Review Article

Effects of Coronavirus on human Cardiovascular System

Abstract

SARS-CoV-2 has many biological traits with SARS, the pathogenic virus that caused the acute respiratory syndrome (sars syndrome) epidemic in 2002. A link among both CORONAVIRUS and cardiovascular illness has also been discovered in clinical investigations. . The coronavirus disease is caused by binding of the viral spike protein to angiotensin-converting enzyme 2. It has spread around the world, affecting millions and millions of the people. CORONAVIRUS looks to be connected to even worse results and an elevated risk of mortality in individuals with which were before cardiovascular illness, despite the fact that CORONAVIRUS can cause myocardial damage, arrhythemia, acute coronuarysyndromee, and veneousthromboembolism. Possible drug–disease interactions are becoming more of a concern for individuals with CORONAVIRUS and associated cardiac and vascular illnesses. By the integrating our expertise of the virus's genetic makeups with clinical findings, we can gain a better grasp of the possible processes causing CO VID-19. This might pave the door for the creation of medicinal and pharmaceutical and products. Pulmonary embolism is one of most common acquired condition in generalized hospitalized patient but a uncommon finding in patients with Covid.It has a proclivity for thrombosis by which is fueled by at least two separate yet interconnected processes i)A state of the hypercoagulability and is Large vessel thrombosis and thromboembolism.ii)Direct vascular and endovascular damage.In situ microvascular thrombosis is caused by endothelial damage.PE can be presented in patients admitted to the ICU despite standard prophylactic anticogulation.Deep vein thrombosis is present is significant cases of PE.

Keywords Cardiovascular disorders, Myocardial injury,Arrhythmia,Angiotensin converting enzyme,Covid Complications.

Introduction

Reports says first time in the history of world CORONAVIRUS coronavirus case was found in Wuhan, wgich is located and is important part of China in Decembeer 2019. Since then, it is spreading rapidly worldwide and has became global pandemic. On March 11, the World Heealth Organisation announced CORONAVIRUS as a worldwide pandemic.

Seveereacuuterespiiratorysyndroome coronavirus 2, also called (SARS-CoV-2) is the causitive agent of CORONAVIRUS. The virus attacks on the upper respiiratory also called (UR) and gastrointestinal also called (GI) tracts in humansCoughing, difficulty breathing, temperature or breathlessness, muscular pains, diarrhea, lack of flavor and aroma, and weariness are some of the symptoms you may experience. are all common symptoms in most patients. (1)

As the arisen of covid 19 SARS-CoV-2, researchers are attempting to characterise the virus's characteristics through genomic sequence analysis and analysis of viral protein structure. Coronaviruses are viruses having only one strand of rna that are not previously recognised as constantly pathogenic virus before the covid 19SARS pandemic. It's likely that we'll be able

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to use some of our knowledge about SARS's biological structure and pathogenesis to understanding SARS CoV-II. For reference, SARS-CoV& SARS-CoV-2 both usesACE2 as receptor which provide place to attach to go inside the cells of patient, on the other hand MERS-CoVfound as dipeptidyl peptidase 4 user as itsjoining receptor. (2)

Coronavirus And Cardiovascular System

SARS-CoV-2covid 19communicate with theACE2 and infiltrate the cells of the host, including alveolarepithelial cells and cardiac myocytes . The ACE2 gene has a wide range of expression in the body of patient, with particularly strong function in the heart, gastrointestinal tractGIT, lungs and both kidneys. ACE2 is also involved in the neurohumoral control of the CVS.The alteration in ACE2 signalling pathways caused by SARS-CoV-2 found on to ACE2 produces immediate cardiac and pulmonary damage . Because it converts angiotensin II to angiotensin 1 to 7), ACE2 protects The renin-angiotensin-aldosterone system affects the heart (RAAS). Angiotensin II is a proinflammatory and vasoconstrictor that damages endothelium of capillaries, whereas angiotensin1-to 7) is a vasodialator and anti-inflammatory mediator.The virus, on the other hand, reasons ACE2 to bedecreased and angiotensin II levels to rise, resulting in increased heart damage. As a result, while Increasing the density of ACE2 receptors will increase viral load. , it is still expected to reduce heart injury.In COVID- 19 cases, individuals with cardiovascular disorders are experiencing an increase in morbidity. Infection impacts cardiac biological pathways like the ACE2 transmission system, heart muscle strength, coagulation processes, promotes a break in plaque related to the stent, and aggravates cardiac dysfunction and dysfunction.The diagnosis was made using nasal scrapings and fine-needle aspiration cytology, which revealed fungal hyphae.

