International Journal for Multidisciplinary Research (IJFMR)
E-ISSN: 2582-2160 ● Website: www.ijfmr.com ● Email: editor@ijfmr.com

Clinical Characteristics Associated in COVID-19 With Mortality: A Retrospective Observational Study

Abhoy Roy[1], Rupam Debnath[2], Jhumpa Debnath[3], Anwesa Das[4], Himshikhar Sarma[5]

[1,2,3]Lecturer, Department of Pharmacognosy, Bir Bikram College of Pharmacy
[4,5]Assistant Professor, Department of Pharmacognosy, Bir Bikram College of Pharmacy

Abstract:
In late 2019, a pneumonia-like outbreak in Wuhan, China, led to the identification of SARS-CoV-2, the virus responsible for COVID-19. The disease rapidly spread worldwide, prompting the World Health Organization (WHO) to designate it as COVID-19. Following the initial cases in China, infections soared globally, with the first cases outside China reported in Thailand on January 11, 2020. By March, India recorded its first death linked to COVID-19, with the virus spreading to every state except Sikkim by mid-April. Our study assessed factors associated with COVID-19 mortality among 50 patients, finding higher death rates among males (62%) and those with comorbidities like hypertension (64%) and diabetes (54%). Older adults (ages 61–80+) were particularly vulnerable. Hypoxic shock with cardiac arrest was the primary cause of death (60%), and viral pneumonia was prevalent (74%).

Keywords: COVID-19, Coronavirus, Pneumonia, Survey.

Introduction:

In late 2019, a pneumonia-like outbreak emerged in Wuhan, China, leading to the identification of a new virus: severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). As the virus spread globally, the World Health Organization (WHO) officially termed the illness caused by this virus COVID-19. Not long afterward, cases were detected across all 50 states in the United States and in four of its overseas territories, underscoring the critical role of community transmission in the rapid expansion of the outbreak.[1], [2]
With symptoms that affect almost every organ system, COVID-19 poses a variety of treatment problems. More than half of hospitalised patients in China had fever, exhaustion, and dry cough, according to preliminary data. Muscle soreness, dyspnoea, and appetite loss were found to be common complaints in subsequent research. Taste and smell loss were distinct markers as the disease spread over the world. Nausea, vomiting, and diarrhoea were among the digestive problems that either manifested alone or in conjunction with respiratory symptoms [3]. Additionally, dermatological symptoms like hives and generalised skin abnormalities have been noted. This is made worse by the fact that cardiac problems, including as heart failure and myocarditis, have been identified as major consequences among patients, while neurological symptoms, such as stroke and altered consciousness, have been observed in extreme cases.[4], [5]

More asymptomatic COVID-19 instances have been found as a result of increasing diagnostic testing. An increasing corpus of research indicates that patients who are asymptomatic may have aberrant radiologic results on chest imaging, even if the incidence of these cases is unknown. [6]

The sickness spread quickly after the first report from China, and the number of cases skyrocketed. After the first case outside of mainland China was reported in Thailand on January 11, the sickness quickly spread to every continent except Antarctica. On January 30, 2020, India reported its first COVID-19 case. On March 12, 2020, the first COVID-19-related death in India was documented. The illness has spread to every Indian state but Sikkim by the second week of April.[7][8]

Initially, zoonotic transmission was considered a likely cause since most early cases were linked to wet markets. However, by late January 2020, an increasing number of cases emerged in people without any known exposure to these markets or contact with symptomatic individuals. The spread of the disease among people who hadn't visited Wuhan, as well as among healthcare workers, pointed toward person-to-person transmission. While the precise transmission route of the virus remains unknown, it is likely spread mainly by respiratory droplets, either directly or indirectly through contaminated surfaces. Currently, there is no evidence to support airborne transmission. Although the virus has been found in stool samples of both symptomatic and asymptomatic individuals, the possibility of faecal-oral transmission remains uncertain.[9]

The predicted number of secondary cases that could result from a single case in a susceptible population is known as the basic reproduction number (R_0). R_0, which shows the likelihood of an epidemic spreading, is the foundation of infectious disease epidemiology. The R_0 for SARS-CoV2 has been estimated by the majority of investigations to be between 2.0 and 3.0. The secondary attack rate is another factor that affects infectivity.[10]

Coronaviruses (CoVs), a group of single-stranded, positive-sense RNA (+ssRNA) viruses characterized by their spike glycoprotein-induced crown-like appearance under electron microscopy, are classified within the *Orthocoronavirinae* subfamily of the *Coronaviridae* family into four genera: Alphacoronavirus, Betacoronavirus, Deltacoronavirus, and Gammacoronavirus.[11], [12]

While COVID-19 can affect individuals of any age, those aged 65 and older are at increased risk, with particularly high vulnerability in individuals aged 85 and above [13]. The likelihood of severe COVID-19 complications is also elevated for residents of nursing homes and those with pre-existing health conditions, including moderate to severe asthma, chronic lung disease, hypertension, heart disease, diabetes, liver disease, and renal failure.[14], [15]

1. Pathophysiology of SARS-CoV-2 infection:

In early SARS-CoV-2 infection, the virus binds to ACE2 receptors on cells, with TMPRSS2 enabling viral entry. Target cells like alveolar type II cells express ACE2 and TMPRSS2. SARS-CoV-2 can cause lymphopenia by destroying T lymphocytes. Studies found no link between ACE inhibitor use and increased COVID-19 risk or mortality [16]. In advanced SARS-CoV-2 infection, rapid viral replication damages the epithelial-endothelial barrier, infecting both epithelial and pulmonary capillary endothelial cells. This intensifies inflammation, with neutrophil and monocyte influx, endothelialitis, and alveolar thickening. Ground-glass opacities on CT reflect edema, progressing to early ARDS, impaired oxygen exchange, and bradykinin-related angioedema. Severe COVID-19 can trigger intense coagulation, leading to microthrombi, deep vein thrombosis, pulmonary embolism, and arterial thrombosis. Inflammatory lung tissue and viral sepsis may further contribute to multiorgan failure.[17], [18]

2. Diagnosis of SARS-CoV-2 infection:

The primary diagnostic method for SARS-CoV-2 is a real-time PCR (RT-PCR) assay on a nasopharyngeal swab, with FDA-approved commercial PCR tests available for various sample types under EUAs. Antibody tests support COVID-19 surveillance, while additional lab tests, such as CBC and inflammatory markers, offer further clinical insights.[19]

