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Perspective

The COVID-19 Pandemic Malefic Impact on Acute Myocardial Infarction

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DESCRIPTION

Myocardial Infarction (MI), sometimes known as "heart attack," occurs when blood flow to an area of the myocardium is reduced or completely stopped. Myocardial infarction can be "silent," resulting in hemodynamic decline and sudden death, or it might be a catastrophic occurrence. Non-ST Segment Elevation Myocardial Infarction (NSTEMI), coronary spasm, or unstable angina is all examples of ST Segment Elevation Myocardial Infarction (STEMI). The coronavirus virus outbreak of 2019 (COVID-19) has fast expanded over the globe. COVID-19 is linked to substantial direct and indirect cardiovascular repercussions in addition to respiratory difficulties, with the latter being more important, especially in the case of time-dependent cardiovascular emergencies. A recent study demonstrated that during the COVID-19 pandemic, hospital admissions for Acute Myocardial Infarction (AMI) dropped dramatically worldwide, owing to patients' refusal to activate emergency medical systems because hospitals were perceived as dangerous places in terms of infection risk. Furthermore, patients with AMI had a considerably greater in-hospital mortality rate during the COVID-19 pandemic compared to those admitted prior to COVID-19, possibly due to late arrival at the hospital. Finally, no agreement has been achieved on the best appropriate healthcare management pathway for AMI, and unified guidance on how to address AMI patients during the pandemic is still needed.

During all three COVID-19 progressive stages, the cardiovascular system appears as both a primary target and the most important secondary co-morbidity component. The heart itself may be a direct target for SARS-CoV-2 virus infection, according to mounting data. Prior studies into the cardiovascular effects of viral respiratory infections during influenza epidemics reported a six-fold increased risk of acute myocardial infarction within seven days after infection, owing to enhanced prothrombotic activity and intracoronary thrombotic events. Hypotension and tachycardia will exacerbate a sick heart's metabolic requirement. Overstated experience the ill effects, as seen by significantly elevated circulating levels of archetypal inflammatory indicators such as IL-6, IL-2, TNF alpha, MCP-1, or CRP, has been shown to contribute to cardiac damage in the absence of hypoxemia.Indeed, in retrospective clinical series of COVID-19 patients hospitalized in countries, some of these indicators were linked to high mortality, indicating possible catastrophic bystander effects on other organs, including the heart.

Increased inflammatory markers do correlate with electrocardiographic abnormalities and indicators of cardiac injury, implying collateral harm to the heart. Finally, elevated cardiac biomarkers indicating cardiac involvement are not only common in COVID-19, but they are also linked to a much worse clinical outcome. In a severely ill cohort hospitalized in multiple nations, myocardial injury and heart failure were responsible for nearly 40% of deaths. The mortality risk linked with acute cardiac injury was significantly higher than age, chronic pulmonary illness, or prior history of cardiovascular disease, according to Cox regression models. In addition to the severe acute respiratory distress syndrome, both direct and indirect mechanisms of cardiovascular injury are anticipated to play a key part in the negative consequences of SARS-CoV-2 infection (ARDS).

The COVID-19 epidemic caught the world's healthcare system off guard, diverting physicians' focus away from the treatment of cardiovascular diseases, particularly time-sensitive crises, with serious consequences for the efficacy of life-saving treatments and patient prognosis. Following the first shock, physicians understood that even during big unanticipated events, rapid management of cardiac emergencies with proper standards of care should be ensured. This can be accomplished by swiftly implementing remedies to this unique and severe emergency, with the goal of preventing widespread and long-term health and societal consequences. Health authorities should, in particular, create protocols that can respond to the index emergency while also ensuring the optimum treatment plan for AMI, based on rapid changes in the hub and spoke interplay.

During the COVID-19 pandemic, delays in therapy delivery were also a source of major worry, limiting the effectiveness of life-saving AMI medications. Because of their concern of COVID-19, patients have been hesitant to attend to the hospital, with many patients with AMI not seeking care at all or just late in the course of the acute episode. This has contributed to the mortality toll rising above the levels directly linked to SARS-CoV-2 infection. Although many problems remain unanswered and more evidence is needed, we believe that scientific societies, health authorities, and the public media should make everyeffort to persuade patients not to delay life-saving therapies, especially during dynamic crises.

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