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A Review on Cardiovascular Disease effect in Corona Patients

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Abstract

Cardiovascular disease (CVD) is a hearts cardiovascular disease includes coronary artery such as angina heart attack cardiomyopathy co morbidities, hypertensive disease. Cardiovascular disease effect in covid-19 many patients with corona virus disease have underlying cardiovascular disease or develop acute cardiac injury during the large of the illness adequate underlying of the interplay between covid-19 (CV) disease is required for optimum management of these patients, cardiovascular disease is responsible of more number of death world-wide, the muscle and vessels of heart and blood transporting roods become vulnerable patients in most of the (CVD) the role of hypertension and cholesterols of different density triglycerides in induction and progression of cardiovascular disease, Besides this the patent biomarkers such as mono cytosine , fibrinogen, D-dimmer and thrombin/ anti thrombin interleukin, severe acute respiratory syndrome-corona virus (SARS-CoV-2) can manifest acutely and persist into convalescence and possibly beyond.

Coronavirus disease 2019 (COVID-19), caused by a strain of corona virus known as severe acute respiratory syndrome corona virus 2 (SARS-CoV-2), has become a global pandemic that has affected the lives of billions of individuals. Clinical studies have also reported an association between COVID-19 and cardiovascular disease. Pre-existing cardiovascular disease seems to be linked with worse outcomes and increased risk of death in patients with COVID-19, whereas COVID-19 itself can also induce myocardial injury, arrhythmia, acute coronary syndrome and venous thromboembolism. Potential drug–disease interactions affecting patients with COVID-19 and co morbid cardiovascular diseases are also becoming a serious concern.

Keywords: Corona, CVD, Patients

Introduction

Cardiovascular disease is a very common ,Cardiovascular disease (CVD) is a collective term designating all types of application effecting the blood circulatory system , including heart and vasculature , the cardiovascular system is node up of the heart and blood vessels, and defined as any serious abnormal condition of the heart or blood such as (arteries and veins) which respectively displaces and conveys the blood , this multifactorial disorder encompasses numerous congenital and acquired maladies (CVD) represent the leading non communicable causes of death in Europe (50% of all death;- 30%) of all death worlds wide. In 2008, 9 million people died of non-communicable disease.

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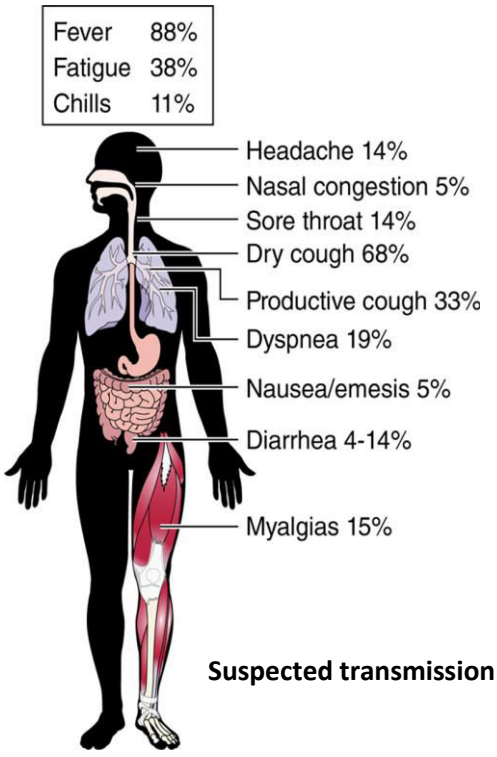
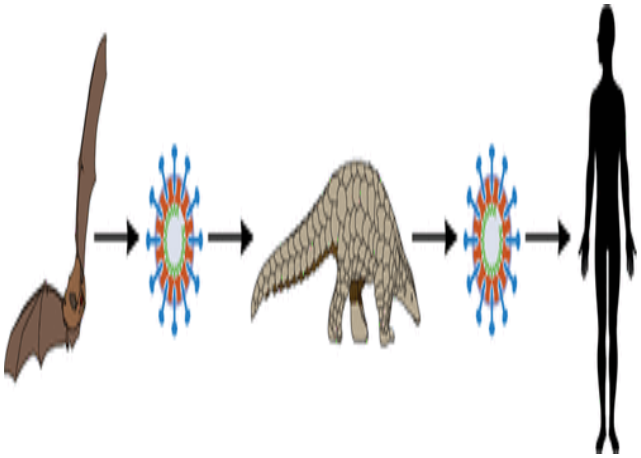
A program plan to develop heart disease in West Virginia was developed as a resource to help improve the cardiovascular health of west Virginians, preventing (CVD) and improving the health. CVD was common co morbidity in patients with COVID-19 predecessors SARS and MERS. In SARS, the prevalence of DM and CVD was 11% and 8%, respectively, and the presence of either co morbidity increased the risk of death 12-fold. DM and hypertension were prevalent in ≈50% of cases of MERS; CVD was present in ≈30% of patients. The increased presence of cardiovascular co morbidities holds true for COVID-19 as well, most notably among those with more severe disease. In 1 cohort of 191 patients from Wuhan, China, any co morbidity was present in 48% (67% of no survivors), hypertension in 30% (48% of nonsurvivors), DM in 19% (31% of no survivors), and CVD in 8% (13% of no survivors).



