- Heart diseases / Virus

'COVID 19 - A POISON FOR THE HEART' PROF DR. M WALI

Medgate Today · 15 Sep 2021 · 30

Coronavirus Disease (COVID-19) is an infectious disease caused by Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-COV-2) virus. It's already second year since World is fighting with COVID-19 caused pandemic. COVID-19 affects significantly respiratory and cardiovascular systems. First, those COVID-19 patients with preexisting cardiovascular disease have an increased risk of severe disease and death. Mortality from COVID-19 is strongly associated with male sex, advanced age, presence of hypertension, diabetes mellitus, cardiovascular diseases and cerebrovascular diseases, as well as complications of acute cardiac injury, cardiomyopathy, and heart failure. The coexistence of coronary heart disease and the myocardial injury was associated with the highest mortality rate (10.5%). Second, therapies under investigation for COVID-19 may have cardiovascular side effects like various arrhythmias. Third, COVID-19 is associated with multiple direct and indirect cardiovascular complications. Associated with a high inflammatory burden related to cytokine release, COVID-19 can induce vascular inflammation, acute myocardial injury, myocarditis, arrhythmias, venous thromboembolism, metabolic syndrome and Kawasaki disease. Understanding the effects of COVID-19 on the cardiovascular system is essential for providing comprehensive medical care for cardiac and/or COVID-19 patients. (fig.1)
Furthermore, SARS-COV-2 virus uses angiotensin-converting enzyme 2 (ACE2) as the receptor to enter the host cell. The cardiovascular disorders share an underlying renin-angiotensin system (Ras)-related pathophysiology and pharmacologic RAS inhibitors both increase ACE2 levels, which may increase the entry of SARS-COV-2 into the lungs and heart. Thus, the infection may have a direct impact on cardiovascular diseases. The detailed cardiac events of comorbidity, complications and relevant mortality are tabulated below (fig.2).

Myocarditis

Acute cardiac injury determined by elevated high-sensitivity troponin levels is commonly observed in severe cases. In a study of 120 Sars-cov-2-infected patients, elevated levels of

N-terminal pro-brain natriuretic peptides (NTPROBNP) (27.5%) and cardiac troponin T (TNT) (10%) were associated with dramatically increased plasma IL 6 levels. Patients with high TNT levels also had higher inflammatory biomarkers, such as leukocytosis, lymphopenia, d-dimer, CRP, and procalcitonin. Myocardial injury is an important prognostic factor in COVID-19 and is strongly associated with mortality.

Double roles of ACE2: "Skeleton in the closet"?

Normally, angiotensin I is converted to angiotensin II via ACE, which could be inhibited by ACE inhibitors. ACE2 antagonizes the activation of the classical RAS and protects against organ damage, especially in patients with hypertension, diabetes, and cardiovascular disease. The ACE2 converts angiotensin I to angiotensin 1–9 and angiotensin II to angiotensin 1–7, which have anti-inflammatory effects. The proinflammatory effects, vasoconstriction, and the genesis of atherosclerosis of angiotensin II are mediated through angiotensin type 1 (AT1) receptor, which is attenuated by AT1 receptor blockers (ARBS). Angiotensin II binding to the AT1 receptor allows ACE2 degradation. ARBS block angiotensin II binding to the AT1 receptor and prevent ACE2 degradation. Chronic use of ARBS would increase ACE2 expression and thus promote anti-inflammatory benefits by conversion angiotensin II to angiotensin 1–7. Overall, by this pathway of ARBS or ACEI, the promotion of ACE2 benefits lung from anti-inflammation. The dual