3. Treatment of SARS-CoV-2 infection:

Current therapeutic options for COVID-19 include FDA-approved and investigational treatments such as immunomodulators (e.g., tocilizumab, baricitinib), antivirals (e.g., remdesivir, hydroxychloroquine, ivermectin), anti-SARS-CoV-2 monoclonal antibodies (e.g., REGN-COV2, bamlanivimab/etesevimab, sotrovimab), convalescent plasma, and anti-inflammatory drugs (e.g., dexamethasone).[20], [21], [22]

4. Prevention of SARS-CoV-2 infection:

In areas with widespread SARS-CoV-2 transmission, wearing a mask in public indoor spaces is essential. Avoid close contact with sick individuals and minimize touching your eyes, nose, and mouth [23]. Stay home if you feel unwell, and cover coughs or sneezes with a tissue, disposing of it immediately. Regularly clean frequently touched surfaces, and wash your hands frequently with soap and water to reduce the risk of infection.[24], [25]

Methodology:

Design:

The mortality data for COVID-19-related deaths from April 2020 to November 2020 from various wards of Ajara Hospital in Warangal, Telangana State, were used in this retrospective observational analysis. We saw a second wave of epidemics in India during this time.

Data sources and data:

The patient case profiles were used to carry out this study and all Microsoft Excel was used to assess the results.

Results:

In our study "**Clinical characteristics associated in COVID-19 with Mortality: A retrospective observational study**" of total of 50 patients in which 31 (62%) were males and 19 (38%) were females. (**Table:1, Fig:1**). The prevalence of death due to COVID-19 being higher in males than that of females because of high expression of Corona Receptors (ACE2) in Male and because of lifestyle, such as higher level of smoking and drinking among male as compared to female.

Table 1: Sex ratio of patients who died of COVID-19

Sex	**No.**
F	19
M	31

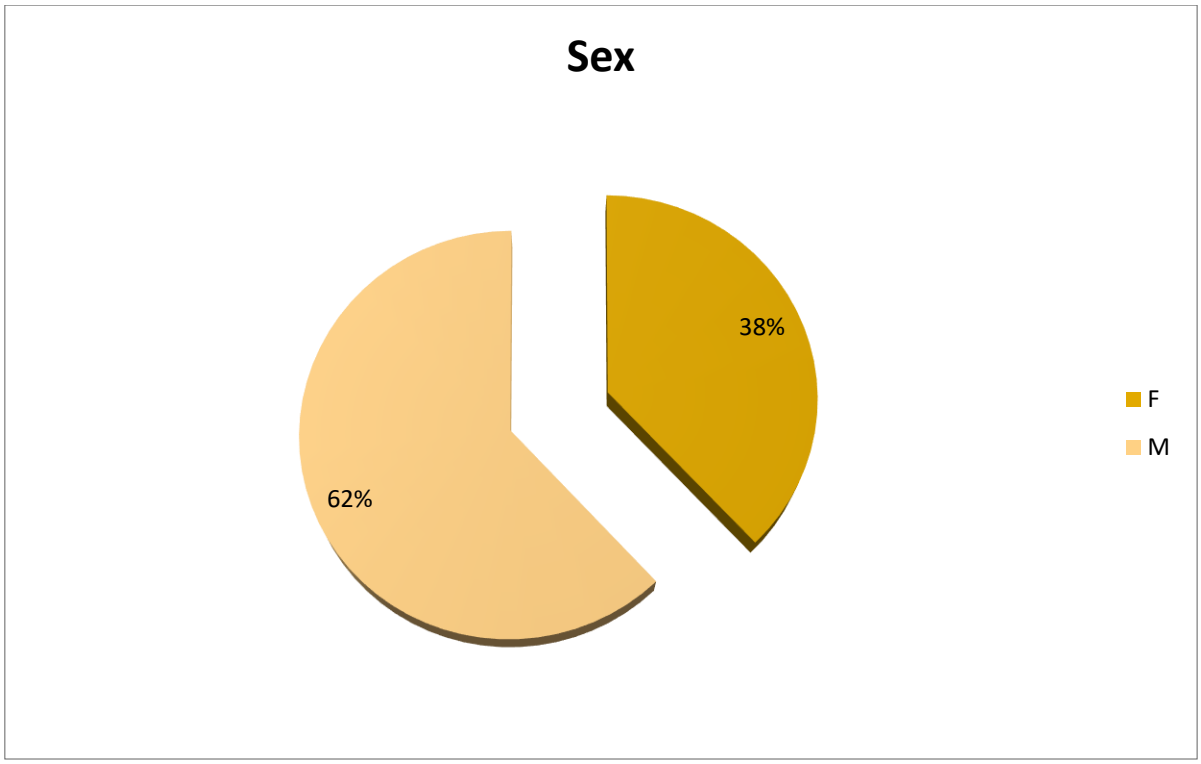

Figure 1: Ratio of male and female patients died due to COVID-19

Out of 50 deaths, 32% are of age 61-70, 26% are of age 51-60, 16% are of age 71-80, 16% are of age 41-50, 6% are of age 80+ and 2% are of age 31-40, 2% are of age 10-20 and no deaths recorded of age 21-30 (**Table:2, Fig:2**). According to our study people above 50 years are undergoing death more than below 50 years. This is because as age increases, the immune system weakens, which makes people more vulnerable to infections also when the immune system gears up in older people, there is also a higher likelihood of a phenomenon called a cytokine storm. This is where the immune system overreacts and produces too many chemicals to fight infection. So older people get severe inflammatory reactions that cause damage to the body which leads to organ failure and finally death.

