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Corona Virus Global Emergency: An Overview

Shoaeb Mohammad Syed[1]* and R. P. Marathe[2]

[1]Dr. Vedprakash Patil Pharmacy College, Aurangabad, (M.H.) – India

[2]Govt. College of Pharmacy, Ratnagiri, (M.H.) - India

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Abstract

The aim of current review was to explain the novel COVID-19 infection. It covers the basic and history about the virus, how the spreading of infection is possible, the probable mechanism of action, signs & symptoms and methods of prevention. This review also illustrates the how the world is getting affected due the deadly corona virus.

Keywords: Corona virus, COVID-19, Symptoms & treatment

Introduction

History

The first human cases of COVID-19, the disease caused by the novel coronavirus causing COVID-19, subsequently named SARS-CoV-2 were first reported by officials in Wuhan City, China, in December 2019. Retrospective investigations by Chinese authorities have identified human cases with onset of symptoms in early December 2019. While some of the earliest known cases had a link to a wholesale food market in Wuhan, some did not. Many of the initial patients were either stall owners, market employees, or regular visitors to this market. Environmental samples taken from this market in December 2019 tested positive for SARS-CoV-2, further suggesting that the market in Wuhan City was the source of this outbreak or played a role in the initial amplification of the outbreak. The market was closed on 1 January 2020. (Data as received by WHO from national authorities by 10:00 CEST,23 may 2020 World Health Organization). [1-3]

What is Corona Virus?

Corona Virus Disease 2019 (COVID-19) is an RNA virus, with a typical crown-like appearance under an electron microscope due to the presence of glycoprotein spikes on its envelope [4]. It is not the first time that a coronavirus causing an epidemic has been a significant global health threat: in November 2019, an outbreak of coronaviruses (CoVs) with severe acute respiratory syndrome (SARS)-CoV started in the Chinese province of Guangdong and again, in September 2012 the Middle East respiratory syndrome (MERS)-Co V appeared [5]. There are four genera of CoVs: (I) α-coronavirus (alphaCoV), (II) β-coronavirus (betaCoV) probably present in bats and rodents, while (III) δ-coronavirus (deltaCoV), and (IV) γ-coronavirus (gammaCoV) probably represent avian species [4–6].

***Corresponding Author**
E.mail: ybccpsh@gmail.com

The virus has a natural and zoonotic origin: two scenarios that can plausibly explain the origin of SARS-CoV2 are: (i) natural selection in an animal host before zoonotic transfer; and (ii) natural selection in humans following zoonotic transfer [5,6]. Clinical features and risk factors are highly variable, making the clinical severity range from asymptomatic to fatal [7]. Understanding of COVID-19 is on-going.

Mode of Action

To address the pathogenetic mechanisms of SARS-CoV-2, its viral structure and genome must be considered. Coronaviruses are enveloped positive strand RNA viruses with the largest known RNA genomes—30–32 kb—with a 50 -cap structure and 30 -poly-A tail. Starting from the viral RNA, the synthesis of polyprotein 1a/1ab (pp1a/pp1ab) in the host is realized [14]. The transcription works through the replication-transcription complex (RCT) organized in double-membrane vesicles and via the synthesis of subgenomic RNAs (sgRNAs) sequences. Of note, transcription termination occurs at transcription regulatory sequences, located between the so-called open reading frames (ORFs) that work as templates for the production of subgenomic mRNAs [15]. In the atypical CoV genome, at least six ORFs can be present. Among these, a frameshift between ORF1a and ORF1b guides the production of both pp1a and pp1ab polypeptides that are processed by virally encoded chymotrypsin-like protease (3CLpro) or main protease (Mpro), as well as one or two papain-like proteases for producing 16 non-structural proteins (nsps) [15]. Apart from ORF1a and ORF1b, other ORFs encode for structural proteins, including spike, membrane, envelope, and nucleocapsid proteins and accessory proteic chains [14,15]. Different CoVs present special structural and accessory proteins translated by dedicated sgRNAs. Pathophysiology and virulence mechanisms of CoVs, and therefore also of SARS-CoV-2 have links to the function of the nsps and structural proteins. For instance, research has underlined that nsps are able to block the host innate immune response [16]. Among the functions of the structural proteins, the envelope has a crucial role in virus pathogenicity as it promotes viral assembly and release.

The pathogenic mechanism that produces pneumonia seems to be particularly complex [14–16]. The data so far available seem to indicate that the viral infection is capable of producing an excessive immune reaction in the host. In some cases, a reaction takes place, which as a whole is labelled a “cytokine storm”. The effect is extensive tissue damage. The protagonist of this storm is interleukin 6 (IL-6). IL-6 is produced by activated leukocytes and acts on a large number of cells and tissues [17]. It is able to promote the differentiation of B lymphocytes, promotes the growth of some categories of cells, and inhibits the growth of others. It also stimulates the production of acute phase proteins and plays an important role in thermoregulation, in bone maintenance and in the functionality of the central nervous system [18]. Although the main role played by IL-6 is pro-inflammatory, it can also have anti-inflammatory effects. In turn, IL-6 increases during inflammatory diseases, infections, autoimmune disorders, cardiovascular diseases and some types of cancer [19]. It is also implicated into the pathogenesis of the cytokine release syndrome (CRS) that is an acute systemic inflammatory syndrome characterized by fever and multiple organ dysfunction [20]. The virus might pass through the mucous membranes, especially nasal and larynx mucosa, then enters the lungs through the respiratory tract. Then the virus would attack the targeting organs that express angiotensin converting enzyme 2 (ACE2), such as the lungs, heart, renal system and gastrointestinal tract [18–20]. The virus begins a second attack, causing the patient’s condition to aggravate around 7 to 14 days after onset. B lymphocyte reduction may occur early in the disease, which may affect antibody production in the patient. Besides, the inflammatory factors associated with diseases mainly containing IL-6 were significantly increased, which also contributed to the aggravation of the disease around 2 to 10 days after onset. The clinical spectrum of COVID-19 varies from asymptomatic or paucisymptomatic forms to clinical conditions characterized by severe respiratory failure that necessitates mechanical ventilation and support in an intensive care unit (ICU), to multiorgan and systemic manifestations in terms of sepsis, septic shock, and multiple organ dysfunction syndromes

