
**"TO STUDY THE CORRELATION OF
FERRITIN/TRANSFERRIN RATIO WITH THE SEVERITY OF
COVID-19 INFECTION – A ONE YEAR CROSS SECTIONAL
STUDY DONE IN KLES DR. PRABHAKAR KORE HOSPITAL
AND MEDICAL RESEARCH CENTRE, BELAGAVI."**

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In partial fulfilment of the requirements for the degree of*

**M.D.
IN
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**JAWAHARLAL NEHRU MEDICAL COLLEGE, KAHER,
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ABSTRACT

Background: The incidence of infections caused due to Severe Acute Respiratory Syndrome-Related coronavirus 2 (SARS-CoV-2) was responsible for causing a worldwide pandemic, this disease which is also known as COVID-19 ended up affecting millions of people. The present study aims to understand the correlation of Ferritin/Transferrin ratio with COVID-19 infection and to assess its predictive value on the severity of the disease.

Materials & Method: This hospital based cross-sectional study was conducted among the patients admitted in the COVID 19 dedicated wards of KLES Dr PRABHAKAR KORE HOSPITAL, BELGAUM and those fulfilling the inclusion criteria of the study were included. Amongst them the blood biomarkers, Ferritin/Transferrin ratio and mortality was assessed and correlated with COVID-19 infection outcome among the patients.

Results: In the present study, a total of 168 patients with COVID-19 were evaluated. In this study, the mean age of patients was found to be 57.35 ± 16.59 years. Also, 84% were male patients and 16% were female patients with male preponderance. There is a significantly higher Ferritin upon Transferrin ratio among the patients with mortality as compared to the patients which survived. The area under the curve (0.885) for the Ferritin/Transferrin ratio was significantly positively related with the prediction of the mortality among the patients with the odds of death increasing by 2.24 (95% CI: 1.77 - 2.99) with the unit increase of Ferritin/Transferrin ratio.

Conclusion: The present study documented the significant correlation of Ferritin/Transferrin ratio with COVID-19 infection and also a higher incidence of mortality among the patients with higher Ferritin, low Transferrin and a significantly higher Ferritin/Transferrin ratio.

Keyword: COVID-19, Mortality, Survived, Ferritin, Ferritin/Transferrin ratio, Predict.

ABBREVIATIONS

ACE-2	ANGIOTENSIN-CONVERTING ENZYME 2 RECEPTOR
AUC	AREA UNDER CURVE
CoV	CORONAVIRUS
COVID 19	CORONAVIRUS DISEASE 19
CFR	CASE FATALITY RATE
HFO	HIGH FLOW OXYGEN
HIF	HYPOXIA-INDUCIBLE FACTOR
ICU	INTENSIVE CARE UNIT
IRE	IRON-RESPONSIVE ELEMENTS
IRP	IRON REGULATED PROTEIN
INR	INTERNATIONAL NORMALISED RATIO
IL	INTERLEUKIN
LDH	LACTATE DEHYDROGENASE
MERS	MIDDLE EAST RESPIRATORY SYNDROME
NIV	NON INVASIVE VENTILATION
RBM	REBREATHER MASK
RA	ROOM AIR

RBS	RANDOM BLOOD SUGAR
RNA	RIBONUCLEIC ACID
RTPCR	REVERSE TRANSCRIPTION POLYMERASE CHAIN REACTION
RR	RESPIRATORY RATE
SARS	SEVERE ACUTE RESPIRATORY SYNDROME
SGOT	SERUM GLATAMIC OXALOACETATE TRANSAMINASE
SGPT	SERUM GLUTAMIC PYRUVIC TRANSAMINASE
SPO2	OXYGEN SATURATION
TMPRS	TRANSMEMBRANE PROTEASE SERINE
UTR	UNTRANSLATED REGION
WHO	WORLD HEALTH ORGANISATION

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OBJECTIVES

Aim

To study the correlation of Ferritin/Transferrin ratio with COVID-19 infection and to assess its predictive value on the severity of the disease.

Objectives

- To assess the blood biomarkers among the COVID-19 patients
- To assess and correlate the predictive value of Ferritin upon Transferrin ratio with the outcome of COVID-19 patients.

INTRODUCTION

The incidence of infection caused due to Severe Acute Respiratory Syndrome related Coronavirus 2 (SARS-CoV-2) was responsible for causing a worldwide pandemic, this disease which is also known as COVID-19, that is coronavirus disease 2019 ended up affecting millions of people.¹

Anemia and its associated disturbances in iron regulation homeostasis is very commonly seen in COVID-19 patients and is associated with a significantly higher mortality in hospital.² The presence of chronic inflammation in COVID-19 along with alterations in iron homeostasis characteristically leads to increased levels of pro inflammatory marker Ferritin and a decreased amount of Transferrin due to sequestration of iron in hepatocytes, bone marrow and macrophages.

Notably, in one study it was found that a Ferritin upon Transferrin ratio of more than 10 predicted a five times higher risk of ICU admission and an eight times higher risk for the need of mechanical ventilation.³ This shows that biomarkers for Iron homeostasis can help with risk stratification of patients and alterations in iron regulation with a higher Ferritin/Transferrin ratio could help to predict the severity of COVID-19 infection.

This study can therefore investigate whether Ferritin/Transferrin ratio could help us to differentiate between COVID-19 patients with or without severe disease.

REVIEW OF LITERATURE

The Coronavirus disease has become a serious illness amongst both people and animals.⁴ Towards the end of 2019, a newly discovered virus was found to be the cause of several pneumonia cases in Wuhan, a city in China's Hubei province. It spread swiftly, resulting in an epidemic in China and an increasing number of cases in other countries throughout the world.⁵ On 5th February 2020, the World Health Organization (WHO) designated the term for this illness as COVID-19, short for Corona-virus disease 2019. Previously known as 2019-nCoV, the virus that causes COVID-19 is now referred to as Severe Acute Respiratory Syndrome Corona-Virus 2 (SARS-CoV-2). The WHO declared COVID-19 a pandemic on March 11, 2020.⁶

Epidemiology

The index case of this disease was reported on 31st December 2019 in the city of Wuhan, Hubei Province, People's republic of China.

Following this, the disease started rapidly spreading to other parts of China followed by its rapid spread internationally. COVID-19 was labelled a Public Health Emergency of International Concern by the World Health Organization on 30 January 2020 and was recognized as a pandemic on 11 March 2020. As of April 23, 2020, it had spread to nearly 185 countries resulting in a global pandemic.

The SARS CoV-2 basic reproduction number (R0) is predicted to be somewhere between 1.40 and 3.90, suggesting its extremely contagious nature. The R0 tends to be higher in public meeting venues such as schools, colleges, cruise ships, academic/business/religious/political congregations and hospitals which are not adequately equipped with personal protective measures such as PPE, gloves, masks

etc. The incubation period for the virus and its serial interval is around 5 to 6 and 8 days, respectively, comparable to both SARS CoV and MERS CoV.⁷ The CFR, which stands for Case Fatality Rate was predicted to lie somewhere between 0.90 and 3.0 percent during the initial period of the pandemic, which was lower than that of previously reported Human Coronavirus SARS CoV (6 percent – 17 percent) and the Middle East Respiratory Syndrome CoV (20 percent – 40 percent). The CFR had increased exponentially in many countries by 24th May 2020.

Unlike SARS CoV, a large proportion of people infected with SARS CoV-2 are asymptomatic or pauci symptomatic, allowing them to avoid identification and become potential carriers.⁸ It is very important to note that not every close contact would be infected, implying that individual genetic susceptibility plays a key role.^{9,10} The virus normally enters humans through the upper aerodigestive tract. SARS CoV-2 was recently also detected from a patient's stool sample raising the possibility of faecal-oral transmission.^{11,12}

In pregnant women, Infection with SARS CoV-2 increases the risk of vertical transmission. Vertical transmission was ruled to be less likely due to negative virus testing on swabs recovered from the cord blood, amniotic fluid, neonatal pharynx, and breast milk of six pregnant women infected with SARS Cov-2.¹³ The possibility of long range transmission via airborne route is also being studied which depends on the ventilation parameters of the given area and flow related dynamics of virus spread from an infected individual.¹⁴ Furthermore, mapping techniques like as cartograms can be used to depict the dissemination and expansion of COVID-19.¹⁵ Understanding the pathways of transmission of SARS CoV-2 can help us to understand and allow use of appropriate containment measures.

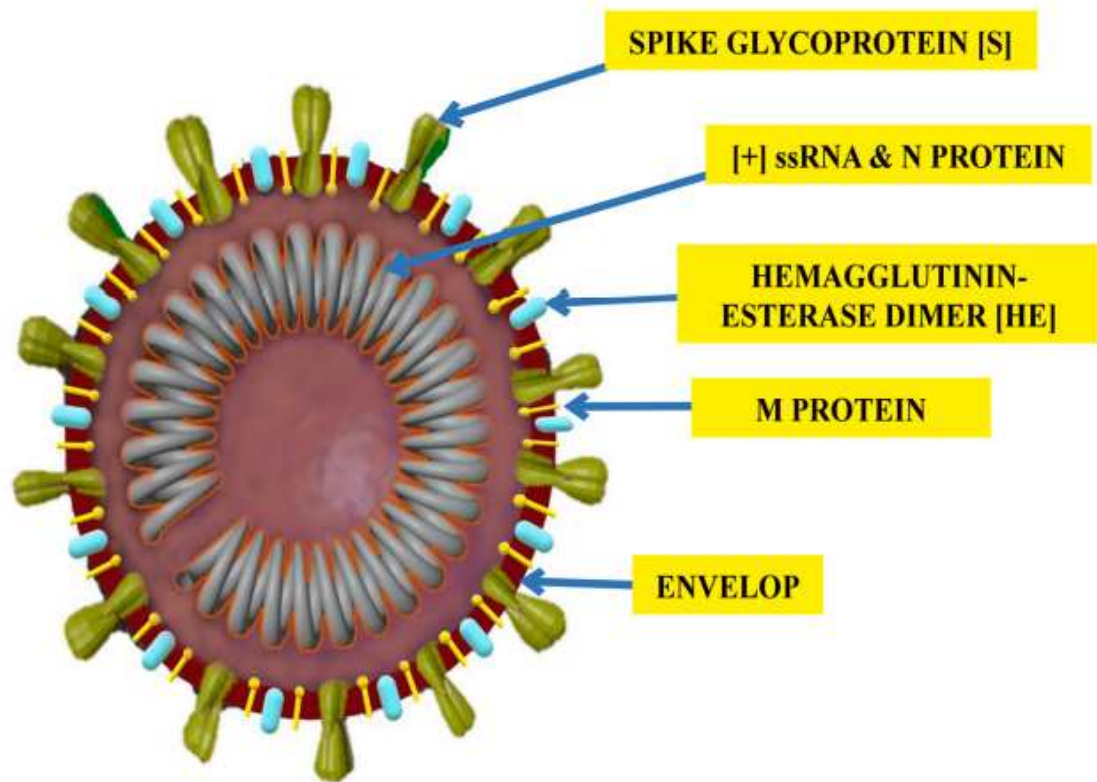


Figure 1: COVID-19 virus structure

Despite the fact that all age groups are susceptible to SARS CoV-2 infection, the severity of the disease and death appears to be lower in children.¹⁶ Women are less typically impacted than males in adulthood due to higher plasma ACE2 levels.¹⁷ The older population, particularly those having co-morbidities like hypertension, stroke diabetes and chronic pulmonary/renal/cardiac disease, are especially prone to very severe infections due to poor immunity against the infection and any underlying age related organ system breakdown.^{1,18} A recent study found that, similar to SARS CoV, the incidence of infection and mortality from COVID 19 was higher among non-O blood groups, notably type A blood group, probably because of lack of protective anti A IgM antibodies. There are still many uncertainties present in the epidemiology of

SARS CoV-2, particularly the interaction between the virus and its host, including vulnerability and epidemic evolution.^{19,20}

The entire family of Coronavirus is divided into α and β which is seen in mammals including humans and γ and δ which is seen in avian species. Their name is derived from the presence a particular spike protein on their envelope and their uncanny resemblance to crown (*Coronam* in Latin) which is usually appreciated only on electron microscopy. These family of viruses can cause infections in the gastrointestinal, respiratory, central nervous system and hepatic system. Both avian and mammalian species are equally predisposed to be infected by these group of viruses.

It is estimated that roughly 2% of the entire population is a healthy carrier of Coronaviruses, and that these viruses seem to be the causative agent for about 5 to 10 percent of acute respiratory infections. It is postulated that when the transfer of these viruses occur from natural hosts to humans via an intermediate amplifying host, they start undergoing recombination and rapid mutation, which results in the emergence of novel Coronaviruses that can be virulent and cause diseases in humans. In the recent few decades, these group of viruses have produced infections such as Severe Acute Respiratory Syndrome (SARS CoV) in China back in 2002–2003 and the Middle East Respiratory Syndrome (MERS CoV) in Saudi Arabia in 2012, culminating in pandemics with extra-pulmonary and pulmonary manifestations. As a result, Coronaviruses have emerged as significant pathogens in rising respiratory illness outbreaks.

Pathogenesis

Because of the similarities between SARS CoV and SARS CoV-2, SARS CoV-2 enters human cells utilising the S1 fraction of its highly glycosylated spike (S) proteins. This protein also possesses a receptor binding domain (RBD), which binds to the ACE-2 Receptor with 10 to 20 times more affinity than SARS CoV. Primarily present in alveolar epithelial cells, this ACE 2 receptor (more expressed in type II than type I). This receptor is expressed by the endothelium, cardiac myocytes, and gastrointestinal (intestinal and esophageal) epithelium. The SARS CoV-2 exploits its specific S1/S2 protease cleavage site with SPRR insertion on the spike protein to assist the fusing of the virus to the host cell membrane after binding to this receptor. It has been shown that TMPRSSs and ACE-2 R are highly co-expressed in the upper esophageal epithelium, type 2 alveolar pneumocytes and absorptive enterocytes, implying that in addition to alveolar epithelium this virus can also enter the host cell via both the intestinal and esophageal epithelium.⁷

Therefore, the potential SARS CoV-2 target tissues should express both TMPRSSs and ACE-2 Receptor. 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. Viral assembly, which occurs just before virions are released from infected cells, entails a nucleocapsid (N) protein binding to RNA molecules, which is then covered by membrane proteins and an envelope to produce full viral particles (virions), which can, as previously discussed, infect a variety of new cells.⁷

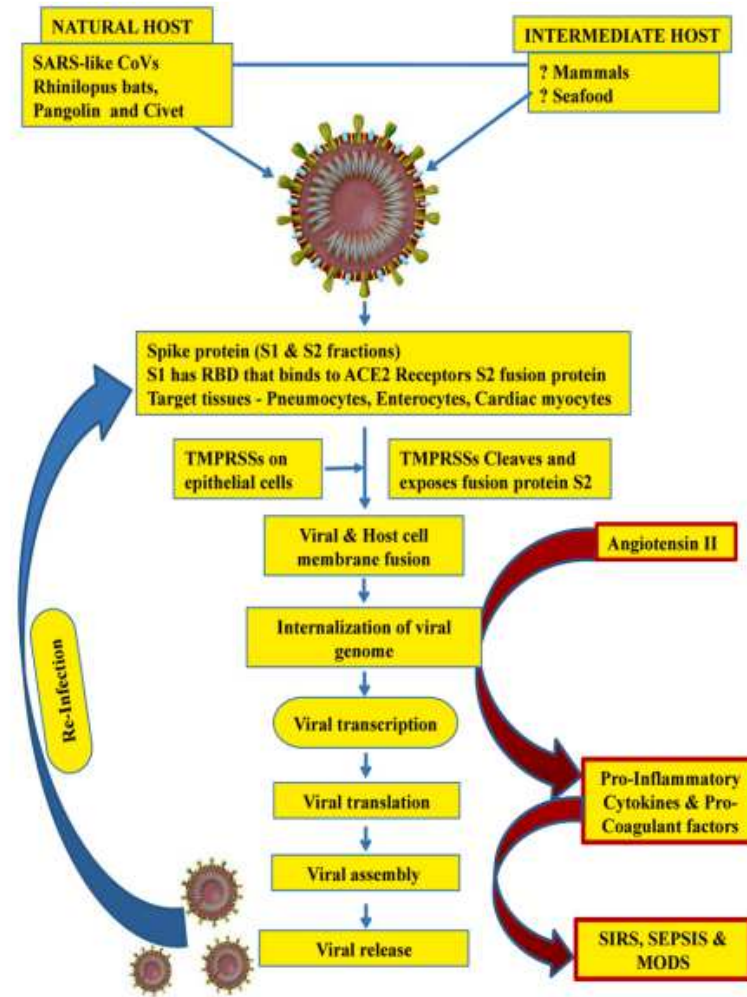


Figure 2: Pathogenesis of COVID-19 infection

Type II pneumocytes which are responsible for surfactant biosynthesis and tissue healing are destroyed after SARS CoV-2 infection, resulting in increased surface tension and dyspnoea. Furthermore, the damage caused to type II pneumocytes impairs the balance between pro-inflammatory and anti-inflammatory alveolar immunologic function by excessively inducing a cascade of both local and systemic inflammatory responses as a result of excessive cytokine production and release, also known as cytokine storm by activated inflammatory cells as a result of angiotensin II accumulation.⁷

Transmission

The secondary attack rate before the emergence of variations among paediatric contacts (less than or equal to 18 years of age) ranged from 4 to 57 percent in the observational studies pertaining to household transmission.^{21,26} A meta-analysis comprised of 87 studies on household transmission involving 1,556,198 household connections from various countries found that there was a secondary attack rate of 30% among adult contacts and 18% among paediatric contacts. More transmissible variations improve household transmission rates. A study of familial clusters found that during the dominant circulation of the Alpha (B.1.1.7 lineage) variant, the secondary attack rate was 72 to 75% in children under the age of 18 and up to 90% in adults.^{27,28}

Close contact with people infected with COVID-19 (typically a household member), having visitors and attending gatherings with people outside the household (social functions, activities with other children) were all linked to SARS-CoV-2 infection in children and adolescents, according to a case-control study.²⁹

There have also been reports of health care associated epidemics and cases of potential transmission from teachers or school employees to kids and among students in the school context.^{30,33} In a case-control investigation, irregular mask use at school was linked to SARS-CoV-2 infection, but not school attendance.²⁹

Although nothing is known about SARS-CoV-2 transmission by truly asymptomatic (as opposed to pre symptomatic) children, transmission from children with confirmed asymptomatic SARS-CoV-2 to household contacts has been described.³⁴ Furthermore, there have been instances of familial clusters involving asymptomatic children, as well as probable transmission from asymptomatic children

to adults outside their family.^{34,37} According to these reports, asymptomatic children may play a role in transmission.^{38,39} Adults' asymptomatic transmission is well documented.

Clinical features

The symptoms of COVID 19 are comparable in children and adults, although their frequency differs, however the symptoms appear to be milder in children as compared to adults; however severe instances in children have been also documented.

The spectrum of infection severity:

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

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

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

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

Similarly, 14 percent of 1.3 million cases reported to the Centres for Disease Control and Prevention (CDC) in the United States by the end of May 2020 were hospitalised, 2% were admitted to the intensive care unit (ICU), and 5% died. The risk of severe disease varies according to age, underlying comorbidities, and vaccination status.⁴⁰

Laboratory findings

The findings of laboratory parameters are variable. In a meta-analysis, the data of following laboratory abnormalities were noted: ^{41,42}

- Elevated C-reactive protein: 54%
- Elevated serum ferritin: 47%
- Elevated lactate dehydrogenase: 37%
- Elevated D-dimer: 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 specimen*	<p>High analytic sensitivity and specificity in ideal settings.</p> <p>Clinical performance depends on the type and quality of the specimen and the duration of illness at the time of testing.</p> <p>Reported false-negative rate ranges from <5 to 40%, depending on the test used.</p>	<p>Time to perform the test ranges from 15 minutes to 8 hours.</p> <p>Turnaround time is influenced by the test used and laboratory workflow.</p> <p>Some assays allow home collection of specimens that are mailed in.</p>
Serology (antibody detection)	Diagnosis of prior infection (or infection of at least 3 to 4 weeks' duration)	Blood	<p>Sensitivity and specificity are highly variable.</p> <p>Detectable antibodies generally take several days to weeks to develop; IgG usually develops by 14 days after onset of symptoms.</p>	<p>Time to perform the test ranges from 15 minutes to 2 hours.</p> <p>Turnaround time is influenced by the test used and laboratory workflow.</p>

			<p>Cross-reactivity with other coronaviruses has been reported.</p> <p>Individual results should be interpreted with caution in settings of low seroprevalence; serologic tests that have high specificity still have a low PPV</p>	<p>It remains uncertain whether a positive antibody test indicates immunity against future infection.</p>
Antigen tests	Diagnosis of current infection	Nasopharyngeal or nasal swabs	<p>Antigen tests are generally less sensitive than nucleic acid tests.</p> <p>Sensitivity is highest in symptomatic individuals within 5 to 7 days of symptom onset.</p>	<p>Time to perform the test is <1 hour.</p>

Table 2: Laboratory features associated with severe COVID-19 infection^{1,18,41,42}

	Possible Limit of Threshold
Elevated levels of :	
▪ D-dimer	More than 1000 ng/mL (normal range: <500 ng/mL)
▪ CRP	More than 100 mg/L (normal range: <8.0 mg/L)
▪ LDH	More than 245 units/L (normal range: 110 to 210 units/L)
▪ Troponin	More than 2 times upper limit of normal (normal range for troponin T: males 0 to 14 ng/L and females 0 to 9 ng/L)
▪ Ferritin	More than 500 mcg/L (normal range: males 30 to 300 mcg/L and females 10 to 200 mcg/L;)
▪ CPK	More than 2 times upper limit of normal (normal range: 40 to 150 units/L)
Decreased levels of:	
▪ Absolute lymphocyte count	<800/microL (normal range for ages more than 21 years: 1800 to 7700/microL)

FERRITIN

Ferritin is the major form in which Iron is stored in both Prokaryotes and Eukaryotes. Being a globular protein complex, it stores intracellular iron in a non-toxic and soluble form. Unbound ferritin which isn't combined with iron molecule is known as Apoferritin.

Ferritin as a biomarker

Ferritin is a protein molecule that binds to iron in the centre of its core. It is found that ferritin also has some role in inflammation. It is a globular protein complex consisting of 24 protein subunits and is the main intracellular iron storage protein in both prokaryotes and eukaryotes, keeping iron in a soluble and non-toxic form. Ferritin which is not combined with iron is called apoferritin.

Ferritin and its structure:

A very important and integral part of iron homeostasis is the iron storing protein Ferritin. It makes iron available for all important cellular processes and even helps to protect protein, DNA and lipids from the potentially harmful effects of iron. It can also play a role in some other conditions which includes inflammatory conditions, neurodegenerative diseases and malignancy.

