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A review of coronavirus disease-2019 (Covid-19)

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ABSTRACT

There is a new public health crises threatening the world with the emergence and spread of 2019 novel coronavirus (2019-nCoV) or the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The disease is transmitted by inhalation or contact with infected droplets and the incubation period ranges from 2 to 14 days. The symptoms are usually fever, cough, sore throat, breathlessness, fatigue, malaise among others. In some people it may progress to pneumonia, acute respiratory distress syndrome (ARDS) and multi organ dysfunction. Many people are asymptomatic. Diagnosis is by demonstration of the virus in respiratory secretions by special molecular tests. Common laboratory findings include normal/ low white cell counts with elevated C-reactive protein (CRP). The computerized tomographic chest scan is usually abnormal even in those with no symptoms or mild disease. Role of antiviral agents is yet to be established. Prevention entails home isolation of suspected cases and those with mild illnesses and strict infection control measures at hospitals that include contact and droplet precautions. The virus spreads faster than its two ancestors the SARS-CoV and Middle East respiratory syndrome coronavirus (MERS-CoV), but has lower fatality. The global impact of this new epidemic is yet uncertain.

Keywords: Nidovirales; Coronavirus; Positive-sense RNA viruses; SARS-CoV

INTRODUCTION

Coronaviruses are a group of enveloped viruses with non-segmented, single-stranded, and positive sense RNA genomes. Apart from infecting a variety of economically important vertebrates (such as Bats, pigs and chickens), six coronaviruses have been known to infect human hosts and cause respiratory diseases [1]. Among them, severe acute respiratory syndrome coronavirus (SARS-CoV) and Middle East respiratory syndrome coronavirus (MERS-CoV) are zoonotic and highly pathogenic coronaviruses that have resulted in regional and global outbreaks. The 2019 novel coronavirus (2019-nCoV), fortunately so far, children have been in frequently affected with no deaths. Since knowledge about this virus is rapidly evolving, readers are urged to update themselves-regularly.

Classification

According to the International Committee on Taxonomy of Viruses, coronaviruses are classified under [4]**.** Prior to the emergence of SARS-CoV, only two HCoVs (HCoV-229E and HCoV-OC43) were known, both causing mild upper respiratory symptoms when inoculated to healthy adult volunteers [5]. Two more HCoVs, HCoV-NL63 and HCoV-HKU1, were identified in 2004 and 2005, respectively [31]. Together, these four globally distributed HCoVs presumably contribute to 15–30% of cases of common cold in humans [6]**.** Although diseases are generally self-limiting, these mild HCoVs can sometimes cause severe lower respiratory infections in infants, elderly people, or immunocompromised patients [7, 19].

