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Review Article

Current Update of Severe Acute Respiratory Syndrome Coronavirus 2

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Abstract

Coronaviruses are group viruses belong to Coronaviridae family the order Nidovirales, and the genus Coronavirus. They are the largest group of viruses causing respiratory infections. There are four genera under this family; Alpha coronavirus, Beta coronavirus, Gamma coronavirus, and Delta coronavirus. However, most of the human coronaviruses produce mild illness in the upper respiratory tract while some strains are lethal and can cause Severe Scute Respiratory Syndrome (SARS), Middle East Respiratory Syndrome (MERS) and coronavirus disease 2019 (COVID-19). The most recent and rapidly evolved coronavirus associated disease in human is COVID-19 caused by SARS-Corona virus-2 (SARSCoV-2).

Keywords: Coronavirus; Beta coronavirus; Respiratory infections; Severe acute respiratory syndrome coronavirus-2.

Introduction SARS CoV 2

Coronaviruses are group viruses belong to Coronaviridae family can produce diseases in human and animal. There are four genera under this family; Alpha coronavirus, Beta coronavirus, Gamma coronavirus, and Delta coronavirus. However, most of the human coronaviruses produce mild illness in the upper respiratory tract while some strains are lethal and can cause Severe Acute Respiratory Syndrome (SARS), Middle East Respiratory Syndrome (MERS) and coronavirus disease 2019 (COVID-19) [1]. The most recent and rapidly evolved coronavirus associated disease in human is COVID-19 caused by SARS-Corona virus-2 (SARSCoV-2) [2]. The nucleotide variations of the COVID-19 causing coronavirus are closely related with SARS coronavirus (SARS-CoV), therefore it has been named SARS-CoV-2 [3]. The SARS-CoV-2 is extremely infectious and contagious compared to SARS-CoV which was affected in 2003 [1]. Genetic analysis of all of these viruses suggests that they all have their ecological origin in bat population, and transmission of the virus to humans has likely occurred through an intermediate animal host. This intermediate animal host might be a domestic animal, a wild animal, or a domesticated wild animal that has not been identified clearly yet.

Structure

Coronaviruses encode four structural proteins that are incorporated into mature enveloped virions: the trans membrane Spike (S), Membrane (M), and Envelope (E) proteins, and the soluble Nucleocapsid (N) protein [13]. Spike protein has been imaged at the atomic level using cryogenic electron microscopy and protrude from the virion, creating the eponymous corona in electron micrographs, and mediate fusion of viral and host cell membranes and responsible for allowing the virus to attach to and fuse with the membrane of a host cell [13,14]. E proteins form cationicviroporins that promote viral assembly and modulate the host immune response. N is an RNA-binding protein that packages the viral RNA genome. M organizes the assembly and structure of new virions and is essential for virus formation, virions are helically shaped and each SARS-CoV-2 virion is 50–200 nanometers in diameter. M is the most abundant membrane protein in the viral envelope and anti-M antibodies are found in plasma of patients infected with SARS-CoV-2 and other coronaviruses [14,15].

The SARS-CoV-2 genome is a non-segmented large positive-sense single-strand RNA virus that belongs to the beta coronavi-

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rus genus and with a length of about 30 kb. It contains a 5'- cap structure and a 3'-poly-A tail [5,10]. The viral genome of SARS-CoV-2 encodes 29 proteins, including 25 multiple non-structural and four structural proteins. The leader sequence and Open Reading Frame (ORF) encode non-structural proteins for RNA replication and transcriptions. The downstream regions of the genome encode structural proteins, including the spike (S) protein, the nucleocapsid (N) protein, the envelope (E) protein, and the membrane (M) protein [3,6]. Remarkably, the genome of SARS-CoV-2 shares 80% sequence homology with that of SARS-CoV, a corona virus that caused a large scale pandemic infection before 18 years ago [7].

Near 15,000 mutations have occurred on SARS-CoV-2 [8]. More than 1000 mutations on the S protein gene have a significant impact on SARS-CoV-2 infectivity [9,10]. These mutations should beput into the perspective that COVID-19 has globally spread. The geographical and demographical diversity of the viral transmission and exogenous and endogenous genotoxins exposures have stimulated SARSCoV-2 mutations. If we consider the average number of mutations per genome, SARS-CoV-2 is mutating slower than other viruses, such as the flu and common cold viruses. This is because SARS-CoV-2 belongs to the coronaviridae family and the Nidovirales order, which has a genetic proofreading mechanism in its replication achieved by an enzyme called non-structure protein 14 (NSP14) in synergy with NSP12, i.e., RNA-dependent RNA polymerase (RdRp) [11]. As a result, SARS-CoV-2 has a relatively high fidelity in its transcription and replication process. In general, Coronavirus mutations are created from three major sources, namely, random errors in replication, such as genetic drift and spontaneous genotoxins, viral replication proofreading and defective repair mechanisms, and host immune responses, such as destructive gene editing [12].

