Journal of Research in Medical and Dental Science
2022, Volume 10, Issue 10, Page No: 230-234

Available Online at: www.jrmds.in
eISSN No.2347-2367: pISSN No.2347-2545

COVID-19: Understanding of Its Pathophysiology, Clinical Presentation and Its Treatment

Swati Gupta, Sarika Dakhode*, Ashok Mehendale

Department of Community Medicine, Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences (Deemed to be University), Sawangi (Meghe), Maharashtra, India

ABSTRACT

Severe Acute Respiratory Syndrome (SARS) 2 coronavirus is a unique virus belonging to its own family, the Coronaviridae family, and is a series of new acute general pulmonary infection which started in Wuhan. It is the cause of the outbreak. Coronavirus disorder 19 or actually the disease caused by this virus, known as COVID-19, progresses at an astonishing pace throughout the sector, with pathophysiology, scientific presentations, and the latest control technology for COVID-19 on March 11, 2020. This section explains. Meanwhile, COVID-19 has evolved around the world, with increasing prevalence and death rates in all population groups. With the lack of accurate and efficient antibody checks, the prognosis is currently based primarily on real time RT PCR of oropharyngeal and nasopharyngeal swab samples. The medical scope of the disease the ability to spread to others nearby, with or without signs, or a mild flu like infection that is indistinguishable from mild upper respiratory tract contamination. I have an illness moderate and extreme cases require hospitalization in addition to comprehensive treatment including antipyretics, antivirals, antibiotics, steroids, as well as invasive airflow and non-invasive drugs. In complex cases, treatment with immunomodulators and plasma over-the-counter tools may also be required.

Although now no longer usually endorsed in viral pneumonia, a superior and powerful antibiotic routine enables save you or control sepsis and secondary bacterial infections. Macrolides including azithromycin are pretty powerful in stopping lung infections in sufferers with viral associated lung infection, similarly to having a giant anti-inflammatory impact in the bronchial tree.

Key words: Azithromycin, Gastrointestinal, Trans Membrane Serine Protease (TMPRSS2)

HOW TO CITE THIS ARTICLE: Swati Gupta, Sarika Dakhode, Ashok Mehendale, COVID-19: Understanding of Its Pathophysiology, Clinical Presentation and Its Treatment, J Res Med Dent Sci, 2022, 10 (10): 230-234.

Corresponding author: Dr. Sarika Dakhode
E-mail: sarikac31@gmail.com
Received: 02-Aug-2022, Manuscript No. JRMDS-22-63313;
Editor assigned: 04-Aug-2022, PreQC No. JRMDS-22-63313 (PQ);
Reviewed: 18-Aug-2022, QC No. JRMDS-22-63313;
Revised: 06-Oct-2022, Manuscript No. JRMDS-22-63313 (R);
Published: 18-Oct-2022

INTRODUCTION

Novel Coronavirus Disease 2019 (COVID-19) is caused by Severe Acute Respiratory syndrome Coronavirus 2 (SARS-CoV-2) severe virus has caused the worldwide spread of was caused by multi-organ damage. Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), originally recognized in the People's Republic of China, caused COVID-19. SARS-CoV-2 infection can be without any symptom or cause a huge range of signs, including increased airway contamination and mild signs of severe sepsis. COVID-19 first became known in December 2019, but a population of patients with unexplained inferior lung infection was identified in Wuhan. By July 10, 2020, the virus had infected in more than 199 countries and recognised more than ten million cases with a mortality rate of 607,000, according to various government data. This assessment summarizes the latest evidence of related to pathophysiology, transmission pathways, analysis or control of coronavirus disease [1]. Coronavirus life cycle and infiltration of its host cells. The virus spreads from men or women to by inhalation of aerosols and droplets. When the virus enters the body, it attaches to the host receptor and enters the host cell *via* endocytosis or membrane fusion. This virus is composed of four kinds of proteins that make up its structure, including Envelope (E), Spike (S), New creocapsid (N), Membrane (M), and protein is called [2] S-proteins are visible when they emerge from the viral floor and are the most important proteins for binding and penetrating the host membrane. It attaches to human cell by a protein made up of 2 subunits named S_1 and S_2.

