

An Update in the United Kingdom of the Current, Emerging and Future Treatment Strategies Regarding COVID-19 in the Face of the Global Pandemic

Carl Dowling*

BSc Paramedic Science Student, UK

*Corresponding author: Dowling C, BSc Paramedic Science Student, UK; E-mail: lyonhart37@hotmail.co.uk

Received: July 21, 2020; Accepted: August 05, 2020; Published: August 14, 2020



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Abstract

The recent outbreak of the novel coronavirus known as Covid-19 has caused many people from different parts of the world to become critically ill. At the start of the global pandemic, there was not enough information known about the novel virus to help fight the infection. As time has passed, with the death toll continuously rising, more research into current and future treatments have been conducted. This has helped healthcare professionals and scientists develop a better understanding of the novel virus, with how it affects the body and how current, emerging, and future treatment strategies can help to manage symptoms and reduce mortality.

Keywords: Covid-19; Comorbidities; Vaccine; Treatment strategies; Cytokine storms.

1. Introduction

According to the European Centre for Disease Prevention and Control (ECDC) [1] In late 2019, the first cases caused from the novel coronavirus known as SARS-CoV-2 (COVID -19) first emerged on 11th March 2020 due to the rapid spread and severity of the virus, The World Health Organization (WHO) declared a global pandemic. By the 30th June 2020 The Department of Health and Social Care [2] stated there was a total of 43,730 deaths reported in the United Kingdom (UK) caused by COVID-19, which has now affected 215 countries worldwide. This article will further examine the comorbidities of health that are at greater risk when contracting COVID-19 and evaluate current and upcoming treatments related to tackling the virus.

2. About COVID-19

He et al [3] states the virus is transmitted through air droplets and human contact. Additionally, He et al [3] explains that an individual who has contracted COVID-19 has spread it on to approximately 2 – 3 people with an incubation period between 7 – 14 days. According to Jin et al [4] Clinical features of COVID-19 include fever, continuous cough,

Citation: Dowling C. An update in the United Kingdom of the current, emerging and future treatment strategies regarding COVID-19 in the face of the global pandemic. Case Rep Rev Open Access. 2020;1(2):114.

fatigue, sputum production, headache, haemoptysis, and diarrhoea. Furthermore, Public Health England (PHE) [5] explains that other clinical features include loss of appetite, anosmia, ageusia, shortness of breath, nausea and vomiting. PHE [5] states that current data on the severity of COVID-19 specified that 40% of patients experience mild symptoms without hypoxia or pneumonia, 40% have moderate symptoms with non-severe pneumonia, 15% have symptoms with severe pneumonia and 5% have critical symptoms with associated life-threatening complications. Jin et al [4] explains that elderly people and individuals with comorbidities and underlying health conditions such as respiratory disease, cardiovascular disease, diabetes, transplants and cancer are at more risk of contracting COVID-19 leading to acute respiratory distress syndrome (ARDS). According to the European Society of Cardiology (ESC) [6] there is a link between ethnicity and the susceptibility of outcomes related to COVID-19 as within the UK, one third of patients admitted to intensive care from contracting COVID-19 were from an ethnic minority background.

3. How COVID-19 Affects the Body

According to Yuki et al [7] the cycle of the virus affecting the host consists of five steps: attachment, penetration, biosynthesis, maturation, and release. Kaur et al [8] mentions that evidence suggests when an individual first contracts COVID-19, the virus membrane spike can affix itself to the host's angiotensin converting enzyme (ACE2) receptor which is located within the lung's epithelium tissue. According to Mo et al [9] the lungs are the most effected organ by COVID-19 as pathologies include diffuse alveolar epithelium destruction, capillary damage and pulmonary consolidation. George et al [10] explains that one of the major risk factors associated with contracting COVID-19 is fibrosis where lung function persistently declines leading into (ARDS) followed by respiratory failure. Furthermore, Kaur et al [8] states that another major risk factor associated with COVID-19 is the rapid multiplication of the virus within the host, which can cause a hyperinflammatory response triggered by cytokine storms, as cytokine forms part of the body's innate immune response, however, too much cytokine production can lead to further tissue damage and possibly sepsis followed by multiple organ failure and death. Furthermore, Rao et al [11] states that the innate immunity is the body's first line of defence against an invader, where interferon molecules are produced by macrophages and discharged from infected cells, as a result from the infection, as interferon are able to activate an antiviral response in surrounding cells. However, Rao et al [11] explains that if the first line of defence is unsuccessful in defeating the infection within 4 – 7 days, Adaptive immunity where B-cells and T-cells are activated via lymphocytes to help fight the invader, this corresponds within the timeframe of individuals with COVID-19 who are showing worsening of symptoms. According to the European Society of Cardiology (ESC) [6] in immunity, T-cells are the chief inflammation cells which are crucial for virus clearing from the body, however, COVID-19 in mild cases T-cells were seen to have decreased, this was more prominent in severe cases.

