

## CASE REPORT

## Pulmonary Embolism Related to COVID-19 Quarantine

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### Abstract

The mandatory quarantine imposed in response to the COVID-19 pandemic has been related to an increase in cases of thromboembolism in non-COVID-19 patients. Particularly in countries like Argentina where strict quarantine was mandated, individuals were far less likely to risk being outside or

observing healthy practices that would maintain the exacerbation of diseases such as thromboembolism. We report the case of a patient with pulmonary thromboembolism without usual triggering causes during the quarantine period, related to a previously undiagnosed hypercoagulable condition.

### Introduction

Quarantine imposed by different countries due to the COVID-19 pandemic has resulted in increased cases of thromboembolism in non-COVID-19 patients.[1, 2] In Argentina, to prevent the spread of the coronavirus, the president announced mandatory quarantine and citizens with jobs described as non-essential had to be confined to their usual residences or to the place where they were at when he announced nationwide lockdown. Citizens had to refrain from attending their workplaces except for essential workers and emergency care providers. Moreover, any public or private event or activity as well as schooling and social gatherings were cancelled. People were not allowed to travel on the roads and freeways unless they had to carry out the essential jobs described on the official list, like the case of transportation of essential cleaning supplies, medicine, and food.[3]

### Case Presentation

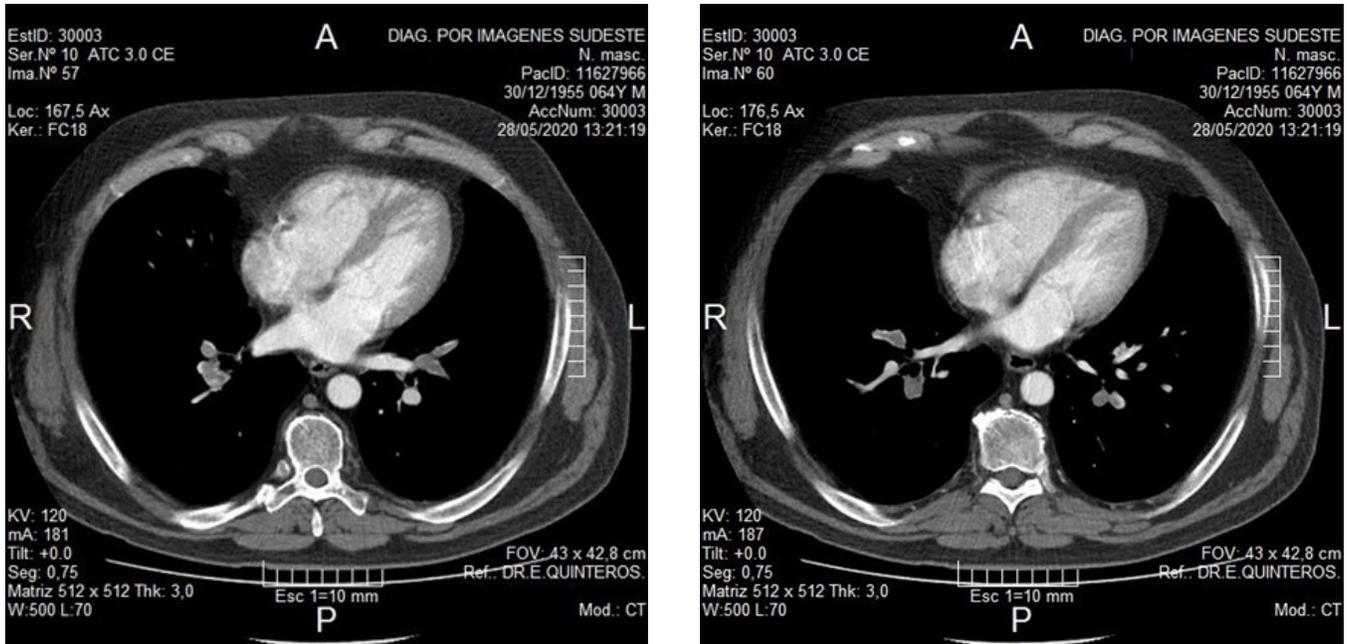
Thirty-seven days after the mandatory quarantine imposed in Argentina after March 2020, which forced everyone except for essential workers and employees of other emergency care situations into lockdown, a 64-year-old man living in Departamento Unión of Córdoba Province (without community viral circulation) consulted his primary care physician for acute dyspnea to minor habitual efforts.[3] The patient realized the symptoms after a short postprandial walk.

