
**“SIGNIFICANCE OF NEUTROPHIL TO LYMPHOCYTE
RATIO AND PLATELET TO LYMPHOCYTE RATIO
FOR PREDICTING CLINICAL OUTCOME AND
SEVERITY IN COVID-19 PATIENTS ADMITTED IN
KLE’S DR. PRABHAKAR KORE HOSPITAL,
BELGAVI, KARNATAKA”**

BY

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Submitted to

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In

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KAHER, BELAGAVI – 590010
KARNATAKA.**

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KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH,
BELAGAVI, KARNATAKA.


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DR. REKHA S PATIL MD
Professor and Head,
Department of General Medicine,
J. N. Medical College,
Nehru Nagar, Belagavi – 10

Date : 30/12/2022
Place : Belagavi




DR.(Mrs.) N.S. MAHANTSHETTI MD
Principal **PRINCIPAL**
J.N. Medical College,
BELAGAVI- 590 010
J. N. Medical College,
Nehru Nagar, Belagavi – 10

Date : 2/1/2023
Place : Belagavi

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Accredited 'A+' Grade by NAAC (3rd Cycle)

Placed in Category 'A' by MHRD (GoI)

Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA

0831 - 2471350



0831 - 2470759



www.jnmc.edu



principal@jnmc.edu

Ref No: MDC/PG/

Date: 14-12-2022.

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Principal,
J. N. Medical College, Belagavi.

To,
Reg. No. BG0120011,
Postgraduate Student,
2020-21 Batch,
Department of General Medicine,
J. N. Medical College, Belagavi.

ETHICAL CLEARANCE CERTIFICATE



K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)

Accredited 'A' Grade by NAAC (2nd Cycle)

Placed in Category 'A' by MHRD (GoI)

JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)

Website: <http://www.jnmc.edu>
E-Mail : dome@jnmc.edu

Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/

Date: 25/01/2021

To,
REG NO: BG0120011
PG student in Medicine,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

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(Dr. Smita Sonoli)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Harsha Hegde)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

ABBREVIATIONS

ACE	ANGIOTENSIN CONVERTING ENZYME
ARDS	ACUTE RESPIRATORY DISTRESS SYNDROME
CBC	CELL BLOOD COUNT
CDC	CENTER FOR DISEASE CONTROL
CFR	CASE FATALITY RATE
CRP	C-REACTIVE PROTEIN
CT	COMPUTED TOMOGRAPHY
LDH	LACTATE DEHYDROGENASE
MERS	MIDDLE EAST RESPIRATORY SYNDROME
NLR	NEUTROPHIL LYMPHOCYTE RATIO
PLR	PLATELET LYMPHOCYTE RATIO
SARS	SEVERE ACUTE RESPIRATORY SYNDROME
WHO	WORLD HEALTH ORGANISATION

ABSTRACT

Background: Blood cell Interactions are essential in the pathophysiology of immune responses; inflammation and haemostasis. Study aimed to find the Significance of neutrophil to lymphocyte ratio and platelet to lymphocyte ratio for predicting clinical outcome and severity in COVID 19 patients admitted in Dr. Prabhakar Kore Hospital; KLE university belagavi”

Methodology: The present hospital based cross sectional study was conducted among the patients admitted and getting treated for COVID-19 infection aged more than 18yrs of age. Patients admitted at KLES Dr Prabhakar Kore hospital & MRC with Covid19 lab confirmed patients Consenting for the study were enrolled and Informed consent was taken. A detailed history was documented and clinical features assessed. 15cc of venous blood was drawn and sent for basic laboratory tests complete blood counts including Neutrophil count; lymphocyte count platelets count, PT/INR renal function tests.

Result: In the present study, the mean age of patients was found to be 58.20 ± 15.20 yrs of age, majority were in age of 50-80yrs. There was male preponderance with 76.3% and female 23.70% with male to female ratio of 3.21:1. The majority of patients are slightly symptomatic to asymptomatic, with a mean age of 37.7 years, male (41.9 percent), and female (58.1 percent). There is higher mean of NLR and PLR among the patients included in the present study.

Conclusion: There is a significant higher NLR and PLR in predicting the outcome of the patients. The mean level of the NLR and PLR was higher among the patients with correlating to other markers. NLR and PLR are easily measurable, available, cost-

effective and reliable parameters which can be used in continuous monitoring which could help to predict the outcome in the treatment of COVID -19.

Keyword: COVID-19, Mortality, Neutrophil-lymphocyte, Platelet-lymphocyte, C-reactive protein.

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INTRODUCTION

Since December 2019, an unexplained pneumonia emerged in Wuhan China and soon spread globally, the etiology was thought to be a new virus and named as CORONA VIRUS disease or COVID-19 by WHO based on phylogeny, taxonomy and established problem, this virus was identified as severe acute respiratory syndrome virus by the corona virus study group. SARSCOV-2 is most probably originated from Bat and have the ability to spread from person to person causing the pneumonia and severe respiratory distress syndrome with a typical findings on imaging.

CORONA VIRUS has become a great cause of mortality and morbidity. Although majority of patients are mild, may deteriorate to ARDS , MODS also mortality. Thus, finding high risk factors for the progression of Corona virus disease is necessary to regress the progression of the disease.

Earlier studies have shown that patients in geriatric age group with comorbidities are most likely to progress and a distorted immune inflammatory response may be a major factor in the severity of the illness..² Studies have been done showing the change in CBC findings like neutrophilia, lymphopenia and thrombocytopenia in infected individuals of severe Covid-19 versus patients having mild infection.

Low lymphocyte percentage, low lymphocyte count, high neutrophil percentage, and high neutrophil count are signs of severe COVID-19 suggesting the viral load and immune state. A study done by Huang C et al. and Yang X stated in their articles that 85% of the critically ill patients of their study group with COVID-19

were found with lymphopenia Therefore, lymphopenia as an indicator of severe COVID-19 was confirmed.^{3,4} So various studies have been Showing Neutrophil to lymphocyte ratio and Platelet to lymphocyte ratio higher in Patients with severity. Therefore; a study needs to be conducted to evaluate the clinical signs and symptoms and the Neutrophil to Lymphocyte ratio and Platelet to lymphocyte ratio and correlate the same with severity of Covid-19 infection.

Blood cell Interactions are essential in the pathophysiology of immune responses; inflammation and haemostasis. Studies suggested that Neutrophil to lymphocyte ratio and Platelet to lymphocyte ratio are inflammatory markers of immune mediated; metabolic; prothrombotic and neoplastic etiology. So we can analyse antibody easily available marker for prognostication of patient affected with COVID 19.

AIMS & OBJECTIVES

Aim

Significance of neutrophil to lymphocyte ratio and platelet to lymphocyte ratio for predicting clinical outcome and severity in COVID 19 patients admitted in Dr. Prabhakar Kore Hospital; KLE university belagavi”

Objective

Primary objective is to see the Neutrophil to lymphocyte ratio and Platelet to lymphocyte ratio of Covid-19 infected Patients.

Secondary objective is to correlate the Neutrophil to lymphocyte ratio; Platelet to lymphocyte ratio and the clinical severity of Disease.

REVIEW OF LITERATURE

Coronaviruses are emerged as an major health issue in both humans as well as animals.⁵ Towards the end of 2019, a cluster of pneumonia patients in Wuhan, a city in China's Hubei province, were discovered to be the result of a newly discovered virus. It spread quickly, resulting in an epidemic in China and a significant rise in number of cases in other countries.⁶ In February 2020, the World Health Organization declared COVID-19 as the coronavirus disease of 2019. The severe acute respiratory syndrome coronavirus type 2 (SARS-CoV-2) that causes COVID-19 was formerly known as 2019-nCoV. On March 11 of 2020, the WHO proclaimed COVID-19 to be a pandemic.⁷

Epidemiology

The first case of SARS CoV-2 infection was reported on December 31, 2019, in Wuhan, Hubei Province, China.

Subsequent to this, it rapidly progressed to various regions of Republic of China and globally , affecting almost 185 countries by April 2020, resulting in the present global pandemic. COVID-19 was labeled a Public Health Emergency of International Concern by the World Health Organization on 30 January 2020, and it was recognized as a pandemic on 11 March 2020.

The COVID 19 basic reproduction number (R0) is predicted in between 1.4 and 3.9, suggesting its extreme infective nature. In public gathering places without personal protective equipment, such as hospitals, cruise ships, and religious, political, academic, and business groups, the Reproduction rate is higher. Comparable to Covid

19 and MERS CoV, the incubation period is 5–6 days, and the serial interval is predicted to be 8 days.⁸ At first, it was discovered that the CFR was between 0.9% and 3%, which was lower than the prior HCoV vs SARS Covid 19 (6 percent to 17 percent) and MERS CoV (20 percent –40 percent). However, by May 24, 2020, the CFR has dramatically risen throughout a number of nations.

Unlike SARS CoV, a large proportion of covid 19 infected people are asymptomatic or pauci-symptomatic, allowing them to avoid identification and become potential carriers.⁹ It is significant to note that not all close contacts contract the illness, which suggests that each person's genetic predisposition plays a part.^{10,11} The virus normally enters humans through the upper aerodigestive tract. Also, covid 19 was first found in patients' excreta, indicating the possibility of fecal-oral transmission.^{12,13}

Infection with Covid 19 in pregnancy increases the risk of transmission to the child. Mother to child transmission was unlikely, however, due to negative virus testing on swabs recovered from the six pregnant women from amniotic fluid, cord blood, neonate throat, and breast milk.¹⁴ Stretched distance aerosol transmission is hypothesized, which is dependent by the dynamics of the virus's flow from the diseased individual as well as the ventilation condition of the area.¹⁵ Furthermore, mapping techniques like as cartograms can be used to depict the dissemination and expansion of SARS COV2.¹⁶ Understanding the pathways of transmission of COVID 19 will allow for the use of appropriate containment measures.

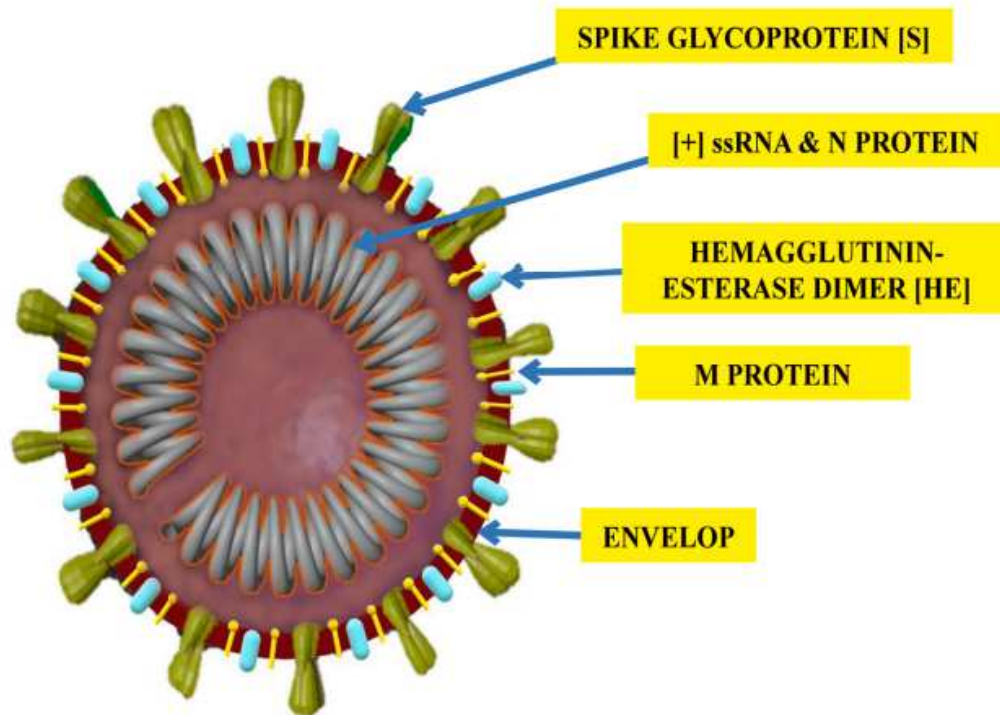


Figure 1: COVID-19 virus structure

Despite the fact that all age groups are at risk to COVID infection, the disease severity, death are lower in children.¹⁷ Women are less typically impacted than males in adulthood due to higher plasma ACE2 levels.¹⁸ The older population, in particular, with diseases like diabetes mellitus, systemic hypertension, CVA, and other pulmonary/cardiac/renal disease, is especially prone to critical infections due to insufficient immune response against infection and other issues.^{19,20} A recent study found that, similar to SARS CoV, non-O blood groups, notably A group, was associated with higher infection and death rates due to a lack of protective antibodies. Numerous uncertainties remain in the COVID-19 epidemiology, particularly the virus-host interaction, including host vulnerability and epidemicity.^{21,22}

The corona viruses (CoVs) can be divided into two categories: and (found in both mammals and humans); and (in avian species). Spike protein which is on its envelope, resemble a crown under an electron microscope, hence the name. Infections of the respiratory, gastrointestinal, hepatic, and central nervous systems in people and other mammals are brought on by them.