Coronavirus disease 2019 (Coronavirus) is an illness caused by coronavirus-2, which causes severe acute respiratory syndrome (SARS-CoV-2). There have been several turns and twists in terms of pathogenesis, diagnosis, therapy, sequelae, and complications since the first case was discovered in December 2019 in Wuhan, China. For effective treatment and improved results, early detection of these high-morbidity diseases is critical.

Mucormycosis sinus infection is a kind of invasive fungal sinusitis that usually affects those who are immunocompromised. Patients with uncontrolled diabetes, acquired immunodeficiency syndrome, iatrogenic immunosuppression, and haematological malignancies are also possible candidates.

Mycotic infiltration of blood vessels, vasculitis with thrombosis, tissue infarction, haemorrhage, and neutrophilic infiltrate are all histological characteristics. Without early identification and treatment, there is a risk of fast development, with death rates of 50–80% reported from intra-orbital and cerebral sequelae.

Evenintervention, therapy is frequently ineffective, resulting in infection spread and mortality. Coronavirus is a life-threatening viral illness that causes a reduction in cluster of differentiation 4 and 8 positive T-helper (CD4+ T and CD8+ T) cell numbers as well as reduced cell-mediated immunity. Fungal co-infections are more common in critically sick patients, notably those hospitalised to intensive care units and those who require mechanical ventilation. Extensive steroid treatment can also decrease immunity, allowing for the colonisation of opportunistic fungal infections.(3)(4)

In patients infected with SARS or MERS, cardiovascular disease is a prevalent comorbidity (with a prevalence of 10 percent and 30 percent, respectively) Similar findings have been observed in a series of papers on the clinical changes in CORONAVIRUS patients.

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Sharma S, Grover M, Bhargava S, Samdani S, Kataria T. Post coronavirus disease mucormycosis: a deadly addition to the pandemic spectrum. J Laryngol Otol 2021; 1–6. https://doi.org/10.1017/ S0022215121000992

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Cardiovascular diseases and risk factors of it, such as diabetes mellitus and hypertension, were identified to be prevalent pre residing diseases in individuals affectedwith CORONAVIRUS in early news from China, however the criteria of cardiovascular diseases cvds used in each research was vague. According to an initial study from Wuhan that included 41 clients who have been hospitalised with coronavirus by second of Jan of the year2020, the incidence of any comorbid conditions was 32percent, with insulin resistance(DM) (20%), high blood pressure(HT) (15%), and other CVDs being the most similar underlying diseases (15 percent Critically sick patients (like those transferred to the ICU) and those who died had a greater incidence of these pre-existing illnesses. According to the National Centers of Disease Control and Management,China as of February 11, 2020, there was a 2.3 percent total death rate for coronavirus (1,025 deaths in 44,673 confirmed cases). In a study of 1,099 mainland Chinese patients, 24 percent had any comorbiidity (37 percent of dengerously sick patients), 14% found hypertensive (25 percent dangerously ill people), and 7% had DM (15 percent of seriously ill patients).

Clinical Pattern of Cardio-vascular activation in coronavirus

individuals with coronavirus infections may also have riskof cardiovascular diseases cvds, like MI, myocardiitis, pericardiitis, vascuiitis, and cardiac arrhythmiias, according to a report found in the Journal of Clinical Infectious Diseases by the American Heart Association (JHA) and the American College of Cardiology.