Early reports suggest the most common symptoms are fever (88%) and dry cough (67.7%), which are shared with many other viral syndromes. Conspicuously, rhino rhea (4.8%) and gastrointestinal symptoms (diarrhoea 4% to 14%, nausea or emesis 5%) appear to be infrequent in COVID-19. Reports from China demonstrate that a significant majority of patients (81%) had mild symptoms (no pneumonia or mild pneumonia) from COVID-19. Among patients with more substantial symptoms, 14% experienced severe symptoms(dyspnoea, respiratory rate ≥30/min, blood oxygen saturation ≤93%, partial pressure of arterial oxygen to fraction of inspired oxygen ratio <300, or lung infiltrates >50% within 24 to 48 hours) and 5% were critically ill (respiratory failure, septic shock, or multiple organ dysfunction or failure).

COVID-19 is caused by the severe acute respiratory syndrome corona virus 2 (SARS-CoV-2). This novel single-stranded enveloped RNA virus is the 7th known human corona virus. SARS-CoV-2 is unlike the corona viruses known to cause the common cold (229E, OC43, NL63, and HKU1), but similar to the zoon tic severe acute respiratory syndrome (SARS) corona virus (SARS-Cove) from 2002 and the Middle East respiratory syndrome (MERS) corona virus from 2012.



Suspected transmission pathway of severe acute respiratory syndrome corona virus 2 to humans

SARS-CoV-2 is believed to have originated in bats, similar to many other corona viruses, because it shares 89% to 96% nucleotide identity with bat corona viruses. Similar to SARS and MERS, it is believed that SARS-CoV-2 moved

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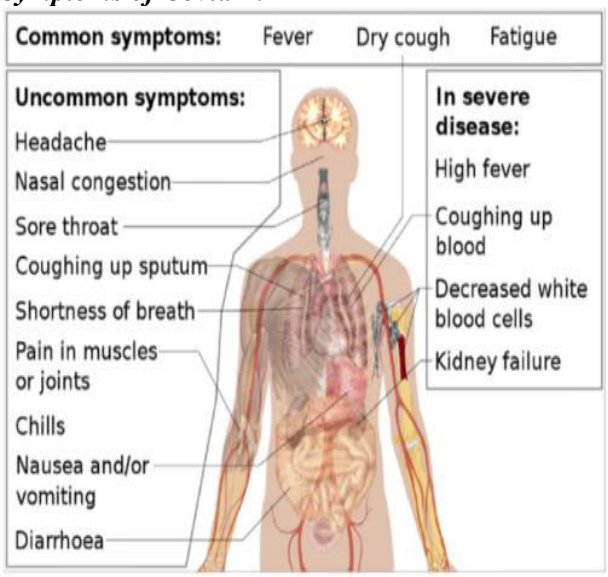
from bats to an intermediate host (possibly a Malayan pangolin, which shares 91% nucleotide identity) and then to humans.

Symptoms of Covid-19



Risk Factors

In order to test differences in cardiovascular health among the cohorts, risk factors for cardiovascular disease, evaluated for each subject at his base-line examination, were analyzed. MantelHaenszel and general-linear-model analysis of individual risk factors showed significant differences among the cohorts, with the 1970 cohort having a much healthier profile than either of the earlier cohorts

Table 6. Selected Risk Factors at Base-Line Examinations of Men Who Were 50 to 59 Years Old at Base Line.*

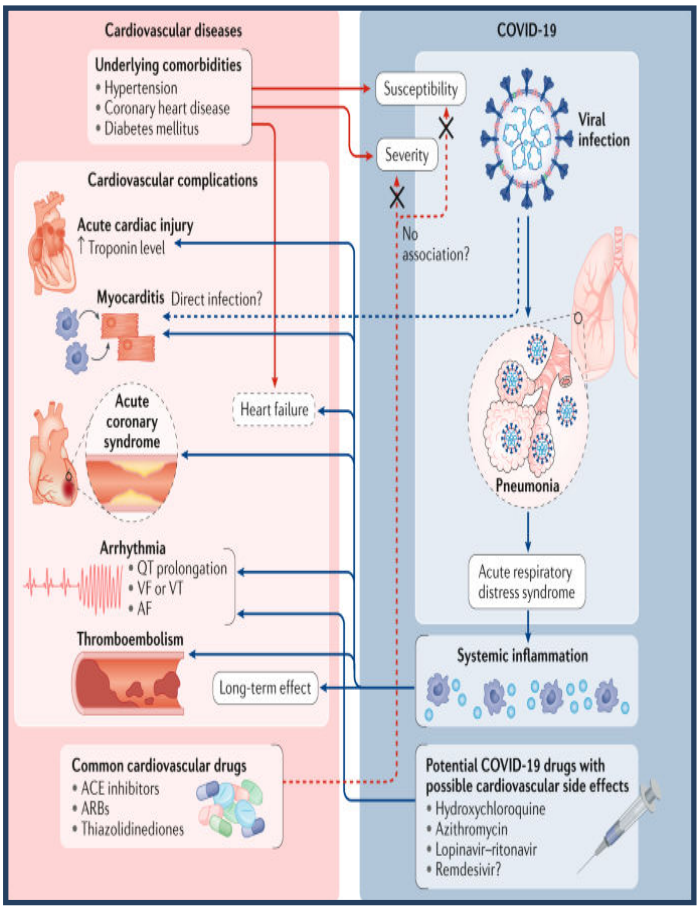
Risk Factor	Free of CVD at Base Line			Incident CVD within 10 Years of Base Line		
	1950	1960	1970	1950	1960	1970
Serum cholesterol (mg/dl)†	228±40	243±37	221±38‡	239±44	246±35	227±40§
Smokers (%)	56	52	34¶	64	60	57
Definite hypertension (%)‖	21	23	15¶	36	41	20¶
Use of antihypertensive medication (%)**	0	11	22¶	0	11	15
Systolic blood pressure (mm Hg)	139±25	137±21	135±19††	152±30	148±22	140±19§
Diastolic blood pressure (mm Hg)	85±13	86±12	84±10‡	91±15	91±12	85±11§
Metropolitan relative weight (%)‡‡	120±15	120±15	123±17‡	121±17	123±15	121±18

