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Commentary

Cardiovascular Manifestations of COVID-19: Lessons Learned and Ongoing Challenges

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DESCRIPTION

The Coronavirus Disease 2019 (COVID-19) pandemic, caused by Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), has profoundly impact global health systems and revealed complex relationships between infectious disease and cardiovascular health. While initially recognized primarily as a respiratory illness, COVID-19 quickly emerged as a multisystem disease with significant cardiovascular implications. More than three years into the pandemic, our understanding of the cardiovascular manifestations of COVID-19 has evolved substantially, informing both acute management strategies and approaches to long-term follow-up of affected patients.

The spectrum of cardiovascular involvement in COVID-19 is remarkably diverse, ranging from direct myocardial injury to indirect effects mediated by systemic inflammation, thrombosis, and disruption of healthcare delivery. Acute cardiac injury, defined as elevation in cardiac troponin above the 99 percentile upper reference limit, has been reported in 8%-62% of hospitalized COVID-19 patients, with higher rates observed in those with severe disease requiring intensive care. This injury appears to result from multiple mechanisms, including direct viral invasion of cardiomyocytes *via* Angiotensin-Converting Enzyme 2 (ACE2) receptors, systemic inflammatory response with cytokine storm, microvascular dysfunction, hypoxemia, and stress-induced cardiomyopathy.

Myocarditis in COVID-19 has been a subject of considerable interest and evolving understanding. Early autopsy studies demonstrated viral particles in myocardial tissue and inflammatory infiltrates in some cases, supporting the plausibility of direct viral myocarditis. However, subsequent evidence suggests that classic viral myocarditis is relatively uncommon in COVID-19, with most cases of myocardial inflammation likely representing a hyperinflammatory or immune-mediated response rather than direct viral infection. Cardiac magnetic resonance imaging studies in patients recovered from COVID-19 initially reported alarmingly high rates of myocardial inflammation, raising concerns about long-term cardiovascular sequelae. However, these findings have been tempered by subsequent studies with appropriate control groups, suggesting more modest rates of persistent cardiac abnormalities.

Acute Coronary Syndromes (ACS) in the context of COVID-19 present diagnostic and therapeutic challenges. The pathophysiology of ACS in COVID-19 appears multifactorial, involving plaque rupture triggered by systemic inflammation, increased sympathetic stimulation, and a prothrombotic state characterized by endothelial dysfunction, platelet activation, and dysregulated coagulation. Additionally, demand ischemia may occur in the setting of increased myocardial oxygen demand and reduced supply due to hypoxemia. Management of these patients is complicated by diagnostic uncertainty, resource constraints during surge periods, and concerns about exposure risk during invasive procedures, necessitating modifications to standard ACS protocols in many centers during the height of the pandemic.

Thrombotic complications represent a hallmark feature of severe COVID-19, with implications for both venous and arterial systems. The increased incidence of venous thromboembolism, including pulmonary embolism, has been well-documented, with reported rates of 20%-30% in critically ill patients despite standard thromboprophylaxis. This heightened thrombotic risk appears to result from the interaction of multiple factors, including endothelial injury, platelet activation, complement activation, and dysregulated coagulation cascades characterized by elevated D-dimer, fibrinogen, and factor VIII levels. These observations led to trials of intensified anticoagulation strategies, with emerging evidence supporting intermediate or therapeutic-dose anticoagulation in selected hospitalized patients.

Heart failure may complicate the course of COVID-19 through various mechanisms, including exacerbation of pre-existing cardiac dysfunction, new-onset cardiomyopathy related to myocarditis or stress, right ventricular failure secondary to pulmonary disease or pulmonary embolism, and volume overload related to acute kidney injury or aggressive fluid resuscitation. The differentiation between cardiogenic and non-cardiogenic pulmonary edema presents a particular diagnostic

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challenge in COVID-19 patients with respiratory failure, as both may present with similar clinical and radiographic features. Point-of-care ultrasound has emerged as a valuable tool in this differentiation, allowing rapid assessment of cardiac function and extravascular lung water at the bedside.

CONCLUSION

Arrhythmias are common in COVID-19, occurring in up to 44% of patients admitted to intensive care units. While sinus tachycardia is most frequent, potentially reflecting fever, anxiety, or appropriate response to hypoxemia or hypovolemia, more malignant arrhythmias including atrial fibrillation, ventricular tachycardia, and ventricular fibrillation have been reported. These rhythm disturbances may result from direct myocardial injury, systemic inflammation, electrolyte abnormalities, hypoxemia, or adverse effects of therapeutic agents, including many repurposed medications initially used in COVID-19 treatment that have since been abandoned due to lack of efficacy and potential harm.