4. Comorbidities of Health

According to Colafrancesco et al [12] COVID-19 can cause cardiovascular risks as the body's inflammatory response can activate the clotting cascade and platelets; this can cause an acceleration in atherosclerosis within the blood vessels of the heart. The ESC [6] states that common complications related to COVID-19 regarding the cardiovascular system are hypotension, myocarditis, arrhythmias and sudden cardiac death. Nosalski et al [13] states that viruses can be associated with metabolic dysfunction, myocardial inflammation and the activation of the sympathetic nervous system, which all can impact on the host's cardiac arrhythmia causing atrial fibrillation (AF), conduction block, ventricular tachycardia and

ventricular fibrillation. These arrhythmias are profoundly observed in viral myocarditis. Zeng et al [14] states that ACE2 plays a vital role in the cardiovascular and immune systems, as ACE2 is associated with the function of the heart and the development of hypertension and diabetes mellitus.

Orioli et al [15] states that individuals who are diabetic and contract COVID-19 are at greater risk of developing severe and critical forms of the virus, as COVID-19 is usually associated with poor glycaemic control causing a higher risk of developing ketoacidosis. Gupta et al [16] further elaborates that poor glycaemic control can impair parts of the innate and adaptive immune response when the body is fighting the virus, there is also a possibility of an increase in viral replication with individuals who are diabetic, as the virus can enter the cell due to an increase in furin, which is a type 1 membrane bound protease. D'Marco et al [17] reiterates that COVID-19 is a major threat to individuals with diabetes and renal impairment such as chronic kidney disease (CKD) or diabetic kidney disease (DKD), as the kidneys are highly vulnerable to damage and are susceptible to ACE2 expression.

Moreover, Yeoh et al [18] explains that an individual who is in an immunocompromised state, such as cancer, transplanted organs, patients taking immunosuppressive agents or have an autoimmune disease are more susceptible to become more severe when contracting COVID-19, as immunocompromised patients are more vulnerable to respiratory infections. Sahu et al [19] relates the low T Cell production from immunocompromised patients to the severity of their symptoms when contracting COVID-19, however, further investigation in this subject is needed to help differentiate the different types of cancer which are more at risk.

5. Current Treatment Guidelines

According to the Centers for Disease Control and Prevention (CDC) [20] all patients who are admitted to hospital are tested for COVID-19 via swab through nasopharynx and at the back of the oropharynx, as there is a stronger chance at detecting RNA shedding. National Health Service (NHS) England [21] state that all tests will be assumed to be positive until confirmed, once an individual test positive, they will remain in isolation under observation and staff will notify the coordination centre for contact tracing. It can take the lab approximately 24 hours to interpret the results of the test. By the end of May 2020, the UK exceeded its target in testing over 200,000 people a day.

According to PHE [5] healthcare workers must follow standard infection control precautions which involves the use of personal protective equipment (PPE) including respirators, FFP3 masks, eye protection/visors, coveralls, aprons which are all disposal and use of hand hygiene techniques before and after every patient. Ortega et al [22] further reiterates that if PPE is properly used, it is highly effective in protecting the person using it and anyone they encounter. However, in April 2020 there was a PPE shortage in certain parts of the country which caused more risk to the healthcare workers and the patients. As according to Artenstein [23] healthcare workers are encountering severe constraints that involve this pandemic and its crucial there is no stone left unturned when dealing with COVID-19 as safety is paramount.

