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Chapter

Impact of Cardiovascular Diseases on the Outcome of Patients with COVID-19

Seeta Devi Akyana and Dipali Dumbre

Abstract

The prevalence of COVID 19 disease cases in India stands too high; this disease is caused by the coronavirus called SARS-CoV-2. Noval coronavirus virus was firstly detected in a group of people suffering from Pneumonia in Wuhan, China. Several studies are conducted to understand the different aspects of novel coronavirus SARS-CoV-2 in causing severe respiratory infections. However, the impact of risk factors on the severity of the symptoms and outcome of COVID 19 is not clearly understood. Similarly, most studies reported that patients who suffer from comorbidities with COVID 19 had a poor prognosis. Most COVID 19 patients who had preexisting medical conditions such as hypertension, diabetes, obesity, smoking habit, etc., required ICU admission and mechanical ventilation. On the other hand, studies reported that COVID 19 infection is responsible for causing the predominant cardiovascular diseases due to myocardial damage, thromboembolism arrhythmias, and ACS.

Keywords: Cardiovascular risk factors, Diabetes, Hypertension, outcome patients, COVID- 19

1. Introduction

Coronaviruses belong to a large family of viruses that causes various illnesses in humans and animals, ranging from the common cold to severe acute respiratory syndrome. COVID 19 is the most recently discovered virus of the coronavirus family. The prominent significant symptoms of coronavirus are cold, cough, fever, headache, and sometimes even diarrhea [1]. It is transmitted from one person to another by tiny droplets from the nose or mouth, expelled by a person with COVID-19 coughs, sneezes, or speaks. These droplets are moderately thick, do not travel far, and quickly sink to the ground [2]. People can also be infected by touching the objects or surfaces with coronavirus and then touching their eyes, nose, or mouth. The person who has contacted a COVID 19 positive and has cold, cough, and fever symptoms needs to be self-quarantined for 14 days to prevent the spread of infection to others within the community. The duration between exposure to COVID 19 and the beginning of symptoms is around five to six days, but it ranges from 2 to 14 days. It is one of the main reasons why the person is quarantined for 14 days after exposure. A research study showed that children and adolescents are as likely to be infected as any other age group and can spread the disease. The severity of the symptoms is equally noticed in all age groups [3].

The effect of COVID 19 among patients with cardiovascular disease conditions and diabetes was found to be high. Similarly, the prevalence of cardiovascular disease conditions was also found to be high among the patients who were infected with COVID 19. The hypothetical reasons are myocardial damage, myocarditis, and cardiomyopathy caused by stress due to COVID 19. The other important causes of cardiovascular problems in COVID 19 are pneumonia, increased cardiac output, electrolyte imbalance, side effects of drugs used to treat the COVID 19 [4].

The infection occurs in myocardial muscles due to damage or injury to the major organs. The inflammation chances will increase, which induces the cytokine storm and vascular hyper-permeability, leading to multi-organ failure and death. The occurrence of cytokines storm is high in patients with diabetes [4].

The thromboembolic action is intense in diabetic patients as it has a significant association with prothrombotic events, which are responsible for causing fibrinolysis and clotting factors. The coagulation activity is likely to be increased further among the patients with COVID 19 diseases. Due to endothelial dysfunction, hypoxia occurs, which can cause intra-vessel coagulation problems [5].

The following are the main cardiovascular risk factors;

1. Diabetes

2. High blood pressure (hypertension)

3. Obesity

4. Family history of CVD

5. Smoking

2. Impact of diabetes on COVID-19 virus

2.1 Pathogenesis of COVID-19 virus

SARS- CoV – 2 is an RNA virus that is the zoonotic source in origin. The transmission of the coronavirus is possible from person to person by respiratory droplets. The symptoms of COVID -19 are range from no signs to mild to moderate. The transmission of coronavirus is possible by specific procedures such as bronchoscopy, endotracheal intubation, and tracheostomy [6].

It enters the human body with S- glycoprotein receptors and binds with the host's ACE2 (Angiotensin-converting enzyme2) receptors, located on the cell membrane. S- Glycoprotein consisting of subunits of S1 and S2 that are found on spikes of the virus [7].

