 doi:10.20944/preprints202008.0688.v1

Pulmonary Embolismo Related To Covid 19 Quarentine

Authors: Eduardo J.Quinteros,MD , Juan Bissonni ,MD

Institutional email :cmayo-administracion@nodosud.com.ar

ABSTRACT : The quarantine imposed as the response to the COVID 19 pandemic has been related to an increase in cases of thromboembolism in Non-COVID19 patients .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

KEYWORDS: lung disease; pulmonary embolis; circulating anticoagulant

To the editor,

Background: Quarantine imposed by different countries due to COVID-19 pandemic has resulted in an increase of cases of thromboembolism in non-COVID-19 patients [1,2].

Objective: To share a case study of a patient who consulted for dyspnea due to pulmonary thromboembolism without the common causes triggering it during mandatory quarantine. The case was related to a previous undiagnosed hypercoagulable state.

Clinical case study: Thirty-seven days after the mandatory quarantine imposed in Argentina since March 2020 (Social, Preventive and Obligatory Isolation [ASPO] throughout the country [3]), which made most people to be into lockdown, except for essential workers and other emergency care situations, a 64-year-old man living in Departamento Unión, a county in Córdoba Province without community viral circulation, consulted his primary care physician for acute dyspnea to minor habitual efforts. The patient realized about the symptoms in a short postprandial walk. Two hours earlier, he had carried out mild effort activities without experiencing any symptoms. His medical records were significant: Type II diabetes, high blood pressure, mild carotid vascular disease, and obesity, with good metabolic and blood pressure checkups, taking his regular medicine except for the last 7 doses of aspirin. Ten years before, he had suffered from deep vein thrombosis during a long flight. On that occasion, he had not been diagnosed with a predictor. The patient did not have any problems with being into lockdown, and his physical activity had fallen significantly since the beginning of the lockdown.

The patient was sent to a cardiology clinic. He attends it 48 hours after the initial symptoms because of his fear of getting the SARS COV-2 and due to the fact that his symptoms continued. He did not have fever or any other respiratory symptoms, or trips to areas with SARS COV-2 viral circulation, 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, his axillary temperature was of 36.4 °C, his radial pulse rate was of 100 bpm , 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 Findings	
Blood pressure	100/70 mmhg
Heart Rate	110 lpm, regular, rhytmic

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Respiratory Rate	18 rpm at rest
SaturaTion O2 Oximetry	99% con 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%*

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

A POCUS pulmonary ultrasound and a POCUS echocardiogram showed an A profile at the pulmonary level (BLUE Protocol) [4], and evidence of Left Ventricular Hypertrophy with hyperdynamic Systolic Function of the Left Ventricle, with dilation and systolic dysfunction of the Right ventricle. Anticoagulation was started.

A formal subsequent venous ultrasound did not find evidence of deep vein thrombosis, and the chest angio-tomography revealed a pulmonary artery caliber of 30mm in the trunk, which 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 and 2

