

EVALUATION OF CLINICAL OUTCOMES IN POST COVID PATIENTS- POST COVID COMPLICATIONS

ORIGINAL RESEARCH ARTICLE

Dr. Mrudula Raj Vanga*¹, Dr. A. Pavan¹, Lavanya Malakalapalli², Ganta Lavanya², Lambu lalitha Naga Sai Sushma², T.Jharika Sri Sravani².

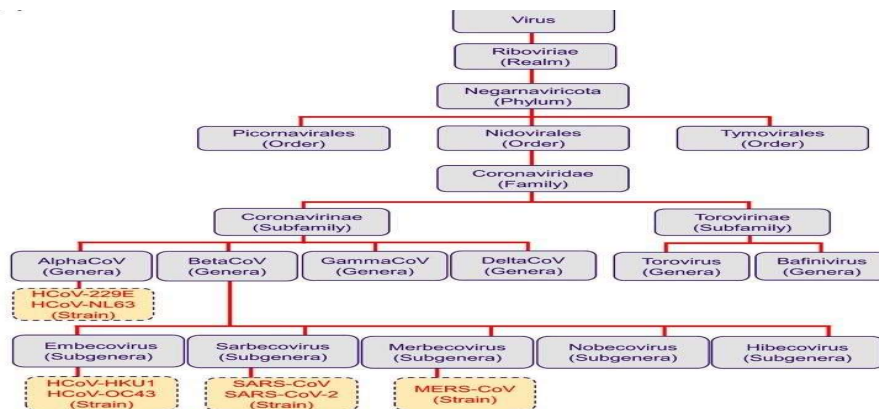
¹Department of Pharmacy Practice, Sir. C. R. Reddy College of Pharmaceutical Sciences, Eluru-534007, Andhra Pradesh, INDIA.

Corresponding Author:- Dr. Mrudula Raj Vanga

ABSTRACT :-Coronaviruses are important human and animal pathogens. At the end of 2019, a novel coronavirus was identified as the cause of a cluster of pneumonia cases in Wuhan, a city in the Hubei Province of China. It rapidly spread, resulting in an epidemic throughout China, followed by an increasing number of cases in other countries throughout the world. In February 2020, the World Health Organization designated the disease COVID-19, which stands for coronavirus disease 2019. The virus that causes COVID-19 is designated severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) previously, it was referred to as 2019-nCoV. This study showed that from the collected data the clinical outcomes varies with the following factors. Males are more effected than females. The infection of COVID-19 varies with their vaccination history. The occurrence of COVID-19 is more in population who have not been vaccinated. The occurrence of the post COVID clinical outcomes varies in different age groups. Patients with other comorbid conditions like Immune disorders, Hypertension, Diabetes mellitus are more prone to post COVID clinical outcomes. The serious post COVID clinical outcomes like Respiratory distress syndrome, Deep vein thrombosis, Migraine are mostly seen in individuals with age group of greater than 40 years.

KEY WORDS:- Covid, Ejection Fraction, Coronary Artery Disease, Coronary Artery Bypass Graft, Percutaneous Coronary Intervention, Left Ventricular Ejection Fraction, Left Anterior Descending Artery, Echo Cardiogram, Vessel Disease, Intra Aortic Balloon Pump.

INTRODUCTION:-Coronaviruses are important human and animal pathogens. At the end of 2019, a novel coronavirus was identified as the cause of a cluster of pneumonia cases in Wuhan, a city in the Hubei Province of China. It rapidly spread, resulting in an epidemic throughout China, followed by an increasing number of cases in other countries throughout the world. In February 2020, the World Health Organization designated the disease COVID-19, which stands for coronavirus disease 2019. The virus that causes COVID-19 is designated severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) previously, it was referred to as 2019-nCoV. [1] Coronaviruses (CoVs) are the largest group of viruses belonging to the *Nidovirales* order, which includes *Coronaviridae*, *Arteriviridae*, *Mesoniviridae*, and *Roniviridae* families. The *Coronavirinae* comprise one of two subfamilies in the *Coronaviridae* family, with the other being the *Torovirinae*. All viruses in the *Nidovirales* order are enveloped, non-seg. They all contain very large genomes for RNA viruses, with some viruses having the largest identified RNA genomes, containing up to 33.5 kilobase (kb) genomes mented positive-sense RNA viruses. The word corona means crown and refers to



the appearance that coronaviruses get from the spike proteins sticking out of them. These spike proteins are important to the biology of this virus. The spike protein is the part of the virus that attaches to a human cell to infect it, allowing it to replicate inside of the cell and spread to other cells. Some antibodies can protect you from SARS-CoV-2 by targeting these spike proteins. Because of the importance of this specific part of the virus, scientists monitor mutations causing changes to the spike protein through a process called genomic surveillance.

