A Study on Impact of Covid 19 on Respiratory System

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Abstract

COVID-19, which emerged in December 2019, is the global hazard acute respiratory tract infection. There are seven distinct coronavirus strains that induce disease in humans. SARS-Cov, MERS-Cov, and SARS-CoV are three of seven respiratory viruses that can have severe effects. Similar protein structure was discovered in COVID-19 and acute respiratory distress syndrome (ARDS), leading to the coronavirus being dubbed severe acute respiratory syndrome coronavirus-2 (SARS- CoV-2). The initial symptoms of COVID-19 were fever, sore pharynx, a cold, and a dry cough. In addition to vomiting, diarrhea, fatigue, and migraines, some COVID-19 patients also exhibit these symptoms. Polymerase chain reaction (PCR) can be used to diagnose COVID-19 by detecting the coronavirus. Until July 2020, articles were retrieved from MEDLINE, Pubmed, Health STAR, and Google scholar for this study. COVID-19, nCOVID-19, SARS Cov-19, Lungs and COVID-19, Respiratory system and COVID-19, ARDS and COVID 19 were among the key terms. Original research papers, review articles, in vitro studies, clinical studies, and epidemiological studies were included. When article holder websites were analyzed on this topic, more than 500 articles were discovered; however, the number of articles was reduced to 150 based on inclusion and exclusion criteria. When time and other factors were considered, only 69 articles were relevant. This article is reviewed among the 69 collected articles. Today, COVID 19 awareness is crucial for the entire global population. This review may assist the public in gaining a comprehensive understanding of how coronavirus affects the airways.

KEY WORDS: COVID-19, Respiratory System, Polymerase chain reaction (PCR), Real-Time.

INTRODUCTION

COVID-19, which emerged in December 2019, is the global hazard acute respiratory tract infection. The initial manifestation of the infection was in Wuhan, China. It is rapidly spreading to other regions of China, at which point it becomes a significant international warning and affects over 200 countries. The World Health Organization has declared that COVID-19 is a pandemic disease. There are seven distinct coronavirus strains that induce disease in humans. SARSCov, MERSCov, and SARSCoV are three of seven respiratory viruses that can have severe effects. There are four recognized coronavirus subfamilies: alpha-, beta-, delta-, and gamma-coronaviruses. Beta-coronaviruses can cause severe illness in humans. In 2007, a virus was identified as SARS that only effects the respiratory system. Similar protein structure was identified in COVID-19 and acute respiratory distress syndrome (ARDS), resulting in the coronavirus being dubbed severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2). These coronaviruses are transmitted between animals and humans. The primary method by which COVID-19 was transmitted from one person to another was through coughing and sneezing that produced airborne droplets. It can also be transmitted by touching the mouth, eyes, or nostril of an infected person. The initial symptoms of COVID-19 are fever, sore throat, cold, and dry cough. In addition to vomiting, diarrhea, fatigue, and migraines, some COVID-19 patients also exhibit these symptoms. In addition, COVID-19 symptoms include shortness of breath, anosmia, ageusia, congestion, and nasal discharge. As of August 2020, there are 18 million confirmed cases worldwide, 10.7 million recovered cases, and 689 thousand deceased cases.

Polymerase chain reaction (PCR) can be used to diagnose COVID-19 by detecting the coronavirus. Reverse transcription polymerase chain reaction (RT-PCR), real-time reverse

transcription polymerase chain reaction (rRT-PCR), and reverse transcription loop-mediated isothermal amplification (RT-LAMP) are PCR types. Moreover, respiratory tract specimens (such as nasopharyngeal, nasal, and oropharyngeal swabs, sputum, saliva, and bronchoalveolar lavage fluid) are the most obtained clinical specimens for SARSCoV2 detection by real-time quantitative reverse transcription PCR (RT-qPCR).

METHODS

This analysis was conducted using articles retrieved from various databases, including PubMed, PubMed central, Web of Science, Embase, WHO, and Google scholar. They were collected on a time-restricted basis between December 2019 and July 2020. COVID 19, nCOVID-19, SARS Cov-19, Lungs and COVID-19, Respiratory system and COVID-19, ARDS and COVID-19 were the search terms used to locate the articles. Original research papers, review articles, in vitro studies, clinical studies, epidemiological studies, as well as articles with pros and cons, were included. This investigation also included articles that were published in English and Chinese. Retracted articles took exclusion criteria into consideration. All the articles were chosen in accordance with COVID-19 and lungs. They are determined by the article's title, abstract, and entirety. Using a quality assessment instrument, the quality of the utilized articles was evaluated and graded as strong, moderate, or poor.

