Mini Review

CLINICAL INVESTIGATION

Coronavirus disease (COVID-19) and SARS (CoV-2)

Erica Bronen*

Editorial Office, Journal of Clinical Investigation, London

*Author for correspondence: clinicalinvestigation@escienceopen.com

Abstract

Coronavirus Disease 2019 (COVID-19) is a global health emergency caused by the Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2). Virus transmission, asymptomatic and presymptomatic virus shedding, diagnosis, therapy, vaccine development, virus origin, and viral pathogenesis are among the nine most essential research concerns discussed here. The Chinese authorities initially labelled the 2019-nCoV as causing an ongoing outbreak of lower respiratory tract sickness known as Novel Coronavirus Pneumonia (NCP). The World Health Organization later suggested the term COVID-19 for the condition. Meanwhile, the International Committee on Virus Taxonomy called 2019-nCoV SARS-CoV-2.

Keywords: SARS-CoV-2 • Novel coronavirus pneumonia

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Introduction

A group of physicians and scientists from the University of Hong Kong produced the first tangible proof for human-to-human transmission of SARS-CoV-2 in the city of Shenzhen, near Hong Kong. This is a great illustration of how a high-quality clinical study can have a big impact on policymaking. This study also established a number of critical clinical characteristics of COVID-19. First, an attack rate of 83 percent within the family context is concerning, showing that SARS-CoV-2 is highly transmissible. Second, COVID-19's clinical manifestations in this family range from mild to moderate, with older patients experiencing more systemic symptoms and more severe radiological abnormalities [1]. COVID-19 looks to be less severe than SARS in general. Third, ground-glass opacities were discovered in an asymptomatic child's lung, as well as SARS-CoV-2 RNA in his sputum sample. Finally, the gastrointestinal involvement in SARS-CoV-2 infection and fecal-oral transmission in two young adults from the same family raises the likelihood of gastrointestinal participation in SARS-CoV-2 infection and transmission. The research has paved the way for COVID-19 control and management. The job was completed on schedule, and the investigators displayed remarkable courage and leadership at a challenging period when the Chinese government failed to detect substantial SARS-CoV-2 transmission before January 20, 2020 [2-4].

In the last few weeks, a number of fascinating studies on SARS-CoV-2 and COVID-19 have been published, reporting on the evolutionary reservoir, probable intermediate host, and genomic sequence of SARS-CoV-2, as well as clinical aspects of COVID-19. In light of these discoveries and the critical need for SARS-CoV-2 and COVID-19 prevention and control, we present our personal thoughts on the most pressing research questions in the field in this commentary.

The first question concerns how SARS-CoV-2 is now propagated in Wuhan's epicenter. Since January 23, 2020, China has closed down Wuhan and adjacent cities to prevent the spread of SARS-CoV-2. The exceptional control measures, which included the stoppage of all urban transportation, appear to have prevented SARS-CoV-2 from spreading to neighboring cities.

The number of confirmed cases in Wuhan, on the other hand, has continued to climb. It's critical to figure out if the increase is due to a significant number of sick people before the lockdown and/or a failure to prevent extensive intra-familial, nosocomial, or community transmission [4-6]. On January 25, 2020, it was estimated that there would be more than 70,000 people infected with SARS-CoV-2 in Wuhan based on the number of cases transported from Wuhan to places outside of mainland China.

As stated below, this should be determined experimentally in Wuhan, and it will disclose whether the true numbers of infected patients and asymptomatic carriers are severely underestimated. Measurement of IgM and IgG antibodies,

as well as antigens, would be highly useful in addition to viral RNA detection. To get a big picture, several sample residential regions should be chosen for extensive investigation. The analysis should encompass all healthy and diseased people in the vicinity, with the goal of identifying people who have recovered from or are still infected. It's also important to figure out how many asymptomatic carriers there are. The analysis should be expanded to detect influenza viral RNA and antigen.

It will be interesting to see if the flu season has ended and how many patients with fevers are now infected with the influenza virus. Based on the findings of this study, precision control measures for SARS-CoV-2 should be tailored-made for high-risk groups. In a hospital context, distinguishing persons with the flu and preventing them from infecting with SARS-CoV-2 could be crucial.

The second question concerns SARS-transmissibility CoV-2's and pathogenicity in tertiary and quaternary human transmission. The fact that SARS-CoV-2 is still being transmitted in Wuhan shows that tertiary and quaternary spreading has occurred. Has the transmission rate increased and the pathogenicity decreased compared to primary and secondary spreading when SARS-CoV-2 was transmitted from animal to human and human to human? Is the virus, on the other hand, less transmissible in people after numerous passages? A review of all confirmed instances in Wuhan in the past should be highly instructive [7].

The answers to the aforementioned questions are crucial to the outbreak's outcome. The outbreak may eventually come to an end if the transmission is decreased, and SARS-CoV-2 is eradicated from people. On the contrary, if successful transmission can be maintained, the chances of SARS-CoV-2 becoming another community-acquired human coronavirus, similar to the other four human coronaviruses (229E, OC43, HKU1, and NL63) that cause just the common cold, are raised. SARS-fundamental CoV-2's reproductive number (R0) has been calculated to be 2.68, implying a 6.4-day pandemic doubling time.

