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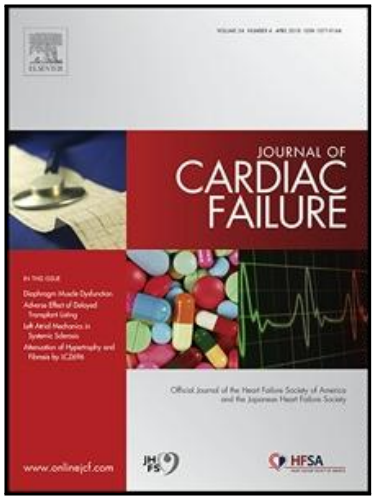

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Recognizing Right Ventricular Dysfunction in COVID-19 Related Respiratory Illness

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To the editor:

We have read the article by Tersalvi and colleagues detailing mechanisms of elevated troponin in patients with Coronavirus Disease 2019 (COVID-19) (1), and we write to encourage recognition of acute right ventricular (RV) strain as an additional possibility.

Acute respiratory distress syndrome (ARDS) is a recognized complication of COVID-19 and a known etiology of acute cor pulmonale (ACP). The mechanism of ACP in ARDS is established as refractory hypoxemia, pulmonary edema, and microvascular thrombosis acutely increase RV afterload (2). Among COVID-19 patients, elevations in troponin and brain natriuretic peptide levels have been shown to be both correlative with elevations in D-dimer and predictive of mortality (3). These laboratory abnormalities may not be a coincidence as emerging autopsies on COVID-19 patients have identified small vessel pulmonary thrombosis and RV dilation (4).

At a time when formal echocardiograms may not be performed due to limitations of exposure risks among healthcare personnel, providers must understand the findings of ACP on point of care ultrasound. RV enlargement may be noted in the apical four chamber view with an RV area greater than 60% of the left ventricular area in end-diastole (5). A “D-shaped” ventricular septum may be visualized in the parasternal short axis window but may only be seen at end-systole due to prolonged RV contraction. A reduced tricuspid annular plane systolic excursion or McConnell’s sign may also be noted.

Alveolar ventilation must be optimized to limit hypoxic vasoconstriction while considering the hemodynamic consequences of high positive end-expiratory pressure (PEEP) on RV preload and afterload. Utilization of prone ventilation is one means in which this is accomplished while minimizing PEEP. A pulmonary arterial catheter may limit comorbid cardiogenic edema while also aiding in optimization of RV preload, cardiac output, and pulmonary vascular resistance for which inotropic support and inhaled pulmonary vasodilators should be considered. In the absence of a pulmonary

arterial catheter, central venous pressure monitoring and evaluation of central venous oxygen saturation is encouraged for optimizing RV preload, kidney perfusion pressure gradients, and cardiac output. Management of microvascular thrombi remains unclear however close monitoring of D-dimer and cardiac biomarkers may have implications on empiric anticoagulation however this warrants further investigation.

As therapeutic trials are ongoing for coronavirus infections, we must not forget that management remains primarily supportive care at this time. This requires optimization and support for perhaps the most impacted side of the heart in critical COVID-19 infections, the right ventricle.

Statements

1) The presented work has not been previously published and is not currently under consideration for publication at any other journal.
2) Conflicts of interest: All authors have no conflicts of interest to disclose
3) Authorship: All authors have contributed meaningfully to the work, agree with its content, and consent to publication if accepted.

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