NHS England [24] states that during this pandemic, there has been an increase in patients needing supplemental oxygen therapy, via assisted ventilation and advanced respiratory support, such as Continuous Positive airway Pressure (CPAP) or Extracorporeal Membrane Oxygenation (ECMO). Furthermore, during the COVID-19 outbreak. NHS England [24] has

aimed to maintain their oxygen prescribing targets ranging from 94%-98%. Fan et al [25] states that when tackling ARDS, personalised lung-protective ventilation reduces mortality by using various ventilatory strategies depending on the patient as one size does not fit all. National Institute for Health (NIH) [26] states that all patients with COVID-19 receiving supplemental oxygen via nasal cannula/mask recommends close observation, whereas patients who are not maintaining oxygen saturation stabilisation due to acute hypoxemic respiratory failure needs to be intubated then mechanically ventilated. Furthermore, patients who are intubated and ventilated and are not stabilising have had their body position changed from supine position to awake prone position, as it helps the fluid drain from the back of the lungs and can increase oxygen saturation, however, NIH [26] recommends not using the change in position to avoid intubation and mechanical ventilation in patients, and to only use it as a last resort if intubation and mechanical ventilation is ineffective on its own.

According to Boretti and Banik [27] it has taken time to find the right treatments to tackle COVID-19, such as antivirals and vaccines, which has caused the healthcare system to look deeper into alternative treatments. Furthermore, Boretti and Banik [27] explains that Intravenous Vitamin C (IV Vit-C) is used as an alternative treatment in tackling COVID-19 as it helps manage cytokine storms in patients with ARDS. Furthermore, Boretti and Banik [27] explain that IV Vit-C allows the immune system to become stronger, causes a reduction in the number of cytokines which are produced in innate immunity and inhibits the oxidative processes. Additionally, Hemila and Chalker [28] stated in a meta-analysis of 8 trials within the United States of America, Intravenous Vitamin C made the duration less in patients who required long term ventilation, as it has been highlighted that there is a dramatic decline in vitamin C in patients who are COVID-19 Positive and critically ill.

6. Emerging Treatments

Since the outbreak, the UK along with the rest of the world has been working tirelessly in finding various treatment strategies to help tackle the ongoing pandemic. Grein et al [29] states that Remdesvir (antiviral drug) works by inhibiting viral RNA polymerases, which blocks further synthesis of RNA, this affects the replication process of the virus, thus stopping the virus from reproducing. Furthermore, Grein et al [29] states that Remdesvir has broad spectrum activity against members of other virus families. Nosalski et al [13] states that Remdesvir is an antiviral that is part of the cardiovascular consideration therapies. However, Dixon et al [30] states that in clinical trials, the drug has been well tolerated, but self-limiting hepatotoxicity and nephrotoxicity is still possible. NIH [25] states that Remdesvir is used with patients who are COVID-19 positive and require supplemental oxygen, but are not mechanically ventilated or on ECMO, these recommendations are currently still being revised.

Dexamethasone is another emerging treatment that is being revised in the UK. According to the Chemocare Drug bank [31] Dexamethasone is a corticosteroid anti-inflammatory drug that helps relieve inflammation in various parts of the body and works well in treating lung conditions such as pneumonia. According to Lane and Fauci [32] in an open label trial where patients who had contracted COVID-19 were receiving oral and intravenous Dexamethasone, which resulted in lower 28-day mortality, compared to the individuals who were under invasive mechanically ventilation with oxygen supplementation. Additionally, Soy et al [33] states that antiviral treatment in some patients who experience severe symptoms is not enough and should be combined with another type of anti-inflammatory treatment to help reduce the

effects of cytokine storms and mortality. NIH [25] recommends that Dexamethasone is used in patients who are mechanically ventilated and patients who are not ventilated, but still require supplemental oxygen.

According to Jalkanen et al [34] another drug which is showing promising results is inhaled interferon beta therapy via nebuliser, as interferon beta is an antiviral protein. It is currently seen as one of the potential leading therapeutics to tackle COVID-19. Balfour [35] explains that COVID-19 is able to evade the bodies initial immunity response, whereas interferon beta counteracts COVID-19 by restoring the lungs ability to neutralise the virus, furthermore, the results of the clinical trial concluded that in a period of 16 days, ventilation treatment or caused death was reduced by 79% and it increased the chances of hospital discharge to patients who were admitted in hospital showing signs of severe symptoms. University Hospital Southampton [36] explains that they will be taking the findings forward from the pilot trial to accelerate their discovery to help tackle COVID-19.

