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Electrocardiographic signs of acute right ventricular hypertrophy in patients with COVID-19 pneumonia: A clinical case series.

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Abstract

This paper reports 6 cases of patients affected by coronavirus disease 2019 bilateral pneumonia with associated acute respiratory distress and signs of acute right ventricular hypertrophy on electrocardiography despite the absence of acute pulmonary embolism or signs of severe pulmonary hypertension on transthoracic echocardiography. These cases suggest a possible connection between acute elevated right ventricular afterload and acute respiratory distress in patients affected by SARS-CoV-2.

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Introduction

Coronavirus disease 2019 (COVID-19) is a rapidly expanding global pandemic due to Severe Acute Respiratory Syndrome Coronavirus-2 (SARS-CoV-2) resulting in significant morbidity and mortality. This major morbidity and mortality are largely due to acute viral pneumonia that evolves to acute respiratory distress syndrome (ARDS). Reports suggest that among those infected with SARS-CoV-2, up to 20% develop severe disease requiring hospitalization [1], and up to 40% cases are associated with ARDS [2]. On the other hand, right ventricular (RV) overload and RV failure are common in patients who develop ARDS [3]. However, until now, the incidence and prognostic implications of this phenomenon have not been well described in patients with ARDS secondary to COVID-19 pneumonia. We present a clinical case series of 6 patients affected by COVID-19 bilateral pneumonia and ARDS associated with signs of acute RV hypertrophy (RVH) on electrocardiography (ECG) despite the absence of acute pulmonary embolism (PE) or signs of severe pulmonary hypertension (pH) on transthoracic echocardiography (TTE).

Clinical case series

The basal characteristics of the patients are described in Table 1. All patients were admitted to the hospital between March 15, 2020, and April 10, 2020, with a diagnosis of SARS-CoV-2 bilateral pneumonia, acute respiratory failure and radiology signs of ARDS. In all cases, electrocardiographic signs of RVH and RV overload were detected. Specifically, incomplete or complete right bundle branch block (RBBB) and the SQII pattern were the most frequent signs detected (5 of 6 patients), followed by inverted T waves in the right precordial leads (Fig. 1A). Other signs, such as enlargement of the R wave in V1 or the S wave in V5-V6, were not demonstrated. Previous ECG registers were detected in medical records only in 2 patients, and in both cases, these signs of RVH were not demonstrated. A TTE study was performed in all patients. The main findings were dilatation of the right cavities in four patients with normal ejection fraction of the RV and the absence of severe PH in all studies (systolic pulmonary artery pressure [aPAP] estimated by continuous wave Doppler measurements of the RV-right atrial pressure gradient reflected in the peak tricuspid regurgitation velocity and mean pulmonary pressure using the pulmonary acceleration time measured from the pulsed Doppler profile of the pulmonary valve) (Table 1). In all cases, PE was excluded by computed tomography. All patients were managed with hydroxychloroquine, lopinavir/ritonavir, azithromycin, ceftriaxone, low-molecular-weight heparin and high doses of prednisone. Indeed, oxygenation with high-flow systems was required in all cases to meet oxygenation goals, although invasive mechanical ventilation was not required in any of them. Unfortunately, 2 patients died during admission due to septic shock, and 4 patients were discharged after verifying radiological improvement of pneumonia and oxygenation parameters. In the follow-up, a new ECG register and TTE study could be performed in 3 patients one month after discharge (Table 2). ECG signs of RVH and mild dilatation of the right cavities were still present in all cases despite improvement in the values of sPAP (Fig. 1B).

Discussion

RVH results from increased RV afterload from PH or other heart, lung, or sleep disorders, which are associated with significant morbidity and mortality [4]. Electrocardiography, although specific, lacks sensitivity for a diagnosis of RVH [5]. The current recommended AHA criteria for
RVH by surface ECG are based on older studies with small study populations of severe RVH from advanced cardiopulmonary disease [4]. However, recent studies have demonstrated that the ECG screening criteria for RVH are not sufficiently sensitive or specific for the screening of mild RVH in adults without clinical cardiovascular disease [4]. In patients with ARSD secondary to acute lung injury, RVH and RV failure are common and are predictors of mortality [3]. However, ECG findings of RVH in this setting have not been used for prognostic objectives, although they provide evidence of advanced disease that may be difficult to manage in critically ill patients [6]. In our cases, the presence of ECG signs of RVH was a predictor of dilatation of the RV in TTE and a hallmark of severity of COVID-19 pneumonia severity. Indeed, the SIQIII pattern was the most frequent sign detected, a finding important because this pattern is usually also found in patients with massive PE. In fact, it is known that COVID-19-affected patients have a heightened inflammatory state that increases their thrombotic risk, specially of venous thromboembolic events and EP [7]. In light of this, although detection of a S\textsubscript{QIII} pattern is probably secondary to increased RV afterload in patients with SARS-CoV-2 infection complicated by ARDS due to pulmonary vasoconstriction as a result of hypoxia and acute lung injury, early detection of EP is recommended.

On the other hand, the temporal evolution of ECG signs of RVH after an acute PH secondary to ARSD or other lung injuries is not well known. In patients affected by chronic thromboembolic PH or pulmonary arterial hypertension, the ECG parameters most strictly linked to the reverse remodeling of the right ventricle are the S wave amplitude in V1, the R:S wave ratio in lead V6 and the S\textsubscript{QIII} pattern. However, in previous studies, in patients affected by chronic thromboembolic PH, these ECG signs did not improve 1 month after improvement of the hemodynamic parameters of PH with pulmonary endarterectomy but did improve late after surgery (1 year) [8]. In our patients, ECG signs of RVH and RV overload were still present after 1 month. Future follow-up will allow us to clarify the temporal evolution of ECG signs in RVH.

In conclusion, our clinical case series shows that signs of acute RVH on ECG are common in patients affected by COVID-19 pneumonia with associated ARDS and that they could be a predictor of RV dilatation and a hallmark of severity of COVID-19 pneumonia.

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