COVID-19 is caused by a virus called SARS-CoV-2. It is part of the corona virus family, which include common viruses that cause a variety of diseases from head or chest colds to more severe (but more rare) diseases like severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome (MERS). Bioinformatics analyses showed that SARS-CoV-2 had characteristics typical of corona virus family. It belongs to the beta corona virus 2B lineage. Early in the pneumonia epidemic in Wuhan, scientists obtained the complete genome sequences from five patients infected with SARS-CoV-2. These genome sequences share 79.5% sequence identity to SARS-CoV. Obviously, SARS-CoV-2 is divergent from SARS-CoV. It is considered to be a new beta coronavirus that infects human. Scientists aligned the full-length genome sequence of SARS-CoV-2 and other available genomes of beta coronavirus. Results indicate the closest relationship of SARS-CoV-2 with the bat SARS-like coronavirus strain BatCov RaTG13, with an identity of 96%. These studies suggest that SARS-CoV-2 could be of bat origin, and SARS-CoV-2 might be naturally evolved from bat coronavirus RaTG13. However, most ORFs annotated from SARS-CoV-2 are conserved. The spike gene exhibits larger dS (synonymous substitutions per synonymous site) values than other genes, which could be caused either by natural selection that accelerates synonymous substitutions or by a high mutation rate. One study analyzed the genomes of SARS-CoV-2 and similar isolates from the GISAID and NCBI. spike protein (S), nucleoprotein (N), and polyprotein (P) from other SARS-CoV-2, with 4, 2, and 22 variations in S, N, and P at the level of amino acid residues respectively. The results show that at least two SARS-CoV-2 strains are involved in the outbreak. Researchers obtained 103 SARS-CoV-2 genomes to recognize the genetic variants. Among the 103 strains, a total of 149 mutations are identified and population genetic analyses indicate that these strains are mainly divided into two types. Results suggest that 101 of the 103 SARS-CoV-2 strains show significant linkage between the two single nucleotide polymorphisms (SNPs). The major types of SARS-CoV-2 (L type and S type) are distinguished by two SNPs which locate at the sites of 8,782 and 28,144. L type accounts for 70% of the 103 strains and S type accounts for 30%, suggesting L type is more prevalent than the S type. However, S type is the ancestral version of SARS-CoV-2. To date, 13 mutations in the spike protein have been identified. The mutation D614G should be paid special attention. In early February, the mutation Spike D614G began spreading in Europe. When introduced to new regions, it rapidly replaced the original strain to become the dominant strain. The D614G mutation in the spike protein would increase infectivity. S^{G614} is more stable than S^{D641} and less S1 shedding are observed, so the SARS-CoV-2 with S^{G614} could transmit more efficiently. One study shows that in multiple cell lines, the SARS-CoV-2 carrying the D614G mutation is eight times more effective at transducing cells than wild-type spike protein, providing evidence that the D614G mutation in SARS-CoV-2 spike protein could increase the transduction of multiple human cell types. The D614G mutation could also decrease neutralization sensitivity to the sera of convalescent COVID-19 patients.⁽⁴⁾

STRUCTURE.

A key set of the proteins in the outer membrane project out from the particle and are known as spike proteins (S). It is these proteins which are recognized by receptor proteins on the host cells which will be infected.^[5]

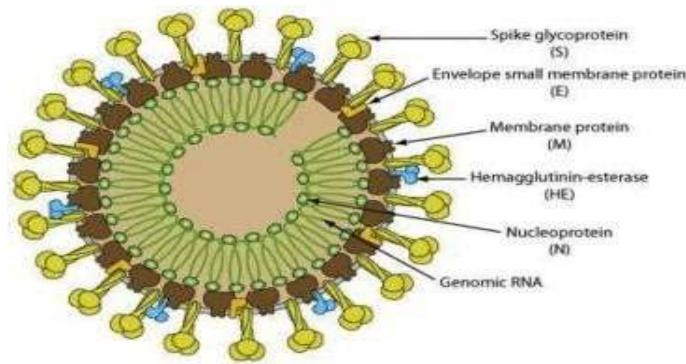


Fig:-1.2 Structure of corona virus

VARIANTS:

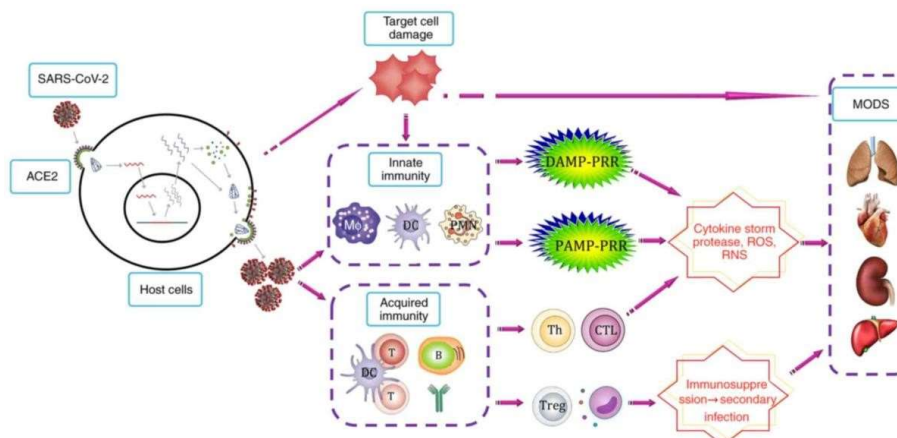
New variants will continue to emerge. Some variants spread more easily and quickly than other variants, which may lead to more cases of COVID-19. Even if a variant causes less severe disease in general, an increase in the overall number of cases could cause an increase in hospitalizations, put more strain on healthcare resources and potentially lead to more deaths.^[3]

Types of variants: Coronavirus Mutations

1. Alpha.(B.1.1.7)
2. Beta (B.1.351).
3. Gamma (P.1)
4. Delta (B.1.617.2)
5. Mu (B.1.621).
6. Omicron, Epsilon, Theta, and Zeta

Epidemiology: Since the initial report from China, the disease spread rapidly, and the number of cases increased exponentially. On January 11, the first case was reported outside mainland China in Thailand, and within months, the disease spread to all the continents except Antarctica. India reported its first case of COVID-19 on January 30, 2020. This rose to three cases by February 3, 2020. No further cases were reported in February 2020. However, by mid-March, the number of infected cases started to increase, and many cases were reported from all over India. The first COVID-19 related death in India was reported on March 12, 2020. By the second week of April, the disease spread to all states in India except Sikkim.^[7]

Etiology: Complete viral genome analysis reveals that the virus shares 88% sequence identity with two bat-derived severe acute respiratory syndromes (SARS)-like coronaviruses, but is more distant from the severe acute respiratory syndrome coronavirus (SARS-CoV). Hence, it was temporarily called 2019-novel coronavirus (SARS-CoV-2). Coronavirus is an enveloped and single-stranded ribonucleic acid named for its solar corona like appearance due to 9–12 nm-long surface spikes. There are four major structural proteins encoded by the coronaviral genome on the envelope, one of which is the spike (S)



Protein that binds to the angiotensin-converting enzyme 2 (ACE2) receptor and mediates subsequent fusion between the envelope and host cell membranes to aid viral entry into the host cell. On 11 February 2020, the Coronavirus Study Group (CSG) of the International Committee on Taxonomy of Viruses finally designated it as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) based on phylogeny, taxonomy and established practice. Soon after, WHO named the disease caused by this coronavirus as Coronavirus Disease 2019 (COVID-19). Based on current data, it seems that bats might initially host COVID-19, which might have been transmitted to humans via pangolin or other wild animals sold at the Huanan seafood market, with subsequent spread via human-to-human transmission.[8]

Mode Of Transmission: Broadly, two modes of transmission of COVID-19 exist—direct and indirect.

