID-19 has been described as the cause for a proinflammatory and hypercoagulable state that induces thrombotic vascular lesions and, in more severe cases, disseminated intravascular coagulation. Increased values of d-dimers are related to the severity of the disease and are associated with worst prognosis. Intensive care studies reported an increased risk of pulmonary embolism and venous thrombosis diseases in COVID-19 compared with the historical control group even in patients who underwent the low-molecular-weight heparin (LWMH) prophylaxis. Patients with COVID-19 who have a stable clinical condition do not require hospitalisation and are treated at home with symptomatic therapy. LWMH is reserved for those with reduced mobility. In this case report, we describe a COVID-19 patient with pulmonary artery thrombosis treated at home.

Keywords
COVID-19 • d-dimers • pulmonary artery thrombosis • interstitial pneumonia

Rezumat

Romanian:

Cuvinte-cheie
COVID-19 • D-dimeri, tromboză arterială pulmonară • pneumonie intersticiată

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Introduction

Arising in China in the winter of 2019, the infection with the novel coronavirus – severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), was described in people who had been exposed to seafood market in Wuhan. Since then, there has been a quick spread of the disease, leading to a global pandemic of coronavirus disease (COVID-19) (1). After an incubation period of 4–5 days, patients who become symptomatic have fever, cough, sore throat, malaise and myalgias. A minority of cases occur with gastrointestinal symptoms (anorexia, nausea and diarrhoea) or neurological symptoms (anosmia and ageusia). Most patients (80%) have mild or moderate disease (including those with mild pneumonia), 15% had severe disease (diffuse lung injury) and 5% had critical illness (2). Patients with respiratory failure are usually monitored in hospital, while those with mild illness usually recover at home with supportive care and isolation. Self-monitoring of oxygen saturation is a useful tool for people who are at high risk for complications (lung diseases, cardiovascular diseases, diabetes and obesity) (3). COVID-19 has been well described as the cause of a proinflammatory and hypercoagulable state. After 7–14 days from the onset of the symptoms, there is a systemic increase of inflammatory mediators and cytokines (CK) (4). The inflammatory response induces a procoagulant effect and a diffuse endothelial damage that determine thrombotic vascular lesions and disseminated intravascular coagulation (DIC) in patients with critical illness (5,6). Increased values of d-dimers are related to the severity of the disease and are associated with the worst prognosis. Intensive care studies reported an increased risk of pulmonary embolism (PE) and venous thrombosis diseases (VTD) in COVID-19 compared with the historical control group even in patients who underwent the low-molecular-weight heparin (LMWH) prophylaxis (7). Monitoring d-dimers blood value has to be useful in identifying cases with a poor prognosis and higher risk of thrombotic phenomena (8). Stable patients infected with COVID-19 do not require hospitalisation and are treated at home (9). LMWH is reserved for those with reduced mobility. Home-treated patients do not undergo laboratory exams or radiological investigations in any phases of the disease. In this case report, we describe a COVID-19 patient with pulmonary artery thrombosis, treated at home with hydroxychloroquine.

Case presentation

A 57-year-old male with a history of virus B hepatitis (VHB) was admitted to our hospital after 3 days history of fever and chest pain. He was an active person and did not have any history of chronic medication. The diagnosis of COVID-19 was based on the detection of SARS-CoV-2 by means of reverse transcriptase polymerase chain-reaction (RT-PCR) assay on nasopharyngeal swab 1 month before admission. After the diagnosis, he started hydroxychloroquine (400 mg/day) for 10 days.

On examination, he was breathing regularly without dyspnoea with body temperature 37.7°C, heart rate 88 beats/min, blood pressure 145/65 mmHg, respiratory rate 16 breaths/min, oxygen saturation 96% at room air and body mass index (BMI) 26 kg/m². Heart sounds were regular and lung auscultation was clear in both lungs. Laboratory test showed: white blood cell count (WBC) = 12.960 mmc, neutrophils 10.730 mmc, C-reactive protein (CRP) 53 mg/L (normal value (NV) < 5), alanine aminotransferase (ALT) 214 U/L, d-dimers 3,742 mg/L (NV < 500), HIV-negative. Arterial blood gases (ABG) in breathing air showed a pH of 7.45, PaCO₂ 40 mmHg, PaO₂ 89 mmHg, satO₂ = 95%. Electrocardiography (EKG) was unremarkable. Doppler ultrasonography (DU) of the lower limbs was negative.

Figure 1. CT thorax with contrast (sagittal view) showing bilateral lower arterial thrombosis (red arrows).
for deep venous thrombosis (DVT) while a computed tomography pulmonary angiography (CTPA) demonstrated a bilateral arterial thrombosis associated with ground glass opacity (GGO) in the lower lobes (Figure 1). His nasal swab resulted positive for COVID-19. The patient started anticoagulation with LWMH 8,000 U twice a day. Abdominal ultrasonography did not demonstrate any signs of cirrhosis, hepatic focal lesions or portal hypertension. Echocardiogram showed normal left and right ventricular wall motion, left ventricle ejection fraction 60% and a normal pulmonary artery pressure (20 mmHg).

After 5 days of hospitalisation, laboratory exams showed WBC 8,720 mmc, ALT 207 U/L and d-dimer 1,425 mg/L. The patient had stable clinical condition without dyspnoea and fever, and the chest pain gradually improved. During discharge (13 days later), he continued anticoagulation therapy with warfarin. His nasal–pharyngeal swab was negative for COVID-19.

### Discussion

This article describes the case of a patient with mild COVID-19 and pulmonary artery thrombosis initially treated at home. Our patient never performed blood chemistry tests or radiological investigation during the home treatment period of COVID-19 infection. For this reason, it was not possible to determine when the thrombotic event occurred. CTPA showed a distal bilateral thrombosis of the lower pulmonary arterial branches where the pulmonary inflammation is most diffuse. It is noteworthy that Doppler ultrasound was negative for DVT. As current data suggest, coagulation disorders are significantly increased in COVID-19 patients, especially among those with severe disease. Indeed, it was observed that COVID-19 may predispose to both venous and arterial thrombosis due to inflammatory cascade, hypoxia and endothelial injury (10). Post-mortem evidence shows microvascular platelet-rich thrombotic depositions in small vessels of the lungs and other organs (11). The endothelial vascular damage inside the injured areas may predispose to the development of a primitive pulmonary artery thrombosis. A recent study reported the findings of 12 autopsies where the authors noted a high incidence of PE with or without underlying DVT (12,13).

In conclusion, we described a patient with mild COVID-19 disease who developed a pulmonary vascular injury, strictly correlated with an elevation of d-dimer values, without any signs of respiratory failure. Based on these studies, elevation of d-dimer levels could identify patients at risk for concurrent thrombotic diseases. We suggest a periodical monitoring of coagulation parameters and prophylactic dose of LMWH even in home patients with mild COVID-19 disease. Further studies will be needed to address this issue.

### Ethical approval

Inform consent was obtained from the patient in order to participate to write the article.

### Conflicts of interests

The authors declare that they have no conflicts of interest.

### References