Table 2: Ratio of age groups patients died due to COVID-19

Age Group	No. of Patients	Percentage
10-20	1	2
21-30	0	0
31-40	1	2
41-50	8	16
51-60	13	26
61-70	16	32
71-80	8	16
80+	3	6

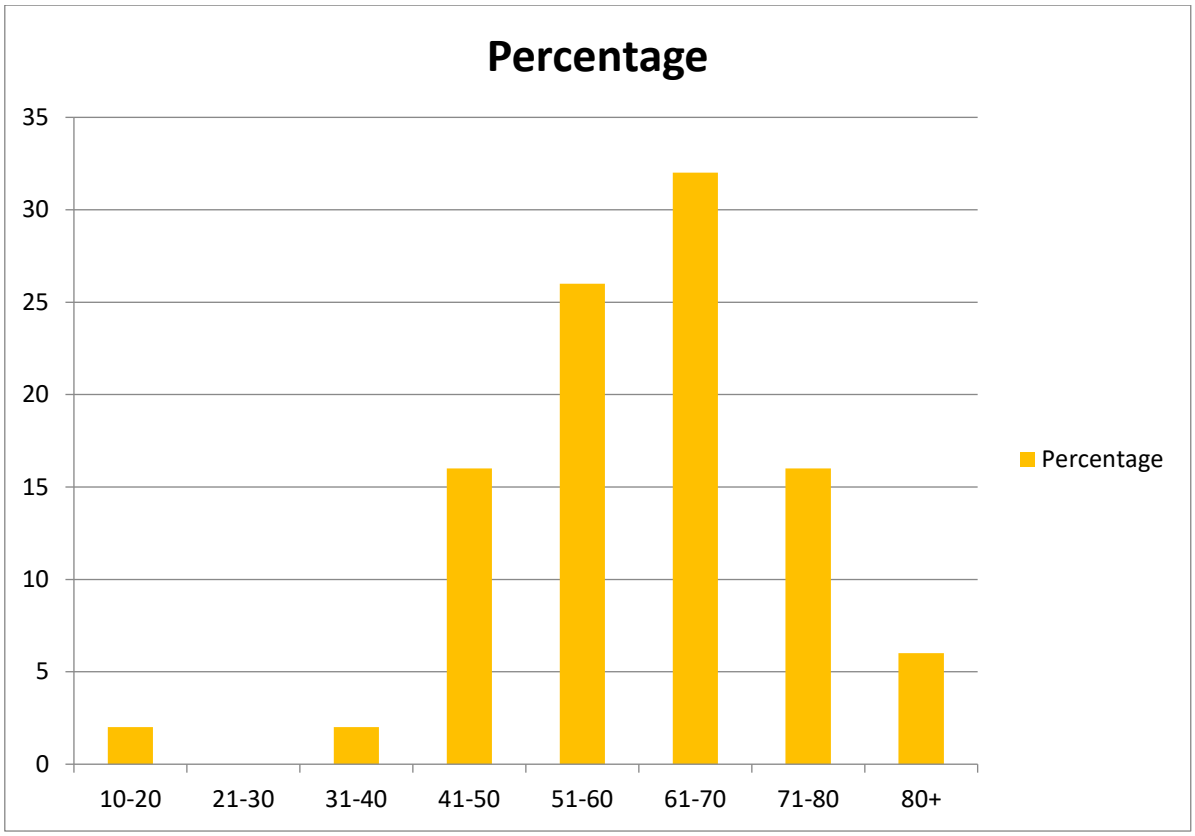

Figure 2: Age group of patients died due to COVID-19

Our study findings indicate that the most prevalent chief complaints of patients include Fever (78%), SOB (76%), and Cough (70%). (**Table:3, Fig:3**) These chief complaints were the most frequently found in COVID-19 patients.

Table 3: Percentage of patients with various chief complaints

Chief Complaints	No. of Patients	%
Fever	39	**78**
SOB	38	**76**
Cough	35	**70**
Cold	7	**14**
Myalgia	12	**24**
Weakness	10	**20**
Loss of Appetite	4	**8**
Others	20	**40**