SARS-CoV-2, an envelope, single stranded positive-sense RNA virus, belongs to beta-coronavirus and is related to two highly pathogenic coronaviruses, SARS-CoV and Middle East respiratory syndrome coronavirus. Along with M, the coronavirus E protein is one of the major membrane components in SARS CoV-2. E is a small, 8.5 kDa protein consisting of 75 amino acid residues. In coronaviruses, E is a cationic selective viroporin, forming a channel across the Endoplasmic Reticulum Golgi Intermediate Compartment (ERGIC) membrane. In SARS-CoV, E mediates the budding and release of viruses [13,16].

Replication

SARS-CoV-2 has sufficient affinity to the receptor Angiotensin Converting Enzyme 2 (ACE2) on human cells to use them as a mechanism of cell entry [17]. ACE2 could act as the receptor for SARS-CoV-2 [18]. Studies have shown that SARS-CoV-2 has a higher affinity to human ACE2 than the original SARS virus strain [19]. SARS-CoV-2 may also use basigin to assist in cell entry [20]. The spike protein are key determinants for virus attachment and entry into target cellsand priming by Trans Membrane Protease, Serine 2 (TMPRSS2) is essential for entry of SARS-CoV-2 [21]. After a SARS-CoV-2 virion attaches to a target cell, the cell's protease TMPRSS2 cuts open the spike protein of the virus, exposing a fusion peptide in the S2 subunit, and the host receptor Angiotensin-Converting Enzyme 2 (ACE2). SARS-CoV 2 cell entry is independent of ACE2 catalytic activity. Entry involves 2 spike protein subunits, which mediate distinct functions. The S1 subunit mediates ACE2 attachment through the receptor-binding domain. The S2 subunit, containing the fusion peptide and trans membrane domains, drives fusion of viral and host cell membranes. After fusion, an endosome forms around the virion, separating it from the rest of the host cell. The virion escapes when the pH of the endosome drops or when cathepsin, a host cysteine protease, cleaves it. The virion then releases RNA into the cell and forces the cell to produce and disseminate copies of the virus, which infect more cells [21,22].

Antigenic Variation

There is a significant variation in minimal receptor-binding domain of S-glycoprotein suggests that SARS CoV 2 may have alteration in virus binding capacity and infectivity into the host cell receptor. The novel glycosylation sites in the spike glycoprotein of 2019-nCoV suggesting that virus may utilize different glycosylation to interact with its receptors. The appearance of the virus was accompanied by several distinct antigenic changes, specifically spike protein which is a key element for host cell entry of virus and major target of currently developing vaccines. Some of these mutations enable the virus to attach to receptors. The Omicron SARS-CoV-2 variant of concern (VOC lineage B.1.1.529), which became 8 dominant in many countries during early 2022, includes several sub variants with strikingly 9 different genetic characteristics [22].

Transmission Pattern

SARS-CoV-2 can infect cells of multiple mammalian host species 7–9, primarily due to the conservation of the Angiotensin-Converting Enzyme 2 (ACE2), the primary host cell receptor used for viral entry, across mammals [10–12]. Another essential trait is the ability to transmit efficiently within the populations of the novel host. Infections of host populations that do not efficiently transmit the pathogen further, also known as 'dead-end' hosts, may quickly lead to pathogen extinction within that population. Dogs, which are susceptible to SARS-CoV-2 infection but do not efficiently transmit the virus1 are a possible example of a dead-end host. On the other hand, human-to-human transmission is rapid, with early estimates of the mean number of subsequent infections produced by an infectious person in a totally naïve population [23].