The size of Ferritin molecule is about 450 kDa protein and consists of 24 subunits. It is present in every kind of cell. In mammalian species this molecule consists of 2 subunits, which are known as the light (L) and the Heavy (H) type with a molecular weight of 19 kDa and 21 kDa respectively. Given below is the structure of ferritin molecule. Apoferritin (iron free form of protein) is spherical in shape. The ferric iron is stored in form of ferrihydrite mineral iron containing form, known as

ferritin or holoprotein. Iron is toxic to the cell as it has the propensity to generate ROS, also known as reactive oxygen species which causes direct damage to DNA and proteins.⁴³

Genetic diseases hyperferritinemia occurs because of the abnormality in ferritin L-gene which results in drastically increased levels of serum ferritin. Increase in levels of H-ferritin is seen in some pathophysiologic conditions like malignancy.⁴³

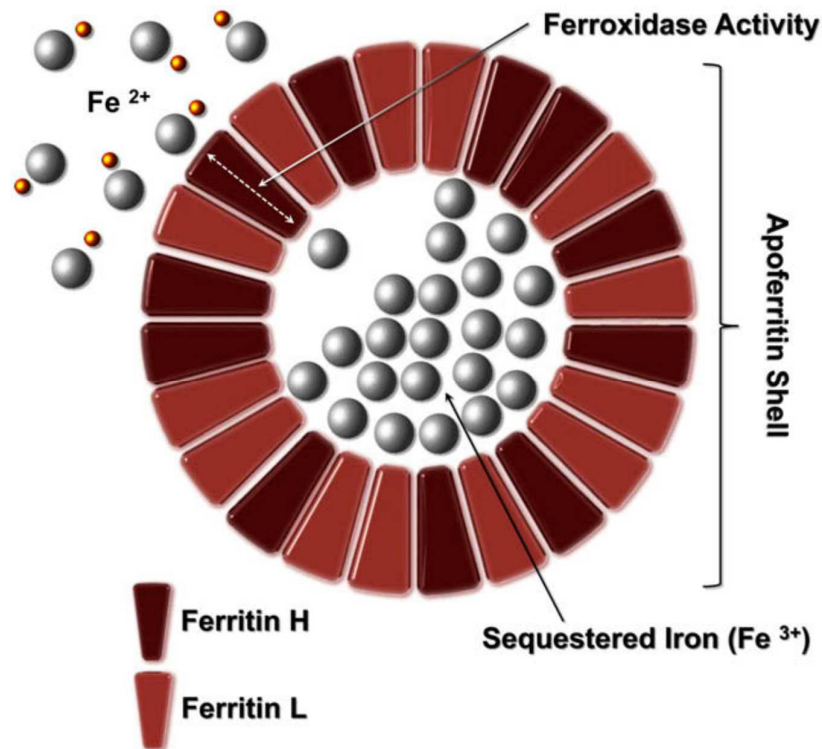


Figure 3: Structure of Ferritin

Physiological role of ferritin:

Ferritin is present both intracellular as well as extracellular compartment and act as iron binding protein. Extracellular ferritin acts as a marker of iron status and also helps in maintaining iron homeostasis as shown in figure below.⁴³

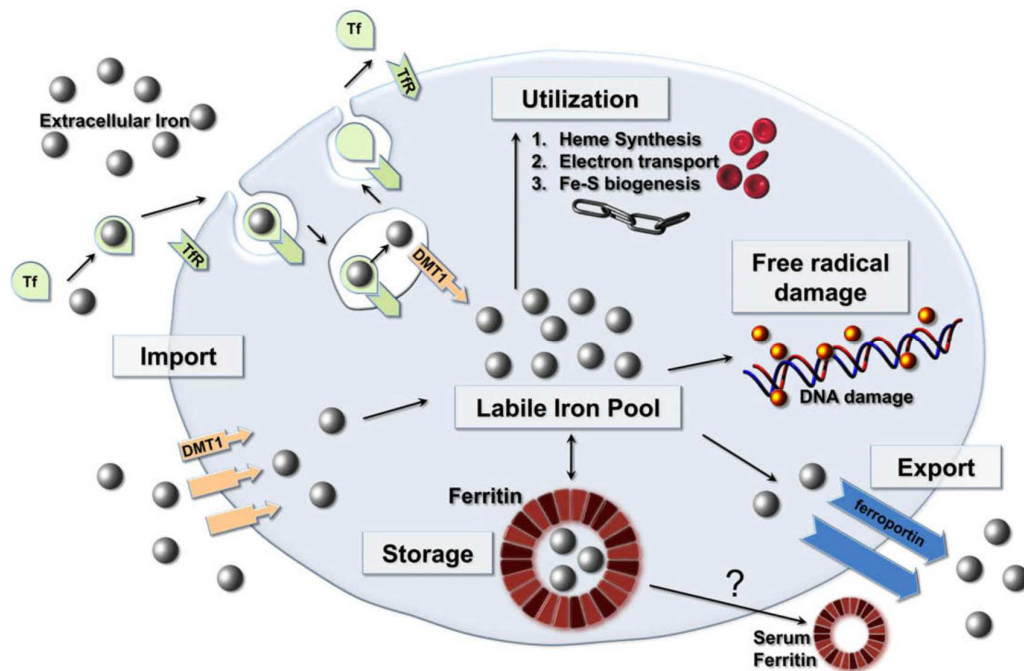


Figure 4: Role of Ferritin

Ferritin acts as a buffer in intracellular iron pool. Ferritin also acts as a parameter to help diagnose iron deficiency. Ferritin also decreases in hypothyroidism and ascorbate deficiency and can be useful in diagnosing conditions of iron overload. A physiological process to excrete excess iron from the body does not exist. Excess iron can collect in liver and heart which eventually causes free radical injury.⁴³

Iron in its free form is toxic to cells because of its propensity to form free radicals. Its role as a catalyst in the Fenton Reaction leads to the production of free radicals from reactive oxygen species which are dangerous to cellular organelles. In order to protect themselves from such dangerous free radicals, organisms have adapted by evolving an elaborate set of various protective mechanism which binds to iron and compartmentalizes the iron in the tissues to its non-toxic form. Inside the cells, iron is bound and complexed to proteins such as hemosiderin or ferritin. Free ferrous iron is bound to Apoferritin and is stored in the ferric state. Hemosiderin are

protein aggregates formed by the accumulation of ferritin within the cells of the reticuloendothelial system.

Iron can be extracted for release from Hemosiderin or Ferritin by the Reticuloendothelial cells, more readily from ferritin as compared to hemosiderin. Under normal conditions, serum ferritin correlates and acts as a surrogate marker of the total iron stores in the body and hence it cements its place as a diagnostic laboratory marker to estimate total iron stores.

Apart from its biological function, Ferritin also finds its use in engineering applications such as development of carbon nano tubes. It also finds its use in the synthesis of iron nano particles which have many emerging applications in nanobiotechnology. The role of ferritin in mitochondria is still being investigated.

Metabolism of iron and role of Ferritin:

Iron is absorbed in the proximal part of intestine. Dietary iron is in the ferric form which is first reduced by iron reductase present in the brush border epithelium. Iron on reaching inside enterocyte has two fates which depends on iron requirement. If iron demand is low in the body then iron binds to ferritin and will eventually be lost by sloughing off of the enterocyte. However if the iron requirement is high then the iron will be transferred into the circulation through ferroportin. The iron then binds to transferrin and is transported to different organs where it gets stored in the form of ferritin.⁴⁴

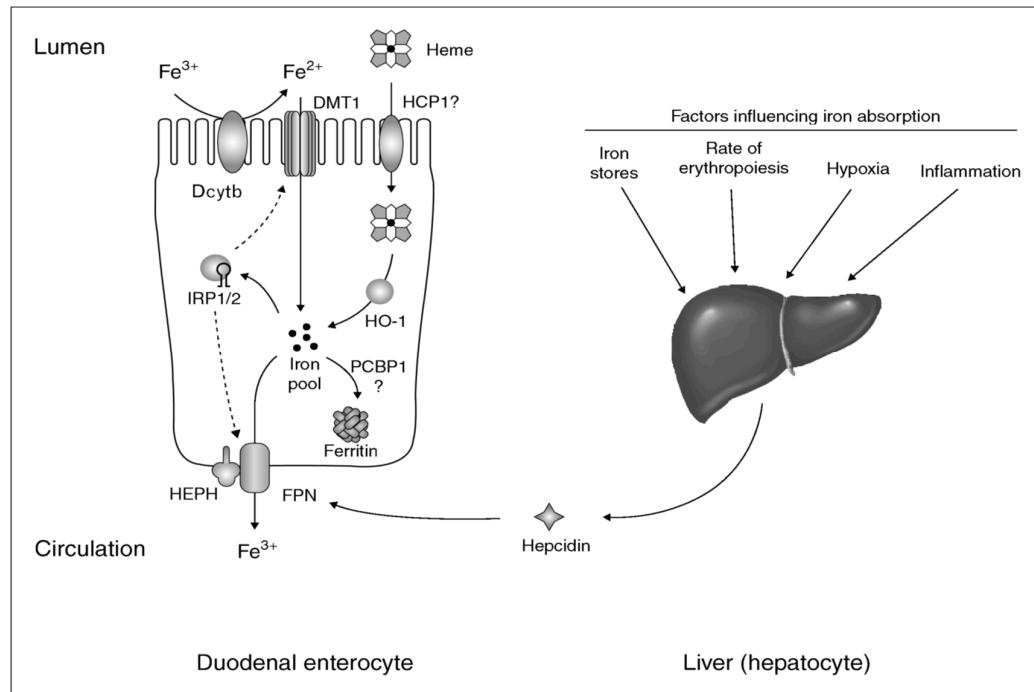


Figure 5: Iron absorption and transport⁴⁴

Role of Hepcidin in Iron Homeostasis

Hepcidin is a protein molecule closely involved in Iron homeostasis and its levels are influenced by various factors such as erythroid activity, iron stores, inflammation and hypoxia. Positive regulators include inflammation and increased iron level and negative regulators include increased erythropoiesis and lower iron levels. All these factors affect the levels of hepcidin by causing transcriptional modification of the protein molecule which in turn affects the levels of Hepcidin. The direct effect of increased hepcidin causes decrease in the absorption of iron from the gut and increased recycling of iron into the macrophages. As hepcidin is also an acute phase reaction protein, its levels are increased in states of acute and chronic inflammation via cytokines and chemokines such as Interleukin-6. The end effect is an increased sequestration of iron in macrophages and decreased absorption of iron from the gut.⁴⁶

Intracellular regulation of iron homeostasis:

The regulation of intracellular iron homeostasis occurs at the transcriptional level and by altering the stability of mRNA. The mRNA which codes for proteins involved in iron regulation has within it loop like domains in the untranslated regions, aka UTR in the 5' and 3' region. These regions of the mRNA are known as IRE, that is Iron Responsive Elements. IRPs are Iron Regulated Proteins which bind to the IREs present on the mRNA. There are mainly two kinds of IRP, IRP-1 and IRP-2. Although they are regulated in different ways, they have the similar function. In the 5' and 3' untranslated regions of the target mRNA, interaction of IRP and IRE takes place. Binding of IRP to the 5' end IRE results in decreased translation. Amongst the various iron regulatory proteins, erythroid ALA synthase (ALAS2), ferritin (Both L and H chain), mitochondrial aconitase, Hypoxia Inducible Factor (HIF 2) and Ferroportin have IREs in the 5' end.⁴⁷

The binding of IRP on the 3' end however results in the stabilization of mRNA and increased translation. The IREs for the proteins Divalent Metal Transporter (DMT-1) and Transferrin Receptor 1 (TFR1) are present on the 3' end of its respective mRNA. By modifications at the level of transcription and translation, the binding of IRP at the 3' end increases the production DMT1 and Tfr1.⁴⁸

The induction of Iron-regulatory proteins takes place whenever iron levels decrease. When the levels of Iron are on the higher side, the free iron binds to sulphur molecules and these iron sulphur cluster inhibit the binding IRPs to their respective IREs by competitive inhibition. Binding of IRPs to iron sulphur cluster promotes their ubiquitination and proteasomal degradation.⁴⁵ These iron sulphur clusters aren't formed when the levels of cellular iron is low. Due to lack of competitive binding on

the IRPs, they can freely bind to the IREs. This results in increase in the cellular uptake with decrease in the storage and utilization of iron.⁴⁵

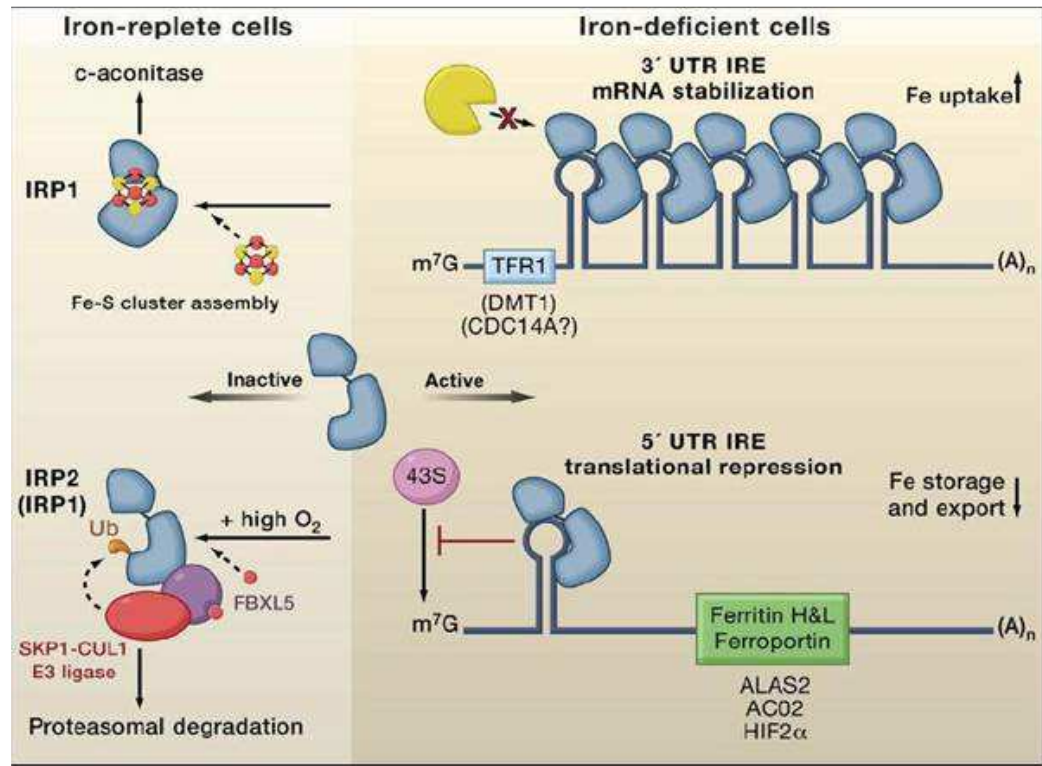


Figure 6: Regulation of Intracellular iron levels.⁴⁵

Role of Ferritin in inflammation:

Along with its role in iron homeostasis, ferritin is also serves as a key serum marker in inflammatory conditions. Ferritin, which is also an acute phase reactant is found to be increased in acute inflammatory conditions such as rheumatological, infections, hematological and cancer associated illnesses.

Increase in the levels of serum ferritin will cause increase in inflammation through inflammatory mediators such as IL-1 β , iNOS, RANTES and ICAM produced by stellate liver cells. Ferritin also acts as an immunosuppressant via increase in IL-10, decrease in B cell proliferation, decrease in T cell multiplication and decrease in IgG production. Therefore, it has been postulated that high ferritin levels are not just

the product of inflammation but rather may have a pathogenic role through its effect on signalling pathway as a part of the innate immune response and modulating the functioning of lymphocytes.

Apart from the role in iron homeostasis, ferritin also serves to protect the host against foreign pathogens by limiting the bioavailability of iron to the pathogens. During an episode of active infection, decreased availability of iron causes decrease in the proliferation of pathogens. It has also been observed that oral iron supplementation during an infection leads to increased mortality and morbidity in humans. It is common to observe a high serum level of ferritin during the active phase of an infection where viremia and bacteraemia predominates. In In vitro and In vivo conditions, Hepatocytes (Liver), Kupfer cells (Liver), proximal tubules (Kidney) and macrophages have all been noted to secrete ferritin. It has also been noted that increased ferritin positively correlates with increased mortality regardless of the underlying condition.⁴⁹

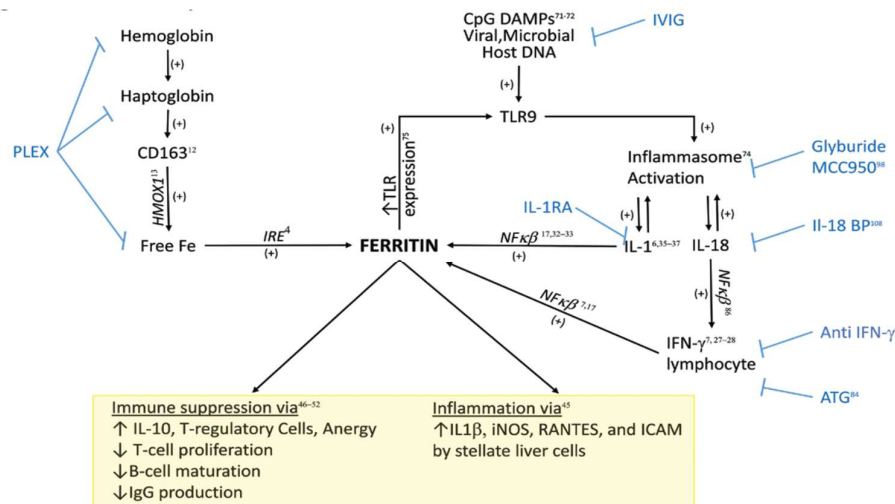


Figure 7 : Role of Ferritin in Inflammatory Positive Feedback

Table 3: Normal value of Ferritin⁵⁰

Age	Serum ferritin (normal)- ng/ml
0-6 weeks	0-400
7weeks- 1year	10-95
1-9 year	10-60
10-18 year (male)	30-300
10-18 year(female)	15-200

Ferritin as a Diagnostic marker

Serum ferritin is routinely included in the work up for patients suffering from Anaemia. It is also used in many rheumatological conditions where it acts as a surrogate marker of ongoing active inflammation. In anaemia, ferritin levels often reflect the total amount of storage form of iron in the body and is classically elevated in anaemia of chronic diseases.

For reference purposes, the normal levels of Ferritin in serum range from 30 to 300ng/ml and 15 to 200ng/ml in females.

Lowered

Iron deficiency anaemia is classically associated with lower levels of ferritin. Serum ferritin serves as one of the most sensitive laboratory test in the diagnosis of iron deficiency anaemia. Lower levels of ferritin and its association with Restless Leg Syndrome is also well documented in various studies.

In one of the studies conducted in Paris, France lower levels of iron and ferritin had a direct correlation with the severity of ADHD symptoms.⁵¹

Elevation in Serum Ferritin

Excess amount of iron can cause elevation in the levels of Serum Ferritin which would otherwise be excreted in stool. In disorders causing iron overload, such as Porphyria and Hemochromatosis, the levels of Serum Ferritin are found to be abnormally on the higher side which also serves as a diagnostic and prognostic indicator of the disease.

In a state of acute infection, Serum Ferritin is found to be on the higher side due to it being an acute phase reactant. A simultaneous normal value of C reactive protein can help to exclude serum ferritin elevations caused due to acute phase reactions.

Acute malnourishment may also cause elevation in the levels of Serum Ferritin.⁵²

Ferritin as an acute phase protein:

There are two main important roles of ferritin in the body, its primary role is in the intracellular sequestration of iron and its secondary role is its storage function during the activation of immune system. Macrophage, Cancer cells and hepatocytes all produce some amount of ferritin and its levels are classically elevated in acute infections. This summarises the role of ferritin under both steady state conditions and during an acute phase response.

Ferritin is also found in the circulation and through its release from damaged cells and through rough endoplasmic reticulum in normal cells, it contributes to increase in plasma ferritin levels. Clearance of plasma ferritin is mainly by hepatocytes.

Apart from the amount of iron storage and inflammatory activity, other conditions which affect the level of serum ferritin include tissue necrosis, infections, malnutrition, increased red blood cell turnover and surgeries. With all of these conditions considered, the direct simple correlation between the concentration of serum ferritin and the storage amount of iron in the body is no longer possible to estimate. In neoplastic diseases increase in ferritin levels are caused due to a higher production of ferritin by the cancerous cells. Solid tumours such as those of pancreas, liver and breast have a propensity to produce increased amount of ferritin, especially the H subunit predominant variant.

Increased level of serum ferritin is also seen in some diseases like still's disease (a systemic inflammatory disease), hemophagocytic syndrome, (a disorder associated with autoimmunity), patients with risk factor of coronary artery diseases and those with a history of myocardial infarction.

Lower levels of serum ferritin are seen in certain neurological disorders like restless leg syndrome, neuroferritinopathy (disorder of basal ganglia) and also in certain neurogenic causes of syncope in children.

Role of Ferritin to Transferrin ratio in COVID-19 patients to assess the severity

In a study conducted by Rosa Bellmann-Weiler, Lukas Lanser et al., at Department of Internal Medicine, Centre for Infectious Disease, Immunology and

Rheumatology, Medical School of Innsbruck, Austria, they found that dysregulated Iron homeostasis and Anaemia, especially with an increased Ferritin/Transferrin ratio is associated with a higher risk for the need of mechanical ventilation and a higher incidence of ICU admission in COVID-19 patients, thus establishing a relation of Iron Profile markers with COVID-19 severity. Studies trying to establish a particular Ferritin/Transferrin ratio have so far been inconclusive although in the above study, a Ferritin/Transferrin ratio of more than 10 suggested a higher risk of admission in the ICU and a higher incidence of patients requiring mechanical ventilation. The limitation of this study was however the short cohort observational period from 25th February 2020 to 20th May 2020. Also the study was not able to explore whether the anaemia which could be pre-existing before the onset of COVID-19 disease was already was a risk factor for more severe disease or a fatal outcome.³

In a study conducted by Katie-May McLaughlin et al., it was found that transferrin was upregulated in cells infected with SARS-CoV-2 as compared to normal non infected cells. Their investigations further revealed transferrin as an important factor playing a role in COVID-19 related coagulopathy. In cells infected with SARS-CoV-2, the transferrin expression was found to be much higher, especially in older age groups and male population. Moreover, a rise in the levels of transferrin levels was seen to have a direct correlation during the progression of COVID-19 infection further cementing the link between Iron Profile biomarkers like Transferrin and Ferritin and the pathogenesis and severity of COVID-19 infection. The above study also found out that locally produced Transferrin, independent of circulating Transferrin levels may contribute to the pathology of COVID-19 disease. For example, Transferrin is locally produced by neurological tissue like brain and spinal cord and higher levels are associated with increased incidence of ischemic

stroke, haemorrhagic stroke and hypercoagulability. Apart from hypercoagulability which predisposes COVID-19 patients to ischemic stroke, transferrin also causes cytotoxic damage associated with haemorrhagic stroke by increasing cellular uptake of iron. Higher levels of transferrin are found to be associated with Metabolic syndrome and Diabetes which have shown to exacerbate the severity of COVID-19 disease. However, one of the drawbacks of the above study was the limitation in sample size which was just 980. Also since it was a gene analysis study, it would make it more difficult to replicate in future due to resource limited settings in most centres.⁵³

In a Systematic Review and Meta-analysis study conducted by PE Taneri et al., on the correlation between Anaemia and Iron Metabolism in COVID-19 disease, published in European Journal of Epidemiology clearly emphasizes the need for future studies to explore the impact of iron metabolism and anaemia in the pathophysiology, prognosis, and treatment of COVID-19. Although the study analysed almost 57,563 COVID-19 patients, the study period was limited only up to 3rd August 2020. Also, it was found that the follow up was incomplete in almost half of the patients. The analysis was also limited to data only on human subjects and articles published in English only. The observational study used for the meta analysis also didn't include case reports and case series.⁵⁴

In a study conducted by Basting A et al., to assess the effect of iron supplement or iron chelator on the severity of COVID-19 infection, the levels of Iron, saturated transferrin and haemoglobin was found to be reduced in all the subjects as compared to the control group, although the level of ferritin was found to be higher in the patient group. After carefully adjusting the data for various parameters like age

and sex, the odds ratio (OR) of the disease was found to be higher in almost all the groups including moderate, severe, and critical groups which were 2.95, 6.1 and 8.34 respectively. Similarly, the Odds ratio was found to be reduced with lower levels of iron in the mild, moderate, severe and critical groups which were 0.96, 0.96, 0.94 and 0.95 respectively. Although both Iron and Ferritin have a higher AUC for COVID-19 disease prognosis, when it comes to disease severity, Serum Ferritin has the highest AUC. This indicated that routine measurement of Serum Iron and Ferritin can help us to determine the prognosis and severity of the disease. Based on the results of the above study, the usage of iron chelators to decrease the amount of iron intake can be of significant therapeutic benefit.⁵⁵

In one of the study by Claise C et al., to assess the effect of low transferrin level on inflammation in patients with COVID-19, it was observed that in severe COVID-19, there was a large rise in IL-6, CRP, and ferritin levels, as well as a drop in transferrin and iron levels. The levels of Transferrin adversely predicted differences in IgG and IgM levels ($P < 0.001$), along with an increase in IL-6 and CRP levels of 34.4% and 36.6% respectively ($P < 0.005$). Reduced serum Iron and Transferrin levels, as well as elevated CRP and Ferritin, were substantially linked to COVID-19's heightened inflammatory and immunological status. Based on the results of the above study, both Ferritin and Transferrin can be effectively used in the prediction of disease progression and severity.⁵⁶

In a study by Benoit JL et al., Anaemia and COVID-19 severity, it was found that future studies should include a larger sample size of cohort to assess the prognostic value of Anaemia during the course of illness.⁵⁷

In a retrospective study by Banchini F et al., to assess the serum ferritin level in inflammation. The median (interquartile range) serum ferritin level in group A was 674ng/mL, which was two times the threshold of 300 ng/mL in nine out of seventeen cases (52%). The median (IQR) ferritin level in group B total blood samples was 231, and 149 in the subgroup with leucocytosis. Group A had a considerably higher ferritin median level than group B overall (two-tailed Mann-Whitney test, $p < 0.0001$) and the subgroup with leucocytosis ($p < 0.0014$). The above study indicates that Iron metabolism appears to have a direct role in COVID-19 infection and that Serum Ferritin can be used as a diagnostic and prognostic marker in COVID-19 infection.⁵⁸

In order to better understand the link between various Iron parameters and the severity of COVID-19 infection, a study was conducted by Yanling LV et al wherein the median age of the participants was 63 years (IQR – 54 to 73) and the average length of hospital stay duration was about 28 days (IQR 17 to 40). After adjusting for pre-existing comorbidities, gender, age and IL-6, the adjusted risk ratios of severe COVID 19 infection was found to be 0.19 [0.08 0.48 at 95% CI], 4.38 [1.86 – 10.33 at 95% CI] and 0.42 [0.22 – 0.83 at 95% CI] for Transferrin, Serum Ferritin and Serum Iron respectively. Apart from predicting the severity, these indices also had a significant correlation with a heightened risk of Acute Liver Injury, Coagulopathy, Acute Myocardial Damage, ARDS, Acute Kidney Injury as well as a higher propensity to cause cytokine elevations, thereby predisposing to cytokine storm. The risk of Multiple Organ Damage as well as increased severity was seen with patients with lower levels of serum Iron. The findings of this study concluded that the above association between Iron parameters and COVID-19 severity may facilitate the use of Iron parameters as a valuable biomarker for assessing the risk factors and prognosis of COVID-19.⁵⁹

MATERIALS AND METHODS

Source of data: Patients admitted in the COVID 19 dedicated wards of KLES Dr PRABHAKAR KORE HOSPITAL, BELGAUM and fulfilling the inclusion criteria of the study were included.