7. Future Treatment Strategies

Bushra and Aslam [37] state that Ibuprofen was introduced in 1969, and it has been rated as the safest conventional non-steroidal anti-inflammatory drug in the UK. However, Mazaleuskaya et al [38] explains that there are risks involved with the use of Ibuprofen, as there is with other NSAIDs, such as serious gastrointestinal and cardiovascular adverse events. Rinott et al [39] explains that on March 14th 2020, the French Minister of Health published a recommendation to avoid the use of NSAIDs against COVID-19 as it can aggravate the infection. Furthermore, Rinott et al [39] states the hypothesis behind the French Minister's recommendation is due to COVID-19 having a high affinity for ACE2 receptors, which are in the lungs, and that ACE2 production along with ACE2 expression could be increased via the use of Ibuprofen. However, Moore et al [40] reiterated that there is no existence of any scientific data which supports the hypothesis that Ibuprofen increases the risk or worsens the novel infection, known as COVID-19. According to WHO [41] NSAIDs are a diverse group of medications with various risk profiles for different individuals and their conditions, and there is no direct evidence of any difference with adverse events, long term survival or quality of life regarding the use of NSAIDs for COVID-19. Torjesen [42] states that in April, UK Medicines Agencies have stated Ibuprofen can be used to treat patients with symptoms of COVID-19, such as fever or headache, following a review by the Commission on Human Medicines. NIH [43] states the Kings College London is currently carrying out a clinical trial on the use of lipid Ibuprofen in patients who are COVID-19 positive.

According to NIH [44] in March 2016, an open label dose ranging study of the Moderna Vaccine (mRNA-1273) had begun in 120 healthy adults. Drug Bank [45] states that mRNA-1273 is a novel mRNA-based vaccine which has a full-length pre-fusion stabilized spike (S) protein. NIH [46] further explains that the vaccine is designed to produce counteracting antibodies, which are aimed at the spike protein of the coronavirus. This is the part of the virus that can bind onto and enter human cells. Gallagher [47] announced in May 2020, positive interim phase 1 clinical data relating to the RNA Vaccine mRNA-1273 against COVID-19. Jackson et al [48] explains that on completion of interim phase one of the trial, the vaccine was immunogenic, as it induced strong binding antibody responses. Furthermore, Jackson et al [48] reiterates that protection from COVID-19 infection has not yet been determined through mRNA-1273, however, the current findings support advancement of the vaccine to further stage clinical trials. According to the University of Oxford [49] another vaccine that is showing promising results is the AZD1222 vaccine, as it has proven to be safe and

generate an immune response in the early stages of clinical trials. According to the National Herald India [50] the serum Institute in India are in the process of applying for the licensure trials of the oxford vaccine, where they will begin with further trials and start manufacturing the vaccine in large volumes.

8. Discussion

At the beginning of the pandemic, not much was known about COVID-19 and as a result, countries were on the backfoot whilst trying to maintain control of the global threat. Over the past few months, there has been a lot of research done where key elements have been discovered through various forms of research. More is known now about COVID-19 and which individuals are more at threat when contracting the novel virus, such as geriatrics, ethnic minorities and other vulnerable adults/children with comorbidities and pre-existing medical conditions, which the virus can worsen through the infection process. At the start of the pandemic, treatment options were limited, but after various clinical trials, there are now certain drugs that are showing promising results such as Remdesivir and Dexamethasone which can help manage symptoms and reduce the effects of the infection. However, like all drugs that are prescribed by healthcare workers in the healthcare setting, certain drugs must be given at certain times to be fully effective and lessen the risk of affecting individuals with comorbidities which the virus can have a devastating impact on. The drugs mentioned above are specifically used to manage symptoms, whereas future treatments such as the mRNA 1273 vaccine and the Oxford vaccine is hoping to be capitalised as a long term treatment, or possibly a prevention for the world to be able to further control and maybe even eradicate COVID-19, like other vaccines have done with other illnesses previously. There are also many other trials being conducted on other types of vaccines and drugs, which is happening all around the world to help fight this pandemic. It is vital that research carries on improving knowledge within the subject of COVID-19 so that healthcare workers can best utilise on updated research in order to deliver the best possible care to the patients.

9. Conclusion

These are unprecedented times where at the start of the pandemic, not much was known about COVID-19 and what effect it has on the body within a cellular level. As time has gone on, more research has been conducted which has helped the health sector better understand how the virus works and the extent of damage it can cause to an individual, especially those with comorbidities. Through crucial research, there have been new discoveries with various drugs which can be further utilised in the fight against COVID-19 by increasing recovery rates and improving hospital discharge. There are also vaccines that are showing promise which could turn the tide by helping increase an individual's immunity against this novel virus.