Consequences of COVID-19 for Health

The novel coronavirus pandemic is the world's largest public health emergency in more than a century. The extremely infectious and highly contagious SARS-CoV-2 causes bioaerosols that transport pathogenic microorganisms, thereby influencing public health [12]. COVID-19 is characterized by respiratory or cardiovascular complications. Bouhanick et al. (2020) hypothesize that diabetic patients with an advanced proinflammatory and prothrombotic state are more susceptible to severe pneumonia. Cardiovascular disease is also a risk factor for the progression and prognosis of COVID-19, When infected with the disease, the latter may exhibit severe pneumonia. In fact, the release of enzymes associated with tissue injury exposes the patient to a higher risk of cytokines by causing hypercoagulability. The status of coronaviruses and their effects on human reproduction, in particular the behavior of male and female gametes. SARSCoV2, a member of the large coronavirus family, causes precise respiratory distress.

Nutritional Support for Patients With COVID-19

Good nutrition is essential for the regulation of immunity in COVID-19-infected patients. The optimal selection of nutrients helps to maintain the immune system's equilibrium and optimize its function. Moreover, an optimal nutritional diet can control oxidative stress positively. For this reason, it is recommended to choose a diet rich in antioxidant-rich vegetables, to prioritize foods with a low glycemic load, to prefer cooking foods with gentle steam, to favor organic food without contaminants, to practice intermittent fasting, and to maintain good life hygiene (exercise regularly, avoid alcohol and tobacco, meditate, and think positively).

ENTRY MECHANISM INTO LUNGS

SARS-CoV-2 predominantly affects the lungs of an infected individual before progressively affecting the other organs. The endocytosis virus enters cells after attaching to the receptor. The viral content is then discharged into the cell, and RNA replication commences. Viruses replicate and mature in host cells in this manner. The coronavirus employs a surface glycoprotein and enters lung cells via angiotensin-converting enzyme 2 (ACE2), a receptor presents on most type II alveolar cells. Angiotensin-converting enzyme 2 (ACE2) has been identified as a functional SARSCoV receptor. Some organs, including the lung, heart, ileum, kidney, and bladder, express ACE2 at elevated levels. On lung epithelial cells, ACE2 was highly expressed in the lung. Below the epithelium are dendritic cells. Macrophages are located on the epithelium's apical surface. Until adaptive immunity develops, innate immune cells like dendritic cells and macrophages fight against viruses.

Thrombosis and pulmonary embolism have also been observed in severe cases. Additionally, endothelial cells express ACE2. Endothelium occupies a significant portion of the lung. Initially, COVID-19 primarily impacts those over 50 years of age, and a high mortality rate has been observed among the elderly. Compared to adult patients, the symptoms of pediatric COVID-19 patients were relatively milder. Based on the correlation between viral burden and age in recent reports, children may have a lower viral load on their host cells. This may be owing to the different levels of ACE 2 expression in the lungs of adults and children. The lung and its epithelial cells continue to develop only after birth. Therefore, ACE 2 expressions in infants may be reduced. ACE2 expression may also be influenced by gender. The levels of circulating ACE 2 are lower in women than in males. This may account for the disparity in morbidity and mortality between males and females in both the adult and pediatric populations. The second possibility is that adolescents respond differently to the SARSCoV2 virus than adults do. Continuous antigen stimulation and thymic involution cause a transition from naive T cells to central memory T cells, effector T cells, and effector memory T cells because of aging. Thirdly, the concurrent presence of other viruses in the mucosa of the lungs and airways, which is common in young children, may allow the SARSCoV2 virus to compete with them and limit its development.