*The data are from the Framingham Heart Study, 1950 through 1979. Plus–minus values are means ±SD. CVD denotes cardiovascular disease.
†To convert values for serum cholesterol to millimoles per liter, multiply by 0.02586.
‡P<0.001 for the comparison of the three cohorts, by the general-linear-models procedure.
§P<0.01 for the comparison of the three cohorts, by the general-linear-models procedure.
¶P<0.05 for the comparison of the three cohorts, by the Mantel–Haenszel test.
‖Defined as systolic blood pressure >160 mm Hg, diastolic blood pressure >95 mm Hg, or both. See Gordon and Shurtleff.[18]
**Percentages refer to all subjects with hypertension (systolic blood pressure >140 mm Hg and diastolic blood pressure >90 mm Hg[18]).
††P<0.05 for the comparison of the three cohorts, by the general-linear-models procedure.
‡‡The measured body weight divided by the average U.S. age-specific and sex-specific weight for height, based on data from the Metropolitan Life Insurance Company.[18]

The mean serum cholesterol levels at base line decreased by 0.57 mmol per liter (22 mg per deciliter) from a high of 6.28 mol per liter (243 mg per deciliter) in the 1960 cohort to a low of 5.72 mmol per liter (221 mg per deciliter) in the 1970 cohort. An intermediate value of 5.90 mmol per liter (228 mg per deciliter) was observed in the 1950 cohort. The percentage of the men who smoked declined from 56 to 34 percent and the percentage with definite hypertension decreased from 21 to 15 percent between the 1950 and 1970 cohorts.

Bidirectional interaction between cardiovascular disease and Covid-19

Cardiovascular comorbidities such as hypertension and coronary artery disease are associated with high mortality in patients with corona virus disease 2019 (COVID-19). Drugs used to reduce cardiovascular risk such as angiotensin-converting enzyme (ACE) inhibitors and angiotensin II receptor blockers (ARBs) have numerous effects that might influence susceptibility to or the severity of COVID-19.



Furthermore, although the main presentation of COVID-19 is viral pneumonia, COVID-19 can

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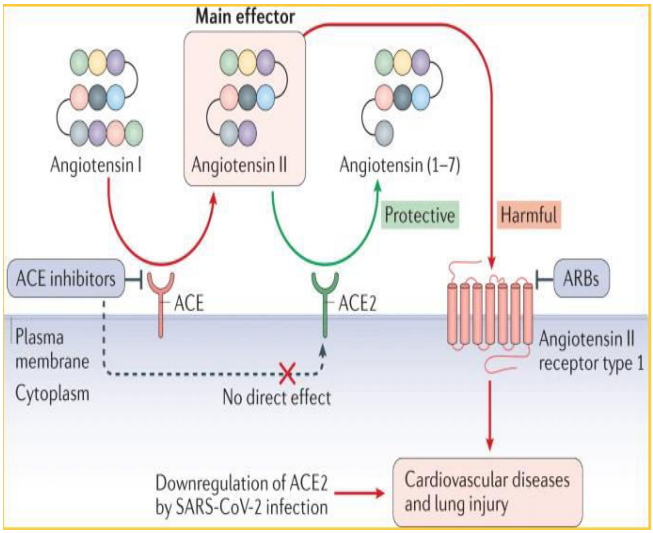
also induce cardiovascular manifestations including myocardial injury, myocarditis, arrhythmias, acute coronary syndrome and thromboembolism. Among these cardiovascular manifestations, myocardial injury has been independently associated with high mortality among patients with COVID-19. Finally, medications that have been proposed as treatments for COVID-19 such as hydroxychloroquine and azithromycin have pro-arrhythmic effects.

Effect of RAAS inhibitors on covid-19

Given that ACE2 is a receptor for SARS-CoV-2, clinicians have expressed concern that medications that up regulate the cell surface expression of ACE2 might be harmful. ACE inhibitors and ARBs have been shown to increase the expression of ACE2 in animal models. Given that both ACE inhibitors and ARBs are commonly used worldwide for the treatment of hypertension and other CVDs, whether these drugs should be discontinued during the COVID-19 pandemic has become a pertinent question. Importantly, indiscriminate withdrawal of these drugs could harm high-risk patients. Several medical societies including the ACC, AHA, Chinese Society of Cardiology, ESC and the Heart Failure Society of America have issued statements recommending continuation of RAAS antagonists for those who are currently prescribed these agents



These inconsistent results are likely to be attributable to the indirect effects of ACE inhibitors or ARBs on ACE2, which depend on conditions such as baseline expression levels of ACE2, dosing and treatment periods. Second, whether potential up regulation of ACE2 is harmful or protective is uncertain. Even if ACE inhibitors or ARBs can up regulate ACE2, no direct evidence has been found to show that up regulation of ACE2 affects susceptibility to viral infection. Furthermore, as shown in *Ace2*-knockout mice and rhACE2 studies, ACE2 is considered to be protective rather than harmful in settings of lung injury and CVDs. Together, these findings indicate no benefit of withdrawal of ACE inhibitors or ARBs in protecting against COVID-19.