The direct mode includes (1) transmission via aerosols formed via surgical and dental procedures and/or in the form of respiratory droplet nuclei; (2) other body fluids and secretions, for example, feces, saliva, urine, semen, and tears; and (3) mother-to-child. Indirect transmission may occur via (1) fomites or surfaces (e.g., furniture and fixtures) present within the immediate environment of an infected patient and (2) objects used on the infected person (e.g., stethoscope or thermometer).^[10] Very small droplets or aerosols that stay in the air for several minutes or hours — called airborne transmission. Can also spread from someone who is infected but hasn't developed symptoms yet. This is called presymptomatic transmission.

RISK FACTORS: Age, Immune system -- body's defence against germs – weakens heart problems, Long-term kidney disease. Dialysis can weaken immune system. Cancer, Chronic obstructive pulmonary disease (COPD), Diabetes, Asthma, Weakened immune system because of an organ transplant, Mental health.

Pathophysiology: The Host Defense Against SARS-CoV-2

Early in infection, SARS-CoV-2 targets cells, such as nasal and bronchial epithelial cells and pneumocytes, through the viral structural spike (S) protein that binds to the angiotensin-converting enzyme 2 (ACE2) receptor.

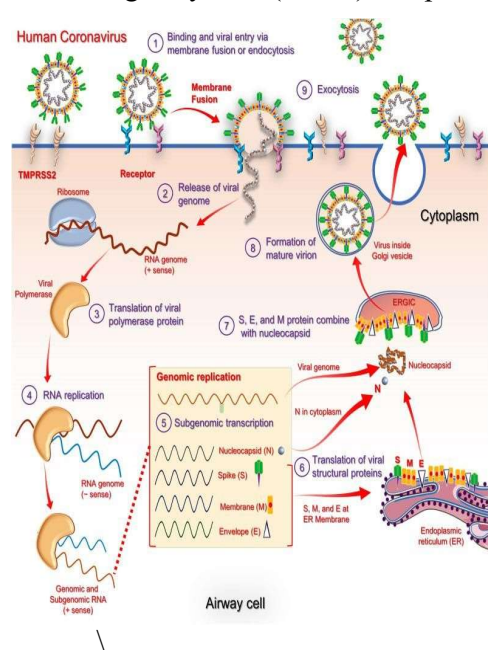


Fig:-1. 6 Pathophysiology of COVID-19

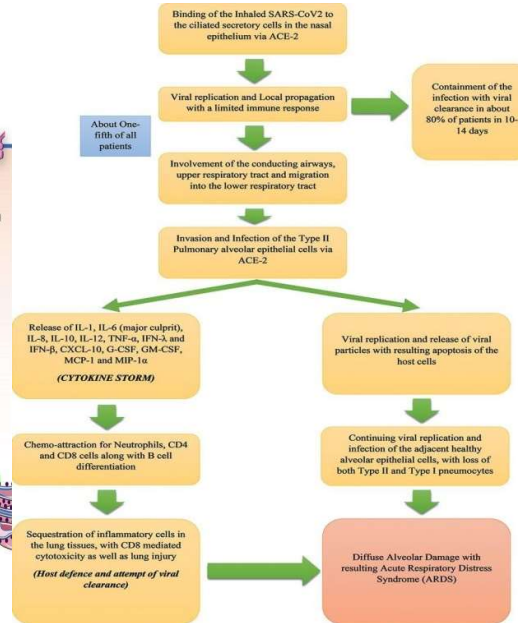


Fig:-1.7 Cytokine action in body

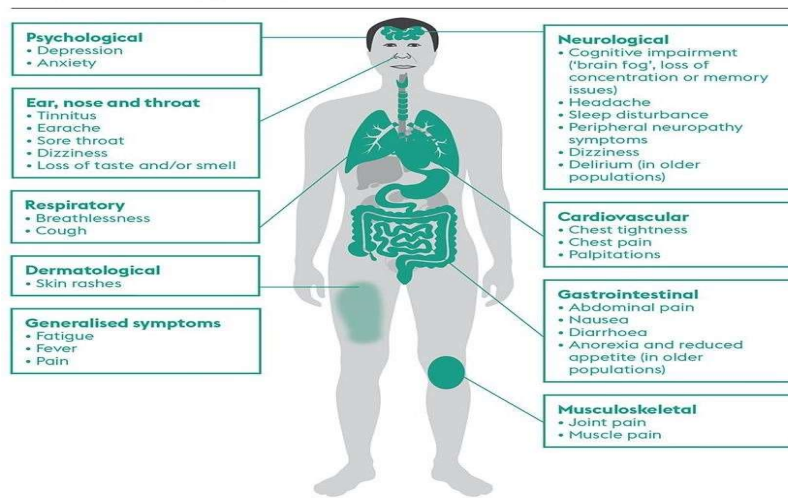


Fig:-1.8Clinical features in COVID

Diagnosis: If the person have COVID-19 symptoms

- At least 5 days after known or suspected close contact to COVID-19
- For screening (schools, workplaces, congregate settings, etc.)
- Before and after travel

When asked by a healthcare professional or public health official

Types of Viral Tests :-Laboratory Test ,Rapid Test If Positive Result

Isolate for at least 5 days. Learn more about isolation timelines and precautions Seek a confirmatory, follow-up laboratory test if recommended by healthcare profession