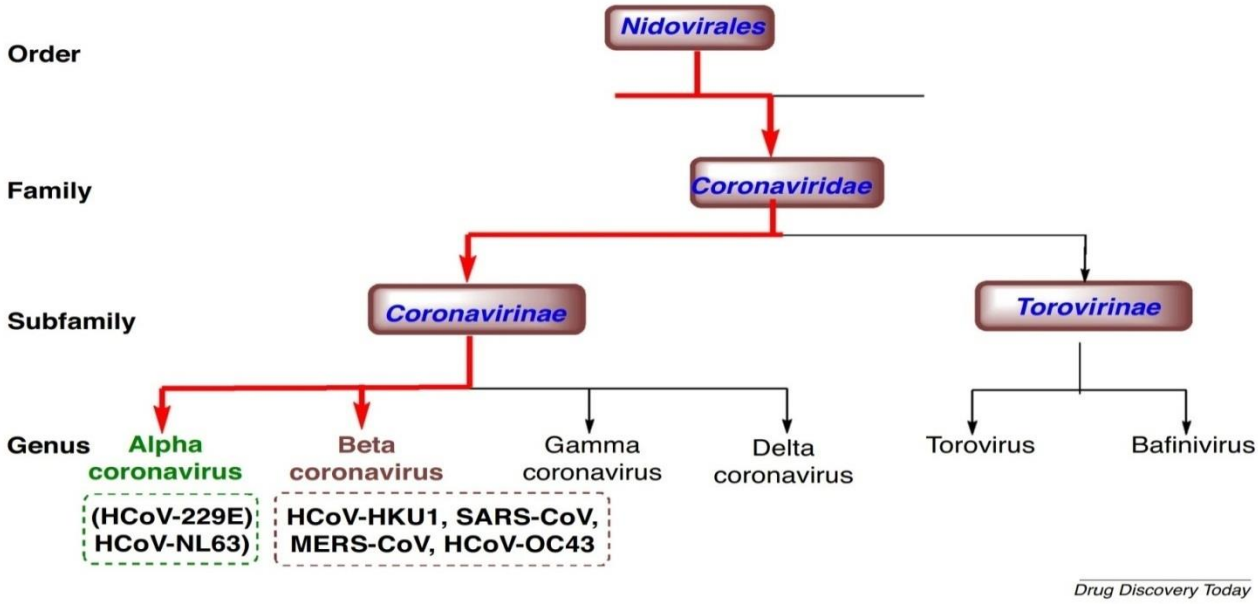

Figure 1: Taxonomy of HCoVs: the updated classification scheme of HCoV and other coronaviruses.

Similar to SARS-CoV and MERS-CoV, HCoV-NL63 and HCoV-229E originated in bats, whereas HCoV-OC43 and HCoV-HKU1 likely originated in rodents [8]. Importantly a majority of alpha corona viruses and betacoronaviruses were identified only in bats, and many coronaviruses phylogenetically related to SARSCoV and MERS-CoV were discovered in diverse bat species. Therefore,emerging zoonotic HCoVs such as SARS-CoV and MERS-CoV likely originated in bats through sequential mutation and recombination of bat coronaviruses, underwent further mutations during the spillover to intermediate hosts,and finally acquired the ability to infect human hosts [8]

Tabel 1 : Classification,discovery, cellular response, and natural host of the coronavirus

HCoV genera	Coronaviruses	Discovery	Cellular receptors	Natural Host(s)
α - Coronaviruses	HCoV-229E	1966	Human aminopeptidase	Bats
	HCoV-NL63	2004	N(CD13)	Palm
	NCoV	2019	ACE2	Civets,Bats
			ACE2	Bats
β- Coronaviruses	HCoV-OC43 HCoV-HKU1 SARS-CoV MERS-CoV	1967	9-O-Acetylated sialicacid	Cattle
		2005	9-O-Acetylated sialicacid	Mice
		2003	ACE2	PalmCivets,
		2012	DPP4	Bats,Camels

History

Coronaviruses are enveloped positive sense RNA viruses ranging from 60 nm to 140 nm in diameter with spike like projections on its surface giving it a crown like appearance under the electron microscope; hence the name coronavirus [9]. There have been two events in the past two decades where in crossover of animal betacorona viruses to humans has resulted in severe disease. The first such instance was in 2002– 2003 when a new coronavirus of the β genera and with origin in bats crossed over to humans via the intermediary host of palm civet cats in the Guangdong province of China. This virus, designated as severe acute respiratory syndrome coronavirus affected 8422 people mostly in China and Hong Kong and caused 916 deaths (mortality rate 11%) before being contained [10]**.** Almost a decade later in 2012, the Middle East respiratory syndrome coronavirus (MERS-CoV), also of bat origin, emerged in Saudi Arabia with dromedary camels as the intermediate host and affected 2494 people and caused 858 deaths and South Korea (2015) (fatality rate 34%) [11]

