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Novel Coronavirus Disease 2019 (COVID-19): A Review of Recent Literature

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ABSTRACT: Coronaviruses are responsible for various diseases in mammals and birds ranging from enteritis in cows and pigs and respiratory tract infections in chickens to potentially deadly human respiratory infections. Every decade has witnessed the evolution of a new coronavirus epidemic since the last three decades. Coronaviruses are known to have high transformation and recombination rates, which may permit them to cross species boundaries and adjust within new hosts. The human airway route fills in as the primary passage point for these respiratory virus infections. The objective of this review article was to have an initial opinion about the COVID-19, the methods of diagnosis & treatment, and prevention in this early stage of the disease.

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INTRODUCTION

In December 2019 several pneumonia patients of unknown origin were admitted in hospitals of Wuhan City, Hubei Province, China and most patients have a history of visiting Wuhan South China Wholesale Seafood Market [1]. Health authorities of Wuhan city and Chinese Center for Disease Control and Prevention (China CDC) reported an outbreak of pneumonia of unknown cause in Wuhan City On 31 December 2019 [2,3]. On 7th January, 2020, the Chinese Center for Disease Control observed a novel coronavirus from collected samples of patient's lower respiratory tract and disclosed the genomic sequence on 11th January2020 [4]. After that novel coronavirus named severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) but the World Health Organization (WHO) named this infection, caused by SARS-CoV-2 identified in 2019 as Coronavirus Disease 2019 (COVID-19) [5].

In the 1930's numerous coronaviruses were identified in domestic poultry, cause animal respiratory, liver, gastrointestinal and neurological diseases. Among humans only seven coronaviruses are known to cause disease. Four of the 7 coronaviruses cause indications of the normal cold most of the time. Coronaviruses 229E and OC43 cause the normal cold; the serotypes NL63 and HUK1 have likewise been related with the common cold. Once in a while, extreme lower respiratory tract contaminations, including pneumonia, can happen, basically in new born children, older people and the immunocompromised. Three of the 7 coronaviruses cause significantly more extreme, and sometimes fatal, respiratory diseases in people than different coronaviruses and have caused significant outbreaks of fatal pneumonia in the 21st century. SARS-CoV was identified in 2002 as the cause of an outbreak of severe acute respiratory syndrome (SARS). MERS-CoV was identified in 2012 as the cause of Middle East respiratory syndrome (MERS). SARS-CoV2 is a novel coronavirus identified as the cause of coronavirus disease 2019 (COVID-19) that began in Wuhan, China in late 2019 and spread worldwide. These coronaviruses that cause serious respiratory diseases are zoonotic pathogens, which start in contaminated creatures and are transmitted from creatures to individuals [6].

NOVEL CORONAVIRUS 2019 (COVID-19)

Coronaviruses are encompassed positive sense single-stranded RNA viruses measured 90-200nm. in diameter. The wrap

bears crown-like, 20nm. long spikes that look like crown of the sun under electron microscopy, henceforth given its name coronavirus. The infection can cause ailment both in animal and in human. It conveys the biggest genome among the presently known RNA infections [7-9]. Coronaviruses are members of the subfamily *Coronavirinae* in the family *Coronaviridae* and the order Nidovirales. This subfamily divides into four genera – Alphacoronavirus, Betacoronavirus, Gammacoronavirus and Deltacoronavirus. Comparison of the genome sequences of the COVID-19, SARS-CoV, and MERS-CoV showed that 2019-CoV has a better sequence identity with SARS-CoV than the MERS CoV [10].

The COVID-19 amino acid sequence varies from other coronaviruses exclusively in the regions of lab polyprotein and surface glycoprotein or S-protein. S-protein has two subunits with one subunit binding directly to the host receptor aiding the virus entry into cells. The RNA binding domain of the S-protein in COVID-19 has a higher homology with SARS CoV. Though some of the residues critical for binding the receptor are different, overall, the non-identical residues did not alter the structural conformation. Studies suggest that the human receptor for COVID-19 could be angiotensin-converting enzyme 2 (ACE2). Other coronaviruses including SARS-CoV gain entry into human cells through ACE29 [11-14].

HUMAN-TO-HUMAN TRANSMISSION [15]

Human-to-human transmission of SARS-CoV-2 has been confirmed during the 2019-20 coronavirus pandemic [16-18]. Transmission occurs primarily via respiratory droplets from coughs and sneezes within a range of about 2 metres (6.6 ft) [19]. Indirect contact via contaminated surfaces is another possible cause of infection [20]. Preliminary research indicates that the virus may remain viable on plastic and steel for up to three days, but does not survive on cardboard for more than one day or on copper for more than four hours; the virus is inactivated by soap, which destabilizes its lipid bilayer [21, 22]. Viral RNA has also been found in stool samples from infected people [23, 24].

CLINICAL CHARACTERISTICS OF COVID-19

The median incubation period was 5.1 days [25], ranged from 2-14 days [26]. An analysis of household transmissions revealed that fever and respiratory symptoms appeared 3-7 days after exposure to the virus. Fever, dry cough, and fatigue were more commonly reported, whereas nasal congestion, rhinorrhoea, sore throat, and myalgia were relatively rare [27]. Occasionally, non-respiratory symptoms such as palpitation, diarrhoea, or headache preceded respiratory symptoms. Some patients were initially afebrile. Clinical spectrum of COVID-19 ranged from asymptomatic to fatal pneumonia. The rate of asymptomatic infection is yet to be defined, since most initially asymptomatic infections eventually turned symptomatic [28-30]. Risk elements of serious pneumonia or demise incorporate ages 60 or more older, and clinical comorbidity, for example, hypertension, diabetes mellitus, cardiovascular sickness, or malignancy. Lab trial of the affirmed COVID-19 cases indicated leukopenia, lymphopenia, mellow raised C-receptive protein. But the patients with extreme pneumonia had elevations in leukocytes, neutrophils, and creatinine kinase. Computer tomography (CT) uncovered ground glass appearance, interstitial penetration, or multiple patchy consolidations in both lungs [31-34].

