Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Review

COVID-19 versus SARS: A comparative review

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ABSTRACT

The two genetically similar severe acute respiratory syndrome coronaviruses, SARS-CoV-1 and SARS-CoV-2, have each been responsible for global epidemics of vastly different scales. Although both viruses arose from similar origins, they quickly diverged due to differences in their transmission dynamics and spectrum of clinical presentations. The potential involvement of multiple organs systems, including the respiratory, cardiac, gastrointestinal and neurological, during infection necessitates a comprehensive understanding of the clinical pathogenesis of each virus. The management of COVID-19, initially modelled after SARS and other respiratory illnesses, has continued to evolve as we accumulate more knowledge and experience during the pandemic, as well as develop new therapeutics and vaccines. The impact of these two coronaviruses has been profound for our health care and public health systems, and we hope that the lessons learned will not only bring the current pandemic under control, but also prevent and reduce the impact of future pandemics.

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Introduction

The coronavirus is no new guest to humans, and neither are humans a new host. The first strain of coronavirus was identified in 1965 as a significant cause of the common cold [1]. Since then, a total of seven human-infecting strains have been discovered of which four generally cause mild symptoms, and three cause potentially severe disease. The latter includes the 2002 Severe Acute Respiratory Syndrome (SARS), 2012 Middle East Respiratory Syndrome (MERS), and the 2019 Coronavirus Disease (COVID-19) [2]. The global SARS epidemic, caused by SARS-CoV-1, redirected significant attention to the Coronaviridae family [3]. Years after the disruptive SARS epidemic, there were many warnings about the possibility of the re-emergence of SARS and other novel viruses. One report in 2007 described the large reservoir of SARS-CoV-like viruses in bats, coupled with the trade of wild animals in China, to be a "time bomb" [4]. Likewise, after the MERS outbreak, a multidisciplinary group of scientists re-iterated the potential risk of SARS-CoV's from bat populations [5]. Years later, SARS-CoV-2, responsible for the current COVID-19 pandemic, emerged to affirm the prior warnings. The disease followed a similar pattern to SARS, starting in China around a wet market selling wild animals and spreading to over 200 countries [6,7]. Both diseases have similar clinical manifestations, predominantly causing a respiratory illness [8]. Ongoing research on SARS-CoV-2 has revealed unique features about its genome, pathogenesis, clinical course, transmissibility, and control measures. By comparing and contrasting SARS-CoV-2 to SARS-CoV-1, we aim to understand the current pandemic and its future trajectory. Herein, we present a comprehensive comparative review of both viruses to provide a resource for clinicians, scientists, and public health experts managing the COVID-19 pandemic and guide future efforts to deal with subsequent coronavirus outbreaks. We used multiple search terms including "SARS", "SARS-CoV", 2019 novel coronavirus" and "COVID-19" to identify published articles and preprints on PubMed and Google Scholar, as well as additional search terms specific to each subtopic, and information from public health agency websites including the World Health Organization and the Centers for Disease Control and Prevention as of March 24, 2021.

Background and epidemiology

The two viruses responsible for SARS and COVID-19 outbreaks mirror one another in many different ways; not only are they genetically related, but the stories behind their emergence are strikingly similar. Both SARS and COVID-19 began as outbreaks of atypical pneumonia located around wet markets [9]. Both outbreaks were reported to the World Health Organization (WHO) when excess cases of pneumonia were detected. Although SARS was reported almost four months after the outbreak had begun, COVID-19 was reported after only one month [10,11]. The etiological agents of SARS and COVID-19 are believed to have originated from bats and transmitted to humans via intermediate hosts [12]. The intermediate host for SARS-CoV-1 is thought to be the Asian palm civet, but the identity of the COVID-19 intermediary is still not confirmed. Some studies suggest pangolins may have played a role in the evolution of SARS-CoV-2 but this remains an active area of investigation [12–14].

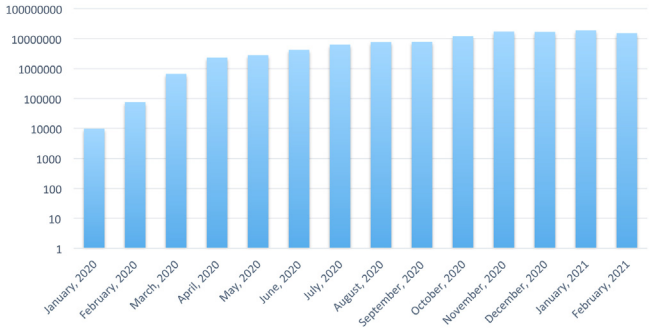

Fig. 1. Global incidence of new COVID-19 cases [22].