If Negative Result

- If up to date on vaccines: return to normal activities. Wear a mask indoors in areas where the COVID-19 Community Level is high.
- If not up to date on vaccines and have symptoms or exposure: quarantine for at least 5 days.
- If not up to date on vaccines and have no symptoms or exposure: return to normal activities. Take steps to get up to date on vaccines to protect yourself and others.
- Testing is very important to help reduce the spread of COVID-19. The patient should always discuss test results with healthcare provider ,Viral Tests
- Rapid Point-of-Care tests Laboratory tests include RT-PCR,CT other types of NAATs. Antibody Test Molecular tests (RT-PCR) [3] ,Serology ,Blood tests ,Chest X-ray

Treatment:The treatment is mainly symptomatic and supportive in most cases. Initially, the patient presenting to the emergency is categorised into mild, moderate or severe according to the symptoms on presentation. Most patients present with mild-to-moderate symptoms such as fever, persistent dry cough, body aches and occasional breathlessness. A small fraction of patients may also present with acute respiratory failure and acute respiratory distress syndrome with associated sepsis or multiorgan failure. The complete management protocol for patients with COVID-19 is depicted.^[15]High-flow nasal oxygen (HO) therapy and NIV. Severe cases (SpO₂ levels ≤90% in room air or patients with ARDS) Endotracheal intubation and mechanical ventilation :-Other Therapies for Covid-19 1. **Antibiotics** 2. **Corticosteroids** 3. **Antiviral drugs**

The following antiviral drugs have been put to use for COVID-19 patients so far.

- Remdesivir (CIPREMI/COVIFOR)
- Lopinavir/ritonavir (KALETRA)
- Oseltamivir (TAMIFLU)
- Favipiravir (FABIFLU)

Immunomodulatory drugs

Tocilizumab

Chloroquine and hydroxychloroquine

Plasma exchange via convalescent plasma.

Supplementary therapies.^[15]

Complications :

Respiratory system involvement :- single ground-glass opacity (GGO) to bilateral diffuse heterogeneous consolidation with air bronchogram and bronchiectasis, the 'white lung'. By contrast, intact angiotensin II stimulates pro-inflammatory responses and increases vascular permeability of the lung tissue, which leads to ARDS. Flow cytometric analysis of critically ill COVID-19-infected patients demonstrated that significant lung injury is accompanied by a substantial decrease in the number of CD4⁺, CD8⁺ T lymphocytes and NK cells. Cytokine release syndrome (CRS), which finally results in ARDS and multiorgan failure

Cardiovascular involvement Hypertension, diabetes, heart failure and coronary artery disease patients. pro-inflammatory state treatment with ACE inhibitors/angiotensin II receptor blockers (ARB) and high level of ACE 2 expression could result in delayed viral clearance and propensity of these individuals to COVID-19. Cardiomyopathy myocarditis was observed as a primary manifestation. In one case report, viral myocarditis associated with COVID-19 occurred without any sign and symptoms of pneumonia. The invasion of the viruses in the bloodstream mediated by ACE 2 receptors highly distributed in the heart and endovascular system stimulates CRS

Kidney involvement :- Acute kidney injury. Similar to SARS-CoV and MERS, the kidneys are potential targets for COVID-19. Podocytes and proximal tubular epithelial cells highly enriched with ACE 2 receptors, are distinctive targets for SARS-CoV-2. glomerular and tubular injury associated with COVID-19 infection. Therefore, AKI appears to be closely associated with both the severity and prognosis of COVID-19 patients. The pathophysiology of AKI associated with COVID-19. AKI during hospitalization.

Hematologic involvement:- Thrombocytopenia associated with COVID-19 reflects the severity of the disease. Coronavirus may invade the hematopoietic cells or cause abnormal hematopoiesis secondary to immune system response. In addition, virus-induced alveolar damage affects the resident megakaryocytes in the lungs (decrease platelet production). The presence of active CD61⁺ megakaryocytes in autopsy examinations of the lungs confirms this theory. Finally, endothelial damage related to coronavirus infection and mechanical ventilation could lead to platelet aggregation as well as thrombus formation (increased platelet consumption).

Coagulopathy:- thrombosis in pulmonary vessels in severe COVID-19. The median time from hospital admission to DIC development was 4 days (range, 1–12 days).

Electrolyte imbalance:- urinary potassium loss. Moreover, in patients with severe diarrhea and/or vomiting, extrarenal potassium loss could also cause or aggravate hypokalaemia.

Liver involvement :- AST elevation occurs in 20% of the COVID-19 patients

Gamma-glutamyltransferase (γ -GT) and lactate dehydrogenase (LDH) has also been found to be elevated in COVID-19 cases, respectively. Elevated alkaline phosphatase (ALP) level was observed in only one out of 56 (1.8%) patients during hospitalization. Biopsy analysis of three patients infected with SARS-CoV suggests direct viral invasion of the virus to the hepatocytes. Limited information exists on the infectivity of SARS-CoV-2 in the liver.

Endocrine involvement:- diabetic ketoacidosis and hyperglycemia. Dysregulation of ACE 2 pathways may cause alterations in glucose metabolism. mild pancreatic injury have been observed in 1–2% and 17% of mild and severe COVID-19, respectively.

Obstetric & gynecologic complications:- Limited evidence suggests that vertical transmission of COVID-19 during late pregnancy is possible. Increased oxygen demand and physiologic anaemia during pregnancy are the potential factors that could exacerbate the severity of COVID-19.

GI tract involvement:- GI symptoms infrequently accompany COVID-19 pneumonia. About 2–10% of patients with COVID-19 had GI symptoms such as diarrhea, abdominal pain and vomiting. Interestingly, diarrhea could be one of the initial presentations of the disease. Epithelial cells of the GI tract are enriched with ACE 2 receptors and SARS-CoV-2 RNA has been detected in stool specimens of the infected patients

Neuromuscular involvement:- Neurologic manifestations suggestive of the central nervous system

(CNS), peripheral nervous system (PNS) and musculoskeletal involvement were reported. dizziness, headache, impaired consciousness, cerebrovascular disease, ataxia and epilepsy In a small number of patients (2–10%), hypogeusia, hyposmia and neuralgia were detected Epithelial cells of the nasal and oral cavity are enriched with ACE 2 receptors.

