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COVID-19 and SARS: Differences and similarities

Dear Editor

SARS-CoV (causative pathogen of Severe Acute Respiratory Syndrome or SARS) and SARS-CoV-2 (causative pathogen of Coronavirus Disease 2019 or COVID-19) are positive-sense RNA viruses belonging to the family of Coronaviridae, able to cause severe respiratory diseases.[1-4]

Despite some similarities, they have many differences, especially in terms of epidemiology. The main differences and similarities are summarized in Table 1.

Genomic characterization has shown that SARS-CoV-2 share almost 80% of the genome with SARS-CoV but it contains additional gene regions (10b, 13, 14).[5]

SARS originated in China's Guangdong province on November 27, 2002. It presented as a respiratory disease caused by the SARS coronavirus (SARS-CoV). At the end of the epidemic in June, the infection affected 8422 individuals leading to 916 deaths and a case-fatality ratio of 10.9% across 29 countries.[2]

On the other hand, COVID-19 began in Wuhan (China), the largest city in Hubei province, in central China in the last week of December 2019. To date, a cumulative 512 701 cases with 23 495 deaths (case-fatality ratio of 4,6%) were reported across 202 countries[6] and, based on available data, the transmission rate might be higher for COVID-19 than for SARS.

The incubation period for SARS was from 2 to 10 days (with mean of 4-5 days) while the average incubation period for COVID-19 is 5.1 days, with a range of 1 to14 days. The average latency of COVID is slightly longer than SARS.[4]

The World Health Organization (WHO) estimates an average basic reproduction number (R0) of COVID-19 of 1.4 to 2.5, with a median of 1.95. In other words, each patient transmits the infection to an additional 1.95 people. The R0 of the SARS epidemic were approximately 3.[3] In contrast with these values, a study shows that the COVID-19 is already more widespread than SARS because its real average R0 is 3.28.[7] This data indicates that COVID-19 may be more transmissible than SARS.

There was a predominance of female patients affected by SARS, with a male to female ratio of 1:1.25. Instead COVID-19 is much more prevalent among males, with a male to female ratio of 2.7:1.[8] Data show that the COVID-19 patients' median age is 59 years, with a range of 15 to 89 years while the median age of patients with SARS was 35 years, with a range of 0 to 92 years and the highest age-specific incidence was in patients with 65-69 years.[2]

The early symptoms of SARS and COVID-19 are very similar, including fever, cough, headache, shortness breath and breathing difficulties. Diarrhea was reported in about 20-25% of patients with SARS, while intestinal symptoms were rarely described in patients with COVID-19. In addition, most patients with SARS and COVID-19 developed lymphopenia with high-levels of proinflammatory cytokines including interleukin (IL)-1b and IL-6.[2]

TABLE 1 Main aspects of coronavirus disease 2019 (COVID-19) and severe acute respiratory syndrome (SARS)

	COVID-19	SARS
Location of first detection	Wuhan, China	Guangdong, China
Start date	December 2019	November 2002
Incubation period	2-10 years (mean of 4-5 days)	1–14 years (mean of 5.1 days)
Global cumulative incidence	512 701 cases (to date)	8422 cases
Deaths	23 595 (to date)	916
Mortality	4.6%	10.8%
Median age	59 years (range of 15 to 89 years)	35 years (range of 0 to 92 years)
Male to female ratio	2.7:1	1:1.25
Possible natural reservoir	Bat	Bat
Possible intermediate host	Pangolins	Civet cats
R0	1.4 to 2.5 (median of 1.95)	3
Intestinal Symptoms	rare	20%-25% of cases
Predominant cellular receptor	ACE2	ACE2

Abbreviation: ACE = Angiotensin-converting enzyme.

The possible pathogens are both derived from wild animals: SARS-CoV was transmitted from civet cats to humans. Previous studies showed that bats were the most likely reservoir for SARS-CoV-2 as it is very similar to a bat coronavirus.[8] However, there are no evidences of direct bat-human transmission; instead pangolins are the possible intermediate host for COVID-19. The common aspect is that SARS and COVID-19 infect lung alveolar epithelial cells using receptor-mediated endocytosis via the angiotensin-converting enzyme II (ACE2) as an entry receptor.[9]

The rapid development of this pandemic requires comparisons with previous epidemic, to analyze infection trends and to find the right prevention and treatment measures, as was done in the past for similar cases.

CONFLICT OF INTEREST

The authors declare no potential conflict of interest.

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