Other R0 estimates range from 2 to 4, which is greater than the SARS-CoV estimate of 2. Determining the true R0 will reveal whether or not infection control strategies are effective, and to what extent.

The third question concerns the significance of asymptomatic and presymptomatic viral shedding in the transmission of SARS-CoV-2 [8]. Infection control is complicated by asymptomatic and presymptomatic viral shedding. Furthermore, patients with vague or weak symptoms are difficult to detect and quarantine. Notably, the absence of fever in SARS-CoV-2 infection (12.1%) is more common than in SARS-CoV (1%) and Middle East respiratory syndrome coronavirus (MERS-CoV; 2%).

As a result, the efficacy of employing fever detection as a surveillance approach should be reconsidered. According to earlier investigations of influenza viruses and community-acquired human coronaviruses, asymptomatic carriers' viral levels are generally low. If this holds true for SARS-CoV-2, the risk should be modest. There is a critical need for research into the natural history of SARS-CoV-2 infection in people. Identifying a cohort of asymptomatic carriers in Wuhan and tracking their viral loads, clinical presentations, and antibody titers over time will reveal how many of the subjects develop symptoms later on, whether virus shedding is indeed less robust, and how frequently they might transmit SARS-CoV-2 to others. The fourth question concerns the role of the fecal-oral pathway in the transmission of SARS-CoV-2. Fecal-oral transmission of SARS-CoV has been proven to be relevant in specific circumstances, in addition to transmission via droplets and close contact. The relevance of the fecal–oral pathway in SARS-CoV-2 transmission is supported by gastrointestinal involvement of SARS-CoV-2 infection and isolation of SARS-CoV-2 from faecal samples of patients. Although diarrhoea was rarely seen in big cohort studies. The risk of SARS-CoV-2 transmission via sewage, waste, contaminated water, air conditioning systems, and aerosols should not be underestimated, especially in cases like the Diamond Princess cruise ship, which had 3,700 passengers, of whom at least 742 were confirmed to have been infected with SARS-CoV-2 as a result of a super spreading event.

The fifth question is about how COVID-19 should be identified and what diagnostic tools should be provided. At the start of the outbreak, the sole specific diagnostic technique is RT-PCR-based SARS-CoV-2 RNA detection in respiratory samples. It was significant in the early detection of SARS-CoV-2-infected patients outside of Wuhan, implying that widespread virus infection occurred in Wuhan at least as early as the beginning of 2020 [9].

As a result, the Chinese government has been forced to admit the gravity of the issue. Due to sample concerns and other technical issues with this test, clinically diagnosed patients with typical ground glass lung opacities in chest CT were counted as confirmed cases at one time in early February in order to have the patients identified and quarantined as soon as feasible. More recently, ELISA kits for detecting IgM and IgG antibodies against N and other SARS-CoV-2 proteins have been available. This has allowed for more precise diagnosis of current and previous infections. IgM antibody seroconversion usually happens a few days before IgG antibody seroconversion.

The sixth question revolves with how COVID-19 should be treated and what therapeutic choices should be accessible. In more than 80% of patients, COVID-19 is a self-limiting condition. According to research with large cohorts of patients, severe pneumonia occurred in roughly 15% of cases. As of February 25, 2020, the global gross case fatality rate was 3.4 percent. Patients in Wuhan have a rate of 4.4 percent, Hubei has a rate of 4.0 percent, and patients outside of Hubei have a rate of 0.92 percent. The high fatality rate in Wuhan could be explained by institutional failure, a large number of misdiagnosed patients, poor care, or a combination of these factors. We still don't have any specific anti-SARS-CoV-2 drugs, although remdesivir, an anti-Ebola medicine, may hold promise. Remdesivir, a nucleotide analogue, has been found to suppress MERS-

CoV replication in monkeys. When the medication was given before or after infection with MERS-CoV, the severity of the disease, viral replication, and lung damage were all reduced. These findings lay the groundwork for a quick assessment of remdesivir's positive effects in COVID-19. Ribavirin, protease inhibitors lopinavir and ritonavir, interferon 2b, interferon, chloroquine phosphate, and Arbidol are all antiviral medicines that should be studied further in the clinic. However, we must take into account the antiviral drugs' negative effects. Interferons of type I, such as interferon 2b and interferon, are widely known for their antiviral properties. Their anti-infective actions are predicted to be beneficial in the early stages of infection. However, if administered later, they run the risk of exacerbating the cytokine storm and aggravating inflammation. Steroids have been widely used experimentally in the treatment of SARS, and some Chinese physicians still prefer them in the treatment of COVID-19. It is reported to be capable of preventing lung fibrosis and stopping the cytokine storm. However, the time frame in which steroids may be useful to COVID-19 individuals is relatively limited. To put it another way, steroids can only be administered after SARS-CoV-2 has been destroyed by the human immune system. Otherwise, SARS-CoV-2 replication will be accelerated, resulting in worsening symptoms, significant viral shedding, and a higher risk of nosocomial transmission and secondary infection. In this regard, it will be interesting to see if reports of fungal infection in the lungs of any Wuhan patients can be linked to steroid abuse [10]. Nonetheless, through identifying new medicines, small-molecule compounds, and other agents with significant anti-SARS-CoV-2 properties, new and improved lead compounds and agents that could be effective in the treatment of COVID-19 will be discovered.

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