The Impact of COVID-19 on General and Dental Health

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Abstract: COVID-19 (Coronavirus disease 2019) is a contagious infection caused by novel coronavirus SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2). This novel virus was publicly announced as an infectious pathogen by the “Chinese Centre for Disease Control and Prevention” on 8 January 2020. The World Health Organization named COVID-19 a pandemic crisis all over the world on March 11, 2020. Aged people and medically compromised people like patients with diabetes, cardiovascular disease, chronic respiratory disease, and cancer are the vulnerable populations for developing an illness. A guideline has been postulated and described in the form of a journey map to avoid exposure of dental health care professionals to COVID-19. This review provides a comprehensive outlook for the current pandemic situation, its origin, spread, and preventive measures to be utilized in general and in dental practice.

Keywords: COVID-19, Pandemic, Preventive Measures, Dental Practice, Respiratory disease, Virus.

1. INTRODUCTION

The recent outbreak of COVID-19 is not a surprising news as everyone is aware of this new war: a war of its kind, a war with a micro-organism! This situation is named an epidemic (when a disease spreads to involve many individuals in a short duration). But clearly, COVID-19 is not an ordinary epidemic. For, this viral disease has spread on the international level and has caused tremendous casualties and a massive economic slowdown across the world. So, we need another term for such a disease that affects humankind on a huge scale globally. A pandemic is “an epidemic occurring worldwide, or over an extensive area, crossing international boundaries and usually affecting a large number of people” [1, 2].

Throughout history, humanity has faced many significant pandemics of diseases; some of the deadliest among them are tuberculosis and the Spanish flu. The Black Death (also known as The Plague) is said to be the most devastating pandemics that occurred in the 14th century and killed about 75-200 million people [3]. One such disease caused by the virus is COVID-19. This disease generally causes mild symptoms but, in several cases, may prove fatal [4]. The significance of knowledge about the co-morbid conditions associated with COVID-19 is dual [5]. Initially, it allows clinicians to modify treatment for their patients, who are vulnerable to severe disease. After that, it helps administrations to develop a facility categorically to address the differential need of patients. This strategic approach can ascertain safety and timely treatment for the vulnerable as well as the general population.

2. WHAT IS A CORONAVIRUS?

Coronavirus is a microbial pathogen with a single-stranded RNA virus in an enveloped capsule. They are capable of infecting many mammals, including humans. They belong to the Coronaviridae family in the order Nidovirales. Tyrell and Bynoe (1966) [6] were the first to describe this virus. They collected the swab from the patients who were ill with common colds. They cultured the viruses and named them coronaviruses based on their morphology. Coronaviruses show crown-like spikes on their outer surface, hence the name Coronavirus (Latin: corona = crown). Coronavirus are comprised of single-stranded RNA. The size of this nucleic material ranges from 26 to 32kbs in length. The size of the whole coronavirus is almost 65–125 nm in diameter. There are seven subtypes of coronaviruses that can contaminate humans. Among them, beta-coronaviruses are the deadliest and may result in fatalities. On the other hand, alpha-coronaviruses mostly result in asymptomatic or mildly symptomatic infections.

Then, a new Coronavirus disease outbreak occurred. The first case was reported in the year 2019. This disease was
termed COVID-19. It is caused by the newly found virus, which is SARS-CoV-2. It is strongly linked with the SARS-CoV and is grouped in the B lineage of the beta-coronaviruses [7 - 9]. Most of the infected people may recover with the basic symptomatic treatment and may not show any major respiratory illness. Aged people and medically compromised people are the vulnerable populations for developing serious illnesses [10].

3. CLINICAL PRESENTATIONS

Lungs are the primary target of the coronavirus. Fever and dry cough are the two main symptoms, which can sometimes lead to breathing problems. If someone coughs continuously for about an hour or has three or more intermittent yet major coughing episodes within 24 hours then the individual could be potentially infected with SARS CoV-2 [11]. This is a different type of cough, unlike the usual one. It is a continuous dry cough with a runny nose.

