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THE 2019 NOVEL PANDEMIC OF CORONAVIRUS DISEASE (COVID-19): A STUDY OF EXISTING EVIDENCE IN THE INDIAN POPULATION

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ABSTRACT

A novel spillover coronavirus (nCoV), with its epicenter in Wuhan, China's People's Republic, has emerged as an international public health emergency. This began as an outbreak in December 2019, and till November eighth, 2020, there have been 8.5 million affirmed instances of novel Covid disease2019 (COVID-19) in India, with 1,26,611 deaths, resulting in an overall case fatality rate of 1.48 percent. Coronavirus clinical signs are fundamentally the same as those of other respiratory infections. In different parts of the world, the quantity of research center affirmed cases and related passings are rising consistently. The COVID-19 is an arising pandemic-responsible viral infection. Coronavirus has influenced huge parts of the total populace, which has prompted a global general wellbeing crisis, setting all health associations on high attentive. This review sums up the overall landmass, virology, pathogenesis, the study of disease transmission, clinical introduction, determination, treatment, and control of COVID-19 with the reference to India.

KEYWORDS: COVID-19, Novel Pandemic, Pathogenesis, Cytokine, Indian population.

INTRODUCTION

Numerous emerging viral diseases have a strong impact on our health and property. Some proofs of our misfortune is the viral episodes, for example, hemorrhagic fever in Crimean Congo, Ebola, Lassa fever, Marburg infection, Severe acute respiratory syndrome corona virus (SARS-CoV), Middle East respiratory syndrome coronavirus (MERS-CoV), Nipah, Zika infection, Rift Valley fever infection, and COVID-19. Many of these outbreaks are strongly affected by global life; they have been declared by the WHO as an international public health emergency; H1N1 (2009), Polio (2014), Ebola in West Africa (2014), Zika (2016) and Ebola in Congo (2019) (1) Coronaviruses (CoVs) are a major group of viruses that, through zoonotic transmission, mostly affect humans. It is the third instance of the appearance of a novel coronavirus in the last two decades, following serious acute respiratory syndrome (SARS) in 2003 and coronavirus (MERS-CoV) in 2012 (2,3). The main causes of concern were the recurrent emergence and global transmission rate, large numbers of deaths, infection and mortality of care providers and healthcare workers (HCWs), and higher risk of death in vulnerable or prone groups (4). Attributable to transmission from indicative/asymptomatic transporters, high portability rates, and fast extension through the world travel framework, the occurrence of COVID-19 contaminations is expanding quickly. High bandwidth and significant death rates are responsible for COVID-19's high potential to turn into a pandemic agent (5,6). In India, most COVID-19 cases have travel history in the infected parts or close to contact with a contaminated individual, transmission to the population is doubtful until now.

AGENT: SEVERE ACUTE RESPIRATORY SYNDROME CORONAVIRUS 2 (SARS-COV-2) AND ITS PATHOGENICITY

The SARS-CoV-2 is a beta-coronavirus belonging to the family of *Coronaviridae*. Essentially azoonotic disease, the first human coronavirus outbreak was recorded in 1965 - HCoV-229E, followed by two outbreaks of similar capacity - SARS-CoV and MERS-CoV in 2003 and 2012, respectively (3,7,8). A novel RNA virus was identified as the causative pathogen by metagenomic sequencing of RNA samples isolated from bronchoalveolar lavage (BAL) fluid in patients with serious acute respiratory disease (SARI) in the city of Wuhan. To date, 11 complete SARS-CoV-2 isolated genome sequences are available. Six of the whole SARS-CoV-2 genome arrangement was confined from different parts of China, and five were disengaged from Japan (9). Seven coronaviruses (229E, NL63, OC43, HKU1, SARS, MERS, and, COVID-19) can contaminate people naturally. Of these four (229ECoV, NL63-CoV, OC43-CoV, HKU1-CoV) are answerable