The SARS outbreak started as atypical pneumonia associated with wet markets in Guangdong, China, in mid-November 2002 [10]. By February of the following year, China reported to the WHO that the disease had affected 305 individuals, 5 of whom had died [15]. China became ground zero for the international outbreak after a 65-year-old physician from Guangzhou, who had been treating patients with what was thought to be atypical pneumonia in China, arrived at a hotel in Hong Kong on 21 February 2003 [16]. Similarly, the outbreak of COVID-19 was believed to have originated around a wet market in Wuhan, China, in November 2019 [17]. Consequently, the market was closed on 1st of January 2020. On 7th of January, the etiological agent of the outbreak was identified as a novel betacoronavirus, and by 21 January, a total of 270 cases were reported in Wuhan [18].

The global spread of both diseases was primarily mediated by commercial travel. Much of the worldwide spread of SARS could be traced back to the pivotal transmission event in the Hong Kong hotel where many international visitors were also staying, bringing the infection back to their respective countries, including Vietnam, Canada, Singapore, USA, Philippines, and Australia [16]. The SARS epidemic lasted approximately nine months, spreading to 29 countries, infecting 8096 people and resulting in 774 deaths [19]. In contrast to 2003, China's connectivity to the rest of the world in 2020 was far more significant, and thus COVID-19 was able to spread much faster. This was compounded by the Chinese New Year celebrations' coincidental timing that involved millions of people traveling within and outside China [7,20]. In a period of 1 year, the world witnessed an exponential rise in cases, with approximately 120 million people infected and over 2 million fatalities [21]. The initial increase in the period from December 2019 to mid-March 2020 reflected the rise and wane of the outbreak within China, followed by outbreaks in the rest of the world, most notably in the Americas, Europe, and Asia. (Fig. 1). More recently, the emergence of new genetic variants of SARS-CoV-2 have contributed to further spread of the virus [22].

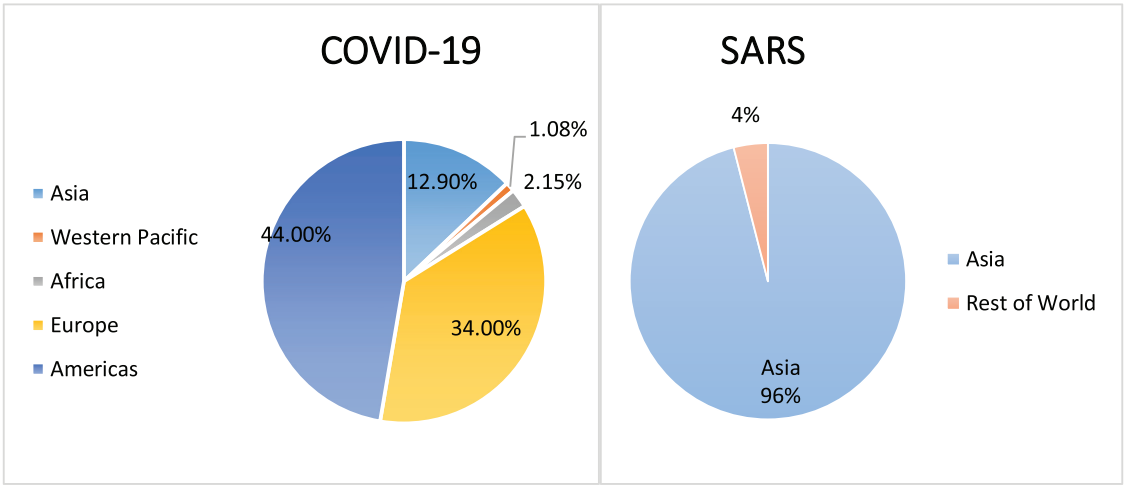

Fig. 2. Geographic distribution of COVID-19 and SARS cases (March 14, 2021).

From a geographical perspective, the SARS outbreak was concentrated mainly in Asia, accounting for 96% of all cases globally (Fig. 2), with 7775 cases and 729 deaths in Asia compared to 321 cases and 45 deaths in the rest of the world [19]. In contrast, the widespread and sustained transmission of COVID-19 is occurring on all continents. After the initial outbreak in China, the disease's incidence was concentrated in Europe and the Americas. The Americas currently account for almost half of the total COVID-19 cases worldwide (44%) (Fig. 2) [21].

