RESEARCH PAPER

An Observational Study On Cocktail Antibodies Treatment In Severe Acute Respiratory Syndrome Coronavirus 2

K Saikrishna[1*], Karthik P N[2], Manisha Kodali[3], S Bhavana[4], Dr. P Vishnu Rao[5], Mitta Sandeep Goud[6]

[1]Assistant Professor, [2, 3, 4]Student, Sri Venkateshwara College of Pharmacy, Madhapur, Hyderabad-500081.
[5]MBBS, MD, FNB (Infectious diseases) CICK, DRCPath, Apollo Hospitals, Jubilee Hills
[6]Assistant Professor, Department of Pharm D, Vaagdevi Pharmacy College, Bollikunta Warangal, Telangana, 506002, India.

ABSTRACT

Background:
Coronavirus disease (COVID-19) has caused a global pandemic with high mortality, particularly among patients who develop acute respiratory distress syndrome (ARDS). Severe disease is often associated with cytokine release syndrome, characterized by elevated inflammatory markers such as interleukin-6 (IL-6). Tocilizumab, a monoclonal antibody that inhibits the IL-6 receptor, is widely used in rheumatological conditions and has been explored as an off-label therapy for moderate to severe COVID-19 to control hyperinflammation.

Aim:
To evaluate the role of Tocilizumab in patients with severe COVID-19 infection.

Methods:
A retrospective observational study was conducted on patients aged above 18 years who were RT-PCR positive for COVID-19 and received Tocilizumab therapy during hospitalization between May 2020 and May 2021. Demographic data, inflammatory markers (IL-6, C-reactive protein, D-dimer, white blood cell count), and drug-related details such as dosage, frequency, and duration were collected using a structured data collection form. Statistical analysis was performed to evaluate treatment outcomes.

Results:
Among the study population, 76% were male and 24% were female. Patients aged 41–60 years and those above 61 years each constituted 46% of the sample. Higher mortality was observed in patients above 61 years. Diabetes mellitus was the most common comorbidity, followed by hypertension and coronary artery disease. Most patients had moderate to severe CT severity scores. ARDS was the most frequent complication. All patients received intravenous Tocilizumab 400 mg, with most receiving a single dose. Significant reductions in IL-6, CRP, and D-dimer levels were observed after treatment. Clinical improvement was noted in 72% of patients.

Conclusion:
Tocilizumab therapy was associated with improved clinical outcomes and reduced inflammatory markers in patients with severe COVID-19, particularly in those below 60 years and without major comorbidities. Larger randomized controlled trials are required to confirm these findings.

Keywords: COVID-19, Cytokine storm, Tocilizumab and ARDS.

How to cite this article: Saikrishna K, Karthik PN, Kodali M, Bhavana S, Rao PV, Goud MS. An Observational Study On Cocktail Antibodies Treatment In Severe Acute Respiratory Syndrome Coronavirus 2. Int J Drug Deliv Technol. 2026;16(6s): 481-489; DOI: 10.25258/ijddt.16.6s.72

Source of support: Nil

Conflict of interest: None

INTRODUCTION

A novel corona virus first identified in Wuhan, China on December 2019, is the causative agent of corona virus disease 2019 (COVID -19). Since then, the disease has spread worldwide, resulting in a pandemic1. According to WHO, there were 34,808,886 cases of COVID-19 with 480,592 deaths in India from 3rd January 2020 to 29th December2 .The symptoms of infection ranges from sore throat, cough, fever, pneumonia and Acute Respiratory distress syndrome (ARDS) to new symptoms like loss of smell, dry mouth, rashes and many others3.When an infected person coughs, sneezes, speaks the virus spread in microscopic liquid particles from their mouth or nose .These particles range from larger respiratory droplets to smaller aerosols4.