Symptoms like a sore throat with irritation, headache, nausea, and diarrhoea can also be associated with the primary symptoms [12]. Other symptoms include loss of smell and taste. The incubation period (the period between exposure to an infection and the appearance of the first symptoms) is five days, on average. In some cases, it may take up to 14 days for symptoms to appear, and in some patients, it may remain asymptomatic, who eventually could be the potential carrier of the disease [13, 14].

4. WHY IS COVID-19 HIGHLY CONTAGIOUS?

SARS-CoV-2 has respiratory transmission potential. But the SARS-CoV was almost equally potent. So why is SARS-CoV-2 more contagious than the other?

A single viral particle having the potential for respiratory transmission is not the only factor that contributes to the virulence nature of the pathogen. There are additional aspects, like the count of virus particles emitted by the patient and the surviving ability of the virus (the time for which it can remain unharmed in the atmosphere). Within the family of coronaviruses, this particular coronavirus has the hardest and the toughest external shell as compared to the other viruses. This outer shell exhibits a crucial role in shielding the inner virion. This rigid covering protects the virus and offers higher resistance to the atmosphere and mucus, salivary enzymes present in the oral cavity, various other digestive enzymes, and other bodily fluids. So, the virus can survive and remain viable for an extended period if it has a harder outer shell. Therefore, one can be easily infected even with a slighter number of virulence particles. Moreover, an infected body is probable to discharge more communicable particles as the virus remains viable [15].

Other categories of viruses also exhibit the protective role of the outer shells. Saliva-associated viruses (e.g., Yellow Fever Virus, Zika Virus, Equine Infectious Anaemia, rabies) and viruses associated with the oro-fecal transmission (e.g., the virus causing poliomyelitis) have rigid shell covers. Also, viruses as the smallpox viruses that last for a longer time in the environment, have hard outer shells [16].

5. ORIGIN OF THE VIRUS/COVID-19

By the end of the year 2019, the government of China informed WHO about occurrences of fatal pneumonia with unknown etiology. The first case of the disease was reported from areas around the seafood market in the Chinese city of Wuhan. The disease rapidly infected more than 45 people. Live wild animals like snakes, birds, bats, frogs, marmots, and rabbits are frequently sold at this market [17].

From the analysis of the sample (nasal swab) obtained from the patients, the virus was identified as a novel coronavirus. The diagnosis of viral infection was confirmed based on genetic sequencing. Primarily, it was observed that the people were affected by some virus that leads to fatal pneumonia. On tracing the index case, it was thought that people visiting the seafood market, where live animals were slaughtered and sold, or people may have ingested infected animals or birds are suffering from the novel viral infection [18]. Later, on investigations, it was observed that some of the people are getting affected even without visiting the seafood market. These observations exhibit that this virus was capable of inter-human transmission, which led to the spread of infection worldwide [19].

The spread of the virus from one person to another occurs due to direct contact with an individual infected with the virus. Also, the virus was carried into the droplets of the infected person, and an individual exposed to these droplets via coughing, sneezing, respiratory droplets or aerosols are at high risk for developing the disease. These virus-loaded droplets have the ability to penetrate the nasal mucosa and can travel to the lungs affecting the respiratory system [20].

6. WHO’S AT BIGGER RISK?

A person is at higher risk for contracting SARS-CoV-2 if he/she comes in contact with someone else who is carrying the pathogen, especially when he/she has been exposed to the other person’s expelled salivary droplets or has been in close contact with them while they coughed or sneezed. People with weak immunity remain at greater risk.

Without taking proper preventive measures, the risk significantly increases with [21, 22]:

- Living with a person who has exposed to the virus
- Home care providers who are taking care of the infected person
- Spouse of an infected person
- Elderly and systemically compromised people are also at a higher risk of getting infected and manifesting severe complications if they are exposed to the virus.