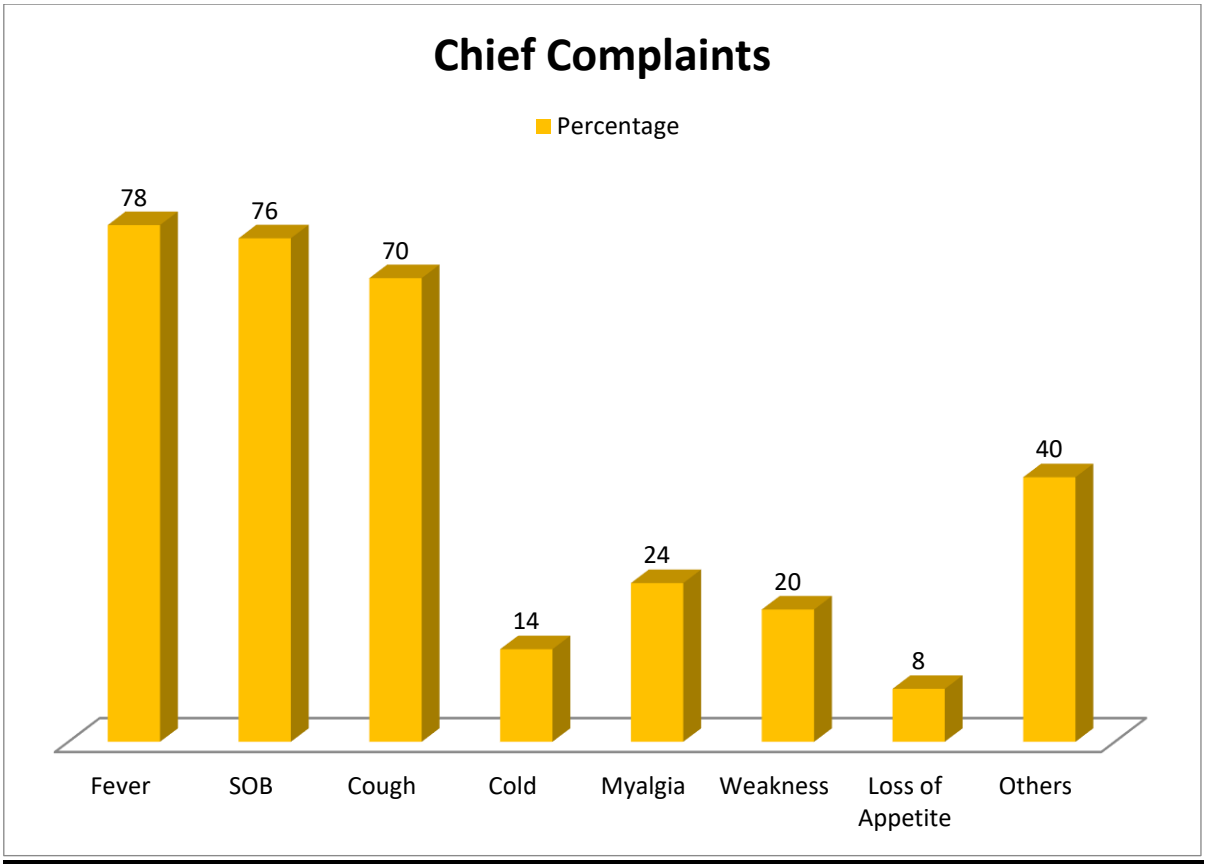

Figure 3: Percentage of patients having various chief complaints

According to our study patients with Hypertension (64%), Diabetes (54%) (**Table:4, Fig:4**) died more than healthy individuals. This is because the link between hypertension, diabetes, and SARS Cov-2 is Angiotensin Converting Enzyme 2 (ACE 2). ACE 2 is expressed in the lungs, intestine, kidney, blood vessels, and epithelial cells. Levels of ACE 2 are higher in diabetes and hypertension when compared to healthy individuals. This is due to the natural pathogen of the diseases, and treatment of these patients either ACE 1 inhibitor or Angiotensin receptor antagonists which further increases ACE 2 levels. These high levels of ACE 2 in diabetes and hypertension patients facilitate increased viral entry & replication leading to severe disease and finally death.

Table 4: Past medical history of patients

Medical History	No. of patients	Percentage
Diabetes	27	54
Hypertension	32	64
Others	10	20

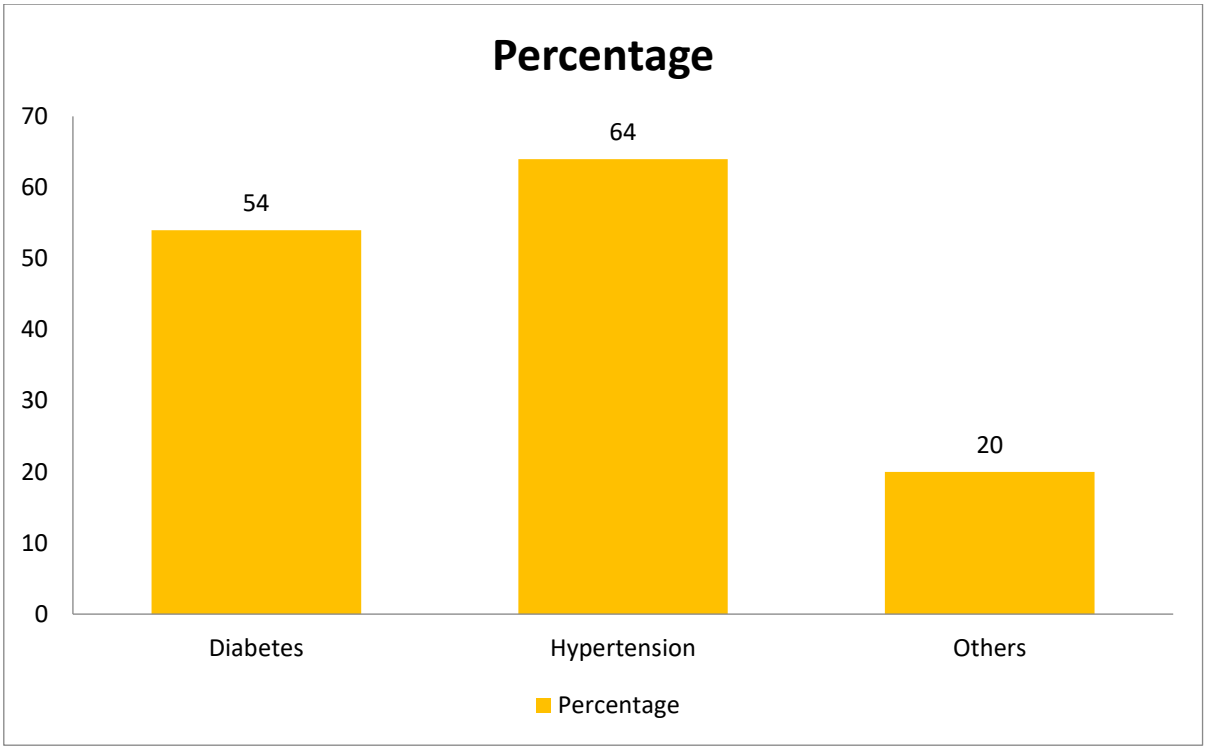

Figure 4: Past medical history of patient died due to COVID-19

According to our study, out of 50 deaths, vaccinated deaths are 6 (12%) and non-vaccinated deaths are 44(88%) (**Table:5, Fig:5**). In our study we found a low risk of COVID-19 deaths after vaccination because vaccine makes the body's immune system trigger and helps the body to fight against infections.

Table 5: Vaccination status

Vaccination Status	No. of Patients	Percentage
Vaccinated	6	12
Non vaccinated	44	88