for gentle upper respiratory contaminations while SARS-CoV, MERS-CoV, and COVID-19 are known for their mellow upper respiratory tract infections. Genome sequences of different isolates are extremely similar and displayed a sequence identity of more than 99 percent. The SARS-CoV-2 genome harbors 10 coding sequences (CDS), which encode polyprotein, surface glycoprotein, membrane glycoprotein, and phospho-protein nucleocapsid. The polyprotein orf1ab, encoded by the isolated from Japanese patients SARS-CoV-2 virus genome, has 24 nucleotide deletion. Genome deletion In the Japanese SARS-CoV-2 virus the untranslated region (UTR) locus and extreme 3'end of the genome were also observed. There is a 27–32 kb capsid, containing a single-stranded RNA envelope-protected genome with positive polarity also presents (13). Coronaviruses contain four sorts of spikes; some long (20 nm) S-spikes of glycoprotein in all Coronaviruses; little spikes of hemagglutinin esterase (HE) in some infections; few transmembrane glycoprotein; and protein spikes in the envelope (14,15). Based on their genome structure the coronaviruses can be divided into three serogroups. Serogroups 1 and 2 may infect mammals and 7 of them (229E-CoV, NL63-CoV, OC43-CoV, HKU1-CoV, SARS-CoV, MERS-CoV, and COVID-19) may infect humans while serogroups 3 may infect avian viruses. The 229E-CoV, NL63-CoV, OC43-CoV, HKU1-CoV is liable for people for 30% or more mild upper respiratory part sicknesses, while SARS-CoV, MERS-CoV, and COVID-19 are notable for serious human diseases. Adapted animal coronaviruses have recently triggered a more lethal infection in humans (16,17). Covid is generally species-explicit. They can grow their genetic variety by supporting recombination and transformation during peridomestic creature diseases and now and then an infection can break the hindrance of the species and furthermore cause human contamination (18). The initiation step of the viral replication process after reaching a reasonable host is the binding of the virion to the receptors on the target cells in the human respiratory tract. The genomic examination recommends COVID-19 would be used related receptors used to treat SARS-CoV (19).The COVID-19 is bound to the angiotensin-converting enzyme 2 (ACE-2) receptor, which is found in the lungs, heart, kidney, small intestine and other tissues, while MERS-CoV uses dipeptidyl peptidase 4 (DPP4), and glycoprotein transmembrane as a receptor (19,20). After that,it will reach host cells by fusion of viral envelope with the plasma membrane or endosomal membranes holding with a typical receptor. The viral genome RNA is released into the cytoplasm after entry and translates into polyproteins pp1a and pp1ab. The translation produced the non-structural proteins in twofold layer vesicles and shaped a replication transcription complex (RTC) (21). A nested collection of subgenomic RNAs that encode accessory and structural proteins is continuously replicated and synthesised by the RTC (22). The newly formed genomic RNA, nucleocapsid proteins, and the envelope of glycol proteins assemble and form viral particle buds. Eventually, to activate the virus, the virion-containing vesicle fuses with the plasma membrane. A number of cytopathic effects (CPE) depending on the viral strains and host cells have been caused by coronavirus infections. Attributed to a variety of factors, the CPE mechanism interferes with the signal pathway, cell function derangement, increased cytokine/chemokine expression, transcription inhibition, and cell protein translation. Many coronaviruses cause fusion of cells, while others cause apoptosis. (23).

SEVERE ACUTE RESPIRATORY SYNDROME CORONAVIRUS 2 (SARS-COV-2) PERSPECTIVES ON IMMUNE REACTIONS

Infection of COVID-19 induces two-phase immune responses: Clinically, SARS-CoV-2 Infection induced immune responses are two phases. A specific adaptive immune response is required during the incubation and nonstop stages to eliminate the virus and prevent disease move to critical levels. So strategies to improve immune responses (anti-sera or pegylated IFNα) are important at this point. The host should be in good general health and a suitable genetic history (e.g., Human leukocyte antigens (HLA) that elicits strong antiviral immunity for the development of an endogenous defensive immune response at the incubation and non-severe stages. It is well known that genetic differences lead to the human variations in the immune pathogen response. However, when a protective immune response is impaired, the virus will propagate and massive tissue destruction will occur, particularly in organs with high ACE2 expression, such as intestines and kidneys (24). The damaged cells cause endogenous lung inflammation which is primarily mediated by pro-inflammatory macrophages and granulocytes. Lung inflammation is the principal cause of serious stage life-threatening respiratory disorders (20). So good general health may not be beneficial for patients who have progressed to a severe stage: once serious lung damage occurs, efforts should be made to suppress inflammation and manage the symptoms. Alarmingly, some patients remain/return viral positive after hospital discharge, and others even relapse. This indicates that it can be difficult to induce a virus-eliminating immune response to SARS-CoV-2 at least in some in these individuals' patients and vaccines cannot work. Those recovered from the non-severe stage should be monitored along with T-cell/B-cell responses for the virus. These scenarios should be

considered when determining the vaccine development strategies. Additionally, there are other coronavirus types or subtypes. And if you take vaccines direct SARS-CoV-2 targeting proves difficult to establish, the Edward Jenner method should be considered (24).

