Clinical Case Reports International

Review Article
Published: 18 Feb, 2023

Cardiovascular Disease Risk in COVID-19 Patients: A Review

*Sunil GD**

College of Agriculture and Bioresources, University of Saskatchewan, Canada

Abstract

The clinical syndrome of Coronavirus Disease of 2019 (COVID-19) is caused by SARS (Severe Acute Respiratory Syndrome) is the etiology of this condition, which uses angiotensin-converting enzyme 2 to enter the cells. While the virus predominantly affects the respiratory system, it can also induce a number of severe cardiac instances, such as myocarditis, heart failure, acute coronary syndrome. COVID-19 treatment is more likely to succeed with Remdesivir and convalescent blood products, based on early findings from clinical trials, the National Institutes of Health (NIH), and the Food and Drug Administration (FDA) recommendations. Umifenovir, hydroxychloroquine and chloroquine, favipiravir, colchicine are additional possible treatments. As a result, all of these medications are undergoing further research. Furthermore, before administering the suggested medications, drug-drug interactions and safety risks must be considered. The development of innovative therapeutics for COVID-19 is a top goal. Because SARS-CoV-2 has such a devastating effect, delaying the spread of infections will benefit the health-care system, particularly in terms of the number of Intensive Care Unit visits (ICU). Several clinical studies are now underway all around the world.

Keywords: Coronavirus; ACE-2; NIH and FDA; ICU; Remdesivir

Introduction

Wuhan, Chinese city, in December 2019, emerges to be the epicenter of an outburst of pneumonia of unidentified cause [1]. After segregation of the virus, it was established by high-throughput sequencing as 2019 novel Coronavirus (2019-nCoV), and later named formally by the World Health Organization (WHO) as the Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), causing coronavirus disease known as COVID-19 [2]. This new single-stranded enveloped RNA virus is the 7th well-known human coronavirus. SARS-CoV-2 is not like the coronaviruses known source of common cold (229E, OC43, NL63, and HKU1), but alike to the zoonotic Severe Acute Respiratory Syndrome-Coronavirus (SARS-CoV), (2002) and the Middle East Respiratory Syndrome (MERS) coronavirus (2012). Following transmembrane protease serine 2 initiation of the viral surface spike protein. The tie up of the viral surface spike protein to the human Angiotensin-Converting Enzyme 2 (ACE2) receptor, a component of the Renin-Angiotensin System (RAS), initiates SARS-CoV-2 infection. The lung (mainly type II alveolar cells 7) expresses ACE2 and appears to be the main entrance point [3]. The beneficial effects of ACEIs/ARBs on ACE2 expression may reduce the harmful effects of ACE2 downregulation caused by COVID-19 infection, providing pulmonary (and cardiac) protection [4]. RAS is a complex web facilitating cardiovascular and renal function. The ACE2 is a membrane protein that inhibits RAS activity by changing the vasoconstrictor Angiotensin-II (Ang-II) to the vasodilator angiotensin-(1-7). In the lungs and heart, it is highly expressed. A fall in ACE2 is connected to the development of type 2 diabetes and cardiac hypertrophy [5]. Though respiratory symptoms are generally the most prominent sign of COVID-19, these individuals may also develop cardiovascular problems, which can lead to decease [6]. In this review we will further discuss following cardiac complications: Arrhythmias, Acute coronary syndrome, heart failure, and myocarditis.

ACE2's Function in Administration of Coronavirus Disease 2019

In the year 2000, ACE2 was identified as a homologous Angiotensin-Converting Enzyme (ACE). ACE2 is an 805-amino-acid transmembrane protein of type I. It has two domains, one amino-terminal and the other carboxy-terminal domains catalytic. ACE2 is encoded by the *Xp22.2 gene*, which is found on the X chromosome. ACE2 is found in the kidney, lung, heart, liver, testis, gut, and other organs, according to evidence [7]. ACE2 is a cleavable extracellular enzyme that is found on the cell surface membrane, similar to ACE. A member of the metalloproteinase family,

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Correspondence:
Gor Darshil Sunil, College of Agriculture and Bioresources, University of Saskatchewan, Saskatoon, Saskatchewan, Canada,
E-mail: darshilgor150@gmail.com

Received Date: *18 Jan 2023*
Accepted Date: *14 Feb 2023*
Published Date: *18 Feb 2023*

Citation:
Sunil GD. Cardiovascular Disease Risk in COVID-19 Patients: A Review. Clin Case Rep Int. 2023; 7: 1484.