Angiotensin II, the main effect or molecule in the renin–angiotensin–aldosterone system (RAAS), is unregulated in many pathological conditions, for which inhibition of angiotensin II by RAAS inhibitors is a common therapeutic strategy.Angiotensin-converting enzyme (ACE) produces angiotensin II from angiotensin I, whereas ACE2 inactivates angiotensin II by converting it to angiotensin (1–7). Therefore, ACE2 has a protective effect against cardiovascular disease and lung injury.

Adverse cardiovascular effects of potential drugs to treat COVID-19

Drug	Mechanism of action	Cardiovascular adverse effects
Inhibitors of endocytosis		
Camostat mesylate	Inhibition of TMPRSS2	Not common
Chloroquine and hydroxychloroquine	Blockade of virus entry by multiple mechanisms	QT interval prolongation
Umifenovir	Inhibition of S protein–ACE2 interaction and membrane fusion	Not common, but limited clinical data
Inhibitors of synthesis of non-structural proteins		
Lopinavir–ritonavir	Inhibition of 3-chymotrypsin-like protease	Atrioventricular block and cytochrome P450 3A4-related drug–drug interaction
Inhibitors of viral RNA replication		
Favipiravir	Inhibition of RdRP	Not common, but limited clinical data
Remdesivir	Inhibition of RdRP	Not common, but limited clinical data
Ribavirin	Inhibition of RdRP	Not common
Others		
Azithromycin	Macrolide antibiotic, used in combination with chloroquine or hydroxychloroquine	QT interval prolongation
Tocilizumab	IL-6 inhibition	Hypertension

ACE2, angiotensin-converting enzyme 2; COVID-19, coronavirus disease 2019; RdRP, RNA-dependent RNA polymerase; S protein, spike protein.

How does COVID-19 affect the heart?

If you have a heart condition, you may have already asked your doctor how COVID-19 affects you. If you haven't done this, you may want to do it soon and see if you need to take any extra precautions. We know the novel corona virus is a respiratory disease that primarily affects the lungs. But it can also affect the heart and other major organs in severe ways. People with heart disease — especially those with serious heart conditions — are among the people most at risk for complications if infected, according to the Centres for Disease Control and Prevention (CDC). People with heart disease in general are at risk for complications from a viral infection. The flu virus, for example, can trigger a heart attack, which is why vaccination is advised to reduce the risk.

With COVID-19, the virus can damage the respiratory system and force the heart to work faster and harder to supply oxygen-rich blood to major organs. COVID-19 can also cause blood clots in your heart's arteries. It can also attack and weaken the heart muscle.

The Long-term Cardiovascular Impact of COVID-19

With nearly a year of experience with the **COVID-19 (SARS-CoV-2)** virus, it has been found that some post-COVID patients experience long-term lingering coughs, cognitive issues and other complains months after they recover from the virus. These long-haulers are also appear to be having the heart issues as well. Determining the long-term impact of corona virus will certainly be one of the top cardiology issues discussed in 2021**.**

COVID patient cadaver studies also raised questions on the long-term damage to the heart after it was found COVID kills cardio myocytes. But the question has been what is the long-term impact of this cardiac injury. I think early on in the COVID pandemic, it became quite clear that hospitalized patients, particularly those that were sick enough to make it to the ICU, a significant minority of them, anywhere from 20 to 30%, had evidence of heart injury. And that heart injury was really related to two different types of pathos physiology. The first was a response to the high levels of circulating stress hormones. And the second was direct viral invasion myocarditis. And that became an important therapeutic target for us early on in the course of this disease. But I think the question many of us are asking now is what would the long-term implications of this be, and perhaps more importantly, would we be seeing heart injury in folks that were infected with COVID-19.

Last summer, physicians began suspect there may be long-term damage not just in lungs, but in the heart, immune system, brain and other organs. Evidence from previous coronavirus outbreaks, especially the 2002 severe acute respiratory syndrome (SARS) epidemic, suggested these effects may last years.

COVID-19 Long-hauler Cardiac disease

"There have been many high-profile cases of athletes at the collegiate and professional levels showing myocarditis after COVID-19," cardiologist and chief science and medical officer for the American Heart Association. "Research and data are key to answering the ongoing debate in college sports about the safety of return to play and guidelines on the appropriate assessment of the athletes."

Waggish is involved with the registry. He is an expert is sports medicine and involved in the Boston Marathon and the professional sports teams in Boston. He also helped author a *JAMA* article on recommendations for when it is safe for athletes infected with COVID to return to play.