REFERENCES

1. European Centre for Disease Prevention and Control. Event Background COVID-19, 2020. [Online]. Available: <https://www.ecdc.europa.eu/en/novel-coronavirus/event-background-2019>
2. Department of Health and Social Care Coronavirus Cases in the UK: Daily Updated Statistics, 2020.
3. He F, Deng Y, Li W. Coronavirus disease 2019: What we know? J Med Virol. 2020;92(7):719-725.

4. Jin Y, Yang H, Wu J, et al. Virology, epidemiology, pathogenesis, and control of COVID-19. *Viruses*. 2020;12(4):372.
5. Public Health England. COVID-19: Epidemiology, Virology and Clinical Features, 2020.
6. European Society of Cardiology. ESC Guidance for the Diagnosis and Management of CV Disease During the COVID-19 Pandemic, ESC. 2020.
7. Yuki K, Fujiogi M, Koutsogiannaki S. COVID-19 pathophysiology: A review. *Clin Immunol*. 2020;20:108427.
8. Kaur G, Lungarella G, Rahman I. SARS-CoV-2 COVID-19 Susceptibility and Lung Inflammatory Storm by Smoking and vaping. *J Inflamm*. 2020;17(21):doi:10.1186/s12950-020-00250-8.
9. Mo X, Jian W, Su Z, et al. Abnormal pulmonary function in COVID-19 patients at time of hospital discharge. *Eur Respir J*. 2020;55(6):2001217.
10. George MP, Wells U, Jenkins RG. Pulmonary fibrosis and COVID-19: The potential role for antifibrotic therapy. *Lancet Respir Med*. 2020;doi:10.1016/S2213-2600(20)30225-3.
11. Rao SV, Arakeri G, Subash A. Rao, et al. COVID-19: Loss of bridging between innate and adaptive immunity? *Med Hypothesis*. 2020;144:109861.
12. Colafrancesco S, Scrivo R, Barbati C. Targeting the Immune System for pulmonary Inflammation and Cardiovascular Complications in COVID-19 Patients. *Front Immunol*. 2020; doi:10.3389/fimmu.2020.01439.
13. Nosalski R, Murray CE, Guzik B, et al. COVID-19 and the cardiovascular system: Implications for risk assessment, diagnosis and treatment options. *Cardiovasc Res*. 2020; 116(10):1666-1687.
14. Zeng YY, Ma YT, Xie X. COVID-19 and the cardiovascular system. *Nat Rev Cardiol*. 2020;17(5):259-260.
15. Orioli L, Hermans PM, Thissen PJ, et al. COVID-19 in diabetic patients: Related risks and specifics of management. *Annales d'endocrinologie*. 2020;81:2-3.
16. Gupta, R, Hussain, A, Misra, A. Diabetes and COVID-19: Evidence, current status and unanswered research questions. *Eur J Clin Nutr*. 2020;74(6):864-870.
17. D'Marco L, Puchades J, Romero-Parra V, et al. Diabetic kidney disease and COVID-19: The crash of two pandemics. *Front Med*. 2020;7:199.
18. Yeoh BC, Lee J, Rieth K, et al. COVID-19 in the cancer patient. *Anesth Analg*. 2020; doi:10.1213/ANE.0000000000004884
19. Sahu K, Jindal K, Siddiqui V, D, et al. Facing COVID-19 in the haematopoietic cell transplant setting: A new challenge for transplantation physicians. *Blood Cells Mol Dis*. 2020;83:102439.
20. Centers for Disease Control and Prevention. Coronavirus Disease 2019: Clinical Case Guidance, 2020.
21. National Health Service England. Guidance and Standard Operating Procedure: COVID-19 Virus Testing in NHS Laboratories, 2020.
22. Ortega R, Gonzalez M, Nozari A, et al. Personal protective equipment and COVID-19. *N Engl J Med*. 2020;25:382(26):e105.
23. Artenstein WA. In pursuit of PPE. *N Engl J Med*. 2020;30:382(18):e46.
24. National Health Service England. Clinical Guide for the Optimal Use of Oxygen Therapy During the Coronavirus Pandemic, 2020.
25. Fan E, Beitler RJ, Brochard L. et al. (2020) COVID-19-associated acute respiratory distress syndrome: Is a different approach to management warranted? *Lancet Respir Med*. 2020;8(8):816-821.

