WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
SJIF Impact Factor 8.084
Volume 9, Issue 5, 889-900. Review Article ISSN 2277– 7105

CORONA VIRUS DISEASE 2019 (COVID-19): A REVIEW

Bhagyashri B. Sakhare*, Nandkishor B. Bavage, Shyamlila B. Bavage, Gali Vidyasagar

*Latur College of Pharmacy, Hasegaon, Ausa, Latur, Maharashtra – 413512, India.

Article Received on
10 March 2020,

Revised on 31 March 2020,
Accepted on 20 April 2020,

DOI: 10.20959/wjpr20205-17421

***Corresponding Author**
Bhagyashri B. Sakhare
Latur College of Pharmacy, Hasegaon, Ausa, Latur, Maharashtra – 413512, India.

ABSTRACT

COVID-19 has spread through out to most countries in the world. The impact of the corona virus disease is different in different countries. These differences are assigned to differences in all cultural norms mitigation efforts and health infrastructure. Coronavirus disease (COVID-19), which appeared in December 2019, presents a global challenge, particularly in the rapid increase of critically ill patients with pneumonia and absence of definitive treatment. The mortality appears to be around 2%; early published data indicate 25.9% with SARS-CoV-2 pneumonia required ICU admission and 20.1% developed acute respiratory distress syndrome. There is presently no vaccine or specific anti-viral drug regime used to treat critically ill patients. The management of patients mainly focuses on the provision of supportive care, e.g., oxygenation, ventilation, and fluid management. Combination treatment of low-dose systematic corticosteroids and anti-virals and atomization inhalation of interferon have been encouraged as part of critical COVID-19 management. This paper aggregates and consolidates the epidemiology, clinical manifestations, diagnosis, treatments and prevention of this new type of coronavirus. The impact of this new epidemic globally is yet uncertain.

KEYWORDS: Coronavirus, SARS-CoV-2, 2019-nCOV, Treatment, Review.

INTRODUCTION

Coronaviruses (CoVs), a large family of single-stranded RNA viruses, can infect animals and also humans, causing respiratory, gastrointestinal, hepatic, and neurologic diseases.[1] As the largest known RNA virus. Coronaviruses belong to the family Coronaviridae in the order Nidovirales.[10] They can be classified into four genera: Alphacoronavirus, Betacoronavirus, Gammacoronavirus, and Deltacoronavirus. Among them, alpha and betacoronaviruses infect mammals, gammacoronaviruses infect avian species, and deltacoronaviruses infect both

mammalian and avian species. Representative alphacoronaviruses include human coronavirus NL63 (HCoV-NL63), porcine transmissible gastroenteritis coronavirus (TGEV), PEDV, and porcine respiratory coronavirus (PRCV). Representative betacoronaviruses include SARS-CoV, MERS-CoV, bat coronavirus HKU4, mouse hepatitis coronavirus (MHV), bovine coronavirus (BCoV), and human coronavirus OC43. Representative gamma and deltacoronaviruses include avian infectious bronchitis coronavirus (IBV) and porcine deltacoronavirus (PdCV), respectively.[3] Coronaviruses are large, enveloped, positive-stranded RNA viruses.[4] They have the largest genome among all RNA viruses, typically ranging from 27 to 32 kb. The genome is packed inside a helical capsid formed by the nucleo capsid protein (N) and further surrounded by an envelope.[23] Associated with the viral envelope are at least three structural proteins: The membrane protein (M) and the envelope protein (E) are involved in virus assembly[11], whereas the spike protein (S) mediates virus entry into host cells. Some coronaviruses also encode an envelope-associated hemagglutinin-esterase protein (HE).[16, 17]