After becoming infected, most persons have few or no symptoms despite having high viral loads, and their condition can be managed on an outpatient basis. In a smaller number of persons, hypoxia develops, leading to hospitalization and receipt of supplemental oxygen5. Viruses like SARS-CoV-2 continuously evolve as changes in the genetic code (genetic mutations) occur during replication of the genome. A lineage is a genetically closely related group of virus variants derived from a common ancestor. A variant has one or more mutations that differentiate it from other variants of the SARS-CoV-2 viruses. As expected, multiple variants of SARS-COV-2 have been documented in the United States and globally throughout this pandemic6.

The SARS-CoV-2 begins its life cycle when its S protein binds to the host angiotensin converting enzyme-2 (ACE2) cellular receptor. Following this step, there occurs a conformation change in the S protein which facilitates viral envelope fusion with the host cell membrane utilizing the endosomal pathway. Then SARS-CoV-2 RNA releases its genetic material, RNA into the host cell. The genome RNA is translated into viral replicase polyproteins pp1a and 1ab, which are then cleaved into small by-products with the aid of viral proteinases. The polymerase yields a series of sub-genomic mRNAs by disjointed transcription and finally translated into relevant viral proteins. These viral proteins and genome are then soon assembled into virions in the endoplasmic reticulum and Golgi and then transported via vesicles and finally released out of the host cell7.

The main pathophysiology involved in COVID-19 disease is that when the severe acute respiratory syndrome corona virus -2 (SARS-CoV-2) attacks the human body enters via conducting airways, upper respiratory tract followed by lower respiratory tract. it activates alveolar macrophages which in turn releases the cytokines such as IL-6, TNF, IL1, IL-12 and other inflammatory mediators via ACE-2 and the continuous viral replication and infection of adjacent healthy alveolar cells leads to the apoptosis of both type-II followed by type-I pneumocytes leading to a condition called cytokine storm and finally resulting in acute respiratory distress syndrome (ARDS). The virus also affects the blood vessels causing vasculitis and lastly ARDS8.

COVID-19 has a variety of effects on different people. The majority of infected people will experience mild to moderate symptoms and recover without the need for hospitalization. The most prevalent symptoms are fever, coughing with a dry throat, tiredness. Less common symptoms are aches and pains, throat irritation, diarrhoea, conjunctivitis, headache, loss of taste or fragrance, a cutaneous rash or discoloration of the fingers or toes. Severe symptoms are: breathing difficulties or shortness of breath, Pain or pressure in the chest, speech or movement problems. Symptoms usually appear 5–6 days after a person is infected with the virus9.

Coronaviruses (CoVs) are positive-stranded RNA (+ssRNA) viruses with a crown-like appearance under an electron microscope (coronam is the Latin term for crown) due to the presence of spike glycoproteins on the envelope. The subfamily Orthocoronavirinae of the Coronaviridae family (order Nidovirales) classifies into four genera of CoVs:

- ✓ Alpha coronavirus (alphaCoV)
- ✓ Beta coronavirus (betaCoV)
- ✓ Delta coronavirus (deltaCoV)
- ✓ Gamma coronavirus (gammaCoV)

BetaCoV genus is further divided into five sub-genera or lineages.10 Genomic characterization has shown that bats and rodents are the probable gene sources of alphaCoV and betaCoV. On the contrary, avian species seem to represent the gene sources of deltaCoV and gammaCoV. CoVs have become the major pathogens of emerging respiratory disease outbreaks. Members of this large family of viruses can cause respiratory, enteric, hepatic, and neurological diseases in different animal species, including camels, cattle, cats, and bats. For reasons yet to be explained, these viruses can cross species barriers and can cause, in humans, illness ranging from the common cold to more severe diseases such as MERS and SARS. To date, seven human CoVs (HCoVs) capable of infecting humans have been identified. Some of the HCoVs were identified in the mid-1960s, while others were only detected in the new millennium. In general, estimates suggest that 2% of the population is healthy carriers of a CoVs and that these viruses are responsible for about 5% to 10% of acute respiratory infections10.

