LETTER TO THE EDITOR

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ACUTE CORONARY SYNDROME DUE TO CORONAVIRUS DISEASE (COVID-19) IN A HEALTHY MALE

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Dear Editor,

Coronavirus disease 2019 (COVID-19), caused by SARS-CoV-2 (severe acute respiratory syndrome coronavirus type 2) often manifest with respiratory symptoms such as cough, fever and difficulty in breathing.[1] The SARS-CoV-2 has also been shown to present with pathology related to cardiovascular system. The spectrum of cardiovascular involvement in COVID-19 patients is highly variable with cardiac injury as a common feature of the disease process.[2] Patients with cardiac risk factors and established cardiovascular disease (CVD) are more susceptible to COVID-19. They are also at an additional risk of severe disease with worse clinical outcomes.[3] Pathophysiologic basis of cardiovascular injury in COVID-19 include direct myocardial injury due to impaired hemodynamic stability and hypoxemia, inflammatory myocarditis, stress cardiomyopathy, microvascular dysfunction or thrombosis due to hypercoagulability, or systemic inflammation (cytokine storm), which may also destabilize coronary artery plaques.[2,3] Severe COVID-19 is associated with significant increase in cardiac output due to fever, increase metabolic demands or hypoxia due to lung disease.[4] Patients with viral Pneumonia and influenza infections are at six-fold risk of acute coronary syndrome.[5] The risk of acute coronary syndrome (ACS) is even more profound in SARS-CoV-2 infection due to disproportionately increased hypercoagulability that could potentially lead to thrombotically mediated damage.[6] However, it is often difficult to distinguish the presentation of atherosclerotic plaque-rupture myocardial infarction (MI) from myonecrosis due to supply–demand mismatch (type 2 MI) in the setting of severe hypoxia, hemodynamic instability and myocarditis due to SARS-CoV-2 viral infection.[7]

A 40-year-old commercial driver presented at Federal Medical Centre Nguru, a tertiary health facility in northeastern Nigeria, on the 13th September 2020, with two hours history of sudden severe and sharp left sided chest pain and heaviness radiating to the throat and jaw region. He had three episodes of non-projectile vomiting and excessive sweating. There was no associated fever, cough or difficulty in breathing. At presentation, he looked anxious, had diaphoresis, tachypneic with respiratory rate of 26 cycles/minute, capillary oxygen saturation at ambient air was 98%, had tachycardia, pulse rate 106 beats/minute and blood pressure of 120/80mmHg. His electrocardiography (ECG) tracing showed pathological Q wave with extensive ST segment elevation in V1-V6 and poor R wave progression. The ECG features persisted on serial ECG on the 6th day. The chest radiography (CXR-PA), done after 24 hours showed normal heart size with upper lobe diversion of blood vessels. Fasting lipid profile were within normal limits. HIV screening test, Hepatitis B surface Antigen and Hepatitis C virus antibodies were non-reactive. Nasopharyngeal and nasal swab for COVID-19 tested positive for SARS-CoV-2 by real time - polymerase chain reaction (RT-PCR). Patient was managed as a case of acute coronary syndrome due to possibly COVID-19. Patient responded to the management of acute coronary syndrome and the cocktail management for COVID-19 that included ZnSO4, Vitamin C and Lopinavir/Ritonavir. Patients clinical condition improved remarkably and was discharged after 12 days on admission.

Clinicians should be mindful of the extrapulmonary presentation of COVID-19. It may involve the cardiovascular system inform of myocardial infarction even in the absence of cardiac risk factors or cardiovascular disease. Patients with complaints suggestive of ACS should be thoroughly evaluated for possible cause, while ensuring standard and transmission-based precautions in the COVID-19 pandemic era.

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