Origin and Spread of COVID-19

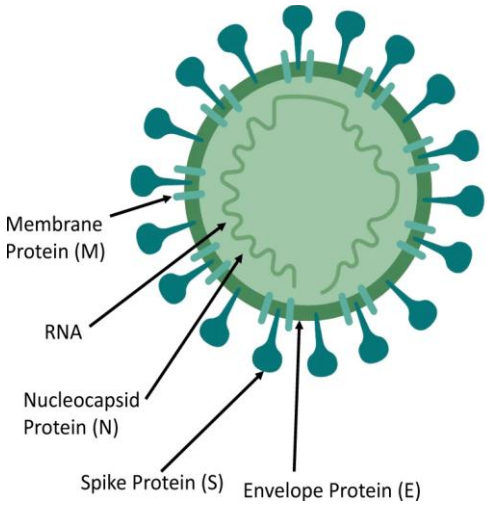
December 2019, adults in Wuhan, capital city of Hubeiprovince and a major transportation hub of China started presenting to local hospitals with severe pneumonia of unknown cause. Many of the initial cases had a common exposure to the Huanan wholesale seafood market that also traded live animals. The surveillance system (put into place after the SARS outbreak) was activated and respiratory samples of patients were sent to reference labs for etiologic investigations. 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 SARS-CoV. Environmental samples from the Huanan sea food market also tested positive, signifying that the virus originated from there.[7] The number of cases started increasing exponentially, some of which did not have exposure to the live animal market, suggestive of the fact that human-to-human transmission was occurring.[8] 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 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.[9,18] 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 day and tested them for the virus. Cases continued to increase exponentially and modeling studies reported an epidemic doubling time of 1.8 d.[10] 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 by15,000 in a single day.[6] As of 05/03/2020 96,000 cases worldwide (80,000 in China) and 87 other countries and 1international conveyance (696, in the cruise ship Diamond Princess parked off the coast of Japan) have been reported. It is important to note that while the number of new cases has reduced in China lately, they have increased exponentially in other countries including South Korea, Italy and Iran. Of those infected, 20% are in critical condition, 25% have recovered, and 3310 (3013 in China and 297 in other countries) have died.[2] India, which had reported only 3 cases till 2/3/2020, has also seen a sudden spurt in cases. By 5/3/2020, 29 cases had been reported; mostly in Delhi, Jaipur and Agra in Italian tourists and their contacts. 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 un-certain. Pangolins and snakes are the current suspects.

Classification

Coronaviruses (CoVs) are the largest group of viruses belonging to the Nidovirales order, which includes Coronaviridae, Arteriviridae, and Roniviridae families. The Coronavirinae comprise one of two subfamilies in the Coronaviridae family, with the other being the Torovirinae. The Coronavirinae are further subdivided into four groups, the alpha, beta,

gamma and delta coronaviruses.[19, 20, 22] The viruses were initially sorted into these groups based on serology but are now divided by phylogenetic cluster.[24]



Structural Protein	Function of Protein
Nucleocapsid Protein (N)	Bound to RNA genome to make up nucleocapsid
Spike Protein (S)	Critical for Binding of host cell receptors to facilitate entry of host cell
Envelope Protein (E)	Interacts with M to form viral envelope
Membrane Protein (M)	Central organizer of CoV assembly Determines shapes of viral envelope

Fig. Structure of Corona Virus.

Clinical features

Incubation period: The exact incubation period is not known. It is presumed to be between 2 to 14 days after exposure, with most cases occurring within 5 days after exposure.[8, 9, and 10]

The spectrum of illness severity: Most infections are self-limiting. COVID-19 tends to cause more severe illness in elderly population or in patients with underlying medical problems. As per the report from Chinese center for disease control and prevention that included approximately 44,500 confirmed Infections with an estimation of disease severity.[11, 26, 27] Mild illness was reported in 81% patients. Severe illness (Hypoxemia, >50% lung involvement on imaging within 24 to 48 hours) in 14%. Critical Disease (Respiratory failure, shock, multi-organ dysfunction syndrome) was reported in 5 percent.[31,32] Overall case fatality rate was between 2.3 to 5%.

