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Concomitant myocardial infarction and pulmonary embolism in a patient with SARS-CoV-2 infection

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Coronavirus disease (COVID-19) is caused by severe acute respiratory syndrome (SARS-CoV-2). The infection can have a direct impact on the cardiovascular system in different ways. The lack of studies and data collection makes it difficult to estimate the prevalence of cardiovascular disease in patients with COVID-19. However, a small number of available studies suggest a strong association between the existence of cardiovascular disease and COVID-19 infection.

It has been reported that a history of cardiovascular disease increases the risk of infection severity and consequently, also mortality. In patients affected by coronavirus disease is described a prevalence of cardiovascular disease, hypertension and diabetes of 16.4%, 17.1% and 9.7% respectively[1-2], and it is observed that patients requiring admission to Intensive Care Units (ICU) are more likely to have these comorbidities, with an increase mortality[3-5].

Moreover, COVID-19 infection has been associated with an increased risk of cardiovascular events, including acute coronary syndrome, myocarditis, arrhythmias and venous thromboembolism, especially in patients with elevated inflammatory parameters, such as D-dimer or interleukin-6 (IL-6). For this reason, the need for anticoagulant treatment in these patients is under discussion[4-6]. Otherwise, we must consider that the infection is also associated with coagulopathy and increased risk of major bleeding.

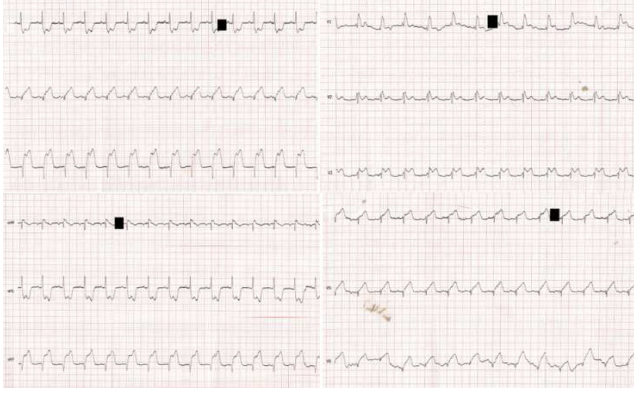
In this paper, we describe a case that presented two thrombotic events simultaneously despite receiving anticoagulant treatment with low-dose enoxaparin.

A 52-years-old male patient, without previous pathological history, was admitted to our center with symptoms of respiratory infection and high fever, being diagnosed of SARS-CoV-2 pneumonia. The initial PaFi was 280, and the analysis showed an elevation of the inflammatory parameters (IL-6 of 107 pg/ml and fibrinogen of 812 mg/dl, although an initial normal value of D-dimer). Initial electrocardiogram showed sinus rhythm without acute ischemia or repolarization changes. Due to progressive respiratory failure, he required endotracheal intubation, pronation sessions and admission to the ICU for 12 days. He also presented respiratory distress syndrome. During his stay in the ICU, he was hemodynamically stable and on prophylactic treatment with 60 mg of enoxaparin per day.

Subsequently, he remained respiratory stable (with low-flow nasal cannulas as the only support) and was discharged to the conventional pneumology ward. Laboratory tests prior to discharge showed a decrease in septic parameters and negative nasopharyngeal PCR for SARS-CoV-2. However, there was a significant increase in D-dimer (31,958 ng/ml). Fibrinogen levels normalized. After six hours of being in the ward, he suddenly presented

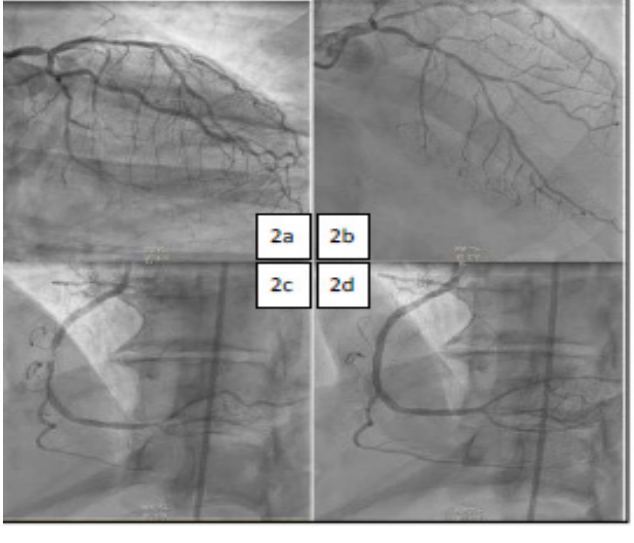
severe desaturation up to 55% and an oppressive chest pain, without initial hemodynamic instability. An electrocardiogram showed sinus tachycardia at 110 bpm with right bundle branch block and inferolateral subepicardial lesion (Figure 1),

Figure 1. (1) Electrocardiogram performed during clinical worsening, with associated chest pain, and with diagnosis of inferolateral STEMI.