LITERATURE REVIEW

Pathophysiology

This virus is a single-stranded RNA virus discovered surrounded by capsular virus and large virus. With humans and various mammals including birds, chickens, dogs, cats, cows and pigs. This virus causes neurological, respiratory and gastrointestinal problems.

Host Defence against SARS-CoV-2 at the onset of contamination, COVID-19 target cells, including lung cells or bronchial and nasal epithelial cells, bind to Angiotensin Converting Enzyme 2 receptor (ACE-2) [3]. Uptake of virus by cleaving the ACE-2 receptor and activating the viral S protein in the host cell with type 2 Trans Membrane Serine Protease (TMPRSS2) which is present in the host cell may also allow the COVID-19 virus to invade the host cell to increase [4]. Alveolar epithelial type two cells are the main target cells of the host, and these two enzymes are present. Like many other respiratory infections, including the influenza virus, people infected with COVID-19 can develop severe lymphocytopenia. On the other hand, SARS-CoV-2 infects and kills T lymphocytes. In addition, lymphocyte apoptosis is increased and lymphocyte formation is affected by adaptive and spontaneous immune responses, including cell-mediated and humoral immunity, respectively. Some authors believe that ACE inhibitors and angiotensin-receptor blockers increase susceptibility to SARS-CoV-2 contamination, and a large observation cohort is not associated with these agents and is associated with COVID-19 contamination or clinical death. The risk has been established. For example, previous treatment with angiotensin receptor blockers and ACE inhibitors was no longer associated with death rates. In addition to epithelial cells, pulmonary capillary endothelial cells are infected with SARS-CoV-2 infection. This increases the inflammatory response and causes the influx of neutrophils and monocytes.

Post mortem reports have a give us a look that diffuse thickening of the walls of alveoli by macrophages and monocytes infiltrated the airspace and inflamed endothelial cells. Acute Respiratory Distress Syndrome (ARDS) occurs when the alveolar region is filled with fluid due to pulmonary edema and vitreous formation occurs in different segments. Disordered, dysfunctional alveolar capillary oxygen transfer is a function of COVID-19. In extreme COVID-19, severe initiation of coagulation pathway and intake of coagulation elements arise [5]. A record from earlier study shows us that 72% of 184 those who succumbs of COVID-19 met standards for inability of blood to clot and stop bleeding. Damaged lung tissues may also bring about small thrombus formation and make contributions to the excessive prevalence of thrombotic headaches, consisting of pulmonary embolism, and thrombotic arterial headaches, deep venous thrombosis. (Example, ischemic stroke, myocardial infarction, limb ischemia) in seriously sick sufferers [6]. The improvement of viral sepsis, described as life threatening organ disorder due to a dysregulated host reaction to contamination, may also in addition make a contribution to multi organ failure.

Clinical features of COVID-19

The common incubation duration of COVID-19 is 14 days. The maximum not unusual place scientific signs and symptoms of COVID-19 and SARS are dry cough, fever, and fatigue. The common age of inflamed sufferers in one of a kind research ranged from forty five to fifty six years. At around 87% and more than 92% of COVID-19 and SARS sufferers, respectively, have odd chest x ray. In addition, 6.1-32.0% of patients with COVID-19 wanted mechanical airflow. These statistics are broad because the proprietary health centre protocol is used throughout the study [7].

The universal death accusation is currently pending, as many of the affected people are currently receiving remedy and post admission care. The predicted death rate within the early range of the outbreak varied to 12-16% in many countries, but it is no longer a common price tag as the most severely ill patients with excessive signs and symptoms were screened for a period of time. In addition, too many asymptomatic patients limited the ability to accurately measure these variables. The most non-abnormal severity of COVID-19 is respiratory distress, which targeted 3.5% of inflamed patients and 12-16% of extremely affected patients (15.7-17.1%). Lymphocytopenia is not uncommon in patients with very serious illnesses and in patients with mild signs. Computed tomography of the chest shows consolidation, opacity of frosted glass on both sides, and mottled shadows near or on both sides. Signs and symptoms of 4,444 GI are not uncommon in patients with COVID-19, and meta-assessment confirms that these signs occur in 18.7% of inflamed patients and are more severe in excessive patients (excessive patients). Similarly, approximately 26% of SARS and MERS patients have gastrointestinal symptoms [8].