The volume of Furin is more in diabetic patients, which is believed to enhance the viral entry into the human body. The enrichment of the viral entry occurs by the S1 and S2 subunits of spike proteins. Other factors include an acidic environment, and the presence of proteases in diabetic patients increases the replication of the virus [8].

The entry of coronavirus in the body occurs via the pulmonary system. However, it spreads to the other organs comprised of lungs, kidneys, heart, and intestines [9]. Severe acute respiratory syndrome coronavirus (SARS-CoV) and Middle East respiratory syndrome coronavirus (MERS-CoV) cause dangerous respiratory symptoms while other coronaviruses cause the common cold (**Figure 1**) [10].

After entry of the coronavirus into the human body, inflammation begins with the development of cytokines and chemokines. The presence of leukocytosis and

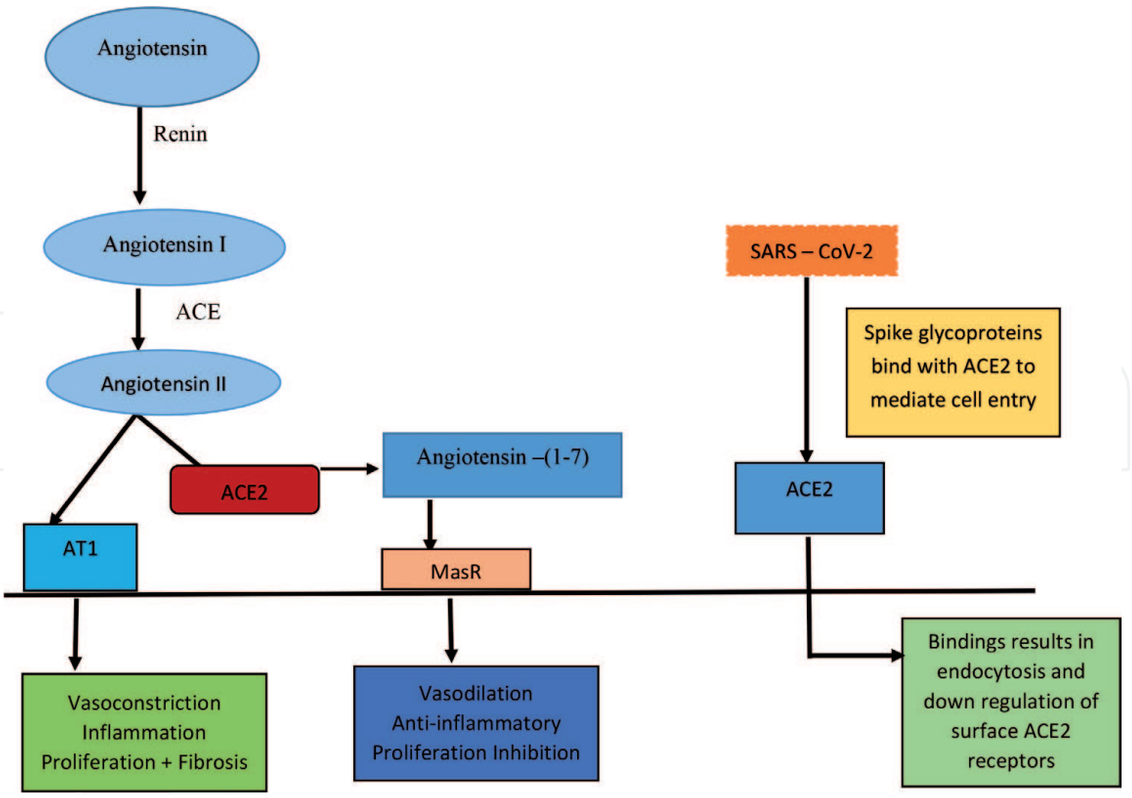

Figure 1.
The role of ACE2 in the regulation of angiotensin system and SARS-CoV-2 infection.

elevated ESR, C- reactive protein levels is commonly observed in laboratory investigation, indicating coronavirus progression in the body [11].