Thr estimations in general , imply that roughly 2% of the population is a carrier of CoVs, and they cause approximately around 5%–10% of acute respiratory tract infections. The hypothesis says that when these viruses transfer to humans from host via an intermediary amplifying host, they go through a speedy mutation and recombination, resulting in the emergence of covid viruses that are dangerous for humans . The majority of the time, CoVs affected are sometimes linked to various respiratory tract infections, yet they can sometimes be the only pathogen that infects children with common long-term illnesses.. As a result, CoVs have emerged as significant pathogens in rising respiratory illness outbreaks.

Pathogenesis

The highly glycosylated SARS CoV-2 S1 fraction with receptor binding domain (RBD), which binds to the angiotensin-converting enzyme 2 receptor (ACE-2 R) with 10–20 fold higher affinity than SARS CoV, is thought to infect human cells. The viruses SARS CoV and SARS CoV-2 are closely related. The primary source of ACE-2 R is type II rather than type I human alveolar epithelial cells. Endothelial cells, GIT epithelial layers, and cardiac myocytes all express the ACE-2 With SPRR insertion on the spike protein, transmembrane protease serine (TMPRSSs) expressed on host cells recognise and break the distinctive polybasic S1/S2 protease cleavage site, exposing the fusion protein (S2 part), allowing the viral and host cell membranes

to converge. It has been demonstrated that upper esophageal epithelium, absorptive enterocytes, and type 2 pneumocytes all considerably co-express TMPRSSs and ACE-2 R, suggesting that SARS CoV-2 can enter the host through these tissues in addition to alveolar epithelium.

Therefore, the tissues that could serve as viral targets should express both TMPRSSs and ACE-2 R. Viral RNA can internalise into the cytoplasm of the host cell thanks to this membrane fusion, where it is copied and translated to produce new viral proteins. The final phase before virions can infect other cells is viral assembly, which takes place right before virions are discharged from infected cells. During this stage, (N) proteins bind to RNA before being encased in an envelope and membrane protein.⁸

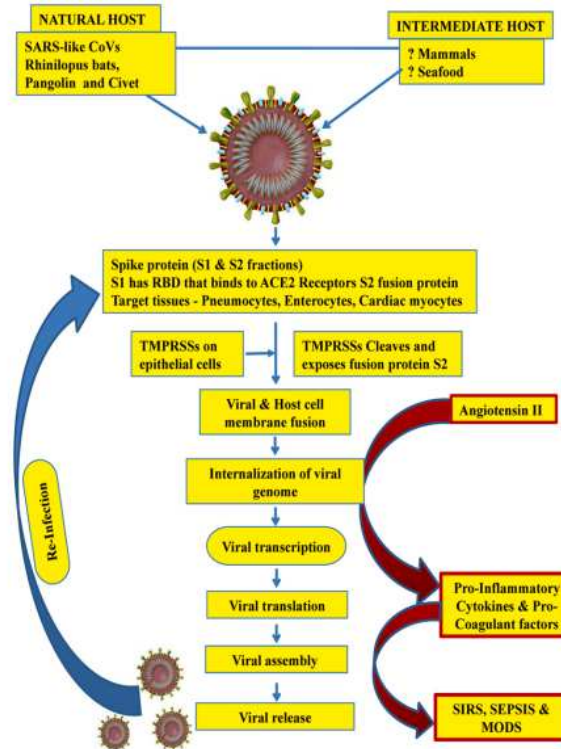


Figure 2: Pathogenesis of COVID-19 infection

Type II pneumocytes, which are in charge of tissue regeneration and surfactant production, are destroyed as a consequence of covid infection, increasing surface tension thereby causing dyspnea. Furthermore, the enhanced cytokine production and release (cytokine storm) by activated inflammatory cells brought on by angiotensin II buildup compromises the alveolar immunologic equilibrium mechanism in these damaged type II pneumocytes.⁸

Transmission

Observational studies of home transmission were conducted prior to the discovery of variants, and the SAR in paediatric contacts (upto 18 years) ranging from 4 to 57 %.²³⁻²⁸ A meta-analysis of 87 home transmission studies with 1,249,163 household links from different c found regions of world found that the SAR was 18 percent in contacts who were under the age of 18 and 30% among contacts who were adults. More transmissible variations result in higher household transmission rates. Children under the age of 18 had a secondary attack rate of 72-75%, whereas adults suffered 90% of these attacks, according to a study of familial clusters carried out during the dominant circulation of the Alpha (B.1.1.7 lineage) variant..^{29,30}

A case-control study found that SARS-CoV-2 infection in kids and teenagers was associated with contact with people infected with COVID-19 in social gatherings.³¹

There have also been stories of epidemics linked to health care, as well as instances where children or students may have contracted a disease from teachers or other school personnel. Regular use of preventative measures at school was connected

in a case-control study to SARS-CoV-2 infection, although regular attendance at school was not.³¹

Children with confirmed asymptomatic infection have been reported to transmit the virus to family members, despite the fact that nothing is known about transmission by truly asymptomatic children. 36 Additionally, there have been cases of familial clusters involving asymptomatic children as well as a possible channel of transmission from asymptomatic children to adults who are not family members. 36–39 such results indicate that asymptomatic children might contribute to transmission. 40,41. Asymptomatic transmission in adults is well known.

Clinical features

Although they occur at different frequencies in children and adults, COVID-19 symptoms are similar in both. COVID-19 affects children lesser compared to adults; however severe instances in children have been documented.

The spectrum of infection severity:

Mild disease (no or mild pneumonia) was reported in 81 percent.

Severe disease (eg, with dyspnea, hypoxia, or >50 percent lung involvement on imaging within 24 to 48 hours) was reported in 14 percent.

Critical disease (eg, with respiratory failure, shock, or multiorgan dysfunction) was reported in 5 percent.

The overall case fatality rate was 2.3 percent; no deaths were reported among noncritical cases.

Comparably, 2% of the 10.3lakh cases reported to (CDC) in the United States by the end of May 2020 were admitted to critical care unit , 5% of those instances resulted in death. Of those cases, 14% were hospitalised. Age, preexisting comorbidities, and immunisation history all affect the likelihood of developing a serious illness..⁴²

Initial presentation:

The most typical reported symptoms with COVID-19 were headache, myalgias, and cough. Other characteristics include having diarrhoea, a sore throat, or abnormalities in taste or smell. The most frequent serious infection-related symptom is pneumonia, which is typically characterised by rise in temperature , coughing, shortness of breath , and bilateral infiltrates on chest imaging.

Symptoms that may be seen in patients with COVID-19⁴³
Cough
Fever
Myalgias
Headache
Dyspnea (new or worsening over baseline)
Sore throat
Diarrhea
Nausea/vomiting
Anosmia or other smell abnormalities
Ageusia or other taste abnormalities
Rhinorrhea and/or nasal congestion
Chills/rigors
Fatigue
Confusion
Chest pain or pressure

Severe complications:

The complications described as

Respiratory failure

Cardiac and cardiovascular complications

Thromboembolic complications

Neurologic complications

Inflammatory complications

Secondary infections

Infection fatality rate

Among cases that have been recorded, the case fatality rate simply shows the mortality rate. There is a lot of heterogeneity by location and across risk groups, however some investigations of unvaccinated persons have estimated the infection fatality rate, or the predicted mortality rate among all individuals with illness, to be between 0.15 and 1 percent. This is due to the fact that many mild infections go untreated and many severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infections are asymptomatic.⁴⁴

A analysis of 27 studies from resource-rich locations found that infection mortality rates rose exponentially with age (0.002 percent at age 10, 0.01 percent at age 25, 0.4 percent at age 55, 1.4 percent at age 65, 4.6 percent at age 75, 15 percent at age 85, and >25 percent at age 90).

Through seroprevalence surveys or extensive tracing programmes, these research assessed the overall number of community infections up to September 2020.. These ageing issues changes appear to account for much of the documented geographic variance in infection fatality rates (i.e., areas with higher median population ages reported higher fatality rates).⁴⁵

Comorbidities associated with risk factors for severe COVID-19^{46,47}

Comorbidities that have been linked to severe COVID-19 in at least one meta-analysis or systematic review (starred conditions), in observational studies, or in case series, constitute established, plausible, and potential risk factors.

- Cancer
- Cerebrovascular accident
- Children with specific underlying illnesses
- Chronic kidney injury
- Long term pulmonary diseases (COPD, ILD, pulmonary thromboembolism, pulmonary artery hypertension, bronchopulmonary dysplasia, bronchiectasis, cystic fibrosis)
- Chronic liver disease (cirrhosis, NAFLD, ethanol use disorder, autoimmune hepatitis)
- Diabetes both, type 1 and type 2
- Down syndrome
- Cardiac manifestations (like heart failure, ischaemic heart disease, or cardiomyopathies)
- Human immunodeficiency virus
- Mental health disorders (mood disorders including depression, schizophrenia spectrum disorders)
- Degenerative CNS manifestations, including Alzheimers
- Obesity (BMI ≥ 30 kg/m²) and overweight (BMI 25 to 29 kg/m²)
- Recent/current pregnancy
- Smoking
- Sickle cell disease or thalassemia
- Solid organ or blood stem cell transplantation
- Substance abuse
- Tuberculosis
- Immunosuppressive medications

Possible risk factors but evidence is mixed (comorbidities have been associated with severe COVID-19 in at least 1 meta-analysis or systematic review, but other studies had reached different conclusions):

- Asthma
- Hypertension
- Other immune deficiencies

Danger signs for serious disease

- The majority of studies evaluating these tools are constrained by the bias risk and have not been sufficiently validated for clinical treatment. Several prediction tools have been proposed to identify patients who are more likely to have critical illness based on epidemiologic, clinical, and laboratory features.
- Advanced age
- Comorbidities
- Socioeconomic background and gender

Laboratory findings

The findings of laboratory are variable. In meta-analysis, the data of following laboratory abnormalities were noted down

- Elevated C-reactive protein: 54%
- Elevated serum ferritin: 47%
- Elevated lactate dehydrogenase: 37%
- Elevated D-dimers: 35%
- Elevated procalcitonin: 21%

- Elevated erythrocyte sedimentation rate: 19%
- Elevated leukocytes: 20%
- Lymphocytopenia: 19%
- Lymphocytosis: 8%
- Elevated serum aminotransferases: 30%
- Elevated creatine kinase myocardial bands: 25%

Table 1: Diagnostic tests for COVID 19

Test category	Primary clinical use	Specimen type	Performance characteristics	Comments
NAATs (including RT-PCR)	Diagnosis of current infection	Respiratory tract specimens*	<ul style="list-style-type: none"> ▪ High analytic sensitivity and specificity in ideal settings. ▪ The kind and quality of the specimen, as well as the severity of the sickness at the time of testing, all affect clinical performance. ▪ Reported false-negative rate ranges from <5 to 40%, depending on the test used.[¶] 	<ul style="list-style-type: none"> ▪ Time to perform the test ranges from 15 minutes to 8 hours.^Δ ▪ Turnaround time is influenced by the test used and laboratory workflow. ▪ Some assays allow home collection of specimens that are mailed in.
Serology (antibody detection)	Diagnosis of prior infection (or infection of at least 3 to 4 weeks' duration)	Blood	<ul style="list-style-type: none"> ▪ Sensitivity and specificity are highly variable. ▪ IgG typically develops 14 days following the onset of symptoms, but detectable antibodies typically take several days to weeks to develop. ▪ There have been 	<ul style="list-style-type: none"> ▪ Time to perform the test ranges from 15 minutes to 2 hours. ▪ Turnaround time is influenced by the test used and laboratory

			<p>reports of cross-reactivity with other coronaviruses.</p> <ul style="list-style-type: none">▪ In environments with low seroprevalence, individual results should be interpreted cautiously because serologic tests with great specificity still have a low positive predictive value.	<p>workflow.</p> <ul style="list-style-type: none">▪ It remains uncertain whether a positive antibody test indicates immunity against future infection.
Antigen tests	Diagnosis of current infection	Nasopharyngeal or nasal swabs	<ul style="list-style-type: none">▪ Antigen tests are generally less sensitive than nucleic acid tests.▪ Within 5 to 7 days following the onset of symptoms, sick people have the highest levels of sensitivity.	<ul style="list-style-type: none">▪ Time to perform the test is <1 hour.