Study design: One Year hospital based Cross-Sectional study.

Study period: SEPTEMBER 2020 to DECEMBER 2021 (One year).

Sample Size: 138

Sample method: Formula used for sample size calculation:

$$n = \left[\frac{(Z_{\alpha/2} + Z_{\beta})}{C} \right]^2$$

Where C is,

$$C = 0.5 \times \log_e \left[\frac{(1+r)}{(1-r)} \right]$$

In the above formula, r is the correlation coefficient, at 95% confidence level $Z_{\alpha/2}$ is 1.96 and Z_{β} value for 85% power is 1.0364.

We assume that there is a positive correlation is present between Ferritin and CRP levels (from below reference).⁵⁹ Correlation coefficient assumed as 0.25. This correlation coefficient is used in the sample size calculation for this study.

With 85% power, 95% confidence level and with above correlation coefficient, sample size required is $137.63 \approx 138$. Minimum sample size required is **138** subjects.

STATISTICAL ANALYSIS

METHODS:

Data is analysed using statistical software R version 4.2.1 and Microsoft Excel. Categorical variables given in the form of frequency tables. Continuous variables given in Mean \pm SD / Median (Min, Max) form. Chi square test is used check the association between categorical variables. Two sample t test is used to compare means over outcome. Mann Whitney U test is used to compare the distribution of variables over outcome. Applicability of Ferritin/Transferrin ratio to predict outcome is checked by Logistic regression and Receiver Operating Characteristic (ROC) curves. P-value less than or equal to 0.05 indicates statistical significance.

RESULTS

Data contains measurement on 168 subjects. Out of them, 3 (1.79%) subjects got worsened and 1 (0.595%) got discharged from hospital in the study period. Hence, remaining 164 subjects are considered whose age ranges from 20 to 90 years with mean age 57.35 ± 16.59 years. The following table gives the distribution of demographic variables over outcome.

Table 4: Distribution of demographic variables over outcome.

Variables	Sub Category	Outcome		Total	p-value
		Died	Survived		
Age (years)	Mean \pm SD	64.93 \pm 12.09	49.59 \pm 17.03	57.35 \pm	< 0.001 ^{MW*}
	Median	66 (32, 90)	52 (20, 85)	16.59	
	(Min, Max)			60 (20, 90)	
Gender	Female	10 (12.05%)	16 (19.75%)	26 (15.85%)	0.1768 ^C
	Male	73 (87.95%)	65 (80.25%)	138 (84.15%)	

Abbreviation: MW – Mann Whitney U test, C – Chi square test, * indicates statistical significance.

From Mann Whitney U test, it is observed that, there is significant difference in the distribution of age over outcome. Further, it can be observed that, the mean age of mortality group is more than the survival group.

From Chi square test, it is observed that, there is no significant difference in the distribution of gender over outcome.

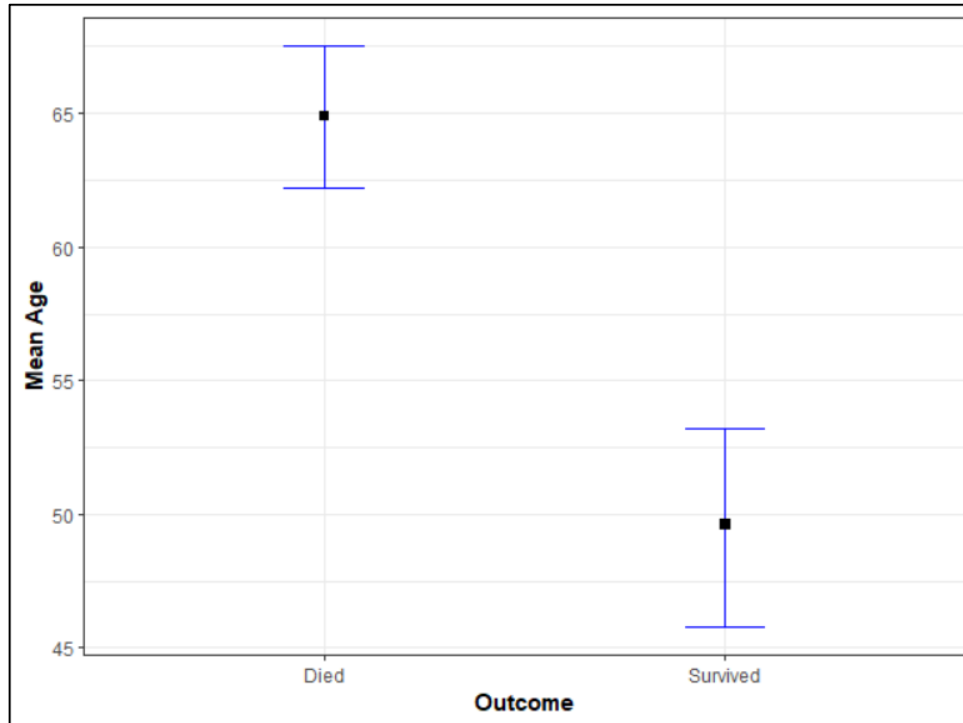


Figure 1: Mean plot of age over outcome.

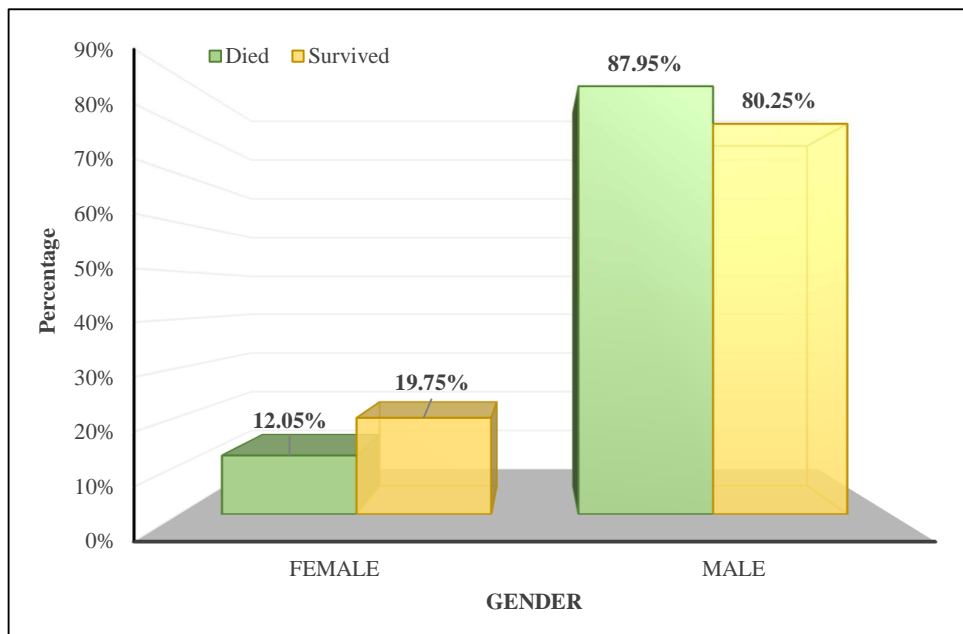


Figure 2: Distribution of gender over outcome.

The following table gives the comparison of symptoms with outcome.

Table 5: Comparison of symptoms over outcome.

Symptoms	Outcome		Total	p-value
	Died	Survived		
Fever	51 (61.45%)	57 (70.37%)	108 (65.85%)	0.2282 ^C
Cough	52 (62.65%)	59 (72.84%)	111 (67.68%)	0.1631 ^C
Breathlessness	74 (89.16%)	49 (60.49%)	123 (75%)	< 0.001 ^{C*}
Myalgia	20 (24.1%)	28 (34.57%)	48 (29.27%)	0.1406 ^C
Vomiting	2 (2.41%)	0	2 (1.22%)	0.5067 ^{MC}
Loose Stools	1 (1.2%)	2 (2.47%)	3 (1.83%)	0.6192 ^{MC}
Weakness	5 (6.02%)	12 (14.81%)	17 (10.37%)	0.0648 ^C
Abdominal Pain	1 (1.2%)	0	1 (0.61%)	1 ^{MC}
Low Urine	1 (1.2%)	0	1 (0.61%)	1 ^{MC}
Decreases Appetite	5 (6.02%)	0	5 (3.05%)	0.069 ^{MC}
Hematuria	1 (1.2%)	0	1 (0.61%)	1 ^{MC}
Sore Throat	1 (1.2%)	0	1 (0.61%)	1 ^{MC}
Chest Pain	2 (2.41%)	3 (3.7%)	5 (3.05%)	0.6917 ^{MC}
Altered sensorium	1 (1.2%)	0	1 (0.61%)	1 ^{MC}

*Abbreviation: C – Chi square test, MC – Chi square test with Monte Carlo simulation, * indicates statistical significance.*

From Chi square test, it is observed that, there is significant difference in the distribution of breathlessness over the outcome. Further, it can be noted that breathlessness symptom was observed more in cases of subjects which didn't survive. There is no significant difference in the distribution of other symptoms over outcome.

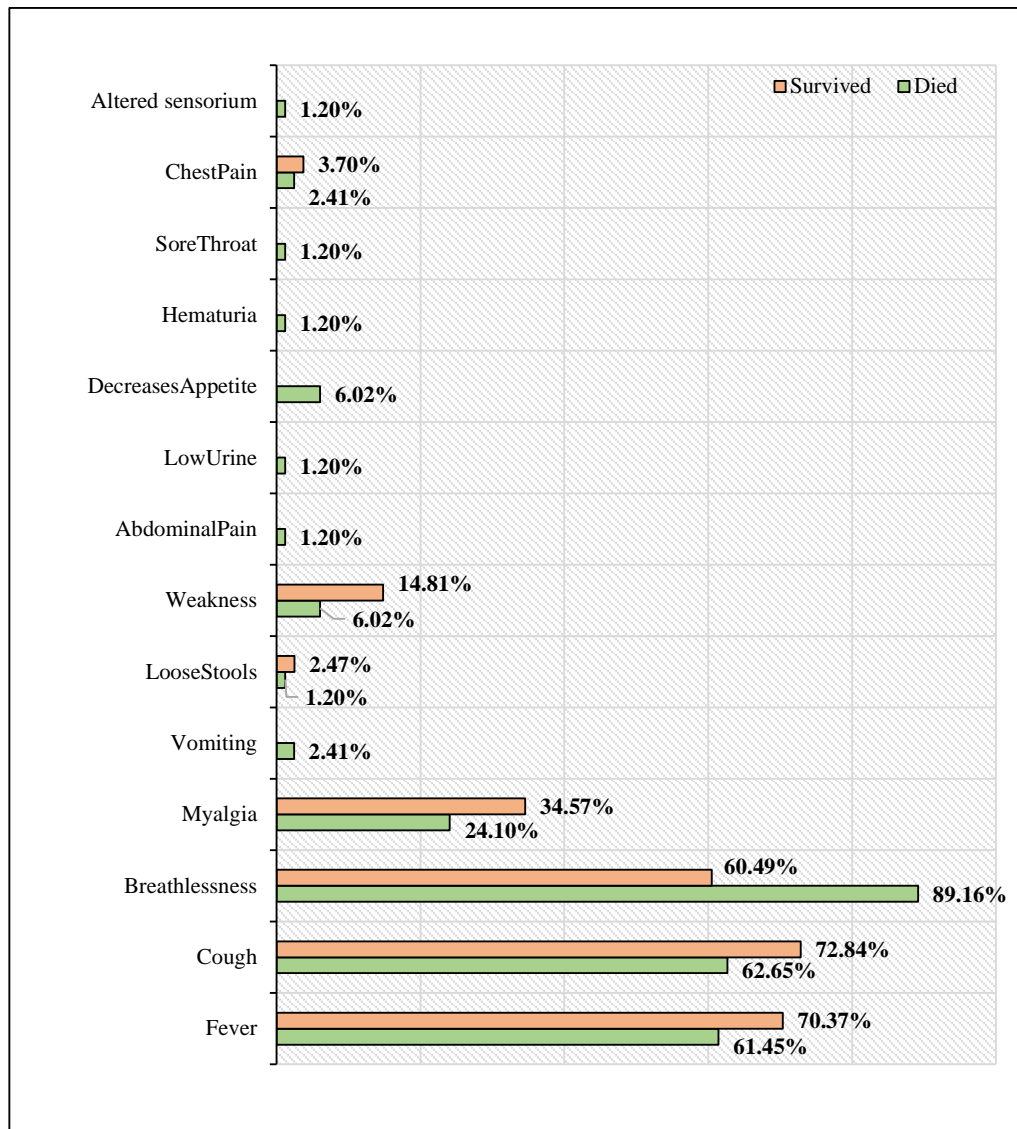


Figure 3: Distribution of symptoms over outcome.

The following table gives the comparison of O2 modality over outcome.

Table 6: Comparison of O2 modality over outcome.

O2 Modality	Outcome		Total	p-value
	Died	Survived		
HFO	1 (1.2%)	0	1 (0.61%)	< 0.001^{MC*}
NIV	22 (26.51%)	0	22 (13.41%)	
O2 MASK	13 (15.66%)	36 (44.44%)	49 (29.88%)	
RA	0	41 (50.62%)	41 (25%)	
RBM	47 (56.63%)	4 (4.94%)	51 (31.1%)	

Abbreviation: MC – Chi square test with Monte Carlo simulation, * indicates statistical significance.

From Chi square test, it is observed that, there is significant difference in the distribution of O2 modalities over the outcome.

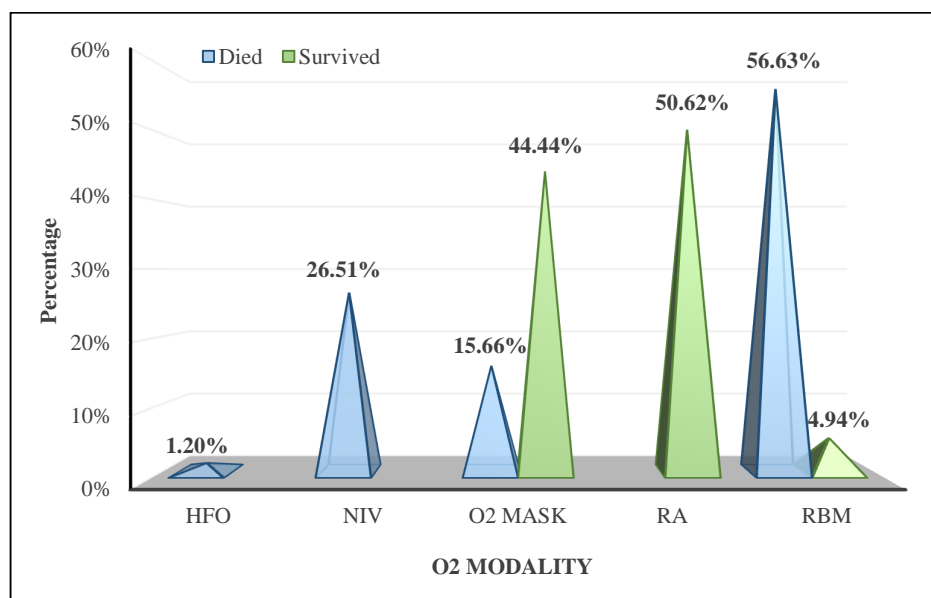


Figure 4: Distribution of O2 modality over outcome.

The following table gives the comparison of time to Hospital from Symptom Onset over outcome.

Table 7: Comparison of Time to Hospital from Symptom Onset over outcome.

Variable	Outcome		Total	p-value
	Died	Survived		
Time to Hospital from Symptom Onset	4.24 ± 2.51 4 (1, 20)	3.59 ± 1.74 3 (1, 10)	3.92 ± 2.18 3 (1, 20)	0.0583 ^{MW}

Abbreviation: MW – Mann Whitney U test.

From Mann Whitney U test, it is observed that, there is no significant difference in the distribution of time to hospital from symptoms onset over outcome.

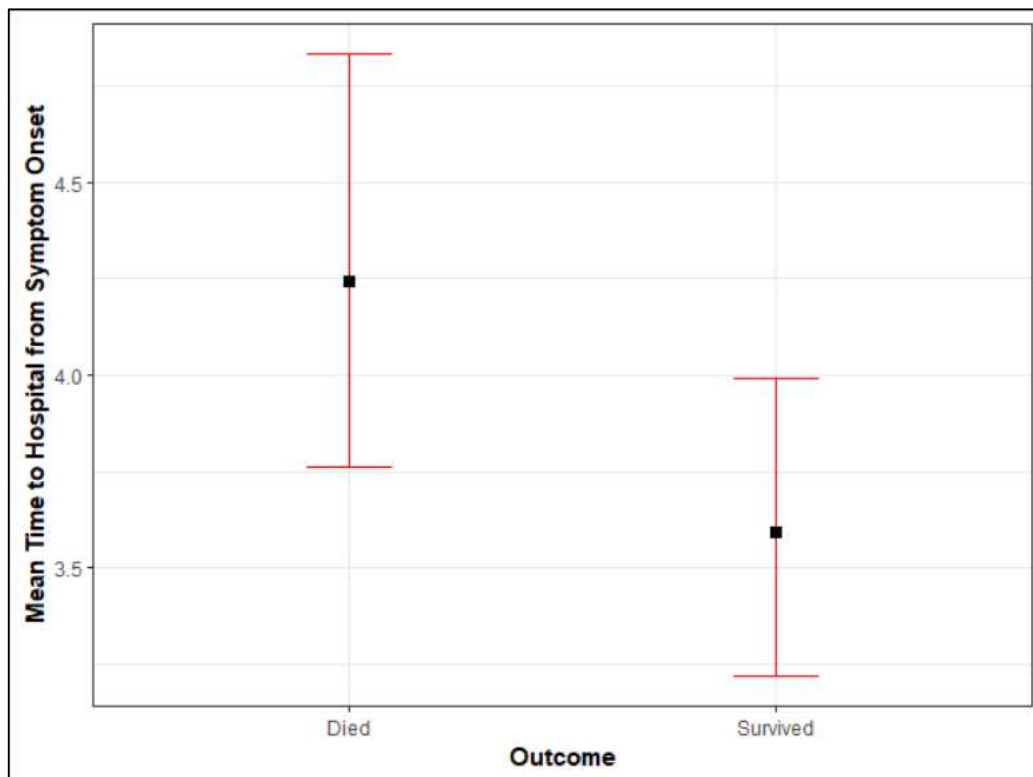


Figure 5: Mean plot of Time to Hospital from Symptom Onset over outcome.

The following table gives the comparison of Respiratory rate and SPO2 at admission over outcome.

Table 8: Comparison of Respiratory rate and SPO2 at admission over outcome.

Variable	Outcome		Total	p-value
	Died	Survived		
Respiratory rate	27.81 ± 5.86 27 (12, 45)	18.8 ± 5.16 18 (9, 32)	23.36 ± 7.12 23 (9, 45)	< 0.001 ^{MW*}
SPO2	84.52 ± 14.82 88 (31, 100)	94.05 ± 4.47 96 (77, 100)	89.23 ± 11.96 93 (31, 100)	< 0.001 ^{MW*}

Abbreviation: MW – Mann Whitney U test, * indicates statistical significance.

From Mann Whitney U test, it is observed that, there is significant difference in the distribution of respiratory rate and SPO2 at admission over outcome. Further, it can be noted that mean respiratory rate is more in those who didn't survive and mean SOP2 admission is more in those who survived.

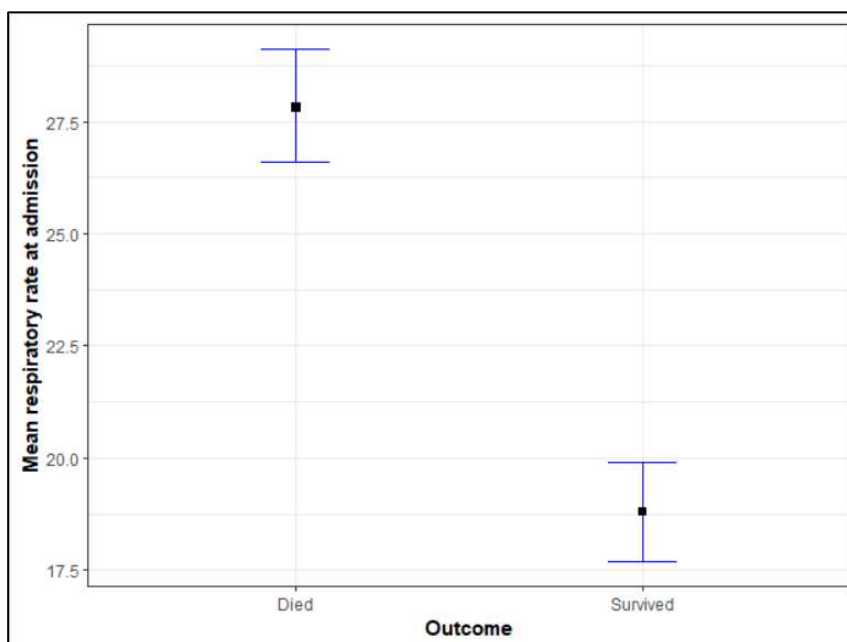


Figure 6: Mean plot of Respiratory rate at admission over outcome.

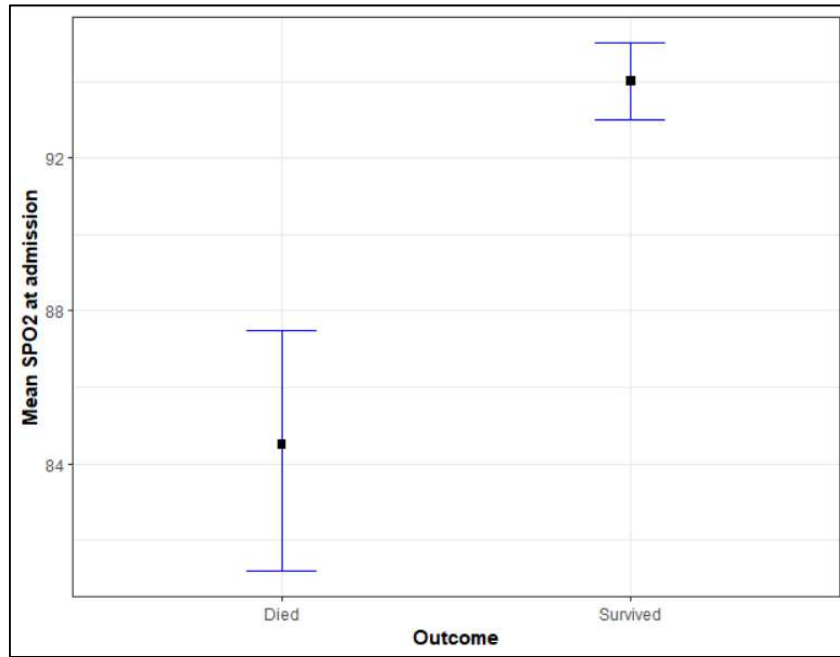


Figure 7: Mean plot of SPO2 at admission over outcome.