"When we first started learning about the relationship between COVID and heart injury, we were obviously very concerned that when student athletes would get back onto the playing field, particularly those that had significant COVID infection that they would harbour heart injury or in worst case, active myocarditis that would put them at risk for sudden death," Baggies said. "What we started to see dealing with many athletes, literally hundreds of athletes at the professional and collegiate level, is that we weren't seeing anything in the way of cardiac injury in people that had mild or asymptomatic disease. So we made a call to really focus the screening to those that had either moderate or severe COVID infection."

I suspect we will have a similar dialogue around COVID-19, in which there will be providers that identify this as a legitimate diagnosis and some

that think about this more as a as a supratentorial diagnosis, meaning a diagnosis that patients may bring, but don't really have any firm pathophysiologic background. So I just think we need to keep a very open mind about what it means when a patient comes into the office and complains of long lasting symptoms

"The cellular damage that this virus causes is incredible. We haven't seen anything like this before, Hurst explained. "I've seen a case or two of people that seem to have ongoing heart muscle weakness or cardiomyopathy after COVID infection. Now, is it really cause and effect, or is even associated? That's impossible to answer at this time; we just need more time and more data. But the number of people and the number of cases that are being reported, it's certainly concern."

Side effects of cardio patients in covid-19

Myocarditis is inflammation of the heart muscle, while pericarditis is inflammation of the two-layered sac surrounding the heart.

Reported symptoms included chest pain and difficulty breathing, which typically began within a week after vaccination. Patients also showed abnormal electrocardiogram and blood test results. Dr. Parana Patel, chief of cardiology and an interventional cardiologist with UCI Health, says myocarditis and pericarditis can occur naturally due to viral and other infections Trusted Source.

"But the rate seen after [the mRNA] vaccines is slightly higher than would be expected for younger individuals," he said. "This is what caused the CDC to look more closely into this." Most cases reported to the CDC occurred in people under age 30 — particularly men and after the second dose. Researchers do not know why. However, this side effect of vaccination is uncommon.

In 12- to 39-year-olds, heart inflammation occurred at a rate of 12.6 cases per million second doses given. The rate after the first dose was 4.4 cases per million doses administered. Both rates were measured within 21 days after vaccination. The rates were higher among people who received the Modern-NIAID vaccine compared to Pfizer-Biotech.

Health officials in the U.S. Defence Department and Israel have reported similar cases of myocarditis or pericarditis among younger men following vaccination with an mRNA vaccine. Patel says that because the chance of heart inflammation is extremely low, the CDC is still recommending that all people 12 years old and older receive a COVID-19 vaccine.

However, if a person develops myocarditis or pericarditis after the first dose of an mRNA vaccine, the agency recommends that their second dose be delayed.

Once their heart is fully healed, the CDC says a second dose of an mRNA vaccine could be considered under certain circumstances.

The Food and Drug Administration (FDA) is expected to add a warning to mRNA vaccines authorized in the United States explaining that cases of heart inflammation are rare but a potential side effect of the vaccine, according to Reuters.

Cases more common in younger people and after second dose

So far, 1,226 cases of myocarditis or pericarditis have been reported to the CDC's vaccine safety monitoring system. This is out of 318 million vaccine doses administered in the United States as of June 21, according to data presented at the meeting of the reported cases, 484 occurred in people between 12 and 29 years old. Health officials are still investigating to confirm some of these.

Among the verified cases in younger people, 309 were hospitalized. As of June 11, only 9 were still hospitalized, with 2 in intensive care, the agency said.

About 81 percent of those who have been discharged from the hospital had recovered from their symptoms at the time of the report. No deaths have been associated with these vaccine-related conditions. Compared to myocarditis not related to a vaccine, the cases that occurred after mRNA vaccination were milder, with a shorter duration and minimal treatment needed, the CDC's Dr. Matthew Ouster told the committee.

Similarly, Dr. Michael Chan, an interventional cardiologist with Providence St. Joseph Hospital in Orange, California, says most cases of myocarditis after the COVID-19 vaccination that he's seen at his hospital have been mild. "These children have not been severely involved with the myocarditis," he said, "like some of the patients that I've seen with seasonal viral myocarditis."

These patients didn't require medications to support their blood pressure or to treat an abnormal heart rhythm. Instead, they were given a non-steroidal anti-inflammatory medicine such as ibuprofen. After that, "their chest pain went away after a day or two, the heart muscle tests trended down toward normal, and they were discharged," said Chan.

Officials at the CDC meeting said warnings about the potential risk of myocarditis and pericarditis should be added to the fact sheets provided to healthcare professionals and vaccine recipients

Effects of Corona virus on Heart Patients

In this unimaginable time, the entire world in limbo, medical facilities stretched to the hilt and frontline workers (medical and non medical) are putting their lives on the line each day, they go out to battle the common enemy: Corona virus (Covid-19). The world today, naturally, is taken in and has taken up one common task; breaking and eliminating the line of corona virus transmission in society's accords the world and healthcare professionals across many countries are reportedly unable to provide ample attention and care to those patients who are not suffering due to corona virus. This article will seek to provide a few points on what you should know, if you or anyone else has pre-existing cardiovascular issues and how corona virus poses certain, increased risks owing to the same.

The first question anybody with any cardiovascular complications will ask "Am I more likely to contract the virus because of my condition?" The answer is simply "No" as the virus does not discriminate and has infected people across the spectrum of earth. The theoretical proposition for those with heart diseases in case they get corona virus: they will most likely display and experience much more severe symptoms in case they are infected.