- There are three main transmission routes for the COVID-19:

1) Droplets transmission,

Droplets transmission was reported to occur when respiratory droplets (as produced when an infected person coughs or sneezes) are ingested or inhaled by individuals nearby in close proximity [23];

2) Contact transmission, and

Contact transmission may occur when a subject touches a surface or object contaminated with the virus and subsequently touch their mouth, nose, or eyes [23];

3) Aerosol transmission

Aerosol transmission may occur when respiratory droplets during aerosol generating procedure mix into the air, forming aerosols and may cause infection when inhaled high dose of aerosols into the lungs in a relatively closed environment [23].

Pathogenesis

The pathophysiology of SARS-CoV-2 involves a highly aggressive inflammatory response that primarily damages the respiratory tract. Aside from the viral infection itself, the host response

has also been found to play a key role in determining the severity of coronavirus disease 2019 (COVID-19). The pathophysiology of severe SARS-CoV-2 infection includes virus damage, endotheliopathy, and thrombosis, immune-inflammatory response, and Renin–Angiotensin–Aldosterone (RAAS) system disorder. Direct Viral Damage Through the S protein, SARS-CoV-2 binds to the Angiotensin-Converting Enzyme 2 (ACE2) receptor [23,24]. The cleavage of S protein by Trans Membrane Protease Serine 2 (TMPRSS2) makes coronavirus gain access to host cells. ACE2 and TMPRSS2 are abundantly expressed in pneumocytes, cardiac myocytes, small intestine epithelial cells, and renal proximal tubules, and direct viral injury is a possible mechanism in severe COVID-19. Individuals diagnosed with SARS-CoV-2 show symptoms of different classes from fever or mild cough to pneumonia and severe involvement of various organ dysfunctions with a mortality rate from 2% to 4%. In general, the SARS-CoV-2 causes fever, dry cough, generalized weakness, respiratory distress, muscle ache, rhinorrhoea, pleuritic chest pain and to some extent headache, nausea, vomition, haemoptysis and diarrhoea. A range of sickness in children has been reported from SARS-CoV-2 infection. SARS-CoV-2 has also been implicated in causing damaging effects on the Central Nervous System (CNS) as observed while investigating viral encephalitis cases. Systemic Inflammatory Response Syndrome (SIRS) may well be unusually triggered in severe pneumonia caused by CoV disease, although early anti-inflammatory treatments can effectively prevent immune damage and reduce the impact of damage to the nervous system. Moreover, the rampant inflammation in response to SARS-CoV-2 infection resulted into cytokine storm associated with release of pro-inflammatory cytokines and chemokines like IFN-g, IL-1b, IP-10, MCP-1, TNF-a, G-CSF, MCP-1, IP-10, and MIP-1A, which is reported to severely damage the pulmonary tissues and leads to death in severe COVID-19. The blood profile of the COVID-19 affected individuals exhibits leukopenia, lymphopenia, thrombocytopenia, RNAaemia along with increased hypersensitive troponin I and aspartate aminotransferase levels7 [24].

Epidemiology

In the first pandemic of covid-19 over 30 million cases have been diagnosed globally with more than 946,000 fatalities. The rate of covid-19 has been reported from South Sudan 70 (10.5%) of the cases had travel history. The study verified that 230 (34.5%) of the cases had contact history. Concerning reasons for testing, about 41.1% were suspected whereas 233 (34.9%) and 129 (19.3%) were found confirmed from among the contact and high-risk groups. However, a few numbers of cases were obtained from dead body and discharged from quarantined settings. There have been around half a billion confirmed cases, with approximately 5 million deaths attributed to the ongoing COVID-19 pandemic, while the confirmed cases in Southeast Asia are in the range of 50 million.

Clinical Manifestation

Patients with suspected infection usually go to health centers with symptoms such as fever over 38.5°C, dry cough, shortness of breath and diarrhoea, which should be examined for respiratory symptoms [25]. The complete clinical manifestation is not clear yet, as the reported symptoms range from mild to severe, with some cases even resulting in death. The most commonly reported symptoms are fever, cough, myalgia or fatigue, pneumonia, and complicated dyspnea, whereas less common reported symptoms include headache, diarrhea, hemoptysis, runny nose, and phlegm-producing cough. Patients with mild symptoms were reported to recover after 1 week while severe cases were reported to experience progressive respiratory failure due to alveolar damage from the virus, which may lead to death. Cases resulting in death were primarily middle-aged and elderly patients with pre-existing diseases (tumor surgery, cirrhosis, hypertension, coronary heart disease, diabetes, and Parkinson's disease). Case definition guidelines mention the following symptoms: fever, decrease in lymphocytes and white blood cells, new pulmonary infiltrates on chest radiography, and no improvement in symptoms after 3 days of antibiotics treatment. The associated clinical presentation include fever, cough, malaise and Acute Respiratory Distress Syndrome (ARDS), which is reported to be a major cause of increased mortality among these immune compromised patients [23].