2.2 Pathogenesis of diabetes mellitus

A lack of insulin causes type 1 diabetes, and type 2 diabetes is caused by insulin resistance. The long-term rise of blood glucose levels causes glucotoxicity in body tissues. This mechanism causes chronic complications to include diabetic ketoacidosis (DKA). The treatment of insulin and sulfonylurea cause hypoglycemia [12].

The infection is the primary responsible factor in causing diabetic ketoacidosis and hyperglycemia; thus, it increases the requirement of insulin, leading to cause uncontrolled hyperglycemia. The presence of C-reactive proteins, plasminogen activator inhibitor-1 in laboratory investigations indicate the association of inflammation and diabetes [13].

3. Relationship between diabetes and COVID 19

3.1 Dipeptidyl peptidase-4

Typically incretins, including the Glucagon peptide and gastric inhibitory peptide, are essential to increase insulin secretion. Nonetheless, these incretin values in type 2 diabetes are reduced due to degraded levels of dipeptidyl peptidase-4 in the small intestinal gut. The dipeptidyl peptidase-4 is a transmembrane glycoprotein that exists in the body as a dimer. The primary function of D dimer is to block the degrading enzyme and enhance the activity of incretins in insulin secretion [14].

This enzyme has been indicated as one of the components responsible for the entry receptors of the coronavirus. The place of the enzyme is at the entry of the bronchial tree.

3.2 ACE2 receptors

These receptors play a crucial role in facilitating the entry of SARS-CoV-2 into the human body cells. ACE2 receptors are mainly located on the cell membranes of the lungs, intestine, kidney, and vessels. ACE2 levels are known to be increased in diabetic patients, which is also associated with non-communicable diseases such as diabetes, hypertension, stroke predisposing to develop the SARS-CoV 2 infection [15, 16].

3.3 ACEi and ARBs

ACEi and ARBs are commonly used antihypertensive medications by patients with diabetes and hypertension. These medications are believed to increase the ACE2 levels within the body. However, clinical evidence is yet to be proved [17].

4. Potentials of spreading COVID-19 infection to diabetes individuals

There is adequate evidence to say that people with diabetes at high risk get COVID – 19 infection than the general population. Diabetic patients are more likely to develop complications if they are infected with COVID 19 infection. Their condition worsens if they have other comorbidities, including coronary heart disease, above 60 years old.

Diabetic patients with COVID 19 are at high for the development of serious complications. They are most likely to suffer from moderate to severe symptoms and other complications. These symptoms and complications are less in the patients who have controlled and managed their blood glucose levels well. COVID 19 infection can cause inflammation and endothelial damage; this inflammation is responsible for generating complications in people with diabetes.

The differences in the complications results depend on viral load, host immunity, patient's age, and other long-term diseases. The mortality and morbidity rate are similar in diabetic patients with the corona infection of SARS and MERS.

Diabetic Patients with COVID 19 are at high risk for uncontrollable inflammation due to hypercoagulable response [18]. In Type 2 Diabetes, inflammation induces poor regulation in the homeostatic glucose levels and peripheral insulin sensitivity [19]. Many other factors are also responsibly increasing the severity of SARS-CoV-2 disease in diabetic patients.

5. Effect of diabetes on COVID-19

Patients with diabetes are at high risk for the acquired infection of viruses and bacteria, which affect the respiratory system. The main responsible factor for this increased risk is less function of leukocytes in DM patients. Further, it increases the risk of SARS-CoV-2 inclinations in patients. The lung's deference in terms of gaseous exchange will be impaired due to microangiopathy. This mechanism causes the proliferation of the microorganism in the airway, especially SARS-CoV-2. These changes in the respiratory tract will petrogenetically affect the lung capacity and diffusing capacity of the pulmonary system [20].

Muniyappa and Gubbi concluded that the following mechanisms are responsible for causing the mortality and morbidity of SARS-CoV-2 among patients with diabetes [21].

a. Cellular binding capacity raises the entry of the virus.

b. Reduced leukocyte and the function of T- cells.

c. Vulnerability increased to high inflammation.