Virology

Structure and genome

Coronaviruses are taxonomically classified under the subfamily Orthocoronavirinae, in the family Coronaviridae, and the order Nidovirales [23]. They are enveloped viruses, composed of a single-stranded, positive-sense RNA genome [3,24]. In contrast to other RNA viruses, coronaviruses are uniquely characterized by their long, non-segmented genome [25]. The large genome, varying from 26 to 32 kb, allows coronaviruses to acquire more genetic diversity and thus greater capacity for cross-species transmission [26]. Currently, there are four known genera (alpha, beta, gamma, and delta) with all human coronaviruses falling under the genus betacoronavirus [23]. The only coronaviruses documented to have caused human epidemics have been SARS-CoV-1, MERS-CoV, and SARS-CoV-2, but there is evidence suggesting that HCoV-OC43 may have caused a pandemic at the end of the 19th century [27,28]. All human coronaviruses are speculated to have arisen through recombination and other mutational events in their original hosts, bats and rodents, as well as through intermediary animals [29].

SARS-CoV-2 was given its name because its genetic sequence was more than 80% identical to SARS-CoV-1 [30]. The RNA genome has several open reading frames that encode for several structural and non-structural proteins [26]. The viral transmission capacity and cellular tropism is mainly determined by the spike protein which also gives these viruses their distinctive crown-like appearance under electron microscopy [31]. Both viruses use the spike protein to gain entry to cells via angiotensin-converting enzyme 2 (ACE2) [32]. The sequence of the receptor binding domain (RBD) of the SARS-CoV-2 spike protein is similar to that of SARS-CoV-1 [33]. However, the RBD of the spike protein of SARS-CoV-2 exhibits greater affinity for binding to ACE2 than the SARS-CoV-1 RBD, which may explain the higher intra-host replication and inter-host transmission efficacy of SARS-CoV-2 [32].

Transmission

The primary mode of transmission for both SARS-CoV-1 and SARS-CoV-2 is via direct or indirect contact with infectious droplets, released when coughing, sneezing, or even speaking [34,35]. Both viruses can also remain viable in aerosols and on inanimate surfaces, which allows for indirect transmission [36]. The stability of each virus is similar to one another in various environmental conditions, so differences in transmissibility between SARS-CoV-1 and SARS-CoV-2 are mostly due to differences in receptor affinity and replication efficiency. Additionally, the superspreading effect, when an individual transmits the virus to a disproportionate number of contacts, has been an important driver in both outbreaks [37,38]. WHO estimates of the basic reproductive number (R_0) for SARS-CoV-2 range from 1.4 to 6.9 with a mean of 3.28, while the R_0 estimates for SARS-CoV-1 range from 2 to 4 [34,39]. However, the R_0 estimates of both viruses are not reflective of the difficulty in controlling the spread of each virus. Patients with SARS were maximally infectious during the second week of illness, whereas COVID-19 patients are most infectious in the pre-symptomatic and early symptomatic phase of illness. The control of SARS-CoV-2 is further complicated by a population of infectious individuals who are asymptomatic at the time of transmission, both from pre-symptomatic individuals and individuals who remain asymptomatic throughout the course of infection. As a result, significantly greater effort has been required to reduce the effective reproductive number (R_e) of SARS-CoV-2 compared to SARS-CoV-1 [34,40,41]. Other routes of transmission, including fecal, transplacental, and sexual have also been documented but their overall importance has been limited to specific scenarios for both COVID-19 and SARS [42–46]. Additionally, nosocomial transmission of both viruses played an important role in amplifying each outbreak, especially in the early stages, when insufficient protection was used by health care workers (HCWs) attending to infected patients, compounded by transmission amongst HCWs themselves [47,48].

Clinical course

Clinical manifestation

The incubation period of SARS-CoV-2 is between 2 and 14 days, with anecdotal reports of periods up to 24 days. The virus produces a spectrum of disease severities, ranging from asymptomatic, to mild, moderate, and critical, life-threatening infection [49]. In comparison, the incubation period of SARS-CoV-1 is slightly shorter, at

Table 1
Comparative summary between SARS and COVID-19.

