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A BRIEF REVIEW ON CORONA VIRUS (COVID 19) AND CARDIOVASCULAR DISEASE

Pharmacy

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ABSTRACT

Coronavirus disease 2019 (COVID-19) could be a international pandemic impacting nearly a hundred and seventy countries/regions. COVID-19 is caused by the Severe Acute respiratory Syndrome Coronavirus-2 (SARS-CoV-2), that invades cells through the angiotensin converting enzyme 2 (ACE2) receptor. Among those with COVID-19, there's a better prevalence of disorder and over seven-membered of patients suffer heart muscle injury from the infection (22% of the critically ill). Despite ACE2 serving because the portal for infection, the role of ACE inhibitors or angiotensin receptor blockers needs additional investigation. COVID-19 poses a challenge for heart transplantation, impacting donor choice, immunological disorder, and post-transplant management. Infection with the novel coronavirus, SARS-CoV-2, produces a clinical syndrome referred to as COVID-19. once severe, COVID-19 could be a general sickness characterised by hyperinflammation, cytokine storm and elevations of cardiac injury biomarkers. Here we tend to review what's proverbial concerning the pathophysiology of COVID-19, its cardiovascular manifestations, and rising therapeutic prospects. a considerable minority of patients hospitalized develop Associate in Nursing Acute COVID-19 vessel Syndrome (ACovCS) which will manifest with a spread of clinical displays, however usually presents as Associate in Nursing acute cardiac injury with cardiomyopathy, ventricular arrhythmias and hemodynamic instability within the absence of preventative arteria disease.

KEYWORDS

Coronavirus, cardiovascular disease, angiotensin converting enzyme 2, myocardial injury

INTRODUCTION:

In December 2019, an outbreak of pneumonia caused by a novel coronavirus occurred in Wuhan, Hubei province, and has spread rapidly throughout China, with an ongoing risk of a pandemic1. After virus identification and isolation, the pathogen for this pneumonia was originally called 2019 novel corona virus (2019- nCoV)2 but has subsequently been officially named severe acute respiratory syndrome coronavirus 2 (SARS- CoV-2) by the WHO. On 30 January 2020, the WHO declared the outbreak of SARS- CoV-2 a Public Health Emergency of International Concern. While having widespread effects on various systems, COVID-2019 interacts with cardiovascular system at various levels and has direct and indirect damage. Similarly, preexisting cardiovascular disease predisposes to serious COVID-2019 infection with In this document, we focus on a prominent myocarditis-like syndrome involving acute myocardial injury often associated with reduced left ventricular ejection fraction in the absence of obstructive coronary artery disease. This syndrome can be complicated by cardiac arrhythmias and/or clinical heart failure with or without associated hemodynamic instability including shock increased morbidity and mortality. Cardiovascular diseases and/or risk factors are highly prevalent in severe forms of coronavirus 2019 (COVID-19) infection.Clinical manifestations are principally respiratory, but some patients may also show cardiovascular complications. Early reports during the coronavirus disease 2019 (COVID-19) pandemic emphasized theoretical concerns that the continued use of medications that block the renin-angiotensin-aldosterone system (RAAS), including angiotensin-converting enzyme (ACE) inhibitors and angiotensin II receptor blockers (ARBs), may influence disease severity and mortality. Respiratory involvement, presenting as mild flulike illness to potentially lethal acute respiratory distress syndrome or fulminant pneumonia, is the dominant clinical manifestation of COVID-19. However, much like any other respiratory tract infection, preexisting cardiovascular disease (CVD) and CV risk factors enhance vulnerability to COVID-19. Further, COVID-19 can worsen underlying CVD and even precipitate de novo cardiac complications. This review is aimed at providing overview of various CV manifestations in patients presenting with COVID-19. The impact of pre-existing CVD and new onset cardiac complications on clinical outcomes in these patients is also discussed. Since our understanding on this subject is only evolving at this stage, the information contained in the subsequent text is based mainly on the limited early experience with COVID-19 and learnings from the previous coronavirus illnesses, namely SARS and Middle-East Respiratory Syndrome (MERS).