Laboratories features associated with severe COVID-19 infection^{19,20,48,49}

Abnormality	Possible threshold
Elevations in:	
▪ D-dimer	>1000 ng/mL
▪ CRP	>100 mg/L
▪ LDH	>245 units/L
▪ Troponin	>2× the upper limit of normal
▪ Ferritin	>500 mcg/L
▪ CPK	>2× the upper limit of normal
Decrease in:	
Absolute lymphocyte count	<800/microL (normal range for age ≥21 years: 1800 to 7700/microL)

Viral factors

Despite some studies finding no correlation between viral RNA levels and severity of disease, patients having critical illness have greater viral RNA levels in throat swabs as compared to patients having less severe illness. Blood coagulopathy, organ damage, and severe disease death have all been linked to viral RNA in the blood.⁵⁰⁻⁵²

Genetic factors

The potential link of host genetic variables with severe illness is also being investigated. The genome-wide association study connected the polymorphism in the ABO blood group gene to SARSCOV2 respiratory failure. A decreased risk of infection and serious disease has been associated with type O.^{53,54}

Radiological findings

Chest Xray:

Chest Xray can be normal when a disease is just beginning. Retrospective analysis of 64 infected SARS COV2 individuals in Hong Kong revealed that 20% of them never had any abnormal chest radiographs throughout the duration of their illness. Consolidation and ground-glass opacities were frequently observed on abnormal radiographs for distributions in the bilateral, peripheral, and lower lung zones. The disease's lung involvement increased with time, reaching a peak in severity 10 to 12 days following the start of symptoms.

Chest CT

Even while CT Thorax is superior than a chest x-ray and chest CT also can be suggestive of infection, none can completely confirm or rule out the potential of infection.

Ground-glass opacities – 83%

Ground-glass opacifications(GGO) and mixed consolidation – 58 %

Pleural thickening – 52 %

Thickening of interlobular septum – 48 %

Air bronchograms sign – 46 %

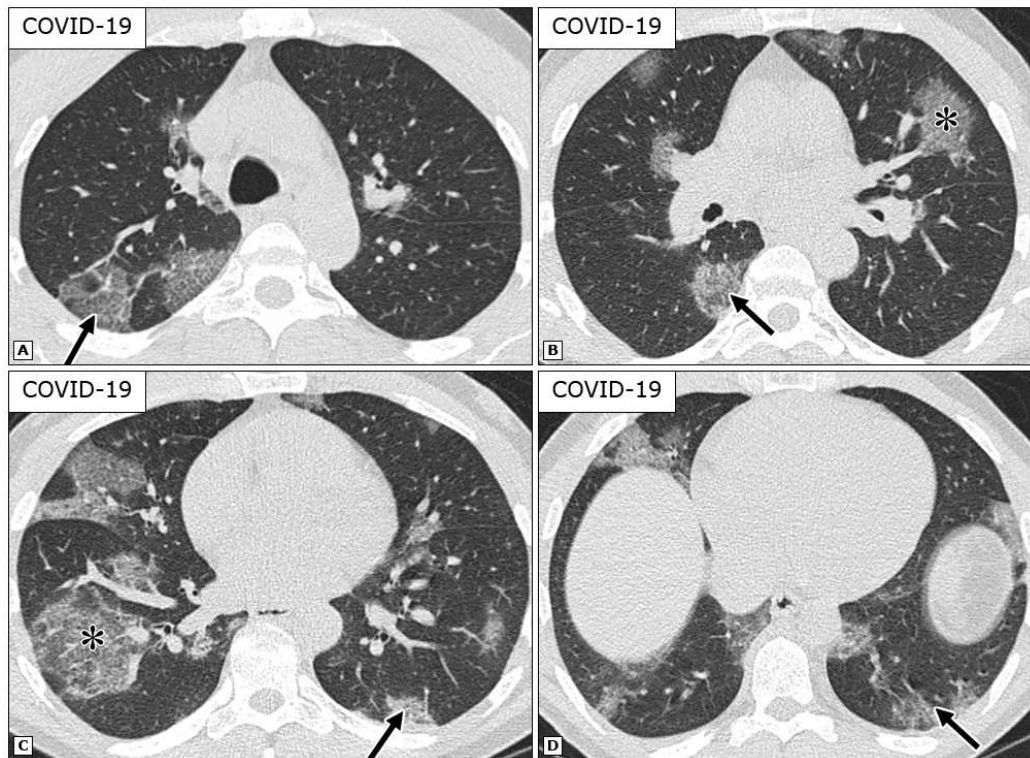


Figure 3: Chest CT findings related to COVID-19⁵⁶

Figure 3 showing: For COVID-19, typical CT imaging characteristics. The lungs of a 52-year-old man with a positive RT-PCR are obvious in unenhanced thin-section axial images (A to D) as having bilateral, multifocal rounded (asterisks) and

peripheral GGO with superimposed interlobular septal thickening and discernible intralobular lines ("crazy-paving"). Most professional organisations including the US Centers for Disease Control and Prevention do not currently advise routine CT screening for the diagnosis or exclusion of COVID-19.⁵⁶

Other less common abnormalities included bronchiectasis, pleural effusion, pericardial effusion, lymphadenopathy, and a crazy paving pattern .

COVID-19 chest CT abnormalities frequently involve the lower lobes, are bilateral, and have a peripheral distribution.

Lung Sonography:

One method for assessing lung involvement in individuals with probable COVID-19 is lung sonography. In addition to discrete, multifocal, or confluent B lines visible under the pleura, patchy, and nodular consolidations, also air bronchogram signals inside the consolidations, patients with COVID-19 exhibit pleural line thickening, discontinuing, and disruption.^{57,58} [188-190]. Despite the fact that ultrasound seems to be somewhat sensitive for diagnosing COVID-19, other investigations have found that it is not very specific.

Role of inflammatory markers like NLR and PLR in COVID-19

Neutrophil Lymphocyte ratio (NLR)

In normal circumstances, the human blood contains 4000-11000 WBCs per microlitre. The most numerous are these granulocytes. Granulocytes have horseshoe-shaped nuclei that become multilobed as they age. The majority of them have neutrophilic granules.

Biologically active chemicals that contribute to inflammatory responses are found in the cytoplasmic granules of neutrophils. The typical half-life of a neutrophil in the blood is 6 hours. They roll along the endothelium surface after being drawn there by selectins. Selectins can be bound to neutrophil adhesion molecules from the integrin family. They enter the capillary walls by a process called diapedesis. The GI tract absorbs a large portion of those that bypass the circulatory system and eliminates them from the body.

Numerous proteases, as well as enzymes like NADPH oxidase, catalase, and myeloperoxidases, are present in neutrophilic granules. The "Respiratory burst," a rapid rise in oxygen consumption and metabolism in neutrophils associated with NADPH oxidase, results in a significant release of free O-radicals. The enzyme myeloperoxidase catalyses the transformation of cyanides and halides into acid forms. These acids themselves act as strong oxidants.

Neutrophil granules contain two metalloproteinases, an elastase, myeloperoxidase, and NADPH oxidase. Total body neutrophils come in two different varieties: circulation pool (CGP) and marginating granulocyte pool (MGP). The cells of these two pools are of identical size and are always in an equilibrium state. MGP, which indicates neutrophils involved in adhesion and rolling along endothelial cells in postcapillary venules, is not present in blood collected after venepuncture. As a result, the neutrophil content contains almost half of the vascular compartment's neutrophils.

Lymphocytes are movable, non-phagocytic cells. Numerous lymphocyte subpopulations communicate with one other and with monocyte macrophage system cells. Both humoral and cell-mediated immunity are supported by them. Proliferating lymphocytes have higher concentrations of the n-terminal deoxyribonucleic acid

transferase enzyme. It is absent from mature lymphocytes but is present in thymocytes and immature lymphoid cells in bone marrow. T-lymphocytes contain large amounts of adenosine de aminase, which is required for immunological action.

Inflammation

A natural defence against the invasion of microorganisms and poisons is inflammation. Vascular and cellular responses are the two basic parts of the inflammatory response. Both reactions are mediated by chemical elements taken from plasma proteins or cells created as a result of an inflammatory response.

A protracted period of active inflammation, tissue deterioration, and repair define chronic inflammation. Endogenous hazardous plasma lipid components can increase the chronic inflammatory processes of atherosclerosis and vascular disease, which affect the arterial wall.

Chronic inflammation morphological features

- Tissue destruction
- Mononuclear cell infiltration
- Healing by connective tissue replacement
- Fibrosis
- Vascular endothelial growth factors and other angiogenic factors

NLR in subclinical inflammation

A high neutrophil lymphocyte ratio is a sign of latent inflammation in various vascular disease conditions. Even though it may also be influenced by systemic infections, atherosclerosis, hypertension, chronic renal illness, and diabetes, NLR refers to the systemic inflammatory response brought on by chronic disease.

Mechanism

The onset of inflammation is signaled by endothelial dysfunction caused by the cellular response of blood components. Endothelial dysfunction impairs nitric oxide and prostacyclin production. This depletes the vascular endothelium's anti-atherogenic, antithrombotic, and vasodilator properties.

In the control population, the normal d-NLR is <2.0.

To evaluate the PLR connection as a significant prognostic factor among patients with Coronavirus illness, Qu R et al. (2020) conducted a study. Patients with platelet peaks during therapy experienced lengthier hospital stays on average than those without platelet peaks (P.05). Patients who experienced platelet peaks during therapy on average were older than patients who did not (P.05). Patients whose platelets dramatically increased with therapy stayed in the hospital longer on average. Furthermore, the average number of hospital days was longer in individuals who had higher PLR during therapy. The volume of platelets and their dynamic changes throughout treatment may be able to predict the severity and prognosis of the disease, according to a single-center case series of 30 hospitalised patients with confirmed COVID-19 at Huizhou Municipal Central Hospital. The cytokine storm may be responsible for the patient with noticeably increased platelets and a longer than usual

hospital stay. A potential measure for the monitoring of COVID-19 patients may be the PLR of patients, which represents the intensity of the cytokine storm.²

RDW, NLR, PLR, and CRP were evaluated as biomarkers in laboratory-confirmed COVID-19 patients in the study by Seyit M et al. (2020), which also looked at the best diagnostic biomarkers and cutoff values. In people with positive Sars CoV-2 PCR results, CRP ($p = 0.0001$), LDH ($p = 0.038$), PLR ($p = 0.0001$), and NLR ($p = 0.001$) levels remained significantly higher. In contrast, Sars CoV-2 negative individuals had significantly higher eosinophil ($p = 0.0001$), lymphocyte ($p = 0.0001$), and platelet counts ($p = 0.0001$). The results showed that COVID-19 positive patients had significantly higher levels of CRP, LDH, PLR, and NLR, while COVID-19 negative patients had significantly higher levels of eosinophils, lymphocytes, and platelets.⁶⁰

In study by Zhu C et al., (2020) to assess the predictive value of NLR and PLR in COVID-19 patients. The conclusion that results from merely adding NLR/PLR to a linear model may be biased because there should be a nonlinear relationship between these parameters and outcomes. For instance, using information from the MIMIC database, we hypothesised that NLR/PLR and death in critically ill patients with lung infection would be unadjustedly linked (Fig. 1). The non-linear correlation somewhat supports our theory even if the cohort is different from the one in the current study. If so, as both high and low NLR/PLR were associated with greater mortality, NLR/predictive PLR's power in AUC can be jeopardised.⁶¹

In study by Jimeno S et al., (2020) to assess the prognostic implications of NLR in COVID-19 patients. In study 45 (12.1 percent) of the patients had severe acute respiratory failure that need breathing assistance⁴⁷ (12.6%) of the patients

passed away. Older patients ($P = 0.002$), those with considerably higher NLR upon admission ($P = 0.001$), higher rises in Peak NLR ($P = 0.001$), and patients with a faster rate of NLR growth ($P = 0.003$) were the patients who had worse outcomes in comparison to those who received follow-up therapy. Age, cardiovascular disease, C-reactive protein at admission, and Peak NLR were all found to be substantially linked with death by multivariable logistic regression. NLR is an easy-to-measure, generally accessible, affordable, and reliable parameter whose ongoing monitoring could help in the identification and management of COVID-19.⁶²

