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PRACTICE | FIVE THINGS TO KNOW ABOUT ...

# Cardiac considerations in patients with COVID-19

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## 1 Cardiac injury is seen in about one-quarter of patients with coronavirus disease 2019 (COVID-19) who are admitted to hospital

Between 8% and 28% of inpatients have myocardial injury, troponin levels greater than the 99th centile of the upper limit of normal (ULN), from either indirect (e.g., owing to hypoxemia, sepsis or cytokine release) or direct myocardial damage.[1,2] Higher rates are seen in patients requiring intensive care.[1,2] Progressive elevation in troponins (Figure 1) and natriuretic peptides (NPs) have been shown to parallel disease acuity, with peak levels about 2–3 weeks from symptom onset.[2,3]

## 2 Cardiac biomarkers are useful for risk stratification

In patients admitted to hospital, periodic measurement of troponin and NP levels may identify patients at increased risk of developing left ventricular dysfunction or arrhythmias. If levels are elevated, consider monitoring the patient in a higher-acuity setting. Consider assessment using cardiac point-of-care ultrasonography if moderate elevation (> 3 times the ULN) or hemodynamic and/or electrical instability occur.[2]

## 3 Myocardial infarction (MI) may occur without obstructive coronary artery disease (CAD)

Patients may present with MI secondary to supply–demand mismatch or microvascular thrombosis. Myocarditis may mimic acute coronary syndrome, and these patients are at increased risk of cardiogenic shock.[2] For patients with refractory or recurrent chest pain, moderate elevation of troponin level or focal electrocardiogram changes, consider performing cardiac point-of-care ultrasonography to assess for left ventricular dysfunction and coronary computed tomography (CT) angiography to rule out CAD.[2,4]

## 4 Treatment should be directed at specific causes of myocardial injury

Manage heart failure, ST-elevated MI and left ventricular dysfunction in a patient with COVID-19 as per protocol using appropriate personal protective equipment, including use of guideline-directed medical therapy. Invasive hemodynamic monitoring should be limited to patients whose volume status cannot be determined clinically.[2] Evidence for management of fulminant myocarditis is scarce, and treatment decisions should be made by multidisciplinary teams.[1,2] Patients taking angiotensin-converting-enzyme inhibitors and angiotensin II receptor blockers should not stop therapy.[5]

## 5 Cardiac complications are associated with poor outcomes

Up to 40% of COVID-19 deaths may be at least partially attributed to heart failure.[2] Among patients admitted to hospital who develop heart failure, mortality rates may be as high as 60%.[3] Sustained ventricular tachycardia or ventricular fibrillation may occur in up to 17% of patients with cardiac injury and are more common among patients in intensive care.[2]

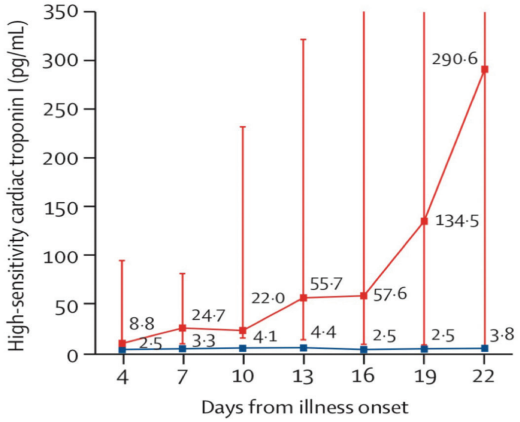


**Figure 1:** Troponin trajectories among hospitalized survivors (blue line) and nonsurvivors (red line) with coronavirus disease 2019. Reprinted from *The Lancet*, Vol. 395, Zhou F, Yu T, Du R, et al. Clinical course and risk factors for mortality of adult inpatients with COVID-19 in Wuhan, China: a retrospective cohort study, 1054-62, Copyright (2020), with permission from Elsevier.[3]

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