In case someone gets the virus, they might want to know, if the virus will infect them with more severe and serious symptoms as compared to those who were otherwise healthy prior to contracting COVID-19. Again, the virus is infecting indiscriminately via droplets in the air or on surfaces, transmitted by coughing, sneezing or talking in close proximity. If a person gets corona virus, the first thing it attacks is the lungs , setting of an inflammatory response that can stress the cardiovascular system of the body. First, the oxygen levels in the body will.

Naturally fall when infected with a respiratory disease and then, the inflammatory nature of COVID-19 will cause a drop in blood pressure. Naturally the heart will have to beat and pump harder in both scenarios in order to supply oxygen to the body. This can become serious in cases with cardiovascular issues. Other groups susceptible to lower lung function include those patients who have undergone an organ transplant, those receiving chemotherapy, those with concomitant leukaemia or lymphoma suffering heart diseases are theoretically at the highest risk of contracting corona virus on heart patients and succumbing to the same. The elderly are most susceptible and this is even more so in the case of elderly people with cardiovascular disease. The same applies to pregnant women. Patients with serious heart problems like heart failure, dilated cardiomyopathy; to name a few, are at highest risk.

In case of very severe inflammatory problems, arrhythmia may be aggravated or aerial fibrillation triggered; this can affect the heart. The best way to fight corona virus is by self-isolation and following proper hygiene protocols.

Can other diseases like diabetes or hypertension coupled with heart issues lead to more risk and higher mortality?

Evidence provided by reports from corona virus wards in Wuhan Province has shed some light on this: a rather significant portion of those who died had pre-existent co morbidities like diabetes and hypertension. One explanation is that both diabetes and hypertension are fairly common among people aged over sixty and the elderly are at highest risk from corona virus. This is just based on numbers from China and the study still remains unsubstantiated, like most studies on COVID-19 as of now

TREATMENT FOR HEART PATIENTS DURING CORONA VIRUS

Ever since Corona virus disease 2019 (COVID-19) has been declared a pandemic by the World Health Organization, it is becoming abundantly clear that this disease behaves differently among different subsets of patients. While the disease tends to be relatively mild and self-limiting in

younger individuals, it tends to take on a rather sinister course in the elderly and several others with risk factors. Pre-existing cardiovascular disease and hypertension in addition to age and diabetes have emerged as fairly strong associates of a poor outcome in patients with this disease. Consequently, I have been contacted on several recent occasions by patients or their relatives and even a few colleagues about various issues pertaining to patients with heart disease. I will try and address them in the following paragraphs:

There was some concern about likely problems with a group of anti-hypertensive during the corona virus epidemic. These medicines belong to either of the two groups viz. Angiotensin-Converting Enzyme Inhibitors (ACEI) such as ramipril, perindopril, lisinopril, etc. or Angiotensin Receptor Blockers (ARBs) such as losartan, telmisartan, olmesartan, azilsartan, etc.

The rationale for this concern was an assumption that since the virus is internalized by the cells through the ACE 2 receptors and since the ACE 2 receptors are increased in patients on these drugs, a higher number of viruses are likely to be internalized in patients on these drugs.

Contrary to these assumptions, there are some animal studies that have shown that these drugs may have a protective value in animals exposed to ACEI/ARBs. There is no human data supporting this.

Therefore, several premier societies such as the American College of Cardiologists (ACC), the European Society of Hypertension have emphasized that there is no need to stop treatment with ACEI or ARBs. Treatment with these drugs should be continued in all patients as per evidence available at present.

Can corona virus infection cause a heart attack? There have been some recent reports of patients with COVID-19 infection presenting as an acute myocardial infarction. When they were subsequently subjected to coronary angiography it was found to be normal.

Any flu-like illness including corona virus infection involves the heart in 3 different ways direct or immunological injury-causing myocarditis, stress-related acute coronary syndrome and electrolyte and acid-base disturbances leading to arrhythmias (including sudden cardiac death). It must be remembered that the former two wiz acute coronary syndromes and myocarditis can have a similar presentation with chest discomfort, ECG changes, and cardiac enzyme elevation.

Hence it is imperative for patients with known coronary artery disease to be extremely diligent with drug compliance and avoid or control any known existing risk factors of coronary artery disease.

Firstly, it is important to remember that no specific drug has been proven to have a definite therapeutic role in corona virus infection. A few drugs have been randomly mentioned (more on social media than in scientific literature) including hydroxychloroquine and azithromycin.

Azithromycin is an antibiotic from the microcline group and can cause prolongation of QT interval on ECG which may predispose individuals to dangerous and life-threatening electrical abnormalities on ECG.

Hence, none of these drugs should be used as self-medication and should be consumed only when prescribed by a physician.