Structurally and phylogenetically, SARS-CoV-2 is similar to SARS-CoV and MERS-CoV and is composed of four main structural proteins: spike (S), envelope (E) glycoprotein, nucleocapsid (N), membrane (M) protein, along with 16 non-structural proteins, and 5-8 accessory proteins11. The surface spike (S) glycoprotein, which resembles a crown, is located on the outer surface of the virion and undergoes cleavage into an amino (N)-terminal S1 subunit, which facilitates the incorporation of the virus into the host cell and a carboxyl (C)-terminal S2 subunit containing a fusion peptide, a transmembrane domain, and cytoplasmic domain is responsible for virus-cell membrane fusion12.The S1 subunit is further divided into a receptor-binding domain (RBD) and N-terminal domain (NTD), which facilitates viral entry into the host cell and serves as a potential

target for neutralization in response to antisera or vaccines13. The RBD is a fundamental peptide domain in the pathogenesis of infection as it represents a binding site for the human angiotensin-converting enzyme 2 (ACE2) receptors. Inhibition of the renin-angiotensin-aldosterone system (RAAS), as previously hypothesized, does not increase the risk of hospitalization for COVID-19 and severe disease.

SARS-CoV-2 gains entry into the hosts' cells by binding the SARS-CoV-2 spike or S protein (S1) to the ACE2 receptors abundantly on respiratory epithelium such as type II alveolar epithelial cells. Besides the respiratory epithelium, ACE2 receptors are also expressed by other organs such as the upper oesophagus, enterocytes from the ileum, myocardial cells, proximal tubular cells of the kidney, and urothelial cells of the bladder14. The viral attachment process is followed by priming the spike protein S2 subunit by the host transmembrane serine protease 2 (TMPRSS2) that facilitates cell entry and subsequent viral replication endocytosis with the assembly of virions15.

In summary, the spike RBD allows the binding to the ACE2 receptor in the lungs and other tissues. The spike protein of an amino acid site (polybasic site) allows the functional processing of the same by the human enzyme furin (protease). This process enables the exposure of the fusion sequences and, therefore, the fusion of the viral and cell membranes, a necessary passage for the virus to enter the cell16.

COVID-19 can be diagnosed by RT-PCR or other nucleic acid tests of contaminated secretions. Chest CT scans, in addition to laboratory tests, may be useful in diagnosing the extent of COVID-19 in people who have a strong clinical suspicion of infection17.

Treatment mostly includes home isolation and symptomatic treatment (with Antipyretics, Antitussives), Doxycycline, Ivermectin, Hydroxychloroquine for mild cases and for moderate to severe cases Remdesivir, Methylprednisolone, NIV, ICU care. Clinical trials are under way and drugs to treat are still being explored18.

In the midst of COVID-19 pandemic, a variety of prophylactic and therapeutic treatments are being developed to combat COVID-19. India has reached a mile stone of dosing 10 million doses of vaccines for prevention of COVID-19 spread. While vaccination drive yet continues, India has been considering alternative therapies for treatment of COVID-19 spread. On May 5th, 2021 the central drugs standard's control organization (CDSCO) provided emergency use of authorization for the antibody cocktail (CASIRIVIMAB and IMDEVIMAB) in India. Monoclonal antibodies (mabs) that can bind to and 'neutralize' the virus infected patients are a novel class of antiviral intervention19. Treatment focuses on immunosuppressive drugs and anti-inflammatory compounds20. For more severe cases or when patients are hemodynamically unstable, interventions like transcatheter arterial embolization or surgery may be necessary to stop active bleeding and prevent further complications21.