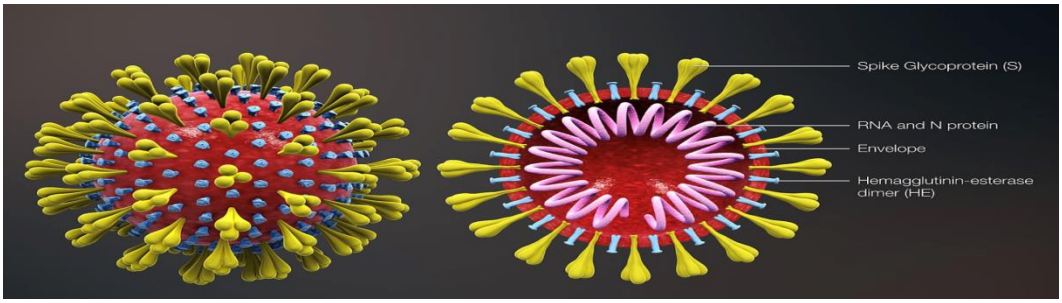

Figure2: Structure of Novel Corona virus (nCoV)

Origin and Spread of COVID-19

In December 2019, adults in Wuhan, capital city of Hubei province and a major transportation hub of China started presenting to local hospitals with severe pneumonia of unknown cause. On December 31st 2019, China notified the outbreak to the World Health Organization and on 1st January the Huanan sea food market was closed. On 7th January the virus was identified as a coronavirus that had >95% homology with the bat coronavirus and>70% similarity with the SARSCoV. Environmental samples from the Huanan sea food market also tested positive, signifying that the virus originated from there [12]**.** The first fatal case was reported on 11th Jan 2020. The massive migration of Chinese during the Chinese New Year fuelled the epidemic. Cases in other provinces of China, other countries (Thailand, Japan and South Korea in quick succession) were reported in people who were returning from Wuhan. Transmission to healthcare workers caring for patients was described on 20th Jan, 2020. By 23rd January, the 11 million population of Wuhan was placed under lock down with restrictions of entry and exit from the region. Soon this lock down was extended to other cities of Hubei province. Cases of COVID-19 in countries outside China were reported in those with no history of travel to China suggesting that local human-to-human transmission was occurring in these countries [13]**.** Airports in different countries including India put in screening mechanisms to detect symptomatic people returning from China and placed them in isolation and testing them for COVID-19. Soon it was apparent that the infection could be transmitted from asymptomatic people and also before onset of symptoms. Therefore, countries including India who evacuated their citizens from Wuhan through special flights or had travellers returning from China, placed all people symptomatic or otherwise in isolation for 14 days and tested them for the virus. Cases continued to increase exponentially and modelling studies reported an epidemic doubling time of 1.8 day [14]. In fact on the 12th of February, China changed its definition of confirmed cases to include patients with negative/ pending molecular tests but with clinical, radiologic and epidemiologic features of COVID-

19 leading to an increase in cases by 15,000 in a single day [15]. It is important to note that while the number of new cases has reduced in USA, Italy, Spain and China, lately, they have increased exponentially in other countries including South Korea, Iran. One case was reported in an Indian who traveled back from Vienna and exposed a large number of school children in a birthday party at a city hotel. Many of the contacts of these cases have been quarantined. These numbers are possibly an underestimate of the infected and dead due to limitations of surveillance and testing. Though the SARS-CoV-2 originated from bats, the intermediary animal through which it crossed over to humans is uncertain.

Attachment and Entry

Coronavirus replication is initiated by the binding of S protein to the cell surface receptor(s). The S protein is composed of two functional subunits, S1 (bulb) for receptor binding and S2 (stalk) for membrane fusion. Specific interaction between S1 and the cognate receptor triggers a drastic conformational change in the S2 subunit, leading to the fusion between the virus envelope and the cellular membrane and release of the nucleocapsid into the cytoplasm [16].