Studies conducted in Italy proved that ischemic heart disease and hypertension are commonly observed among patients with comorbidities. A survey conducted in Wuhan regarding the feature of COVID 19 results revealed that the prevalence of COVID 19 about 2–20% is high among the patient's DM and created the requirement of ICU admission about 7.1%. The study results on the clinical features on COVID 19 patients with diabetes reported that approximately 20% of cases reported with COVID 19 positive, and 7% of the cases required the ICU admission. In another study also similar results were observed that 17% and 12.1% accordingly [22].

Hyperglycemia plays a vital role in damaging the endothelial function, causing the cytokine storm and injuries to multiple organs. Patients with COVID 19 and hyperglycemia cause a reduced expiratory volume of the lungs. Phillips et al. [22] results revealed that high blood glucose levels in blood on the respiratory system would reduce its distinctive immune capacity. The cardiovascular mortality rate is also increased due to hyperglycemia by enhancing the inflammatory process in the endothelial system and platelet aggregation [23], in case-controlled blood glucose levels worsen the mortality rate [24].

6. Effect of COVID-19 on diabetes

COVID 19 infection in patients multiplexes the glucocorticoids and catecholamines in the circulation. These are two factors causing uncontrolled glucose levels in the blood, damage the majority of vital organs, and worsen the outcome of the disease.

6.1 Effect of corticosteroids

In case of severe infection, to suppress the inflammatory progress, corticosteroids include hydrocortisone. However, the complications due to administration of these drugs found high comprise as raising 80% of blood glucose levels among diabetic patients and lesser in non-diabetic patients. The mortality rate was higher among diabetic patients with CIVID 19 infection after administration of corticosteroids due to uncontrolled blood glucose levels, necrosis, and psychosis [25, 26]. Glucose monitoring is essential for the patients who all are on the corticosteroids [27].

7. Relationship between hypertension and COVID 19

Hypertension is characterized by increased blood pressure (BP) to the extent of 140/90 mmHg. The essential modifiable factors are associated with the development of cardiovascular illnesses with high BP. Hypertension is one of the primary diseases causing worldwide non-communicable infections [28].

Coronavirus infection mainly influences respiratory tract disease. The spreading of infection is extreme in patients who are at risk of cardiovascular diseases. Hypertension causes massive pathophysiological changes in the cardiovascular system includes left ventricular hypertrophy and fibrosis, which makes parson more susceptible to SARS-CoV-2 [28].

8. Regulation of blood pressure by RAAS

Angiotensin-converting enzyme 2 is a biochemical pathway of the Renin-Angiotensin-Aldosterone System (RAAS), which plays an essential role in regulating blood pressure.

In reaction to stimulation of the sympathetic nervous system, the blood flow to the kidneys and serum sodium levels are reduced. Renin is emitted from the juxtaglomerular apparatus in the kidney.

Renin is an enzyme that converts the angiotensinogen to angiotensin I. Angiotensin-converting enzyme (ACE) converts angiotensin I into angiotensin II (A-II), a potent vasoconstrictor that increases vascular resistance; thus, it increases blood pressure levels.

Angiotensin-II increases blood pressure by stimulating the adrenal cortex to produce aldosterone, which causes sodium and water preservation by the kidneys, bringing about expanded blood volume and expanded cardiac output.

Due to vasoconstriction in blood vessels, blood pressure values that cause atherosclerosis, renal illness, heart hypertrophy) [29].

9. Link between coronavirus and RAAS

RAAS is the neurohormonal pathway that controls blood pressure and fluid balance. The mechanism of the rennin angiotensin is the formation of angiotensin 2, a vasoactive hormone attached to the receptor of the type I angiotensin, which is present in the renal, cardiac, and respiratory systems. It has a vital role in causing the hypertrophy of the myocardium and fibrosis. Further, it causes inflammation, remodeling of vascular endothelium, and formation of atherosclerosis plaque. The Angiotensin-converting enzyme 2 is present in human body tissues, and it affects the mechanism of angiotensin II and the reducing of vasoconstriction effect [29].