MATERIALS AND METHODS

STUDY OBJECTIVES:

1. To observe the efficacy and safety of cocktail antibodies (Casirivimab and Imdevimab) in mild to moderate COVID-19 patients

2. To observe the severity, hospitalization and mortality in COVID-19 Patients.

STUDY METHODOLOGY:

Study design: Prospective Observational, Single Centered study

Study duration: 6 months

Review Period: 1 Year

Study site: Apollo Health City, Jubilee Hills, Hyderabad

SELECTION OF PATIENTS:

Inclusion Criteria:

1. Patients who were accepted to participate in the study and diagnosed as COVID-19 positive.

2. Patients who received monoclonal antibody cocktail (Casirivimab and Imdevimab) for the treatment of mild to moderate COVID-19

3. Patients who are able to communicate through phone call after 1st, 2nd and 3rd week from the date of receiving COVID-19 cocktail antibodies (Casirivimab and Imdevimab) therapy

4. Age >18 years

Exclusion criteria:

1. Patients requiring high-flow oxygen or mechanical ventilation 2.Critically ill and severe COVID-19 Patients

2. Pregnant and lactating women

3. < 18 years of age

STUDY PROCEDURE

A prospective, observational and single centered study will be conducted in Apollo hospital to assess the efficacy and safety of the cocktail antibodies given in COVID-19 patients.

Records of the patient will be studied and demographic details along with relevant information including Suspected Adverse Drug Reactions will be monitored with the help of Suspected Adverse Drug Reaction Reporting Form will be collected from patient profile.

Patients receiving cocktail antibodies will be monitored for 21 days with a gap of 7 days through phone call or revisits. Data collected will be tabulated in MS-EXCEL sheet and analyzed.

RESULTS AND DISCUSSION

Table 1 DISTRIBUTION OF PATIENTS BASED ON GENDER, AGE GROUP AND BMI

GENDER	**NO. OF PATIENTS**
Males	53
Females	27
Total	80
AGE GROUP	**NO. OF PATIENTS**
18-40	18
41-60	28
>61	34
Total	80
BMI	**No. of Patients**
<18 (Under Weight)	1
18-25(Healthy)	41
26-30(Over Weight)	24
>30(Obese)	14
Total	80

Based on Gender distribution majority of the patients were found to be males, (65% males vs. 35% females). Not much difference was seen in gender regarding health outcomes post Cocktail antibodies administration. Based on Age we have categorized our patients into 3 age groups. They are 18 years -40 years (22.5) %, 41-60 years (35%) and >60 years (75%).

In our study most of the patients are Healthy (51.25%) and overweight accounting for 30% followed by patients who were obese 17.5% and underweight 1%. Highest mortality was seen in patients who were obese