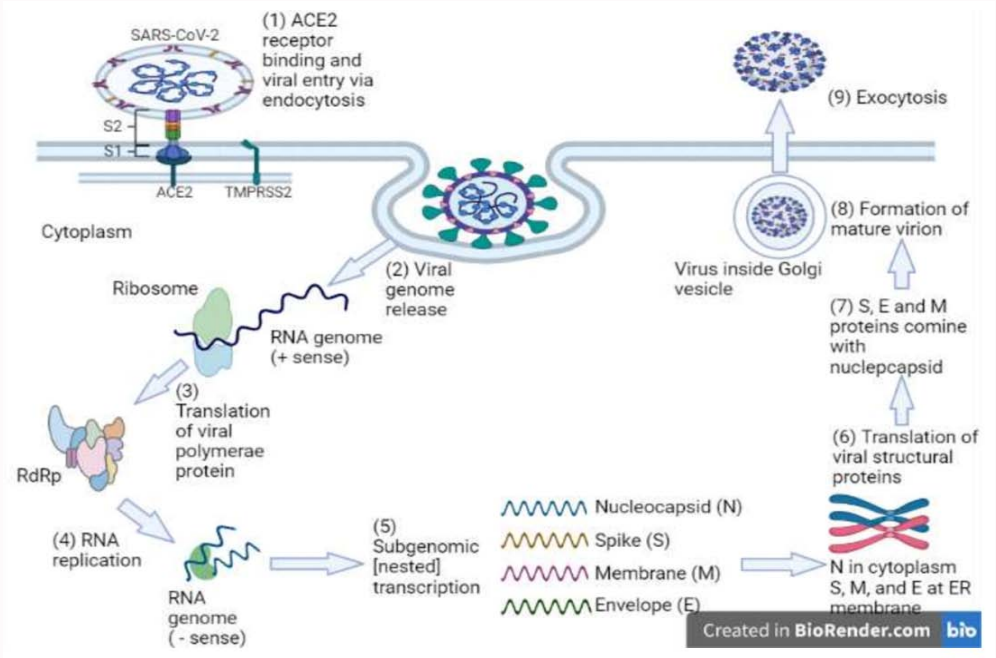

Figure 1: Inside the host cell SARS-CoV-2 life cycle [13].

ADAM17, may degrade membrane-bound ACE2 and discharge the extracellular portion into the bloodstream as soluble ACE2 (sACE2), which absences transmembrane and cytoplasmic domains but retains activity. The presence of an endogenous inhibitor in the pattern of a yet-to-be-identified positively charged small molecule has been discovered to lower sACE2 activity [8].

RAS component ACE2

RAS is one of the utmost chief hormonal systems for sustaining homeostasis in the human body, controlling fluid volume, blood pressure, and sodium-potassium balance. RAS, which is found in the body circulation and local tissues, has been associated to a variety of illnesses, including cardiovascular and cerebrovascular disease, diabetes, and others [8]. In the classical RAS, Renin cleaves angiotensinogen to create the decapeptide Angiotensin I (Ang-I), and subsequently ACE removes two amino acids from the carboxyl terminus of Ang-I to yield Ang-II. Angiotensin Type 1 Receptor (AT1R) binds to Ang-II and induces cell proliferation, vasoconstriction, blood coagulation, inflammatory responses, and extracellular matrix remodeling, while Angiotensin Type 2 Receptor (AT2R) neutralize AT1R's effects. Angiotensin-(1-7) is a heptapeptide formed by removing the carboxy-terminal phenylalanine from Ang-II. Furthermore, angiotensin-(1-7) can be produced without Ang-II under the alternating actions of ACE2 and ACE. Ang-I is initially digested by ACE2 to generate angiotensin-(1-9), which is subsequently hydrolyzed by ACE to form angiotensin-(1-7) in this metabolic pathway. Endopeptidases and oligopeptidases can directly convert Ang-I to angiotensin-(1-7) [9]. Ang 1-7, unlike Ang II, binds to a G-protein-coupled Mas receptor and has unmistakable vasodilatory, anti-inflammatory, anti-fibrotic, and natriuretic effects. When this system is perturbed, the opposing actions of Ang derivatives maintain a tight physiologic balance, with Ang 1-7 acting to offset the undesirable side effects of unrestrained Ang II activity [10].