COVID 19 AND ACUTE RESPIRATORY STRESS SYNDROME (ARDS)

Nearly 80% of infected individuals develop influenza-like symptoms, while the remaining 20% may develop pneumonia or severe acute respiratory syndrome. Pneumonia, which is commonly observed in elderly individuals with corona due to diminished lung capacity, diminishes the immune system and affects numerous pulmonary structures. Syndrome of acute respiratory distress (ARDS) is caused by a lung infection or pulmonary trauma. ARDS is diffuse alveolar damage (DAD), so fluid retention in the lungs causes excess, which in turn causes lung damage. ARDS is a condition characterized by a heterogeneous combination of diverse disease processes that results in decreased lung compliance and severe hypoxemia. This results in difficulty breathing and a lack of oxygen reaching the circulation in the affected individual. Reduced oxygen supply to the brain, organs, and tissues of the body can result in a decline in function. Acute respiratory failure caused by COVID-19 was not always ARDS. Some COVID-19-associated ARDS patients who met ARDS Berlin criteria may have relatively normal lung compliance. In addition, the lung compliance of some COVID-19-associated ARDS patients was relatively high, which was inconsistent with the severity of hypoxemia. COVID19 ARDS results in the characteristic ARDS pathological alterations of diffuse alveolar damage in the lungs [19]. Other reports conducted a cohort study on ARDS patients and found that respiratory failure in COVID-19-affected patients has comparable gas exchange, respiratory system dynamics, and response to prone ventilation. Mortality rate from COVID-19 ARDS is caused by respiratory failure (approximately 50%), respiratory failure combined with cardiac failure (approximately 30%), other minor cardiovascular problems (5-10%), and a small number of unidentified causes.

The radiology of ARDS is standard, but COVID-19 pneumonia appears to have distinctive characteristics, such as frosted glass opacities and vascular hypertrophy or enlargement. As the severity of COVID-19 increased, both lungs were afflicted. In imaging, opacities become spherical and are referred to as " COVID balls". COVID-19 begins to recover, with lung opacity also diminishing.

The Berlin definition is used to clinically define ARDS, which includes onset within a week, the presence of diffuse lung opacities on chest radiography, and confirmation of hypoxemia as defined by a ratio of partial pressure of arterial oxygen to fraction of inspired oxygen (PaO2/FiO2) 300 mmHg, at a minimum positive end-expiratory pressure (PEEP) of 5 cmH20. Based on the Berlin oxygenation index, it can be divided into three stages: mild, moderate, and severe. This classification aids in understanding the severity of the disease and the treatment protocol. The clinical characteristics of COVID-19-associated ARDS are still unknown.

Acute respiratory distress syndrome treatment for the most severely afflicted Covid-19 patients will play a crucial role in reducing the disease's mortality rate. About one-sixth of Covid-19 patients have trouble breathing, and approximately forty percent of those with difficulty breathing develop ARDS. Patients with moderate-to-severe COVID-19-associated ARDS should be positioned prone, and patients with severe ARDS may require Ven venous extracorporeal membrane oxygenation (V-V ECMO). WHO advised that high-flow nasal oxygen (HFNO) is safe for COVID- 19-related ARDS patients and that non-invasive ventilation may also be used. Essentially, restore physiological equilibrium by increasing oxygenation and pulmonary compliance and decreasing ventilator-induced lung injury. A positive end-expiratory pressure (PEEP) of at least 5 cmH20 or higher was administered to ARDS patients with enhanced survival rates. WHO recommended that COVID-19 or COVID-19-related ARDS patients should not receive corticosteroids routinely. Although reports indicate that although treatment with methylprednisolone may be advantageous for COVID-19-associated ARDS patients, the effect of corticosteroids in such patients remains unknown and requires further study. Numerous COVID-19 patients receive antiviral or immunosuppressive treatment. In addition, hypertonic saline gargles and nasopharyngeal lavage can be used for the prevention and treatment of COVID-19. This peptide has a potential function in anti-cytokine therapy, such as interleukin (IL)-6 and IL-6R antagonists, which were developed in the United States. In addition, it modulates the immune response of an infected person. Remestemcel-L, a product of Mesoblast Limited, is also being studied for the treatment of ARDS in Covid-19 patients. It reduces the production of pro-inflammatory cytokines while increasing the production of anti-inflammatory cytokines and cells.