TABLE 1. Rate of death to Date of induals With coronavirus Infection and Specific already existing Conditions (W H OData) (5)

Already existing conditions	Death rate %
CVD	10.5
DM	7.3
respiratory disease	6.3
HT	6.0
Cancer	5.6

Drug-Induced Cardiovascular Complications

Hydroxychlorohhquine and azitthhromycin have been utilised for CORONAVIRUS infection prophyylaxis. The worldewidecommon antibiotic azithromyciin has been discovered as a rare reason of QT prolongation, severe arrhithhmias, and an increased cases of sudden death; risk factors include greater age and female sex. This antibiotic can also produce polymorphic ventricular tachycardia that is not pause-dependent. Both medicines can elicit proarrhythhmia via mechhanisms other than the inhibition of IKr involved in typical csases of torsaade de pointes, according to electrophysiological investigations. A few authors, on the other hand, have highlighted the safety considerations for utilising hydroxychlooroquine with azithroomycin in medical usse [166]. There isn't enough clinical evidence to recommend whether chloroquine or hydroxychloroquine should be used to treat CORONAVIRUS. However, if taken, clinicians should keep an eye on patients for side effects, particularly prolonged QT intervals.(6)(7)(8)

Microvascular Coagulopathy and Plateltes in CORONAVIRUS

CORONAVIRUS-induced coagulopathy is caused by a cytokkine storm, uncontrolled inflammmation-meediatedendoothelial damage, plateletts, and RASdysregullation.

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Antiphosspholipidssyndrosme (APS) is an autoiimmune condition that causes blood clot in the artery and vein on a regular basis. Lupus anticoaagulant, anticardiollipin, and anti-β2-glycoprotein antibodies, also known as triplle-posiitiveantiboodies, are a distinct subtype of APS. Antibodies to these proteins are high in viral infections, syphilis, and autoimmune disorders including systemic lupus erythematosus. Lupus anticoagulant was found in 50% of CORONAVIRUS patients. Only endothelial cells and megakaryocytes produce Von Willebrand factor (VWF), which is either released into the plasma or retained inside intracellular organelles. VWF has been found to bind to platelet receptors and influence platelet adhesion and aggregation. Thrombotic microangiopathy, the hallmark of thrombocytopenic purpura, is caused by this abnormal VWF. VWF's interaction with inflammation or complement activation to cause coagulopathy is unknown. (9)(10)

The Effect of coronavirus on the Heart

With increased troponin, between 9% and 27% of patients with CORONAVIRUS infections show indications of cardiac damage. Cardiomyopathy patients exhibited a much higher mortality rate, showing the importance of the cvs in these patients' prognosis. (11)(12)

Many CORONAVIRUS patients die of cardiac arrest, most likely as a result of a combination of main cardiac involvement and systemic manifestations such acute hypoxia, multiorgan failure syndrome, or systemic inflammatory response syndrome, among others.

During the 2003 SARS outbreak, Oudit from our group found SARS-CoV virus in 7 of 20 hearts of SARS victims on autopsy. The expression of ACE2 and TMPRSS2 receptors presumably promoted viral growth, although ACE2 expression was significantly lowered post infection, perhaps contributing to the increased inflammation. (13)

Middle East Respiratory Syndrome

In June 2012, the MERS-CoV pandemic broke out in Arab countries. Infected dromedary camels were found to be intermediate host, the virus spread through contact with the camel, to the people. MidleEastrespiratory syndrome. CoVis of the group of viruses that use a serine peptiidase as the receiver to pass into thecell. It spreads from respiratory secretions of person who is already infected, to others through near contact. It has period of incubation 3 - 14 days. The MERS-CoV said to be have first observed in bats and then might have introduced to dromedary camels. MERS-CoV is thought to be spread into the environment and transferred from ambient surfaces to hands, where it can infect the nose, eyes, and mouth. (14)

As recorded, until the date 30th November, 2019, nearly 2500 confirmed cases of MERS-CoV infections has been recorded, with around 860 deaths with fatality rate of 35%, most of cases were recorded in Saudi Arabia (2102 cases, fatality rate of 37%). In South Korea and Saudi Arabia, R0 of MERS-CoV epidemics was estimated to be found between 2 and 5, that concludes, each person infected with MERS-CoV is anticipated to infect 2 to 5 other persons in a completely infection prone community. Older age, male sex, and underlying medical problems such as diabetes mellitus, heart disorders, chronic renal disease, pulmonary disease, and hypertension were all clinical risk factors for MERS mortality. (15)(16)

Others

Infection of COVID-19 can cause a hypercoagulable condition, which is well-documented.In addition, PE symptoms may be mistaken for pneumonia which make it difficult to differentiate between the symptoms of COVID-19. Therefore, doctors perform outpatient or inpatient evaluation of COVID-19-positive patient.It is important that the criterion for

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,Acute ventricular hypertrophy and signs of right ventricular overload.