ACE2: Receptor for SARS-CoV-2

SARS-CoV-2 may enter cells thanks to the efficient binding of the Spike (S) viral protein, a 1,273 amino acid long protein that belongs to the viral envelope and protrudes outwards with a spike. The (ACE2) has a 'corona' look. (ACE2) receptors are a kind of receptor found in the body [11]. Concluded over *in vitro* studies, S1 domain of the SARS-CoV (S) protein is efficiently bound by ACE2; a soluble version of ACE2, but not ACE1, prevented the S1 domain from binding to ACE2; In ACE2-transfected 293T cells, SARS-CoV reproduced effectively, but not in mock-transfected cells; and Anti-ACE2 antibodies, but not anti-ACE1 antibodies, stopped viral multiplication in Vero E6 cells from African green monkey kidneys, a cell susceptible to SARS-CoV, MERS-CoV, and SARS-CoV-2 infection. In addition, SARS-CoV replication can be supported by exogenous ACE2 expression in refractory cell lines. These findings strongly suggest that ACE2 is a functioning SARS-CoV receptor [12].

Function of ACE2 in pathogenesis of COVID-19

SARS-CoV-2 binds to the receptor of ACE2 in human cells with a high affinity, establishing a connection between COVID-19 and the RAS. SARS-CoV-2 and SARS-CoV are identical save for 380 amino acid changes. Additionally, 27 amino acid changes were discovered in the 1,273-amino-acid S protein, including six in Receptor-Binding Domain (RBD). The van der Waals forces are maintained by a 3-D structure in the S protein, and RBD has a Receptor-Binding Determining Region (RBDR) that targets ACE2. The 394-glutamine residues in the receptor binding domain region of SARS-CoV-2 is known by the crucial lysine 31 residue on the human ACE2 receptor [13]. The preliminary stage in the viral entrance procedure is the attachment of the viral protein unit S1's N-terminal region to a pocket of the angiotensin-converting enzyme 2 receptor. Protein breakdown between the S1 and S2 units, which is carried out by the Hepsin/TMPRSS subfamily member receptor Transmembrane Protease Serine 2 (TMPRSS2), is considered to be necessary for viral entry. Following S1 separation, the remaining viral S2 unit undertakes a

conformational alteration that promotes and completes the viral-cellular fusion, allowing the virus to enter the cell, release its content, replicate, and infect other cells. The fact that camostat mesylate, a TMPSRR2 inhibitor, partially blocks SARS-CoV and SARS-CoV-2 entrance into cells supports the significance of TMPSRR2 [11].

The virion's S glycoproteins attach to the cellular receptor angiotensin-converting enzyme 2, allowing it to go in target cells by an endosomal pathway. In the cytoplasm, the viral RNA is revealed. The viral polymerase protein gets translated from genomic RNA when the viral genome is released. New ssRNA (+) is produced during replication. A nested collection of sub-genomic RNAs (sgRNAs) is created during transcription. Nucleocapsids are formed in the cytoplasm after the structural proteins of SARS-CoV-2 are produced, and then budded in the lumen of the Endoplasmic Reticulum (ER) Golgi intermediate compartment. A mature virion is formed. Finally, virions exit the cell through the cell's constitutive exocytic pathway (Figure 1) [13].