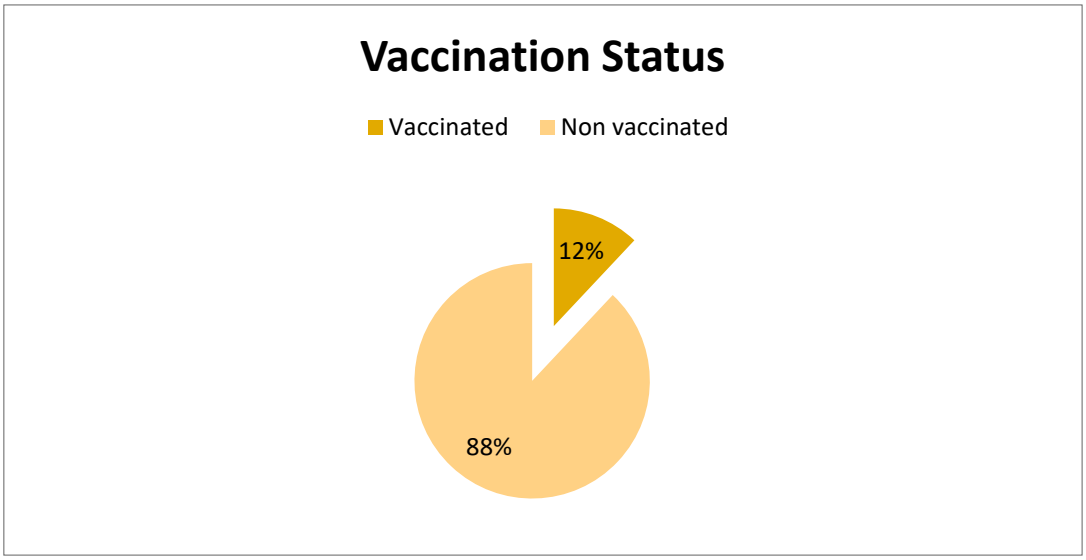

Figure 5: Vaccinated and non-vaccinated ratio

According to our study, we have observed 12 (24%) patient's CT severity scores of 11-20, 9(18%) patient's CT severity score is 5-10, and 1(2%) patient's Ct severity score is 21-25 (**Table:6, Fig:6**)

Table 6: CT Severity Score

CT severity score	No. of patients	Percentage
5-10	9	18
11-20	12	24
21-25	1	2
Unknown	28	56

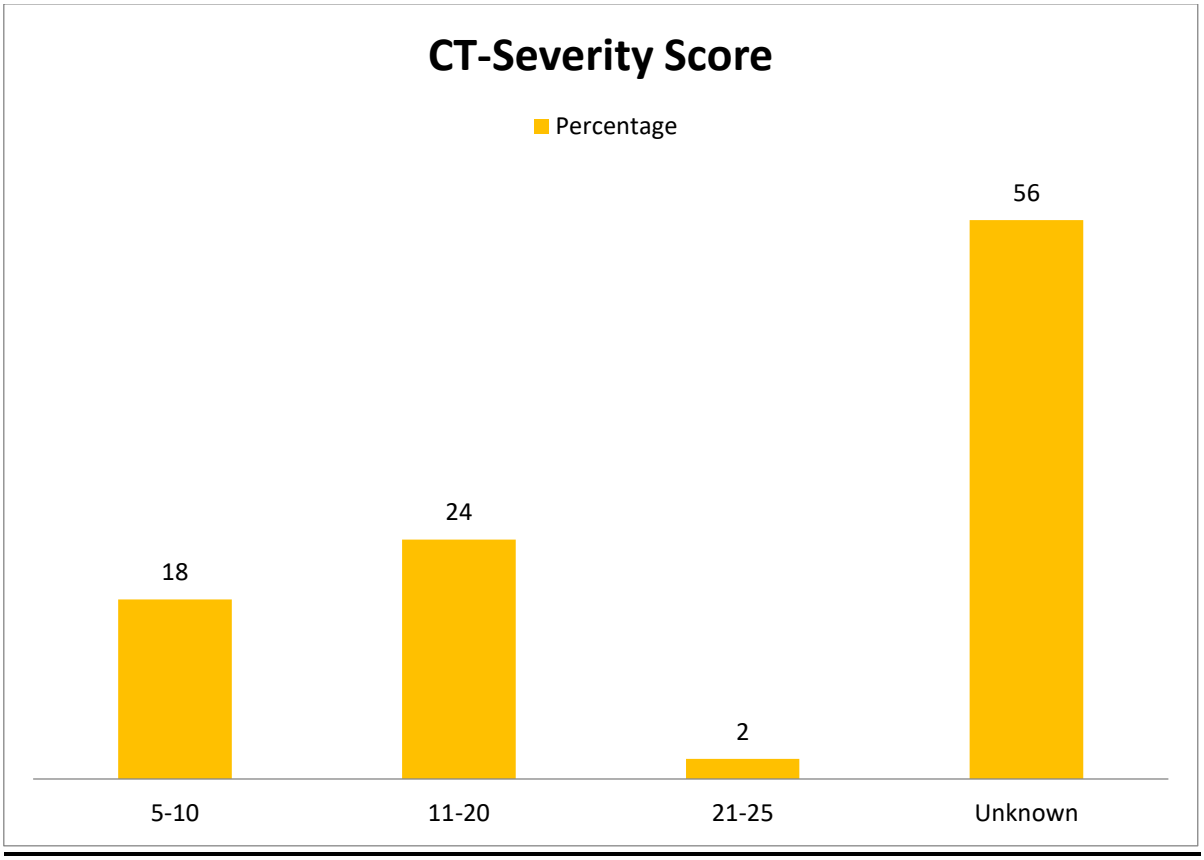

Figure 6: CT Severity Score

According to our study, out of 50 deaths, 30 (60%) are due to Hypoxic shock with cardiac arrest, 9 (18%) are due to Cardiopulmonary failure, 3 (6%) are due to Septic shock and 8 (16%) are due to other reasons (**Table:7, Fig:7**). We found that patients with chronic comorbidities particularly those with previous cardiovascular metabolic diseases like Hypoxic shock with cardiac arrest are more prone to death because of impaired immunity in them.

Table 7: Reason of death patients

Reason of death	No. of patients	Percentage
Hypoxic Shock with Cardiac arrest	30	60
Cardiopulmonary Failure	9	18
Septic shock	3	6
Others	8	16