COVID-19 in Patients with Cardiovascular Disease

Cardiovascular disease was a common comorbidity in patients with COVID-19 predecessors SARS and MERS. In SARS, the prevalence of DM and CVD was 11% and 8% respectively, and the presence of either comorbidity increased the risk of death twelvefold. DM and HTN were prevalent in about 50% of cases of MERS, while CVD was present in approximately 30% of patients.The increased presence of cardiovascular comorbidities holds true for COVID-19 as well, most notably among those with more severe disease. In one cohort of 191 patients from Wuhan, China, any comorbidity was present in 48% (67% of non-survivors), HTN was present in 30% (48% of non-survivors), DM in 19% (31% of non-survivors), and CVD in 8% (13% of non-survivors). In a cohort of 138 hospitalized patients with COVID-19, comorbidities were similarly prevalent (46% overall, 72% in patients requiring an ICU), as were cardiovascular comorbidities: HTN in 31% (58% in patients requiring an ICU), CVD in 15% (25% in patients requiring an ICU), and DM in 10% (22% in patients requiring an ICU). Analysis of an outpatient and inpatient cohort of 1,099 patients with COVID-19 reported that 24% had any comorbidity (58% among those with intubation or death), with 15% having HTN (36% among those with intubation or death), 7.4% with DM (27% among those with intubation or death), and 2.5% with coronary heart disease (9% among those with intubation or death).Data from the National Health Commission (NHC) of China demonstrated that 35% of patients diagnosed with COVID-19 had HTN and 17% had coronary heart disease. Lastly, a recent meta-analysis of eight studies from China including 46,248 infected patients showed the most prevalent comorbidities were HTN (17±7%, 95% CI 14-22%) and DM (8±6%, 95% CI 6-11%), followed by cardiovascular diseases (5±4%, 95% CI 4-7%). The mechanism of these associations remains unclear at this time.

COVID-19 and Myocardial Injury

Myocardial injury, proven by elevated cardiac biomarkers, was recognized among early cases in China. within the same study of 138 hospitalized patients with COVID-19 in Wuhan China, cardiac injury (elevated high sensitivity Troponin I [hs-cTnI] or new cardiogram or echocardiographic abnormalities) was present in 7.2% of patients overall, and 22% that needed unit care. The report from the NHC of China rumored that just about 12-tone music of patients while not notable CVD had elevated troponin levels or cardiac arrest throughout the hospitalization. Reports recommend that the middle East respiratory syndrome- connected coronavirus (MERS- CoV) will cause acute myocardial inflammation and heart failure. SARS- CoV-2

and MERS- CoV have similar pathogenicity, and also the cardiac muscle injury caused by infection with these viruses without doubt will increase the issue and complexness of patient treatment. cardiac muscle injury related to the SARS- CoV-2 occurred in five of the primary 41 patients diagnosed with COVID-19 in Wuhan, that principally manifested as a rise in high- sensitivity viscus troponin I (hs- cTnI) levels (>28 pg/ml).

COVID-19 and Hypertension

Early reports from the outbreak in Wuhan, China indicated that a large proportion of patients with severe COVID-19 had hypertension, raising the concern that hypertension was a risk factor for more-severe disease. Similar data have emerged from preliminary analyses from Italy and the United States.6, 7 However, in all of these reports, patients with severe COVID-19 were older – age 65 and above – making it challenging to separate the impact of age from comorbid conditions, such as hypertension. Indeed, the proportions of children and adolescents in these early reports were less than two percent. These early reports have been descriptive case series, describing one-way associations without employing appropriate epidemiological or statistical methods to estimate the independent association of hypertension with COVID-19 outcomes. It is well known that hypertension prevalence, and thus antihypertensive medication use, increases with age. Until the results of more-sophisticated analyses that account for age, comorbid conditions, baseline medication use, and other potential confounding factors become available, one cannot conclude that hypertension is an independent risk factor for SARS-CoV-2 infection or more-severe COVID-19. There has been additional speculation that ACE inhibitors and ARBs may also increase the risk of SARS-CoV-2 infection and COVID-19. Like the original SARS-CoV described in 2003, data suggest that SARS-CoV-2 binds to ACE2 to gain entry to host cells on the respiratory tract epithelium. As select animal studies have demonstrated increased ACE2 expression with ACE inhibitor or ARB administration, a potential mechanistic link between SARS-CoV-2 infection.