Comment [c102]: Please change it to PE testing should be conducted in patients with moderate, short-term symptoms, but also syncope, shock or minimal pulmonary infiltrates, symptoms of acute respiratory failure, acute ventricular hypertrophy and signs of right ventricular overload.

D-dimer has been used to help identify coronavirus patients which are susceptible to venous thromboembolism in many case studies.In study of group of patients D-dimer levels were shown to be linked with 198 cases. There is a 50 percent increase in the probability of contracting VTE. study of 400 COVID inpatients At the age of 1,001, a high D-dimer was As much as 2,500ng/mL showed an OR for thrombos with a hazard ratio of 3.04 (95 percent confidence interval, 1.27-7.31)

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OR of 6.79 was found for D-dimer > 2,500 ng/mL.In this study, 95 percent confidence intervals ranged from 2.39 to 19.30, and P <.001 .But this study found that elevation of D-dimer is also indication of bleeding complications.So there is lack of evidence showing elevated D-dimer level for diagnosis of PT. Since Pulmonary thromboembolism is leading covid complication thromboprophylaxis should be considered in vulnerable individuals. Prophylaxis therapy to prevent cogulation within 24 hrs was associated with a lower mortality rate with compared to prophylactic with no anticoagulation.Prophylactic anticoagulant is recommended by multiple society guidline with patients who do not have contraindications. In individuals with COVID-19, however, normal dosages of preventive anticoagulation are likely to be inadequate to prevent VTE; many VTE episodes are caused within 24 hours of admission. Number of cytogenetic abnormality have been detected in cases of coronavirus. Infact the generation time of coronavirus is somewhat prolonged rahtherthan shortened.

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Conclusions

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Infection of COVID-19 can cause a hypercoagulable condition, which is well-documented.In addition, PE symptoms may be mistaken for pneumonia which make it difficult to differentiate between the symptoms of COVID-19 . Therefore, doctors perform outpatient or inpatient evaluation of COVID-19-positive patient.It is important that the criterion for admission to an inpatient contemplation of simultaneous PE.PE testing should be conducted in patients with moderate, short-term symptoms, but also syncope, shock or minimal pulmonary infiltrates,symptoms of acute respiratory failure

,Acute ventricular hypertrophy and signs of right ventricular overload.

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Comment [c113]: Please delete this paragraph that already mentioned under OTHERS section damage, HF, arrhhythmias, stress-caused cardioomyopathy, cooronary syndrome, and DIC, according to multiple investigations. Concerns have been raised about how drugs used to treat CORONAVIRUS individuals may cause cardiovascular issues, thus their use should be carefully evaluated before treating CORONAVIRUS patients. Several attempts have already been undertaken to better understand the diagnosis and prognosis of these cardiovascular problems, but the available information is limited.Direct viral infection, hypertension, hypoxemia, immunological response, cytokine storm, stress-induced cardiomyopathy and DIC are all thought to have a role in CORONAVIRUS-induced cardiovascular problems, although the specific pathways remain unknown.

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Due to the few research focusing on cardiovascular clinical information on CORONAVIRUS exposure, a slew of concerns remain unanswered: (1) How do genetic and non-genetic variables impact CORONAVIRUS severity, susceptibility and clinical outcomes? (2) What are the molecuar and celular processes of post-CORONAVIRUScarrdiovascularcomplcations? (3) What medicines or treatments are available to guard against cardiovascular problems caused by CORONAVIRU?. Hundreds of vaccine development projects based on adenovirus-associated virus (DNA), modified mRNA and inactivated virus are now underway throughout the world, with dozens of them in clinical stages 1 and 2, and 11 vaccination programmes in clinical stages 3 and 4..While preliminary human clinical studies have shown humoral and T-cell responses to vaccination injections, there are still many unanswered concerns about the vaccines' effects. It's difficult to say how long these vaccinations will keep people safe from SARS, or how effective they will be.

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Immune protection might be fleeting, may be lasting months or maybe a lifetime, and the virus could develop resistance to it. Continued research and development of animal models to replicate human disease, with a particular focus on CORONAVIRUS's cardiovascular effects, might provide new insights into these and other challenges. (17-27)

Comment [c122]: No references cited in conclusions section

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Font size, 12
Style, Normal
And typical to reference style of the journal especially references from number 20-27