(MODS) [15]. Asymptomatic infections have also been described, but their frequency is unknown. The main symptoms are reported in Table 1. Pneumonia appears to be the most frequent serious manifestation of infection, characterized primarily by fever, cough, dyspnea, and bilateral infiltrates on chest imaging. [22] There are no specific clinical features that can yet reliably distinguish COVID-19 from other viral respiratory infections. Other, less common symptoms have included headaches, sore throat, and rhinorrhea. In addition to respiratory symptoms, gastrointestinal symptoms (e.g., nausea and diarrhea) have also been reported, and in some patients they may be the presenting complaint. Respiratory droplet transmission is the main route and it can also be transmitted through person-to-person contacts by asymptomatic carriers [20].

Main COVID-19-associated symptoms include,

- Fever
- Cough
- Dyspnea
- Headache
- Sore throat
- Rhino rhea

Diagnosis

For patients with suspected infection, the following diagnosis techniques are utilised: performing real-time fluorescence (RT-PCR) to detect the positive nucleic acid of SARS-CoV-2 in sputum, throat swabs, and secretions of the lower respiratory tract samples. In patients with COVID-19, the white blood cell count can vary. Leukopenia, leukocytosis, and lymphopenia have been reported, although lymphopenia appears most common. Elevated lactate dehydrogenase and ferritin levels are common, and elevated aminotransferase levels have also been described. On admission, many patients with pneumonia have normal serum procalcitonin levels; however, in those requiring ICU care, they are more likely to be elevated. High D-dimer levels and more severe lymphopenia have been associated with mortality. Imaging findings—Chest computed tomography (CT) in patients with COVID-19 most commonly demonstrates ground-glass opacification with or without consolidative abnormalities, consistent with viral pneumonia. Others study have suggested that chest CT abnormalities are more likely to be bilateral, have a peripheral distribution, and involve the lower lobes. Less common findings include pleural thickening, pleural effusion, and lymphadenopathy. Chest CT may be helpful in making the diagnosis, but no finding can completely rule in or rule out the possibility of COVID-19. An oropharyngeal swab can be collected but is not essential; if collected, it should be placed in the same container as the nasopharyngeal specimen [18-20]. An oropharyngeal swab is an acceptable alternative if nasopharyngeal swabs are unavailable [20]. Expectorated sputum should be collected from patients with productive cough; induction of sputum is not recommended. A lower respiratory tract aspirate or bronchoalveolar lavage should be collected from patients who are intubated. Data from this study suggested that viral RNA levels are higher and more frequently detected in nasal compared with oral specimens, although only eight nasal swabs were tested. SARS-CoV-2 RNA is detected by reverse-transcription polymerase chain reaction (RT-PCR) [14,20]. A positive test for SARS-CoV-2 generally confirms the diagnosis of COVID-19, although false-positive tests are possible. If initial testing is negative but the suspicion for COVID-19 remains, the WHO recommends resampling and testing from multiple respiratory tract sites [34]. The accuracy and predictive values of SARS-CoV-2 testing have not been systematically evaluated. Negative RT-PCR tests on oropharyngeal swabs despite CT findings suggestive of viral pneumonia have been reported in some patients who ultimately tested positive for SARS-CoV-2. Serologic tests, once generally available, should be able to identify patients who have either current or previous infection but a negative PCR test [20].

Treatment

There is no specific antiviral treatment recommended for COVID-19, and no vaccine is currently available. The treatment is symptomatic, and oxygen therapy represents the major treatment intervention for patients with severe infection. Mechanical ventilation may be necessary in cases of respiratory failure refractory to oxygen therapy, whereas hemodynamic support is essential for managing septic shock . Different strategies can be used depending on the severity of the patient

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and local epidemiology.(12-20)

Fig. 1: Treatment of COVID-19

How the world has affected due to COVID-19?

Till 30 June 2020 the total confirmed cases has been risen to 10, 268, 839 with 506 064 total deaths affecting nearly 206 countries and union territories according to WHO latest updates.[3]

United State is the first amongst all the countries with highest number of positive cases and highest number of deaths followed by Brazil.

Conclusion

The new COVID-19 or corona virus disease spreading at much faster rate and affected the world, currently there is no direct medication available the research and trial is going on the best medicine is to maintain the protocols of social distancing and cleanliness and stay safe also the economy has been fallen low all over the world, the protocols of WHO and concerned govt. should be followed to defense this new COVID-19.

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