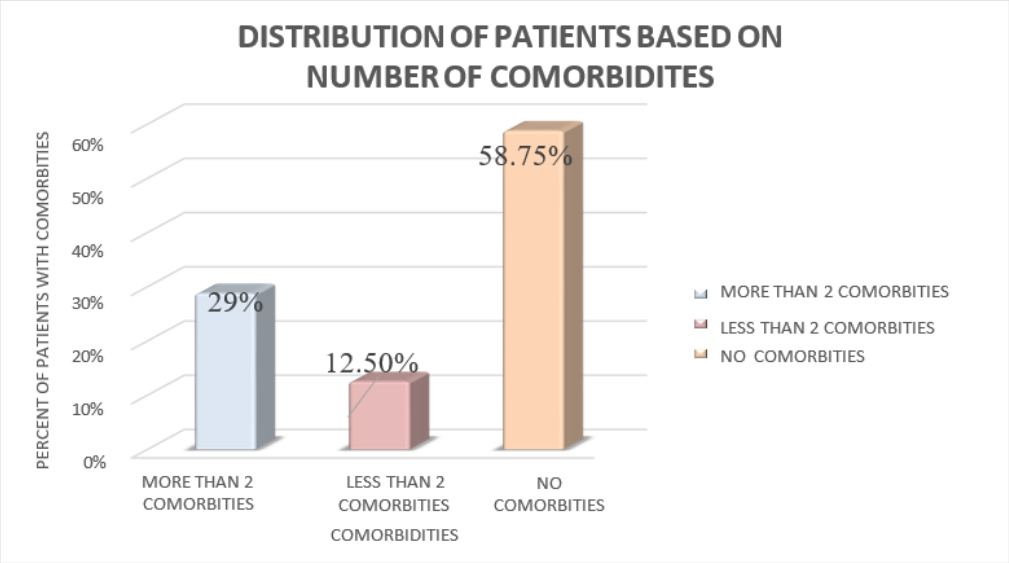

Fig 1 Distribution of patients based upon comorbidities

Patients with No comorbidities were found to be 58.75% with less than 2 comorbidities were found to be 12.50% and more than 2 comorbidities were 29%. The most common comorbidities are Diabetes (20%) followed by Hypertension (6.25%) coronary artery disease (7.5%), Hypothyroidism 3.75%, ulcerative colitis 1%, Urinary tract infection 1%, and microscopic polyangiitis 1%.

Table 2 DISTRIBUTION OF PATIENTS BASED ON CO-RADS SCORE

CO-RADS SCORE	NO. OF PATIENTS
CO-RADS 3	23
CO-RADS 4	19
CO-RADS 5	20
CO-RADS 6	18

Most of the patients have CO-RADS score of >3 accounting for 28.75%.

Table 3 DISTRIBUTION OF PATIENTS BASED ON CT- SEVERITY SCORE

CT SEVERITY SCORE	NO. OF PATIENTS
MILD (0-7)	44
MODERATE (8-17)	23
SEVERE (18-25)	13

CT severity of majority of patients was found to be Mild to Moderate state of COVID-19.

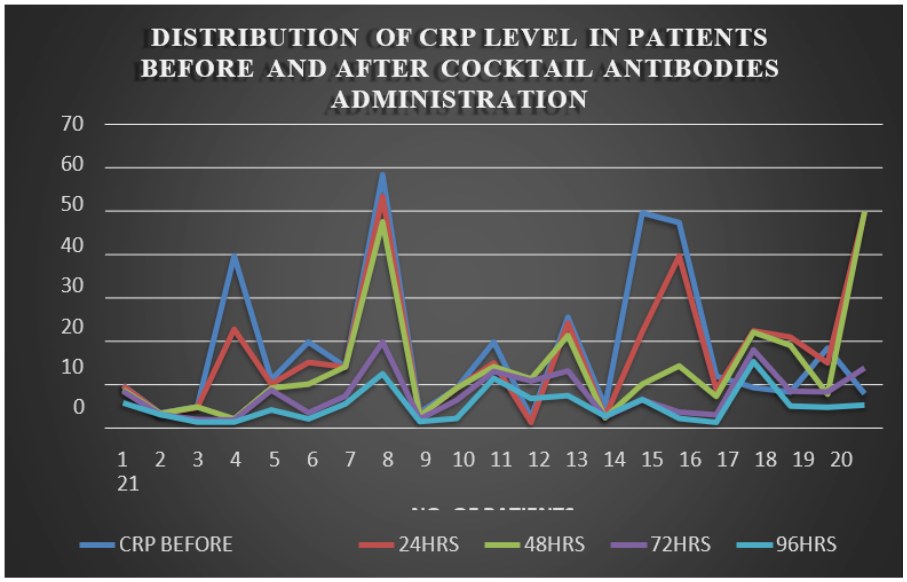

Fig 2 Distribution of CRP level in patients before and After Cocktail Antibodies Administration

C - reactive protein values before Cocktail Antibodies administration were compared with the values of 24 hrs, 48hrs, 72hrs; 96Hrs after Cocktail antibodies administration, reduction in CRP values were seen in about 26.5% of patients.

