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An unusual cluster of severe pneumonia was noted in Wuhan, China, in December 2019. The etiological agent was identified as a precursor virus to humans. The receptor used by SARS-CoV-2 to gain entry to cells is angiotensin-converting enzyme 2 (ACE-2). Rhinolophus genus. What remains unclear is whether there were intermediate hosts that facilitated transfer and adaptation of the virus within the spike protein while RaTG13 does not. The natural reservoir from which SARS-CoV-2 emerged is likely to be bats of the Rhinolophidae family. Closely related viruses have been found in Rhinolophus genus bats, notably RaTG13 and RMYN02, which respectively, share 96.3% and 93% nucleotide identity with SARS-CoV-2. RMYN02 and SARS-CoV-2 both have furin cleavage sites that are characteristic of Coronaviridae Family. A novel coronavirus closely related, but not identical to, the virus that caused SARS in 2003. The virus was named SARS coronavirus 2 (SARS-CoV-2) and the disease coronavirus virus disease 2019 (COVID-19). Within months, the virus spread to cause a pandemic which has had catastrophic impacts on global health, economy and society. SARS-CoV-2, SARS-CoV-1 (causing SARS epidemic in 2003) and related viruses found in Rhinolophid bats are classified within the sub-genus Sarbecovirus, genus Betacoronavirus, Family Coronaviridae. Closely related viruses have been found in Rhinolophus genus bats, notably RaTG13 and RMYN02, which respectively, share 96.3% and 93% nucleotide identity with SARS-CoV-2. RMYN02 and SARS-CoV-2 both have furin cleavage sites within the spike protein while RaTG13 does not. The natural reservoir from which SARS-CoV-2 emerged is likely to be bats of the Rhinolophus genus. What remains unclear is whether there were intermediate hosts that facilitated transfer and adaptation of the precursor virus to humans. The receptor used by SARS-CoV-2 to gain entry to cells is angiotensin-converting enzyme 2 (ACE-2).

The median incubation period of SARS-CoV-2 infection is around 5 days (range 2–14 days) and the reproduction number (Ro) is estimated to be 2.5. Infected persons may transmit infection from 1 to 2 days prior to onset of symptoms to around 7–10 days after symptom onset. However, severely ill patients and immunocompromised individuals may be infectious for longer periods of time. Pre-symptomatic as well as asymptomatic infections may lead to transmission. The virus is transmitted via large respiratory droplets or respiratory aerosols, predominantly over close range (a few meters) although there are occasional instances of transmission over greater distances. Crowded indoor environments are more conducive to transmission and singing or loud speaking by infected individuals increases the risk of transmission. The virus remains viable for many hours on smooth surfaces (stainless steel, glass, plastic) but survival is much shorter on non-porous surfaces such as cloth or paper. Therefore, indirect transmission from contaminated surfaces via hands to eyes, nose or mouth may potentially contribute to transmission. While the virus RNA can be detected in feces for prolonged periods, infectious virus has infrequently been detected and the degree of infectiousness of feces remains unclear. Super-spreading events are prominent drivers of transmission. In the early stages of the pandemic, non-pharmaceutical interventions including case detection, isolation, contact tracing, quarantine, physical distancing, reduction of mobility and travel related measures were successfully used to reduce transmission.

Symptoms include fever or chills, cough, shortness of breath or difficulty in breathing, fatigue, muscle or body aches, headache, sore throat, congestion or runny nose, nausea, vomiting or diarrhea. Loss of smell or taste is frequently reported and is associated with infection and damage of olfactory neurones in the nasopharynx. Progression of clinical disease may lead to hypoxia and acute respiratory distress syndrome (ARDS). Radiological changes include bilateral ground glass opacities and alveolar exudation. Lymphopenia with increased serum transaminase, C-reactive protein and d-dimer levels are commonly seen in severe cases. Progression of disease is associated with difficulty in breathing, leading to ARDS, sometimes leading to a fatal outcome.

The overall infection fatality risk increases progressively with age; those aged 15–44, 65–74 and >75 years having infection fatality risks of 0.03%, 3.1% and 11.6%, respectively; males roughly having twice the risk of females across age spectrums. COVID-19 infection in children and young adults is often mild or asymptomatic. The presence of co-morbidities including heart, respiratory, renal and liver diseases, cancer, diabetes and obesity increase the risk of severe infection and fatal outcome.

Molecular detection of SARS-CoV-2 RNA is the mainstay of diagnosis. Detection of viral protein (usually nucleoprotein) by rapid antigen detection tests give more rapid results and are sensitive in detecting specimens with high viral load who have highest transmissibility of infection. Antibody responses to multiple viral proteins (spike, nucleoprotein, ORF8) and virus neutralizing antibodies are progressively detectable towards the end of the first week after onset of symptoms, and are detectable in most
patients by the end of the third week of infection. Neutralizing antibodies target the spike protein and is protective. CD4 and CD8 T cell responses are also elicited following infection but their role in protection remains to be elucidated.

Direct viral damage as well as immunopathology contribute to pathogenesis, a hyper-inflammatory state being observed in severely ill patients. An intravascular coagulopathy also contributes to pathogenesis, often involving the microvasculature but sometimes leading to thrombosis of large blood vessels with poor prognosis.

Supportive care in the management of patients include provision of supplemental oxygen or mechanical ventilation as and when required. Randomised clinical trials are beginning to identify specific therapies with proven clinical efficacy and this is a fast-moving area of knowledge. There is emerging consensus for the beneficial use of corticosteroids in those patients who require supplemental oxygen or mechanical ventilation. The antiviral drug remdesivir improves time to recovery but does not appear to provide survival benefit when used by itself. However, a combination of remdesivir with immunomodulators (e.g., JAK inhibitors such as baricitinib) may provide improved benefit.

There has been a rapid progress in developing and evaluating COVID-19 vaccines. These have included protein subunit, viral vectored (e.g., adenoviral vectors) and RNA vaccines targeting the viral spike protein; and inactivated whole virus vaccines which elicit immune responses against the structural proteins of the virus. By the end of the year 2020 phase 3 trial data show acceptable levels of efficacy and safety with RNA and adenoviral vectored vaccines targeting the virus spike protein providing evidence that the viral spike is a protective antigen. In December 2020 some countries have started to vaccinate their populations. The duration of vaccine induced protection remains unknown. Most clinical trials evaluate protection from virologically confirmed symptomatic clinical disease and it is unclear whether there will be comparable impact in reducing transmission, a question of key relevance in disease control and population immunity.

**Further Reading**

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