Age affected: Mostly middle aged (>30 years) and elderly. Symptomatic infection in children appears to be uncommon, and when it occurs, it is usually mild.[42]

Clinical Presentation: In a study describing 1099 patients with COVID-19 pneumonia in Wuhan, the most common clinical features at the onset of illness.[41]

- Fever in 88%
- Fatigue in 38%
- Dry cough in 67%

- Myalgias in 14.9%
- Dyspnea in 18.7%

Pneumonia appears to be the most common and severe manifestation of infection. In this group of patients breathing difficulty developed after a median of five days of illness.[39] Acute respiratory distress syndrome developed in 3.4% of patients.[29]

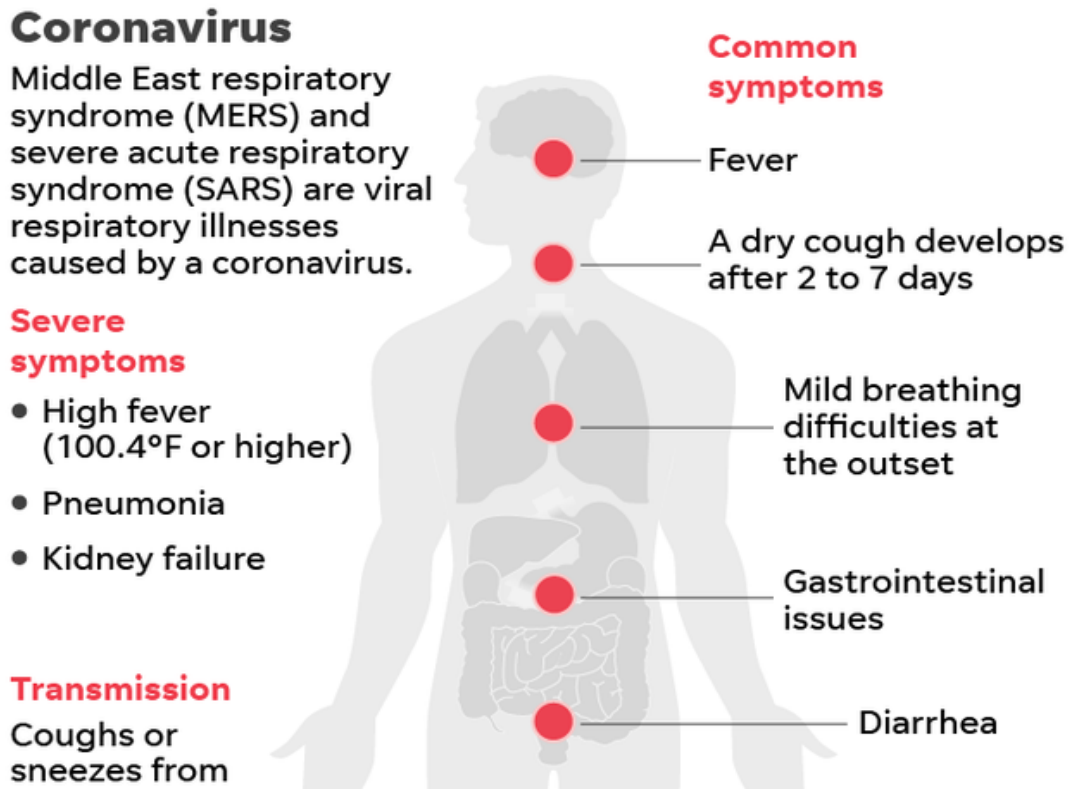


Fig. Symptoms of coronavirus disease.