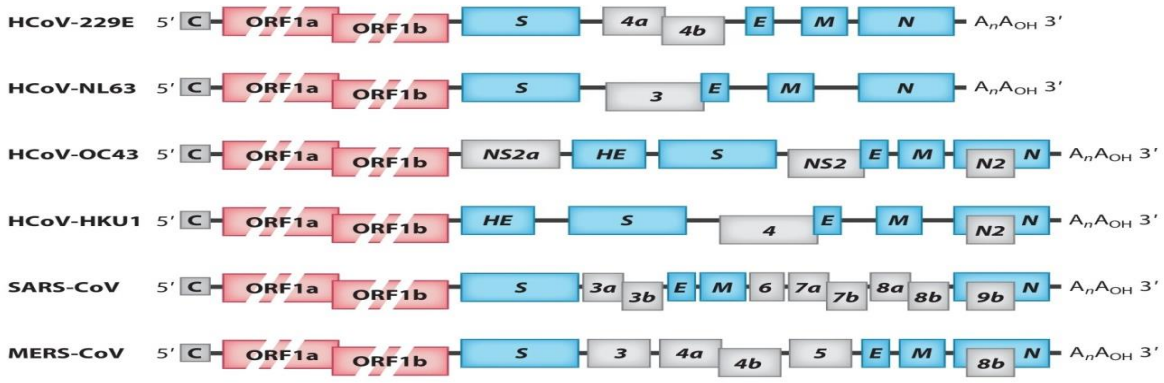

Figure 3: Genome structure of human coronaviruses (HCoVs).

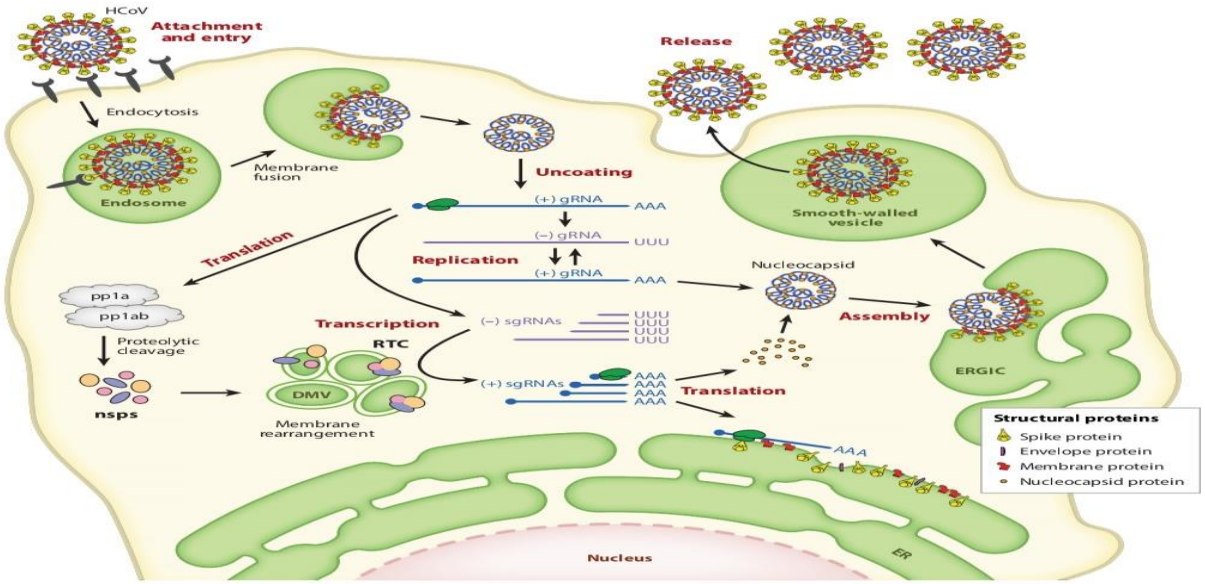

Figure 4: Replication cycle of human coronaviruses (HCoVs). Schematic diagram showing the general replication cycle of HCoVs

Replication cycle of human coronaviruses (HCoVs). Schematic diagram showing the general replication cycle of HCoVs. Infection starts with the attachment of HCoVs to the cognate cellular receptor, which induces endocytosis. Membrane fusion typically occurs in the endosomes, releasing the viral nucleocapsid to the cytoplasm. The genomic RNA (gRNA) serves as the template for translation of polyproteins pp1a and pp1ab, which are cleaved to form non-structural proteins (nsps). nsps induce the rearrangement of cellular membrane to form double-membrane vesicles (DMVs), where the viral replication transcription complexes (RTCs) are anchored. Full-length gRNA is replicated via a negative-sense intermediate, and a nested set of sub genomic RNA (sgRNA) species are synthesized by discontinuous transcription. These sgRNAs encode viral structural and accessory proteins. Particle assembly occurs in the ER-Golgi intermediate complex (ERGIC), and mature virions are released in smooth-walled vesicles via the secretory pathway [17]**.**