Central nervous system involvement:- More than three-fourths (88%) of the patients with severe COVID-19 displayed neurologic manifestations, including acute cerebrovascular diseases and encephalopathy. lymphopenia, elevated ferritin and LDH levels were not worthy

The virus is believed to enter the CNS via the systemic circulation in the case of severe infection.

Skin involvement Cutaneous changes due to COVID-19 infection are infrequently observed. Several skin conditions, including erythema, papules, maceration and scaling accompanied with symptoms of burning, itching and stinging, are mostly related to personal protective equipment and personal hygiene measures. Recently, a report of skin rash with petechiae was described as a possible early sign of COVID-19 in Thailand widespread urticaria (16.67%) and chickenpox- like vesicles (5.56%). These skin lesions were either along with mild itching or pruritus was absent.

Multiorgan failure :-After the internalization, SARS-CoV-2 replicates over a few days, and the innate immunity fails to combat the virus.

Kawasaki-like syndrome :-The first case of Kawasaki-like syndrome associated with COVID-19 was a 6-month-old infant who presented with fever, fussiness, maculopapular rash, conjunctivitis, swelling of the extremities and tongue papilla. The result of RT-PCR was positive for COVID-19. These symptoms occurred 3 weeks after her probable exposure to SARS-CoV-2 [16]

The four main types of COVID-19 vaccine

There are four categories of vaccines in clinical trials: WHOLE VIRUS, PROTEIN SUBUNIT, VIRAL VECTOR and NUCLEIC ACID (RNA AND DNA). Some of them try to smuggle the antigen into the body, others use the body's own cells to make the viral antigen.

WHOLE VIRUS :- Many conventional vaccines use whole viruses to trigger an immune response. There are two main approaches. Live attenuated vaccines use a weakened form of the virus that can still replicate without causing illness. Inactivated vaccines use viruses whose genetic material has been destroyed so they cannot replicate, but can still trigger an immune response.

PROTEIN SUBUNIT :- Subunit vaccines use pieces of the pathogen - often fragments of protein - to trigger an immune response. Doing so minimises the risk of side effects, but it also means the immune response may be weaker. This is why they often require adjuvants, to help boost the immune response. An example of an existing subunit vaccine is the hepatitis B vaccine.

NUCLEIC ACID :- Nucleic acid vaccines use genetic material either RNA or DNA to provide cells with the instructions to make the antigen. In the case of COVID-19, this is usually the viral spike protein. Once this genetic material gets into human cells, it uses our cells' protein factories to make the antigen that will trigger an immune response. The advantages of such vaccines are that they are easy to make, and cheap. Since the antigen is produced inside our own cells and in large quantities, the immune reaction should be strong.

VIRAL VECTOR :- Viral vector vaccines also work by giving cells genetic instructions to produce antigens. You may get Johnson & Johnson's COVID-19 vaccine in some situations.

- **Pfizer** – A COVID-19 vaccine made using mRNA technology. Two doses are needed. The vaccine is recommended for people 5 years of age and older.
- **Moderna** – A COVID-19 vaccine made using mRNA technology. Two doses are needed. The vaccine is recommended for people 18 years and older.
- **Johnson & Johnson (also known as J&J and Janssen)** – A COVID-19 vaccine made using viral vector technology. One dose is required. This vaccine is authorized for people 18 years and older.

SCOPE OF THE STUDY

Coronavirus disease 2019 (COVID-19) is a communicable disease caused by novel severe acute respiratory syndrome coronavirus 2 (SARS- CoV-2). COVID -19 has spread throughout the world, leading to a global pandemic. The World Health Organisation (WHO) consequently declared COVID-19 as a public health emergency of international concern. Importantly after viral infection different types of damage occur in different body organs. In addition peripheral

and central inflammatory responses may be triggered by the infection and lead to long lasting problems in the individuals. the goal of our present study was to investigate and characterize the clinical outcomes which appear after eradication of the coronavirus and its relation to disease severity. Finally with this study, we like to conclude that the presence of clinical outcomes after recovery from COVID-19. The prevalence of the clinical outcomes vary the age.

AIM AND OBJECTIVES

Aim of the study:

To evaluate the clinical outcomes in the post COVID -19 patients, their prevalence and the severity of the outcomes in the effected individuals

Objectives of the study:

- ❑ To assess the prevalence of impaired health status and physical health symptoms among individuals after SARS CoV- 2 Infections.
- ❑ To evaluate the severity of the outcomes in different individuals based upon their age and gender of the individual.
- ❑ To assess the quality of life in the affected individuals.
- ❑ To evaluate the prevalence of outcomes based upon their vaccination status.
- ❑ To create awareness among the health care individuals and patients.
- ❑ **MATERIALS AND METHODS**
- ❑ **STUDY DESIGN:** Retrospective observational study, which involves active observation of patients recovered from COVID -19 .
- ❑ **STUDY DURATION:** 6 months duration i.e., from November 2021 to April 2022.
- ❑ **STUDY SITE:** Government general hospital, Eluru Sir. C. R. College of Pharmaceutical sciences.
- ❑ **POPULATION SIZE:** The study population size is 200.

SOURCES OF DATA

We used the case report form (CRF) and google form for collecting the data from the patient for the project.

- Patient demographic details
- Chief complaints of COVID
- Past and present medical and medication history
- Social history
- Vaccination details
- Laboratory investigation for COVID positive reports

→ Data collection occurred between November 2021 to April 2022. Of 211 individuals with COVID were studied. 200 were eligible for recruitment. The reason for exclusion were 7 (n=7) individuals indicated that they did not wish to participate in the study, while remaining participants (n=4) because they had not given required data for our work. The participant recruitment flow diagram is presented in plan of work.