The following table gives the comparison of comorbidity over outcome.

Table 9: Comparison of Comorbidity over outcome.

Comorbidity	Outcome		Total	p-value
	Died	Survived		
DM	54 (65.06%)	36 (44.44%)	90 (54.88%)	0.008^{C*}
HTN	43 (51.81%)	31 (38.27%)	74 (45.12%)	0.0816 ^C
IHD	13 (15.66%)	4 (4.94%)	17 (10.37%)	0.0243^{C*}
CKD	5 (6.02%)	2 (2.47%)	7 (4.27%)	0.4598 ^{MC}
OSA	1 (1.2%)	0	1 (0.61%)	1 ^{MC}
Obesity	1 (1.2%)	0	1 (0.61%)	1 ^{MC}
Asthma	1 (1.2%)	3 (3.7%)	4 (2.44%)	0.3638 ^{MC}
BPH	0	1 (1.23%)	1 (0.61%)	0.5062 ^{MC}
CA Breast	0	1 (1.23%)	1 (0.61%)	0.5062 ^{MC}
Parkinson's	0	1 (1.23%)	1 (0.61%)	0.5062 ^{MC}
AKI	1 (1.2%)	0	1 (0.61%)	1 ^{MC}
CVA	1 (1.2%)	0	1 (0.61%)	1 ^{MC}
TB	2 (2.41%)	1 (1.23%)	3 (1.83%)	1 ^{MC}
Hypothyroidism	2 (2.41%)	0	2 (1.22%)	0.5067 ^{MC}

Abbreviation: C – Chi square test, MC – Chi square test with Monte Carlo simulation, * indicates statistical significance.

From Chi square test, it is observed that, there is significant difference in the distribution of DM and IHD over the outcome. Further, it can be noted that the proportion of DM and IHD is more in case of subjects who didn't survive. There is no significant difference in the distribution of other comorbidity over outcome.

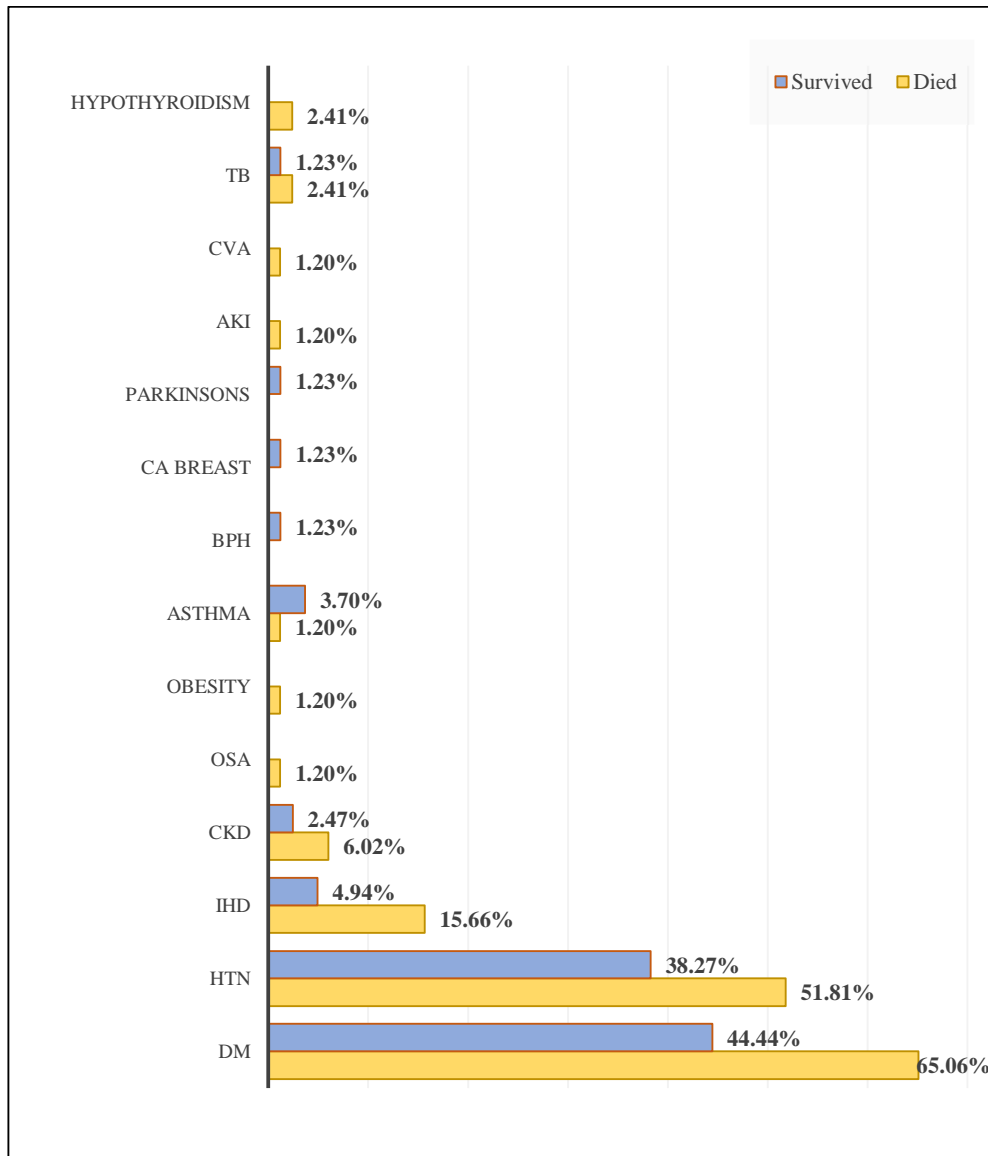


Figure 8: Distribution of comorbidities over outcome.

The following table gives the comparison of different parameters over outcome.

Table 10: Comparison of different parameters over outcome.

Variable	Outcome		Total	p-value
	Died	Survived		
CT severity score	19.35 ± 4.12 20 (5, 25)	12.91 ± 5.11 13 (4, 26)	16.17 ± 5.64 16 (4, 26)	< 0.001 ^{MW*}
Hb	12.87 ± 2.05 12.6 (8.5, 18)	12.53 ± 2.23 12.4 (7.3, 18.3)	12.7 ± 2.14 12.55 (7.3, 18.3)	0.2985 ^t
TLC	12.5 ± 7.53 11.6 (2.6, 47.1)	8.26 ± 3.5 7.2 (4.1, 21.6)	10.4 ± 6.25 9.15 (2.6, 47.1)	< 0.001 ^{MW*}
Neutrophil (%)	84.01 ± 10.12 87 (62, 98)	69.26 ± 13.57 70 (41, 95)	76.73 ± 14.02 77 (41, 98)	< 0.001 ^{MW*}
Lymphocyte (%)	11.49 ± 10.1 8 (1, 64)	22.53 ± 12.2 20 (3, 48)	16.95 ± 12.45 14 (1, 64)	< 0.001 ^{MW*}
Platelet (in thousand)	208.73 ± 98.5 195 (38, 464)	235.76 ± 84.15 229 (2.29, 439)	222.09 ± 92.41 214 (2.29, 464)	0.055 ^{MW}
Ferritin	1217.9 ± 561.05 1133 (46.52, 3037)	643.16 ± 363.05 629 (27.38, 2000)	934.03 ± 553.28 847.3 (27.38, 3037)	< 0.001 ^{MW*}
Transferrin	201.6 ± 60.69 190 (102, 396)	279.07 ± 56.79 273 (165, 398)	239.87 ± 70.32 243 (102, 398)	< 0.001 ^{MW*}
Ferritin/Transferrin ratio	6.67 ± 3.58 6.22 (0.19, 18.52)	2.34 ± 1.35 2.24 (0.13, 8.03)	4.53 ± 3.47 3.26 (0.13, 18.52)	< 0.001 ^{MW*}
LDH	673.76 ± 435.42 630 (126.1, 3650)	369.37 ± 160.3 353 (105, 1012)	522.5 ± 361.65 425 (105, 3650)	< 0.001 ^{MW*}
hsCRP	171.52 ± 100.99 178 (11, 652)	64.75 ± 53.06 43 (7, 221)	118.79 ± 96.85 89 (7, 652)	< 0.001 ^{MW*}
IL-6	96.94 ± 59.92 89 (10.05, 289)	69.29 ± 56.35 52.2 (4.54, 314.8)	83.28 ± 59.64 69.95 (4.54, 314.8)	< 0.001 ^{MW*}
d-Dimer	2749.53 ± 1682.17 2345 (244, 5000)	1221.6 ± 1060.87 893 (344, 5000)	1994.88 ± 1600.94 1259 (244, 5000)	< 0.001 ^{MW*}
Urea	64.34 ± 44.16 48.5 (18, 265)	32.53 ± 16.09 31 (8, 85)	48.53 ± 36.84 36 (8, 265)	< 0.001 ^{MW*}
Creatinine	1.54 ± 1.72	1.1 ± 0.71	1.32 ± 1.33	0.0020 ^{MW*}

	1.1 (0.6, 11.2)	0.98 (0.49, 6.4)	1.02 (0.49, 11.2)	
Bilirubin	1.01 ± 1.31 0.68 (0.14, 8.26)	0.84 ± 0.99 0.68 (0.13, 9)	0.93 ± 1.16 0.68 (0.13, 9)	0.793 ^{MW}
SGOT	146.68 ± 818.43 40.5 (12, 7437)	31.86 ± 19.13 28 (11, 116)	89.63 ± 581.73 32 (11, 7437)	< 0.001 ^{MW*}
SGPT	102.79 ± 583.85 31.5 (11, 5318)	29.38 ± 16.51 27 (10, 116)	66.31 ± 414.65 29 (10, 5318)	0.0433 ^{MW*}
RBS	223.66 ± 100.12 212 (45, 509)	163.9 ± 73.69 137 (26, 380)	193.96 ± 92.69 175 (26, 509)	< 0.001 ^{MW*}

Abbreviation: MW – Mann Whitney U test, t – Two sample t test, * indicates statistical significance.

From Mann Whitney U test, it is observed that, there is significant difference in the distribution of CT severity score, TLC, Neutrophil, Lymphocyte, Ferritin, Transferrin, Ferritin/Transferrin ratio, LDH, hsCRP, IL-6, d-Dimer, Urea, Creatinine, SGOT, SGPT and RBS over outcome. There is no significant difference in the distribution of Bilirubin over outcome.

From two sample t test, it is observed that, there is no significant difference in mean HB over outcome.

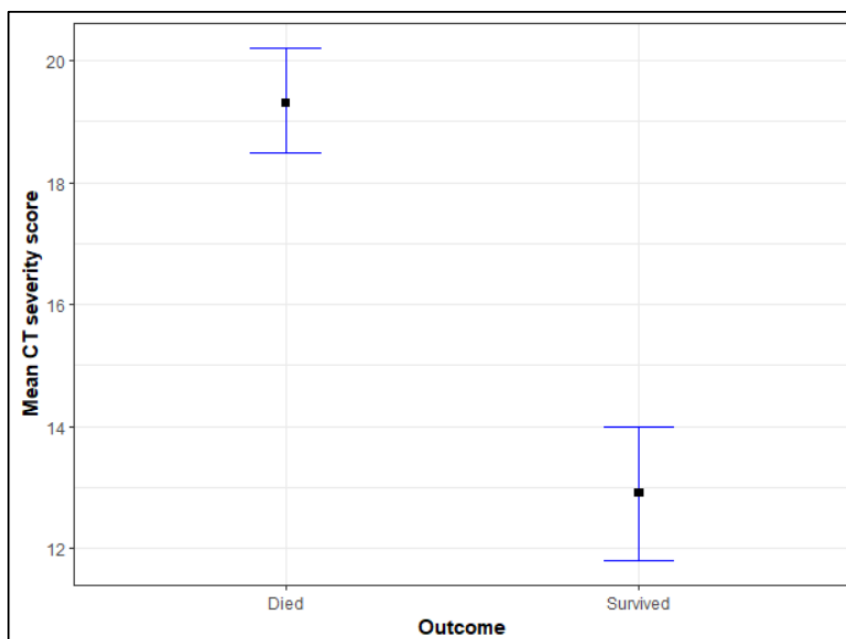


Figure 9: Mean plot of CT severity score over outcome.

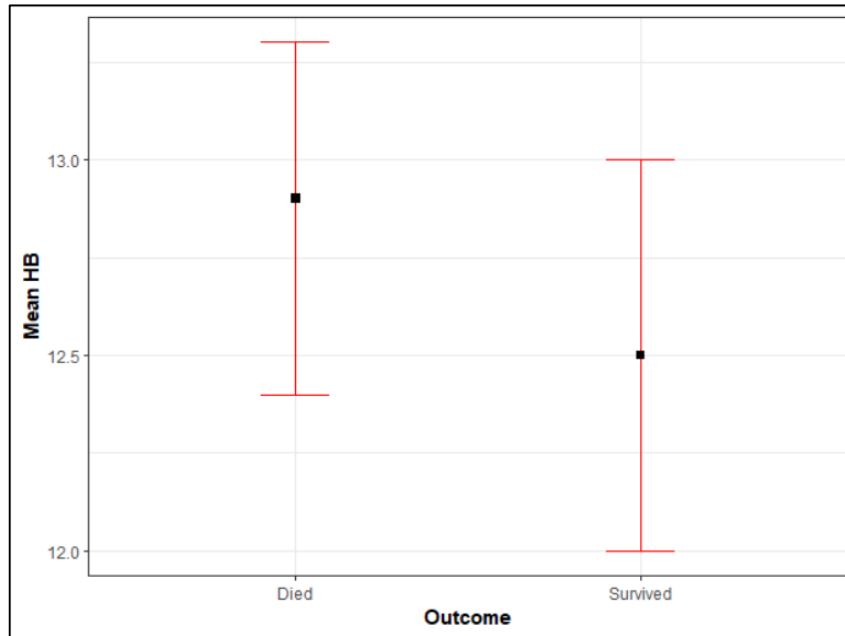


Figure 10: Mean plot of HB over outcome.

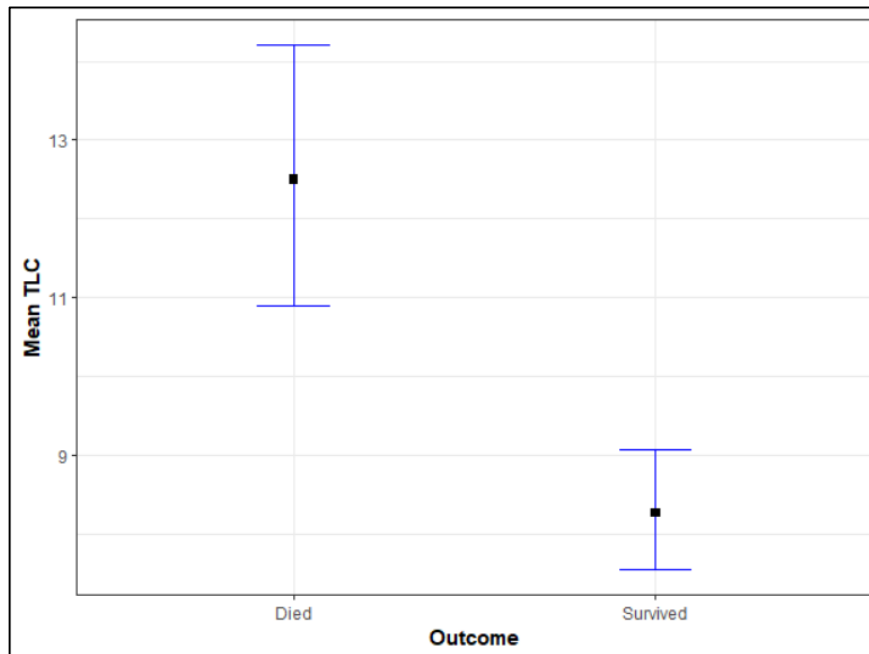


Figure 11: Mean plot of TLC over outcome.

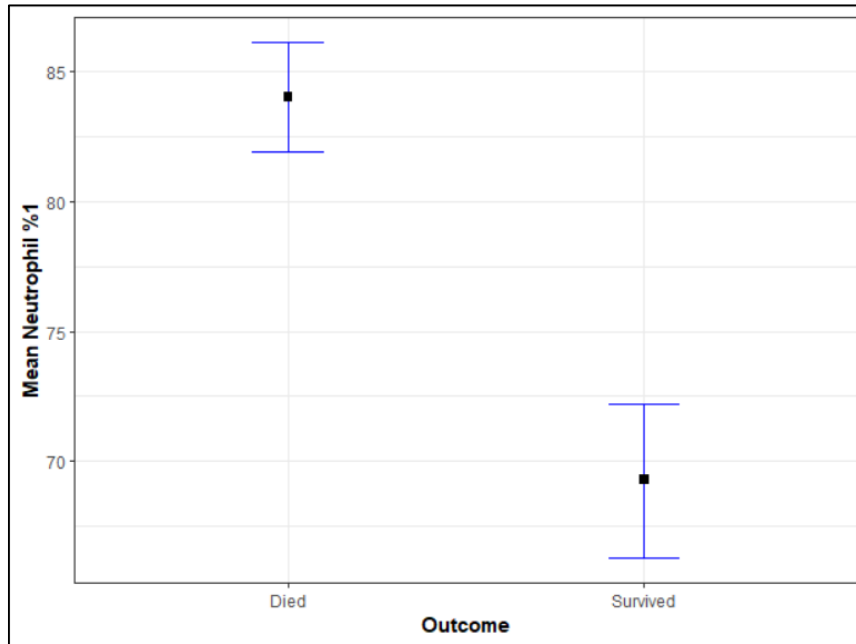


Figure 12: Mean plot of neutrophil over outcome.

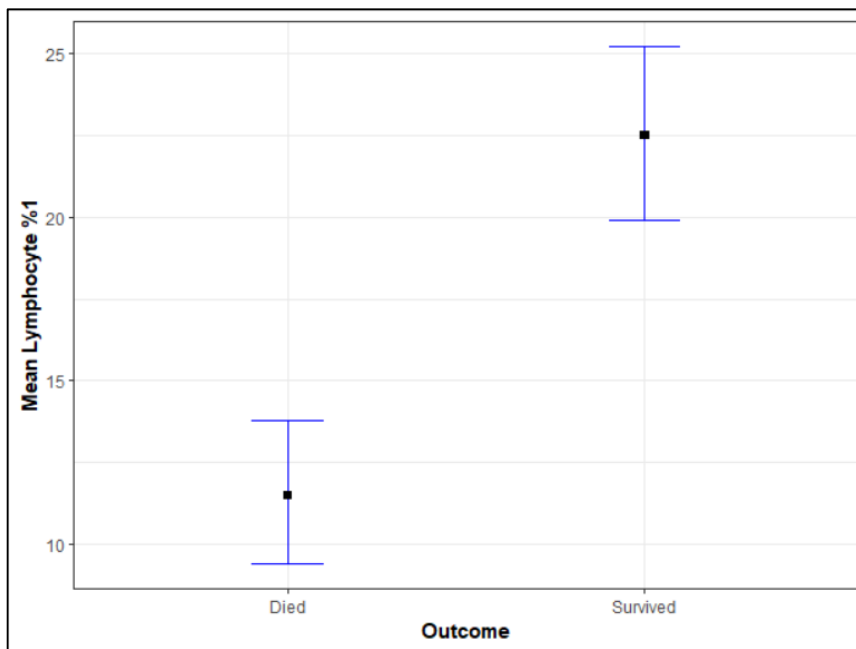


Figure 13: Mean plot of lymphocyte over outcome.

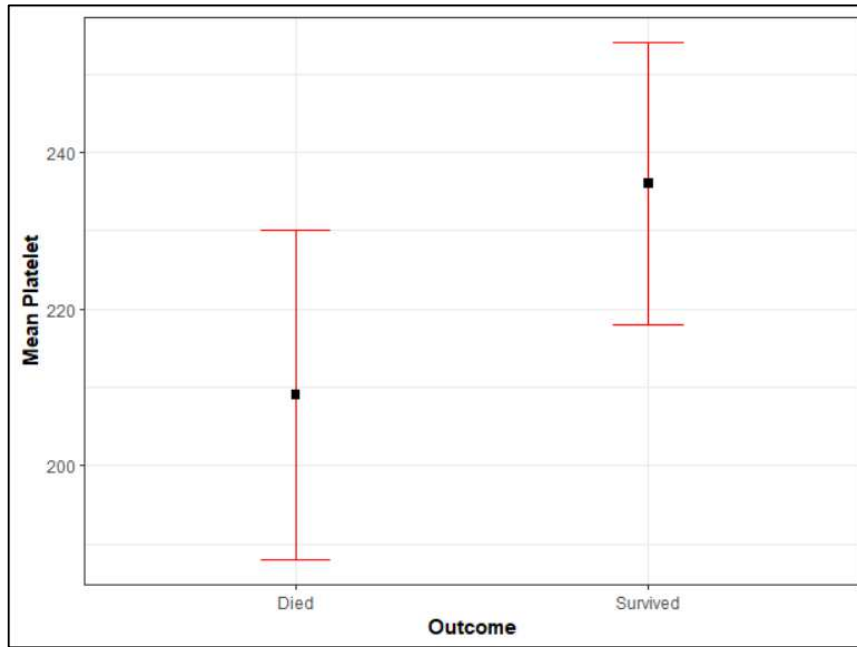


Figure 14: Mean plot of platelet over outcome.

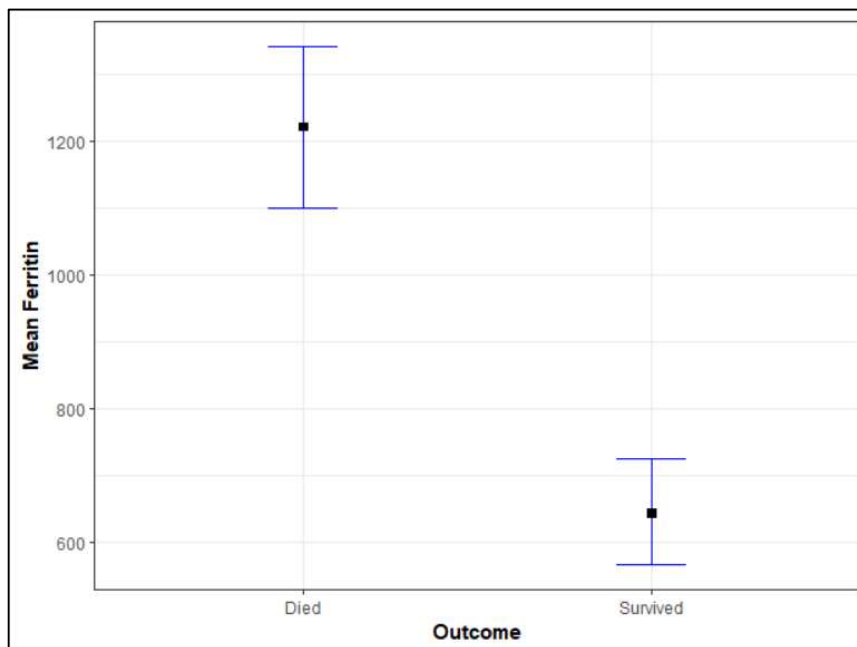


Figure 15: Mean plot of Ferritin over outcome.

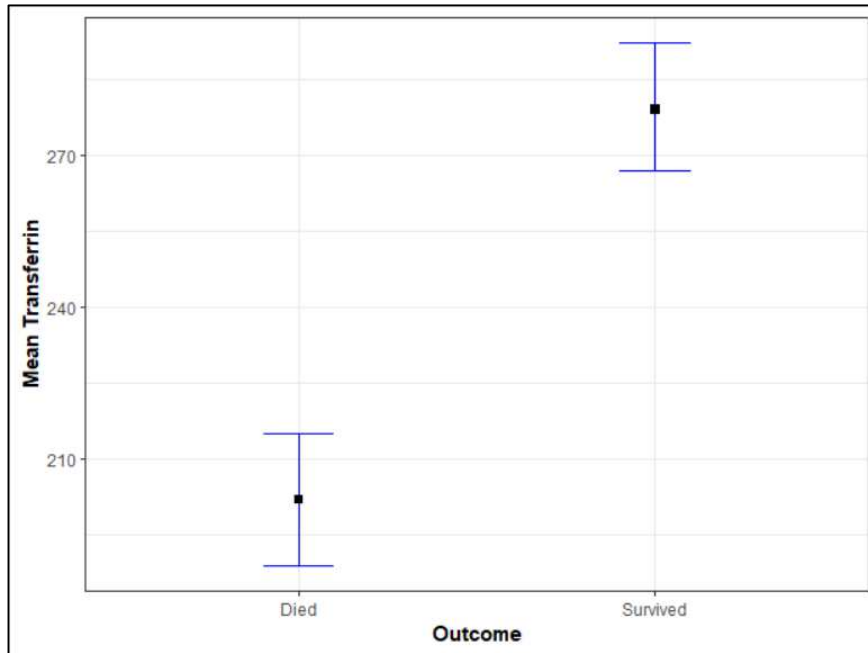


Figure 16: Mean plot of Transferrin over outcome.