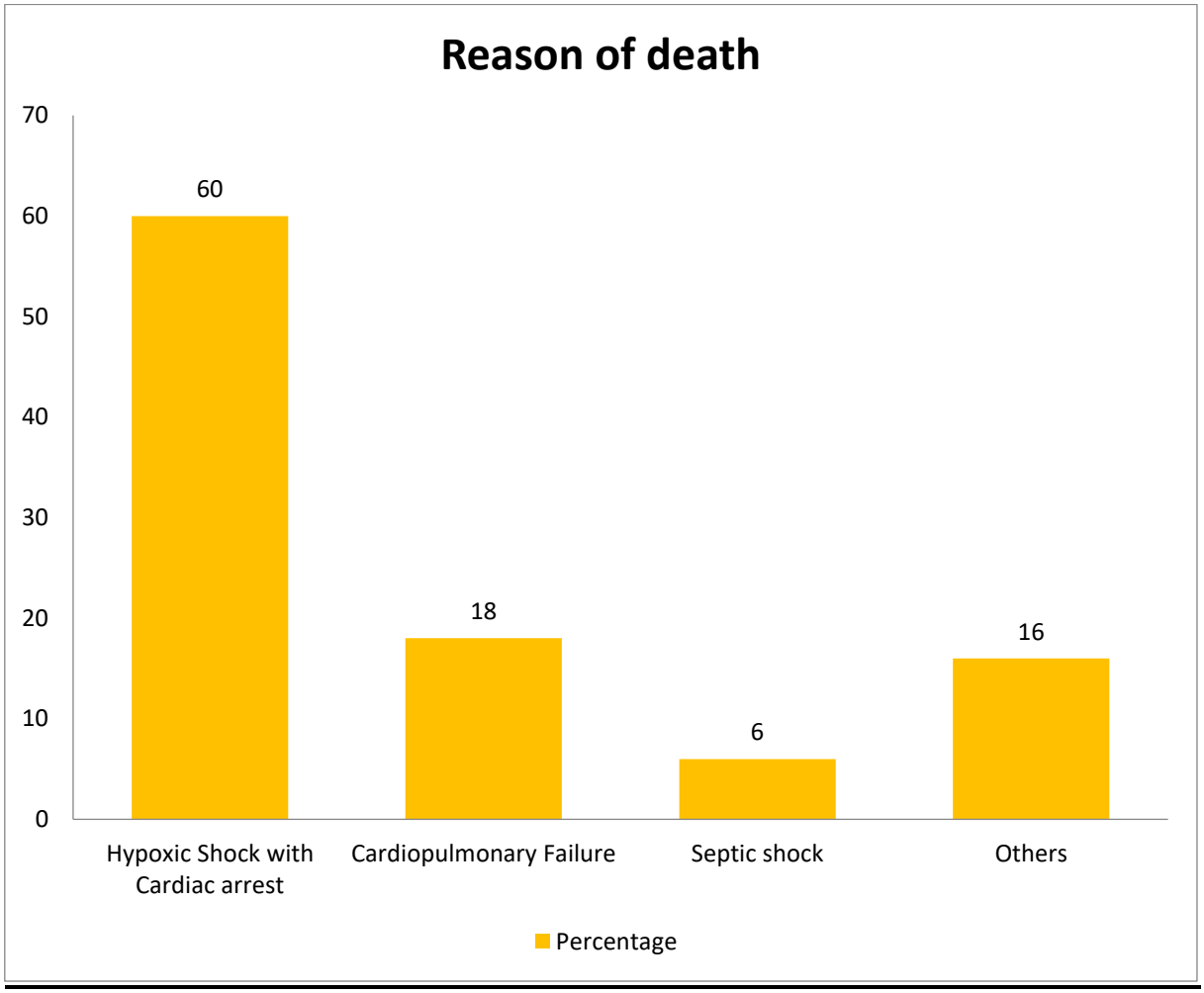

Figure 7: Reason of death of patients

According to our study, we have observed the most final diagnosis is Viral pneumonia with COVID-19. Out of 50 patients, 37 (74%) patients are diagnosed with COVID-19 (**Table:8, Fig:8**).

Table 8: Final Diagnosis of patients

Final Diagnosis	No. of patients	Percentage
VIRAL PNEUMONIA WITH COVID-19	37	74
VIRAL PNEUMONIA WITH 2◦ BACTERIAL SEPSIS	1	2
VIRAL PNEUMONIA WITH COVID-19 AND CYTOKINE STROME	1	2
VIRAL PNEUMONIA WITH COVID-19 AND CKD	2	4

VIRAL PNEUMONIA WITH COVID-19 AND SEPSIS WITH MULTI ORGAN DYSFUNCTION SYNDROME	1	2
VIRAL PNEUMONIA, CVA, UROSEPSIOS, ASPIRATIONAL PNEUMONIA	1	2
VIRAL PNEUMONIA WITH COVID-19 AND DKA	2	4
POST COVID PNEUMONIA WITH Lt PNEUMOTHORAX	1	2
DYSELECTROLYTEMIA WITH POST COVID-19 STATUS AND SVT	1	2
VIRAL PNEUMONIA WITH COVID-19, SEPTIC SHOCK & MODS	1	2
VIRAL PNEUMONIA WITH COVID-19, SEPTIC SHOCK WITH MODS, CKD & SEVERE ASTHMA	1	2
VIRAL ENCEPHALOPATHY WITH COVID-19	1	2