Two hours earlier, he had participated in mild physical activities without experiencing any symptoms. His medical records were significant: Type II diabetes, high blood pressure, mild carotid vascular disease, and obesity. The patient was current with all scheduled metabolic and blood pressure checkups as well as regularly administered doses of aspirin, except for the last seven days, which he had forgotten. Ten years before, he had suffered from deep vein thrombosis during a long flight. On that occasion, he had not been diagnosed with any predictor. The patient did not have any problems with being in lockdown, but his physical activity had fallen significantly since the beginning of the lockdown.

The patient was sent to a cardiology clinic, where he was admitted 48 hours after the initial symptoms because of continuing symptoms. The delay in admittance was due in large part because of his fear of contracting SARS-CoV-2. He did not have fever or any other respiratory symptoms, or trips to areas with SARS-CoV-2, viral outbreak, or close contact with a patient with a COVID-19 diagnosis. He had no angina, palpitations, syncope, or dyspnea at rest. The patient did not have any other symptoms at rest, and his axillary temperature was 36.4 °C and his radial pulse rate was 100 bpm. The patient had slight arterial oxygen desaturation by oximetry (89% with ambient air) and an S3 on auscultation, without physical signs of venous thrombosis. The findings of his examination and laboratory tests are summarized in **Table 1**.

**Table 1.** Patient examinations and laboratory findings.

Blood pressure	100/70 mmhg
Heart Rate	110 lpm, regular, rhythmic
Respiratory Rate	18 rpm at rest
Saturation O <sub>2</sub> Oximetry	89% with ambient air
Temperature	36.4 °C
ECG	Sinusal tachycardia
D dimer	198 ng/ml (VN<200 ng/ml)
Protrombine Time	21.1 seg RIN 1,66
PCR	<6 mg/l
Glucemia	128 mg%



**Figure 1.** Lung angio-tomography.

The diagnostic impression was hypoxic respiratory failure secondary to acute pulmonary thromboembolism (PE). The risk chances of PE were calculated with the GENEVA Score at 8 points (moderate risk), the Wells Score at 9 points (high risk of PE), and the PESI Score at 114 points (high risk).

A POCUS pulmonary ultrasound and a POCUS echocardiogram showed an “A” profile at the pulmonary level (BLUE Protocol).[4] It also showed evidence of a left ventricular hypertrophy, with hyperdynamic systolic function of the left ventricle and dilation and systolic dysfunction of the right ventricle. At this, the patient received anticoagulation.

A formal subsequent venous ultrasound did not find evidence of deep vein thrombosis. However, a chest angio-tomography revealed a pulmonary artery caliber of 30mm in the trunk that was found to be patent as well as the right and left main branches. Hypodense

material which partially blocked the vascular lumen, compatible with acute PE, was found in the lobar and segmental branches of all lobes (Figure 1).

A SARS-CoV-2 PCR was not performed because the patient did not present with the local case definition criteria at the time of admission. Anticoagulation with sodium heparin, oxygen at 3L/minute, and boluses of saline crystalloids were administered with good outcome. The subsequent workup for other patient-related factors predisposing him for venous thromboembolism revealed Antiphospholipid Syndrome with evidence of a circulating anticoagulant (lupus anticoagulant) (Table 2). It was decided that the patient was going to remain anticoagulated in the long term.[5]

**Table 2.** Subsequent workup for other patient-related factors.

Circulating anticoagulant	LA1 126 LA2 50, normalized reason 2.54
Protein C	112%
Ac Against phospholipids IgC	1.7 (normal)
Ac Agains phospholipids IgM 1	Normal
Ac Anticardiolipin igG,IgM	Negative
Ac Antinuclear	Negative
Rheumatoid Factor	Negative
Protein S	88% normal

## Discussion

This case study of acute PE contributes to the field by offering new evidence of the increased risk of high-risk thromboembolic situations. In this case, the situation presented in a patient with unusual triggering factors under the conditions of decreased physical activity due to the SARS-CoV-2 pandemic quarantine with the presence of Antiphospholipid Syndrome.[1-3] A delay in consulting a physician due to fear of becoming in-

fectured while visiting health care areas increased this risk. Cases such as the one described above show the need to evaluate the risk/benefit to health outcomes from strict/mandatory quarantines in areas without viral circulation, and the need to give sound advice to patients with known risks. In the case study described, the risk of developing thromboembolism in quarantine can be equated with the risk a person can have during a long flight.

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