		SARS (SARS-CoV-1)	COVID-19 (SARS-CoV-2)
Epidemiology	Source of infection	Zoonotic	
	WHO classification	Epidemic	Pandemic
	Duration	November 1, 2002–July 31, 2003	Decemeber 2019 - ongoing
	Number of cases	8096 [19]	119,512,530[22] (March 14, 2021)
	Number of deaths	774 [19]	2,642,612 [22] (March 14, 2021)
	Total countries affected	29 [19]	200+ [21]
Transmission	Primary mode of transmission	Infectious droplets, aerosols	
	Viability on various surfaces	Similar viability	
	Basic reproductive number (R_0)	2–4 [34]	1.4–6.9 [39]
	Additional modes of transmission	Faecal	Faecal, and potentially vertical, and sexual [42–46]
	Nosocomial transmission	Yes	
	Peak of contagiousness	Symptomatic phase [34]	Asymptomatic, presymptomatic, and early symptomatic phases [40]
Clinical Course & Complications	Incubation period	2–10 days [50]	2–14 days [49]
	Case Fatality Rate (As per confirmed cases and deaths)	14–15% [58]	2% [21]
	Most common cause of death	Acute respiratory distress syndrome (ARDS)	
	Extra-pulmonary disease	Cardiovascular diseases, renal impairment, coagulopathy	Cardiovascular diseases, renal impairment, coagulopathy
	Cytokine storm	Associated with ARDS	Associated with ARDS and other complications, more so than in SARS
Therapeutics and Vaccines	Antiviral drugs	No approved drugs	Remdesivir (emergency use authorisation) [136]
	Other treatments	No evidence of benefit from corticosteroid therapy No anticoagulant protocols needed Convalescent plasma therapy may have benefit	Corticosteroid therapy e.g., dexamethasone [140] Anticoagulant protocols crucial [126]
	Vaccines	Approximately 30 vaccines initially developed with no clinical trials conducted [154]	150+ vaccines developed; more than 30 candidates have reported results from clinical trials [153] As of March 14, 2021, 6 vaccines have been deployed globally
Prevention	Public health measures	Isolation of cases Quarantine of close contacts Closure of businesses and schools Restrictions on mass gatherings Cordon sanitaire of high-risk areas	
	Infection control precautions	Standard Contact Droplet Airborne	

2–10 days [50]. Patients over the age of 60 and those with comorbidities, such as diabetes, cardiovascular disease, and hypertension, are at higher risk of developing severe disease with both viruses [51,52]. For COVID-19, symptomatic individuals can be classified into three major groups based on severity. In a Chinese study of 55,924 confirmed cases, approximately 80% of patients had mild to moderate illness, 13.8% had severe disease, and 6.1% were critically ill [53]. Symptoms of mild to moderate disease include fever, cough, sore throat, and mild pneumonia. Symptoms of severe disease include severe pneumonia (SpO_2 < 93%), shortness of breath, and respiratory distress [24,54]. Critical cases are defined as patients with respiratory failure, multi-organ dysfunction, sepsis, or shock [24]. The case severity of SARS was much reportedly higher, with 50–85% of SARS patients needing oxygen supplementation, while intensive care unit (ICU) admission rates ranged from 19% to 32% [55–57]. The overall case fatality rate (CFR) for SARS is 14–15%, which is significantly higher than the CFR estimate of approximately 2% for COVID-19 (Table 1) [21,58].

A significant proportion of COVID-19 patients are asymptomatic, with estimates ranging from 4% to 41% [59]. In contrast, asymptomatic patients were not as evident during the SARS outbreak, but subsequently, asymptomatic seropositive HCWs have been identified [60,61]. Symptoms that overlap between the two infections include fever, cough, headache, and shortness of breath, with fever being the most common symptom for both infections [62,63]. According to two meta-analysis studies, the incidence of respiratory failure or acute respiratory distress syndrome (ARDS) in hospitalized COVID-19 patients ranged from 14.8% to 19.5%, with two studies reporting a prevalence of around 32% to 42%. As for SARS, there is a paucity of data, but studies reported the prevalence of ARDS to range from 20% to 49% [64–69]. Gastrointestinal (GI) symptoms have been reported in COVID-19 patients, with diarrhea being the most common manifestation, followed by vomiting and GI bleeding. Some patients reported GI symptoms without any respiratory symptoms. COVID-19 patients with GI symptoms had a longer incubation period, higher liver enzymes, and prolonged coagulation times [70,71]. Neurological manifestations have also been reported in COVID-19. The most common neurological symptoms were asthenia, headache, anosmia followed by encephalopathy, seizures, and stroke. In severe cases, encephalitis and coma have been reported [72–74]. In a systematic review, headaches were reported in 7.5% of COVID-19 patients, while 6.1% reported dizziness [75]. Other less common neurologic manifestations include cerebral haemorrhage and cerebral venous thrombosis (0.5%) [76]. In comparison, the most common neurological manifestation in SARS were neuromuscular. Other reported

complications included axonopathic polyneuropathy, myopathy, and large artery ischemic stroke [77,78]. Skin lesions have been widely reported as a potential manifestation of COVID-19, but not SARS [79]. The finding of orchitis remains fairly unique to SARS patients, but its prevalence is still being studied in COVID-19 patients [80].

