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LETTER TO THE EDITOR

Letter by Vazgiourakis et al Regarding Article, "Spectrum of Cardiac Manifestations in COVID-19: A Systematic Echocardiographic Study"

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To the Editor:

We read with great interest the study by Szekely et al[1] presenting a systemic echocardiography evaluation in 100 patients with coronavirus disease 2019 (COVID-19). In 68 patients, abnormal studies were reported, with right ventricular (RV) dysfunction being the most common one. In 20 patients, repeated echocardiogram was performed because of clinical deterioration, with 16 of them presenting respiratory deterioration, warranting mechanical ventilation. The authors report that the only echocardiographic variables presenting significant deterioration were RV-related. These findings are interesting because, until now, all the available information for cardiac involvement in patients with COVID-19 relied on clinical and biochemical markers.[2] However, we were surprised to find that nothing is reported concerning the effect of mechanical ventilation on RV deterioration, although the authors acknowledge that increased positive end-expiratory pressure (PEEP) is a contributing factor.

PEEP levels induce compression of the pulmonary vasculature, leading, finally, to right heart dilation. This physiological consequence is exaggerated when the lung compliance is relatively normal, as has been reported in COVID-19 pneumonia. Therefore, high PEEP, according to current guidelines,[3] may further raise pulmonary vascular resistance and lead to dead-space ventilation (transition of West Lung Zone 2 to 1). Therefore, the authors' comments on the mechanical ventilation variables and other parameters of the patient's respiratory system (PEEP, compliance, Pa_{O_2}/fraction of inspired oxygen) would be valuable.

RV dilatation and dysfunction are increasingly recognized as a distinct feature in COVID-19 pathophysiology. Recently, pulmonary vascular endothelialitis and thrombosis have been described, indicating that primary cardiovascular complications, increased pulmonary vascular resistances, and RV dysfunction may be fundamental aspects of the COVID-19 clinical spectrum.[4] When the pulmonary vasculature is injured (microthrombosis), additional factors such as pulmonary thromboembolism and PEEP application may further increase pulmonary vascular resistances. The authors report that 5 of the 12 patients with RV deterioration presented deep venous thrombosis; therefore, pulmonary embolism cannot be excluded. However, in the rest of the patients, in whom the clinical condition and probably RV function did not worsen (a second echocardiogram examination was not performed), deep vein thrombosis or pulmonary embolism presence was not evaluated. Thromboembolic events may have been present in the rest of the patients, indicating that these events maybe contributing factors but not the major event per se. In this context, data on PEEP levels used would be highly informative because they could at least partly explain the worsening in RV performance. Moreover, could RV dilation be minimized after manipulation of the PEEP levels (if such a maneuver was performed)? In our patients, PEEP de-escalation improved RV function significantly.

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RV dysfunction may be present from the initial phases of the disease, resulting from endothelialitis. Mechanical ventilation may be an additional factor implicated in the clinical course of RV dilation and dysfunction in COVID-19. Echocardiography may significantly influence decision-making for adjusting the ventilator settings in COVID-19 as "the right heart only knows."[5]

ARTICLE INFORMATION

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Disclosures

None.

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