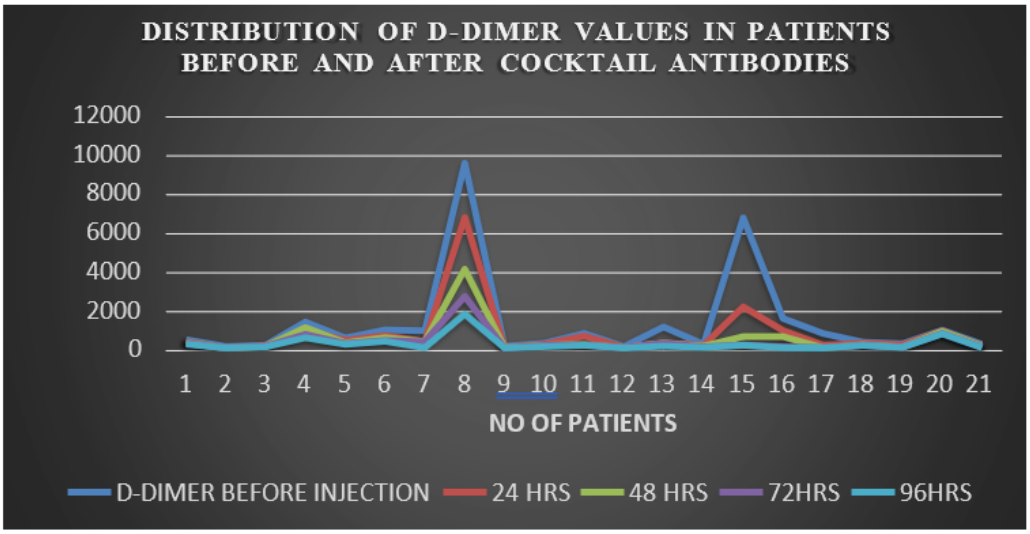

Fig 3 Distribution of D-dimer values in patients before and after Cocktail antibodies administration

D-dimer is a fibrin degradation product that is often used to measure and assess clot formation. Elevated D-dimer levels have been associated with disease severity and mortality trends. In our study much difference was observed in D-dimer values after Cocktail antibodies administration when compared with the initial values. A significant reduction was found in the D-dimer values after 48 to 72 Hrs of Cocktail antibodies administration.

Table 4 IF YOU HAVE FACED ANY SYMPTOMS WHAT SYMPTOMS THAT BOTHERED YOU THE MOST?

SYMPTOMS	NO OF PATIENTS
JOINT PAINS	23

UNABLE TO SLEEP	7
UNABLE TO SLEEP, CHEST PAIN	3
SOB	2
CHEST DISCOMFORT	1
JOINT PAINS, UNABLE TO SLEEP	1
NO ADRS	43

Here 28.7% of the patients suffered with joint pains 8.75%were unable to sleep, 3.75 % were unable to sleep, chest pain, 2.5% suffered with shortness of breath and 1.25% suffered with joint pains, unable to sleep and chest discomfort remaining 53.75 % of the patients have no ADRs.

Table 5 ADRs AFTER ADMINISTRATION OF COCKTAIL ANTIBODIES

ADRs	1st WEEK	2nd WEEK	3rd WEEK
BODY PAINS	21	5	0
FEVER	6	4	0
FEVER, BODY PAINS	23	4	0
HEADACHE	10	1	0
HEADACHE, BODY PAINS	12	1	0
NO ADRS	7	67	79
DEATH	1	0	0
CHEST PAIN	4	0	0
SOB	2	0	0
DEATH	1	0	0
NO ADRs	73	79	79
UNABLE TO SLEEP, DEPRESSED	11	0	0
GOOD, NO ADRs	69	79	79
GI IRRITATION	7	1	0
HEART BURN	3	1	0
VOMITINGS	4	2	0
NO ADRs	66	76	79
MYALGIA	0	0	0
FATIGUE	0	0	0
JOINT PAINS	38	0	0
NO ADRS	42	79	79

Most of the patients suffered with Fever, Body pains, Headache during the 1st week after administration of Cocktail antibodies it gradually decreased during the 2nd week and during the 3rd week most of them were stable. Most of the patients suffered with Chest pain, Shortness of breath during the 1st week after administration of cocktail antibodies and it gradually decreased during 2nd and 3rd week. Here 13.7% of patients were Depressed, Unable to sleep during the 1st week after cocktail antibodies administration and remaining were stable

Here 8.75% of the patients were suffered with GI irritation, 3.75% of the patients suffered with Heart Burn, 5% of the patients suffered with vomiting are during 1st week and they recovered during 2nd and 3rd week. Here 47.5% of patients suffered with Joint pains during 1st week and remaining were stable.