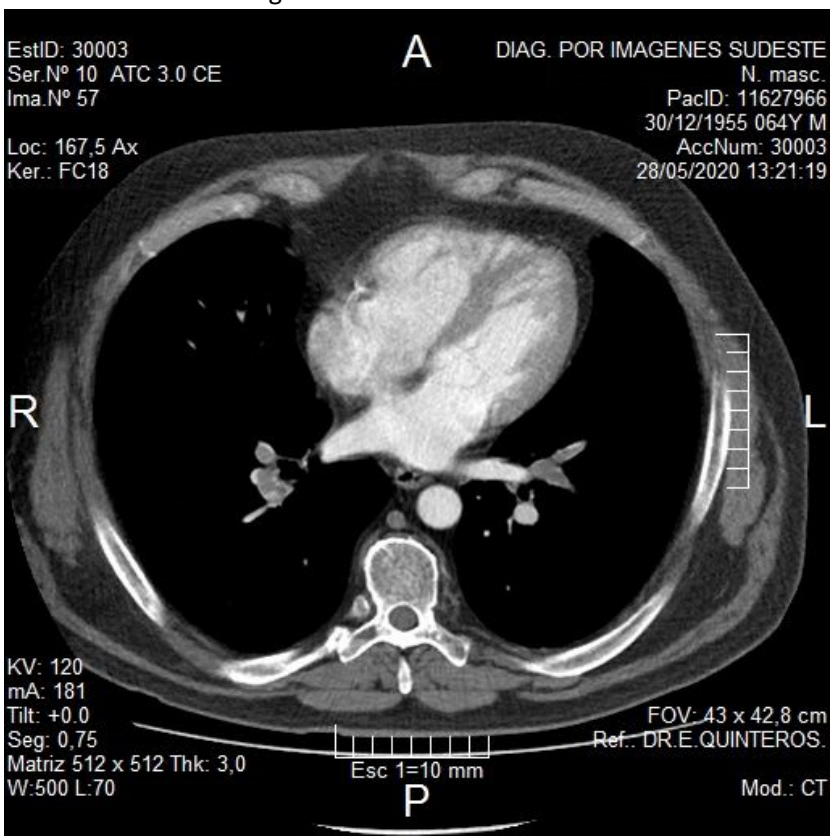

Figure 1

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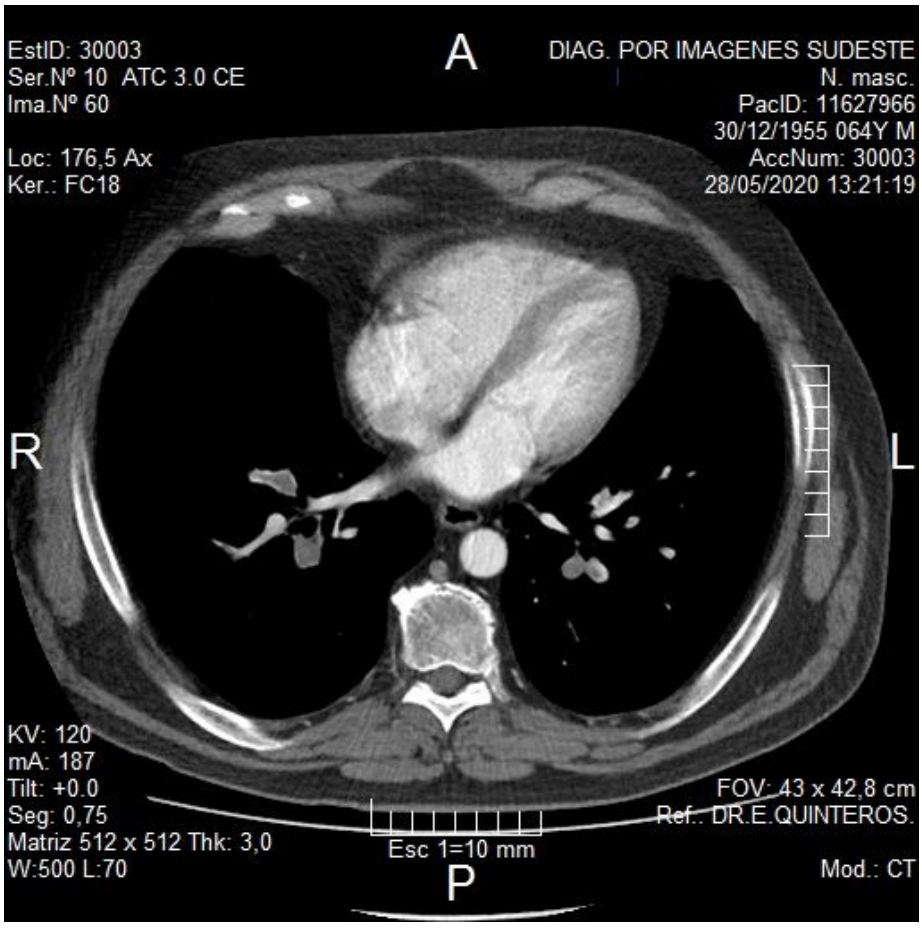

Figure 2

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

Tabla 2 – workup

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
Reumatoid Factor	Negative
Protein S	88% normal

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Discussion: The present case study of acute PE makes a contribution to the field offering new evidence of the increased risk of high-risk thromboembolic situations in patients with unusual triggering factors, under the limited conditions and decrease in physical activity due to the SARS-COV2 Pandemic quarantine[1,2,3], in this case, the presence of a Antiphospholipid Syndrome. A delay in consulting a physician due to a fear of becoming infected in health care areas increases this risk. Cases such as the one described above, show the need to evaluate the risk / benefit to health of strict / mandatory quarantines in areas without viral circulation, and the need to give sound advice to patients with known risk. 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.

Eduardo Quinteros, MD. Board Certified in Internal Medicine and Cardiology. Clínica Privada Mayo, Bell Ville, Argentina

Juan Bisonni, MD. Board cetified in Images diagnosis/Radiology. Diagnóstico por Imágenes Sudeste, Bell Ville,Argentina

Our thanks to professor Mariana Falco, Msc, National University of Villa María, for the revision of the text in English

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