Treatment

Mild cases (SpO_2 range 94%-97% in room air: High flow oxygen treatment with Simple face mask/venturi mask/nasal cannula/non-rebreathing mask accident with COVID-19 for management of patients with breathlessness The room should be equipped with a functional oxygen system, pulse oximeter, disposable oxygen converter, along with a nasal cannula, easy face mask, venturi mask, Non-Rebreathing (NRB) mask, mask with reservoir bag. They are involved in the disaster and are performed according to the severity of the presentation. An oxygen mask can be used to oxygenate patients with mild dyspnea and SpO_2 levels of 94% to 97%. Patients with SpO_2 30 min or chronic respiratory distress should be given oxygen *via* a 40% venturi mask for a more stable supply of oxygen. The reassessment, if solid, should be completed again after 10 minutes and 6 hours. If 6 venturi masks develop little after 6 hours, Non-Invasive airflow (NIV) should be considered. It offers safer options for additional oxygen supply [9].

Moderate cases (90%-94% SpO_2 stage in room air Mild illness (90%-94% SpO_2 in room air: Patients are removed to prevent the virus transmission included. The designated medical record will be carried along with the existing comorbidity record. Key symptoms and symptoms and oxygen saturation (SpO_2 region) should be tracked in relation to examinations such as x-rays, EKGs, and tests throughout the chest. Use when SpO_2 cannot always be maintained above 92%. Or, there is no

onset of dyspnoea through the face mask from fashionable oxygen means. The oxygen content of the drift charge of HFNO drugs is about 30-40 L/min and should always be adapted to the scientific reaction of the parties involved. It has also been observed to be useful for non-stop interruptions with significant airway pressure (CPAP) between cycles, and for critically ill patients who require the assistance of fibre optic tracheal intubation 52. It also has a high risk of aerosolization and should be used most effectively in lightly loaded rooms [10]. Patients with drift >50 L/min and FiO_2 >70% with no increase after 1 hour are advised to switch to NIV. Severe cases (SpO_2 stage in ambient air ≤ 90% or ARDS patients).

In patients having critical symptoms oxygen supplementation should be started immediately at 6000 ml/min and the dose should be reduced to the SpO_2 ≥ 90 target. Titrate% for non-pregnant females and SpO_2 ≥ 93–96% for pregnant females. High flow nasal oxygen is highly effective in decreasing the need of ventilation and endotracheal tube intubation that classical artificial lungs attached through nasal mask. Intratracheal intubation in case of increase carbon dioxide level in blood due to pulmonary edema, multi-organ failure, hemodynamic instability, normal intellectual reputation or less than 90% decrease in oxygen saturation only invasive airflow due to intubation should be considered [11].

Endotracheal tube Intubation of endotracheal tube endotracheal tube is usually performed by a specialist after all private shielding systems including full size gowns, N95 masks and goggles are fitted. The CPAP method provides 5 minutes of pre-oxygenation with 100% oxygen and rapid extraction intubation is recommended if possible. Mechanical airflow begins with a decrease in tidal volume (5-9 ml/kg frame weight) and a decrease in inspiratory pressure (plateau expansion <29 cm H_2O). Vulnerable airflow of 16-18 hours depending on the day is recommended for patients with excessive ARDS, but sufficient personnel and expertise are required to handle it safely. In sufferers with slight or extreme ARDS, better Positive End Expiratory Pressure (PEEP) is usually recommended which has the advantages of reducing trauma because of Lung collapse and improved Conscription of alveoli, however can reason headaches because of lung over distension and growth with inside the resistance due to blood vessels of lungs.

Extracorporeal Membrane Oxygenation (ECMO) for sufferers with non-responding lack of oxygen in blood notwithstanding intra tracheal tube intubation and air flow have to be taken into consideration if feasible. In COVID-19 sufferers, ECMO may also constitute a green guide in case of cardiogenic/septic shock and refractory hypoxemia unresponsive to maximal remedy.

Other therapies for COVID-19

Antibiotics: Although now no longer usually endorsed in viral pneumonia, a superior and powerful antibiotic routine enables save you or control sepsis and secondary bacterial infections. Macrolides including azithromycin are pretty powerful in stopping lung infections in sufferers with viral associated lung infection, similarly to having a giant anti-inflammatory impact in the bronchial tree.