Cardiovascular complications in COVID-19 patients

Arrhythmias: Arrhythmia is also a possibility in COVID-19 patients. Sinus tachycardia is the most prevalent arrhythmia seen in COVID-19 patients. It's unclear if sinus tachycardia is caused by increased cardiac output triggered by fever, hypoxia, inflammatory stress, or medicines, or by anatomical alterations in the heart. In a study of 700 COVID-19 infected patients, there were 25 cases of Atrial Fibrillation (AF), 9 bradyarrhythmia's, and 10 Non-Sustained Ventricular Tachycardia (NSVTs). In addition, ICU hospitalization was linked to AF and NSVT events [14]. Myocarditis, hypoxia, aberrant host immunological response, myocardial strain, myocardial ischemia, electrolyte derangement, intra-vascular volume abnormalities, and medication side effects are all possible causes of arrhythmia in COVID-19 patients [15]. A retrospective analysis of 187 individuals found a connection between underlying CVD and myocardial damage and death outcomes in COVID-19 patients. Eleven individuals (5.9%) had ventricular tachycardia/ventricular fibrillation, according to the 9+ study. Malignant arrhythmias were more conjoint in patients with greater cardiac Troponin (Tn) levels than in the sick with normal (Tn) levels (11.5% *vs.* 5.2%; P<0.001) [16].

Acute coronary syndrome: The prevalence of coronary artery disease in COVID-19 individuals has been found to range from 4% to 25%. Although the specific mechanism has yet to be determined, direct and indirect effects on the cardiovascular organization, for instance SARS-CoV-2 infection, hypoxia, following cytokine storm, coronary spasm, microthrombi, and an overactive immune system, are all probable causes. Acute coronary syndrome may arise as a consequence of these responses in COVID-19 individuals [17]. With severe viral infection, such as SARS and influenza, plaque rupture and thrombus development have been reported, resulting in both ST-elevation and non-ST-elevation myocardial infarction [18]. Because of the near resemblance of diseases, COVID-19 creates a challenge for clinicians treating patients with ACS [19]. Furthermore, cardiac Troponin (cTn) I and T, regulatory components of myocardial cells contractile machinery, are the most often utilized biomarkers for assessing myocardial damage. Any form of cardiac damage can cause substantial cTn release into the bloodstream, but cTn elevation does not allow for differentiation of the pathophysiological processes at play. Several clinical situations involving a mismatch in oxygen supply and demand, such as respiratory failure (predominantly hypoxemia) and infectious illness (especially sepsis), can cause or contribute to type 2 MI [20].

Myocarditis: Acute viral infections are known to cause cardiac damage and acute myocarditis. In a recent study from the PRC's National Health Commission, mononuclear cell infiltrates and myocyte necrosis were present in heart muscle postmortem tissues. Of many cases, there has been an increase in extremely proinflammatory CCR6+ Th17 in CD4+ T cells, which are important inflammatory mediators in myocarditis. The frequency, clinical significance, and mechanism(s) of myocardial inflammation in COVID-19 disease, however, are unknown. Myocarditis can lead to fulminant illness in certain people. Conduction block, tachyarrhythmias, and left ventricular dysfunction can all happen as an end result of myocarditis. If characteristic acute myocardial damage signals are detected on cardiac Magnetic Resonance Imaging (MRI), the diagnosis is typically confirmed. The gold standard diagnostic technique, Endomyocardial Biopsy (EMB), can show mononuclear cell infiltrates and myocyte necrosis directly [21]. Fever, dyspnea, and chest discomfort were the most prevalent symptoms. T-wave variations and ventricular tachycardia were among the EKG abnormalities. There was a lot of LV dysfunction. Severe myocarditis has been connected to a cytokine storm. HF medications, as well as immunosuppressants, glucocorticoids, and IV immunoglobulins, were given to the patients. However, none of these therapies have been put through rigorous testing [22]. Some instances have been linked to fulminant myocarditis. Despite the fact that EMB only indicated moderate inflammation and the COVID-19 RNA was only found in the inflammatory cells, a case of fulminant myocarditis along with cardiogenic shock needing mechanical circulatory support was described [23].