DATA COLLECTION PROCESS : Patient specific proforma was designed, the patients consent is taken by oral questioning and required data for the study is collected. (Annexure-I) By interacting with patient post COVID clinical features were assessed.

DATA MANAGEMENT AND STUDY PROCEDURE: The obtained patient specific data was entered into collection form

STATISTICAL PLAN:

All the data was recorded in Microsoft excel statistics were carried out using anovatest

RESULTS

1.Distribution based on age of patients (n=200)

S.No	Age group (in years)	No of patients (n=200)	Percentage (%)
1.	11-20	29	14.5
2.	21-30	96	48
3.	31-40	52	13.5
4.	41-50	18	9
5.	51-60	12	6
6.	61-70	10	5
7.	71-80	4	2
8.	81-90	4	2

Table 7.1 : Distribution based on age of patients

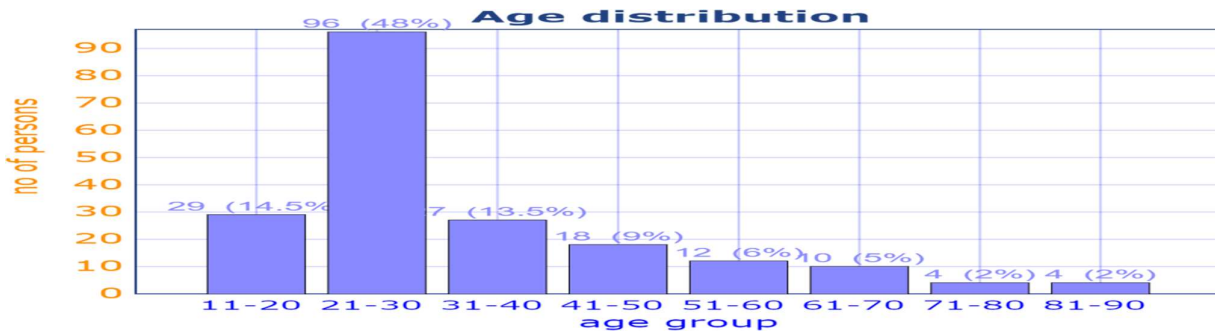


Fig: 7.1 Distribution based on number of patients

2.Distribution based on gender of patients (n=200)

S.No	Age group	Male	Male %	Female	Female %
1.	10-20	8	4	21	10.5
2.	21-30	55	27.5	41	20.5
3.	31-40	13	6.5	17	8.5
4.	41-50	14	7	4	2
5.	51-60	11	5.5	1	0.5
6.	61-70	9	4.5	1	0.5
7.	71-80	3	1.5	1	0.5
8.	81-90	2	1	2	1
	Total	115	57.5	85	42.5

Table :7.2 Distribution based on Gender of patients(n=200)

Gender

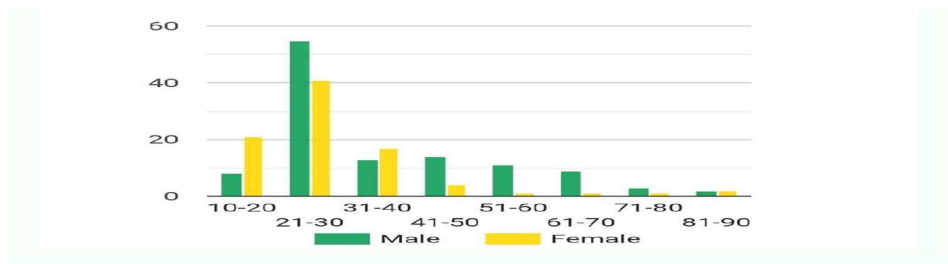
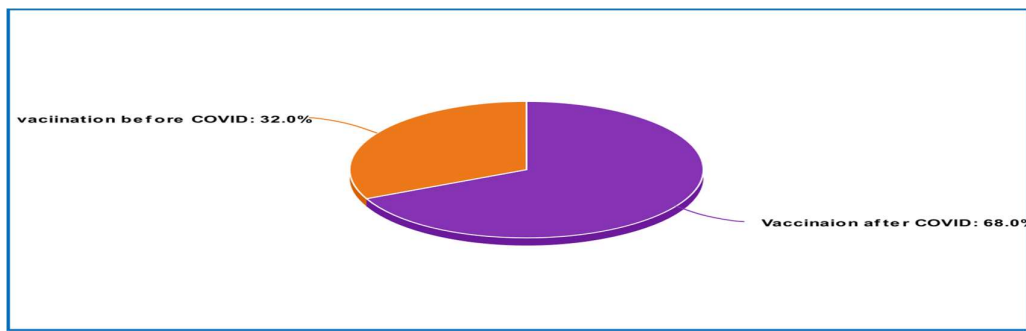


Fig:7.2 :Distribution based on Gender of patients (n=200)

3. Distribution of patients based on COVID effected before or after vaccination

S.No	Age group	Vaccinated before COVID	Vaccinated after COVID
1	11-20	8	21
2	21-30	35	61
3	31-40	5	22
4	41-50	6	12
5	51-60	4	8
6	61-70	3	7



S.No	Age group	Vaccinated before COVID	Vaccinated after COVID
7	71-80	1	3
8	81-90	2	2

Table: 7.3 Distribution of patients based on COVID effected before or after vaccination

Fig:7.3 Distribution based on vaccination

4. Distribution of patients based on type of vaccination

S.No	Age group	covishield	Covaxin
1	11-20	21	8
2	21-30	71	25
3	31-40	17	10
4	41-50	7	11
5	51-60	9	3
6	61-70	7	3
7	71-80	3	1
8	81-90	3	1