DISCUSSION

We included a total of 80 Patients who were diagnosed with Mild to Moderate COVID-19 Pneumonia and were treated with Casirivimab 600mg and Imdevimab 600mg Cocktail antibodies IV in the tertiary care hospital in our study. The data was collected from Medical Record Department of the hospital.

Based on Gender distribution majority of the patients were found to be males, (65% Males vs 35% Females). Not much difference was seen in gender regarding health outcomes post Cocktail antibodies administration. Based on Age we have categorized our patients into 3 age groups. They are 18 years -40 years (22.5) %, 41-60 years (35%) and >60 years (75%). From table 4.3 in our study most of the patients are Healthy (51.25%) and overweight accounting for 30% followed by patients who were obese 17.5% and underweight 1%. Highest mortality was seen in patients who were obese. Patients with No comorbidities were found to be 58.75% with less than 2 comorbidities were found to be 12.50% and more than 2 comorbidities were 29%. The most common comorbidities are Diabetes (20%) followed by Hypertension (6.25%) coronary artery disease (7.5%), Hypothyroidism 3.75%, ulcerative colitis 1%, Urinary tract infection 1%, and Microscopic polyangiitis 1%. Most of the patients have CO-RADS score of >3 accounting for 28.75%. CT severity of majority of patients was found to be Mild to Moderate state of COVID-19. C - reactive protein values before Cocktail Antibodies administration were compared with the values of 24 hrs, 48hrs, 72hrs; 96Hrs after Cocktail antibodies administration, reduction in CRP values were seen in about 26.5% of patients. D-dimer is a fibrin degradation product that is often used to measure and assess clot formation. Elevated D-dimer levels have been associated with disease severity and mortality trends. In our study much difference was observed in D-dimer values after Cocktail antibodies administration when compared with the initial values. A significant reduction was found in the D-dimer values after 48 to 72 Hrs of Cocktail antibodies administration. Serum electrolytes sodium, potassium, creatinine values were also analysed but there was no significant change in their values after Cocktail antibodies administration .so it may not have much effect on electrolytes and Kidneys. Most of the patients suffered with Fever, Body pains, Headache during the 1st week after Cocktail antibodies administration and it gradually decreased during 2nd week and the patients were normal from 3rd week.

CONCLUSION

The majority of patients demonstrated clinical improvement and was successfully discharged from hospital after receiving cocktail antibodies. We have observed that cocktail antibodies are effective in patients of 60 years of age and more effective in patients of age below 60 years. Cocktail antibodies showed good results in patients of normal BMI and with comorbidities. According to our study we have noted that cocktail antibodies have reduced mortality rate in Mild to Moderate COVID-19 Patients.

REFERENCES

1. Zhu N, Zhang D, Wang W, et al. A novel coronavirus from patients with pneumonia in China, 2019. N Engl J Med 2020; 382:727-733.
2. WHO COVID-19 Explorer. Geneva: World Health Organization, 2020, accessed 4 august 2021, https://covid19.who.int/region/searo/country/in.
3. Parasher A, COVID-19: Current understanding of its Pathophysiology, Clinical presentation and Treatment, Postgraduate Medical Journal 2021; 97:312-32
4. L, YC, BAI, HASHIKAWA, JOURNAL OF virology, 2020, Jan, 92(6)-552-5.
5. Muhammad Adnan Shereen, Suliman Khan, Abeer Kazmi, Nadia Bashir, Rabeea Siddique. COVID-19 infection: Emergence, transmission, and characteristics of human coronaviruses: Journal of Advanced Research, Volume 24, July 2020.
6. Goyal P, Choi JJ, Pinheiro LC, et al. Clinical characteristics of Covid-19 in New York City. N Engl J Med 2020; 382:2372-2374.
7. Volz E, Hill V, McCrone J, et al. Evaluating the Effects of SARS-CoV-2 Spike Mutation D614G on Transmissibility and Pathogenicity. Cell 2021; 184(64-75). Doi: https://doi.org/10.1016/j.cell.2020.11.020external.
8. Muhammad Adnan Shereen, Suliman Khan, Abeer Kazmi, Nadia Bashir, Rabeea Siddique. COVID-19 infection: Emergence, transmission, and characteristics of human coronaviruses: Journal f Advanced Research, Volume 24, July 2020.