CYTOKINE STORM, DAMAGE TO THE LUNGS

Cytokine release syndrome (CRS) appears to affect severely affected patients. Since lymphocytopenia is frequently seen in severe COVID-19 patients, leukocytes other than T cells have to mediate the CRS caused by the SARS-CoV-2 virus, as in patients receiving Chimeric antigen receptor (CAR-T) therapy; a high White blood cell (WBC) -count is common, suggesting it in conjunction with lymphocytopenia, as a differential diagnostic criterion for COVID-19 (24). Blocking IL-6 can in any case be effective. IL-1 and TNF blockades may also be of interest to patients. While the use of mesenchymal stromal / stem cells (MSCs) in serious cases with COVID-19 infection has been confirmed at various clinical sites in China, solid results are yet to be seen (24).One caveat is that in order to exert its anti-inflammatory effects, which may be absent in severely affected patients as T cells are not well activated by SARS-CoV-2 infection, MSCs need to be activated by interferon-γ (IFNγ) (25). To enhance effectiveness, one could consider employing the "licensingapproach": pretreat MSCs with IFNγ with/without TNF (tissue necrosis factor) or IL- 1(25). Such cytokine-licensed MSCs may be more effective in suppressing the hyperactive immune response and promoting tissue repair since licensed MSCs are effective in acute lung damage caused by lipopolysaccharide (LPS) (26). Lung injury in those serious patients is a significant barrier to recovery. By producing diverse growth factors, MSCs may help regenerate damaged lung tissue (24). It is important to note that different studies demonstrated this in animals models of bleomycin-induced lung injury, vitamin B3 (niacin or nicotinamide) are very effective in preventing damage to the lung tissue (27).

COVID-19 POTENTIAL IN INDIA

The COVID-19 was initiated in India from Kerala by three students returning from Wuhan, China. By, 9th November 2020, more than 8.5 million confirmed cases of COVID-19 from diverse parts of India have been identified. Most of them have a history of travelling to areas affected. Meanwhile the first casualty on 12 March 2020 was confirmed. The first victim was a man who had migrated to Saudi Arabia, aged 76. COVID-19 speeds up the infection rate, more than 8.5 million on November 8th, 2020, and the death toll crossed 1, 26,611on November 8th, 2020. The number of cases is rising steadily compared to other nations, as India is the second most populated, developing nation in the world with insufficient medical facilities. More than 11 crore persons were screen, out of which 8.5 million are positive for COVID-19 by laboratory-confirmed assays, of which 1,26,611 (1.48%) died, 79,17,373 (92.56%) recovered/discharged included one; migrated and 5,96,73(5.96%) active patients admitted to various hospitals (https://www.mohfw.gov.in/seen on November 9th 2020). Maharashtra (1.71M,20.23%), Tamil Nadu (7,41000,8.71%), Delhi (4,31000,5.07%), Telangana (2,50000,2.94%), Rajasthan (2,09000,2.4%), Uttar Pradesh (4,95,000,5.82%), Andhra Pradesh (8,41,000,9.89%),Bihar (2,21,000,2.6%), Karnataka (8,44,000, 9.92%), Gujarat (1,80,000, 2.11%), are most affected states contained 33,41000 out of 8.5M (39.30%) of total laboratory-confirmed cases of COVID-19 (Figure 1).

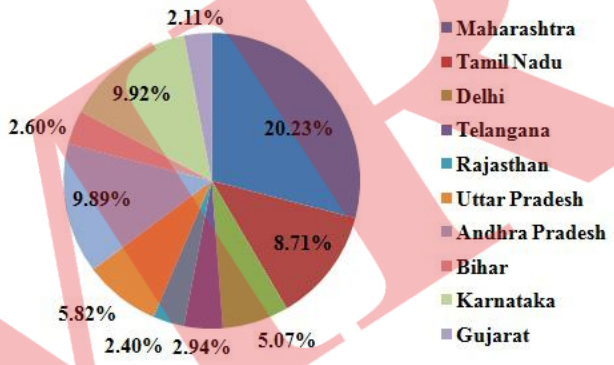

Fig. 1: Distribution Pattern Of Most Affected States Of India. (Source:www.mohfw.gov.in seen on 9th Novemver, 2020).

