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# AMPATH LAB UPDATE

Dr Marita du Plessis, Chemical Pathologist

# Role of cardiac troponin testing in COVID-19

## Introduction

- Increased **high-sensitive cardiac troponin (hs-cTn) is a marker of myocardial injury**, **irrespective of the mechanism**, and **does not equate to acute myocardial infarction (MI).**
- An increase of hs-cTn may be due to **ischaemic** (e.g. myocardial infarction) or **non-ischaemic** (e.g. myocarditis) **causes** of myocardial injury[1].
- A rise and/or fall of hs-cTn alone is insufficient to make the **diagnosis of acute MI**, which should be based on **clinical judgement** using a **combination of hs-cTn, signs and symptoms, and ECG changes[2].**
- On review of reports from China, **elevated hs-cTnI/T** was observed in a considerable number of patients (12–28%) admitted with COVID-19. These patients were older and had more comorbidities, including hypertension, coronary artery disease (CAD) and diabetes.
- Patients with higher troponin levels were more likely to be **admitted to ICU** and showed **higher in-hospital mortality[3]**.

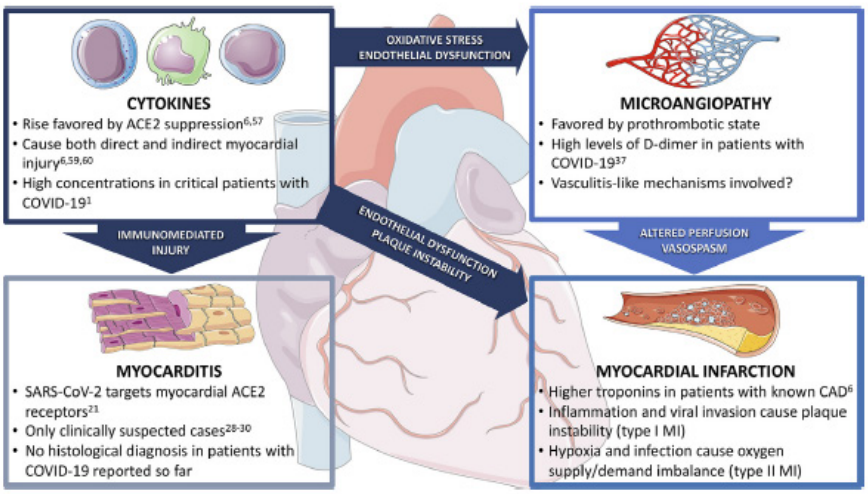
## Mechanisms of cardiac troponin elevation in COVID-19

The mechanisms explaining myocardial injury in patients with COVID-19 are not yet fully understood. Therefore, the proposed mechanisms are based on experience with the first SARS coronavirus. Direct (non-coronary) myocardial damage is probably the most likely cause.

The myocardial damage appears to be mediated mainly through binding of the SARS-CoV-2 virus to the angiotensin-converting enzyme 2 (ACE2) receptor mediating cellular entry and direct myocyte injury, as well as downregulation of ACE2 expression.

This causes angiotensin II upregulation and a subsequent angiotensin II/Angiotensin 1-7 imbalance, which contributes to endothelial dysfunction, cytokine storm, oxidative stress and coagulopathy, as well as indirect myocardial damage. The proposed mechanisms are summarised in Figure 1.

**Figure 1: Possible mechanisms explaining troponin elevation in patients with COVID-19**




*Journal of Cardiac Failure 2020, 26(6): 470–475*[3]

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## Potential use of cardiac troponins in COVID-19

- Patients with **pre-existing CAD** and those with **risk factors for cardiovascular disease (CVD)** are at **increased risk** of developing acute coronary syndrome during acute infection.
- **Type 2 MI** may be caused by an imbalance of oxygen supply and demand, secondary to hypoxic respiratory failure and the associated fever, tachycardia and endocrine dysregulation of COVID-19. This might further lead to unmasking of stable underlying CAD.
- COVID-19 can also precipitate **Type 1 MI**, caused by plaque rupture and thrombus formation[3].
- Although the initial recommendation from the American College of Cardiology was to limit troponin testing to patients with pre-existing CAD[2], several reports have shown the benefit of using hs-cTn testing to identify myocardial injury and potentially **risk stratify** patients with COVID-19 on admission[1].
- A study done at Wuhan University on 461 patients, showed that patients with cardiac injury required more non-invasive (46.3% vs 3.9%) and invasive **mechanical ventilation** (22.0% vs 4.2%)[4].
- **Complications** such as acute respiratory distress syndrome (ARDS), acute kidney injury and coagulation disorders were also more common in patients with elevated hs-cTn levels[4].
- In a cohort of 191 COVID-19 patients, the univariable odds ratio for **death** when hs-cTnI levels were above the 99th percentile upper reference limit was 80.1 (95% confidence interval [CI] 10.3 to 620.4), compared to an odds ratio of 20.04 (95% CI 6.52-61.56) for a D-dimer level >1 mg/l. Unfortunately, hs-cTnI was not included in multivariable analysis[5].
- **Determination of hs-cTn should be considered "an ally and a crucial diagnostic and prognostic aid" during the COVID-19 pandemic** according to cardiologists in the United Kingdom. Further testing following an elevated level should however be carefully considered, and the hs-cTn result and level of increase interpreted in combination with clinical and ECG findings[1].

### References


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