These health conditions include [23, 24]:

- COPD (Chronic Obstructive Pulmonary Disease), asthma, and other respiratory diseases
- Cardiac diseases
- Compromised immunity in case of HIV
- Neoplasm that requires management
- Overweight individual
- Individuals with uncontrolled diabetes, renal disease, or hepatic disease.
7. HOW DOES COVID-19 DAMAGE THE KIDNEYS?

The virus has the ability to infect the renal cells by direct attachment to the receptors. This enables the new coronavirus to invade and self-replicate in the cell destroying the tissues. Similarly, cardiac and pulmonary cells are also predisposed to viral infection because of the presence of such receptors targeted by the viruses causing tissue injury [25].

Another reason in patients with kidney problems is low oxygen saturation due to pneumonia in severe cases of COVID-19. The kidneys work as a filter that excretes waste product out of the body. This function of the kidney is impaired by COVID-19 as it results in the formation of tiny clots in the blood vessels, which further may eog the microcirculation of the kidney [26].

8. HOW DIABETES AND COVID-19 ARE RELATED?

First, to enter into the individual, the SARS-CoV-2 virus affects the target cells. These viruses take over through the endocrine pathway to affect the mechanism of blood regulation, metabolism and cause inflammation [27]. The receptor of coronavirus i.e. ACE2 (Angiotensin-converting-enzyme 2) is found on the spike protein, which leads to an inflammatory cascade. COVID-19 viral infection reduces ACE2 expression that results in cell damage, increases inflammation and thereby, induces respiratory distress [28].

Acute hyperglycemia leads to the upregulation of cellular expression of the ACE2 receptor, causing ingress of the virus. Conversely, chronic hyperglycemia leads to downregulation of ACE2 expression that also causes inflammation & adversely damages the cell. Thus, the effect of ACE2 on pancreatic β cells directly hampered the β cell function [29]. Italian colleagues and co-workers recommended that the function of β cell gets affected, which causes insulin deficiency in diabetic ketoacidosis individuals. There are few observations for the requirement of insulin in patients with COVID-19 through various paths of the infection. Up to what extent COVID-19 has been found in relation directly to high insulin resistance is unclear [30].

Secondly, there is a correlation between diabetes and the SARS-CoV-2 virus, which explains the participation of the dipeptidyl peptidase-4 (DPP-4) enzyme in type 2 diabetic individuals. In various cell studies, the authors stated that DPP-4 was found to be a functionally active receptor present for human coronavirus-Erasmus Medical Center (hCoV-EMC) for Middle East Respiratory Syndrome [31]. Antibodies used against DPP-4 inhibit hCoV-EMC infection of primary cells. DPP-4 expressed type II transmembrane glycoprotein with the upregulation of glucose & insulin metabolism. It leads to higher inflammation in patients with type 2 diabetes.

9. HYPERTENSION

Hypertension is another risk factor. The pathway for the virus to enter the body cells is the ACE2 receptor (a target for treating hypertension).

Lei Fang and colleagues (2020) [32] stated that SARS-CoV-2 binds to the ACE2 receptor, facilitating host cell entry, disease severity, and mortality due to COVID-19. Therefore, patients taking angiotensin-converting enzyme inhibitors (ACEIs) and angiotensin receptor blockers (ARBs) are more susceptible to COVID-19 as the ACE2 receptor might be upregulated by ACEIs and ARBs. Thus, patients are at higher risk who are on drugs that are ACE2 inhibitors for severe COVID-19 infection.

10. THE TWO-WAY RELATIONSHIP BETWEEN COVID-19 AND CARDIOVASCULAR DISEASES (CVD)

COVID-19 leads to clot formation, which can adversely affect CVD. Therefore, cardiovascular comorbidities like hypertension, myocardial infarction, coronary artery disease, and thrombo-embolism can be linked with the high mortality of individuals associated with COVID-19. CVD patients on ACE inhibitors and ARB’s drugs are also severely affected by the COVID-19 [33].