In a study published in 2020 by Tiwari et al., the authors assessed the changes in NLR, PLR, and haematological markers of COVID-19 patients' clinical outcomes in India. Patients have a mean age of 37.7 years, are mostly slightly symptomatic to asymptomatic (41.9 percent), and are male (58.1 percent). The NLR and PLR at baseline between male and female patients do not differ statistically significantly, according to the 95 percent confidence range. A statistically significant rise in lab values is seen during follow-up visits. Most of the patients are female, younger, and present with modest clinical symptoms. Patients in paediatrics only have mild symptoms. The initial CBC values reveal mild lymphopenia, eosinopenia, neutrophilia, and thrombocytopenia. There was an increase in CBC values and NLR in the subsequent cases. Anemia was not detected in either the baseline or follow-up CBCs. A single PLR does not indicate disease progression.¹

In a study published in 2020 by Huang S et al., the importance of NLR and PLR for predicting clinical outcomes in COVID were evaluated. Males make up a greater percentage of the population in severe instances than in less severe ones. There is a significant age difference between the two groups ($p=0.022$). More

comorbidities, like hypertension, diabetes mellitus, chronic obstructive pulmonary disease (copd), and fatty liver, were present in severe patients than in non-severe patients. There was a substantial difference between NLR and PLR ($p < 0.001$). Diabetes, fatty liver, coronary heart disease, and NLR were all substantially related with severe COVID-19 patients. NLRs were 1.729 times greater in the severe group of patients compared to the non-severe group. NLR is a distinct risk factor for patients with critical COVID-19. PLR and NLR significantly differed between people with critical and mild problems, indicating that evaluating PLR and NLR may be useful for identifying COVID-19 patients who are at high risk.⁶⁴

In study by Chan et al., (2020) to assess the use of NLR and PLR in COVID-19. A total of 20 trials with a total of 3,508 patients were included in the analysis. Nineteen studies revealed NLR values, while five studies reported PLR values comparing COVID-19 individuals with severe and non-severe disease. Patients with severe disease had higher levels of NLR (SMD: 2.80, 95 percent CI: 2.12 - 3.48, $P < 0.001$) and PLR (SMD: 1.82, 95 percent CI: 1.03 - 2.61, $P < 0.001$) than those with non-severe disease. In COVID-19, NLR and PLR can be employed as independent prognostic indicators of disease severity.⁶⁵

In a study by Ozsari S et al., (2021), the NLR, PLR, and mean platelet volume with PCR test were evaluated and contrasted in covid patients. The polymerase chain reaction levels and neutrophil lymphocyte ratio in group C were statistically higher than those in group B ($p < 0.001$ in both cases). There was no statistically significant difference in the mean platelet volume between the groups ($p > 0.005$ for all). Neutrophil lymphocyte ratio and C-reactive protein had a significant positive correlation ($r = 0.421$, $p < 0.001$).

The PCR and CRP had a favourable correlation ($r=0.243$, $p=0.001$) as well. The thorax tomography result can be seen before the polymerase chain reaction test. The polymerase chain reaction test's sensitivity varies based on the tester, the test's execution, and the test's overall quality. As a result, patients with negative polymerase chain reaction results and thorax tomography findings should have their neutrophil lymphocyte ratio and platelet lymphocyte ratio levels checked, and they should be monitored if a COVID-19 diagnosis is suspected.⁶⁶

In study by Man MA et al., (2021) to assess the NLR, PLR and Eosinophils correlation with high resolution CT severity score among the COVID-19 patients. 139 patients with verified corona virus disease and 149 age-matched controls underwent blood testing, and COVID-19 patients underwent HRCT thorax scans. Both the severity of a positive chest CT scan and NLR and PLR have been connected. Both the severity of a positive chest CT scan and NLR and PLR have been connected. When the NLR value is larger than 5.04, the chance of substantial CT alterations is only 50%, however when the NLR value is less than 5.04, the CT score is less than 3 with a 94% likelihood. Chest CT severity of 2 is correlated with an eosinophil value of 0.35 percent (Se = 0.88, Sp = 0.43, AUC = 0.661, 95 percent CI (0.544; 0.779), $p = 0.021$). NLR and PLR scores were considerably higher in COVID-19 patients. NLR = 2.90 and PLR = 186 in the study had strong specificity (0.89, $p = 0.001$ and 0.92, $p0.001$, respectively). A thorax CT should be ordered if the NLR and PLR levels are higher since it may reveal important lesions that could affect the patient's future care.

MATERIAL & METHOD

Source of data: Patients admitted in KLE's Dr. Prabhakar Kore Hospital and MRC, Belagavi fulfilling inclusion criteria.

Study design: A one year hospital based cross sectional study

Sample size formula:-

The prevalence rate-based formula for the minimal sample size is

$$n = \frac{Z\alpha^2 p (1-p)}{d^2}$$

where P is the prevalence percentage and d is the likelihood of the prevalence difference in percentage.

Z and significance level are related. Z = 1.96 at the 5% level of significance.

With P = 34% and d = 25% of P = 8.50%, the sample size is 119.

Inclusion criteria:

- Lab confirmed covid19 Patients admitted in
- KLE Dr Prabhakar Kore Hospital
- AGE >18 YEARS

Exclusion CRITERIA:

- Age <18 years
- Tuberculosis
- Neoplasms

METHODOLOGY

Patients admitted at KLES Dr Prabhakar Kore hospital & MRC with Covid19 lab confirmed patients Consenting for the study were enrolled and Informed consent was taken. A detailed history was documented and clinical features assessed. 15cc of venous blood was drawn and sent for basic laboratory tests complete blood counts including Neutrophil count; lymphocyte count platelets count, PT/INR renal function tests. Based on the clinical picture and test results patients were divided into severe cases and mild cases.

There is a link between severe cases and the neutrophil to lymphocyte and platelet to lymphocyte ratios.

STATISTICAL ANALYSIS

Since the study is of observational study the plan of analysis was followed. For the continuous quantitative variables mean and standard deviation was calculated. For The purpose of comparison. The continuous variables were compared using Student's unpaired t- test. The pre and post treatment measures were compared using student's paired t test Discrete variables were represented by median. The categorical data were expressed in terms of rates, ratios and percentages. The association between the outcome, clinical and Demographic characteristics were tested using Chi-square test, test of proportion or Fisher's exact test. For discrete variables nonparametric tests was used. For all the tests the value of p less than 5% (0.05) was considered significant and all the data was analysed using SPSS v21 operating on windows 10.

RESULTS

In the present study, the mean age of patients was found to be 58.20 ± 15.20 yrs of age, majority were in age of 50-80yrs. There was male preponderance with 76.3% and female 23.70% with male to female ratio of 3.21:1. The majority of patients are slightly symptomatic to asymptomatic, with a mean age of 37.7 years, male (41.9 percent), and female (58.1 percent).

Table 1: Agewise distribution of patients

AGE	NUMBER	%
20 - 29	4	2.96
30 - 39	13	9.63
40 - 49	21	15.56
50 - 59	33	24.44
60 -69	32	23.70
70-79	21	15.56
≥ 80	11	8.15
TOTAL	135	100.00

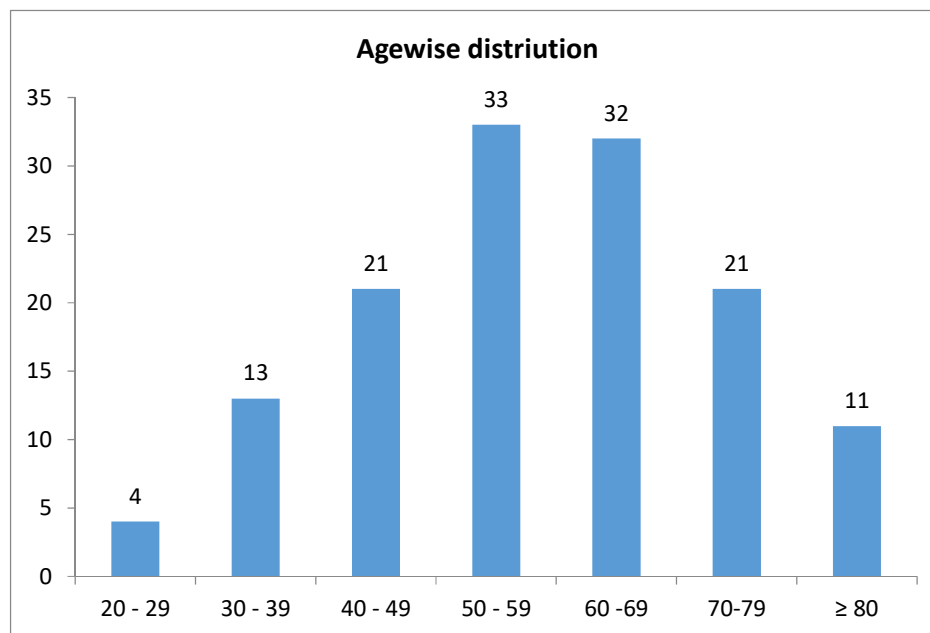


Figure 1: Agewise distribution of patients

Table 2: Mean age of participants

	MEAN	S.D.	MIN	MAX
AGE	58.20	15.20	24	102

Table 3: Showing the distribution of patients gender in the study

GENDER	NUMBER	%
Female	32	23.70
Male	103	76.30
TOTAL	135	100.00

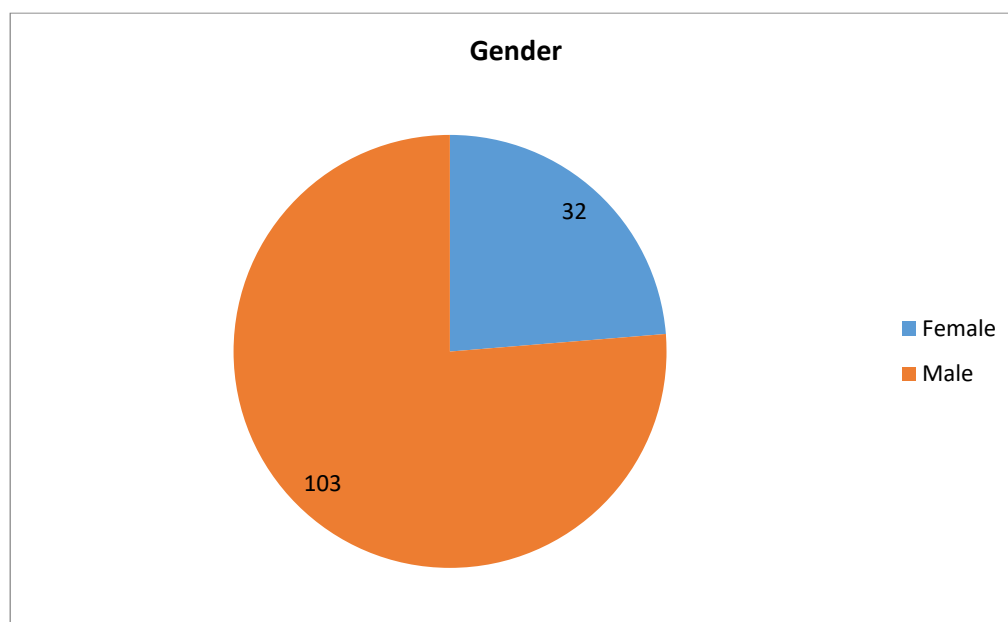


Figure 2: Showing the distribution of patients gender in the study

Table 4: Showing the symptoms of patients at presentation

SYMPTOMS	NUMBER	%
Cough	88	65.2
Fever	79	58.5
Breathlessness	107	79.2
Myalgias	17	13.3
Loss of smell	1	0.7
Loss of taste	1	0.7
Nil	3	2.1