Host Immune Response

Host innate immune response against SARS-CoV-2 Accumulating evidence indicates that SARS-CoV-2 infects the epithelial cells of the respiratory tract and lungs, as well as epithelial and no epithelial cells of other organs that express the ACE2 receptor, which initiates an antiviral immune response upon detection of the virus. As an evolutionarily conserved system of cellular and chemical defenses, the innate immune system is crucial to pathogen detection and restriction as well as the subsequent activation of an adaptive immune response [26].

T Cell Response

Once an individual becomes infected with SARS-CoV-2, the virus will remain in its incubation period for between 4 and 5 days before the patient begins to develop symptoms. Approximately one week after COVID-19 symptoms begin, both B and T cell responses can be detected in the blood. Early studies conducted on some of the first COVID-19 patients found that mononuclear cells, which most likely included monocytes and T cells, accumulated within the lungs, whereas low levels of hyperactive T cells were identified in the peripheral blood. The presence of T cells in such low levels within the blood suggests that rather than remain within the bloodstream, T cells travel from the blood into the infected organs to mitigate the immune response [26].

B Cell Response

Humoral immunity depends on the ability of B cells to successfully transform into plasmocytes and produce antibodies. The production of antibodies to COVID-19 is largely dependent upon the response by T follicular helper cells, which typically occurs one week after COVID-19 symptoms begin. To date, a total of 19 neutralizing antibodies are produced by B cells in COVID-19 patients. Whereas nine of the identified neutralizing antibodies to SARs-CoV-2 bind to the Receptor-Binding Domain (RBD) of the Spike (S) protein of this virus molecule, eight other neutralizing antibodies target the N-terminal region of the upper S protein and the remaining two antibodies bind to other nearby regions.

The COVID-19 antibody response has been identified to begin between 4 and 8 days after symptoms begin; however, the neutralizing activities of these antibodies do not begin until at least 2 weeks after symptom onset. Although the long-term stability of these neutralizing antibodies has not yet been confirmed, they have the potential, with the assistance of memory B cells, to prevent against re-infection if a patient encounters SARS-CoV-2 again in the future. Several monoclonal antibodies have been developed from the memory B cells of former CO-

VID-19 patients as potential therapies. Although the neutralization capabilities of certain antibodies are believed to be effective against SARS-CoV-2 and thereby have protective effects, B cells can also produce non-neutralizing antibodies. The release of non-neutralizing antibodies could potentially initiate a process known as Antibody-Dependent Enhancement (ADE) of the disease. Rather than offer protective effects against SARS-CoV-2, these alternative non-neutralizing antibodies could instead enhance SARS-CoV-2 infection by ADE [26].

Laboratory Diagnosis

Molecular Technique

Detection of SARS-CoV-2 nucleic acid: There are two available categories for RNA amplification tests for SARS-CoV-2; rRT-PCR and Loop-mediated isothermal Amplification (LAMP). For SARS-CoV-2 detection, protocols of rRT-PCR were approved by WHO and the US Food and Drug Administration (FDA), whereas the isothermal amplification assays have not been authorized. For diagnosis of RNA virus infections, RT-PCR is the most common tool due to its accuracy and popularity. Additionally, less common amplification assays other than PCR are also used in the detection of RNA viruses. Nonetheless, the accuracy of nucleic acid amplification tests is ultimately affected by mutations in the sequences targeted by test primers [27].

Different qualitative rRT-PCR protocols for SARS-CoV-2 diagnosis were developed by different countries. Two diagnostic rRT-PCR-based assays are accepted for procurement under Emergency Use Listing procedure; Genesig Real-Time PCR Coronavirus (Primerdesign Ltd, Southampton, UK) and Cobas SARS-CoV-2 6800/8800 system (Roche Molecular Systems). The Cobas 6800 SARS-CoV-2 system from Roche was recently evaluated by Roche-independent researchers and found to be reliable for detecting SARS-CoV-2 RNA in nasopharyngeal specimens [227].