ZINC'S ROLE IN RESPIRATORY INFECTIONS AND COVID 19

By regulating the proliferation, differentiation, and maturation of immune cells, zinc plays a vital function in the immune system. It also plays a vital role in numerous enzyme functions, protein catabolism, and DNA synthesis. Normal zinc levels in adults are between 2 and 3 grams, while the recommended zinc intake ranges from 2 mg for newborns to 8 mg for adolescents aged 9 to 13 years. It has been observed that the prevalence of zinc deficiency has increased globally and that it has a significant impact on population health. A severe zinc deficiency can reduce lymphocyte proliferation and, consequently, the body's immune function. Due to zinc deficiency, rheumatoid arthritis, diabetes, and atherosclerosis can occur. Variation in zinc levels can impact immune reactions, leading to increased susceptibility to diseases such as measles, pneumonia, malaria, and tuberculosis. Additionally, animal products such as meat, shellfish, and poultry, as well as fortified breakfast cereal, beans, nuts, and seeds, contain zinc. Vegetables and cereals can reduce zinc absorption, so vegetarians and vegans may require 50 percent more zinc in their diet. Zinc also controls the production of pro-inflammatory cytokines, which regulates the inflammatory response. By modifying the capillary epithelium, cells inhibit the migration of plasma proteins and reduce edema and inflammatory responses. Zinc plays a crucial function in stabilizing the cell membrane, thereby preventing the entry of viruses. Zn supplementation increases the production of interferons (alpha and gamma) and decreases the production of tumor necrosis factor (TNF), according to in vitro and in vivo investigations. Zinc inhibits caspases and increases cellular resistance to apoptosis as a result.

Zinc has a potential function in the direct inactivation of the Varicella-Zoster virus, according to in vitro studies. Zn may also prevent the membrane fusion of respiratory syncytial virus by binding to a specific histidine residue released on the viral E1 protein at low endosomal pH. This is perceived by binding to a specific histidine residue on the viral E1 protein at low endosomal pH. Zn cation reduces RNA replication by inhibiting the RNA polymerase activity of SARS-CoV and other proteins essential for virus life cycle resolution. SARS-CoV and other human coronaviruses, artery-viruses, and porcine reproductive and respiratory syndrome virus (PRRSV)

belong to the Nidoviruses, a large group of viruses with positive-strand RNA (+RNA). Zinc inhibits RNA synthesis and modifies the RdRp activity of nido-viruses in in vitro studies. RdRp and 3CLpro protease of SARS-CoV-2 share over 95% sequence similarity with those of SARS-CoV, even though these two viruses share only 79% sequence similarity. Chloroquine can be used to treat SARSCov2. Based on prior research, chloroquine mediates the zinc influx into the cell, thereby enhancing the immune response. In addition to chloroquine, other zinc supplements with minimal toxicity and fewer side effects are administered for SARSCov2. Numerous clinical trials are currently underway to prove the hypothesis. It suggests that Zn supplementation may be beneficial for COVID-19 prevention and treatment. Due to its immune modulating effect and direct antiviral effect, zinc is considered a potential adjunctive treatment for COVID-19 infection. Zinc supplementation on malignancies inhibits vascular generation activity and stimulates proinflammatory cytokines. Zinc supplementation of 50 mg per day for three months was suggested for cancer patients to strengthen the immune system and prevent this severe COVID-19 infection.

By exposing rodents to Zn, researchers observed a decrease in the lung activity of recombinant human ACE2. Infection with the coronavirus reduces the rhythmic frequency of cilia and ciliary movements, resulting in impaired mucociliary clearance. Zn supplementation can enhance the proliferation of cilia in the bronchial epithelium, allowing it to combat viral respiratory infections.

RECEPTOR FOR ACTYLCYCLIC AMP:

No ACE receptor cells are present on the nose's outermost layer. It resides on the secretory cells inside and outside the nasal filaments. This receptor is also found in the mouth and tongue, in addition to the nostrils. SARS-CoV-2 and COVID-19 indicate that hand-to-mouth transmission is the mode of transmission. TMPRSS2 is a protease that breaks down the coronavirus spike, allowing SARS-CoV-2 RNA to enter nasal cells. As soon as the virus penetrated the host cells, it multiplied into a million copies. Both ACE receptors and TMPRSS have been identified as the finest supporting structures for the upper nose nerve cells. It is essential for scent signals to reach the brain. Nearly sixty percent of COVID-19 patients complained of anosmia. Pneumocytes are an ACE2 receptor found in the lungs. This aids in the production of surfactant, which reduces lung surface tension and makes breathing easier. In COVID-19 cases, the virus destroys pneumocytes, reducing the surfactant burden and causing alveoli collapse and consolidation of the lungs. Therefore, most COVID-19 patients have difficulty inhaling and require a ventilator. In healthy individuals, angiotensin converting enzyme 1 (ACE1) activity is regulated by ACE2 receptors. In severe cases of COVID-19, the lungs develop inflammatory reactions and fibrin blockages. In the presence of an infection, ACE1 converts more angiotensin 2 to angiotensin 1. Angiotensin 2 is a potent vasoconstrictor that directly effects the lungs and causes blood to leak. The lungs of COVID-19 and influenza patients share some characteristics, but the blood vessels of COVID-19 patients exhibit unique characteristics. By harming vascular endothelial cells, COVID-19 causes severe endothelial injury. This results in an abundance of blood clots, thrombosis, and a new pattern of COVID-19-associated pulmonary vascular disease. Due to their diminished immune response and inability to restore the damaged epithelium, the elderly is at high risk.