SARS-CoV-2 connected to the ACE2 receptor by releasing the spikes protein into host cells causes the regulation of ACE2 and bringing about the local aggregation of angiotensin II. Respiratory severe illness is a sign of COVID-19 and an essential reason for morbidity and mortality; RAAS is proposed to cause an extreme lung injury. Angiotensin-converting enzyme II is considered as the entry for SARS-CoV-2.

The entry of COVID-19 infection in the human body occurs if the virus comes in contact with the angiotensin-converting enzyme II cell surface. This enzyme acts with SARS-CoV-2 by restricting the receptor binding space of the viral spike protein. In the human lung, type II alveolar epithelial cells, ACE2 and transmembrane protease serine III are viewed as fundamentally liable for virus entry in COVID-19 [29].

10. The possible impact of RAS inhibitors on COVID-19

At the start of the COVID 19 episode, the patients with hypertension utilizing angiotensin-converting enzyme inhibitors might provide unfavorable results. The studies mentioned that mineral corticoid receptor antagonists (MRAs) increase the movement of angiotensin-converting enzyme II in the heart and kidney. The antihypertensive medications initiate the ACE2 increase the chances of the portal of entry for the COVID infection [30].

11. Key comorbidities of hypertension comparable to COVID-19

Many research studies have shown that the death rate in COVID 19 was more in patients aged above 50–60 years with hypertension [31].

Obesity with hypertension is also the determinant factor in increasing the seriousness of the disease, which deteriorates the patient's outcome by affecting the expansion of the diaphragm, decreasing the expiratory reserve volume, compromising the ventilation. Obesity increases the release of increased cytokines, causes atherosclerosis, and leads to cardiac problems, which reduces the recovery of the patients with COVID - 19 [31].

12. Coronavirus and thromboembolic problems

The patients with COVID 19 have shown a higher risk of forming arterial and venous thrombus and embolic complications. The patient with severe clinical infection shows the neurological components and the increment level of the D – dimer values, indicating that thromboembolic complications increase mortality among the COVID 19 patients. Among thromboembolic difficulties, VTE (Venous Thromboembolic complication) was a common problem in hospitalized patients [32].

13. Potential mechanisms of SARS-CoV-2-instigated endothelial injury

The organ damage from the coronavirus was mainly related to the inflammatory response; the stimulation of the inflammatory mediator such as cytokine leads to endothelial injury. The virus damages the endothelial cell, directly and indirectly, leading to the endothelial cell's dysfunction and the formation of the clots. Endothelial cell damage increases the permeability of the cell membrane, leading to complications such as acute respiratory distress syndrome and pulmonary fibrosis and pulmonary edema, which is not related to the cardiac problem but due to the increase in vascular permeability. Thus, endothelial injury due to the COVID virus can affect the patient's respiratory function and impacts the patient's recovery [33].

14. Myocardial injury related with COVID-19

There are different types of cardiovascular problems observed in the patient of COVID-19. The issues include congestive cardiac heart failure, dysrhythmia, cardiogenic shock, myocardial infarction, and myocarditis. The studies also stated that the diagnostic studies related to cardiac system problems such as cardiac injury in COVDI-19 patients indicated a positive test of Troponin T, changes in the ECG, and 2d echo. The pathogenesis of the myocardial injury is indescribable in the situation of COVID 19. Due to the viral infection, the inflammatory mediator triggers the inflammatory process, leading to the rupture of the plaque and development of the thrombus and ending in atherosclerotic diseases. The production of the inflammatory mediator comprises of cytokine causes instability in the plaque results in atherosclerosis, and myocardial dysfunction shows clinical conditions like inflammation of myocardium and cardiomyopathy [33].