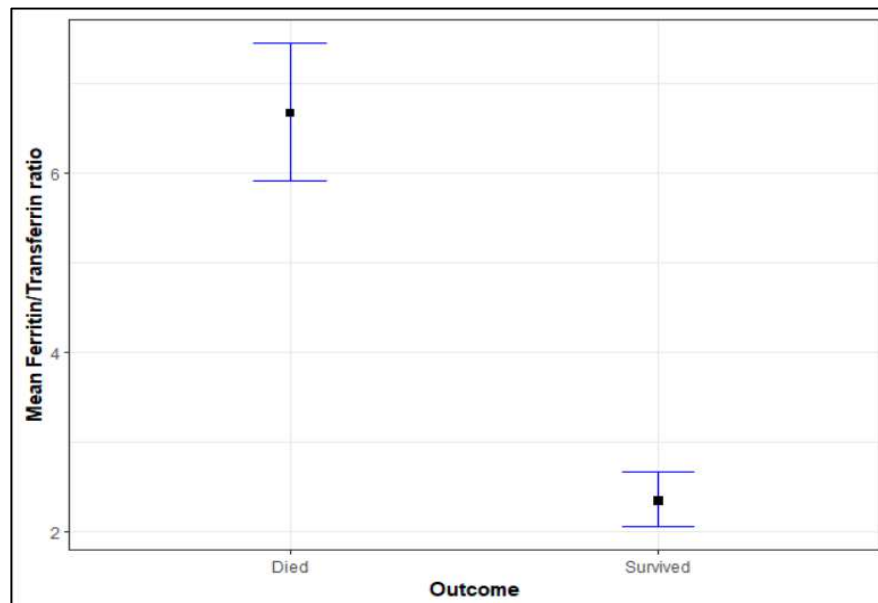


Figure 17: Mean plot of Ferritin/Transferrin ratio over outcome.

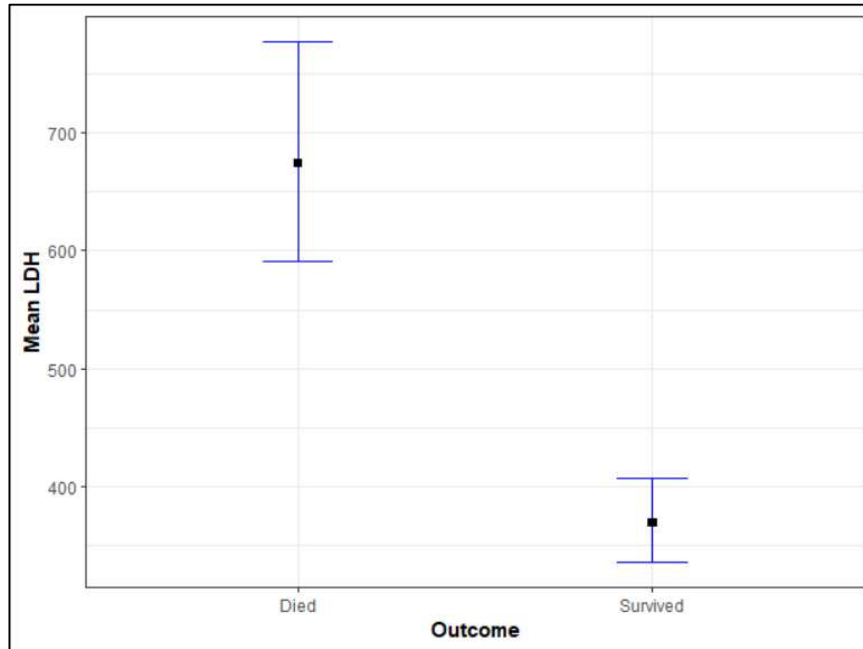


Figure 18: Mean plot of LDH over outcome.

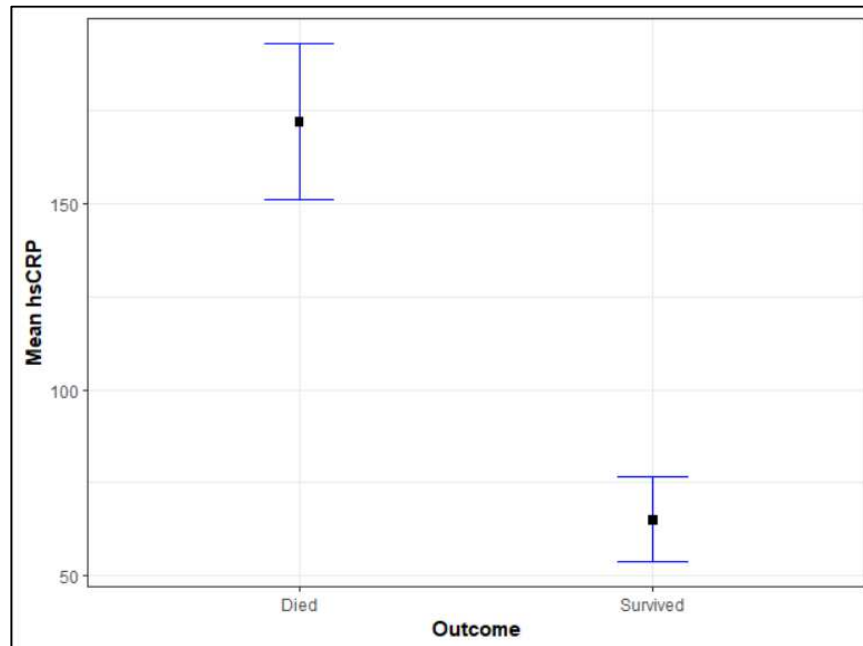


Figure 19: Mean plot of hsCRP over outcome.

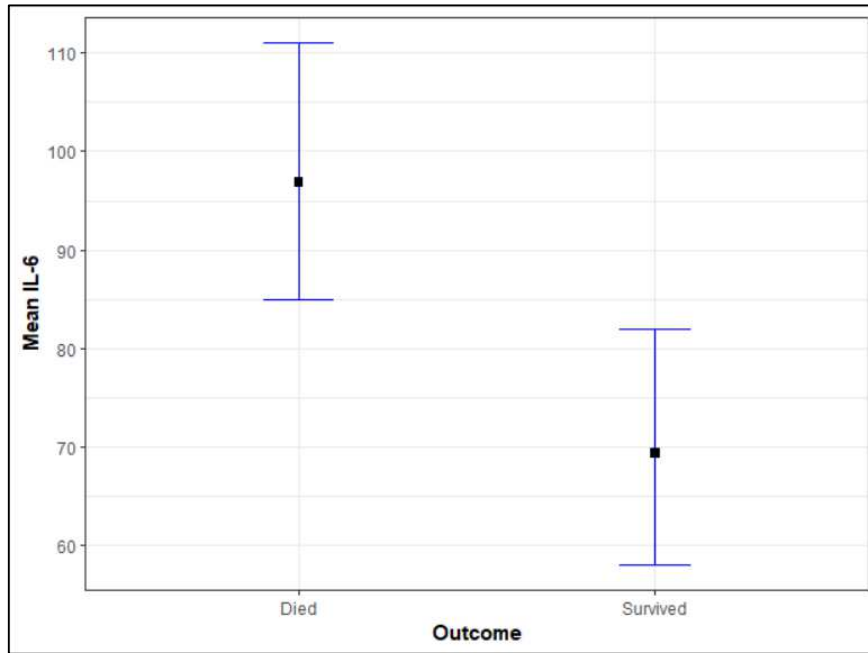


Figure 20: Mean plot of IL-6 over outcome.

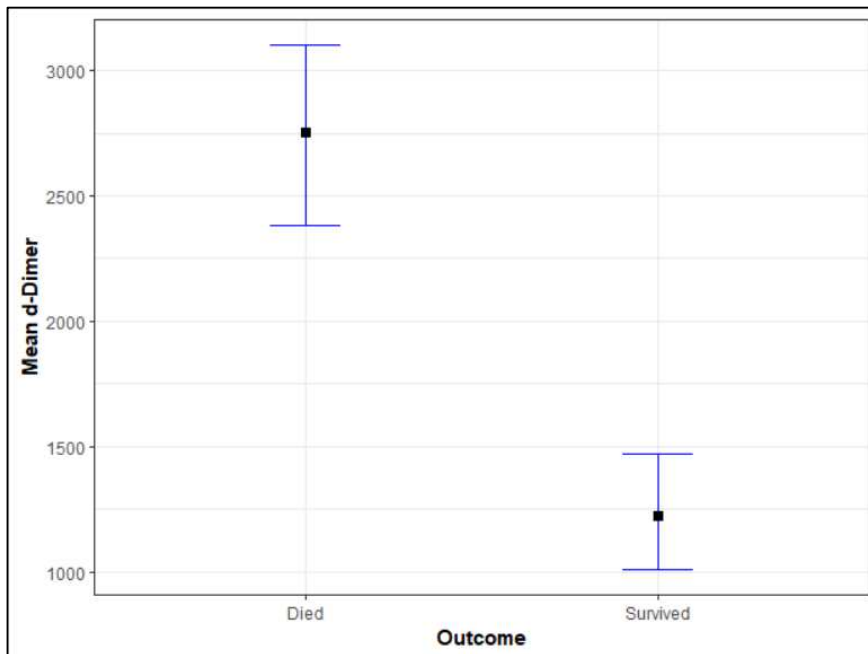


Figure 21: Mean plot of d-Dimer over outcome.

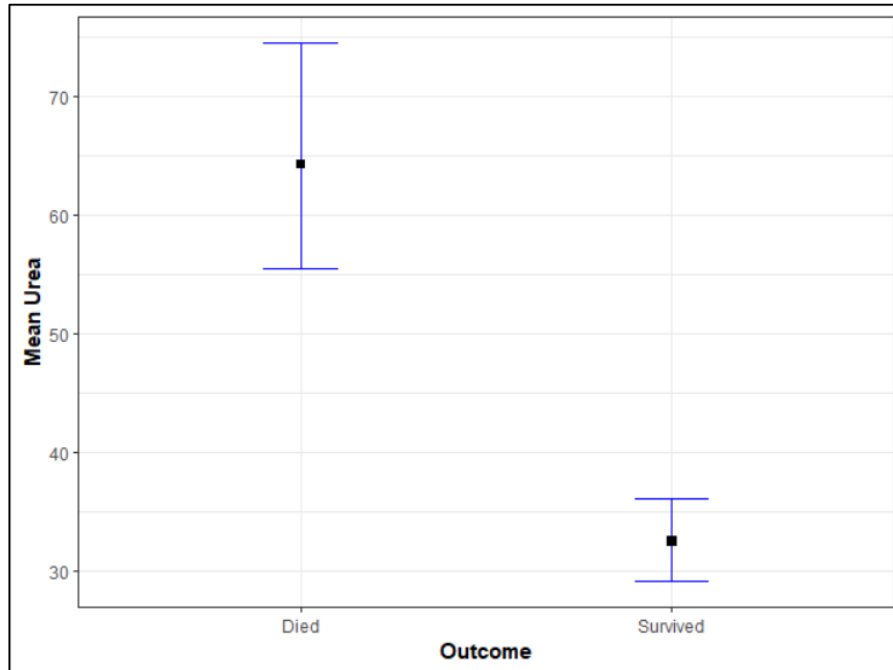


Figure 22: Mean plot of urea over outcome.

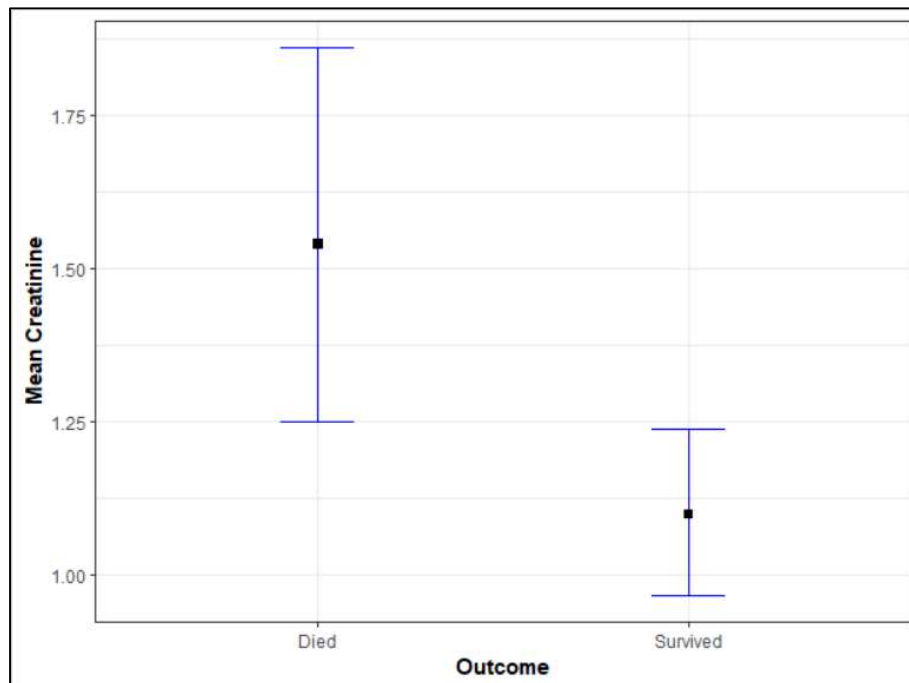


Figure 23: Mean plot of creatinine over outcome.

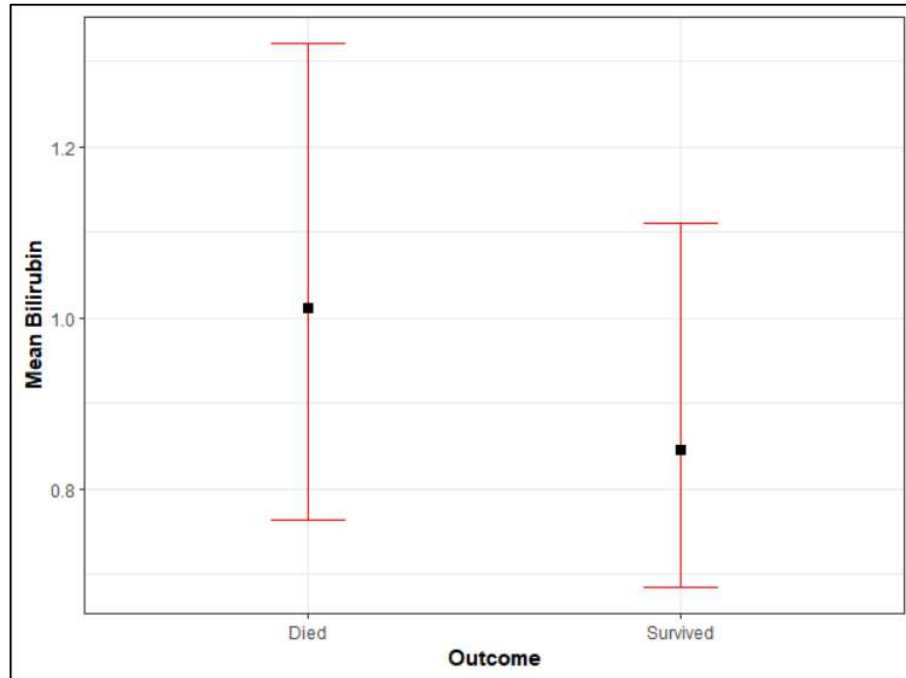


Figure 24: Mean plot of bilirubin over outcome.

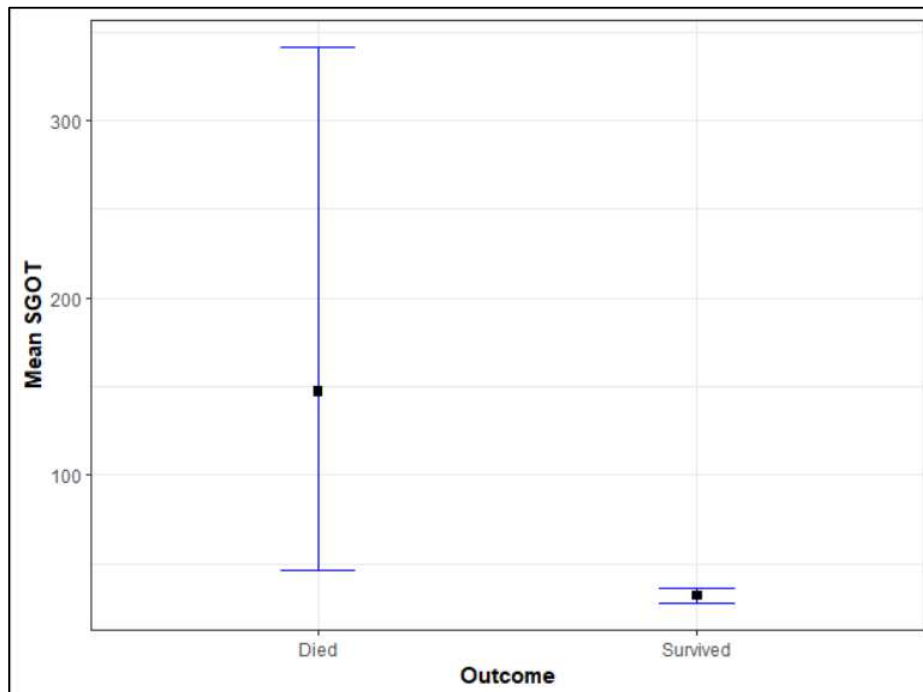


Figure 25: Mean plot of SGOT over outcome.

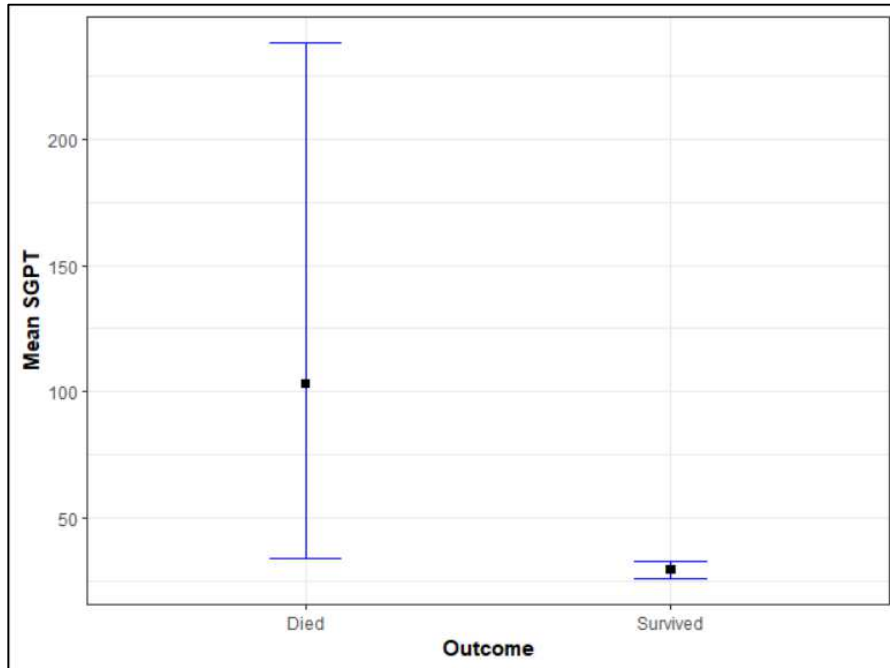


Figure 26: Mean plot of SGPT over outcome.

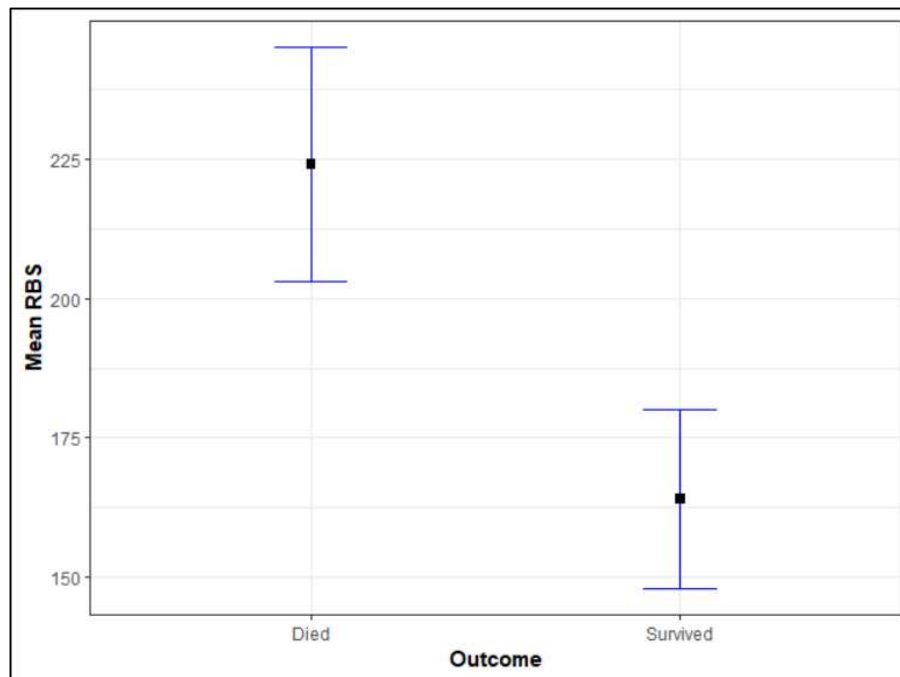


Figure 27: Mean plot of RBS over outcome.

The following table gives the comparison of treatment given over outcome.

Table 11: Comparison of Treatment given over outcome.

Treatment	Sub Category	Outcome		Total	p-value
		Died	Survived		
Remdesivir	No	15 (18.07%)	8 (9.88%)	23 (14.02%)	0.1016 ^C
	Yes	66 (79.52%)	75 (92.59%)	141 (85.98%)	
Tocilizumab	No	80 (96.39%)	57 (70.37%)	137 (83.54%)	< 0.001 ^{MC*}
	Yes	1 (1.2%)	26 (32.1%)	27 (16.46%)	
Type of Steroid used	Dexamethasone	4 (4.82%)	16 (19.75%)	20 (12.2%)	0.0090 ^{MC*}
	Methyl prednisolone	76 (91.57%)	65 (80.25%)	141 (85.98%)	
	Methyl prednisolone, Dexamethasone	1 (1.2%)	0	1 (0.61%)	

Abbreviation: C – Chi square test, MC – Chi square test with Monte Carlo simulation, * indicates statistical significance.

From Chi square test, it is observed that, there is significant difference in the distribution of use of Tocilizumab over outcome. There is significant difference in the distribution of steroid used over outcome. There is no significant difference in the distribution of use of Remdesivir over outcomes.

The following table gives the comparison of hospital stay over outcome.

Table 12: Comparison of Hospital Stay over outcome.

Variables	Outcome		Total	p-value
	Died	Survived		
Duration of Hospital stay	5.8 ± 4.31	6.35 ± 4.23	6.07 ± 4.27	0.294 ^{MW}
	5 (0, 19)	6 (1, 24)	5 (0, 24)	

Abbreviation: MW – Mann Whitney U test.

From Mann Whitney U test, it is observed that, there is no significant difference in the distribution of hospital stay over outcome.