9. Jodie Tester, Imperial College London, UK Cytokines: Introduction: British Society for Immunology. Accessed 4 august 2020, <https://www.immunology.org/public-information/bitesized-immunology/receptors-and-molecules/cytokines-introduction
10. Lei J, Kusov Y, Hilgenfeld R., et al. Nsp3 of coronaviruses: Structures and functions of a large multi-domain protein. Antiviral Res. 2018 Jan; 149:58-74.
11. Jiang S, Hillyer C, Du L., et al. Neutralizing Antibodies against SARS-CoV-2 and Other Human Coronaviruses: (Trends in Immunology 41, 355-359; 2020). Trends Immunol. 2020
12. Du L, He Y, Zhou Y, Liu S, Zheng BJ, Jiang S., et al. The spike protein of SARS- CoV--a target for vaccine and therapeutic development. Nat Rev Microbiol. 2009 Mar;7(3):226-36
13. Jiang S, Hillyer C, Du L., et al. Neutralizing Antibodies against SARS-CoV-2 and Other Human Coronaviruses. Trends Immunol. 2020 May; 41(5):355-359.
14. Song W, Gui M, Wang X, Xiang Y., et al. Cryo-EM structure of the SARS coronavirus spike glycoprotein in complex with its host cell receptor ACE2. PLoSPathog. 2018Aug: 14(8): e1007236.
15. Abajo FJ, Rodríguez-Martín S, Lerma V, Mejía-Abril G, Aguilar M, García-Luque A, Laredo L, Laosa O., et al. MED-ACE2-COVID19 study group. Use of renin- angiotensin-aldosterone system inhibitors and risk of COVID-19 requiring admission to hospital: a case-population study. Lancet. 2020 May 30; 395(10238):1705-1714.
16. Xu H, Zhong L, Deng J, Peng J, Dan H, Zeng X, Li T, Chen Q., et al. High expression of ACE2 receptor of 2019-nCoV on the epithelial cells of oral mucosa. Int J OraL Sci. 2020 Feb 24; 12(1):8
17. Hoffmann M, Kleine-Weber H, Schroeder S, Krüger N, Herrler T, Erichsen S Schiergens TS, Herrler G., et al. SARS-CoV-2 Cell Entry Depends on ACE2 and TMPRSS2 and Is Blocked by a Clinically Proven Protease Inhibitor. Cell. 2020 Apr 16; 181(2):271-280.e8.
18. Wang J, Jiang M, Chen X, Montaner LJ., et al. Cytokine storm and leukocyte changes in mild versus severe SARS-CoV-2 infection: Review of 3939 COVID-19 patients in China and emerging pathogenesis and therapy concepts. J Leukoc Biol. 2020 Jul; 108(1):17-41.
19. From Wikipedia, the free encyclopedia, COVID-19 testing. Accessed 4 august 2021: <https://en.wikipedia.org/wiki/COVID19_testing >.
20. Rohini S. Chiranjit Das, Sandeep Goud Mitta (2024). A Case Report on Arthritis Induced Inflammatory Bowel Disease. Glob Acad J Med Sci.; 6.
21. Parunandi Y, Shenkeshi S, Tanniru S, Mitta SG. A Case of Iliopsoas Hematoma in a Patient on Acenocoumarol. Indian Journal of Pharmacy Practice. 2025 Oct; 18(4):451-4.