Use medicine heart patients in covid-19 Hydroxychloroquine and azithromycin

Chloroquine and hydroxychloroquine have been widely touted as potential treatment strategies for COVID-19. Chloroquine and hydroxychloroquine can potentially block virus entry into cells, particularly via the endosomal pathway, by inhibiting the glycosylation of host receptors, photolytic processing and endosomal acidification. These agents can also mediate immunomodulatory effects through attenuation of cytokine production and inhibition of autophagy and liposomal activity. Both drugs are used in the treatment of malaria and chronic inflammatory diseases such as systemic lupus erythematosus and rheumatoid arthritis. A French observational study involving 36 patients with COVID-19 reported improved virus clearance with hydroxychloroquine treatment. Furthermore, combining hydroxychloroquine with azithromycin in six patients resulted in even better virus clearance than with the use of hydroxychloroquine alone. Importantly, however, several concerns have been raised regarding the characteristics of the control group (which consisted of patients recruited from a separate hospital) and ethics approve. In a randomized study involving 62

patients with COVID-19 in China, patients in the treatment group received hydroxychloroquine (400 mg per day) for 5 days, whereas patients in the control group received standard treatment (oxygen therapy, antiviral agents, antibacterial agents and immunoglobulin, with or without corticosteroids). Hydroxychloroquine improved the time to clinical recovery, body temperature recovery time, cough remission time and pneumonia-related symptoms compared with standard treatment alone. Furthermore, in a retrospective study from Wuhan involving 550 critically ill patients with COVID-19, mortality was significantly lower among patients treated with hydroxychloroquine plus standard treatment (which included other antiviral drugs and antibiotics) compared with standard treatment alone (18.8% versus 47.4%). However, in a large observational study involving 1376 patients from New York City, hydroxychloroquine treatment did not alter the risk of the composite end point of intubation or death. These inconsistent results show that the efficacy of hydroxychloroquine is still controversial and needs to be validated in large, randomized studies.

Although chloroquine and hydroxychloroquine have a long history of clinical use for numerous conditions, these agents are also known to induce arrhythmias. Azithromycin, which has been assessed in combination with hydroxychloroquine as a treatment for COVID-19, is also known to prolong the QT interval. In a cohort study of 90 patients with COVID-19 who received hydroxychloroquine (with or without azithromycin), those who received a combination of hydroxychloroquine and azithromycin had greater QT interval prolongation than those taking hydroxychloroquine alone. Furthermore, in a retrospective cohort study of 1,438 patients hospitalized with COVID-19 in New York, treatment with hydroxychloroquine, azithromycin or both was compared with neither treatment. None of the groups had an increase in in-hospital mortality, but the secondary outcome of cardiac arrest was more likely in patients receiving both hydroxychloroquine and azithromycin than in patients receiving neither drug. Given that some patients with COVID-19 might have impaired renal function owing to systemic illness, frequent electrocardiographic evaluation should be strongly considered in patients treated with hydroxychloroquine and/or azithromycin.

Remdesivir

Remdesivir is a promising investigational nucleotide analogue for the treatment of COVID-19 that has broad-spectrum antiviral activity and functions by targeting Drip'.Remdesivir was originally developed for the treatment of Ebola virus disease. Prophylactic and therapeutic administration of deliver has been shown to improve pulmonary function and to decrease viral load in a mouse model of MERS. In a randomized, double-blind, placebo-controlled trial involving 237 patients with COVID-19 in China, remdesivir was associated with a numerically (but not statistically significant) faster time to clinical improvement than was the placebo. Preliminary results from a double-blind, randomized, multicentre, placebo-controlled trial involving 1,063 patients with COVID-19 indicate that those who received remdesivir had a 31% faster time to recovery than those who received placebo (median time to recovery 11 days versus 15 days). Of note, in light of these preliminary findings, the FDA granted emergency use of remdesivir for COVID-19 in May 2020 to meet the urgent demand for treatment of hospitalized patients. The optimal dosing and duration of treatment is still under investigation. Under the emergency use authorization, a 10-day treatment regimen (200 mg on day 1 followed by 100 mg per day for 9 days) is suggested for patients requiring invasive mechanical ventilation and/or extracorporeal membrane oxygenation, and a 5-day treatment course is suggested for patients with milder symptoms. Although prominent cardiovascular adverse effects associated with remdesivir have not been reported so far, these might become apparent with more widespread use.

Lopinavir–Ritonavir

Lopinavir–ritonavir is a fixed-dose drug combination used for the prevention and treatment of HIV infection and works by inhibiting protease activity. Lopinavir is available only in combination with ritonavir, which functions to slow the breakdown of lopinavir by inhibiting cytochrome P450 3A4. In a randomized, controlled, open-label trial involving 199 patients with COVID-19, no benefit was observed with

lopinavir–ritonavir treatment compared with standard care. Gastrointestinal adverse effects were more frequently reported in the lopinavir–ritonavir treatment group than in the standard group, but adverse cardiovascular effects were not reported in either group. Nevertheless, lopinavir–ritonavir should be used with caution in patients with COVID-19 because this drug combination might interact with common cardiovascular drugs that are metabolized by cytochrome P450 3A4, including ant arrhythmic agents, ant platelet drugs and anticoagulants.

Levocetirizine + Montelukast Uses

Levocetirizine+Montelukastis used for sneezing and runny nose due to allergies, Hay fever and Allergic skin conditions.

How Levocetirizine + Montelukast works

Levocetirizine + Montelukastis a combination of two medicines: Levocetirizine and Montelukast, which relieves sneezing and runny nose due to allergies. Levocetirizine is an ant allergic which blocks a chemical messenger (histamine) responsible for runny nose, watery eyes and sneezing. Montelukast is a leukotriene antagonist. It works by blocking another chemical messenger (leukotriene). This reduces inflammation (swelling) in the airways and nose, and improves symptoms.