Pathogenesis

All ages are susceptible. Infection is transmitted through large droplets generated during coughing and sneezing by symptomatic patients but can also occur from asymptomatic people and before onset of symptoms. Studies have shown higher viral loads in the nasal cavity as compared to the throat with no difference in viral burden between symptomatic and asymptomatic people [18]**.** These infected droplets can spread 1–2 m and deposit on surfaces. The virus can remain viable on surfaces for days in favorable atmospheric conditions but are destroyed in less than a minute by common disinfectants like sodium hypochlorite, hydrogen peroxide etc. [20]**.** Infection is acquired either by inhalation of these droplets or touching surfaces contaminated by them and then touching the nose, mouth and eyes. The virus is also present in the stool and contamination of the water supply and subsequent transmission via aerosolization/ feco oral route is also hypothesized[15].As per current information, transplacental transmission from pregnant women to their fetus has not been described [21]**.** However, neonatal disease due to post-natal transmission is described. The incubation period varies from 2 to 14 days [median 5 d]. Studies have identified angiotensin receptor 2 (ACE2) as the receptor through which the virus enters the respiratory mucosa [22]**.** These animals also show increased levels pro-inflammatory cytokines and reduced T-cell responses, suggesting a possible immunopathological mechanism of disease [23, 24]. More recently, a novel coronavirus named SW1 was identified in a deceased Beluga whale [25]**.** Large numbers of virus particles were identified in the liver of the deceased whale with respiratory disease and acute liver failure. Although, electron microscopic images were not sufficient to identify the virus as a coronavirus, sequencing of the liver tissue clearly identified the virus as a coronavirus [26]**.**

Clinical Features

The clinical features of COVID-19 are varied, ranging from asymptomatic state to acute respiratory distress syndrome and multi organ dysfunction. The common clinical features include fever (not in all), cough, sore throat, headache, fatigue, headache, myalgia and breathlessness. Conjunctivitis has also been described. Thus, they are indistinguishable from other respiratory infections. In a subset of patients, by the end of the first week the disease can progress to pneumonia, respiratory failure and death. This progression is associated with extreme rise in inflammatory cytokines including IL2, IL7, IL10, GCSF, IP10, MCP1, MIP1A, and TNFα [27]. The median time from onset of symptoms to dyspnea was 5 days, hospitalization 7 days and acute respiratory distress syndrome (ARDS) 8 days. The need for intensive care admission was in 25–30% of affected patients in published series. Complications witnessed included acute lung injury, ARDS, shock and acute kidney injury. Recovery started in the 2nd or 3rd wk. The median duration of hospital stay in those who recovered was 10 days. Adverse outcomes and death are more common in the elderly and those with underlying co-morbidities (50–75% of fatal cases). This may either be due to selection bias wherein the cases reporting from Wuhan included only the severe cases or due to predisposition of the Asian population to the virus due to higher expression of ACE2 receptors on the respiratory mucosa.