Apart from this, essential risk factors such as smoking, obesity, hypertension, diabetes, etc., cause atherosclerosis. It increases the viral load in circulation causes

hypoxemia and unusual hemodynamic changes in patients with COVID-19. This mechanism probably acts in corresponding to cause the damage to the cardiovascular system, leads myocardial infarction, ventricular arrhythmias, and congestive cardiovascular failure. Viral infection also affects the respiratory system, which reduces the oxygen levels in the blood. This process alters the hemodynamic parameters, including saturation level, blood pressure changes, leading to cardiovascular complications and atherosclerosis plaque formation. This process of pathogenesis causes significant cardiac damage and significant complications such as myocardial infarction, dysrhythmias, and the cardiac failure [34].

15. Impact of obesity on COVID 19

People with obesity are at high risk for the contraction of COVID – 19 infection. Adipose tissue is increased in ACE2 receptors, which facilitates the SARS-CoV-2 to enter the human cells quickly [35]. The viral load is significantly increased along with the prolonged viremia due to the massive number of adipocytes in obese people. Visceral adiposity causes cytokine proliferation, which causes the low-grade inflammation to advance the cytokines storm in COVID- 19 [36]. Obesity is responsible for reducing individuals' immunity and susceptibility to infection due to various pathogenic organisms. Because increased cytokines in the circulatory system reduce adiponectin levels, thus the immune response to infection is reduced. This mechanism causes damage to the lymphoid tissue and decreasing the B and T cells in the immune system, susceptible to viral infection [37]. Obesity and procoagulant factors are interlinked together that play a vital role in causing thromboembolic complications in patients with COVID-19. Obesity also reduces pulmonary function, reducing reserve volume and respiratory system compliance, at high risk for COVID 19 complications. Obesity with dysfunctional adipose tissue is associated with type 2 diabetes, hypertension, and CVD, which impair individuals' health during COVID 19 infection [38].

16. Impact of family history on COVID 19

There are certain non-modifiable risk factors involved in the pathogenesis of hypertension; one of the essential factors is family history. Various family grounds the inherited character of hypertension examines, exhibiting the relationship of circulatory strain among kin and guardians and youngsters [39].

Hereditary attributes identified with hypertension, for example,

- Counter-Transport in High Level of Sodium and Lithium L,
- Reduced Exertion Of The Urinary Kallikrein,
- Increased Level Of Blood Uric Acid,
- High Blood Sugar Level,
- Changes In The High-Density Lipoprotein And Low-Density Lipoprotein,
- Changes In The Glycemic Index,
- Body Mass Index

- The Environmental Factors Like More Intake Of Sodium In The Diet [40].

The prevalence of CVD and family history are interlinked. The patient with hypertension with a family history is double in value than the patient with no family history [15]. Various types of research indicate that the person having a family history of hypertension leads to premature changes in the cardiac system, including ventricular wall thickness and differences in vascular permeability and stress responses [41, 42].

17. Impact of smoking on COVID 19

The side effect of smoking is prominent, and it causes mortality in all the body system linked with cardiovascular, respiratory systems, and diabetes. Smoking is a significant risk factor for all kinds of cardiovascular infections. It has expanded the risk of getting coronary heart disease. The rate of mortality has increased by 70% from coronary heart disease because of smoking.

It impacts the myocardium, obstructs the blood supply, and increases the chances of atherosclerosis, which directs myocardial infarction and different infections from cardiovascular problems, including cardiomyopathy. The tobacco content incorporated the nicotine and carbon monoxide directly impacts vascular endothelium, which causes inflammation and thrombosis. The majority of individuals with smoking are in danger of developing atherosclerosis.

Smoking has equally been troubled in the progression of corpulmonale. However, a close relationship with congestive cardiovascular breakdown has not been set up.

The proportion of myocardial oxygen supply and demand is influenced by nicotine and carbon monoxide, resulting in vascular endothelial injury, prompting the atherosclerosis plaque's progression [43].

18. Effect of smoking on coronary heart disease

Cigarette smoking is a significant modifiable factor for developing coronary illness. Due to cigarette smoking, the patients breathe tar; this substance contains 4,000 synthetics and cancer-causing agents, including cyanide, formaldehyde, and smelling salts. The nicotine is delivered by smoking which stimulates the sympathetic nervous system, causes peripheral vasoconstriction, and displays tachycardia, hypertension, and increased cardiac workload. Smoking affects the respiratory tract by causing hyperplasia that enhances the abnormal production of mucus. Hyperplasia obstructs the airway due to excessive secretion. Smoking causes the enlargement of the distal air spaces with obliteration of the alveolar walls. Thickening and narrowing of the airway wall cause the inflammatory exudates in the airway lumen.