11. AGE

Mostly elder patients contracted with COVID-19 come to the hospital with just electrolyte disturbances or mental confusion but no fever or respiratory symptoms. They just feel malaise. This may be because, in many infections, elderly patients do not have the ability to develop a fever as an immune response. Even at home, they are kept isolated and are not active, so they do not experience breathlessness. Once they are admitted, they are even less active and are therefore more susceptible to developing clots [34].

12. ASTHMA AND COVID-19

COVID-19 affects the respiratory system and results in symptoms of a varying degree from being asymptomatic to death. This can be attributed to the difference in the levels of SARS-CoV-2 receptor, ACE2, and the spike protein activator transmembrane protease serine 2 (TMPRSS2) in the respiratory tract, where the entry of the virus into the cells depends on S-protein priming by host cell proteases.

Patients with moderate-severe asthma are suggested to be at higher risk for developing the severe disease during COVID-19; however, there are no reported data supporting this finding to date as stated by the Centers for Disease Control and Prevention [35]. Garg et al. (2020) have reported that amongst the age group of 18-49 years, the asthmatic condition of an individual may increase the risk of hospitalization from COVID-19 [36]. Conversely, a report from New York states that asthma was not present in those who died from COVID-19 [37]. Nevertheless, evaluation of cumulative data recorded worldwide could suggest the association of asthma as a risk factor for COVID-19.

13. OBESITY AS A RISK FACTOR FOR COVID-19

Worldwide the prevalence of obesity is higher than 20% [38]. It has been observed that amongst 5700 patients diagnosed with COVID-19, 41.7% of patients were obese [39]. This can be further related to the fact that ACE2 receptors are present in greater numbers in the adipose tissue, which can be a portal for SARS-CoV-2 into the tissue. More adipocytes in PWo results in a higher load of viruses and prolonged viremia. Adipose cells are also known for the release of pro-
inflammatory cytokines as a host response mechanism. This may contribute to the “cytokine storm” observed during COVID-19. Besides, obesity alters the immune response and predisposes an individual to infection from various pathogens. Along with the raised circulating pro-inflammatory cytokines and lower adiponectin levels, immune response to any kind of infection is downregulated. Impaired B and T cell activity in PwO also increases the susceptibility of an individual to viral infection with delayed recovery. Pulmonary functions like expiratory reserve volume and respiratory system compliance are also decreased in obese individuals, which can also be a reason for obesity as a higher risk factor in COVID-19 [40].

15. PREVENTIVE MEASURES

There are following preventive measures [42]:

- Avoiding or limiting meetings with the person exhibiting the symptoms of COVID-19.
- Practicing good hygiene and social distancing is very effective in the fight against coronavirus.
- Frequently washing hands for a minimum of 20 seconds every time with soap and water.
- Avoiding unnecessary touching on the nose, face, mouth, or eyes.
- Wearing a face mask when coming in contact with an infected person.
- Preventing outdoor visits if a person is showing symptoms of the illness.
- Staying no less than 1 meter away from another individual.
- Covering the mouth with an elbow while sneezing or coughing.
- Staying alert all the i,e without getting panic.

CONCLUSION

COVID-19 has affected the worldwide population irrespective of various factors. From the assimilated data, it is obvious that elderly and systemically compromised people are at more risk of getting infected with the COVID-19. Therefore, utmost preventive measures should be practiced by these individuals to avoid getting the infection, as they generally have a poor prognosis. Every individual other than who is at higher risk should also follow the protocol which includes avoiding going in public areas unless it is necessary, frequent washing of hand with soap and water or use of alcohol-based hand sanitizer, maintaining social distancing, wearing a face mask all the time in a public place and avoiding touching the surfaces or any object and person. Similarly, a standard protocol should be followed while treating a patient in general or in the Dental office.

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CONFLICT OF INTEREST

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