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Acute Cardiac Injury in Patients with COVID-19

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Abstract

COVID-19's cardiac problems have the potential to be fatal. Myocardial damage is a multifactorial event in the setting of COVID-19 that has piqued researchers' curiosity. A systematic review of the literature was conducted, followed by a meta-analysis. The databases MEDLINE and EMBASE were combed. The publications were reviewed by two independent reviewers for the outcome myocardial damage. According to recent studies, myocardial damage can occur in patients with COVID-19 with a frequency of 16%. As the pandemic progresses, more study is needed to update these findings and explain the consequences of cardiac injury in the setting of this virus.

Keywords: COVID-19 • Myocardial injury • Acute myocardial infarction • Myocarditis • Troponin

Introduction

The link between Cardio Vascular Disease (CVD) and negative outcomes in COVID-19 patients has been well documented. In severe infections, troponin increases, which indicate myocardial injury, are common. Patients with COVID-19 have a high rate of abnormal troponin results, especially when high-sensitivity troponin is employed. Acute myocardial damage was found in 12% of the first 41 COVID-19 patients, and four of these patients required intensive care therapy, suggesting the higher severity associated with this presentation. The goal of this study was to see how often acute myocardial damage is in COVID-19 individuals. Atherosclerotic plaque rupture (type I acute myocardial infarction), a supply/demand mismatch in the context of severe infection and sepsis (type II myocardial infarction), or direct myocardial damage are all possible mechanisms of myocardial injury in COVID-19. Since the beginning of the pandemic, abnormal troponin readings have been recorded often in individuals with COVID-19, especially when high-sensitivity troponin is used. Five (12%) of the initial 41 Chinese patients with COVID-19 suffered acute myocardial infarction, and four out of five required critical care. The implications of aberrant troponin levels differ, and high troponin in COVID-19 may not always mean that a myocardial infarction or myocarditis has occurred. Nonetheless, the current study finds that myocardial injury can occur in 16% of COVID-19 patients who are hospitalised, a high enough rate to make this complication a priority in clinical management.

Discussion

In COVID-19, cardiac damage is a major consequence that should be considered in patients who experience clinical deterioration and predicted in those with risk factors such as past cardiovascular disease. It is thus desirable to have a good understanding of the prevalence, predisposing factors, and features of cardiac damage in COVID-19. Different test sensitivity and abnormality cut-off values may make it difficult to interpret the results. It's worth noting that the timing of the troponin assays varied between trials, as did the severity of the patients. Atherosclerotic plaque rupture (type I acute myocardial infarction), a supply/demand mismatch in the context of severe infection and sepsis (type II myocardial infarction), or direct myocardial damage are all possible mechanisms of myocardial injury in COVID-19.

Conclusion

As a result, abnormal troponin levels should be examined in the context of the patient's overall clinical condition, rather than as a stand-alone reason for tests like cardiac catheterization or therapeutic measures like the start of antiplatelet therapy. The present rate at which data and new papers are released makes it particularly difficult to keep up with the rapidly changing research. A meta-analysis, on the other hand, would be beneficial for estimating the frequency of COVID-19 adverse events, which, in the case of myocardial damage, is a particularly serious complication to consider.

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