Immunodiagnostic Approaches

Immunodiagnostic point-of-care tests generating rapid results (in less than 1 hour) are less complex than molecular tests. Based on current evidence, seroconversion for SARS-CoV-2 was found to occur between 7 and 11 days after onset of symptoms. Consequently, antibody detection assays might be impractical for diagnosis of acute (current) infection at the early stage. Nonetheless, these tests may be useful in epidemiological surveillance (retrospective evaluation), contact tracing and research studies addressing neutralizing antibodies [27].

Serological Tests

Many commercially available serological kits have been granted Emergency Use Licenses from the FDA to detect antibodies against SARS-CoV-2. Such tests are mostly based on the principles of immunochromatography, chemiluminescence or ELISA to detect IgG or IgG and IgM in serum. Serodiagnosis is believed to be useful in convalescent patients with negative PCR findings, as the accuracy of molecular assays is influenced by viral shedding dynamics. However, cross-reactivity with other antibodies is a major challenge to serological tests. In fact, the profile of the humoral immune response to SARS-CoV-2 is still partially unknown [27].

Rapid Diagnostic Testing for SARS-CoV-2

Rapid Diagnostic Tests (RDTs) that are authorized by the Food and Drug Administration to diagnose Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) infection are either nucleic acid amplification tests to detect genes or antigen-based immunoassays to detect proteins of SARS-CoV-2.RDTs are approved for use in persons with symptoms of coronavirus disease 2019 (Covid-19) and in asymptomatic persons who are close contacts of a person with Covid-19 or who have been in a potential high-risk transmission setting. Symptomatic persons should undergo testing as soon as possible, quarantine while awaiting test results, and consider retesting if they have a negative RDT, particularly if they have a high pretest probability of infection. Asymptomatic persons with a known exposure to SARS-CoV-2 should undergo testing 5 to 7 days after exposure, and if the RDT is negative, they should undergo testing again 2 days later [27].

Treatment

Respiratory support has to be given to patients with hypoxic respiration failure and acute respiratory distress syndrome. At the pandemic time of covid-19 there is no single specific antiviral therapy for COVID-19 and the main treatments are supportive. Recombinant IFN with ribavirin only has limited effects against SARS-CoV-2 infection [28,29]. High-throughput oxygen therapy and Continuous Positive Airway Pressure (CPAP) ventilation are both effective supportive therapies and target blood SpO_2 should be 88-90%. Invasive mechanical ventilation is used as a last resort. The results of a recent study with 100 patients have demonstrated that chloroquine phosphate is more effective than control treatment in inhibiting the exacerbation of pneumonia, improving lung imaging findings, promoting a virus-negative conversion, and shortening the disease course. Previously, tocilizumab, also known as atlizumab, prescribed for two patients with COVID-19 infection in Iran and has shown promising results. This drug is an immunosuppressive humanized monoclonal antibody against the Interleukin-6 receptor (IL-6R) and mainly used for the treatment of Rheumatoid Arthritis (RA) and IL-6 is a cytokine that plays an important role in immune response [29]. Recently, Baricitinib was inhibitor, for the treatment of hospitalized adult patients with COVID-19 requiring supplemental oxygen. Bebtelovimab was for the treatment of symptomatic mild to moderate COVID-19 and it neutralizing human IgG1k monoclonal antibody targeting the spike protein of SARS-CoV2. Dexamethasone was recommended in patients with confirmed COVID-19 who require supplemental oxygen including those who require mechanical ventilation.

Prevention

People with close contacts and suspicious exposure need to be advised to have a 14-day health observation duration, which starts from the last day of contact with the SARS-CoV-2 infected patients or suspicious environmental publicity. When displaying any sign and symptom, mainly fever, respiration signs like coughing, shortness of breath, or diarrhea, they must reach out for medical attention right now. Contact surveillance has to be allotted for people who had been exposed to accidental contact, low-level exposure to suspected or confirmed sufferers, i.e. checking any symptoms whilst concluding everyday activities.

Patients with a suspected infection should be isolated, monitored, and diagnosed in the hospital as quickly as possible. Doctors ought to make suggestions supported the affected person's situation. Patients with mild signs and suspected infection may additionally remember in-home isolation and domestic care. Suspected infected with severe symptoms and those who have to stay in the health facility for remark through physician's judg-

ment have to observe the isolation guidelines for suspected patients. International site visitors should take ordinary precautions while getting into and leaving the affected regions, which includes avoiding near contacts with human beings with acute breathing infection, washing hands frequently, mainly after contacting with the ill or their surrounding environment; following appropriate coughing etiquette [24].

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