Diagnosis

Clinical Diagnosis

The SARS-CoV-2 infected cases have symptoms like fever, fatigue, dry cough, dyspnea etc., with or without nasal congestion, runny nose or other upper respiratory symptoms.[1, 12, 25] Despite the atypical symptoms were reported pointed out that fever is still the typical symptom of SARS-CoV-2 infection.[40]

a) Physical examination: Patients with mild symptoms may not be present positive signs. Patients in severe condition may have shortness of breath, moist rales in lungs, weakened breath sounds, dullness in percussion, and increased or decreased tactile speech tremor, etc.

b) CT imaging examination: The imaging finding vary with the patient`s age, immunity status, disease stage at the time of scanning, underlying diseases, and drug interventions. Chest X-

ray examination In the early stage of pneumonia cases, chest images show multiple small patchy shadows and interstitial changes[14], remarkable in the lung periphery.[21] Severe cases can further develop to bilateral multiple ground-glass opacity, infiltrating shadows, and pulmonary consolidation, with infrequent pleural effusion. While Chest CT scans Pulmonary lesions are shown more clearly by CT than X-ray examination, including ground-glass opacity and segmental consolidation in bilateral lungs, especially in the lung periphery. In children with severe infection, multiple lobar lesions may be present in both lungs. A study of CT scans of 21 patients with SARS-CoV-2 infection showed that three (21%) with normal CT scans, 12 (57%) with ground-glass opacity only, and six (29%) with ground-glass opacity and consolidation at presentation.[42] Another study of 41 patients with confirmed SARS-CoV-2 infection was reported to have bilateral lung involvement on chest radiographs.[13, 14] Overall, the imaging findings reported for CoVID-19 are similar to those reported with SARS[43, 44] and MERS[45, 46] not surprising as the responsible viruses are also coronaviruses.

Laboratory Diagnosis- It mainly should be distinguished from other known viral virus of pneumonia, such as influenza viruses, parainfluenza virus, adenovirus, respiratory syncytial virus, rhinovirus, SARS-CoV, etc.; and also, from mycoplasma pneumonia, chlamydia pneumonia, and bacterial pneumonia. It should be distinguished from non-infectious diseases, such as vasculitis, dermatomyositis, and organizing pneumonia.[9] So, laboratory diagnosis is necessary.

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. No treatment specifically available for COVID-19 and no cure for an infection. Treatments and vaccines are currently study. Treatment focuses on managing symptoms as the virus runs its course. Other coronavirus like SARS and MERS are also treated by managing Symptoms.[34, 35]

Therapies used for this illness include.

- Antiviral or retroviral medications.
- Breathing support, such as mechanical ventilation.
- Steroids to reduce lung swelling.
- Blood plasma transfusions.

- Other antimalarial Drug Hydroxychloroquine used to relieve viral Replication.

The combination regimen of steroids and ribavirin was the treatment protocols for SARS in Hong Kong.[47] Corticosteroid is commonly used as an immunomodulatory agent to modulate inflammatory cytokines to achieve immune homeostasis.[48] Steroid with high dosage was used to treat patients with SARS.[49] Ribavirin can suppress the replication of RNA virus. It has been used in treating hepatitis C virus, lassa fever virus and severe respiratory syncytial virus infection.[50] However, the usage of ribavirin did not reduce the intra-tracheal intubation or mortality rates.[28, 51] It has significant toxicity which may cause hemolytic anemia and electrolyte disturbances.[50]

Chinese guidelines do recommend that the short term therapy with low-to-moderate dose corticosteroids in COVID-19 ARDS.[25,38,39] Detailed guidelines for critical care management for COVID-19 have been published by the WHO.[26] Currently there is, as of now, no approved treatment for COVID-19.[30] Antiviral drugs such as ribavirin, lopinavir-ritonavir 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.[15]

Preventions

To prevent the spread of SARS and COVID-19, measures are based on effective infection control and isolation. The relative measures are mainly focused on 4 aspects, confirmed cases, close contact handling, community prevention as well as protection on healthcare workers.[18]

- Ideal post protective equipment: PPE for at-risk health facilities.
- Airborne precautions for aerosolized generating procedures: Gloves, Gloves nitrile, powder-free, non-sterile.
- Mask (health care worker): Medical mask, good breathability, internal and external faces should be clearly identified.
- Face Shield: Made of clear plastic and provides good visibility to both the wearer and the patient, Adjust-able band to attach firmly around the head and fit snuggly against the forehead, Fog resistant (preferable), Completely cover the sides and length of the face, May be re-usable (made of robust material which can be cleaned and disinfected) or disposable. Particulate respirator, grade N95 or higher N95 or FFP2 respirator or higher Good breathability with design that does not collapse against the mouth.[19, 20]