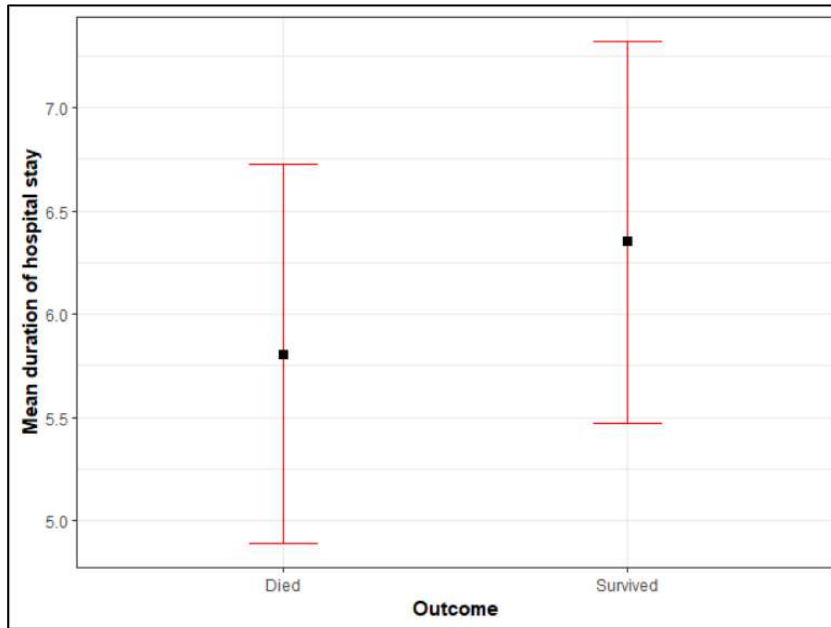


Figure 28: Mean plot of duration of hospital stay over outcome.

The following table gives diagnostic analysis of Ferritin/Transferrin ratio for outcome.

Table 13: Diagnostic analysis of Ferritin/Transferrin ratio for outcome.

	Ferritin/Transferrin ratio
Sensitivity (95% CI)	88.89% (79.95% - 94.79%)
Specificity (95% CI)	83.13% (73.32% - 90.46%)
PPV (95% CI)	83.72% (74.2% - 90.8%)
NPV (95% CI)	88.46% (79.22% - 94.59%)
Accuracy (95% CI)	85.98% (79.7% - 90.9%)
AU-ROC (95% CI)	0.8853 (0.8295 - 0.9412)
p-value	< 0.001*

Abbreviation: * indicates statistical significance.

The AUC for Ferritin/Transferrin ratio is 0.8853 with 88.89% sensitivity and 83.13% specificity in predicting mortality.

From logistic regression, it is observed that, Ferritin/Transferrin ratio has significant effect on mortality (p-value < 0.001). The odds of death increase by 2.24 (95% CI: 1.77 - 2.99) with the unit increase of Ferritin/Transferrin ratio.

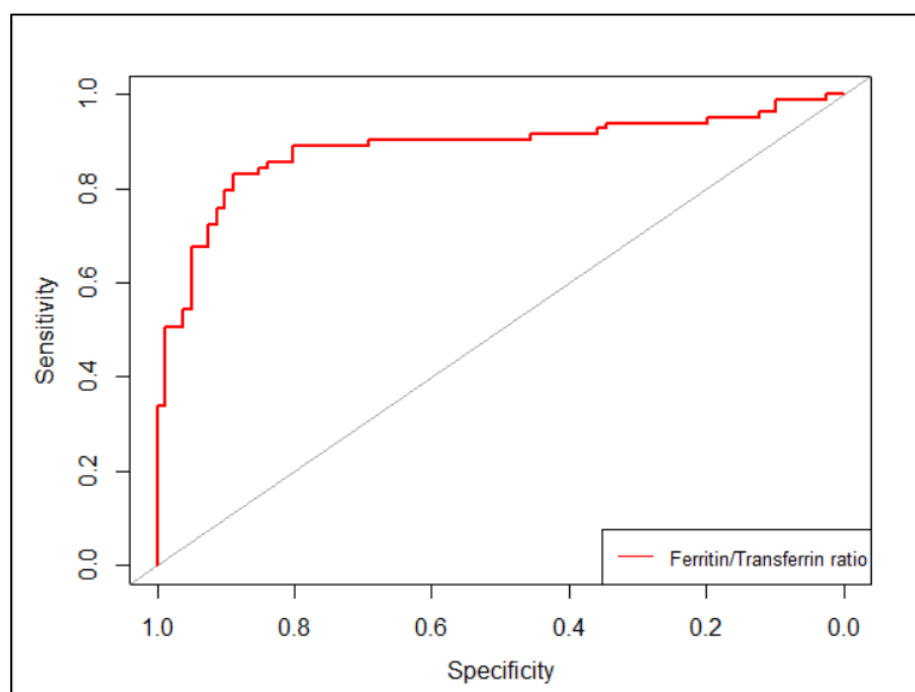


Figure 29: ROC curve of Ferritin/Transferrin ratio for predicting mortality outcome.

DISCUSSION

The expression of transferrin, a known pro-coagulant was upregulated in SARS-CoV-2 infected cells, increased with age and was higher in males than in females. Moreover, a rise of Serum Ferritin levels was observed in patients during COVID-19 disease progression further cementing the link between Iron Profile biomarkers like Transferrin and Ferritin and the pathogenesis and severity of COVID-19 infection. Locally produced Transferrin, independent of circulating Transferrin levels may contribute to COVID-19 pathology. For example, Transferrin is locally produced by neurological tissue like brain and spinal cord and higher levels are associated with an increased incidence of ischemic stroke, haemorrhagic stroke and hypercoagulability. Apart from hypercoagulability which predisposes COVID-19 affected patients to ischemic stroke, Transferrin also causes cytotoxic damage associated with haemorrhagic stroke by increasing cellular uptake of iron. Higher levels of transferrin are also found to be associated with Metabolic syndrome and Diabetes which have shown to exacerbate the severity of COVID-19 disease.⁵³

In the present study, a total of 168 patients with COVID-19 were evaluated and the mean age of patients was found to be of 57.35 ± 16.59 years of age in which 84% were male patients and 16% were female patients with male preponderance. In this study, the mean age of the patients with mortality was higher compared to patients which survived. Among the study participants, the gender distribution was comparable between both the groups.

On assessment of days of admission after onset of symptoms to hospital it was found that there was no significant difference in the distribution of time to hospital from symptom onset over outcome. Similarly, there was no significant difference in the distribution of hospital stay over outcome. However, during the analysis of

comparison of symptoms over outcome, it was observed that there was a significant difference in the distribution of breathlessness over the outcome and breathlessness symptom was observed more in cases of subjects which didn't survive, although there was no significant difference in the distribution of other symptoms over outcome. Among the comorbidities, it was observed that there was a significant difference in the distribution of DM and IHD over the outcome. Further, it was noted that the proportion of DM and IHD was more in cases of subjects which didn't survive. There was no significant difference in the distribution of other comorbidity over outcome.

On assessment of the Respiratory rate and the SPO2 on admission, it was observed that there was a significant difference in the distribution of Respiratory rate and SPO2 at admission over outcome. It was noted that the mean Respiratory rate and mean SPO2 on admission was more in subjects which didn't survive.

In a study by Weiler RB et al., it was shown that increased Ferritin/Transferrin ratio was associated with a higher risk for the need of mechanical ventilation and a higher incidence of ICU admission among COVID-19 patients, thus establishing a relation between Iron Profile markers and COVID-19 severity. Studies trying to establish a particular Ferritin/Transferrin ratio have so far been inconclusive although in their study, a Ferritin/Transferrin ratio of more than 10 suggested a higher risk of admission in the ICU and a higher incidence of patients requiring mechanical ventilation with the odds ratio being 5.702 and 8.054 at 95% confidence interval respectively.³ Reduced serum Transferrin and Iron levels, as well as elevated CRP and Ferritin were substantially linked to COVID-19's heightened inflammatory and immunological status. Therefore, Transferrin, Iron levels, CRP and Serum Ferritin can be utilised as a useful predictor of disease severity and progression.⁵⁶

In this study, it was observed that there was a significant difference in the distribution of CT severity score, TLC, Neutrophil, Lymphocyte, Ferritin, Transferrin, Ferritin/Transferrin ratio, LDH, hsCRP, IL-6, d-Dimer, Urea, Creatinine, SGOT, SGPT and RBS over outcome. However, there was no significant difference in the distribution of Bilirubin over outcome. There were significantly higher neutrophils among the patients of mortality compared to survived and significantly lower lymphocyte and platelet count among the patients which died compared to survived. There was significantly higher Ferritin/Transferrin ratio among the patients with mortality as compared to the patients which survived. Similarly, the mean level of Ferritin was significantly higher and mean Transferrin significantly lower in mortality patients compared to the survived patients. ($p < 0.001$)

In the present study there is a significant difference in the distribution of steroid use over outcome. The mean dose of prednisolone and dexamethasone was significantly higher among the patients with mortality compared to the survived patients. There is no significant difference in the distribution of use of Remdesivir over outcomes. In the study, the AUC for Ferritin/Transferrin ratio is 0.8853 with 88.89% sensitivity and 83.13% specificity in predicting mortality. From logistic regression, it was observed that, Ferritin/Transferrin ratio had a significant effect on mortality ($p\text{-value} < 0.001$) with the odds of death increasing by 2.24 (95% CI: 1.77 - 2.99) with every unit increase of Ferritin/Transferrin ratio.

Yanling LV et al. also documented that the risk of Multiple Organ Damage as well as increased severity was seen with patients with a lower level of serum Iron. The findings of this study concluded that the above association between Iron parameters and COVID-19 severity may facilitate the use of Iron parameters as valuable biomarkers for assessing the risk factors and prognosis of COVID-19.⁵⁹

In a study conducted by Bastin A et al., both Iron and Ferritin had a higher AUC for COVID 19 disease prognosis but when it came to disease severity, Serum Ferritin had the highest AUC. This indicated that routine measurement of Serum Iron and Ferritin can help us to determine the prognosis and severity of the disease. Based on the results of the above study, the use of iron chelators to decrease the amount of iron intake can be of significant therapeutic benefit.⁵⁵

CONCLUSION

The present study documented the significant correlation of Ferritin/Transferrin ratio with COVID-19 infection and a higher incidence of mortality among the patients with higher Ferritin, low Transferrin and a significantly higher Ferritin/Transferrin ratio. There is a significant association of mortality among the patients with a higher Ferritin/Transferrin ratio.

SUMMARY

- In the present study, a total of 168 patients with COVID-19 were evaluated in which the mean age of patients was found to be 57.35 ± 16.59 yrs. of age wherein 84% were male patients and 16% were female patients with male preponderance.
- In our analysis, the mean age of the patients with mortality was higher compared to patients which survived.
- Among the study participants, the gender distribution was comparable between both the groups.
- On assessment of days of admission after the onset of symptoms to hospital, it was found that there was no significant difference in the distribution of time to hospital from symptom onset over outcome.
- On assessment of the Respiratory Rate and the SPO2 at admission among the study participants it was observed that there was a significant difference in the distribution of Respiratory Rate and SPO2 at admission over outcome. Further, it can be noted that mean Respiratory Rate and mean SPO2 admission was more in those who didn't survive.
- It was observed that there was a significant difference in the distribution of O2 modalities over the outcome with a higher need of NIV (ventilatory support) among mortality patients.
- In this study, the CT severity score on admission was significantly higher among the patients with mortality as compared to those who survived.

- In our analysis, there were significantly higher neutrophils among the patients of mortality compared to survived and significantly lower lymphocyte and platelet count among the patients which died compared to survived.
- Among the comorbidities, it was observed that, there is a significant difference in the distribution of DM and IHD over the outcome. Further, it can be noted that the proportion of DM and IHD is more in cases of subjects which didn't survive.
- It was observed that, there is significant difference in the distribution of TLC, Neutrophil, Lymphocyte, Ferritin, Transferrin, Ferritin/Transferrin ratio, LDH, hsCRP, IL-6, d-Dimer, Urea, Creatinine, SGOT, SGPT and RBS over outcome. However, there was no significant difference in the distribution of Bilirubin over outcome
- In the present study there is significantly higher Ferritin upon Transferrin ratio among the patients with mortality as compared to patients which survived. Similarly, the mean level of Ferritin was significantly higher and Transferrin was significantly lower in mortality patients as compared to the survived patients. (p<0.001)
- In the present study, the mean dose of prednisolone and dexamethasone was significantly higher among the patients with mortality as compared to those which survived.
- In this study, the Area Under Curve (0.8853) for the Ferritin/Transferrin ratio was significantly positively related with the prediction of mortality among the patients (p value <0.001) with a sensitivity of 88.89% (95% CI) and a specificity of 83.13% (95% CI).

Limitation Of the Study

1. The mean age of the population 57.35 ± 16.59 yrs, was towards a higher side with an unequal distribution of sex among the participants wherein 84% were male patients and 16% were female patients with a male preponderance.
2. This was a single center observational study with a limited sample size.
3. The duration of the study was spread over for only one year.
4. There was no discernible method to figure out if the alterations in Iron Profile parameters were caused by preexisting anemia and whether it had any bearing on the final outcome.

Future Perspective

The findings of this study concluded that the above association between Iron parameters and COVID severity may facilitate the use of Iron parameters as valuable biomarkers for assessing the risk factors and prognosis of COVID-19. Future studies are warranted utilizing the predictive models using Serum Ferritin, Serum Transferrin and Ferritin/Transferrin ratio at the time of admission to predict ICU admission, management and prognosis among COVID-19 patients.

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ANNEXURE I
INFORMED CONSENT

Dear Mr./Mrs./Dr. _____, you are kindly requested to enroll yourself in a research study titled, **“To Study the correlation of Ferritin/Transferrin ratio with the severity of COVID-19 infection - A One year Cross Sectional Study done in KLES Dr. Prabhakar Kore Hospital and Medical Research Centre. Belagavi.”** being conducted by REG NO: BG0120015, a post graduate student in M.D. General Medicine and the study will be carried out under the direct supervision and guidance of Dr. _____, Associate Professor, Department of General Medicine, Jawaharlal Nehru Medical College, Belgaum.

You have been requested to participate in this as you fit into the laid out criteria for a study ‘subject’/ participant.

Your participation in study is voluntary. During the study you will be asked some questions and you are supposed to answer to the best of your knowledge. Your decision whether or not to participate in the study will not affect your treatment in any form. If you decide to participate you are free to withdraw at any time.

TITLE OF THE STUDY:

“To Study the correlation of Ferritin/Transferrin ratio with the severity of COVID-19 infection - A One year Cross Sectional Study done in KLES Dr. Prabhakar Kore Hospital and Medical Research Centre. Belagavi.”

PURPOSE OF THE STUDY: To study the correlation of Ferritin/Transferrin ratio with COVID-19 infection and to assess its predictive value on the severity of the disease.

PROCEDURES INVOLVED:

If you agree to enroll yourself in my study, you will be interviewed regarding your present, past and family history then you will be clinically examined in detail and investigated accordingly.

Then you will be subjected to a few blood investigations, namely complete blood counts, Iron Profile, random blood glucose, renal function tests, liver function tests, urine routine and microscopy, D dimer, LDH, serum ferritin, IL-6, hsCRP.

RISKS AND BENEFITS:

There are no potential risks involved in this study.

Benefits of taking part in this research:

By taking part in this study, correlation between the Iron Profile, especially Ferritin/Transferrin ratio and the severity of the patient suffering from COVID-19 infection can be found out which may serve as a prognostic indicator for predicting the severity of COVID-19 infection.

VOLUNTARY PARTICIPATION / WITHDRAWAL FROM THE STUDY:

Taking part in the study is voluntary. You may choose not to enroll yourself in this study and may choose to leave the study anytime in between.

ALTERNATIVES:

Your decision regarding participation in study will not change present or future health care services offered to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi. You would simply be excluded from the study if you wish to, and all your details shall be kept confidential and you will get the routine line of management.

PRIVACY AND CONFIDENTIALITY:

All data collected or disclosed by you during the course of participation of study, will be kept fully confidential. If however during the course it becomes necessary for the progress of the course to disclose the identity, it would be done so only after your informed & written consent.

The only people to know that you are a research subject are members of the research team. No information about you will be disclosed to other

without your written permission except:

- In emergency to protect your rights AND welfare.
- If required by law.

AUTHORIZATION TO PUBLISH RESULT:

The results of the study may be used to publish an article. When the results of research published or discussed, in a conference, no information will be displayed that would disclose your identity. Any information obtained in connection with this study and that can be identified with you will remain confidential.

FINANCIAL INCENTIVES FOR PARTICIPATION:

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

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

COMPENSATION:

In the event that you become injured as a result of taking part in this study, treatment will be offered to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum, or you will be given information about where to receive medical care. However, no reimbursement, compensation or free medical care will be given.

QUESTIONS/CONTACT DETAILS:

You shall be free to contact the below mentioned name & addresses anytime during the study period for any clarification or help as you may desire for.

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

1. Dr. _____
Chairman,
J.N.M.C Ethical Committee for
Human Research

2. Dr. _____
Professor and HEAD OF
DEPARTMENT,
Dept. of General Medicine,
JNMC, Belagavi.

3. Dr. _____
Guide,
Associate Professor,
Dept. of General Medicine,
JNMC, Belagavi.

4. REG NO: BG0120015
Investigator,
PG in General Medicine,
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

Date:

Place:

ತಿಳುವಳಿಕೆಯ ಸಮ್ಮತಿ

ಆತ್ಮೀಯ ಶ್ರೀ / ಶ್ರೀ. / ಡಾ. _____, ಓರೆಯಾಗಿರುವ ಸಂಶೋಧನಾ ಅಧ್ಯಯನಕ್ಕೆ ನಿಮ್ಮನ್ನು ಸೇರಿಸಲು ನಿಮ್ಮನ್ನು ದಯೆಯಿಂದ ವಿನಂತಿಸಲಾಗಿದೆ, "ಕೋವಿಡ್ -19 ಸೋಂಕಿನ ತೀವ್ರತೆಯೊಂದಿಗೆ ಫೆರಿಟಿನ್ / ಟ್ರಾನ್ಸ್‌ಫೆರಿನ್ ಅನುಪಾತದ ಪರಸ್ಪರ ಸಂಬಂಧವನ್ನು ಅಧ್ಯಯನ ಮಾಡಲು - ಕೆಎಲ್‌ಇಎಸ್ ಡಾ. ಪ್ರಭಾಕರ್ ಕೋರೆ ಆಸ್ಪತ್ರೆ ಮತ್ತು ವೈದ್ಯಕೀಯ ಸಂಶೋಧನಾ ಕೇಂದ್ರದಲ್ಲಿ ಬೆಳಗಾವಿ ಒಂದು ವರ್ಷದ ಕ್ರಾಸ್ ಸೆಕ್ಷನಲ್ ಸ್ಟಡಿ ಮಾಡಲಾಗಿದೆ". ಎಂ.ಡಿ. ಜನರಲ್ ಮೆಡಿಸಿನ್‌ನಲ್ಲಿ ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿ REG NO: BG0120015 ಅವರು ನಡೆಸಲಿದ್ದಾರೆ ಮತ್ತು ಬೆಳಗಾವಿನ ಜವಾಹರಲಾಲ್ ನೆಹರು ವೈದ್ಯಕೀಯ ಕಾಲೇಜಿನ ಜನರಲ್ ಮೆಡಿಸಿನ್ ವಿಭಾಗದ ಸಹಾಯಕ ಪ್ರಾಧ್ಯಾಪಕ ಡಾ. _____ ಅವರ ನೇರ ಮೇಲ್ವಿಚಾರಣೆ ಮತ್ತು ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ ಈ ಅಧ್ಯಯನವನ್ನು ನಡೆಸಲಾಗುವುದು.

ಅಧ್ಯಯನದ 'ವಿಷಯ' / ಭಾಗವಹಿಸುವವರಿಗೆ ನೀವು ನಿಗದಿಪಡಿಸಿದ ಮಾನದಂಡಗಳಿಗೆ ಸರಿಹೊಂದುವಂತೆ ಇದರಲ್ಲಿ ಭಾಗವಹಿಸಲು ನಿಮ್ಮನ್ನು ವಿನಂತಿಸಲಾಗಿದೆ. ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ. ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ನಿಮ್ಮನ್ನು ಕೆಲವು ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲಾಗುತ್ತದೆ ಮತ್ತು ನಿಮ್ಮ ಉತ್ತಮ ಜ್ಞಾನಕ್ಕೆ ನೀವು ಉತ್ತರಿಸಬೇಕಾಗುತ್ತದೆ. ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಬೇಕೆ ಅಥವಾ ಬೇಡವೇ ಎಂಬ ನಿಮ್ಮ ನಿರ್ಧಾರವು ನಿಮ್ಮ ಚಿಕಿತ್ಸೆಯ ಮೇಲೆ ಯಾವುದೇ ರೂಪದಲ್ಲಿ ಪರಿಣಾಮ ಬೀರುವುದಿಲ್ಲ. ನೀವು ಭಾಗವಹಿಸಲು ನಿರ್ಧರಿಸಿದರೆ ನೀವು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಹಿಂತೆಗೆದುಕೊಳ್ಳಬಹುದು.

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: "ಕೋವಿಡ್ -19 ಸೋಂಕಿನ ತೀವ್ರತೆಯೊಂದಿಗೆ ಫೆರಿಟಿನ್ / ಟ್ರಾನ್ಸ್‌ಫೆರಿನ್ ಅನುಪಾತದ ಪರಸ್ಪರ ಸಂಬಂಧವನ್ನು ಅಧ್ಯಯನ ಮಾಡಲು - ಕೆಎಲ್‌ಇಎಸ್ ಡಾ. ಪ್ರಭಾಕರ್ ಕೋರೆ ಆಸ್ಪತ್ರೆ ಮತ್ತು ವೈದ್ಯಕೀಯ ಸಂಶೋಧನಾ ಕೇಂದ್ರದಲ್ಲಿ ಬೆಳಗಾವಿ ಒಂದು ವರ್ಷದ ಕ್ರಾಸ್ ಸೆಕ್ಷನಲ್ ಸ್ಟಡಿ ಮಾಡಲಾಗಿದೆ".

ಅಧ್ಯಯನದ ಉದ್ದೇಶ:

ಕೋವಿಡ್ - 19 ಸೋಂಕಿನೊಂದಿಗೆ ಫೆರಿಟಿನ್ / ಟ್ರಾನ್ಸ್‌ಫೆರಿನ್ ಅನುಪಾತದ ಪರಸ್ಪರ ಸಂಬಂಧವನ್ನು ಅಧ್ಯಯನ ಮಾಡಲು ಮತ್ತು ರೋಗದ ತೀವ್ರತೆಯ ಮೇಲೆ ಅದರ ಮುನ್ಸೂಚಕ ಮೌಲ್ಯವನ್ನು ನಿರ್ಣಯಿಸಲು.

ಒಳಗೊಂಡಿರುವ ಕಾರ್ಯವಿಧಾನಗಳು:

ನನ್ನ ಅಧ್ಯಯನಕ್ಕೆ ನಿಮ್ಮನ್ನು ಸೇರಿಸಲು ನೀವು ಒಪ್ಪಿದರೆ, ನಿಮ್ಮ ಪ್ರಸ್ತುತ, ಹಿಂದಿನ ಮತ್ತು ಕುಟುಂಬದ ಇತಿಹಾಸದ ಬಗ್ಗೆ ನಿಮ್ಮನ್ನು ಸಂದರ್ಶಿಸಲಾಗುವುದು, ನಂತರ ನಿಮ್ಮನ್ನು ಪ್ರಾಯೋಗಿಕವಾಗಿ ವಿವರವಾಗಿ ಪರಿಶೀಲಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಅದಕ್ಕೆ ಅನುಗುಣವಾಗಿ ತನಿಖೆ ಮಾಡಲಾಗುತ್ತದೆ.

ನಂತರ ನೀವು ಕೆಲವು ರಕ್ತ ತನಿಖೆಗೆ ಒಳಗಾಗುತ್ತೀರಿ, ಅವುಗಳೆಂದರೆ ಸಂಪೂರ್ಣ ರಕ್ತದ ಎಣಿಕೆಗಳು, ಕಬ್ಬಿಣದ ವಿವರ, ಯಾದ್ಯಕ್ಕೆ ರಕ್ತದ ಗ್ಲೂಕೋಸ್, ಮೂತ್ರಪಿಂಡದ ಕಾರ್ಯ ಪರೀಕ್ಷೆಗಳು, ಪಿತ್ತಜನಕಾಂಗದ ಕಾರ್ಯ ಪರೀಕ್ಷೆಗಳು, ಮೂತ್ರದ ದಿನಚರಿ ಮತ್ತು ಸೂಕ್ಷ್ಮದರ್ಶಕ, ಡಿ ಡೈಮರ್, ಎಲ್‌ಡಿಹೆಚ್, ಸೀರಮ್ ಫೆರಿಟಿನ್, ಐಎಲ್ -6, ಎಚ್‌ಎಸ್‌ಸಿಆರ್‌ಪಿ.

ಅಪಾಯ ಮತ್ತು ಪ್ರಯೋಜನಗಳು: ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಯಾವುದೇ ಸಂಭಾವ್ಯ ಅಪಾಯಗಳಿಲ್ಲ.

ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವ ಪ್ರಯೋಜನಗಳು:

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವ ಮೂಲಕ, ಐರನ್ ಪ್ರೊಫೈಲ್, ವಿಶೇಷವಾಗಿ ಫೆರಿಟಿನ್ / ಟ್ರಾನ್ಸ್‌ಫೆರಿನ್ ಅನುಪಾತ ಮತ್ತು ಕೋವಿಡ್ - 19 ಸೋಂಕಿನಿಂದ ಬಳಲುತ್ತಿರುವ ರೋಗಿಯ ತೀವ್ರತೆಯ ನಡುವಿನ ಪರಸ್ಪರ ಸಂಬಂಧವನ್ನು ಕಂಡುಹಿಡಿಯಬಹುದು, ಇದು ಕೋವಿಡ್ - 19 ಸೋಂಕಿನ ತೀವ್ರತೆಯನ್ನು ಹಿಸಲು ಮುನ್ನರಿವಿನ ಸೂಚಕವಾಗಿ ಕಾರ್ಯನಿರ್ವಹಿಸುತ್ತದೆ.

ಸ್ವಯಂಪ್ರೇರಿತ ಭಾಗವಹಿಸುವಿಕೆ / ಅಧ್ಯಯನದಿಂದ

ಹಿಂತೆಗೆದುಕೊಳ್ಳುವಿಕೆ:

ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವುದು ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ. ಈ ಅಧ್ಯಯನಕ್ಕೆ ನಿಮ್ಮನ್ನು ಸೇರಿಸಿಕೊಳ್ಳದಿರಲು ನೀವು ಆಯ್ಕೆ ಮಾಡಬಹುದು ಮತ್ತು ಈ ನಡುವೆ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನವನ್ನು ಬಿಡಲು ಆಯ್ಕೆ ಮಾಡಬಹುದು.

ಪರ್ಯಾಯಗಳು:

ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವ ಬಗ್ಗೆ ನಿಮ್ಮ ನಿರ್ಧಾರವು ಕೆಎಲ್‌ಇಎಸ್ ಡಾ. ಪ್ರಭಾಕರ್ ಕೋರೆ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಬೆಳಗಾವಿ ವೈದ್ಯಕೀಯ ಸಂಶೋಧನಾ ಕೇಂದ್ರದಲ್ಲಿ ನಿಮಗೆ ನೀಡುತ್ತಿರುವ ಪ್ರಸ್ತುತ ಅಥವಾ ಭವಿಷ್ಯದ ಆರೋಗ್ಯ ಸೇವೆಗಳನ್ನು ಬದಲಾಯಿಸುವುದಿಲ್ಲ. ನೀವು ಬಯಸಿದರೆ ನಿಮ್ಮನ್ನು ಅಧ್ಯಯನದಿಂದ ಹೊರಗಿಡಲಾಗುವುದು, ಮತ್ತು ನಿಮ್ಮ ಎಲ್ಲಾ ವಿವರಗಳನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗುತ್ತದೆ ಮತ್ತು ನೀವು ವಾಡಿಕೆಯ ನಿರ್ವಹಣೆಯನ್ನು ಪಡೆಯುತ್ತೀರಿ.

ಗೌಪ್ಯತೆ ಮತ್ತು ಗೌಪ್ಯತೆ:

ಅಧ್ಯಯನದ ಭಾಗವಹಿಸುವಿಕೆಯ ಸಮಯದಲ್ಲಿ ನೀವು ಸಂಗ್ರಹಿಸಿದ ಅಥವಾ ಬಹಿರಂಗಪಡಿಸಿದ ಎಲ್ಲಾ ಡೇಟಾವನ್ನು ಸಂಪೂರ್ಣವಾಗಿ ಗೌಪ್ಯವಾಗಿಡಲಾಗುತ್ತದೆ. ಕೋರ್ಸ್ ಸಮಯದಲ್ಲಿ ಪ್ರಗತಿಗೆ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸುವುದು ಅಗತ್ಯವಿದ್ದರೆ, ನಿಮ್ಮ ಮಾಹಿತಿ ಮತ್ತು ಲಿಖಿತ ಒಪ್ಪಿಗೆಯ ನಂತರವೇ ಇದನ್ನು ಮಾಡಲಾಗುತ್ತದೆ.

ನೀವು ಸಂಶೋಧನಾ ವಿಷಯ ಎಂದು ತಿಳಿದುಕೊಳ್ಳುವ ಏಕೈಕ ಜನರು ಸಂಶೋಧನಾ ತಂಡದ ಸದಸ್ಯರು. ನಿಮ್ಮ ಲಿಖಿತ ಅನುಮತಿಯಿಲ್ಲದೆ ನಿಮ್ಮ ಬಗ್ಗೆ ಯಾವುದೇ ಮಾಹಿತಿಯನ್ನು ಇತರರಿಗೆ ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ:

- ನಿಮ್ಮ ಹಕ್ಕುಗಳು ಮತ್ತು ಕಲ್ಯಾಣವನ್ನು ರಕ್ಷಿಸಲು ತುರ್ತು ಪರಿಸ್ಥಿತಿಯಲ್ಲಿ.
- ಕಾನೂನಿನ ಪ್ರಕಾರ ಅಗತ್ಯವಿದ್ದರೆ.

ಫಲಿತಾಂಶಗಳನ್ನು ಪ್ರಕಟಿಸಲು ಅಧಿಕಾರ:

ಅಧ್ಯಯನದ ಫಲಿತಾಂಶಗಳನ್ನು ಲೇಖನವನ್ನು ಪ್ರಕಟಿಸಲು ಬಳಸಬಹುದು. ಸಂಶೋಧನೆಯ ಫಲಿತಾಂಶಗಳು ಪ್ರಕಟವಾದ ಅಥವಾ ಚರ್ಚಿಸಿದಾಗ, ಸಮ್ಮೇಳನದಲ್ಲಿ, ನಿಮ್ಮ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸುವ ಯಾವುದೇ ಮಾಹಿತಿಯನ್ನು ಪ್ರದರ್ಶಿಸಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ಪಡೆದ ಯಾವುದೇ ಮಾಹಿತಿಯು ಮತ್ತು ಅದನ್ನು ನಿಮ್ಮೊಂದಿಗೆ ಗುರುತಿಸಬಹುದು.

ಭಾಗವಹಿಸುವಿಕೆಗೆ ಆರ್ಥಿಕ ಪ್ರೋತ್ಸಾಹ:

ಈ ಅಧ್ಯಯನದ ಉದ್ದೇಶಕ್ಕಾಗಿ ಯಾವುದೇ ಹೆಚ್ಚುವರಿ ವೆಚ್ಚಗಳು ನಿಮ್ಮ ಮೇಲೆ ಆಗುವುದಿಲ್ಲ. ಇದನ್ನು ಸಂಪೂರ್ಣವಾಗಿ ಸಂಶೋಧನೆಯ ಆಲೋಚನೆಯೊಂದಿಗೆ ಮಾಡಲಾಗುತ್ತದೆ ಮತ್ತು ಅಧ್ಯಯನದ ಎಲ್ಲಾ ವೆಚ್ಚವನ್ನು ತನಿಖಾಧಿಕಾರಿ ಭರಿಸುತ್ತಾರೆ.

ಪರಿಹಾರ:

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಂಡ ಪರಿಣಾಮವಾಗಿ ನೀವು ಗಾಯಗೊಂಡರೆ ಬೆಳಗಾವಿನ ಕೆಎಲ್‌ಇಎಸ್ ಡಾ. ಪ್ರಭಾಕರ್ ಕೋರೆ ಆಸ್ಪತ್ರೆ ಮತ್ತು ವೈದ್ಯಕೀಯ ಸಂಶೋಧನಾ ಕೇಂದ್ರದಲ್ಲಿ ನಿಮಗೆ ಚಿಕಿತ್ಸೆ ನೀಡಲಾಗುವುದು ಅಥವಾ ವೈದ್ಯಕೀಯ ಆರೈಕೆಯನ್ನು ಎಲ್ಲಿ ಪಡೆಯಬೇಕು ಎಂಬ ಬಗ್ಗೆ ನಿಮಗೆ ಮಾಹಿತಿ ನೀಡಲಾಗುವುದು. ಆದಾಗ್ಯೂ, ಯಾವುದೇ ಮರುಪಾವತಿ, ಪರಿಹಾರ ಅಥವಾ ಉಚಿತ ವೈದ್ಯಕೀಯ ಸೌಲಭ್ಯವನ್ನು ನೀಡಲಾಗುವುದಿಲ್ಲ.

ಪ್ರಶ್ನೆಗಳು / ಸಂಪರ್ಕ ವಿವರಗಳು:

ನೀವು ಬಯಸಿದಂತೆ ಯಾವುದೇ ಸ್ಪಷ್ಟೀಕರಣ ಅಥವಾ ಸಹಾಯಕ್ಕಾಗಿ ಅಧ್ಯಯನದ ಅವಧಿಯಲ್ಲಿ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಈ ಕೆಳಗಿನ ಹೆಸರು ಮತ್ತು ವಿಳಾಸಗಳನ್ನು ಸಂಪರ್ಕಿಸಲು ನೀವು ಮುಕ್ತರಾಗಿರಬೇಕು.

ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಅಥವಾ ಭವಿಷ್ಯದಲ್ಲಿ ನೀವು ಈ ಕೆಳಗಿನ ವ್ಯಕ್ತಿಗಳನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು,

ಡಾ. _____

ನೈತಿಕ ಸಮಿತಿಯ ಮುಖ್ಯಸ್ಥ
ಮಾನವ ಸಂಶೋಧನೆ ಜಿಎನ್‌ಎಂಸಿ, ಬೆಳಗಾವಿ.

ಡಾ. _____

ಪ್ರೊಫೆಸರ್, ಮುಖ್ಯಸ್ಥ, ಜನರಲ್ ಮೆಡಿಸಿನ್ ಇಲಾಖೆ,
ಜವಾಹರಲಾಲ್ ನೆಹರು ವೈದ್ಯಕೀಯ ಕಾಲೇಜು,
ಬೆಳಗಾವಿ- 590010.

ಡಾ. _____

ಗೈಡ್, ಸಹಾಯಕ ಪ್ರಾಧ್ಯಾಪಕ,
ಜನರಲ್ ಮೆಡಿಸಿನ್ ಇಲಾಖೆ,
ಜವಾಹರಲಾಲ್ ನೆಹರು ವೈದ್ಯಕೀಯ ಕಾಲೇಜು,
ಬೆಳಗಾವಿ- 590010.

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ಇನ್ವೆಸ್ಟಿಗೇಟರ್, ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿ
ಜನರಲ್ ಮೆಡಿಸಿನ್ ಇಲಾಖೆ,
ಜವಾಹರಲಾಲ್ ನೆಹರು ವೈದ್ಯಕೀಯ ಕಾಲೇಜು,
ಬೆಳಗಾವಿ- 590010.

ಒಪ್ಪಿಗೆ ಪತ್ರ

ಕೆಳಗೆ ಸಹಿ ಮಾಡುವ ಮೂಲಕ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನಾನು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪುತ್ತೇನೆ. ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಹಿಂತೆಗೆದುಕೊಳ್ಳಬಹುದು. ಈ ಫಾರ್ಮ್ ಸಹಿ ಮಾಡುವ ಮೂಲಕ ನಾನು ನನ್ನ ಯಾವುದೇ ಕಾನೂನು ಹಕ್ಕುಗಳನ್ನು ಬಿಟ್ಟುಕೊಡುತ್ತಿಲ್ಲ. ಕೆಳಗಿನ ನನ್ನ ಸಹಿ

येथील डॉ. _____, सहयोगी प्राध्यापक, सामान्य चिकित्सा विभाग, जवाहरलाल नेहरू मेडिकल कॉलेज, बेळगाव यांच्या थेट देखरेखीखाली आणि मार्गदर्शनानुसार केला जाईल .

आपण अभ्यासाच्या 'विषय' / सहभागीच्या निकषांनुसार बसत असल्यामुळे यामध्ये सहभागी होण्याची विनंती केली गेली आहे .

अभ्यासात आपला सहभाग ऐच्छिक आहे. अभ्यासादरम्यान आपल्याला काही प्रश्न विचारले जातील आणि आपल्या सर्वोत्तम उत्तरासाठी आपल्याला उत्तर द्यावे लागेल. अभ्यासामध्ये भाग घ्यायचा की नाही या निर्णयाचा तुमच्या उपचारांवर कोणत्याही प्रकारचा परिणाम होणार नाही. आपण सहभागी होण्याचे ठरविल्यास आपण कधीही माघार घेण्यास मोकळे आहात .

बेळगावी .

अभ्यासाचे शीर्षक:

"कोविड – 19 संसर्गाच्या तीव्रतेसह फेरीटिन / ट्रान्सफेरिन रेशोच्या परस्परसंबंधाचा अभ्यास करण्यासाठी - केएलईएस डॉ. प्रभाकर कोरे रुग्णालय व वैद्यकीय संशोधन केंद्रा बेलागावीत केलेला एक वर्षाचा क्रॉस सेक्शनल स्टडी. "

अभ्यासाचा हेतू:

कोविड -19 संसर्गासह फेरिटिन / ट्रान्सफेरिन रेशोच्या परस्परसंबंधाचा अभ्यास करणे आणि रोगाच्या तीव्रतेवर त्याचे भाकित मूल्यांचे मूल्यांकन करणे.

प्रक्रिया समाविष्ट:

माझ्या अभ्यासामध्ये आपण स्वतःस नावनोंदणी घेण्यास सहमत असल्यास, आपल्यास आपल्या वर्तमान, भूतकाळाच्या आणि कौटुंबिक इतिहासाच्या संदर्भात मुलाखत दिली जाईल, त्यानंतर आपणास वैद्यकीयदृष्ट्या तपशीलवार तपासणी केली जाईल आणि त्यानुसार चौकशी केली जाईल .

त्यानंतर आपल्यास काही रक्त तपासणी, अर्थात संपूर्ण रक्ताची संख्या, लोह प्रोफाइल, यादृच्छिक रक्तातील ग्लूकोज, रेनल फंक्शन टेस्ट, यकृत कार्य चाचण्या, लघवीचे दिनचर्या आणि मायक्रोस्कोपी, डी डायमर, एलडीएच, सीरम फेरीटिन, आयएल -6, एचएससीआरपी असे विषय असतील.

जोखीम आणि फायदे :

या अभ्यासामध्ये कोणतेही संभाव्य धोके गुंतलेले नाहीत.

या संशोधनात भाग घेण्याचे फायदे:

या अभ्यासामध्ये भाग घेऊन, लोह प्रोफाइल, विशेषतः फेरीटिन / ट्रान्सफेरिन प्रमाण आणि कोविड -19 संक्रमणामुळे ग्रस्त झालेल्या रुग्णाची तीव्रता यांच्यात परस्परसंबंध आढळला की कोविड -19 संसर्गाच्या तीव्रतेचा अंदाज लावण्यासाठी रोगनिदान सूचक म्हणून काम करू शकते.

ऐच्छिक सहभाग / अभ्यासामधून पैसे काढणे :

अभ्यासामध्ये भाग घेणे ऐच्छिक आहे. आपण या अभ्यासामध्ये स्वतःची नावनोंदणी न करणे निवडू शकता आणि दरम्यान अभ्यास कधीही सोडणे निवडू शकता.

विकल्प:

अभ्यासात सहभागासंदर्भातील तुमचा निर्णय केएलईएस डॉ. प्रभाकर कोरे हॉस्पिटल आणि वैद्यकीय संशोधन केंद्र, बेळगाव येथे तुम्हाला देऊ केलेल्या सध्याच्या किंवा भविष्यातील आरोग्य सेवा बदलणार नाही. आपली इच्छा असेल तर आपल्याला अभ्यासापासून वगळले जाईल आणि आपले सर्व तपशील गोपनीय ठेवले जातील आणि आपल्याला व्यवस्थापनाची नियमित रूंदी मिळेल.

गोपनीयता आणि गोपनीयता :

अभ्यासाच्या सहभागादरम्यान आपण गोळा केलेला किंवा जाहीर केलेला सर्व डेटा पूर्णपणे गोपनीय ठेवला जाईल. अर्थात कोर्सच्या दरम्यान ओळख जाहीर करणे आवश्यक झाले तर ते तुमच्या माहिती व लेखी संमतीनंतरच केले जाईल.

आपण संशोधन विषय आहात हे फक्त लोकांनाच माहित आहे की ते संशोधन पथकाचे सदस्य आहेत. आपल्या लेखी परवानगीशिवाय इतर आपल्याबद्दल कोणतीही माहिती उघड केली जाणार नाही:

- आपत्कालीन परिस्थितीत आपले हक्क आणि कल्याण यांचे संरक्षण करण्यासाठी.
- कायद्याने आवश्यक असल्यास.

निकाल प्रकाशित करण्यासाठी अधिकृतता:

अभ्यासाचा निकाल लेख प्रकाशित करण्यासाठी वापरला जाऊ शकतो. जेव्हा एखाद्या संशोधनाचे निकाल कॉन्फरन्समध्ये प्रकाशित केले जातात किंवा त्यावर चर्चा केली जाते तेव्हा आपली ओळख उघडकीस आणणारी कोणतीही माहिती दर्शविली जाणार नाही. या अभ्यासाच्या संदर्भात प्राप्त केलेली कोणतीही माहिती आणि ती आपल्याशी ओळखली जाऊ शकते ती गोपनीय राहिल.

सहभागासाठी आर्थिक प्रोत्साहन :

या अभ्यासाच्या हेतूने आपल्यावर कोणत्याही प्रकारची अतिरिक्त किंमत आकारली जाणार नाही.

हे निव्वळ संशोधनाच्या कल्पनेने केले जात आहे आणि अभ्यासाचा सर्व खर्च तपासनीस करेल.

भरपाई :

या अभ्यासामध्ये भाग घेतल्यामुळे आपण जखमी झाल्यास, केएलईएस डॉ. प्रभाकर कोरे हॉस्पिटल आणि मेडिकल रिसर्च सेंटर, बेळगाव येथे तुम्हाला उपचार देण्यात येतील किंवा तुम्हाला वैद्यकीय सेवा कोटून घ्यावी याविषयी माहिती दिली जाईल. तथापि, कोणतेही प्रतिपूर्ती, भरपाई किंवा विनामूल्य वैद्यकीय सेवा दिली जाणार नाही.

प्रश्न / संपर्क तपशील:

अभ्यासाच्या कालावधीत कोणत्याही स्पष्टीकरणासाठी किंवा तुम्हाला पाहिजे असलेल्या मदतीसाठी तुम्ही खाली नमूद केलेल्या नावाने व पत्त्यांशी कधीही संपर्क साधू शकता .

अभ्यासाच्या वेळी किंवा भविष्यातील प्रश्नांच्या बाबतीत आपण खालील व्यक्तींशी संपर्क साधू शकता ,

डॉ. _____

अध्यक्ष, नैतिक समिती मानव संशोधन
जे. एन. एम. सी, बेळगावी .

डॉ. _____

प्राध्यापक, प्रमुख , सामान्य औषध विभाग ,
जवाहरलाल नेहरू मेडिकल कॉलेज , बेळगाव

डॉ. _____

मार्गदर्शन, सहयोगी प्राध्यापक,
सामान्य औषध विभाग,
जवाहरलाल नेहरू मेडिकल कॉलेज , बेळगाव

REG NO: BG0120015

अन्वेषक, पदव्युत्तर विद्यार्थी
सामान्य औषध विभाग,
जवाहरलाल नेहरू मेडिकल कॉलेज , बेळगाव.

संमती फॉर्म

मी खाली स्वाक्षरी करून या अभ्यासात भाग घेण्यास स्वेच्छेने सहमत आहे. मी कधीही माघार घेऊ शकतो. या फॉर्मवर सही करून मी माझा कोणताही कायदेशीर हक्क सोडत नाही. खाली माझी स्वाक्षरी सूचित करते की मी हा संमती फॉर्म वाचला आहे किंवा हा संमती फॉर्म मला वाचला आहे आणि मला सर्व प्रश्नांची उत्तरे दिली आहेत

सहभागी किंवा कायदेशीररित्या अधिकृत प्रतिनिधीची सही / डावा अंगठा प्रिंट

सहभागीचे नाव:

स्वाक्षरी / डावा अंगठा ठसा:

सहभागीचा

कायदेशीररीत्या अधिकृत नाव:

प्रतिनिधी / पालक

स्वाक्षरी / डावा अंगठा ठसा:

साक्षीचे नाव:

स्वाक्षरी / डावा अंगठा ठसा:

अन्वेषकांचे नाव आणि स्वाक्षरी:

तारीख:

ठिकाण:

सूचित सहमति

प्रिय श्री / श्रीमती / डॉ. _____, आपसे विनम्र अनुरोध है कि आप स्वयं को एक शोध अध्ययन में नामांकित करें, " कोविड -19 संक्रमण की गंभीरता के साथ फेरिटिन / ट्रांसफेरिन अनुपात के सहसंबंध का अध्ययन करने के लिए - केएलईएस डॉ. प्रभाकर कोरे अस्पताल और चिकित्सा अनुसंधान केंद्र बेलगावी में किया गया एक साल का क्रॉस सेक्शनल अध्ययन." एमडी जनरल मेडिसिन में स्नातकोत्तर छात्र REG NO: BG0120015 द्वारा संचालित किया जा रहा है और यह अध्ययन डॉ. _____,

सहयोगी प्राध्यापक, जनरल मेडिसिन विभाग, जवाहरलाल नेहरू मेडिकल कॉलेज, बेलगाम के प्रत्यक्ष पर्यवेक्षण और मार्गदर्शन में किया जाएगा।

आपसे यह अनुरोध किया गया है कि आप इसमें एक अध्ययन 'विषय' / प्रतिभागी के निर्धारित मानदंडों में फिट हों।

अध्ययन में आपकी भागीदारी स्वैच्छिक है। अध्ययन के दौरान आपसे कुछ प्रश्न पूछे जाएंगे और आप अपने ज्ञान का सबसे अच्छा जवाब देने वाले हैं। अध्ययन में भाग लेने या न लेने का आपका निर्णय किसी भी रूप में आपके उपचार को प्रभावित नहीं करेगा। यदि आप भाग लेने का निर्णय लेते हैं तो आप किसी भी समय वापस लेने के लिए स्वतंत्र हैं।

अध्ययन का शीर्षक :

" कोविड -19 संक्रमण की गंभीरता के साथ फेरिटिन / ट्रांसफेरिन अनुपात के सहसंबंध का अध्ययन करने के लिए - केएलईएस डॉ. प्रभाकर कोरे अस्पताल और चिकित्सा अनुसंधान केंद्र बेलगावी में किया गया एक साल का क्रॉस सेक्शनल अध्ययन."