Corticosteroids: Steroids may be used for a brief duration of time, that is, 4-6 days in sufferers who display revolutionary decreasing level of oxygen saturation in blood, expanded initiation of pro-inflammatory reaction and fast deterioration of functions on chest radio graphical imaging. Steroid turned into the primary and handiest steroid indicated first of all, at a dose now no longer exceeding 1-2 mg/kg/day for slight instances and 2-3 mg/kg/day for excessive instances. Excessive amounts of drug had been now no longer endorsed in view of the put off in viral clearance because of decreasing immunity due to steroids [12].

Currently, dexamethasone has additionally been observed to be powerful for reducing death rates in intense units and significantly sick instances.

Antiviral drugs: The following antiviral capsules were placed to apply for COVID-19 sufferers so far.

Remdesivir (COVIFOR\CIPREMI): Remdesivir has a taste for multiple RNA viruses (along with Ebola) *in vitro* and may help prevent and treat coronavirus infections, according to some preclinical studies was first shown. Remdesivir is a wide range of antiviral agents that function by blocking the movement of RNA polymerase in the viral RNA structure. This bypasses proofreading by viral exoribonucleases and significantly reduces viral RNA production 60. In a mouse version of SARS-CoV, it was determined that Remdesivir reduces viral load in the lungs and improves lung function. It is the leading case of COVID-19 contamination in the United States, with rapid development confirmed one day after ingestion of Remdesivir.

Favipiravir (FABIFLU): Favipiravir exhibits a hobby as opposed to the RNA virus by converting to ribofuranosyl triphosphate spin-off *via* the host. Selective inhibition of enzymes and then viral RNA based RNA polymerases. It was first discovered for the therapeutic use of cases of resistant influenza. The drug had also been shown to be effective in avian influenza treatments and may replace infections caused by pathogens such as COVID-19 and Ebola virus. Immunomodulators (hydroxychloroquine, tocilizumab, chloroquine).

Tocilizumab: Tocilizumab is IgG1. Which is monoclonal antibody that is humanized, directed against the IL-6 receptor, and is usually used to treat juvenile arthritis, rheumatoid arthritis, and large-scale mobile arthritis. This can be considered in patients with mild to moderate illness with elevated inflammatory markers (IL-6) and gradual increase in oxygen demand, and in patients who are routinely ventilated and do not respond to alleviation. The dose is 9 mg/kg (almost 900 mg at a single dose) and is slowly administered in Normal Saline (NS) over an hour. It can be given twice a day if needed.

Tocilizumab is contraindicated for neutropenia and active tuberculosis. Treatment with 4974 Tocilizumab potentially reduces the risk of invasive mechanical airflow or mortality in patients with excessive COVID-19 related pneumonia, with or without subcutaneous or intravenous administration plasma changes caused by convalescent plasma COVID-19 virus removed from broncho alveolar lavage fluid in severely ill individuals has been shown to be neutralized by a large number of convalescent plasma 85. Longer developmental shows, regardless of steroid use (oxygen demand increases steadily). Some key requirements for this mode include antibody titers in ABO compatibility, convalescent plasma and cross matching of donor plasma. Recipients should be carefully monitored for transfusion-related negative events for hours after transfusion and in patients with Ig allergies and IgA deficiency it should be avoided. A single dose level of 5 to 14 ml/kg, usually 300 ml, is given slowly over 3 hours. Recovery plasma should be discontinued within 2 weeks of recovery to ensure over justification due to excessively high antibody titers COVID-19.

DISCUSSION

Supplemental therapy: Prophylactic anticoagulant therapy with Low Molecular Weight Heparin (LMWH) (e.g. Enoxaparin 40 mg SC) is given to patients with mild to severe illness (depending on the time of day) (depending on the case). Should be given for anticoagulant therapy. Considering the excessive risk of thromboembolism. Comorbidities consisting of associated diabetes, hypertension, and hyperthyroidism should be monitored accordingly. Pregnant women with excessive illness should be consulted as needed by neonatology, obstetrics, and extensive care professionals regarding the recognition of COVID-19, it is necessary to guarantee psychological counselling for those affected by anxiety and tension.

