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2022 Symposium Presentation

COVID-19 Induced Right Ventricular Failure and Right Ventricular Assist Device Support

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Abstract

COVID-19, while primarily recognized for its pulmonary and systemic manifestations, afflicts the cardiovascular system through various abnormalities. Notably, right ventricular (RV) involvement leading to dysfunction and failure is a manifestation seen in up to 20% of severe COVID patients. RV severity correlates with overall COVID severity, serving as a prognostic marker. Data review reveals that RV failure was largely underdiagnosed, particularly early on in the pandemic. The therapy approach for RV failure in patients with COVID should focus on supporting overall RV perfusion pressure, maintaining sinus rhythm, optimizing RV loading conditions and contractility, and addressing anticoagulation and thrombus-related conditions. Beyond medical therapy, cardiac and pulmonary support should be utilized and introduced in a graded, stair-step approach of aggressiveness based on clinical need. This approach is best managed with a care team and defined protocols. Effective devices include right ventricular assistance devices (RVAD), Oxy-RVAD, veno-venous extracorporeal membrane oxygenation, and Impella (Abiomed) devices.

Keywords: RV failure, cytokines, thrombosis, coronavirus, mechanical circulatory support, ECMO

Background

Right ventricular (RV) involvement in COVID-19 was described early in the pandemic. The involvement was mainly attributed to pulmonary embolism and lung disease.¹ Myopericarditis was also described, but these early reports indicated that RV dilation was underdiagnosed.² Additional data showed that RV dilatation could be a mortality marker.³

Mechanism and Pathophysiology

Approximately 20% of patients that went to the hospital were admitted to the intensive care unit. Of those, 10% were supported with extracorporeal membrane oxygenation (ECMO); of the patients on ECMO, ~10% needed RV

support. Around 1-2% of hospitalized patients had direct RV failure. Newer data suggests an even higher degree of RV involvement in severely ill COVID patients, reported in up to 20% of cases. The causes of COVID-19-mediated RV failure include pulmonary parenchymal disease, pulmonary arterial disease, pulmonary arterial thrombosis, pulmonary emboli, RV myocardial disease, and load.

Considering the pathophysiology of RV failure, additional contributors to the disease process included infection, inflammation due to pulmonary infiltration, interstitial congestion/edema leading to pulmonary congestion, vasoconstriction, hypoxia, and pulmonary hypertension and RV afterload. RV failure could also be due to cytokine involvement, pulmonary emboli, or thrombosis.

Vascular thickening was seen in pulmonary arteries, and thrombosis was documented in large, medium, and small vessels.^{4,5} Data out of New York showed that platelet thrombi also play an important role in RV failure.⁶ Cytokines drive thrombosis, leading to increased fibrinogen and related platelet and white cell activation. COVID-19 infection results in a cytokine storm, an exuberant release of cytokines due to hyperactivation of the immune system.⁷ Within this setting, D-dimer is involved as both a driver of thrombosis and a novel marker of thrombosis and disease severity.^{8,9}

As to other cardiac manifestations involving the right heart, a septal shift was described, seen accompanying pulmonary hypertension in patients with COVID-19. Myocarditis was observed, with evident direct myocardial SARS-CoV-2 infiltration noted in several cases. Of note, direct significant myocardial viral infiltration was not the prototypic dominant feature of COVID-19.¹⁰

Clinical Strategies

The clinical presentation of RV failure in patients with COVID-19 consisted of general symptoms and signs consistent with RV failure. Patients presented with elevated central venous pressure, tricuspid regurgitation, pulmonary insufficiency, abdominal distention associated with ascites, peripheral edema, and, if increasingly severe hypotension, syncope, shock, and cardiac arrest. There were several interesting diagnostic indicators related to RV dilation on echocardiography, such as RV/LV ED area > 0.6; RV diameter > 42 mm (base); TAPSE < 17mm; RV FAC < 35%; RV EF < 45%; however, radial dysfunction was primarily unique to COVID patients.

Treatment

The treatment approach focused on 1) supporting overall RV perfusion pressure, 2) maintaining sinus rhythm, 3) optimizing RV loading conditions and contractility, and 4) addressing anticoagulation and thrombus-related conditions.¹¹

The initial recommendation for mechanical circulatory support was a staged approach using a single cannula right ventricular assistance device (RVAD), specifically Protek Duo (LivaNova), paired with a gas exchanger. The next step was support using veno-venous ECMO and an Impella (Abiomed) device.¹²⁻¹⁴ A surgical approach was recommended for special cases only. The downside to using ECMO is the increased risk of inflammation driven by foreign material. Using data from the Specialty Care database of 500 patients, the survival rate on ECMO was 40-45%.

The access point and insertion site are the next issues to consider in the staged approach.¹⁵ In selected cases, support can be added on the left side. Data kindly provided by Dr. A.

El Banyosy from Integris Medical Center in Oklahoma showed that in 87 patients with severe COVID who were on ECMO support, almost 10% (9/87) had severe RV dysfunction requiring support with an Oxy-RVAD. Half of these patients were successfully discharged. The Food and Drug Administration provided emergency use authorization for Impella RP to treat right heart failure in COVID-19 patients and Impella CP in tandem with ECMO for critically ill patients. Field data from Abiomed demonstrated increased use of Impella in patients with COVID-19; 10% (70/700) of patients were supported with ECMO and Impella.

Conclusion

Overall, COVID-19 RV failure was underdiagnosed. The mechanisms behind RV failure are multifactorial, involving thrombotic, embolic, proliferative, inflammatory, and loading pathogenic mechanisms. The initial treatment approach involved maintaining overall pressure, rhythm, optimized RV pre- and afterload, anticoagulation, and inotropes. A graded approach was recommended to avoid increased inflammation, hemolysis, and other complications if mechanical support was considered. Oxy-RVAD with Protek Duo was preferred in 95% of cases reviewed, followed by support with Impella. Understanding mechanisms and addressing all pathophysiologic components will improve our approach to RV failure in COVID. At this point, defining and systematizing an overall approach and optimal treatment strategies, all implemented in a tiered and team fashion, is the best approach for preventing and treating RV dysfunction and failure in COVID.

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