DANGER OF THROBOSIS

Increases the release of cytokines caused by inflammatory responses in COVID-19. These cytokines stimulate the liver to produce more coagulation factors. The normal fibrinogen level is 2-4g/dl, but individuals with COVID 19 who are severely afflicted have 10-14g/dl. Therefore, their blood is too viscous in nature. Thus, they are highly susceptible to developing thrombosis. COVID-19 pneumonia cases develop pulmonary thrombosis, as detected by computed tomography (CT) pulmonary angiograms; this was discovered by researchers. One of the causes

of a high mortality rate is thrombosis. In addition to Ven thrombosis and pulmonary embolism, immune thrombosis with lung destruction owing to inflammation was observed postmortem in covid 19 [47,48]. In COVID-19 patients, abnormal microcirculation contributes to the clinical outcome. In many nations, autopsy studies revealed the presence of significant microvascular thrombosis. This indicates that many blood clots are present in the lungs' tiny blood vessels. Blood blockages result in diminished blood supply, inadequate oxygenation, and ultimately respiratory failure. Reduced oxygen supply causes alveoli to disintegrate, which in turn leads to silent pneumonia and hypoxia. D-dimer levels may indicate the lung's stage of progression. Covid-19 may contribute to the observed proliferation of new blood vessels and intussusceptive angiogenesis. In cases of inflammation caused by COVID-19, elevated D-dimer levels have been linked to an increase in several associated conditions other than thromboembolism. There is a correlation between inflammation and procoagulant alterations in COVID-19 because all the patients exhibited elevated levels of fibrinogen, D-dimer, and IL-6. Although the precise mechanisms by which COVID-19 induces thrombosis have not been identified, at least some of the well-described mechanisms associated with infection/inflammation are likely to be involved. Thromboelastographic and platelet function tests reveal that COVID-19 patients have activated platelets, suggesting that antiplatelet therapy may be useful. In this case, the potential for doing good is theoretically greater, but so is the possibility of doing damage. Neutrophil extracellular traps (NETs) have been identified in COVID-19 vessel mortem specimens. Increased levels of circulating free DNA and histones activate the prothrombotic pathway, which increases thrombin production. There are no distinctions in ACE 2 receptor expression based on age, gender, or disease severity.

LUNGS AND OTHER ORGANS OF COVID-19:

The scope of post-mortem examination varies between studies, ranging from microbiological sampling without autopsy to limited examination of organs of interest to complete autopsy. Inadequate infrastructure, availability of appropriate biosafety conditions and/or personal protective equipment, and efforts to minimize prosecutor exposure to viruses have all contributed to this variation.

A COVID-19 postmortem lung investigation using a needle lung biopsy stained with hematoxylin and eosin. On CT imaging, bilateral ground glass-like opacifications were observed. Histopathology revealed diffuse alveolar injury, viral protein expression between the alveoli, alveolar epithelial cells, and desquamated cells within the alveolar space. Many of the diseased lungs were enormous, incredibly weighty, and delicate. The pleura was pallid and there were numerous hyperemic areas observed. In most cases, individuals who perished from COVID-19 exhibited diffuse alveolar damage (DAD), which serves as a clinical sign but cannot be observed in all cases. Viruses can also be detected in organs such as the kidneys, cerebellum, and liver. Angiogenesis was greater in deceased COVID-19 patients than in influenza patients. Coronavirus destroys immune organs, effects the lung, and causes systemic vasculitis, as well as alterations in systemic toxicity and secondary infections.

Examined COVID-19 lungs from deceased individuals, they observed diffuse consolidation. In the acute and early proliferative phases, diffuse alveolar damage (DAD) during the exudative/proliferative phase was observed. Additionally, necrotizing pneumonia, intravascular fibrin thrombi, hyaline membrane, alveolitis injury, inflammatory cells including multinucleated giant cells, alveolar hemorrhage, and interstitial inflammatory infiltrate were detected. There was viral content in both type I and type II pneumocytes. CD3, CD4, CD20, CD68, and CD8 lymphocytes penetrate the alveolar septum, as determined by immunohistochemistry (IHC) studies. The Rp3 NP protein of SARS-CoV-2 is highly expressed in alveolar cells, according to an immunostaining investigation with an antibody against this protein.