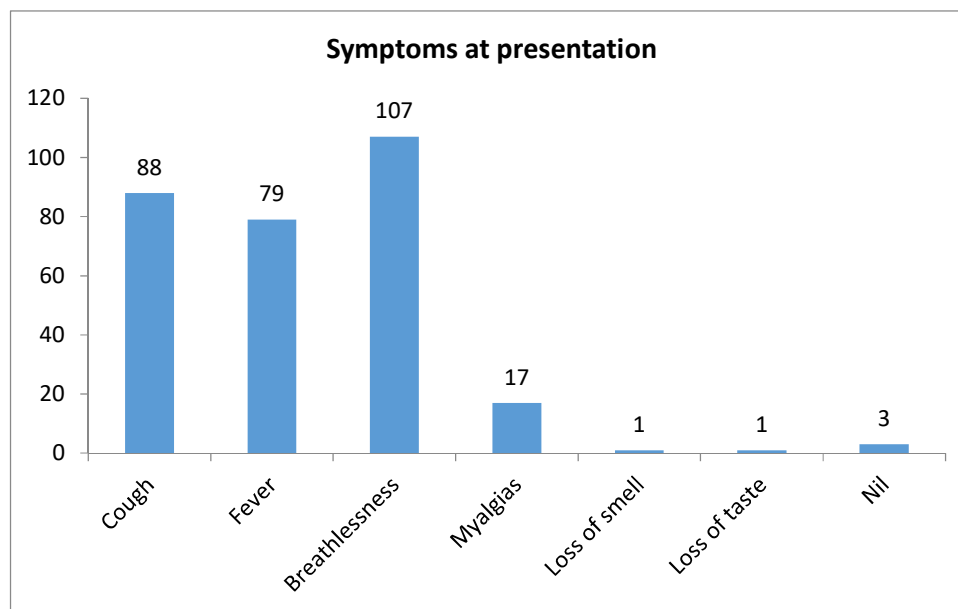


Figure 3: Showing the symptoms of patients at presentation

Table 5: Showing the other symptoms of the patients at presentation

OTHER SYMPTOMS	NUMBER	%
Altered Sensation	1	0.74
Abdomen Pain	1	0.74
Chest Pain	1	0.74
Chest Pain	1	0.74
Drowsiness, Vomiting	2	1.88
Facial Swelling	1	0.74
Headache	3	2.2
Hemoptysis	1	0.74
Loss Of Appetite	1	0.74
Restlessness	1	0.74
Right Eye Swelling X 5 Days	1	0.74
Nil	121	89.63
TOTAL	135	100.00

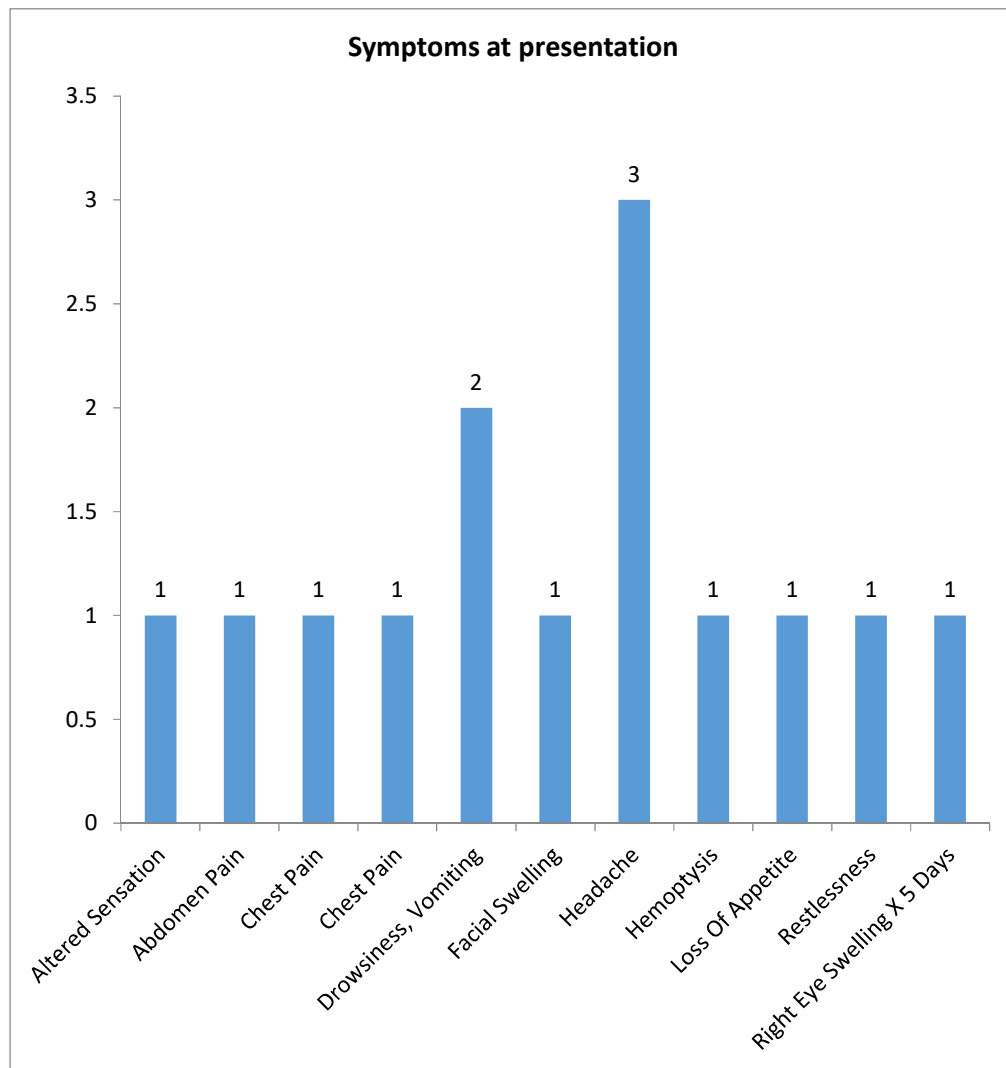


Figure 4: Showing the other symptoms of the patients at presentation

Table 6: Showing the time to hospital from symptoms onset

Time To Hospital From Symptom Onset (DAYS)	NUMBER	%
≤ 2	25	18.52
3 - 4	26	19.26
5 - 6	27	20.00
7 - 8	18	13.33
≥ 8	9	6.67
NOT RECORDED	30	22.22
TOTAL	135	100.00

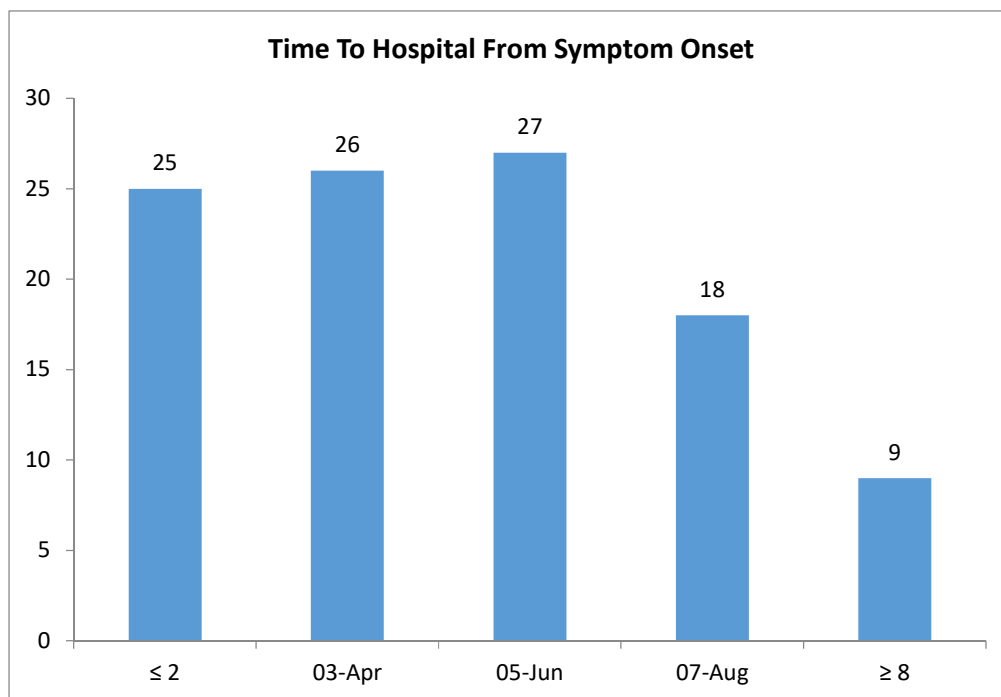


Figure 5: Showing the time to hospital from symptoms onset

Table 7: Showing the distribution of admission respiratory rate

Admission Respiratory rate (RR/min)	NUMBER	%
15- 19	1	0.74
20 - 24	24	17.78
25 - 29	14	10.37
30 - 34	26	19.26
≥ 35	5	3.70
NOT RECORDED	65	48.15
TOTAL	135	100.00

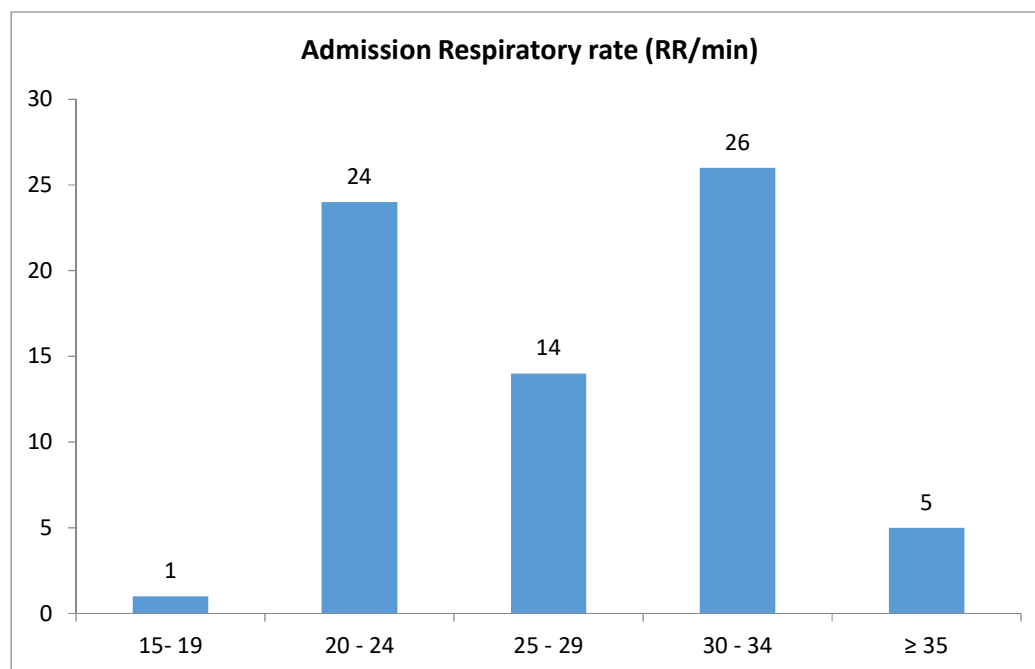


Figure 6: Showing the distribution of admission respiratory rate

Table 8: Showing the distribution of SpO2 at admission

ADMISSION SPO2	NUMBER	%
< 40	3	2.22
40 - 49	4	2.96
50 - 59	6	4.44
60 - 69	7	5.19
70 - 79	9	6.67
80 - 89	39	28.89
90 - 99	51	37.78
100	8	5.93
NOT RECORDED	8	5.93
TOTAL	135	100.00

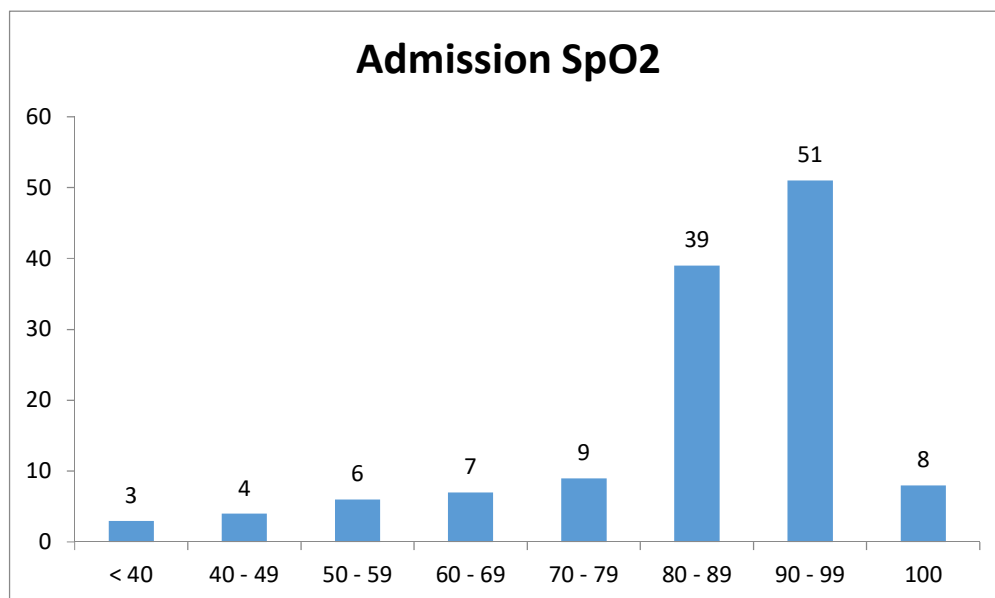


Figure 7: Showing the distribution of SpO2 at admission

Table 9: Showing the mean level of SpO₂ at admission

	MEAN	S.D.	MIN	MAX
ADMISSION SPO ₂	83.48	16.08	27	100

Table 10: Showing the oxygen modality at admission

ADMISSION O ₂ MODALITY	NUMBER	%
CPAP	5	3.70
HFNC	2	1.48
HFO	5	3.70
MASK O ₂	17	12.59
NIV	6	4.44
NRBM	9	6.67
O ₂ SUPPORT	2	1.48
RA	41	30.37
RBM	21	15.56
VENTILATOR	3	2.1
NOT RECORDED	24	17.78
TOTAL	135	100.00