A total of 1,26,611persons died, out of which97,235(76.79%) were from most affected states like Maharashtra (45,240, 35.73%), Gujarat (3,760, 2.96%), Bihar (1,144,0.90%),Delhi (6,989, 5.52%), Tamil Nadu (11,344, 8.95%), Telangana (1,381,1.09%), Karnataka (11,391,8.99%), Andhra Pradesh (6,791,5.36%), Uttar Pradesh (7,206,5.69%), Rajasthan(1,989,1.57%)(www.mohfw.gov.in).

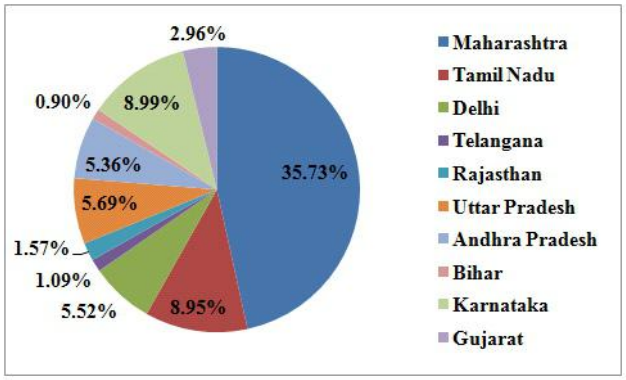

Fig.2: Distribution Pattern Of Death Cases In Most Affected States Of India. (Source www.mohfw.gov.in seen on 9thNovember, 2020)

The distribution pattern of other respiratory tract COVID-19 depends on multiple parameters, such as their basic reproductive number (R0), the nature of the virus, trade/travel in environmental conditions with the affected country, population size and the nature of society. The basic reproductive number (R0) and the existence of the virus are the same for the world as a whole, so the variation in epidemic potential depends on other factors (1). WHO data clearly indicate that colder and wet regions are affected more than warmer and drier regions (https://www.worldometers.info/coronavirus). In India, initial outbreaks have begun in regions that are necessarily related to the affected area through trade/-travel. The number of COVID-19 cases should be higher in expert opinion, but India's diagnostic levels are very small compared to other countries.

CLINICAL SYMPTOMS

The clinical manifestations of COVID-19 are very similar to influenza and other respiratory viruses. COVID-19 can be variable in clinical signs and severity. Three fairly large-scale studies of 278 pooled patients in Wuhan, China suggest that COVID-19 causes viral pneumonia. A latest analysis in Beijing showed that 2 out of the 13 COVID-19 infants aged 2-15 years were pneumonia patients. (28). Co-morbidity including cardiovascular disease, hypertension, diabetes, and age the development of diseases also plays a key role for patients. An adult with no severe co-morbidity was easily recovered while the elderly person was co-morbid with the intensive care needed and a ventilator (26,28). Similar results have been reported in Indian patients. The patients come along infection of the upper respiratory virus unknown. Most patients show fever, cough, sore throat, nasal congestion, malaise, and headache symptoms. The elderly and other patients who are immunocompromised have more and more extreme uncharacteristic symptoms. Though these patients lack dehydration, sepsis, or shortness of breath (kachrooV.Novel Coronavirus (Covid-19) in India). Prognosis of all coronavirus infections is not mandatory because they are self-limited and cause only mild upper respiratory infections.