Table: 7.4 Distribution of patients based on type of vaccination

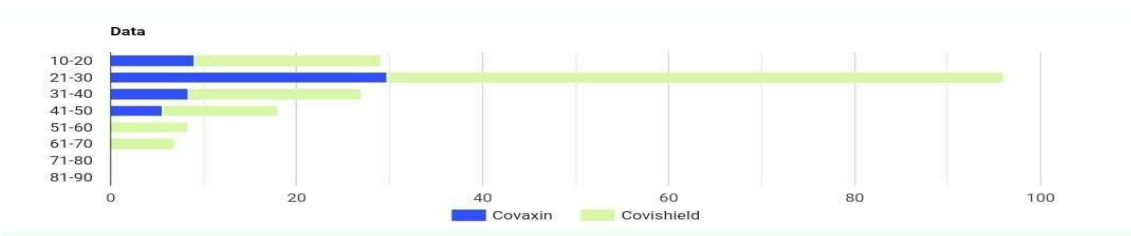


Fig : 7.4 Distribution of patients based on type of vaccination

5. Distribution of patients based on their past medical history

Table: 7.5 Distribution of patients based on type of vaccination

S.No	Age group	With past medical history	Without past medical history
1.	10-20	2	27
2.	21-30	9	87
3.	31-40	4	23
4.	41-50	2	16
5.	51-60	2	10
6.	61-70	3	7
7.	71-80	2	2
8.	81-90	2	2

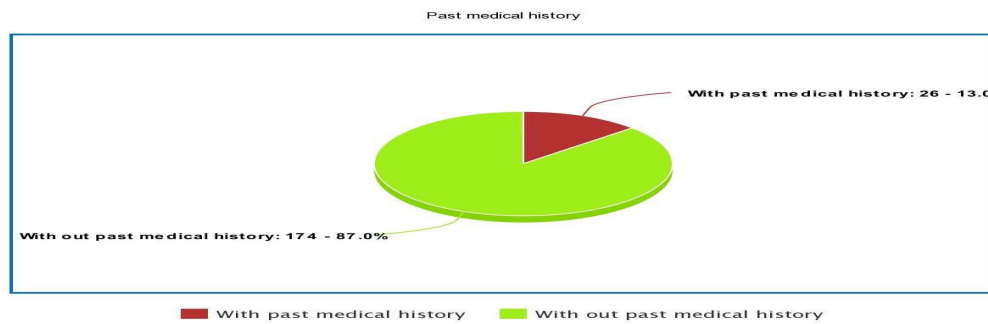


Fig : 7.5 Distribution of patients based on type of vaccination

6. Distribution of patients based on post COVID clinical outcomes

S. No	Age group	Head ache	Pulmonary	Muscle, joint pains	Loss of smell and taste	Weight gain	Weight loss	Hair fall	Fatigue	Gastric problems	CNS	Psychological problems	Cold	Skin infections	Menstrual problems	Stroke
1	11-20	15	9	9	14	2	4	15	13	9	3	1	6	2	2	0
2	21-30	44	33	47	42	10	16	24	40	16	9	21	22	5	5	1
3	31-40	15	15	15	12	8	7	12	20	7	2	8	3	3	4	1
4	41-50	14	14	13	12	5	7	11	17	9	7	8	4	3	2	1
5	51-60	8	9	10	7	4	5	6	10	10	4	3	5	2	1	1
6	61-70	9	10	9	10	3	2	2	8	6	5	5	2	3	0	2
7	71-80	2	2	3	4	2	1	2	4	4	4	2	2	3	0	3
8	81-90	2	4	4	4	2	2	4	4	4	4	2	2	1	0	1
9	Total	79	96	77	105	36	43	76	116	65	38	50	46	19	14	10

Table: 7.6 Distribution of patients based on post COVID clinical outcomes

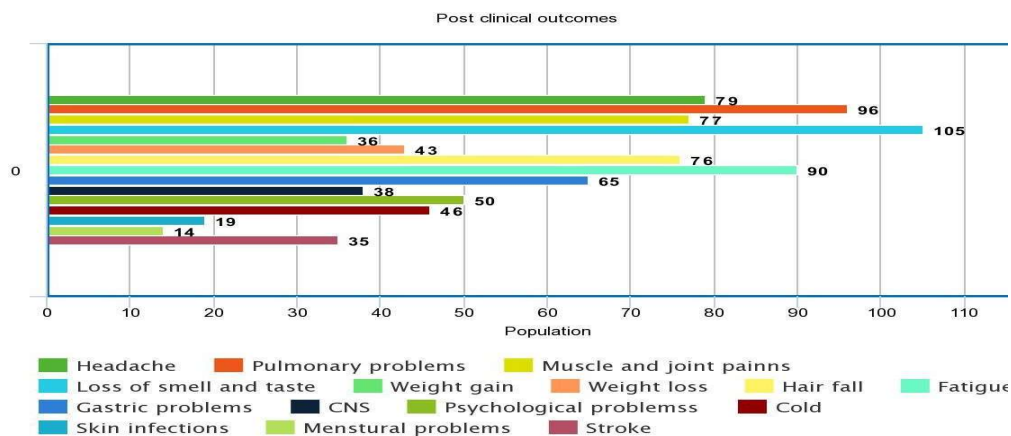


Fig :7.6 Distribution of population based on prescence of post clinicaoutcomes

Groups	N	Mean	Std. Dev.	Std. Error
Group 1	8	13.625	13.3624	4.7243
Group 2	8	12.125	9.4179	3.3297
Group 3	8	13.75	14.028	4.9597
Group 4	8	13.125	12.2526	4.3319
Group 5	8	4.5	3.0237	1.069
Group 6	8	5.5	4.8107	1.7008
Group 7	8	14.5	11.7838	4.1662
Group 8	8	8.125	3.9074	1.3815
Group 9	8	4.75	2.252	0.7962
Group 10	8	6.25	6.5411	2.3126
Group 11	8	5.75	6.7348	2.3811
Group 12	8	2.75	1.165	0.4119
Group 13	8	1.25	0.8864	0.3134
Group 14	8	1.75	1.9086	0.6748
Group 15	8	9.5	7.597	2.6859