Aspirin is used to reduce fever and relieve mild to moderate pain from conditions such as muscle aches, toothaches, common cold, and headaches. It may also be used to reduce pain and swelling in conditions such as arthritis. Aspirin is known as a salicylate and a no steroidal anti-inflammatory drug (NSAID). It works by blocking a certain natural substance in your body to reduce pain and swelling. Consult your doctor before treating a child younger than 12 years. Your doctor may direct you to take a low dose of aspirin to prevent blood clots. This effect reduces the risk of stroke and heart attack. If you have recently had surgery on clogged arteries (such as bypass surgery, carotid endarterectomise, coronary stent), your doctor may direct you to use aspirin in low doses as a "blood thinner" to prevent blood clots.

How to use aspirin oral

If you are taking this **medication** for self-treatment, follow all directions on the product package. If you have any questions, ask your doctor or **pharmacist**. If your doctor has directed you to take this medication, take it exactly as prescribed.

Take this medication by mouth. Drink a full glass of water (8 ounces/240 millilitres) with it unless your doctor tells you otherwise. Do not lie down for at least 10 minutes after you have taken this drug. If stomach upset occurs while you are taking this medication, you may take it with food or milk.

Swallow enteric-coated tablets whole. Do not crush or chew enteric-coated tablets. Doing so can increase stomach upset.

Do not crush or chew extended-release tablets or capsules. Doing so can release all of the drug at once, increasing the risk of side effects. Also, do not split extended-release tablets unless they have a score line and your doctor or pharmacist tells you to do so. Swallow the whole or split tablet without crushing or chewing.

Vitamin C benefit in covid-19 of heart patients

Vitamin C is one of the safest and most effective nutrients, experts say. Though it may not be the cure for the common cold, the benefits of vitamin C may include protection against immune system deficiencies, cardiovascular disease, prenatal health problems, eye disease, and even skin wrinkling. The tolerable upper intake level (or the maximum amount you can take in a day that likely won't cause harm) is 2000 mg a day for adults.

A recent study published in *Seminars in Preventive and* Alternative Medicine that looked at over 100 studies over 10 years revealed a growing list of possible benefits of vitamin C.

"Vitamin C has received a great deal of attention, and with good reason. Higher blood levels of vitamin C may be the ideal nutrition marker for overall health," says study researcher Mark Moy ad, MD, MPH, of the University of Michigan. "The more we study vitamin C, the better our understanding of how diverse it is in protecting our health, from cardiovascular, cancer, stroke, eye health [and] immunity to living longer."

"But," Moy ad notes, "the ideal dosage may be higher than the recommended dietary allowance."

How Much Vitamin C Is Enough?

Most of the studies Moy ad and his colleagues examined used 500 daily milligrams of vitamin C to achieve health results. That's much higher than the RDA of 75-90 milligrams a day for adults. So

unless you can eat plenty of fruits and vegetables, you may need to take a dietary supplement of vitamin C to gain all the benefits, Moy ad says. He suggests taking 500 milligrams a day, in addition to eating five servings of fruits and vegetables.

Conclusion

It is the given that numerous studies have been demonstrated that SARS-CoV-2 shares many biological features with SARS-Cove, our knowledge of the path physiological mechanisms underlying SARS can be used to understand the disease processes involved in COVID-19. Mechanistically, the interaction between the S protein and ACE2 is likely to have a central role in disease pathogenesis, especially in cardiovascular manifestations of this disease, and this interaction is a potential target for the prevention and treatment of COVID-19.

Several hurdles need to be overcome in the study of the mechanisms underlying COVID-19. First, biological experiments using SARS-CoV-2 can be performed only in laboratories with a bio safety level 3 rating. Second, the use of animal models to mimic the disease process is associated with numerous challenges. Given that cellular or tissue tropism is likely to be an important factor contributing to the diverse phenotypes of COVID-19 mouse or rat models are not ideal to study host tropism because they are not as susceptible to SARS-CoV-2 as humans owing to differences in the amino acid sequence of ACE2. To use mice or rats, human *ACE2* needs to be introduced artificially. Transgenic mice expressing *ACE2* infected with SARS-CoV-2 have been reported to show signs of pneumonia, but the overall symptoms experienced by these mice are much milder than those in humans. Therefore, alternative platforms might involve genome-edited mouse or rat models in which *Ace2* is replaced by human *ACE2*, other animal species that are naturally susceptible to SARS-CoV-2 infection (such as ferrets, hamsters and non-human primates) or in vitro models such as induced pluripotent stem cells and or ganoids

The COVID-19 pandemic is changing our lives in unprecedented ways. Given the lack of safe and effective vaccines or proven treatments for COVID-19, our main strategy to combat the pandemic is social distancing. The capacity of health-care systems globally has been severely tested (and in some countries completely overwhelmed), and the effect of this pandemic on social interactions, health-care delivery and the global economy continues to mount. Reduced physical activity owing to lockdown measures might also contribute to poor control of cardiovascular risk factors. Vaccine development is expected to take 12–18 months. To meet the urgent need for effective treatment and preventative strategies, a concerted effort must be made by researchers globally to investigate and integrate biological and clinical findings related to COVID-19.

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