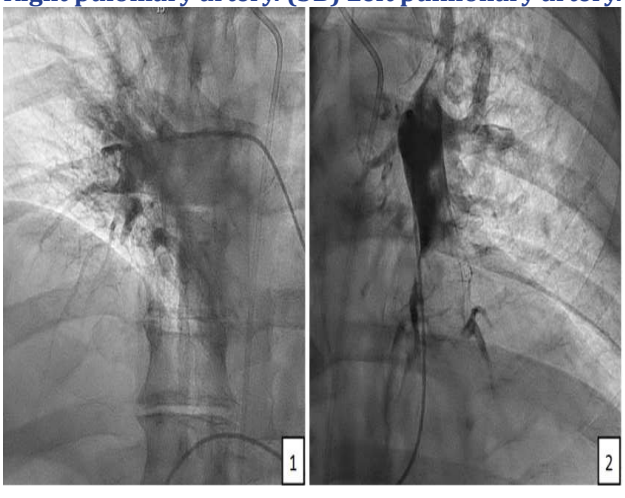
so it was diagnosed as an inferolateral ST-elevation myocardial infarction (STEMI). On physical examination, he did not show signs of left heart failure, but he had right congestion. The severe desaturation was not considered to be justified by STEMI isolated, and concomitant pulmonary thromboembolism was suspected. An echocardiogram showed severe dilatation of right chambers, with significant right ventricular dysfunction. Emergent coronary angiography showed three-vessel disease, the right coronary artery being considered the culprit artery. A drug-eluting stent was implanted covering the lesion (Figure 2).

Figure 2 (2a-c) Diagnostic coronary angiography, with threevessel disease. (2d) Angioplasty on the right coronary artery.



We decided to complete the diagnostic study with an angiography of the pulmonary arteries, which showed a bilateral pulmonary thromboembolism (Figure 3).

Figura 3. Angiography of the pulmonary arteries, that shows bilateral pulmonary thromboembolism. (3A) Right pulomary artery. (3B) Left pulmonary artery.



Mechanical thrombectomy was performed (fibrinolysis was ruled out because hemoptoic sputums during the angiography), persisting residual thrombus of less than 30% in the interlobar artery. A thrombus was also seen in the inferior vena cava, so a vena cava filter was implanted. Due to respiratory failure during the procedure, the patient required orotracheal reintubation and was admitted to the ICU again.

The initial evolution was marked by hemodynamic instability due to right ventricular dysfunction, requiring vasoactive support. He was anticoagulated with enoxaparin 150 mg daily. However, the prognosis depended on a very severe respiratory failure, initially secondary to pulmonary embolism and after complicated with bilateral pneumonia associated with mechanical ventilation due to Pseudomonas aeruginosa. Despite targeted antibiotic treatment, nitric oxide and pronation sessions, the evolution was torpid with persistence of refractory hypoxemia. We decided to implant venovenous extracorporeal membrane oxygenation (ECMO). 12 hours after cannulation, the patient presented refractory multi-organ failure, finally dying.

Few cases of concomitant acute myocardial infarction and pulmonary thromboembolism have been described in the literature, and in most cases there is usually an intracardiac

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communication, such as foramen ovale, which justifies the simultaneity of both events (paradoxical embolism). In the case that we have exposed, an intracardiac shunt was not observed, and we also consider that the physiopathogenic mechanisms of both events were different. On one side, an acute rupture of an atherothrombotic plaque was observed as a cause of the coronary syndrome, and on the other, thrombotic material was visualized at inferior vena cava that explains the pulmonary thromboembolism. However, it should be noted that, despite the SARS-CoV-2 pneumonia was presenting a good evolution, the inflammatory markers (in this case the D-dimer) were increasing. The elevation of inflammatory markers could be a predictor of cardiovascular events, considering that both prothrombotic state and endothelial dysfunction have been associated with systemic inflammation due to COVID-19 infection. A significant elevation of these markers could suggest the need of more aggressive anticoagulant treatment to avoid cardiovascular events.

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