Complications

ARDS is a common sequela in COVID-19 and SARS patients, and is the commonest cause of death in COVID-19 patients [81–84]. Pulmonary inflammation and extensive lung damage in SARS patients were associated with high levels of pro-inflammatory cytokines. Recent studies of COVID-19 showed similar findings (e.g., elevated levels of IL1B, IL6, IL12, IFNγ, IP10, and MCP 1) [84]. Multiple studies show a correlation between higher concentrations of inflammatory mediators and disease severity [84,85]. The same immune reaction brought about by the cytokine storm can also rapidly progress to multiple organ failure [55]. In addition, COVID-19 patients can also die of cardiovascular and renal complications. This has been linked to either direct viral injury due to the expression of the ACE2 receptor in these organs, or indirectly via the cytokine storm [86].

Serious respiratory infections are well-recognized triggers for cardiovascular complications, especially in patients with underlying cardiovascular disease [87,88]. Cardiovascular involvement is highly associated with mortality in COVID-19 patients. Mortality rates were reported to be significantly higher in those with elevated plasma troponin T (TnT) and high-sensitivity cardiac troponin I (hs-cTnI) levels compared to patients with normal plasma levels [89]. While cardiovascular events were documented to occur post SARS infection, cardiovascular complications such as heart failure (23%), acute cardiac injury (17%), and arrhythmia (16.7%) have been widely reported in COVID-19 patients, with a higher prevalence among non-survivors [90,91]. A study of cardiovascular complications of SARS showed that the majority were either self-limiting or reversible, with the most common being tachycardia [92].

Kidney damage has been reported with both coronavirus infections but less commonly in SARS. The pathogenesis of kidney damage includes direct viral injury, indirect damage through deposition of immune complexes or via the cytokine storm [93,94]. Additionally, thrombotic complications, such as pulmonary embolism and deep vein thrombosis, have been documented in SARS and COVID-19 patients, but are significantly more common in COVID-19. These complications are thought to be associated with the exaggerated inflammatory immune response, hypoxia, endothelial injury, or diffuse intravascular coagulation similar to that seen in sepsis [95,96]. An inflammatory shock similar to atypical Kawasaki disease and Kawasaki disease shock syndrome, termed multisystem inflammatory syndrome in children (MIS-C), has also been reported in children with COVID-19, but was not observed in SARS [97].

Investigation and diagnosis

The diagnostic techniques used to identify the presence of SARS-CoV-1 and SARS-CoV-2 are similar. For the diagnosis of infection with SARS-CoV-1, the patient must present with indicative signs and symptoms combined with a positive laboratory test [98]. As for SARS-CoV-2, the diagnostic criteria include epidemiological evidence with indicative clinical signs and symptoms, and a positive laboratory test [99]. In both diseases, reverse transcription-polymerase chain reaction (RT-PCR) is performed primarily on upper respiratory tract samples, such as nasopharyngeal swabs or saliva, to detect the virus. Other sample types include lower respiratory tract samples, such as bronchoalveolar lavages, as well as stool, which may have greater sensitivity than upper respiratory tract samples depending on the clinical presentation and stage of infection. RT-PCR requires the extraction of viral RNA from a sample and uses reverse transcriptase to convert the RNA to cDNA, which is then amplified by PCR targeting genetic targets specific to each virus [98,100]. One study of 213 confirmed COVID-19 patients showed that sputum samples had the highest positivity rate in both mild and severe cases (82.2% and 88.9%), followed by nasal swabs (73.3% and 72.1%) and finally throat swabs (60.0% and 61.3%) [101]. Another study similarly found higher SARS-CoV-2 viral loads in nasopharyngeal swabs compared to oral throat swabs [102]. As for SARS-CoV-1, tracheal aspirates were reported to yield higher positivity rates (66.7%) compared to nasopharyngeal swabs (29.7–40.0%) as most SARS patients had lower respiratory tract infections at the time of diagnosis [103]. It is important to note that the diagnostic yield of the test is affected by the quality of the sample, as well as the timing of the sample collection in relation to the stage of illness. The addition of a lower respiratory tract sample can be useful when an upper respiratory sample is negative for SARS-CoV-2 in cases that are highly suspicious for COVID-19 [104].

