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Right Ventricular Hemodynamics in COVID-19 Patients

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Right Ventricular Hemodynamics in COVID-19 Patients

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Abstract

The right ventricle is highly sensitive to afterload, and pulmonary compromise can increase pulmonary vascular resistance and lead to right ventricular dysfunction. Pulmonary hypertension can also be exacerbated by mechanical ventilation. Patients with COVID-19 pneumonia and respiratory failure, especially those ventilated with positive end-expiratory pressure, are prone to pulmonary hypertension. Understanding their right ventricular hemodynamics can have therapeutic and prognostic implications.

Keywords: RV dysfunction, positive end-expiratory pressure, cardiac output, cardiac index

Background

There are four questions to consider when treating patients with COVID-19 pneumonia and acute respiratory distress syndrome (ARDS):

- Is there evidence of right ventricular (RV) dysfunction?
- Is this mediated by the heart, lungs, or a combination?
- Does this have independent prognostic significance?
- Does it have therapeutic implications?

The right ventricle is highly sensitive to afterload and, as such, may be affected by pulmonary pathophysiology, including hypoxia, hypercarbia, atelectasis, and overdistension. Hypoxia increases pulmonary vascular resistance because of hypoxic pulmonary vasoconstriction, which shunts blood away from deoxygenated areas. Hypercarbia also increases pulmonary vascular resistance.[1] Atelectasis leads to hypoxia and hypercarbia and decreases the number of perfused alveoli, increasing pulmonary resistance.[2,3] Overdistension of alveoli also increases pulmonary vascular resistance. All of these factors can cause increased pulmonary vascular resistance and right-sided afterload.

Effects of Ventilation on Respiratory Failure

Positive end-expiratory pressure (PEEP) is intended to minimize hypoxia and hypercarbia. The optimal PEEP decreases atelectasis without causing overdistension. The goal is to minimize lung stress and decrease pulmonary vascular resistance. The effects of PEEP on the heart depend on right and left ventricular function. PEEP decreases venous return in a normal heart, but this decrease is generally responsive to fluid administration. In left ventricular failure, PEEP decreases afterload since the positive pressure is applied to the chest and thus increases the gradient between the heart and the peripheral vasculature. This can increase cardiac output (CO) since failing left ventricles are afterload-dependent. In contrast, PEEP increases RV afterload in acute RV dysfunction, which can shift the septum to the left and cause a decrease in CO that does not respond to fluid administration.

Ideally, tidal volume and PEEP should be limited to avoid hypercapnia, acidosis, hypoxia, and hypoxic vasoconstriction. Achieving all of these goals may be difficult for severely ill patients. These difficulties may be especially prominent in patients with COVID-19 and severe respiratory failure.

Clinical Data

In a prospectively collected database including 1,997 patients hospitalized in our institution for COVID-19 pneumonia from March 2020 to March 2021, 368 had shock requiring vasopressors. Of these, 327 had echocardiography to assess ventricular function and stroke volume based on clinical indications. Left ventricular ejection fraction and RVFAC (RV fractional area change) were measured; 187 patients had evaluable data on all parameters. Patients were divided into groups with low or preserved RVFAC (cutoff ≤ 35%) and low or normal cardiac index (cutoff ≤ 2.2 L/min/m^2).

The mean right ventricular systolic pressure (RVSP) was 38.8 ± 12.2 mm Hg, and the mean PEEP was 11.0 ± 3.7 cm H_2O. RVSP was higher in patients with low RVFAC than normal RVFAC regardless of cardiac index (CI) (40.5 ± 1.4 mm Hg versus 37.4 ± 1.1 mm Hg, respectively; P = .037). PEEP was higher in patients with low CI than normal CI regardless of RVFAC (11.9 ± 0.4 cm H_2O versus 10.2 ± 0.3 cm H_2O, respectively; P = .037). Hospital mortality was 80% in this group with COVID-19 pneumonia and shock and did not differ among the groups (P = .19).

RV contractile function correlated with RV pressure and not CI in this group, whereas CO correlated with PEEP and not contractile function. Although RV dysfunction has been associated with a worse prognosis, these results suggest a mechanism linked to afterload and pulmonary pathology rather than contractility. Low CI may be related less to impairment of RV contractile function than to right-sided filling influenced by positive pressure ventilation.

Conclusion

In conclusion, RV dysfunction is common in patients with severe COVID-19 and shock and appears to be driven by pulmonary insufficiency and positive pressure ventilation. Whether treatment of RV dysfunction in COVID will improve outcome remains uncertain.

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