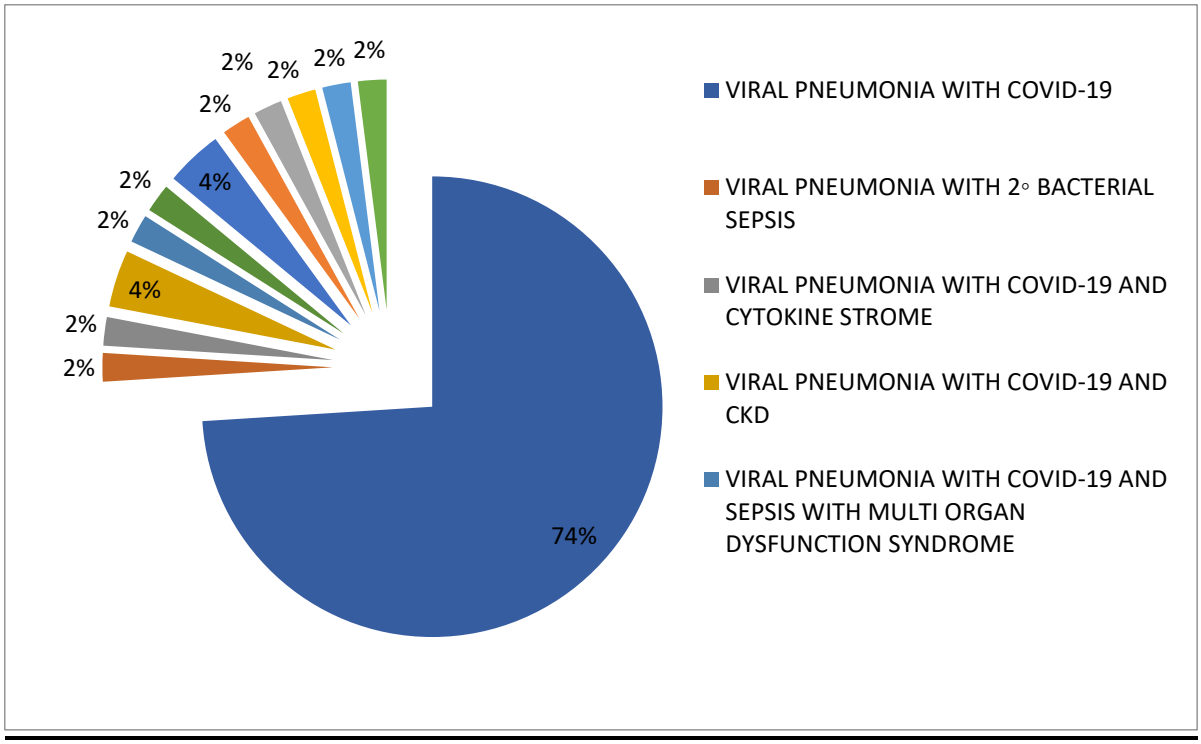

Figure 8: Percentage of Final diagnosis of patients

In our study population (n=50), 37(74%) were treated with Vitamin C (**Table:9, Fig:9**). These drugs are used to treat low levels of Vitamin C. 30(60%) were treated with Thiamine (Vit B1). These are used to treat Vitamin B1 deficiency. 28 (56%) were treated with Pantoprazole. These drugs are used to treat acid reflux. 24(48%) were treated with Dexamethasone. These drugs are used to treat allergic symptoms. 22(44%) patients were treated with Enoxaparin. These drugs are used to prevent blood clots.

Table 9: Prescribed Drugs to patients

Drugs Prescribed	No. of Patients	Percentage
Vitamin C	37	74

Thiamine (Vit B1)	30	60
Pantaprazole	28	56
Dexamethasone	24	48
Enoxaparin	22	44
Atorvastatin	19	38
Colchicine	19	38
Remdesivir	18	36
Vitamin D3	17	34
Methylprednisolone	17	34
Acetylecystein	17	34
Famotidine	15	30
Zinc	15	30
Cefoperazone + Sulbactam	11	22
Ivermectin	11	22
Ondansetron	10	20
Others	15	30

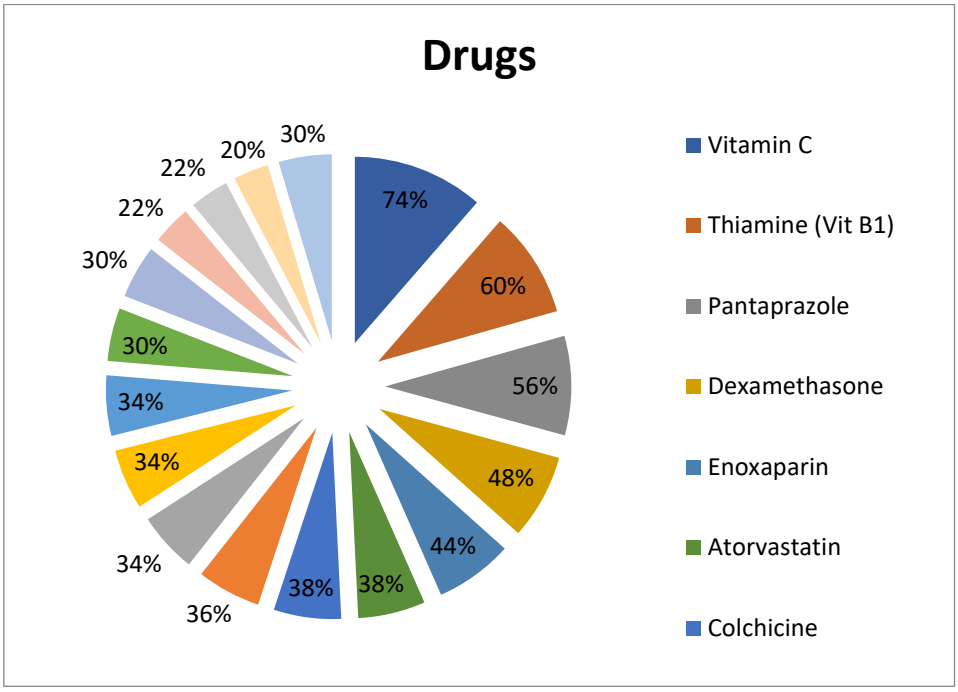

Figure 9: Percentage of Drugs Prescribed to the patients

CONCLUSION:

From the study it was found that, among the selected population (n=50) we found males (62%) are more prone to death with a higher percentage of the population was with co-morbidities like hypertension (64%) and diabetes (54%). The higher percentages of the population were belonging to the age group 61-80+ (54%).

As per the retrospective observational study, we observed that,

1. More deaths due to COVID-19 in the study population are seen as males (62%).

2. According to age, 61-80+ years are affected more because at this age people have a weak immune system.
3. In our study we found that people with co-morbidities like Hypertension (64%) and Diabetes (54%) are more prone to death than people with no co-morbidities.
4. We found that the most prevalent chief complaints reported in patients who died of COVID-19 were Fever (78%), SOB (76%), and Cough (70%).
5. We found that vaccinated people are at low risk of mortality than non-vaccinated ones.
6. We found that the most common reason for death was Hypoxic shock with cardiac arrest (60%) in patients who died of COVID-19.
7. We found that most of the patients who died of COVID-19 were diagnosed with Viral pneumonia with COVID-19 (74%).
8. We found that the most common drug received by the patients who died of COVID-19 were Vitamin C (74%), Thiamine (Vit B1) (60%), Pantoprazole (56%), Dexamethasone (48%) and Enoxaparin (44%).