- Scrubs, tops: Tunic/tops, woven, scrubs, reusable or single use, short sleeved (tunic/tops), worn under-neath the coveralls or gown.
- Scrubs, pants: Trouser/pants, woven, scrubs, reusable or single use, short sleeved (tunic/tops), worn underneath the coveralls or gown.
- Apron, heavy duty: Straight apron with bib, Fabric: 100% polyester with PVC coating, or 100% PVC, or 100% rubber, or other fluid resistant coated material, Waterproof, Sewn strap for neck and back fastening. Minimum basis weight: 300g/m2covering size: 70-90 cm (width) X 120-150cm (height).[21]
- Goggles, protective

Good seal with the skin of the face, Flexible PVC frame to easily fit with all face contours with even pressure, Enclose eyes and the surrounding areas, Accommodate wearers with prescription glasses, Clear plastic lens with fog and scratch resistant treatments, Adjustable band to secure firmly so as not to become loose during clinical activity, Indirect venting to avoid fogging, May be re-usable (provided appropriate arrangements for decontamination are in place) or disposable.[22]

CONCLUSION

The causes, transmissions, symptoms and preventive measures of COVID-19 are reviewed above. However, diagnosis and treatments remain for further study. There is no specific treatment regimen, only has a broad spectrum of antiviral drugs for COVID-19 patients and no vaccine for prevention at present. Nowadays, personal hygiene and protection are the most important for preventing the spread of COVID-19 such as wearing a mask and washing hands as well as reducing social contact including avoiding crowds, working in home, so on. Corona virus disease 2019 was reported as cluster of disease in China in December 2019. WHO declared COVID-19 as a pandemic and it has since spread to all continents except Antarctica. Elderly persons with co-morbidities are more affected. It spreads mainly via Respiratory droplet Pneumonia is the most common complication. Presently there is no standardized treatment or vaccine.

REFERENCES

1. Weiss SR, Leibowitz JL. Coronavirus pathogenesis. Adv Virus Res, 2011; 81: 85-164.
2. Enjuanes L, Almazan F, Sola I, Zuniga S. Biochemical aspects of coronavirus replication and virus-host interaction. Annu. Rev. Microbiol, 2006; 60: 211–30.

3. Perlman S, Netland J. Coronaviruses post-SARS: update on replication and pathogenesis. Nat. Rev. Microbiol, 2009; 7: 439–50.
4. Li Q, Guan X, Wu P, et al. Early Transmission Dynamics in Wuhan, China, of Novel Coronavirus Infected Pneumonia. N Engl J Med 2020.
5. Guan WJ, Ni ZY, Hu Y, et al. Clinical Characteristics of Coronavirus Disease 2019 in China. N Engl J Med 2020.
6. Chan JF, Yuan S, K ok KH, et al. A familial cluster of pneumonia associated with the 2019 novel coronavirus indicating person-to person transmission: a study of a family cluster. Lancet, 2020; 395: 514.
7. Wu Z, McGoogan JM. Characteristics of and Important Lessons from the Coronavirus Disease 2019 (COVID-19) Outbreak in China: Summary of a Report of 72 314 Cases from the Chinese Center for Disease Control and Prevention. JAMA, 2020.
8. Huang C, Wang Y, Li X, et al. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. Lancet 2020; 395: 49740. Clinical Characteristics of Coronavirus Disease 2019 in China. W. Guan, Z. Ni. Yu Hu, W. Liang, C. Ou, J. He, L. Liu.
9. Clinical Characteristics of Coronavirus Disease 2019 in China. W. Guan, Z. Ni, Yu Hu, W. Liang, C. Ou, J. He, L. Liu, H. Shan, C. Lei, D.S.C. Hui, B. Du, L. Li, G. Zeng, K.-Y. Yuen, R. Chen.
10. Wei M, Yuan J, Liu Y, Fu T, Yu X, Zhang ZJ. Novel Coronavirus Infection in Hospitalized Infants Under 1 Year of Age in China. JAMA, 2020 Feb 14.2020.2131.
11. Richman DD, Whitley RJ, Hayden FG, eds. Clinical virology, 4th edition. Washington: ASM Press, 2016.
12. Ksiazek TG, Erdman D, Goldsmith CS, et al. A novel coronavirus associated with severe acute respiratory syndrome. N Engl J Med, 2003; 348: 1953–66.
13. Kuiken T, Fouchier RAM, Schutten M, et al. Newly discovered coronavirus as the primary cause of severe acute respiratory syndrome. Lancet, 2003; 362: 263–70.
14. Drosten C, Günther S, Preiser W, et al. Identification of a novel coronavirus in patients with severe acute respiratory syndrome. N Engl J Med, 2003; 348: 1967–76.
15. de Groot RJ, Baker SC, Baric RS, et al. Middle East respiratory syndrome coronavirus (MERS-CoV): announcement of the Coronavirus Study Group. J Virol, 2013; 87: 7790–92.