Serological testing is an essential tool for both epidemiological studies and vaccine evaluation, as well as to identify patients with higher antibody titers for hyperimmune plasma donation [105]. Furthermore, serological tests are typically less expensive and easier to perform than molecular tests, making them a better option for population level surveillance, especially for the identification of individuals who may have had asymptomatic or subclinical infection [106]. The use of serological testing for SARS-CoV-2 is less clear for the diagnosis of acute infection, as reliable detection of antibodies does not occur until at least 2 weeks post onset. For the detection of recent infection, serology would need to be complemented with viral testing (such as RT-PCR), which may be useful to enhance contact tracing efforts [107]. Serological testing should not be used as definitive evidence of immunity until further studies identify which antibodies and epitopes are most predictive of immunity and how long that immunity lasts [108]. Both the IgM and IgG responses in COVID-19 are detectable earlier than in SARS. For SARS-CoV-1, enzyme-linked immunosorbent assays (ELISAs) can detect serum antibodies (IgM and IgG) with 80–85% sensitivity by the end of the second week, and 100% by the third week [98]. Various commercial tests for SARS-CoV-1 serology have sensitivities ranging from 72% to 100%, and specificities ranging from 98% to 100% [106].

SARS-CoV-1 and SARS-CoV-2 have similar radiological findings in those who have lower respiratory tract infection [98,109]. The most common chest x-ray findings are consolidation and ground glass opacification [110]. In cases where chest x-ray findings are not diagnostic, a CT scan may be indicated. The use of CT to diagnose both infections is crucial in cases that present early in the course of the disease when lung abnormalities are not yet clear on chest X-ray [111]. This will allow for earlier isolation and treatment of patients, especially when viral testing is not available or may be falsely negative. CT scan can also be used to assess the changes during clinical follow-up [98]. The CT findings most commonly associated with both diseases are bilateral pulmonary ground-glass opacities and consolidation. These findings are most prominent 10 days after the onset of symptoms in patients with SARS-CoV-1 and SARS-CoV-2 [109,112]. These CT scan findings are also seen in some asymptomatic SARS-CoV-2 patients, in which unilateral opacifications progress to bilateral consolidations in 7–21 days [109]. At initial presentation in SARS patients, 86% of the cases in one case series presented with a unilateral focal opacity. In severe cases, the virus led to diffuse consolidation, a sign of the development of ARDS. The lesions were mostly located in the lower lung lobes [98].

COVID-19 can affect multiple organs and tissues at the histopathological level. In the lungs, findings include interstitial pneumonia, alveolar hyperplasia, and pulmonary fibrosis [113]. As for the kidneys, one post-mortem study reported that acute kidney injury developed in 94% of COVID-19 deaths, with the most com-

mon pathological finding being acute tubular injury. Other findings related to the kidneys included hypertensive arterionephrosclerosis and diabetic glomerulosclerosis. Focal thrombi were present in 14% of autopsies [114]. In the brain, spongiosis and edema were the most common histopathological findings in COVID-19 [115]. Similar to COVID-19, the lungs of SARS patients also showed alveolar damage, edema, and pulmonary fibrosis. The kidneys of SARS patients also showed acute tubular injury [116]. In SARS, neuronal edema and degeneration were the two most common findings in the brain. Damage to the heart was also reported in SARS with evidence of myocardial fiber edema and atrophy [116]. Likewise, in COVID-19, acute myocyte necrosis along with the presence of inflammatory infiltrates and apoptotic bodies have been reported in autopsies [117]. As for dermatoses related to COVID-19, several features have been identified, including necrotic keratinocytes and acantholytic clefts [117]. This has not been commonly reported in SARS [116].

Management

Patients with SARS and COVID-19 infection with mild symptoms and no risk factors for severe disease development can be managed at home while maintaining infection control precautions [11,118,119]. Patients who are managed at home are advised to continue infection control precautions for 10 days following resolution of fever in the case of SARS and for variable durations in the case of COVID-19 depending on the guidelines of different countries, which may also include clinical or laboratory criteria [11,118,120].

A limited set of therapeutics, including remdesivir, corticosteroids, and monoclonal antibodies, have been indicated for use in critical cases of COVID-19 or cases where there is a high risk of disease progression [121]. Similar therapeutic guidelines have not been developed for SARS due to more limited extent of the outbreak and challenges with demonstrating clinical efficacy; however, similar approaches were used and investigated [122].