Intimate contact transmission from men or women to individuals hastened to intensify the onset of the disease, making it even more difficult to integrate it into the community. Affected individuals may be completely asymptomatic with favourable smear control and also give gifts in case of mild flu-like infections or severe symptoms requiring hospitalization can do. Currently, there is no strong antibody check for fast analysis, but computed tomography chest scan had found to be very tricky or specific. Due to lack of a strong vaccine, this drug is specifically supported by steroids, HCQS, antibiotics, antivirals, and oxygen. Complex cases, as well as those who do not respond to conventional treatments, May also require plasma switching from immunomodulators and convalescent sera in convalescent patients. As many pharmaceutical companies have already begun research on humans, advances in viral sequencing and generation had definitely to make it easier for improved vaccines against COVID-19.

CONCLUSION

The COVID-19 pandemic is now a global fitness emergency. Infection through close contact between a man and a woman to an individual hastened to intensify the onset of the disease, making it even more difficult to integrate it into the community. Affected individuals may be completely asymptomatic with favourable smear control and also give gifts in case of mild flu-like infections or severe symptoms requiring hospitalization can do. Currently, there is no strong antibody check for rapid analysis, but HRCT scans of the chest have been found to be very tricky and specific. In the absence of a strong vaccine, this drug is specifically supported by oxygen scavengers, antivirals, steroids, HCQS, and antibiotics. Complex cases, as well as those who do not respond to conventional treatments, may also require plasma switching from immunomodulators and convalescent sera in convalescent patients. As many pharmaceutical companies have already begun research on humans, advances in viral sequencing and generation have certainly paved the way for improving vaccines against COVID-19.

REFERENCES

1. Parasher A. COVID-19: Current understanding of its pathophysiology, clinical presentation and treatment. Postgraduate Med J 2021; 97:312-320.
2. Ksiazek TG, Erdman D, Goldsmith CS, et al. A novel coronavirus associated with severe acute respiratory syndrome. New England J Med 2003; 348:1953-1966.
3. Cascella M, Rajnik M, Cuomo A, et al. Features, evaluation and treatment coronavirus (COVID-19). Stat Pearls. Treasure Island (FL): Stat Pearls Publishing, 2020.
4. Wiersinga WJ, Rhodes A, Cheng AC, et al. Pathophysiology, transmission, diagnosis, and treatment of coronavirus disease 2019 (COVID-19): a review. JAMA 2020; 324:782-793.
5. Yang N, Shen HM. Targeting the endocytic pathway and autophagy process as a novel therapeutic strategy in COVID-19. Int J Bio Sci 2020; 16:1724.
6. Hoffmann M, Kleine-Weber H, Schroeder S, et al. SARS-CoV-2 cell entry depends on ACE-2 and TMPRSS2 and is blocked by a clinically proven protease inhibitor. Cell 2020; 181:271-280.
7. Walls AC, Park YJ, Tortorici MA, et al. Structure, function, and antigenicity of the SARS-CoV-2 spike glycoprotein. Cell 2020; 181:281-292.
8. Wu CN, Xia LZ, Li KH, et al. High-flow nasal-oxygenation-assisted fibreoptic tracheal intubation in critically ill patients with COVID-19 pneumonia: a prospective randomised controlled trial. BJA: Br J Anaesthesia. 2020; 125:e166.
9. Bai Y, Yao L, Wei T, et al. Presumed asymptomatic carrier transmission of COVID-19. JAMA 2020; 323:1406-1407.

10. Scavone C, Brusco S, Bertini M, et al. Current pharmacological treatments for COVID-19: What's next? British J Pharmacol 2020; 177:4813-4824.
11. Li L, Zhang W, Hu Y, et al. Effect of convalescent plasma therapy on time to clinical improvement in patients with severe and life-threatening COVID-19: a randomized clinical trial. Jama 2020; 324:460-470.
12. Gautret P, Lagier JC, Parola P, et al. Hydroxychloroquine and azithromycin as a treatment of COVID-19: results of an open-label non-randomized clinical trial. Int J Antimicrobial Agents 2020; 56:105949.