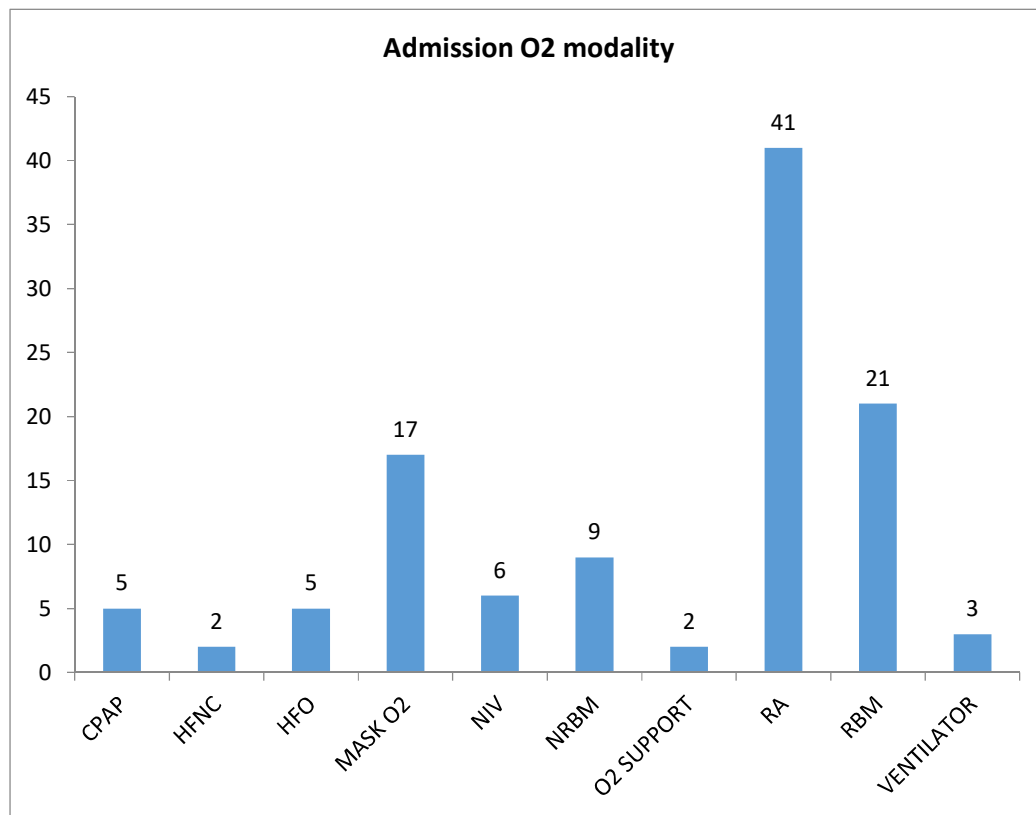


Figure 8: Showing the oxygen modality at admission

Table 11: Showing the mean level of oxygen used at treatment

	MEAN	S.D.	MIN	MAX
Mean Oxygen	14.97	10.66	1.5	60

Table 12: Showing the various comorbidities among the study patients

Co-Morbidities	NUMBER	%
Diabetes mellitus	59	
Hypertension	56	
Chronic kidney disease	9	
Ischemic heart disease	5	
Thyroid disorder	3	
Asthma	2	

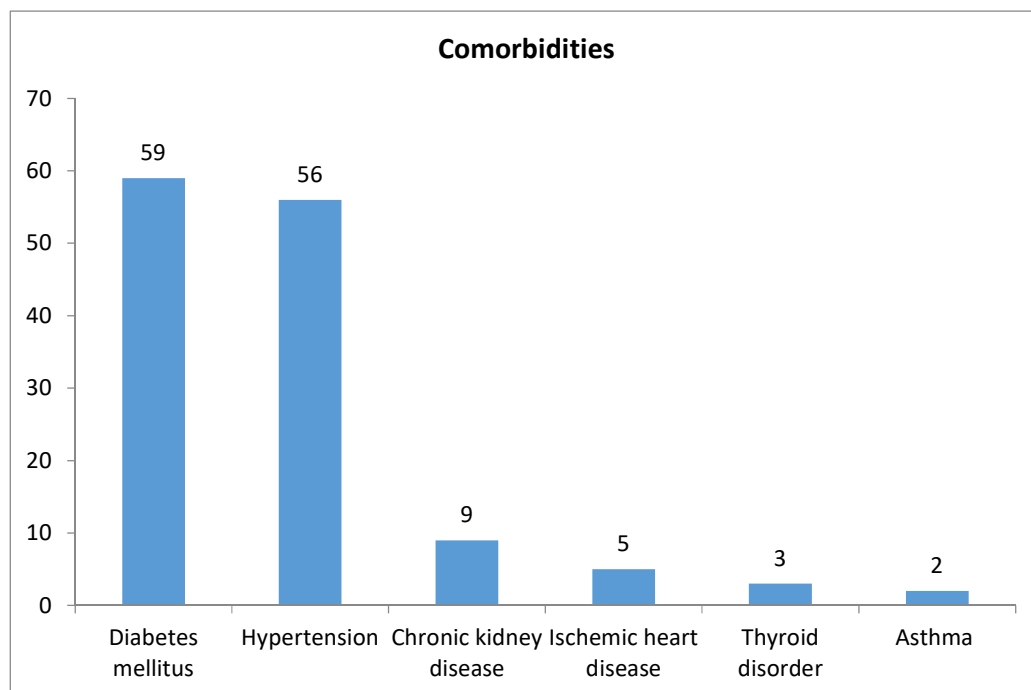


Figure 9: Showing the various comorbidities among the study patients

Table 13: Showing the distribution of duration of hospital stay among the study participants

Duration of hospital stay (DAYS)	NUMBER	%
0	4	2.96
1	13	9.63
2	20	14.81
3	6	4.44
4	17	12.59
5	11	8.15
6	7	5.19
7	10	7.41
8	10	7.41
9	8	5.93
10	7	5.19
≥ 11	19	14.07
NR	3	2.22
TOTAL	135	100.00

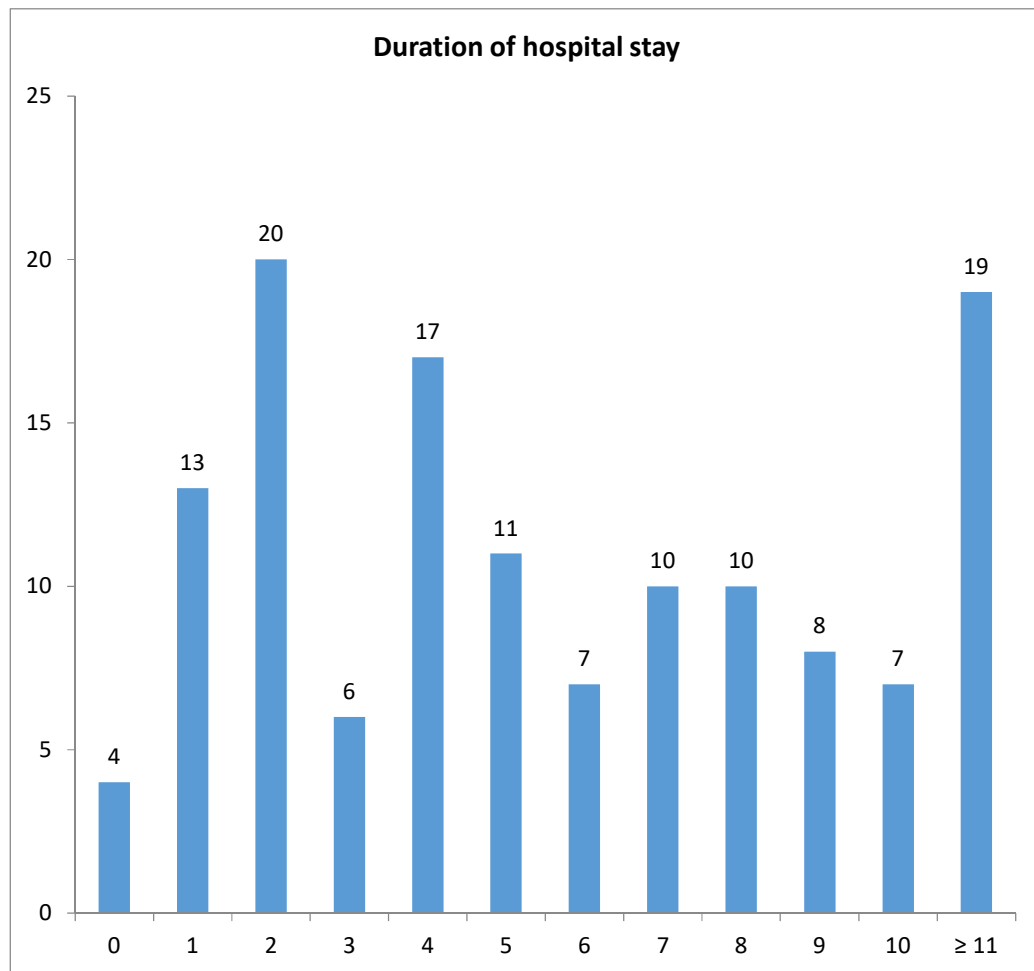


Figure 10: Showing the distribution of duration of hospital stay among the study participants

Table 14: Showing the mean level of duration of hospital stay, and other blood parameters

	MEAN	S.D.	MIN	MAX
Duration of hospital stay	6.62	6.52	0	42
Creatinine	1.93	3.01	0.41	21.74
Bilirubin	0.63	0.51	0.14	3.32
SGOT 1	57.35	40.80	6.1	273
SGPT 1	65.45	161.75	10	1265
SPO2 1	76.66	31.16	0.5	100

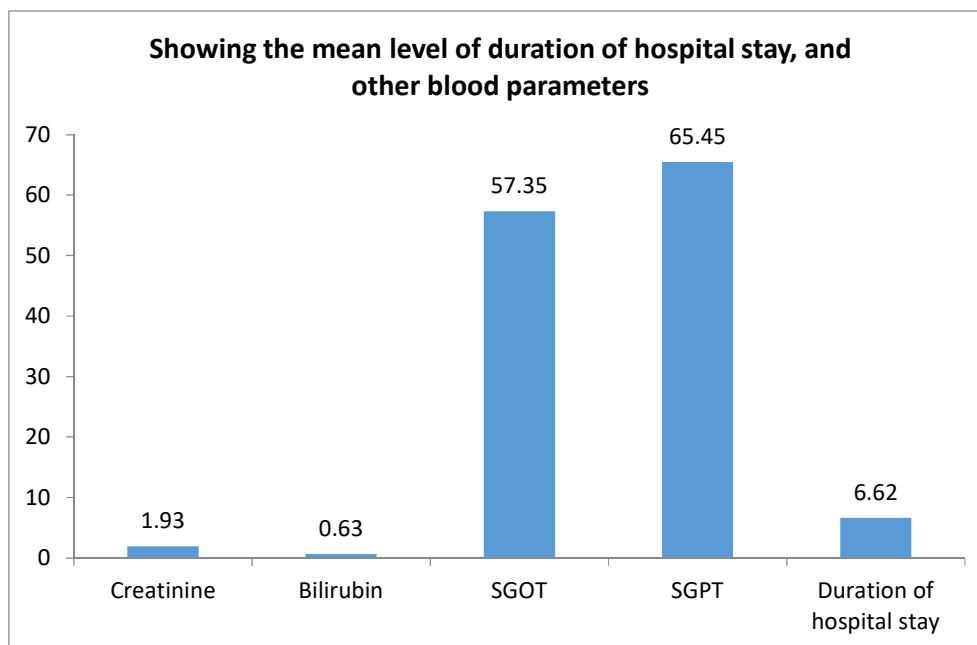


Figure 11: Showing the mean level of duration of hospital stay, and other blood parameters

Table 15: Showing the mean level of neutrophil and lymphocyte among the study participants

	MEAN	S.D.	MIN	MAX
Neutrophil % 1 (n = 89)	84.71	9.35	57	97
Neutrophil % 2 (n = 35)	84.34	19.71	16	98
Lymphocyte % 1 (n = 88)	10.10	7.71	2	34
Lymphocyte % 2 (n = 34)	8.62	11.11	1	42

