

Effect of Novel COVID-19 Infection on Different Organs of Human Body: A Narrative Review

Abdul Qader Hayat¹, Muhammad Sajid Hamid Akash²

¹Student, Department of Pharmaceutical Chemistry, Government College University Faisalabad, Pakistan

²Associate Professor, Department of Pharmaceutical Chemistry, Government College University Faisalabad, Pakistan

ABSTRACT

The COVID-19 (corona virus disease-2019) infection produces detrimental effect on vital organs of the human body leading towards mild to severe organ damage. The most drastic effects associated with COVID-19 infection include respiratory failure, cardiac arrest, liver injury and brain damage. The drugs which are used for the treatment of novel coronavirus are also associated with various side effects, which may prove fatal during treatment. This viral infection also reduces patients' immunity by binding with ACE2 (angiotensin-converting enzyme 2) receptors and modulates immune responses. Older people are particularly at a greater risk. The literature selected for this narrative study was searched and collected from two databases, Google Scholar and PubMed by using specific key words, from March 2020 to June 2020. The objective of this study was to increase understanding about COVID-19, particularly its effects on vital organs so that a better treatment strategy can be established.

Key words: Angiotensin-Converting Enzyme 2, COVID-19, Immunity, Organ damage

Authors' Contribution:

¹⁻²Conception; Literature search; Manuscript design and drafting; Critical analysis and Manuscript review; Manuscript editing.

Correspondence:

Abdul Qader Hayat
Email: Pharmacistqader316@gmail.com

Article info:

Received: April 16, 2020
Accepted: December 25, 2020

Cite this article. Hayat AQ, Akash MS. Effect of Novel COVID-19 Infection on Different Organs of Human Body: A Narrative Review. *J Islamabad Med Dental Coll.* 2020; 9(4): 303-306. Doi: 10.35787/jimdc.v9i4.532

Funding Source: Nil
Conflict of Interest: Nil

Introduction

About 82% gene sequence of 2019 Novel Coronavirus or SARS-CoV-2 (Severe Acute Respiratory Syndrome-Coronavirus 2) resembles SARS-CoV (Severe Acute Respiratory Syndrome-Coronavirus) and 50% with Middle-East Respiratory Syndrome Coronavirus (MERS-CoV).¹ Generally, COVID-19 is a reversible disease with 0.1 to 25% case fatality ratio globally.² In COVID-19, death usually occurs due to massive alveolar destruction and respiratory failure.³ These patients suffer either a

direct liver injury by viral invasion of cells or an indirect injury due to hepatotoxicity from drugs used in treatment.⁴ Studies reveal that SARS-CoV2 has potentially deleterious effects on central nervous system as well.⁵ According to WHO, there have been about 21294845 confirmed COVID-19 cases worldwide, including 761779 deaths till submission of this review. Although this virus can infect all ages, but children, elderly and people with comorbidities are at greater risk.⁶ Literature search for this narrative review was done mainly from two

databases (Google Scholar and PubMed) by using specific key words in English, from March 2020 to June 2020. Key words/phrases used included "Coronavirus", COVID-19", "SARS-CoV", "SARS-CoV-2", "Organ damage in COVID-19", "Liver and COVID-19", "CVS and COVID-19", "Respiratory System and COVID-19", "CNS and COVID-19", and "Angiotensin-Converting Enzyme 2". The objective of this study was to increase understanding about COVID-19, particularly its effects on vital organs so that a better treatment strategy can be established.

Cardiovascular System (CVS)

Novel coronavirus and MERS-CoV produce similar pathogenicity. Cardiac injury produced by novel coronavirus infection was reported in 5 of 41 earliest patients in Wuhan.³ According to NHC (National Health Commission, China), the coronavirus patients first complained about chest tightness and heart palpitations before the onset of typical symptoms such as shortness of breath, flu, cough, fever and sore throat.

Death in many COVID-19 patients has been reported due to cardiac arrest and heart damage during hospitalization.⁷ A survey-based data collected from 25 patients, who had recovered from SARS-CoV-2 infection, showed CVS abnormalities in 44% patients. These patients exhibited an abnormal biochemical profile, indicating high levels of serum free fatty acids (FFA), lysophosphatidyl ethanolamine (LPE), lysophosphatidylcholine (LPC) and phosphatidylglycerol (PG) as compared to normal individuals.⁸ Because the structure of SARS-CoV-2 is similar to SARS-CoV, there is a possibility of similar behavior in affecting CVS. People with comorbidities of CVS are at a higher risk and the death rate is also found to be high in such patients. Therefore, patients already suffering from CVS complications must be monitored carefully during treatment and given special care.⁷ During treatment, the administration of antiviral drugs should also be

carefully monitored because these drugs can lead to arrhythmias, cardiac insufficiency and other related complications.⁹