LABORATORY DIAGNOSIS

Nasal emissions, blood, sputum, and bronchoalveolar lavage (BAL) gathered from suspected patients are utilized as clinical examples. The examples are exposed to explicit serological and molecular tests specific for COVID-19 for laboratory diagnosis. Serological tests utilize enzyme linked immunosorbent assay (ELISA) or Western blots that detects specific COVID-19 proteins. Molecular approaches are based on Real Time-PCR (RT-PCR) or Northern blot hybridization targeting specific COVID-19 genes. Viral antigens present in the clinical specimens are detected by using direct immune fluorescent assay (IFA) [35].

TREATMENT AND MANAGEMENT OF PATIENTS WITH COVID-19 INFECTION

In general, there are few or no treatment options for viral diseases that occur suddenly [36]. In parallel with this knowledge clinical care of suspected patients with 2019-nCoV should focus on recognition of the disease condition at the earliest, isolation and adoption of proper infection control measures, and delivery of optimized supportive care toward the suspected/confirmed cases. For preventive measure, the WHO guideline “Clinical management of severe acute respiratory infection when novel coronavirus (2019-nCoV) infection is suspected” mainly focuses on avoiding close contact with persons suffering from acute respiratory infections, frequent hand wash, and avoidance of unwanted contact with wild animals [37, 38]**.** Today there is no vaccine or effective treatment to prevent COVID-19 infection. Molecules are being tested for COVID-19 in in-vitro and human-based SARS-CoV and MERS-Cov trials. Initially, interferons-α nebulization, broad-spectrum antibiotics, and anti-viral drugs were used to reduce the viral load [39-41]

however, only Remdesivir has shown promising impact against the virus [42]. Remdesivir only and in combination with chloroquine or interferon beta significantly blocked the SARS-CoV-2 replication and patients were declared as clinically recovered [43-45]. Various other anti-virals are currently being evaluated against infection. Nafamostat, Nitazoxanide, Ribavirin, Penciclovir, Favipiravir, Ritonavir, AAK1, Baricitinib, and Arbidol exhibited moderate results when tested against infection in patients and in-vitro clinical isolates [46, 47]. Several other combinations, such as combining the antiviral or antibiotics with traditional Chinese medicines were also evaluated against SARS-CoV-2 induced infection in humans and mice. Recently in Shanghai, doctors isolated the blood plasma from clinically recovered patients of COVID-19 and injected it in the infected patients who showed positive results with rapid recovery [48, 49].

THE SARS-COV-2 VACCINE [50]

There is no available vaccine against COVID-19, while previous vaccines or strategies used to develop a vaccine against SARS-CoV can be effective. Recombinant protein from the Urbani (AY278741) strain of SARS-CoV was administered to mice and hamsters, resulted in the production of neutralizing antibodies and protection against SARS-CoV [51, 52]**.** The DNA fragment, inactivated whole virus or live-vectored strain of SARS-CoV (AY278741), significantly reduced the viral infection in various animal models [53-57]. Different other strains of SARS-CoV were also used to produce inactivated or live-vectored vaccines which efficiently reduced the viral load in animal models. These strains include, Tor2 (AY274119) [58, 59], Utah (AY714217) [60], FRA (AY310120) [61], HKU-39849 (AY278491) [62], BJ01 (AY278488) [63], NS1 (AY508724), ZJ01 (AY297028) [64], GD01 (AY278489) [65] and GZ50 (AY304495) [66]. However, there are few vaccines in the pipeline against SARS-CoV-2. The mRNA-based vaccine prepared by the US National Institute of Allergy and Infectious Diseases against SARS-CoV-2 is under phase 1 trial.

Despite the fact that exploration groups everywhere throughout the world are attempting to research the key highlights, pathogenesis and treatment alternatives, it is considered important to concentrate on competitive therapeutic options and cross-obstruction of other vaccines [67].

CONCLUSION

Inside six months since the revelation of a novel coronavirus in patients with pneumonia COVID-19 has affected quickly all through the world and is beating SARS-CoV and MERS-CoV in the quantity of confirmed cases and deaths. Researchers are looking for powerful and appropriate vaccine candidates and therapeutics for controlling the deadly COVID-19. There are no effective vaccines or specific antiviral drugs for COVID-19. Henceforth, we need to depend solely on enforcing strict preventive and control measures that minimize the risk of possible disease transmission. Present day clinical information and innovation empowered us to expeditiously recognize the earlier obscure pathogen, share the genomic data of the virus, create analytic tools, identify the illness, detach patients and give suitable clinical consideration. In any case, a few unanswered questions stay to be tended to. The world needs to endeavor to slow the spread as well as put resources into improvement of treatment and antibodies for Novel Coronavirus disease 2019. Further research should be directed toward the study of SARS-CoV-2 in suitable animal models for analyzing replication, transmission, and pathogenesis.

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CONFLICTS OF INTEREST

The authors have no conflicts of interest.

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