Carbon monoxide (CO) is a part of tobacco smoke, which absorbs the increased hemoglobin and reduces the O2-carrying capacity to blood. Smoking causes inflammation, vasoconstriction, clot formation, and hypoxia in the endothelial system. Individuals who smoke are at high risk for the development of atherosclerosis. Endothelial injury causes thrombosis [44]. Coronary thrombosis can cause cataclysmic heart damage that leads to sudden death. Nicotine acts on the sympathetic nervous system and decreases myocardial oxygen, causing angina [45]. Tobacco smokers are bound to encounter intense cardiovascular occasions at an early age and prior illness. It has been proved that smoking can adversely affect the lungs, destructing the immune system and making it prone to developing infections.

Smokers are prone to develop pulmonary infections and reduce pulmonary immune function. World Health Organization (WHO) expressed that individual who smokes, carries the fingers to the lips, and that expands the chances of hand to mouth infection transmission, which is generally seen in COVID patients [46].

19. Preventive measures to be followed by patients

- People with diabetes to be careful in preventing the contraction of COVID 19 disease. The recommendations made available for the general public are doubly important for people with diabetes.
- A thorough and regular hand washing is essential.
- Do not touch the face beforehand, washing and drying of the hands.
- The objects or materials are frequently touched to be disinfected thoroughly.
- The food items, clothes, vessels, tools, etc., should not be shared with others.
- The mouth and nose should be closed with tissue when there is coughing and sneezing.
- Avoid exposure with people with symptoms of COVID – 19, such as respiratory infections.
- Try to avoid contact with anyone showing symptoms of respiratory illness such as coughing.
- Unnecessary gatherings and traveling's use of public transportation should be avoided.
- Patients with diabetes to be prepared if they are infected.
- Diabetic patients need to be more cautious in controlling their glucose levels. To prevent diabetic complications, their blood glucose levels to be monitored daily.
- If patients suffer from flu-like symptoms, such as increased temperature, cough, and breathing difficulties, they need to consult health care professionals. If cough consists of phlegm, suspected of infection, they need to get medical advice and treatment urgently.
- Patients need to be ensured for the sufficient supply of diabetic medication; this would help if he has to be quarantined.
- Patients should have enough provisions to correct the hypoglycemia if their blood sugar levels drop down suddenly.
- Diabetic patients need to be advised to adequate sleep and to avoid excessive workout [47].

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20. Conclusion

The amount of the Furin is more in the diabetic patients that promote the entry of virus in the human body.

The enhancement of the viral entry occurs by the S1 and S2 subunits of spike proteins of the virus. ACE2 inhibitors play a crucial role in enriching the entry of SARS-CoV-2 into the human body. They are usually located on the vital organs' cell membranes, including the lungs, intestine, kidney, and vessels. They are known to be more in diabetic and hypertensive patients. ACE2 values are high in the patients using antihypertensive and antidiabetic medications.

Similarly, diabetic patients are at increased risk of acquiring viral infections due to the decreased function of the leukocytes. Further, it enhances the multiplication of SARS-CoV-2. Many studies revealed that diabetes and hypertensive patients infected with COVID 19 causes endothelial destruction and lead to venous thrombus and embolic complications. That leads to an increase in the D – dimer values, which indicates the thromboembolic complications increase mortality among the COVID 19 patients with diabetes and hypertension. The spreading of COVID 19 disease is high in the obesity population, as increased levels of fatty tissues consist of ACE2 receptors that easily facilitate the SARS-CoV-2 entry into the human system. Smoking is also one of the crucial modifiable risk factors in contacting the COVID 19 infection as it destructs the endothelial system and damages the respiratory system. Therefore, through this chapter, we understand an association between cardiovascular risk factors and the outcome of the patients with COVID 19.

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