In-patient care for SARS and COVID-19 is mainly supportive, focusing on managing the complications of infection such as pneumonia, respiratory failure, and sepsis, as well as complications from prolonged hospital stay, including secondary bacterial infections and thromboembolism [123–125]. Because of the importance of endothelial dysfunction in COVID-19, many guidelines recommend prophylactic doses of anticoagulant or antiplatelet therapies for all in-patient cases, including full therapeutic doses for those at higher risk of developing thromboembolic disease. Low-molecular weight heparins have been specifically recommended for their down-regulation of IL-6 and blocking of the SARS-CoV-2 spike protein [126–128]. Recent studies suggest that therapeutic anticoagulation doses in COVID-19 patients with critical illness may worsen mortality outcomes due to the increased risk of bleeding, but rather, prophylactic dosages may be more suitable, unless otherwise indicated by a hypercoagulable state [129,130]. The need for long-term antithrombotic therapy remains unclear. Similar antithrombotic protocols were not recommended for the treatment of SARS.

Treatment and vaccination

Treatment

Other than supportive care and infection control measures, there are no specific or highly effective treatments for SARS-CoV-1 and SARS-CoV-2 [3,11]. Empirical therapy for community acquired pneumonia should be administered in patients suspected to have SARS [3,131]. However, bacterial superinfection has not been a prominent feature of COVID-19, so empiric antibiotic therapy has not been broadly recommended [132].

As many therapeutic and vaccine development projects were not successfully carried to completion during the SARS outbreak, there exist gaps in knowledge to guide the development of vaccines and treatments against COVID-19 [133]. Several antiviral agents have nonetheless been used experimentally for the treatment of both diseases. Remdesivir is a nucleoside analogue that was originally developed against Ebola, but later shown to inhibit SARS-CoV-1 in animal models [134,135]. Remdesivir gained an emergency use authorization (EUA) for the treatment of COVID-19 by the FDA, as clinical trials showed modest benefits in critically ill patients [136]. Preliminary evidence suggested that hydroxychloroquine and chloroquine may have antiviral effects against both viruses, but EUA for the use of both drugs against COVID-19 was revoked by the FDA following findings showing a lack of benefit and concerns of serious side effects [137,138].

Furthermore, various immunomodulators have been used to improve the prognostic outcome of SARS and COVID-19 patients. Corticosteroids were used during the SARS outbreak, but did not seem to be effective [139]. However, the use of dexamethasone in a clinical trial has shown a significant decrease in the mortality of COVID-19 patients requiring respiratory support [140]. In terms of monoclonal antibodies, tocilizumab (IL-6 inhibitor) has been recommended along with dexamethasone in hospitalised COVID-19 cases presenting with rapidly deteriorating respiratory function [141]. Other neutralizing antibodies, such as bamlanivimab and etesevimab, have been considered for outpatients with mild to moderate COVID-19 disease who are at increased risk of disease progression [142]. In the case of SARS, although some SARS-CoV-1 antibodies have been developed, they have not been sufficiently studied [122,143]. Finally, the use of convalescent plasma has shown promise for seriously ill patients for both viruses. However, the FDA has recommended further evidence from controlled clinical studies to determine the efficacy and safety of this approach [144,145].

Vaccination

There have been many efforts to develop effective vaccines against SARS-CoV-1 and SARS-CoV-2 [146–153]. The fact that affected individuals produce high titers of neutralizing antibodies against SARS-CoV-1 suggests that the induction of neutralizing antibodies could be an effective strategy to prevent infection [146]. Studies have explored the use of different classes of vaccines, such as nucleic-acid based, protein-based, and inactivated whole virus vaccines, many of which have successfully demonstrated the induction of neutralizing antibodies [147–151,154]. Although the use of SARS-CoV-1 whole virus vaccine was effective in animals, its use in humans raised serious safety concerns due to the risk of inflammatory responses and potential for SARS-like disease. Therefore, none of the vaccines developed for SARS have been evaluated in clinical trials [152,154]. Conversely, there are currently over 100 vaccine candidates from 8 classes of vaccines being developed and tested in clinical trials against SARS-CoV-2, including whole virus vaccines, viral vectors, and nucleic acid and protein-based vaccines. This accelerated effort has produced several efficacious vaccines, which are now being deployed globally to mitigate the COVID-19 pandemic. Additionally, other efforts are ongoing to study the immunomodulatory effect of existing vaccines, such as the Bacillus Calmette-Guerin (BCG) vaccine for tuberculosis, which may have a protective effect against severe illness in COVID-19 [153,155,156].