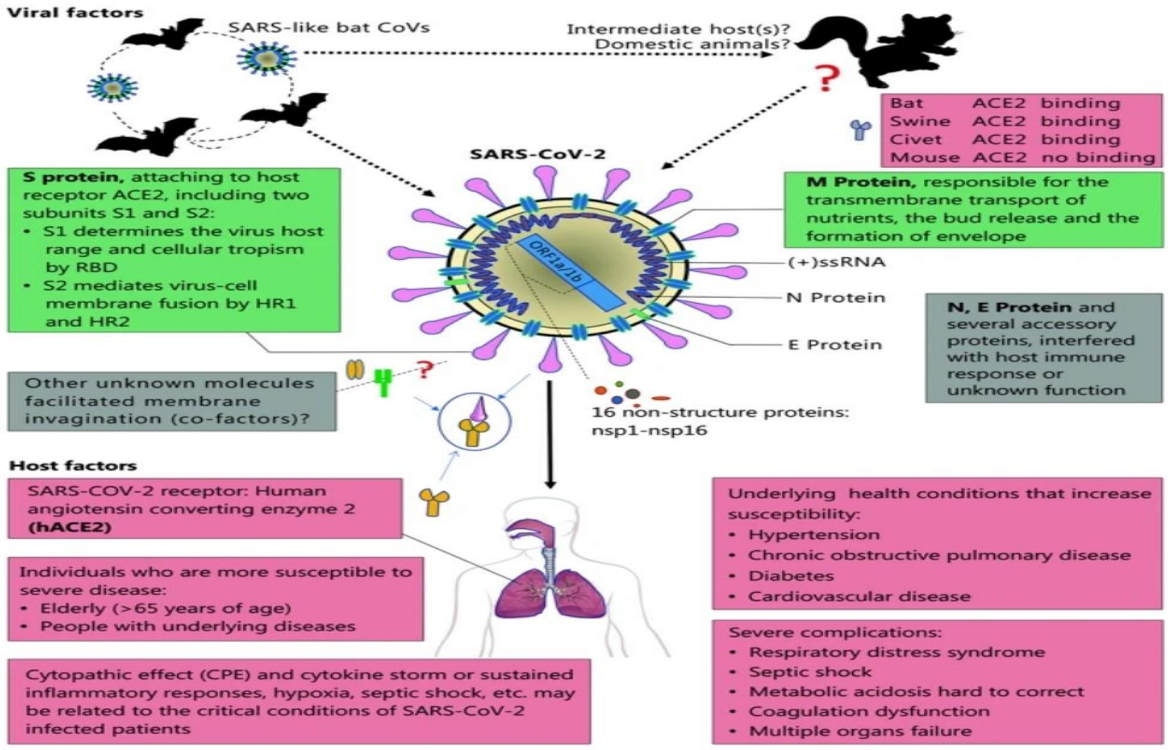

Figure 5: The origin, Transmission and clinical therapies n coronavirus disease 2019

Diagnosis

A suspect case is defined as one with fever, sore throat and cough who has history of travel to China or other areas of persistent local transmission or contact with patients with similar travel history or those with confirmed COVID-19 infection. A confirmed case is a suspect case with a positive molecular test. Specific diagnosis is by specific molecular tests on respiratory samples (throat swab/ nasopharyngeal swab/ sputum/ endotracheal aspirates and bronchoalveolar lavage). Virus may also be detected in the stool and in severe cases, the blood. It must be remembered that the multiplex PCR panels currently available do not include the COVID-19. In a suspect case in India, the appropriate sample has to be sent to designated reference labs in India or the National Institute of Virology in Pune. As the epidemic progresses, commercial tests will become available. Other laboratory investigations are usually nonspecific. The white cell count is usually normal or low. There may be lymphopenia; a lymphocyte count <1000 has been associated with severe disease. The platelet count is usually normal or mildly low. The CRP and ESR are generally elevated but procalcitonin levels are usually normal. A high procalcitonin level may indicate a bacterial co-infection. The ALT/AST, prothrombin time, creatinine, D-dimer, CPK and LDH may be elevated and high levels are associated with severe disease.

The chest X-ray (CXR) usually shows bilateral infiltrates but may be normal in early disease. The CT is more sensitive and specific. CT imaging generally shows infiltrates, ground glass opacities and sub segmental consolidation. In fact, abnormal CT scans have been used to diagnose COVID-19 in suspect cases with negative molecular diagnosis; many of these patients had positive molecular tests on repeat testing [28]**.** The differential diagnosis includes all types of respiratory viral infections [influenza, parainfluenza, respiratory syncytial virus (RSV), adenovirus, human meta- pneumo-virus, non COVID-19 coronavirus], atypical organisms (mycoplasma, chlamydia) and bacterial infections. It is not possible to differentiate COVID-19 from these infections clinically or through routine lab tests [29].