26. National Institutes for Health. COVID-19 Treatment Guidelines: Oxygenation and ventilation, 2020.
27. Boretti A, Banik KB. Intravenous vitamin C for reduction of cytokine storm in acute respiratory distress syndrome. *Pharam Nut.* 2020;21:100190.
28. Hemila H, Chalker E. Vitamin C: A Possible Therapy for COVID-19. *Infect Chemother.* 2020;2(2):222–223.
29. Grein J, Ohmagari N, Shin D, et al. Compassionate use of Remdesivir for patients with severe COVID-19. *N Engl J Med.* 2020;382(24):2327-36.
30. Dixon LD, Tassell V, Vecchie WB. et al. Cardiovascular considerations in treating patients with coronavirus disease 2019 (COVID-19). *J Cardiovasc Pharmacol.* 2020; doi:10.1097/FJC.0000000000000836.
31. Chemocare Drug bank (2020) Dexamethasone: What Dexamethasone is used for.
32. Horby P, Lim WS, Emberson JR, et al. Dexamethasone in hospitalized patients with COVID-19 – preliminary report. *N Engl J Med.* 2020; doi: 10.1056/NEJMoa2021436.
33. Soy M, Keser G, Atagunduz P, et al. Cytokine storm in COVID-19: Pathogenesis and Overview of anti-inflammatory agents used in treatment. *Clin Rheumatol.* 2020; 2020;39(7):2085-2094.
34. Jalkanen J, Hollmen M, Jalkanen S. Interferon Beta-1a for COVID-19: Critical importance of the administration route. *Critical Care.* 2020;24(1):1-3.
35. Balfour H. SNG001 diminished the risk of COVID-19 patients developing severe symptoms, reduced breathlessness and improved recovery rates. *Europ Pharm Rev.* 2020.
36. University Hospitals Southampton. Southampton Researchers Trial Inhaled Therapy for COVID-19, 2020.
37. Bushra R, Aslam N. An overview of clinical pharmacology of Ibuprofen. *Oman Med J.* 2010;25(3):155.
38. Mazaleuskaya LL, Theken NK, Gong L, et al. PharmGKB Summary: Ibuprofen pathways. *Pharmacogenet Genomics.* 2015;25(2):96–106.
39. Rinott E, Kozer E, Shapira Y, et al. Ibuprofen use and clinical outcomes in COVID-19. *Clin Microbiol Infect.* 2020; doi:10.1016/j.cmi.2020.06.003.
40. Moore N, Carleton B, Blin P, et al. Does Ibuprofen Worsen COVID-19? *Drug Saf.* 2020; doi:10.1007/s40264-020-00953-0.
41. World Health Organization. The Use of Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) in Patients with COVID-19. *Scientific Brief,* 2020.
42. Torjesen I. COVID-19: Ibuprofen can be used for symptoms, says UK agency, but reasons for change in advice are unclear. *British Med J.* 2020; doi:10.1136/bmj.m1555.
43. National Institutes for Health (2020) LIBERATE Trial in COVID-19 (LIBERATE).
44. National Institutes for Health. Safety and Immunogenicity Study of 2019-nCoV Vaccine (mRNA-1273) for Prophylaxis of SARs-CoV-2 Infection (COVID-19), 2020.
45. Drug Bank. mRNA-1273: Identification, 2020.
46. National Institutes for Health. Experimental COVID-19 Vaccine Safe, Generates Immune Response, 2020.
47. Gallagher MG. Moderna Shares Positive early Coronavirus Vaccine Data. *Contagion Live: Infectious Diseases Today,* 2020.
48. Jackson AL, Anderson J, Roupael E, et al. An mRNA vaccine against SARS-CoV-2 – preliminary report. *N Engl J Med.* 2020;NEJMoa2022483.
49. University of Oxford. Oxford COVID-19 Vaccine to Begin Phase 2/3 Human Trials, 2020.

50. National Herald India. Serum institute of India to Apply for Oxford Vaccine Trials in India in a week, 2020.

Citation: Dowling C. An update in the United Kingdom of the current, emerging and future treatment strategies regarding COVID-19 in the face of the global pandemic. Case Rep Rev Open Access. 2020;1(2):114.