अध्ययन का उद्देश्य:

कोविड -19 संक्रमण के साथ फेरिटिन / ट्रांसफेरिन अनुपात के सहसंबंध का अध्ययन करना और बीमारी की गंभीरता पर इसके अनुमानित मूल्य का आकलन करना।

शामिल प्रक्रियाएं :

यदि आप मेरे अध्ययन में खुद को नामांकित करने के लिए सहमत हैं, तो आपको अपने वर्तमान, अतीत और परिवार के इतिहास के बारे में साक्षात्कार दिया जाएगा, फिर आपकी चिकित्सकीय जांच की जाएगी और तदनुसार जांच की जाएगी।

फिर आपको कुछ रक्त जांचों, अर्थात् पूर्ण रक्त गणना, आयरन प्रोफाइल, यादृच्छिक रक्त ग्लूकोज, गुर्दे समारोह परीक्षण, यकृत समारोह परीक्षण, मूत्र दिनचर्या और माइक्रोस्कोपी, डी डिमर, एलडीएच, सीरम फेरिटिन, आईएल -6, एचएससीआरपी के अधीन किया जाएगा।

जोखिम और लाभ:

इस अध्ययन में कोई संभावित जोखिम शामिल नहीं हैं।

इस शोध में भाग लेने के लाभ:

इस अध्ययन में भाग लेने से, आयरन प्रोफाइल, विशेष रूप से फेरिटिन / ट्रांसफेरिन अनुपात और कोविड -19 संक्रमण से पीड़ित रोगी की गंभीरता के बीच सहसंबंध पाया जा सकता है जो कोविड -19 संक्रमण की गंभीरता का अनुमान लगाने के लिए एक रोगसूचक संकेतक के रूप में काम कर सकता है।

अध्ययन से स्वैच्छिक भागीदारी / निकासी :

अध्ययन में भाग लेना स्वैच्छिक है। आप इस अध्ययन में खुद को नामांकित नहीं करना चुन सकते हैं और बीच में कभी भी अध्ययन छोड़ने का विकल्प चुन सकते हैं।

विकल्प:

अध्ययन में भाग लेने के बारे में आपका निर्णय के एल ई एस डॉ। प्रभाकर कोरे अस्पताल और चिकित्सा अनुसंधान केंद्र, बेलगाम में आपके लिए पेश की गई वर्तमान या भविष्य की स्वास्थ्य देखभाल सेवाओं को नहीं बदलेगा। यदि आप चाहें, तो आपको अध्ययन से बाहर रखा जाएगा और आपके सभी विवरणों को गोपनीय रखा जाएगा और आपको प्रबंधन की नियमित लाइन मिल जाएगी।

गोपनीयता और गोपनीयता :

अध्ययन की भागीदारी के दौरान आपके द्वारा एकत्र या प्रकट किए गए सभी डेटा को पूरी तरह से गोपनीय रखा जाएगा। हालांकि यदि पाठ्यक्रम के दौरान यह आवश्यक है कि पाठ्यक्रम की प्रगति के लिए पहचान का खुलासा करना आवश्यक है, तो यह आपकी सूचना और लिखित सहमति के बाद ही किया जाएगा। केवल यह जानने के लिए कि आप एक शोध विषय हैं, अनुसंधान टीम के सदस्य हैं। आपके लिखित अनुमति के बिना आपके बारे में कोई भी जानकारी का खुलासा नहीं किया जाएगा :

- अपने अधिकारों और कल्याण की रक्षा के लिए आपातकाल में।
- यदि कानून द्वारा आवश्यक हो।

परिणाम प्रकाशित करने के लिए प्राधिकरण :

अध्ययन के परिणामों का उपयोग एक लेख प्रकाशित करने के लिए किया जा सकता है। जब एक सम्मेलन में प्रकाशित या चर्चा की गई शोध के परिणाम, कोई भी जानकारी प्रदर्शित नहीं की जाएगी जो आपकी पहचान का खुलासा करेगी। इस अध्ययन के संबंध में प्राप्त की गई कोई भी जानकारी और जिसे आप के साथ पहचाना जा सकता है, गोपनीय रहेगी।

भागीदारी के लिए वित्तीय प्रोत्साहन :

इस अध्ययन के उद्देश्य से आपके ऊपर कोई अतिरिक्त लागत नहीं लगेगी। यह विशुद्ध रूप से अनुसंधान के विचार के साथ किया जा रहा है और अध्ययन का सारा खर्च अन्वेषक द्वारा वहन किया जाएगा।

भरपाई :

इस अध्ययन में भाग लेने के परिणामस्वरूप आप घायल हो जाते हैं, तो केएलईएस डॉ। प्रभाकर कोरे अस्पताल और चिकित्सा अनुसंधान केंद्र, बेलगाम में उपचार की पेशकश की जाएगी, या आपको चिकित्सा देखभाल कहाँ प्राप्त होगी, इसके बारे में जानकारी दी जाएगी। हालांकि, कोई प्रतिपूर्ति, मुआवजा या मुफ्त चिकित्सा देखभाल नहीं दी जाएगी।

प्रश्न / संपर्क विवरण :

आप किसी भी स्पष्टीकरण या मदद के लिए अध्ययन अवधि के दौरान किसी भी समय नीचे दिए गए नाम और पते से संपर्क करने के लिए स्वतंत्र हो सकते हैं।

अध्ययन के दौरान या भविष्य में प्रश्नों के मामले में आप निम्नलिखित व्यक्तियों से संपर्क कर सकते हैं,

डॉ. _____

अध्यक्ष, नैतिक समिती मानव संशोधन
जे. एन. एम. सी, बेळगावी.

डॉ. _____

प्रोफेसर प्रमुख, सामान्य चिकित्सा विभाग,
जवाहरलाल नेहरू मेडिकल कॉलेज,
नेहरू नगर, केएलई अस्पताल, बेलगाम

डॉ. _____

मार्गदर्शक, सहयोगी प्राध्यापक,
नेहरू नगर, केएलई अस्पताल, बेलगाम

REG NO: BG0120015

अन्वेषक, स्नातकोत्तर छात्र
सामान्य चिकित्सा विभाग,
जवाहरलाल नेहरू मेडिकल कॉलेज, बेलगाम

सहमति पत्र

मैं स्वेच्छा से नीचे हस्ताक्षर करके इस अध्ययन में भाग लेने के लिए सहमत हूँ। मैं किसी भी समय वापस ले सकता हूँ। मैं इस फॉर्म पर हस्ताक्षर करके अपने किसी भी कानूनी अधिकार को नहीं छोड़ रहा हूँ। नीचे दिए गए मेरे हस्ताक्षर से संकेत मिलता है कि मैंने इस सहमति फॉर्म को पढ़ा है, या यह मेरे लिए पढ़ा गया है, यह सहमति फॉर्म और उत्तर दिए गए प्रश्नों के उत्तर हैं

तिभागी या कानूनी रूप से अधिकृत प्रतिनिधि का हस्ताक्षर / बायाँ अंगूठा प्रिंट

प्रतिभागी का नाम:।

हस्ताक्षर / बाएं अंगूठे का निशान:।

प्रतिभागी का

कानूनी रूप से अधिकृत का नाम:।

प्रतिनिधि / अभिभावक

हस्ताक्षर / बाएं अंगूठे का निशान:।

साक्षी का नाम:।

हस्ताक्षर / बाएं अंगूठे का निशान:।

अन्वेषक का नाम और हस्ताक्षर:।

दिनांक:

जगह:

ANNEXURE - II - PROFORMA

DEMOGRAPHY

IP No :

Date of Admission :

Age :

Sex :

Clinical Parameters At Admission**Symptoms:**

Tick the applicable Box					
Cough		Fever		Other	
Myalgia		Breathlessness		Symptoms	
Time to hospital from symptom onset (Days)					

Oxygenation Parameters at admission

Admission Respiratory rate (RR/min)	
Admission SPO2(%)	
Admission O2 modality (RBM,HFO,NIV, Ventilator)	
Admission O2 rate (in Litres per minute)	
Admission O2 FiO2 (%)	
Admission PO2 (Look at admission ABG)	

Co-morbidities

DM		HTN		IHD		CKD		CLD		CVA		Malignancy	
Others													

Lab Parameters At Admission

Hb		TLC		Lymphocyte%		Platelets	
Ferritin		LDH		hsCRP			
d-Dimer		CRP		Fibrinogen		Urea	
Creatinine		Bilirubin		SGOT		SGPT	
RBS		HbA1c					

CT Severity score (Out of 25)	
--------------------------------------	--

Maximum OXYGENATION Support required during entire stay	
Mode of O2 (RBM,HFO,NIV, Ventilator)	
FiO2%	
SpO2 %	

PO2% (Look at ABG)	

Ferritin, Transferrin and Ferritin/Transferrin Ratio on Admission	
Serum Ferritin	
Serum Transferrin	
Ferritin/Transferrin Ratio	

TREATMENT

Remdesivir (No. of Doses)	
Tocilizumab (No. of Doses)	
LMWH (Max Dose)	
Un-fractionated Heparin (Max Dose)	
Type of Steroid used (methyle pred or dexa)	
Max dose of steroid per day used	
Thymosin 1 Alpha (No of doses)	
Convalescent Plasma (Cycles)	
PO2/FiO2 at the time of convalescent plasma	
Mode of O2 at the time of plasma therapy	
Cytosob (Cycles)	
Plasma Exchange (Cycles)	
Alteplase (Dose)	
Alteplase (tPA)(Number of times Given)	
Pre tPA PO2/FiO2	
Post tPA pO2/FiO2	
Pre tPA Fibrinogen	
Post tPA Fibrinogen	
Pre tPA d-Dimer	
Post tPA D-dimer	

OUTCOME

Outcome	Tick	Date
Improved and discharged		
Died		
Worsened and went AMA		
Improved and went AMA		

										TREATMENT						OUTCOME							
3	FIG 1	2	3	SpO2 1	2	3	PO2 (ABG) 1	2	3	Remdesivir (No. of Doses)	Tocilizumab (No. of Doses)	LMWH (Max Dose)	Unfractionated Heparin (Max Dose)	Type of Steroid used (Methyl ePred or dexam)	Max dose per day used	Thymosine 1 Alpha (No of doses)	Plasma (No. of times used)	Alteplase (Doses)	Improved and Discharged	Died (note- this column should be left empty as this page is only for survivors)	Worsened and AMA	Improved and AMA	Outcome Date
	95			97%						none		80mg		methyl pred	50mg				17-10-2020				17-10-2020
										5		80mg		methyl pred	50mg				18/09/20				18/09/20
NIV	90	80	80	97	89	95	96.6	64.3	75.6	5		80mg		methyl pred	80mg		1	50mg	28/8/20				28/8/20
										7		120mg		methyl pred	500 mg				3-10-20				3-10-20
										8		120mg		methyl pred	120				7-10-20				7-10-20
	NA			99%						6		80MG		methyl pred	500MG				30/09/20				30/09/20
RA	8LO2	2LO2		93%	95%	93%				5		80MG		methyl pred	80mg				28/08/20				28/08/20
RBM	15L	15L		93%	92%		20.4	68.4		9		80 MG		methyl pred	80MG				15/10/20				15/10/20
	6L			95%						2		80MG		methyl pred	80MG				07-10-20				07-10-20
RA				96%	95%	96%				3		40MG		methyl pred	80MG				12-08-20				12-08-20
				94%	96%					2		80 MG		methyl pred	80 MG				09-09-20				09-09-20
RA				85%	91%	94%				6	1	100MG		methyl pred	1000 MG				25/09/20				25/09/20
				94%	96%	97%				5		80MG		methyl pred	80MG				29/09/20				29/09/20
				94%	96%					7		80 MG		DEXA	12 MG				24/08/20				24/08/20
				92%	94%					6		80 MG		methyl pred	80 MG				10-09-20				10-09-20
RA				90%	90%	99				6		80 MG		methyl pred	80 MG				08-27-22				08-27-22
				92%	96%					1		80MG		methyl pred	120MG				15/09/20				15/09/20
				99%						NONE		80 MG		methyl pred	120 MG				12-09-20				12-09-20
				96%						3		40		methyl pred	80				18/07/20				18/07/20
				96%						5		80mg		methyl pred	500mg				25/09/20				25/09/20
				94%	94%	95%				3		40		methyl pred	80MG				13/07/20				13/07/20
3L O2				96%	92%	95%				5		40mg		methyl pred	80mg				22/08/20				22/08/20
				99%						5		80mg		methyl pred	80mg				28/07/20				28/07/20
6L				93%	95%	97%				4		40mg		methyl pred	80mg				24/09/20				24/09/20
				96%	97%					6		40mg		dexa	12mg				23-08-2020				23-08-2020
10L				97%	100%	98%				6		40mg		methyl pred	80mg				15/10/20				15/10/20
RA				96%	99%	96%				6		40mg		methyl pred	80mg				03-08-2020				03-08-2020
1L				94%	92%	95%				5		40mg		methyl pred	80mg				31/08/20				31/08/20
				99%						3		40mg		methyl pred	80mg				25-07-2020				25-07-2020
RA				96%	98%	99%				5		40mg		methyl pred	mg				20-08-2020				20-08-2020
				96%	98%					NONE		40mg		dexa	12mg				25/08/20				25/08/20
				99%	98%					NONE		40MG		dexa	12mg				15/10/20				15/10/20
RA				92%	94%	100%				5		40MG		methyl pred	80MG				12-08-20				12-08-20
RBM	3L	6L	8L	95%	96%	100%				6		40mg		methyl pred	80mg				15-10-2020				15-10-2020
RA				96%	92%	99%				4		40mg		methyl pred	80mg				20-08-2020				20-08-2020
										NONE		40MG		DEXA	12MG				22/08/20				22/08/20
RA				91%	93%	99%				5		80mg		methyl pred	80mg				03-10-2020				03-10-2020
				96%	97%					5		80mg		methyl pred	80mg				15-08-2020				15-08-2020
RA				96%	92%	98%				NONE		40MG		methyl pred	40 MG				30/07/20				30/07/20
				96%	98%					NONE		NA		DEXA	12MG				10-09-2020				10-09-2020
				96%	96%	97%				NONE		NA		DEXA	12MG				06-10-2020				06-10-2020
				92%	99%					3		40mg		DEXA	12MG				09-07-2020				09-07-2020
RA	5L	2L		94%	98%					NONE		80Mg		Methyl prednisone	40Mg tid				1-10-20				1-10-20
				85%	88%	96%	99%			6		40Mg		Methyl prednisone	1000Mg				6-9-20				6-9-20
				95%	95%					6		80Mg		Methyl prednisone	80Mg				04-09-2020				04-09-2020
				98%	99%					4		80Mg		Methyl prednisone					21-09-2020				21-09-2020
RBM	4L	6L	2L	94%	98%	99%				6		80Mg		Methylpred	120mg				19/8/20				19/8/20
	4L			100%	95%					6		80 Mg		Dexamethasone	16mg				1-8-20				1-8-20
				96%	99%					NONE		80 Mg		Methyl prednisolone	80 mg				19/9/20				19/9/20
RBM	10L	15L	2L	95%	97%	99%				6		40 Mg		Methyl prednisolone					2-10-20				2-10-20
				98%	98%					NONE		NONE		DEXA	12MG				30-08-2020				30-08-2020
HFO	60L	60L	60L	94%	90%	89%			50	6		80 Mg		Methyl prednisolone	1000 Mg				30/9/20		Worsened		30/9/20
	10L	15L		90%	85%		47			3		80 Mg		Methyl prednisolone	120 Mg				17/10/20		Worsened		17/10/20
RBM		5L	5L	92%	94%	98%				7		80 Mg		Dexamethasone	12 Mg				24/8/20				24/8/20
RA				94%	96%	98%				4		80 Mg		Methyl prednisolone	80 Mg				5-10-20				5-10-20
RBM	10L	8L	4L	98%	98%	96%				5		40 Mg		Methyl prednisolone	80 Mg				29/9/20				29/9/20
				98%						5		40 Mg		Methyl prednisolone	80 Mg				5-10-20				5-10-20
RBM	2L	4L	6L	94%	94%	94%				6		40 Mg		Methyl prednisolone	80 Mg				29/9/20				29/9/20
O2 MASK	6L	2L	2L	99%	98%	97%				6		40 Mg		Dexamethasone	8 Mg				20/8/20				20/8/20
	10L	8L		99%	92%					10		80 Mg		Methyl prednisolone	1000 Mg		50 Mg		3-9-20		Worsened		3-9-20
RA	4L	2L		96%	96%	98%				5		80 Mg		Methyl prednisolone	500 Mg				30/9/20				30/9/20
RA	5L	2L	NA	92%	94%	98%				7		40 Mg		Methyl prednisolone	40 Mg				29/9/20				29/9/20
				98%	97%					3		80 Mg		Methyl prednisolone	500 Mg				28/9/20				28/9/20
				97%						5		80mg		Methyl prednisone	80mg				3-10-2020				3-10-2020
	4L			99%						3		80mg		Methylpred	80mg				6-9-20				6-9-20
	4L/MIN	2L/MIN		98%	98%					5		80mg		Methyl prednisone	80mg				21/9/20				21/9/20
				96%	98%					2		80mg		Methyl prednisone	80mg				23/9/20				23/9/20
				98%						3		NONE		DEXA	12MG				02-08-2020				02-08-2020
RA		3L/MIN		98%	90%	98%				6		40MG		Methylpred 80mg					25/7/2020				25/7/2020
	48 L			90%			68%			6		80mg		methyl pred	80mg				19/10/2020				19/10/2020
				96%	98%					5		80mg		methyl pred</									

										70										TREATMENT										OUTCOME				
RBS	RBS	RBS	HbA1c	Date #2	Date #3	Date #4	Mode of O2 (RBM,HFO,NIV, Ventilator) 1	2	3	FIO2 1	2	3	SpO2 1	2	3	Remdesivir (No. of Doses)	Toelizumab (No. of Doses)	LMWH (Max Dose)	Unfractionated Heparin (Max Dose)	Type of Steroid used (MethylPred or dexa)	Max dose per day used	Alteplase (Doses)	Alteplase (Number of times Given)	Improved and discharged	Died	Worsend and AMA	Improved and AMA	Outcome Date						
				25-07-2020			NIV			100			92			2				Methyl Pred	1G							25/8/20						
				25-07-2020	01-08-2020	10-08-2020	NIV	NIV		60	80		97%	97%	90%	9	1	40mg		MethylPred	40mg							10-08-2020	10-08-2020					
				27-07-2020	30-07-2020	03-08-2020	RRM	HFO	NIV	12L/MIN	60L/MIN	100%	92%	93%	91%	5	2	40MG		Methyl pred	40mg							04-08-2020	04-08-2020					
				08-05-2020	09-05-2020		RRM	NIV		5L/MIN	100%		96%			3				Methyl pred	80mg							09-05-2020	09-05-2020					
				28-07-2020	30-07-2020	08-08-2020	HFO	NIV	NIV	40L/MIN	80L/MIN	100%	92%	99%	90%	7	1	40MG		Methyl pred	40mg							09-08-2020	09-08-2020					
				21-08-2020	23-08-2020		HFO	NIV		60L/MIN	100%					2				Methyl pred	80mg							23/08/2020	23/08/2020					
				05-09-2020	09-09-2020		NIV	INTUBATION		100%	100%		98	99%		8				MethylP	1G	30MG	1					11-09-2020	11-09-2020					
				04-09-2020	05-09-2020	16-09-2020	HFO	NIV	HFO	60L	100%	60L/min	94%	97%	93%	8	1	40MG		Methyl Pred	1g		2					18/9/2020	18/9/2020					
				20-08-2020	22-08-2020	23-08-2020	HFO	HFO	INTUBATI ON	60L/MIN	70L/MIN	100%	97	95%	91%	7	1	40MG		Methylpred	500mg							24-08-2020	24-08-2020					
				23-08-2020			NRBM			10L			88%			NONE				MethylP	80MG							24/08/2020	24/08/2020					
			10.1	27-07-2020	30-07-2020		HFO	NIV		60L/MIN	100%		96	90%		2	1	80MG		Methyl Pred	80mg							31/7/20	31/7/20					
				13/8	17/8	20/8	HFO	NIV	NIV	50L	60	80	75%	98%	96%	9	2	80mg		MethylP	500MG							22/08/2020	22/08/2020					
				28/8/20	29/8/20		NIV	NIV		100	100		95	92		2				MethylP	1G							29/8/20	29/8/20					
				03-08-2020	05-08-2020		NIV	NIV		100	100		98	92%		6				DEXA	12MG							06-08-2020	06-08-2020					
			6.5	04-09-2020			INTUBATED			100			92			1				METHYPRED	500MG							4-9-2020	4-9-2020					
			8.2	10-08-2020			NIV			100			94			10	2	40mg		Methylpred	1gm							16/8/2020	16/8/2020					
				14-08-2020			INTUBATED			100			98			4				METHYPRED	120MG							15/8/2020	15/8/2020					
				11-09-2020	13-09-2020	14-09-2020	HFO	NIV	INTUBATE D	80L/MIN	100%	100%	92	96%	98%	3		40MG		METHYPRED	1000GM							15/9/2020	15/9/2020					
			10.7	11-08-2020			HFO		HFO	60L/MIN			86			2				DEXA	12MG							13/8/2020	13/8/2020					
				30-07-2020	05-08-2020		NIV	INTUBATED		100			68			9	3	40mg		Methylpred	1g							8-8-2020	8-8-2020					
				07-09-2020			NIV			100			89			5	1	40 MG		METHYPRED	1000MG							8-9-2020	8-9-2020					
			9.8	27-08-2020	30-08-2020		NIV	INTUBATED		100	100		86	91%		5		40mg		Methylpred	500mg							1-9-2020	1-9-2020					
				24-09-2020	25-09-2020	25-09-2020	RRM	HFO	NIV	15L/MIN	60L	100	89	92	89	1				Methylpred	500mg							25/9/2020	25/9/2020					
			7.6	3-9-20			NIV			100			78			2				Methyl Pred	40MG							04-09-2020	04-09-2020					
				07-09-2020			NIV			100			70			NONE				Methylpred	80							08-09-2020	08-09-2020					
				24/9	25/9	29/9	HFO	RRM	NIV	30L/MIN	15L/MIN	100	93	94	95	6				Methyl Pred	40mg	50MG	1					30/9/20	30/9/20					
				10-09-20	11-09-20		NIV	NIV		100	100		92	95		2				Methylpred	1000 mg							11-09-2020	11-09-2020					
				5-9-20	6-9-20	9-9-20	RRM	RRM	HFO	10L/MIN	15L/MIN	100	94	97	60%	5		40MG		Methylpred	500							10-9-20	10-9-20					
				8-9-20			NIV			100			95			2				Methylpred	40							14/9/20	14/9/20					
				25/7/20			NIV			100			90			2	6	40		Methylpred	40							1-8-20	1-8-20					
				15/8/20	19-08-2020		HFO	NIV		60	100		93			4	2	80		Methylpred	500							21/08/2020	21/08/2020					
				10-09-2020	12-09-2020	13-09-2020	NIV	NIV	NIV	100	100	100	97	90	86	5		40		Methylpred	1g							14/9/20	14/9/20					
				27-07-2020	29-07-2020		RRM	NIV	NIV	10L/MIN	100	100	88	92	96	5				Methylpred	80MG							30-07-2020	30-07-2020					
				19/9	24/9	26/9	NIV	NIV	NIV	100	100	100	92	84	80	11	1	80		Methylpred	500							26/9/2020	26/9/2020					
				17-08-2020			NIV	INTUBATED		100	100		76	84		5				Methylpred	1g	50mg	1					23/09/2020	23/09/2020					
				21/8	25/8	26/8	RRM	NIV	NIV	90	90	84	92	92	2					Methylpred	1g							27/08/2020	27/08/2020					
			6.9	29/8	31/8	02-09-2020	NIV	NIV	NIV	100	80	100	93	96	84	5				Methylpred	1g							02-09-2020	02-09-2020					
				16/8	18/8	19/8	RRM	RRM	HFO	10L/MIN	15L/MIN	60L/MIN	94	89	70	2				Methylpred	500							20/08/2020	20/08/2020					
				31-08-2020	01-09-2020		NIV	NIV		100	100		94	94		5				Methylpred	1g							01-09-2020	01-09-2020					
				24-08-2020	29-08-2020		NIV	INTUBATED		100	100		94	96		NONE				Methylpred	1g							25/08/2020	25/08/2020					
				17/8	18/8		HFO	NIV		60	90		80	90%		1	1	40		Methylpred	1g							21/08/2020	21/08/2020					
				31/8			NIV			100			94			1				Methylpred	1g							31/8/20	31/8/20					
				27/8	28/8		NIV	NIV		100	100		92	60		7				Methylpred	1g							29/08/2020	29/08/2020					
				16/08	17-08-2020		HFO	INTUBATED		60	100		90			3				Methylpred	120							17/08/2020	17/08/2020					
				12-8	13/8	14-08-2020	RRM	RRM	NIV	15L/MIN	15L/MIN	100	100	92	4					DEXA	12							15/8/20	15/8/20					
				5-8	7-8	14/8	NIV	NIV	NIV	60	100	50	94	96	92	9	1	40		Methylpred	120							14/08/2020	14/08/2020					
				26-08-2020	27-08-2020		HFO	NIV	NIV	60	100	100	91	92	88	2				Methylpred	1GM							25/8/20	25/8/20					
				30-08-2020	01-09-2020		NIV	INTUBATED		100	100		90	100	100	NONE				Methylpred	80							01-09-2020	01-09-2020					
				18-08-2020	20-08-2020		NIV	NIV		100	100		92	90		3				Methylpred	40							20/8/20	20/8/20					
				13-08-2020			INTUBATED			100			88			1				Dexa	24							13/08/2020	13/08/2020					
				25-08-2020	26-08-2020	27-08-2020	HFO	NIV	INTUBATE D	60	100	100	90	99	92	5				Methylpred	120							28/08/2020	28/08/2020					
				29-08-2020	01-09-2020		NIV	INTUBATED		80	90		93	92		4				Methylpred	80	40	1					02-09-2020	02-09-2020					
				20-08-2020	21-08-2020	22-08-2020	RRM	NIV	INTUBATE D	10L/MIN	100	100	86	94																				