ANOVA Summary

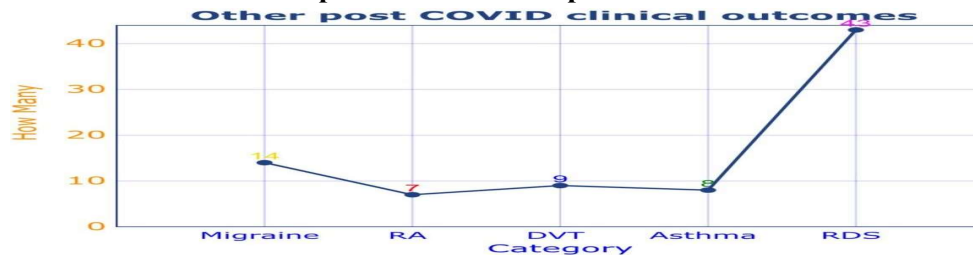
source	Degrees of Freedom DF	Sum of Squares SS	MeanSquar eMS	F-Stat	P-Value
Between Groups	14	2410.9667	172.2119	2.6984	0.002
Within Groups	105	6701.016	63.8192		
Total:	119	9111.9826			

7. Distribution of patients with other post COVID clinical outcomes

S.No	Age group	Migraine	Rheumatoid arthritis	DVT	Asthma	Respiratory distress syndrome
1	11-20	0	0	0	0	0

2	21-30	5	0	0	3	22
3	31-40	1	0	1	1	2
4	41-50	1	1	1	1	7
5	51-60	2	1	2	0	3
6	61-70	3	3	2	2	5
7	71-80	1	1	1	0	2
8	81-90	1	1	2	1	2
	Total	14	7	9	8	43

Table : 7.7 Distribution of patients with other post COVID clinical outcomes



DISCUSSION

In our present study, a total of 200 post COVID patients with different co-morbid conditions were observed. In our study with a total of 200 population with different age groups. Age with 11-20 years were 14.5% (n=29), 21-30 years are 48% (n=96), 31-40 years are 13.5% (n=27), 41-50 years are 9% (n=18), 51-60 years are 6% (n=12), 61-70 years are 5% (n=10), 71-80 years are 2% (n=4), 81-90 years are 2% (n=4). In this study population 32% (n=64) were vaccinated before COVID-19 occurrence, and 68% (n=136) are vaccinated after the COVID-19 occurrence. Almost 69% (n=138) of population has been vaccinated with COVISHIELD whereas 31% (n=62) population has been vaccinated with COVAXIN. Based on our study we observed we observed different clinical outcomes in the post COVID-19 patients. Which include Headache 39.5%, Pulmonary problems 48%, Muscle and Joint pains 38.5%, Loss of Smell and Taste 52.55, Weight gain 18%, Weight loss 21.5%, Hair fall 38%, Fatigue 45%, Gastric problems 32.5%, CNS problems 19%, Psychological problems 25%, Increased frequency of Cold 23%, Skin infections 9.5%, Menstrual problems 7%, Stroke 17.5%, and other severe problems include Migraine 7%, Rheumatoid Arthritis 3.5%, Deep vein thrombosis 4.5%, Asthma 4%, Respiratory Distress Syndrome 21.5%. Although many COVID-19 patients eventually recover, some do not cease experiencing clinical outcomes long after their COVID-19 polymerase chain reaction test turns negative. We observed the presence of different complications in the population. But their severity varies with presence other comorbid condition's Viral infection leads to an aggressive immunological reaction, which directly and indirectly compromises the cardiopulmonary system. However, haematological changes caused by vascular inflammation bring about a microenvironment for thromboembolism formation, affecting other vital organs, such as the nervous system, gastrointestinal tract, liver, and kidneys. late viral infection has been related to other disorders.

CONCLUSION

This study showed that from the collected data the clinical outcomes varies with the following factors

Males are more effected than females. The infection of COVID-19 varies with their vaccination history. The occurrence of COVID-19 is more in population who have

not been vaccinated. The occurrence of the post COVID clinical outcomes varies in different age groups. Patients with other comorbid conditions like Immune disorders, Hypertension, Diabetes mellitus are more prone to post COVID clinical outcomes. The serious post COVID clinical outcomes like Respiratory distress syndrome, Deep vein thrombosis, Migraine are mostly seen in individuals with age group of greater than 40 years.

POST COVID-19 COMPLICATIONS

SUBJECTIVE DATA:

Patient name:

Age/Gender:

Area(rural/urban):

Covid occurrence:

Number of times:

Chief complaints in covid:

Past medical history:

Past medication history:

Vaccination: Yes/No

Doses:

Type:Covid after or before vaccination:

PERSONAL HISTORY AND HABITS :

Height:

Weight:

BMI:

Education:

Occupation:

Diet

(veg/non-veg):

Alcoholi

c/Smoker:PHYSICAL EXAMINATION

SNo	Parameter	Observed
1	Temp(F)	
2	BP(mmof Hg)	
3	RR(cycles/min)	
4	PR(beats/min)	

Diagnosis:

Rapid /CT

scan:

Grading

/Ratio

COMPLICATIONS:

→ **Head ache:**

→ **Vision problems:**

→ **Hearing problems:**

→ **Lungs(SOB, Chest pain, Phlegm):**

→ **Taste:**

→ **Smell:**

→ **Muscle/Joint aches:(walking difficulty)**

→ **Loss of appetite:**

→ **Physical weakness: fatigue**

→ **physiological condition(Anxiety/Depression):**

→ **Gastric problems (ulcers, diarrhoea, constipation):**

→ **Numbness:**

→ **Oedema:**

→ **Frequency of flu:**

→ **Skin infections:**

→ **Fat in stomach**

- CNS
- Dementia:
- Hair fall:
- Weigh loss /weight gain:
- Menstrual problems:
 - Any surgery after covid:
 - Covid medications:
 - Non pharmacological therapy:

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