Heart failure: Patient's sick with COVID-19 had concomitant cardiac failure in 23% to 49% of cases. It was especially linked to a poor prognosis; it was shown to be around 5 times more common in people who did not survive their hospitalization (51.9% *vs.* 11.7%). Increase of B-type Natriuretic Peptides (BNP/NT-proBNP) are linked with a worse outcome in patients with ARDS, similar to troponin. Heart failure in COVID-19 might be due to a worsening of existing cardiovascular illness or the progress of cardiomyopathy for the first time (particularly myocarditis or stress cardiomyopathy). In the existence of pulmonary hypertension and severe ARDS or pulmonary embolism, isolated right-heart failure might be seen [24]. SARS-CoV-2 interacts with cardiac tissue and causes direct myocardial injury by attaching the viral glycoprotein Spike 1, after being activated by the host's serine 2 transmembrane protease and binding to ACE2 receptors, expressed particularly in cardiac pericytes, resulting in immediate tissue injury and, later, downregulation of these receptors. The presence of cardiac disease is a significant risk factor for COVID-19 individuals who have acute myocardial infarction. The higher occurrence of ACE2 receptors in postmortem cardiac pericytes isolated from individuals with heart disease compared to those without heart disease led to the notion of a cumulative impact of prior CV illness and troponin rise [25-30] (Table 1).

Potential Drugs against COVID-19

Arbidol (Umifenovir)

Arbidol is a flu treatment that works by binding to the Hemagglutinin (HA) protein. Any structural or sequence similarity between the SARS-CoV-2 spike (S) glycoprotein and the influenza virus (H3N2) HA might lead to a therapeutic effect. A small section of

Table 1: Pro-inflammatory cytokines and their functions.

Sr no.	Cytokine	Family	Function	Reference
1	TNF-alpha	Cytokine family	~ It is a pyrogen set free during severe infection. ~ Important marker for autoimmune disease and chronic inflammation.	[26]
2	IL-6	Cytokine family	~ Anticipate the end result of chronic heart failure and acute coronary syndrome.	[27]
3	IP-10	Cytokine family	~ Playing the chief role in the SARS-CoV-2-encouraged cytokine storm.	[28]
4	CCL2 (MCP-1)	Cytokine family	~ Improves the buildup of neutrophils. ~ Rises procollagen production by fibroblasts.	[29]
5	IL-10	Cytokine family	~ High in patients with acute myocarditis. ~ Play an immune-activating role in covid19 pathogenesis and a pro-inflammatory role.	[30]

the trimerization domain (S2) of SARS-CoV-2 spike (S) glycoprotein (aa947–aa1027) mimics that of H3N2 HA, according to comparative protein sequence analysis. SARS-CoV-2 spike glycoprotein's outer membrane is required for host cell attachment via human ACE2 and CD26 receptors, and its trimerization is required for host membrane fusion [31]. In addition, compared to untreated patients, SARS-CoV-2 infected hospitalized patients cured with arbidol (0.4 g, thrice a day for nine days) had a higher discharge rate and a lower fatality rate. However, retrospective research found that Umifenovir has no effect on COVID-19 patients' outcomes [32].

Remdesivir (GS-5734)

Remdesivir is a nucleotide analog or nucleoside prodrug that has the potential to be effective against numerous viruses at sub-micromolar doses. The most likely broad-spectrum anti-viral Nucleoside Analogs (NAs), RNA-dependent RNA polymerase (RdRp) inhibitors, have proved beneficial in the cure of numerous viral infections. It has been revealed that remdesivir hinders viral replication in SARS-CoV-2, emphasizing the relevance of remdesivir in the medicaments of CoV infections [33]. On May 1st, 2020, the FDA approved an Emergency Use Authorization (EUA) for the cure of hospitalized COVID-19 patients based on preclinical findings and early indications of rapid recovery in clinical studies [34]. In individuals with severe COVID-19, Remdesivir was given for 5 or 10 days. Patients who received additional possibly active medicines against COVID-19 concurrently (within 24 h of commencing remdesivir therapy) were excluded. Administration of Remdesivir (200 mg once a day on 1st day and 100 mg once a day on succeeding days) for 5 or 10 days. 397 sick patients were randomly assigned to one of two groups (200 in the 5-day group and 197 in the 10-day group). Clinical improvement was observed in 64 percent of patients in the 5-day therapy group and 54 percent of individuals in the 10-day therapy group on day 14. This shows that individuals on mechanical ventilation would benefit from a 10-day remdesivir therapy [35].