COVID-19 and sepsis are characterized by a wide spectrum of multiorgan involvement signs and symptoms. Aside from the lungs, the liver exhibited cirrhosis, steatosis, necrosis, congestion, venous flow obstruction, sinusoidal dilation, and newly organized thrombi. Individuals with COVID-19 have elevated alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels and decreased albumin levels. The afflicted liver displayed a larger gallbladder and increased volume. Microvesicular steatosis, mild lobular activity, and patchy necrosis were observed in the postmortem biopsy. Acute tubular injury, collapsing glomerulopathy, and artery-nephrosclerosis were discovered in the kidney. The viral content was detected in the kidney's tubular epithelium, endothelium, and podocytes.

In regions of epicardial inflammation, microvascular thrombi, chronic ischemic cardiomyopathy, and acute infarction were observed in the heart. Mild interstitial fibrosis and myocardial enlargement were also observed. Hem phagocytosis was identified in the bone marrow and spleen. Identified in the brain were cerebral hemorrhage, ischemic necrosis, inflammation, and vascular obstruction. Acute respiratory failure and systemic coagulopathy are crucial aspects of morbidity and mortality associated with severe infections with SARS-CoV-2. The signs and symptoms include shortness of breath, decreased urine output, increased heart rate, alteration in mental status, and physiological variations of organ include increased bilirubin level, acidosis, coagulopathy, and decreased platelet count.

Like the respiratory tract, the ACE 2 receptor is abundantly expressed in the gastrointestinal tract. Specific receptor expression is higher in proximal and distal enterocytes. Children exhibit diarrhea and vomiting more frequently than adults. In the initial stage of infection, most COVID-19 patients complained of loss of appetite, whereas only a small number reported anorexia. The deceased COVID-19 intestine displayed varying levels of mucosal erosion, degeneration, and necrosis. The viral load was detected in stomach, duodenum, and rectal epithelial cells, but not in esophageal cells. It could spread readily through feces. Several COVID-19 patients also undergo stool testing in addition to sample testing. Found that 30% of individuals who tested negative with a nasal swab remain positive with stool samples.

CONCLUSION

The coronavirus disease (COVID-19), which was discovered in December 2019 in China, has attained pandemic proportions; as of June 2021, it has affected more than 171 million people globally and caused more than 3.5 million deaths. The COVID-19 pandemic as a significant health crisis has attracted the attention of numerous researchers, resulting in the development of a comprehensive quantitative picture of human behavior during the coronavirus outbreak. So far, it has been determined, among other things, the psychological symptoms that can result from confinement and the most prevalent coping strategies. What we lack, however, is an in-depth understanding of the changes in coping strategies during distinct stages of the pandemic. In the following study, we utilized a longitudinal qualitative approach to investigate the challenges during the various phases of the coronavirus pandemic as well as the accompanying coping mechanisms.

An infectious virus entering the upper respiratory tract, including the nose, can produce a common cold. In the interim, the throat, larynx, and sinuses may also be affected, manifesting as congestion, sore throat, nasal discharge, sneezing, headache, and fever. The symptoms of COVID-19, which is caused by a novel coronavirus, can overlap with those of the common cold, allergies, and influenza. Like other forms of colds, the virus initially colonized the nose. Coronaviruses are a group of viruses that can cause a range of diseases, from benign colds to more severe conditions like severe acute respiratory syndrome and COVID-19. COVID-19 has similar symptoms to the common cold and influenza, with the exception that it predominantly affects the lungs and causes fever, dry cough, and shortness of breath. Later designated as the new coronavirus pneumonia (NCP), pneumonia caused by the novel coronavirus spread in

December 2019 in Wuhan, China as a highly contagious disease with clinical characteristics very similar to those of pneumonia. WHO has declared the epidemic a "global public health emergency."

Coronaviruses have a high degree of species specificity. Their disease's pathogenesis in humans is poorly understood. Most animal coronaviruses target the respiratory and gastrointestinal epithelial cells. Infections caused by the coronavirus are typically restricted to the upper respiratory tract.

Serious respiratory ailments, including pneumonia and progressive respiratory failure, marked the 2003 SARS outbreak. The virus appears to have originated in a non-human host and has since acquired the capacity to infect humans.

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