Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Letter to the Editor

Recognizing Right Ventricular Dysfunction in Coronavirus Disease-2019–Related Respiratory Illness

To the Editor:

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

Acute respiratory distress syndrome is a recognized complication of COVID-19 and a known etiology of acute cor pulmonale. The mechanism of acute cor pulmonale in acute respiratory distress syndrome is established as refractory hypoxemia, pulmonary edema, and microvascular thrombosis acutely increase RV afterload.[2] Among patients with COVID-19, 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 patients with COVID-19 have identified small vessel pulmonary thrombosis and RV dilation.[4]

At a time when formal echocardiograms may not be performed owing to limitations of exposure risks among health care personnel, providers must understand the findings of acute cor pulmonale on point-of-care ultrasound examination. RV enlargement may be noted in the apical 4-chamber view with an RV area of 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 owing 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 on RV preload and afterload. The use of prone ventilation is one means by which this is accomplished while minimizing positive end-expiratory pressure. 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. This process 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.

Disclosures

None.

Supplementary materials

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.cardfail.2020.05.003.

Gregory P. Milligan, MD, MPH
Amit Alam, MD
Cesar Guerrero-Miranda, MD
Baylor University Medical Center, Dallas, Texas

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