16. Zaki AM, van Boheemen S, Bestebroer TM, Osterhaus ADME, Fouchier RAM. Isolation of a novel coronavirus from a man with pneumonia in Saudi Arabia. N Engl J Med, 2012; 367: 1814–20.
17. WHO. Summary of probable SARS cases with onset of illness from 1 November, 2002 to 31 July2003. Dec 31, 2003.
18. WHO. Middle East respiratory syndrome coronavirus (MERS-CoV). November, 2019.
19. WHO. Novel coronavirus – China. Jan 12, 2020.
20. WHO. Novel coronavirus – Thailand (ex-China). Jan 14 2020.
21. WHO. Novel coronavirus – Japan (ex-China). Jan 17, 2020.
22. WHO. Novel coronavirus – Republic of Korea (ex-China). Jan 21, 2020.
23. CDC. First travel-related case of 2019 novel coronavirus detected in United States. Jan 21, 2020.
24. Tan W, Zhao X, Ma X, et al. A novel coronavirus genome identified in a cluster of pneumonia cases — Wuhan, China, 2019−2020.
25. Gimeno C, Lloret T, et al. Relationship between the presence of hypoxemia and the inflammatory response measured by C-reactive protein in bacteremic pneumococcal pneumonia. Eur Respir J, 2011; 38(suppl 55): 2492.
26. Kidney disease: improving global outcomes (KDIGO) acute kidney injury work group. KDIGO clinical practice guideline for acute kidney injury. March, 2012.
27. Garner JS, Jarvis WR, Emori TG, Horan TC, Hughes JM. CDC definitions for nosocomial infections, 1988. Am J Infect Control, 1988; 16: 128–40.
28. Gao C, Wang Y, Gu X, et al. Association between cardiac injury and mortality in hospitalized patients infected with avian influenza A (H7N9) virus. Crit Care Med, 2020; published online Jan 20.
29. Perlman S, Netland J. Coronaviruses post-SARS: update on replication and pathogenesis. Nat Rev Microbiol, 2009; 7: 439–50.
30. Lee N, Hui D, Wu A, et al. A major outbreak of severe acute respiratory syndrome in Hong Kong. N Engl J Med, 2003; 348: 1986–94.
31. Assiri A, Al-Tawfiq JA, Al-Rabeeah AA, et al. Epidemiological, demographic, and clinical characteristics of 47 cases of Middle East respiratory syndrome coronavirus disease from Saudi Arabia: a descriptive study. Lancet Infect Dis, 2013; 13: 752–61.
32. Wong CK, Lam CWK, Wu AKL, et al. Plasma inflammatory cytokines and chemokines in severe acute respiratory syndrome. Clin Exp Immunol, 2004; 136: 95–103.

