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Short Communication

Clinical Significance of Cardiac Troponin Measurement in COVID-19 Infection

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DESCRIPTION

The severe acute respiratory syndrome COVID-19 is brought on by the new coronavirus SARS-CoV-2. Due to its rapid human-to-human transmission, COVID-19 is currently a global epidemic and a public health emergency. The consequences are extensive, including forced social exclusion and isolation, negative effects on personal physical activity and mental welfare, adverse effects on young people schooling, and negative economic implications on businesses. Although the majority of COVID-19 patients have mild to moderate symptoms, those who experience rapid illness development are more likely to die. Comorbid cardiovascular disease is clearly linked to greater severity and higher mortality as more is understood about this unique disease. Many individuals who test positive for COVID-19 exhibit elevated cardiac troponin levels, which complicate clinical interpretation. SARS-CoV-2, formerly known as 2019-nCov, is the most recent virus that causes COVID-19, a severe acute respiratory illness. The World Health Organization (WHO) currently views SARS-CoV-2 as a pandemic human viral illness following the initial outbreak in Wuhan, China, in late 2019. Although it has been speculated that the virus was created through genetic engineering, SARS-CoV-2 most likely emerged through natural selection in an animal source. The current SARS-CoV-2 virus exhibits genetic similarities to coronaviruses that are comparable to SARS-CoV in bats. However, there is no proof of direct bat-to-human transfer, pointing to a possible involvement of an intermediary animal host[1]. This follows comparable zoonotic infection pathways used by other coronaviruses to infect humans. People with underlying cardiovascular disorders linked to hypertension, diabetes, coronary artery disease, or cerebral vascular disease are more likely to develop COVID-19's most severe form of the disease, which has the greatest fatality rate. COVID-19 is characterized by pneumonia. Cardiac consequences can include sudden heart failure, Acute Coronary Syndrome (ACS), and arrhythmia. All of these conditions have a poor prognosis and are linked to elevations in cTn12, particularly when high-sensitivity immunoassays are used. People with acute infectious respiratory diseases frequently have elevated cTn levels, and these levels rise in correlation with the severity of the infection. The development of SARS-CoV-2 assays has been accelerated by the *in vitro* diagnostics sector. Patients can receive an early diagnosis of heart disease by having cardiac troponin, the gold standard marker of myocardial damage. The most recent worldwide recommendations suggest cardiac tests for cardiac Troponin I (hs-cTnI) and T (hs-cTnT), which is particularly sensitive for detecting myocardial damage and acute myocardial infarction (MI). As a result, the death rate for patients with underlying cardiovascular disease is approximately three times higher. Also people with a history of cardiovascular disease are more likely to have greater cardiac troponins as biomarkers than infected individuals, particularly those with severe disease, rendering them more vulnerable to the heart damage brought on by SARS-2-CoV. The use of biomarkers in decision-making is crucial for the effective distribution of resources. There are three subunits in cardiac troponins (Troponin C, Troponin T, and Troponin I). Troponin C, also known as TN-C or TnC, is a calcium-binding protein that is produced in skeletal and cardiac muscle from the TNNC1 gene. The inhibitory component known as troponin I prevent myosin from interacting with actin. Troponin I exists in three distinct isoforms, the slow and rapid skeletal isoforms, and the cardiac-specific isoform. The biggest subunit, Troponin T (36 kDa, 288 amino acids), is what causes the heart to contract. Troponin T can be separated into many functional sections, including the N-terminus, also referred to as the T1 region (interacts with tropomyosin), and the C-terminus, often referred to as the T2 region. Adiponectin and soluble Inter Cellular Adhesion Molecule (sICAM)-1 were considerably elevated in blood and saliva after acute myocardial infraction in a patient with high-cardiac troponin levels for prevention of this disease. Viral replication in the heart muscle can set off a chain reaction of inflammatory reactions that eventually result in cardiac necrosis and fibrosis. Cardiac biomarkers can be used as a metric of a worsening clinical scenario or as an indicator of improving response due to cardio-protective intervention due to the rapid development of disease progression around the world and with a better understanding of the mechanisms of cardiovascular complications in COVID-19 [2-4].

CONCLUSION

A better tool to lower COVID-19 death rates based on Coronary Artery Disease (CAD) involvement may be provided by the

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combined use of many biomarkers, which may provide complementing prognostic information. Few investigations, however, have examined the function and levels of very sensitive cardiac troponins in COVID-19 patients. Understanding of clinical and diagnostic presentations is improving as more cases are found. In patients with COVID-19 disease, cardiac biomarkers, in particular cTn and natriuretic peptides are frequently increased. Elevation of cTn is linked to illness severity and a bad prognosis, just like with many other non-ACS disorders. Cardiac troponins have gradually grown in clinical significance for cardiovascular disease patient diagnosis, prognosis, and treatment due to the situation of COVID-19.

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