Lopinavir-ritonavir

Protease enzymes including 3 C-like protease and papain-like protease are required for the persistence of *Orthocoronavirinae* viruses. To aim these protease enzymes, Protease Inhibitors (PIs) such as lopinavir-ritonavir are utilized. In a trial of 199 hospitalized patients with proven SARS-CoV-2 infection, there was no variance in clinical improvement between lopinavir-ritonavir therapy and conventional care. In patients with acute SARS-CoV-2, individual treatment with lopinavir-ritonavir (400 mg/100 mg taken orally two times a day for 14 days) failed to show clinical improvement or lower the virus load [36]. On days 14 and 28, around 45 percent and 40% patients, respectively, were found to have positive viral RNA. Another research had 86 patients who were divided into three groups: i) 34 patients were given Lopinavir/Ritonavir (200 mg/50 mg) twice day; ii) 35 patients were given Umifenovir (100 mg) three times daily; and iii) 17 patients were given no antiviral medication. There were no major variations in improvement rates across groups on many occasions. Despite the absence of statistical significance, the lopinavir-ritonavir group had a lower death rate and spent less time in the Intensive Care Unit (ICU) [37].

Hydroxychloroquine and chloroquine

Chloroquine is a low-cost drug that has been around for a long time, mostly for malaria prevention, with great outcomes and acceptable safety and tolerability. To lower the likelihood of COVID-19 spreading and development, it appears that inhibiting the replication of the SARS-CoV-2 is necessary [38]. SARS-CoV-2 likewise employs ACE2 as a cell entrance receptor, implying that CQ may have a comparable effect on SARS-CoV-2 at this stage of viral replication. CQ may also alter viral replication early on by preventing virus-endosome fusion, most likely by raising endosomal pH. CoVs like SARS-CoV have been found to be able to infiltrate target cells *via* a pH-dependent process in which the lysosome's acidic pH causes the viral and endosomal membranes to fuse resulting in the release of viral nucleic acid into the cytoplasm and the uncoating of viral particles. CQ may also prevent lysosomal protein degradation and autophagosome fusion [39]. In Vero E6 cells, researchers looked at how CQ affected SARS-CoV-2 and half-maximal Effective Concentration (EC50) was found to be 1.13 μM; a Selectivity Index (SI) more than 88.50 and a half-Cytotoxic Concentration (CC50) larger than 100 μM [40]. In COVID-19 type 2 diabetic individuals, HCQ has been demonstrated to produce hypoglycemia. The Food and Drug Administration withdrew the emergency use authorization for CQ and HCQ on June 15th, 2020 in the cure of COVID-19 patients in hospitals, and concluded that neither medicine was likely to be useful in COVID-19 treatment. In addition, substantial use of chloroquine and hydroxychloroquine in COVID-19 treatment was further discouraged by cardiac adverse effects in critical patients and other major adverse effects. However, there is a paucity of reliable clinical data to upkeep the use of CQ and HCQ in COVID-19 therapy. The results of a number of ongoing clinical trials will help to determine the therapeutic implications of Chloroquine (CQ) and Hydroxychloroquine (HCQ) in the treatment of COVID-19 [41].

Favipiravir

Favipiravir (prodrug) is a Nucleoside Analogue (NA) that is transformed to favipiravir Ribofuranosyl-5-Triphosphate (favipiravir–RTP) by metabolic activation via ribosylation and phosphorylation. Viral RdRp then incorporates it into the emerging viral RNA, causing chain termination or the amassing of destructive mutations. Favipiravir attaches to the viral RdRp active site and is mistook for a purine nucleotide, causing the binding pocket to become catalytically non-productive. It stacks on the primer strand's 3' nucleotide and utilizes its amide group to form a non-canonical base pair with the template RNA strand. As a result, favipiravir works as a

chain terminator, limiting viral RdRp by stopping chain elongation at the inclusion site [42]. Favipiravir-RTP acts as a mutagen after RNA viral inclusion, allowing it to avoid coronavirus repair machinery. The favipiravir-RTP therapy increases the stress on CoV nucleotide content, already having cytosine (~17.6%) in SARS-CoV-2 genome. Overall, favipiravir-RTP has a beneficial effect on SARS-CoV-2 by dropping the quantity of viral RNA and infectious particles, as well as increasing the frequency of mutation. With a docking score of -6.925, favipiravir has a significant binding affinity for RdRp. Hence, favipiravir aims the Achilles heel (RdRp complex) of SARS-CoV-2 [43].