References:

1. Y. C. Liu, R. L. Kuo, and S. R. Shih, "COVID-19: The first documented coronavirus pandemic in history," Aug. 01, 2020, *Elsevier B.V.* doi: 10.1016/j.bj.2020.04.007.
2. Z. Liu *et al.*, "Association Between Diabetes and COVID-19: A Retrospective Observational Study With a Large Sample of 1,880 Cases in Leishenshan Hospital, Wuhan," *Front Endocrinol (Lausanne)*, vol. 11, Jul. 2020, doi: 10.3389/fendo.2020.00478.
3. J. Baj *et al.*, "COVID-19: Specific and non-specific clinical manifestations and symptoms: The current state of knowledge," Jun. 01, 2020, *MDPI*. doi: 10.3390/jcm9061753.
4. P. Kelaita, K. Pienaar, J. Keaney, D. Murphy, H. Vally, and C. M. Bennett, "Pandemic policing and the construction of publics: an analysis of COVID-19 lockdowns in public housing," *Health Sociology Review*, vol. 32, no. 3, pp. 245–260, 2023, doi: 10.1080/14461242.2023.2170260.
5. D. Wolff, S. Nee, N. S. Hickey, and M. Marschollek, "Risk factors for COVID-19 severity and fatality: a structured literature review," Feb. 01, 2021, *Springer Science and Business Media Deutschland GmbH*. doi: 10.1007/s15010-020-01509-1.
6. S. Sengupta *et al.*, "All India ophthalmological society - Indian journal of ophthalmology consensus statement on preferred practices during the COVID-19 pandemic," *Indian J Ophthalmol*, vol. 68, no. 5, pp. 711–724, May 2020, doi: 10.4103/ijo.IJO_871_20.
7. M. A. Andrews *et al.*, "First confirmed case of COVID-19 infection in India: A case report," May 01, 2020, *Wolters Kluwer Medknow Publications*. doi: 10.4103/ijmr.IJMR_2131_20.
8. N. Jahan *et al.*, "Entry and initial spread of COVID-19 in India: Epidemiological analysis of media surveillance data, India, 2020," *Clin Epidemiol Glob Health*, vol. 9, pp. 347–354, Jan. 2021, doi: 10.1016/j.cegh.2020.10.008.
9. Z. J. Madewell, Y. Yang, I. M. Longini, M. E. Halloran, and N. E. Dean, "Household Transmission of SARS-CoV-2: A Systematic Review and Meta-analysis," *JAMA Netw Open*, vol. 3, no. 12, Dec. 2020, doi: 10.1001/jamanetworkopen.2020.31756.
10. K. Linka, M. Peirlinck, and E. Kuhl, "The reproduction number of COVID-19 and its correlation with public health interventions," May 06, 2020. doi: 10.1101/2020.05.01.20088047.

11. Z. Zhou, Y. Qiu, and X. Ge, "The taxonomy, host range and pathogenicity of coronaviruses and other viruses in the Nidovirales order," Dec. 01, 2021, *BioMed Central Ltd*. doi: 10.1186/s44149-021-00005-9.
12. R. Perotte, G. Sugalski, J. P. Underwood, and M. Ullo, "Characterizing COVID-19: A chief complaint based approach," *American Journal of Emergency Medicine*, vol. 45, pp. 398–403, Jul. 2021, doi: 10.1016/j.ajem.2020.09.019.
13. L. Huang *et al.*, "Rapid asymptomatic transmission of COVID-19 during the incubation period demonstrating strong infectivity in a cluster of youngsters aged 16-23 years outside Wuhan and characteristics of young patients with COVID-19: A prospective contact-tracing study," *Journal of Infection*, vol. 80, no. 6, pp. e1–e13, Jun. 2020, doi: 10.1016/j.jinf.2020.03.006.
14. E. J. Williamson *et al.*, "Factors associated with COVID-19-related death using OpenSAFELY," *Nature*, vol. 584, no. 7821, pp. 430–436, Aug. 2020, doi: 10.1038/s41586-020-2521-4.
15. G. Suleyman *et al.*, "Clinical Characteristics and Morbidity Associated with Coronavirus Disease 2019 in a Series of Patients in Metropolitan Detroit," *JAMA Netw Open*, vol. 3, no. 6, p. E2012270, Jun. 2020, doi: 10.1001/jamanetworkopen.2020.12270.
16. K. Ejima *et al.*, "Estimation of the incubation period of COVID-19 using viral load data," *Epidemics*, vol. 35, Jun. 2021, doi: 10.1016/j.epidem.2021.100454.
17. W. J. Wiersinga, A. Rhodes, A. C. Cheng, S. J. Peacock, and H. C. Prescott, "Pathophysiology, Transmission, Diagnosis, and Treatment of Coronavirus Disease 2019 (COVID-19): A Review," Aug. 25, 2020, *American Medical Association*. doi: 10.1001/jama.2020.12839.
18. Y. Chen, X. Zhao, H. Zhou, H. Zhu, S. Jiang, and P. Wang, "Broadly neutralizing antibodies to SARS-CoV-2 and other human coronaviruses," Mar. 01, 2023, *Nature Research*. doi: 10.1038/s41577-022-00784-3.
19. L. Falzone, G. Gattuso, A. Tsatsakis, D. A. Spandidos, and M. Libra, "Current and innovative methods for the diagnosis of COVID-19 infection (Review)," *Int J Mol Med*, vol. 47, no. 6, Jun. 2021, doi: 10.3892/ijmm.2021.4933.
20. J. H. Beigel *et al.*, "Remdesivir for the Treatment of COVID-19 — Final Report," *New England Journal of Medicine*, vol. 383, no. 19, pp. 1813–1826, Nov. 2020, doi: 10.1056/nejmoa2007764.
21. L. Falzone, G. Gattuso, A. Tsatsakis, D. A. Spandidos, and M. Libra, "Current and innovative methods for the diagnosis of COVID-19 infection (Review)," *Int J Mol Med*, vol. 47, no. 6, Jun. 2021, doi: 10.3892/ijmm.2021.4933.
22. A. Sharma, S. Tiwari, M. K. Deb, and J. L. Marty, "Severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2): a global pandemic and treatment strategies," *Int J Antimicrob Agents*, vol. 56, no. 2, Aug. 2020, doi: 10.1016/j.ijantimicag.2020.106054.
23. M. Lotfi, M. R. Hamblin, and N. Rezaei, "COVID-19: Transmission, prevention, and potential therapeutic opportunities," Sep. 01, 2020, *Elsevier B.V.* doi: 10.1016/j.cca.2020.05.044.
24. R. Güner, İ. Hasanoğlu, and F. Aktaş, "COVID-19: Prevention and control measures in community," 2020, *Turkiye Klinikleri*. doi: 10.3906/sag-2004-146.
25. O. Dadras *et al.*, "Effects of COVID-19 prevention procedures on other common infections: a systematic review," Dec. 01, 2021, *BioMed Central Ltd*. doi: 10.1186/s40001-021-00539-1.