Treatment

The first step is to ensure adequate isolation (discussed later) to prevent transmission to other contacts, patients and healthcare workers. Mild illness should be managed at home with counseling about danger signs. The usual principles are maintaining hydration and nutrition and controlling fever and cough. Routine use of antibiotics and antivirals such as oseltamivir should be avoided in confirmed cases. In hypoxic patients, provision of oxygen through nasal prongs, face mask, high flow nasal cannula (HFNC) or non-invasive ventilation is indicated. Mechanical ventilation and even extra corporeal membrane oxygen support may be needed. Renal replacement therapy may be needed in some. Antibiotics and antifungals are required if co-infections are suspected or proven. The role of corticosteroids is unproven; while current international consensus and WHO advocate against their use, Chinese guidelines do recommend short term therapy with low-to-moderate dose corticosteroids in COVID-19 ARDS [30, 31]**.** Antiviral drugs such as ribavirin, lopinavirritonavir have been used based on the experience with SARS and MERS. In a historical control study in patients with SARS, patients treated with lopinavir-ritonavir with ribavirin had better outcomes as compared to those given ribavirin alone.

In the case series of 99 hospitalized patients with COVID19 infection from Wuhan, oxygen was given to 76%, noninvasive ventilation in 13%, mechanical ventilation in 4%, extracorporeal membrane oxygenation (ECMO) in 3%, continuous renal replacement therapy (CRRT) in 9%, antibiotics in71%, antifungals in 15%, glucocorticoids in 19% and intravenous immunoglobulin therapy in 27% [27]**.** Antiviral therapy consisting of oseltamivir, ganciclovir and lopinavirritonavir was given to 75% of the patients. The duration of non-invasive ventilation was 4–22 d [median 9 d] and mechanical ventilation for 3–20 d [median 17 d]. In the case series of children discussed earlier, all children recovered with basic treatment and did not need intensive care [32]. More evidence is needed before these drugs are recommended. Other drugs proposed for therapy arearbidol (an antiviral drug available in Russia and China), intravenous immunoglobulin, interferons, chloroquine and plasma of patients recovered from COVID-19 [29, 33, 34].

Prevention

Since at this time there are no approved treatments for this infection, prevention is crucial. Several properties of this virus make prevention difficult namely, non-specific features of the disease, the infectivity even before onset of symptoms in the incubation period, transmission from asymptomatic people, long incubation period, tropism for mucosal surfaces such as the conjunctiva, prolonged duration of the illness and transmission even after clinical recovery [35]. For the corona virus to prevent covering mouth and nose when coughing and sneezing, infection spread includes regular hand washing and also symptoms of respiratory illness. The World Health Organization has advised people to avoid "unprotected" contact with live animals, thoroughly cook meat and eggs, and avoid close contact with anyone with cold or flu-like symptoms.

Isolation of confirmed or suspected cases with mild illness at home is recommended. The ventilation at home should be good with sunlight to allow for destruction of virus. Patients should be asked to wear a simple surgical mask and practice cough hygiene. Caregivers should be asked to wear a surgical mask when in the same room as patient and use hand hygiene every 15–20 min [36]**.** Standard recommend a basic hand hygiene, such as washing your hands with soap and water and respiratory hygiene, such as when you sneeze, sneezing into your elbow. Ways to protect yourself against a potential animal source would be to avoid unnecessary unprotected contact with live animals and to make sure that you wash your hands thoroughly after connecting with animal.

CONCLUSION

Over the past 50 years the emergence of many different coronaviruses that cause a wide variety of human and veterinary diseases has occurred. This new virus outbreak has challenged the economic, medical and public health infrastructure of China and to some extent, of other countries especially, its neighbours. Time alone will tell how the virus will impact our lives here in India. More so, future outbreaks of viruses and pathogens of zoonotic origin are likely to continue. Therefore, apart from curbing this outbreak, efforts should be made to devise comprehensive measures to prevent future outbreaks of zoonotic origin. It appears that bats and birds, the warm-blooded flying

vertebrates, are ideal hosts for the corona virus gene source (with bats for Alpha coronavirus and Beta coronavirus, and birds for Gamma corona virus and Delta corona virus) to fuel corona virus evolution and dissemination.

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