Liver

The chances of hepatic disturbance due to COVID-19 infection are high, probably due to the attack of SARS-CoV-2 on liver cells or adverse effects of the medicines taken by the patients.⁴ Liver injury has been described in a patient infected with novel coronavirus.¹⁰ In another study, elevation of AST (aspartate aminotransferase), a biomarker of liver injury, was recorded in 8 of 13 patients infected with SARS-CoV-2 in the intensive care unit. It was observed that the severe cases are more prone to liver injury than milder ones.⁷ Approximately 2–10% of patients with novel coronavirus infection present with diarrhea, and SARS-CoV-2 RNA has been detected in stool and blood samples.¹¹ However, the pathological investigation of hepatic tissues of a dead coronavirus patient showed no viral inclusions in the liver.¹¹

Drugs used in the treatment of COVID-19 may also produce hepatotoxicity. In addition, the immune-mediated inflammation, including pneumonia-associated hypoxia and cytokine storm might also cause liver injury in corona patients.⁷ The medicines being used to combat novel coronavirus like lopinavir, oseltamivir, ribavirin, ritonavir, hydroxychloroquine sulfate, chloroquine phosphate, and azithromycin are metabolized within hepatic cells. The hepatotoxicity associated with coronavirus can disturb the metabolism of these drugs leading to their higher concentrations in plasma causing increased chances of toxicity.¹²

Central Nervous System (CNS)

Many studies have shown the existence of SARS-CoV in CNS, where most of the viral strains are found in nerves.¹³⁻¹⁵ An experimental study has also revealed that when SARS-CoV and MERS-CoV are given

intranasally, these can enter into the brain via the olfactory nerves and spread rapidly to brain niches, specifically in the brainstem and thalamus.¹⁶ Surprisingly the MERS-CoV strains were isolated from only brain (not in lungs), and the brainstem appeared to be the most prominent site of infection by SARS-CoV-2.^{16,17} The actual route of entry of viral strains into CNS is still not clear, however some evidence suggests SARS-CoV strains first permeate into peripheral nerves, and then enter CNS via synapse-connections.^{18,19} Collectively, the neuroinvasive tendency is a common property of coronaviruses and due to similarity between SARS-CoV2 and SARS-CoV, it is possible that SARS-CoV2 also possesses the same potential. Thus, the virus enters into the brain and damages the medullary neurons. Some neurological symptoms observed in patients of COVID-19 in a study are headaches, loss of smell and taste, vomiting and nausea.⁵

Respiratory System

The novel coronavirus shows great resemblance with Middle-East Respiratory-Syndrome (MERS) and SARS-CoV infection.²⁰ In a study the histological features exhibited bilateral alveolar damage and multinucleated-syncytial cells in alveolar spaces, which was a clear indication of viral infection.¹¹

COVID-19 infection and ACE-2 receptor interaction

Angiotensin-converting enzyme 2 (ACE2) is an aminopeptidase that plays a very important role in immune and cardiovascular system. ACE2 has diverse functions. Most importantly this enzyme participates in the regulation of heart functions and has been implicated as a key receptor for binding of CoVs (coronaviruses) such as SARS-CoV2 and SARS-CoV. The SARS-CoV2 contains various proteins on its surface in a spike-like form and the infection of SARS CoV2 is stimulated when these viral proteins interact with ACE-2. This interaction creates harmful effects on patients' immune system and CVS.²¹ SARS-CoV2

mainly destroys alveolar cells which leads to respiratory symptoms.⁷

Limitation: This review included studies till June 2020, a lot of research must have been available till the publication of this manuscript. The element of bias cannot be ruled out because of unsystematic selection procedure inherent of the narrative reviews.

Conclusion

The current form of coronavirus is capable of destroying vital organs of human body. The heart, liver, CNS and lungs are more prone to the attack of this virus. Now, COVID-19 infection has become a global threat and it has spread worldwide. The population of some countries is at greater risk because of low levels of immunity owing to poor socioeconomic conditions. In many countries the biological researchers are struggling to combat COVID-19 infection. The Pakistani researchers should also play their part and should present their research initiatives to the Government. The government of Pakistan needs to set up proper research centers for this purpose so that we may have better treatment options not only for local community but also for the world in future.

References

1. Chau TN, Lee KC, Yao H, Tsang TY, Chow TC, Yeung YC, Choi KW, Tso YK, Lau T, Lai ST, Lai CL. SARS-associated viral hepatitis caused by a novel coronavirus: report of three cases. *Hepatology*. 2004; 39(2): 302-10. Doi: 10.1002/hep.20111.
2. World Health Organization. WHO: Estimating Mortality from COVID-19. [Online]. Available from: <https://www.who.int/news-room/commentaries/detail/estimating-mortality-from-covid-19>.
3. Chan, J.F.W., Yuan, S., Kok, K.H., To, K.K.W., Chu, H., Yang, J., Xing, F., Liu, J., Yip, C.C.Y., Poon, R.W.S. and Tsoi, H.W., 2020. 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(10223): 514-23. Doi: 10.1016/S0140-6736(20)30154-9.