DIAGNOSIS

The clinical effects of the coronavirus and other respiratory viruses are very similar. It is possible to make their exact diagnosis by laboratory-based assays. Coronavirus can initially be detected by virus isolation, electron microscopy, serological procedures, and molecular assays are prioritized in later stages (after genomic sequences). Virus isolation is not a reliable method of diagnosis, but it has provided a good quantity of viruses that can be used for further antigenic and molecular characterizations. Serological methods are good, but validation involves serum pairs. In modern times, molecular diagnostic assays for viral diagnosis are faster, more sensitive, more specific, and more cost-effective (29,30). COVID-19 was diagnosed with RT-PCR in real-time, after complete availability of genomic virus series (World Health Organization. Laboratory testing for 2019 novelcoronavirus (2019-nCoV) in suspected human cases: interim guidance. Geneva: WHO. 2020). In India the suspected individuals are selected according to country and/or WHO guidelines; the required samples should be obtained during the early infection process. After careful wearing of personal protective equipment (PPE) the samples will be taken together (WHO coronavirus (2019-nCoV) Situation report). The collected samples should be transmitted in chilly conditions in the virology laboratory. In the virology laboratory, the sample is processed and standardised assays are performed for diagnosis. In India, all the diagnoses of COVID-19 made in over one hundred NABL accredit laboratories for virology. COVID-19 is diagnosed by RT-PCR in real-time using the World health organization (WHO) Protocol and Indian Council of Medical Research (ICMR) Guidelines.

TREATMENT AND AVOIDANCE

For any virus, an efficient vaccine or antiviral might be the most appropriate response. Without these, preclusion is merely the choice. MERS, SARS, Nipah (31) Zika (32) outbreaks give us a lesson that social isolation, regular hand-washing, avoiding interaction with animals and infected persons should protect us. COVID-19 is similar to other respiratory viruses and can be transmitted through droplets, tainted body parts and targets from infected to susceptible individuals. Via these infected targets, a susceptible person acquired the infection, amplified the virus, and spread it to other people. In the absence of treatment; the patients may be given quarantine and supportive care (oxygen therapy, fluid management, antibiotic antimicrobials, etc.). Some antivirals have been tried to control 2019-nCoV, such as Remdesivir, lopinavir-ritonavir, and interferon-a 2b, as well as other medications, but predicted results have not been achieved. No specific antiviral against COVID-19 is available.On SARS-CoV-2, chloroquine and hydroxychloroquine were found to be effective and confirmed to be effective in Chinese COV-19 patients (33). Hydroxychloroquine (Dose 400 mg BD for 1 day, 200 mg BD for 4 days) may be given under close medical supervision with the combination of Azithromycin (500 mg OD for 5 days) (https://www.mohfw.gov.in). The Indian Council of Medical Research's (ICMR) National Task Force for Covid-19, in a March 21, 2020 newsletter (34)

recommended the use of hydroxychloroquine for prophylaxis in asymptomatic healthcare workers who care for suspected or confirmed patients and household contacts of confirmed patients. That is cause for concern, both in terms of bioethical approach and good clinical practice. Firstly, no pre-clinical or clinical evidence is yet available to demonstrate the effectiveness of hydroxychloroquine as a prophylactic agent for Covid-19 (https://www.cdc.gov/coronavirus/2019-ncov/hcp/therapeutic-options.html).

CONCLUSION

The emerging COVID-19 virus, initiated in Wuhan, China, has spread to more than 210 countries around the globe, including India. The clinical effects of COVID-19 are very similar to other respiratory viruses. The number of laboratory-confirmed cases and associated mortality is regularly rising in different parts of the world. Luckily, In contrast to SARS, COVID-19 mortality is low and MERS recovers the majority of its cases.

To date, there are 11 complete isolated genome sequences of SARS-CoV-2. Six of the entire genome sequence of SARS-CoV-2 was isolated from various parts of China, and five from Japan. COVID-19 infection induces two-phase immune responses: Clinically, two phases are immune responses induced by SARS-CoV-2 infection.

Cytokine release syndrome (CRS) appears to affect patients who have been severely impacted. Since lymphocytopenia is frequently seen in severe COVID-19 patients, the CRS caused by the SARS-CoV-2 virus must be mediated by leukocytes other than T cells, as in patients receiving CAR-T therapy; a high WBC-count is common, suggesting it in conjunction with lymphocytopenia as a differential diagnostic criterion for COVID-19.

Medical signs of COVID-19 are extremely close to those of influenza and other airborne viruses. Medical signs and frequency of COVID-19 can be variable. Following, the full availability of genomic virus series, COVID-19 was diagnosed with RT-PCR in real-time.

Several antivirals have been attempted to control the 2019-nCoV, such as Remdesivir, lopinavir-ritonavir, among interferon-a 2b, and other drugs, but predicted results have not been achieved. What's more, future outbreaks of zoonotic viruses and pathogens are probably going to start. Hence, apart from curbing this outbreak, efforts should be made to formulate systematic steps to avoid potential zoonotic origin outbreaks.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

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