Chronic cardiovascular damage

A 12- year follow- up survey of 25 patients who recovered from SARS-CoV infection found that 68 had lipemia, 440 yards had circulatory system abnormalities and hour had glucose metabolism disorders. Metabolomics analysis unconcealed that lipid metabolism was dysregulated in patients with a history of SARS- CoV infection. In these patients, the bodily fluid concentrations of free fatty acids, lysophosphatidylcholine, lysophosphatidylethanolamine and phosphatidylglycerol were considerably enhanced compared with people while not a history of SARS- CoV infection. However, the mechanisms by that SARS- CoV infection ends up in disorders of lipid and aldohexose metabolism ar still unsure. as long as SARS- CoV-2 features a similar structure to SARS- CoV, this novel virus may additionally cause chronic injury to the circulatory system, and a spotlight ought to incline to cardiovascular protection throughout treatment for COVID-19.

The Role of Angiotensin-Converting Enzyme 2 in COVID-19

Angiotensin-converting enzyme 2 (ACE2) is a carboxypeptidase that converts angiotensin II into angiotensin. This enzyme is homologous to angiotensin-converting enzyme (ACE) but serves a counterbalancing role in the renin-angiotensin-aldosterone system (RAAS). Beyond its function in cardiovascular homeostasis, ACE2 is also a functional receptor and a portal of entry for both SARS-CoV and SARS-CoV-2. The reader is referred elsewhere for an extensive review of this crucial link in the pathogenesis and pathophysiology of COVID-19. ACE2 is expressed in multiple tissues, including lungs, heart, and kidneys. In animal models, ACE2 expression in the heart is an essential regulator of function, with ACE2 knockout mice developing severe left ventricular dysfunction. SARS-CoV infection appears to downregulate ACE2, which may contribute to myocardial dysfunction. Thus, the link between SARS-CoV and ACE2 provides one theoretical mechanism for cardiac dysfunction in COVID-19: ACE2 downregulation leads to cardiac dysfunction. In addition, the relationship between viral entry and ACE2 forms the basis for the controversy surrounding the use of RAAS antagonists, which increase ACE2 expression in animal studies and, therefore, can theoretically increase susceptibility to infection. But even the directionality of the effects is debated: higher ACE2 levels may be protective, by providing a reservoir of receptors to offset those lost in the course of infection. However, there is, as yet, no convincing, corollary data in humans relating to the virus of interest, SARS-CoV-2.

CONCLUSION:

COVID-19, caused by SARS-CoV-2, could be a international pandemic evolving in real time. vas comorbidities are common in patients with COVID-19 and such patients are at higher risk of morbidity and mortality. SARS- CoV-2 is believed to infect host cells through ACE2 to cause COVID-19, whereas additionally inflicting injury to the heart muscle, though the precise mechanisms are unsure. Patients with underlying CVD and SARS- CoV-2 infection have an adverse prognosis. Therefore, particular attention ought to run to cardiovascular protection throughout treatment for COVID-19. The COVID-19 pandemic has motivated an explosion of recent analysis, that is already providing key insights into the pathogenesis of the disease. yet, several queries stay unanswered. lymphocytopenia, hyperinflammation, and internal organ involvement are all distinguished options of the unwellness and have prognostic worth, however the mechanistic links among these phenomena are ill-defined . Similarly, despite the quickly growing variety of clinical trials, no definitive therapies (other than appurtenant care) are beginning to at this point. New therapeutic paradigms, however, square measure setting out to emerge, and with rigorous investigation can ultimately advance our understanding and treatment of the disease. Even when COVID-19 has become a far off memory, the lessons learned throughout this unsure time can likely inform the assessment and medicine of different syndromes of hyperinflammation affecting the heart and vasculature.

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