4. Zhang C, Shi L, Wang FS. Liver injury in COVID-19: management and challenges. *Lancet Gastroenterol Hepatol.* 2020; 5(5): 428-30. Doi: 10.1016/S2468-1253(20)30057-1.
5. Li YC, Bai WZ, Hashikawa T. The neuro-invasive potential of SARS-CoV2 may play a role in the respiratory failure of COVID-19 patients. *J Med Virol.* 2020; 92(6): 552-5. Doi: 10.1002%2Fjmv.25728.
6. World Health Organization. Coronavirus disease 2019 (COVID-19): situation report, 146. <https://apps.who.int/iris/handle/10665/332403>.
7. Zheng YY, Ma YT, Zhang JY, Xie X. COVID-19 and the cardiovascular system. *Nat Rev Cardiol.* 2020; 17(5): 259-60. Doi: 10.1038/s41569-020-0360-5.
8. Wu Q, Zhou L, Sun X, Yan Z, Hu C, Wu J, et al. Altered lipid metabolism in recovered SARS patients twelve years after infection. *Sci Rep.* 2017; 7(1): 1-2. Doi: 10.1038/s41598-017-09536-z.
9. Sakabe M, Yoshioka R, Fujiki A. Sick sinus syndrome induced by interferon and ribavirin therapy in a patient with chronic hepatitis C. *J Cardiol Cas.* 2013; 8(6): 173-5. Doi: 10.1016/j.jccase.2013.08.002.
10. Yeo C, Kaushal S, Yeo D. Enteric involvement of coronaviruses: is faecal-oral transmission of SARS-CoV-2 possible? *Lancet Gastroenterol Hepatol.* 2020; 5(4): 335-7. Doi: 10.1016/S2468-1253(20)30048-0.
11. Xu Z, Shi L, Wang Y, Zhang J, Huang L, Zhang C, et al. Pathological findings of COVID-19 associated with acute respiratory distress syndrome. *Lancet Respir Med.* 2020; 8(4): 420-2. Doi: 10.1016/S2213-2600(20)30076-X.
12. Rismanbaf A, Zarei S. Liver and kidney injuries in COVID-19 and their effects on drug therapy; a letter to editor. *Arch Acad Emerg Med.* 2020; 8(1): e17. <http://journals.sbmu.ac.ir/aaem>.
13. Ding Y, He L, Zhang Q, Huang Z, Che X, Hou J, et al. Organ distribution of severe acute respiratory syndrome (SARS) associated coronavirus (SARS-CoV) in SARS patients: implications for pathogenesis and virus transmission pathways. *J Pathol.* 2004; 203(2): 622-30. Doi: 10.1002/path.1560.
14. Gu J, Gong E, Zhang B, Zheng J, Gao Z, Zhong Y, et al. Multiple organ infection and the pathogenesis of SARS. *J Exp Med.* 2005; 202(3): 415-24. Doi: 10.1084/jem.20050828.
15. Xu J, Zhong S, Liu J, Li L, Li Y, Wu X, et al. Detection of severe acute respiratory syndrome coronavirus in the brain: potential role of the chemokine Mig in pathogenesis. *Clin Infect Dis.* 2005; 41(8): 1089-96. Doi: 10.1086/444461.
16. Li K, Wohlford-Lenane C, Perlman S, Zhao J, Jewell AK, Reznikov LR, et al. Middle East respiratory syndrome coronavirus causes multiple organ damage and lethal disease in mice transgenic for human dipeptidyl peptidase 4. *J Infect Dis.* 2016; 213(5): 712-22. Doi: 10.1093/infdis/jiv499.
17. Netland J, Meyerholz DK, Moore S, Cassell M, Perlman S. Severe acute respiratory syndrome coronavirus infection causes neuronal death in the absence of encephalitis in mice transgenic for human ACE2. *J Virol.* 2008; 82(15): 7264-75. Doi: 10.1128/JVI.00737-08.
18. Li YC, Bai WZ, Hirano N, Hayashida T, Hashikawa T. Coronavirus infection of rat dorsal root ganglia: ultrastructural characterization of viral replication, transfer, and the early response of satellite cells. *Vir Res.* 2012; 163(2): 628-35. Doi: 10.1016/j.virusres.2011.12.021.
19. Matsuda K, Park CH, Sunden Y, Kimura T, Ochiai K, Kida H, et al. The vagus nerve is one route of transneuronal invasion for intranasally inoculated influenza A virus in mice. *Vet Pathol.* 2004; 41(2): 101-7. Doi: 10.1354%2Fvp.41-2-101.
20. Ding Y, Wang H, Shen H, Li Z, Geng J, Han H, et al. The clinical pathology of severe acute respiratory syndrome (SARS): a report from China. *J Pathol.* 2003; 200(3): 282-9. Doi: 10.1002/path.1440
21. Turner AJ, Hiscox JA, Hooper NM. ACE2: from vasopeptidase to SARS virus receptor. *Trends Pharmacol Sci.* 2004; 25(6): 291-4. Doi: 10.1016/j.tips.2004.04.001.