33. Mahallawi WH, Khabour OF, Zhang Q, Makhdoum HM, Suliman BA. MERS-CoV infection in humans is associated with a pro-inflammatory Th1 and Th17 cytokine profile. Cytokine, 2018; 104: 8–13.
34. He L, Ding Y, Zhang Q, et al. Expression of elevated levels of pro-inflammatory cytokines in SARS-CoV-infected ACE2+ cells in ARS patients: relation to the acute lung injury and pathogenesis of SARS. J Pathol, 2006; 210: 288–97.
35. Faure E, Poissy J, Goffard A, et al. Distinct immune response in two MERS-CoV-infected patients: can we go from bench to bedside? PLoS One, 2014; 9: e88716.
36. Falzarano D, de Wit E, Rasmussen AL, et al. Treatment with interferon-α2b and ribavirin improves outcome in MERS-CoV-infected rhesus macaques. Nat Med, 2013; 19: 1313–17.
37. Stockman LJ, Bellamy R, Garner P. SARS: systematic review of treatment effects. PLoS Med, 2006; 3: e343.
38. Lansbury L, Rodrigo C, Leonardi-Bee J, Nguyen-Van-Tam J, Lim WS. Corticosteroids as adjunctive therapy in the treatment of influenza. Cochrane Database Syst Rev, 2019; 2: CD010406.
39. Arabi YM, Mandourah Y, Al-Hameed F, et al. Corticosteroid therapy for critically ill patients with Middle East respiratory syndrome. Am J Respir Crit Care Med, 2018; 197: 757–67.
40. WHO. Clinical management of severe acute respiratory infection when novel coronavirus (n Co V) infection is suspected, Jan 11, 2020.
41. Chu CM. Role of lopinavir/ritonavir in the treatment of SARS: initial virological and clinical findings. Thorax, 2004; 59: 252–56.
42. Arabi YM, Alothman A, Balkhy HH, et al. Treatment of Middle East respiratory syndrome with a combination of lopinavir-ritonavir and interferon-β1b (MIRACLE trial): study protocol for a randomized controlled trial. Trials, 2018; 19: 81.
43. Harder J, Schröder JM. Antimicrobial peptides role in human health and dis-ease. Springer International Publishing, 2015
44. Vigant F, Santos NC, Lee B. Broad-spectrum antivirals against viral fusion. Nat Rev Micro, 2015; 13(7): 426–37.
45. Taguchi F, Shimazaki YK. Functional analysis of an epitope in the S2 subunit of the murine coronavirus spike protein: involvement in fusion activity. J Gen Viral, 2000; 81(12): 2867–71.

46. Du L, He Y, Zhou Y, Liu S, Zheng B-J, Jiang S. The spike protein of SARS-CoV: a target for vaccine and therapeutic development. Nat Rev Microbiol, 2009; 7(3): 226–36.
47. Hilchie AL, Wuerth K, Hancock REW. Immune modulation by multi-faceted cationic host defense (antimicrobial) peptides. Nat Chem Biol, 2013; 9(12): 761–8.
48. Lu L, Liu Q, Zhu Y, Chan K-H, Qin L, Li Y, et al. Structure-based discovery of Middle East Respiratory Syndrome Coronavirus fusion inhibitor. Nat Commun, 2014; 5.
49. Shuwen L, Shuguang W, Shibo J. HIV entry inhibitors targeting gp 41: from polypeptides to small-molecule compounds. Curr Pharm Des, 2007; 13(2): 143–62.
50. Sainz B, Mossel EC, Gallaher WR, Wimley WC, Peters CJ, Wilson RB, et al. Inhibition of severe acute respiratory syndrome-associated coronavirus (SARS-Co V) infectivity by peptides analogous to the viral spike protein. Virus Res, 2006; 120(1-2): 146–55.
51. Rico-Mata R, De Leon-Rodriguez LM, Avila EE. Effect of antimicrobial peptides derived from human cathelicidin LL-37 on Entamoeba histolytica trophozoites. Exp Parasitol, 2013; 133(3): 300–6.