Colchicine

Colchicine is a medication that may be useful in the fight against SARS-Cov-2 CS. Gout, Behçet's disease, pericarditis prevention and therapy, familial Mediterranean fever, sweet syndrome, scleroderma, and amyloidosis are all treated with colchicine. The anti-inflammatory characteristics of colchicine are the basis for the scientific notion of its usage in SARS-CoV-2. Colchicine appears to have a possible synergistic effect in the therapy of cytokine storm at various trigger point values. Colchicine, in reality, works by reducing inflammation through a variety of methods. The basic method of action is to attach to tubulin and thereby prevent it from polymerizing. Its anti-inflammatory properties have been related to the disintegration of microtubules into neutrophils, which prevents them from migrating [44]. Furthermore, colchicine can affect the distribution of molecule of adhesion on the surface of neutrophils and endothelial cells, resulting in a considerable suppression of white blood cell-endothelial cell contact by interfering with their transmigration. Because of their potential to interact with the inflammatory protein complex (NLRP3), which plays a crucial role in cytokine storm, the key procedure for reducing cytokine storm in patients with SARS-COV-2 is probably suppression of interleukin-1, interleukin-6, and interleukin-18 production. A component of the SARS-Associated Coronavirus (SARS-CoV) is viroporin E, has been demonstrated to activate NLRP3 inflammation and produce Ca-permeable ion channels, according to research. NLRP3 inflammation is triggered by a variety of pathways and is crucial in the progression of the SARS-CoV-2 phase three cytokinin storms. Upstream suppression of NLRP3 inflammation might be a potential strategy for preventing or treating SARS-CoV-2 infection. Several clinical trials are underway to investigate the effectiveness of colchicine in individuals infected with SARS-CoV-2 [45].

Discussion

In December 2019, Wuhan, a Chinese city, became the hub of an unexplained pneumonia outbreak [1]. The virus was identified as 2019 novel Coronavirus (2019-nCoV) by high-throughput sequencing and later recognized formally as, by the World Health Organization as SARS-CoV-2 (Severe Acute Respiratory Syndrome Coronavirus), producing COVID-19 coronavirus illness [2]. The lung (mostly type II alveolar cells 7) expresses ACE 2 and appears to be the major route of entry [3]. Despite the fact that respiratory difficulties are the most prevalent COVID-19 symptom, these people can also develop cardiovascular issues, which can result in death [6]. The effective binding of the Spike (S) viral protein, a 1,273 amino acid long protein that belongs to the viral envelope and projects outwardly with a spike, allows SARS-CoV-2 to enter cells. The attachment of the viral protein unit S1's N-terminal region to an ACE2 receptor pocket is the first step in the viral entrance process. The Hepsin/TMPRSS subfamily member receptor transmembrane protease serine 2 catalyzes protein breakdown between the S1 and S2 units, is considered to be necessary for viral entry (TMPRSS2). Following S1 separation, the rest of the virus S2 unit goes through a conformational change that initiates and completes the fusion of the viral and cellular membranes, permitting the virus to enter the cell, release its content, multiply, and infect new cells. The fact that Camostat mesylate, a TMPSRR2 inhibitor, partially prevents the entrance of SARS-CoV and SARS-CoV-2 into cells adds to the importance of TMPSRR2 [11]. Some cardio vascular complications are mentioned which are the most common beside the respiratory problems in a COVID-19 individual. Many anti-viral treatments which are currently under clinical trials are employed for the cure of Coronavirus Disease (COVID-19) including the use of anti-viral such as Remdesivir (GS-5734), Hydroxychloroquine and chloroquine, Favipiravir, Arbidol, colchicine, and Liponavir-ritonavir.

Conclusion

In this review, I provided the findings together with plausible explanations for the practice of antivirals in the cure of COVID-19 and the associated cardiovascular symptoms. Patients with underlying cardio vascular problems had a greater chance of SARS-CoV-2 infection progressing more aggressively. Some viruses, including SARS-CoV-2, cause cytokine storms, which cause substantial morbidity and death owing to immunopathology. To summarize, SARS-CoV-2 is a viral infectious illness that mostly expresses itself as fever and pneumonia. While antiviral medicines are the gold standard, we feel that treatments that lower the cytokine response, particularly in more severe patients, may be useful.

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