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Scientific letter

Role of echocardiography in the diagnosis of pulmonary embolism during SARS-CoV-2 infection☆

Papel de la ecocardiografía en el diagnóstico de la embolia pulmonar durante la infección por SARS-CoV-2

Dear Editor:

The clinical manifestations of SARS-CoV-2 (COVID-19) infection range from mild respiratory symptoms to acute respiratory distress, requiring admission to an intensive care unit (ICU).[1,2] Cardiac involvement by the virus has been described in 20%–25% of patients and is associated with a worse prognosis.[3,4] This cardiac abnormality has been defined as an elevation of laboratory markers for myocardial damage, but without any accompanying cardiac imaging test.[3]

On the other hand, infection is associated with an increased incidence of thrombosis explained by the cytokine storm and the increased expression of tissue factor produced by infection, added to the severity and immobilisation of any admitted patient.[4]

Echocardiography is the mainstay of cardiac imaging techniques, and its use is of great interest for the monitoring of patients hospitalized in ICUs, as well as for the diagnosis and follow-up of pulmonary embolism (PE) in hemodynamically unstable and stable patients, respectively.[5] However, its use has been limited during the pandemic due to the risk of spreading the infection. The aim of our work is to describe the echocardiographic findings in patients diagnosed with PE in the context of COVID-19.

We report 11 patients diagnosed with SARS-CoV-2 infection who presented with PE and underwent an echocardiography during hospital admission. These patients were 8 men and 3 women with a mean age of 60 years who had obesity (72%), hypertension (54%) and dyslipidaemia (45%) as the most important comorbidities. Diagnosis of infection was made by reverse transcriptase polymerase chain reaction test performed on admission, diagnosis of PE was made by computed tomography angiography (CT angiography) and echocardiography was performed by cardiology and/or intensive care physicians. Five patients (45.5%) were admitted to the ICU, five to a conventional hospital ward and one to the emergency department.

The time from hospital admission to PE diagnosis was 14.36 days, with a minimum of 0 (one patient) and a maximum of 44 (one patient).

According to radiological findings most PEs (54.54%) occurred in segmental arteries and 27.27% in lobar arteries; one patient had involvement of main pulmonary arteries and another of subsegmental arteries. PE was bilateral in 45.45% of cases.

Echocardiography was performed during admission in 11 patients. Right ventricular (RV) involvement was observed in 4 (36.36%) patients, in the form of RV hypocontractility in 2 (18.2%) patients, RV dilatation in one (9.1%) patient and a combination of hypocontractility and dilatation in another patient (9.1%). Signs of pulmonary hypertension were observed in 2 (18.2%) patients and left ventricular dysfunction only in one (9.1%) patient. In terms of laboratory tests, at the acute time of PE diagnosis (48 h peri-diagnosis), peak values of markers for myocardial damage (troponin T and N-terminal pro-brain natriuretic peptide) were observed in 4 patients (36.36%), 3 of whom also had radiographic and echocardiographic RV involvement.

Although cardiological abnormalities (mainly RV hypocontractility) could be found at the time of PE diagnosis in almost 40% of patients, this did not influence the therapeutic approach and it was observed that these abnormalities were corrected in subsequent ultrasound scans during admission. Furthermore, there seems to be a correlation between radiological, echocardiographic and laboratory findings of cardiac involvement, such that all patients with signs of RV involvement on CT angiography had RV involvement on echocardiography, and almost all had elevated markers of myocardial damage on blood tests. Thus, cardiac involvement in PE could be inferred from the radiological and analytical findings that we see in patients without SARS-CoV-2.[5]

The current findings support the fact that the presence of PE in the context of SARS-CoV-2 infection does not justify a change in the indications for echocardiography already established by the 2019 clinical practice guidelines.[5] This may be due to the fact that most of these events occur in secondary arteries and/or are small in size and therefore do not usually cause haemodynamic instability. However, according to our results, the finding of RV hypocontractility in patients with COVID-19 could indicate undiagnosed PE. Finally, we believe that the use of echocardiography in PE during SARS-CoV-2 infection should be limited as a diagnostic and monitoring tool to haemodynamically unstable patients, or those who while stable show clinical deterioration.

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