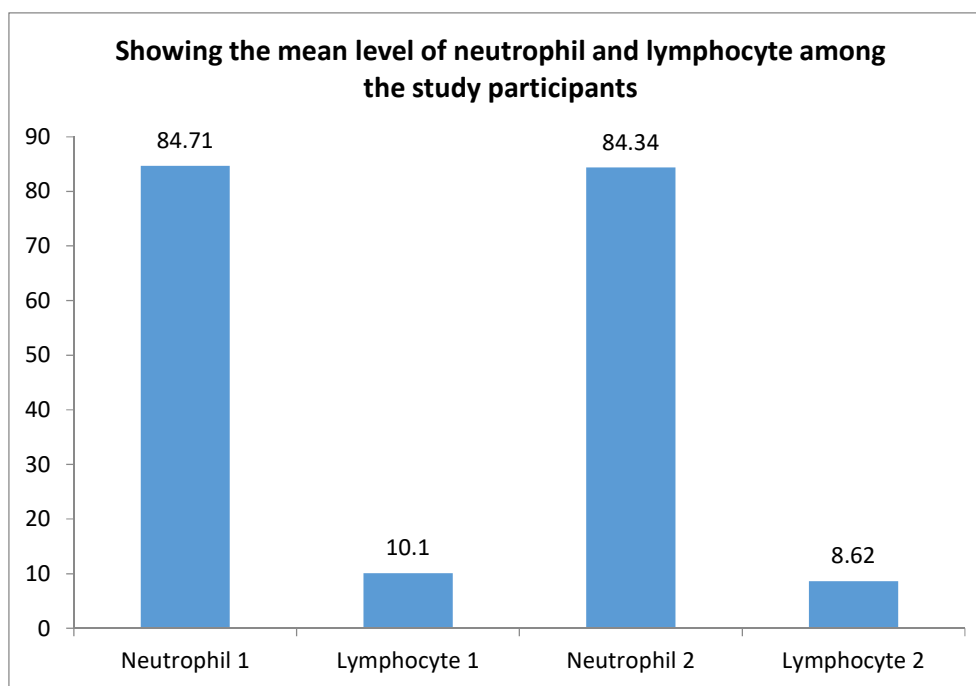


Figure 12: Showing the mean level of neutrophil and lymphocyte among the study participants

Table 16: Showing the distribution of NLR ratio among the study participants

N/L RATIO 1	NUMBER	%
< 5	13	9.63
5 – 10	29	21.48
10 – 15	11	8.15
15 – 20	10	7.41
20 – 25	8	5.93
25 – 30	0	0.00
30 – 35	12	8.89
≥ 35	4	2.96
NR	48	35.56
TOTAL	135	100.00

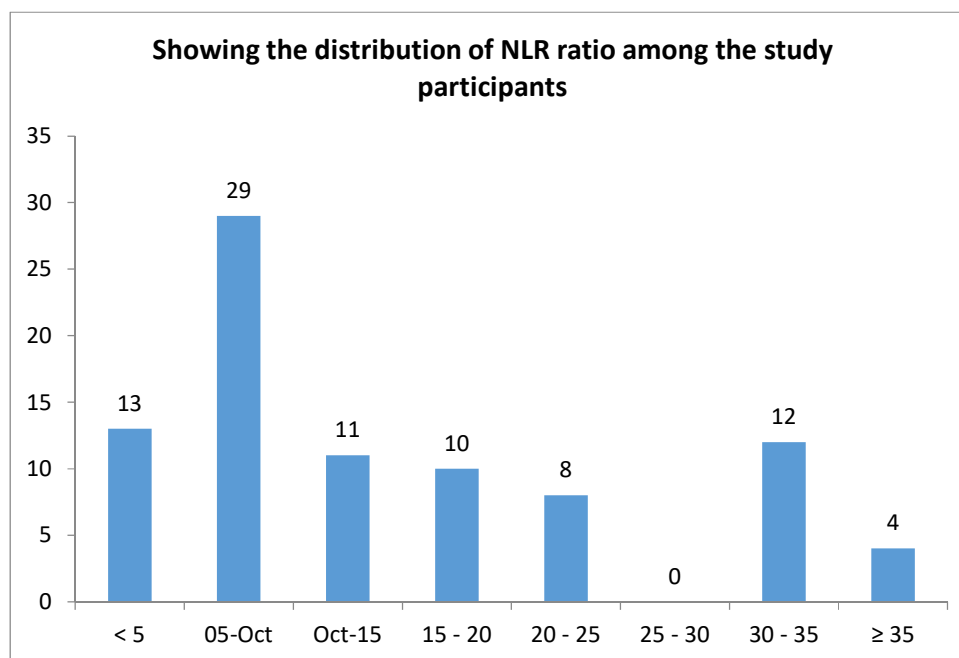


Figure 13: Showing the distribution of NLR ratio among the study participants

Table 17: Showing the mean level of NLR among the study participants

	MEAN	S.D.	MIN	MAX
N/L RATIO 1 (n = 87)	14.98	11.61	1.71	48.5
N/L RATIO 2 (n = 34)	25.98	22.9	0.89	98

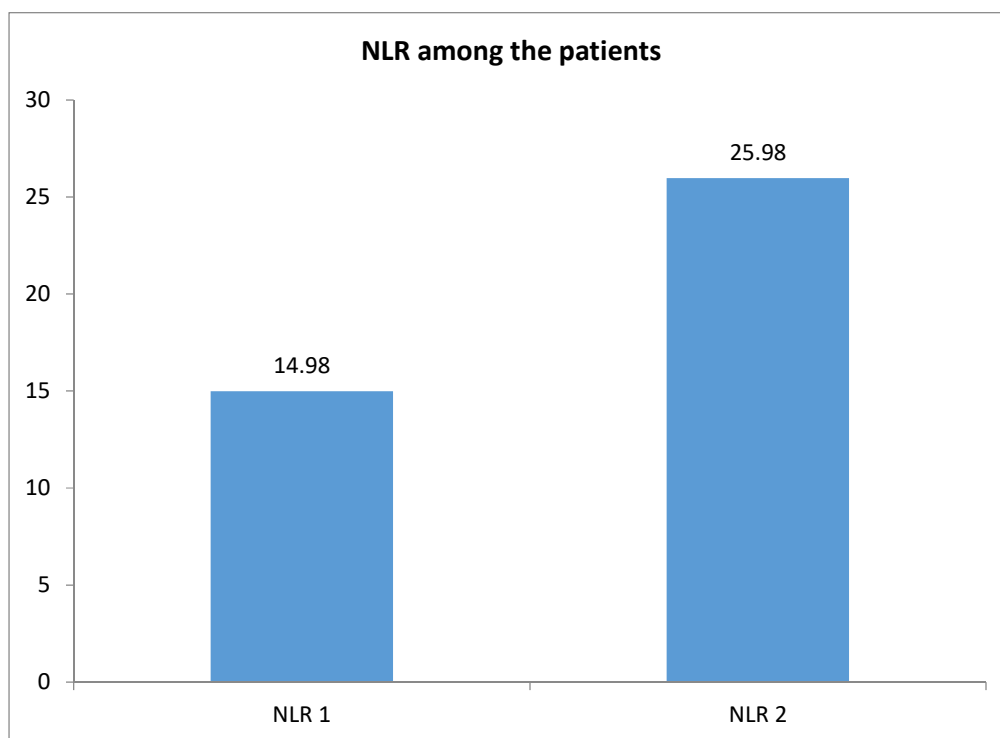


Figure 14: Showing the mean level of NLR among the study participants

Table 18: Showing the distribution of NLR among study participants

N/L RATIO 2	NUMBER	%
< 5	6	4.44
5 - 10	2	1.48
10 - 15	1	0.74
15 - 20	6	4.44
20 - 25	5	3.70
25 - 30	0	0.00
30 - 35	7	5.19
≥ 35	6	4.44
NR	102	75.56
TOTAL	135	100.00

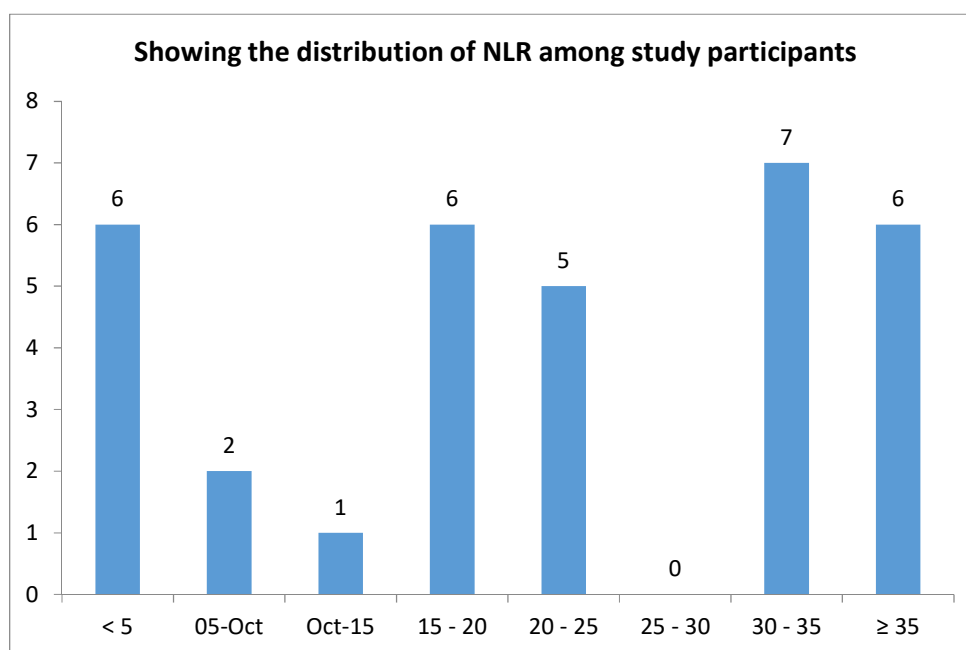


Figure 15: Showing the distribution of NLR among study participants

Table 19: Showing the mean level of platelet lymphocyte ratio among study participants

	MEAN	S.D.	MIN	MAX
PLATELET TO LYMPHOCYTE RATIO 1 (n = 84)	35.54	36.89	0.09	185.5

Table 20: Showing the distribution of PLR among the study participants

PLATELET/ L RATIO 1	NUMBER	%
0 - 10	17	12.59
10 - 20	21	15.56
20 - 30	16	11.85
30 - 40	6	4.44
40 -50	2	1.48
50 - 60	2	1.48
60 -70	5	3.70
70 - 80	5	3.70
80 - 90	2	1.48
≥ 90	8	5.93
NR	51	37.78
TOTAL	135	100.00