Prevention

Prevention in the community

In addition to the current emergency approval and distribution of COVID-19 vaccines, public health containment measures are the only effective strategies to prevent or slow down human transmission in the community [20]. Hand hygiene is strongly advised in

the setting of coronaviruses, as these viruses are efficiently inactivated via handwashing with soap and water or alcohol-based sanitizers [157]. Good respiratory hygiene, social distancing, and the universal use of facemasks are also fundamental elements of controlling these viral outbreaks [158,159]. The detection and isolation of confirmed cases, along with the tracking and quarantine of close contacts, is essential to prevent community transmission. These methods resulted in the elimination of SARS from the human population and remain important tools for controlling the COVID-19 pandemic [20,160,161]. Restrictions on movement have also been imposed worldwide, both within countries as well as between countries, with individuals from high-incidence regions prevented from traveling or having to go through additional screening or quarantine on arrival [162–164].

Prevention in hospitals

SARS and COVID-19 have been shown to spread in the hospital setting [165,166]. Appropriate infection prevention methods such as standard, contact, and droplet precautions must be practiced by HCWs (Table 1). Additionally, the use of N95 respirators in the setting of aerosol-generating procedures has been greatly emphasized [167,168]. Disinfection of hospital areas and patients' items has been shown to be useful in preventing hospital-acquired infection [169]. Due to the magnitude of the COVID-19 outbreak, some countries resorted to building new temporary hospitals, and converting public venues such as stadiums and parking lots into shelter hospitals for treatment and isolation. These methods have been shown to be effective in preventing the nosocomial spread of disease while maintaining health care services for non-COVID-19 patients [170].

Preventing future zoonotic outbreaks

The SARS and COVID-19 outbreaks have emphasized the need to be able to predict and prevent future outbreaks. Regulatory oversight of wet markets and the wild animal trade must be one key component of strategies to prevent future zoonotic outbreaks [171]. Although these markets are an essential source of food and income for many parts of the world, additional safety measures need to be implemented along with enforcement of these rules. Surveillance efforts to monitor and characterize animal viruses in the environment must continue to provide early warnings for potential zoonotic pathogens. The capacity of the health care system, including clinical laboratory testing and stockpiles of resources such as masks, to deal with emergencies such as a pandemic must be maintained [172]. In addition, it is essential that communicable disease prevention and control programs and agencies continue to be supported even when global outbreaks are not in the headlines [173]. The current pandemic has also demonstrated the successful incorporation of new technology for public health, with the use of mobile applications to assist with contact tracing. One study in Spain has shown that the use of digital contact tracing was almost twice as effective as manual contact tracing techniques [174].

Limitations of comparative reviews

It is worth noting that the amount of literature available on COVID-19 is much larger than that on SARS. In fact, a PubMed search using the term "COVID-19" provides over 100,000 results from the period of 2019 to early-2021 alone. In contrast, a search using the term "SARS" between 2002 and 2019 provides a list of 9,000 results. Therefore, a comparative discussion using the available literature is prone to selection and/or reporting bias. Additionally, since the SARS outbreak was more concentrated in Asia compared to the global distribution of COVID-19, the findings related to SARS may not be representative of the potential spectrum of clinical disease, complications and public health implications, had it spread globally to more diverse populations. Finally, a larger dedicated international effort has been directed towards studying COVID-19 compared to the SARS epidemic. Again, this highlights the need for a cautious approach when drawing parallels between SARS and COVID-19, despite the obvious and significant similarities between both viruses.

Future perspective

The past is the present's gift for the future; if we choose to accept it, we may be carving the path for well-informed, proactive measures for the management of future health crises. These measures cannot focus only on health, but must involve a collaborative effort that takes into consideration global and community-level inequalities. It is evident, as the scientific and clinical community tackles the COVID-19 pandemic with incomplete knowledge, that previous epidemics, such as SARS, can guide us to better understand the trajectory of the outbreak, the expected pathogenesis, and potential complications of the disease, both clinically and at the population level. Global health issues of this magnitude require multidisciplinary efforts from governments, research and scientific bodies, health care systems, and international entities. As evident from the current situation and past experiences, it is imperative that strict measures for virus source control are implemented, including the banning, or at least regulation, of the wild animal trade, and global monitoring and surveillance efforts for zoonotic viral reservoirs and significant viral mutations. Without significant changes in the global approach to zoonotic diseases, another coronavirus outbreak in the next few years would not be unthinkable. Viral outbreaks and infectious diseases have plagued humanity for centuries, and it is inevitable that they will continue to do so. However, we hope that lasting changes will result from this unprecedented modern-day outbreak, and that the next comparative review on the topic will be discussing the proactive infection prevention measures, successful containment, efficient global response, and rapid activation of public health emergency systems in the effort to reduce the magnitude of damage from the next pandemic.

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