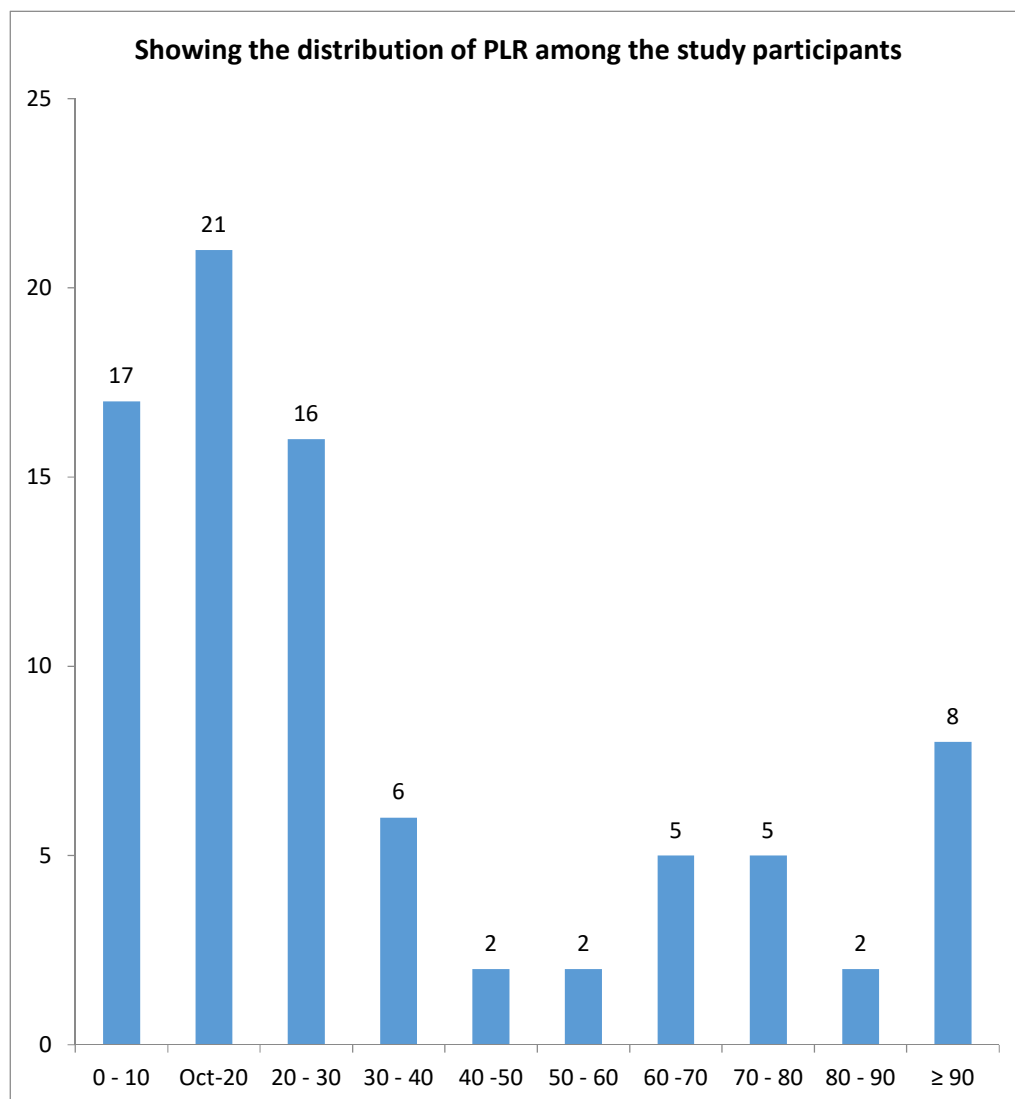


Figure 16: Showing the distribution of PLR among the study participants

DISCUSSION

The present hospital based cross sectional study was conducted among the patients admitted and getting treated for COVID-19 infection aged more than 18yrs of age. The purpose of the study was to evaluate the value of NLR and PLR in predicting the course and severity of COVID-19 in patients admitted to the Dr. Prabhakar Kore Hospital at the KLE University in Belagavi.

The bulk of the patients in the current study were between the ages of 50 and 80, with a mean age of 58.20 15.20 years. Males outnumbered females by a ratio of 3.21:1, with 76.3% of the population being male and 23.70% female. The average age of the patients is 37.7 years, and they are mostly mildly symptomatic to asymptomatic. Male patients make up 41.9% of the patient population (58.1 percent). The baseline CBC levels, NLR, and PLR between male and female patients are not statistically different, according to the 95 percent confidence range. There is a statistically significant rise in lab readings during follow-up visits. 1 Males make up a greater percentage of the population in severe instances than in less severe ones.. The age difference between the two groups is significant ($p=0.022$).⁶⁴

According to a study by Qu R et al., patients with platelet peaks during therapy had longer average hospital stays than those without them (P 0.05). Patients who experienced platelet peaks during medication on average were older than patients who did not (P0.05). Patients whose platelets dramatically increased with therapy stayed in the hospital longer on average. Additionally, the average number of hospital days was longer in individuals with higher PLR during therapy.²

Cough, fever, shortness of breath, myalgias, loss of smell, and loss of taste were studied. According to Ozsari S's research, fever was seen in 88 out of 203 patients (43%), along with cough, dyspnea, and non-respiratory symptoms in 34 (17%) and 41 (20%) patients, respectively. In our study, additional symptoms included joint discomfort (1%), headache (1%), sore throat (1%), anosmia (2%), diarrhoea (1%), and weakness (4%)⁶⁸

According to the report, an average hospital stay was 6.62 days. The bilirubin was 0.63, the creatinine was 1.93 mg/dl, and the SGOT and SGLT values were within the normal range. According to Seyit M et al research, 's COVID-19 positive patients had significantly greater levels of CRP, LDH, PLR, and NLR, while COVID-19 negative patients continued to have significantly higher levels of eosinophils, lymphocytes, and platelets. ⁶⁰ Beginning on day 5 after hospitalisation, a study by Seyit M et al. found a positive association between NLR and the length of hospitalisation, indicating that NLR was slightly correlated with hospitalisation days and implicated in predicting the outcome of COVID-19 patients. ⁶⁰

The neutrophil and lymphocyte counts in the study were within normal limits. With the NLR at 14.98 11.61, 25.98 was higher. Patients with COVID-19 have higher mean NLR and PLR levels, which is associated with patient mortality. The amount of platelets and their dynamic changes throughout treatment may be able to predict the severity and prognosis of the disease, according to a single-center case series of 30 hospitalised COVID-19 patients at Huizhou Municipal Central Hospital. The patient's dramatically higher platelets and longer-than-average hospital stay may be linked to the cytokine storm. The PLR of patients, which can serve as a novel indicator in the monitoring of COVID-19 patients, shows the intensity of the cytokine storm.²

According to Zhu C et al research, 's adding these parameters to a linear model could result in biased results because NLR/PLR should have a nonlinear relationship with results. For instance, we predicted an unadjusted link between NLR/PLR and death in critically ill patients with lung infection using information from the MIMIC database. The non-linear connection, despite the fact that the cohort in the current study is different, in some respects supports our claim. If so, the predictive usefulness of NLR/PLR in AUC may be compromised because both high and low NLR/PLR were linked to higher mortality⁶¹ Jimeno S and other people Older patients ($P = 0.002$), those with considerably higher NLR upon admission ($P = 0.001$), higher rises in Peak NLR ($P = 0.001$), and patients with a faster rate of NLR growth ($P = 0.003$) were the patients who had worse outcomes in comparison to those who received follow-up therapy. By using multivariable logistic regression, it was discovered that death was significantly correlated with age, cardiovascular disease, C-reactive protein upon admission, and Peak NLR. Continuous monitoring of NLR, an easily measurable, widely available, reasonably priced, and trustworthy indication, could aid in the detection and management of COVID-19.⁶²

According to Nalbant A. et al., their study had an NLR of 2.4, an AUC value of 0.660 ($P=0.021$), a sensitivity of 69.01 percent, a specificity of 65.40 percent, an LR+ and LR- of 1.98 and 0.48, a PPV of 80.43 percent, and an NPV of 50.00 percent. When NLR was 2.4 ($P=0.007$), the logistic regression revealed that the probability of COVID-19 was 20.3-fold higher. NLR is a reliable indicator for the diagnosis of COVID-19.⁶³

Huang S et al., severe patients had more comorbidities than non-severe patients, including hypertension, diabetes, chronic obstructive pulmonary disease, and fatty liver. The difference between NLR and PLR was significant ($p < 0.001$). Patients with severe COVID-19 disease were much more likely to have diabetes, fatty liver, coronary heart disease, or NLR. Patients in the severe group had NLRs that were 1.729 times higher than those in the non-severe group. For patients with severe COVID-19, NLR is a separate risk factor. PLR and NLR were significantly different between individuals with severe and mild conditions, suggesting that measuring PLR and NLR may help identify COVID-19 patients at high risk..⁶⁴

Some studies demonstrate the PLR and NLR are reliable markers of immune mediated, metabolic, prothrombotic and neoplastic disease. The fluctuations are related to immune inflammatory reactions and correlate positively with other markers of inflammation like CRP..⁶²

CONCLUSION

The COVID-19 is a disease with a wide range of clinical signs that is quickly spreading. The number of coexisting conditions, as well as the haematological parameters, were associated to the disease's severity. The NLR and PLR are much greater when predicting the patients' outcomes. Among patients whose NLR and PLR mean levels correlated with other markers, they were greater on average. When employed in continuous monitoring, NLR and PLR are simple to measure, accessible, affordable, and reliable metrics that could aid in COVID-19 therapy outcome prediction.

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ANNEXURE I – CONSENT FORM

TITLE OF RESEARCH STUDY:

**“SIGNIFICANCE OF NEUTROPHIL TO LYMPHOCYTE RATIO AND
PLATELET TO LYMPHOCYTE RATIO FOR PREDICTING CLINICAL
OUTCOME AND SEVERITY IN COVID 19 PATIENTS ADMITTED IN DR.
PRABHAKAR KORE HOSPITAL; KLE UNIVERSITY BELAGAVI”**

Principal Investigator: -

DR. MOHAMMAD SHAN ANSARI

Post graduate student,
Department of General Medicine,
JNMC, Belgavi.

Guide: -

Dr. RAJU H. BADIGER

Professor, Dept. of General Medicine
JNMC-Belagavi.

Introduction and Purpose: - Hyponatremia has been shown to be a predictor of CVS mortality among patients with heart failure. Hyponatremia is common after Myocardial Infarction and clinical outcome is accompanied by rise in plasma sodium concentration. This study is being undertaken to determine the incidence of hyponatremia in setting of Acute ST Elevation MI and determine its effect on the outcome

PROCEDURE:

If you agree to be part of the research study, you will be asked the relevant history and will be subjected to relevant clinical examination and investigations. You will also have to give blood for the necessary investigations.

Risk and Benefits:

The only risk and possible discomfort you might get is while taking blood from your arm for the investigations. It may cause swelling, pain, redness (rarely happens) at the site from where the blood is drawn.

You may not be benefitted by these investigations but you will be part of this study which is going to be useful to others in the future.

Alternatives:

Taking part in this study is voluntary. You may choose not to take part in this study.

If you decide to take part and later change your mind to withdraw from the study. Your decision will not change the present or future health care or other services that you receive. The study doctor or sponsor may stop your participation in this study at any time. If you choose not to take part in the study, you will receive the standard treatment for patients for your condition.

Privacy and Confidentiality:

All information collected about you during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify you in this research record. Information from this study may be published but your identity will be confidential in any publication.

Institution / Sponsor's policy:

Does not apply to this research

Financial incentives for participation:

No additional costs shall be incurred upon you for the purpose of this study.

Its purely being done with the idea of research and all the cost of the study will be borne by the investigator.

Authorization to publish the results:

The results of the study would be forwarded to the KLE University, Belagavi as part of requirement towards the completion of MD degree, review and publication.

In case of the queries during study or in future you may contact following persons

1. Dr. MOHAMMAD SHAN ANSARI

Investigator
Post Graduate Student,
Department of General Medicine,
JNMC, Belagavi.

2. Dr. RAJU H. BADIGER

Professor, Dept. of General Medicine
JNMC-Belagavi.

3. Dr. HARSHA HEGDE

Head of Ethical Committee for Human Research
JNMC, Belagavi

CONSENT FORM

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read this consent form, or it has been read to me, this consent form and have had all the questions answered

Signature / Left Thumb print of the Participant or legally authorized representative

Participant's name: _____

Signature / Left thumb impression: _____

of the participant

Name of the legally authorized _____

representative / guardian

Signature / Left thumb impression: _____

Witness' name: _____

Signature / Left thumb impression: _____

Investigator's name and signature: _____

ANNEXURE II – PROFORMA

**SIGNIFICANCE OF NEUTROPHIL TO LYMPHOCYTE RATIO AND
PLATELET TO LYMPHOCYTE RATIO FOR PREDICTING CLINICAL
OUTCOME AND SEVERITY IN COVID 19 PATIENTS ADMITTED IN DR.
PRABHAKAR KORE HOSPITAL; KLE UNIVERSITY BELAGAVI**

CASE NO: _____

NAME: _____

AGE/SEX: _____

IP NO.: _____

ADDRESS: _____

OCCUPATION: _____

COMPLAINTS AT PRESENTATION: _____

PAST HISTORY: _____

FAMILY HISTORY: _____

PERSONAL HISTORY: _____

TREATMENT HISTORY: _____

PHYSICAL EXAMINATION: _____

GENERAL CONDITION: _____

PALLOR- YES/NO: _____

ICTERUS-YES/NO: _____

LYMPHADENOPATHY-YES/NO: _____

CYANOSIS- YES/NO: _____

CLUBBING-YES/NO: _____

EDEMA-YES/NO: _____

VITALS: _____

TEMPERATURE: _____

PULSE: _____

RESPIRATORY RATE: _____

BLOOD PRESSURE: _____

SYSTEMIC EXAMINATION: _____

R. S.: _____

C.V.S.: _____

P.A.: _____

C.N.S.: _____

INVESTIGATIONS:

HB –	T. BILIRUBIN -
NEUTROPHILS –	D.BILIRUBIN-
LYMPHOCYTES-	ALBUMIN -
PLATELET COUNT-	ALP –
PCV–	SGOT -
RBC-	SGPT -
WBC -	INR –
ESR-	Creatinine –
Monocytes -	Urea -
Eosinophils -	
Basophils –	

ANNEXURE III – MASTER CHART

