"CORRELATION OF ABO BLOOD GROUPING AND OUTCOME IN SEPSIS PATIENTS ADMITTED IN INTENSIVE CARE UNIT AT KLE DR. PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELAGAVI -A CROSS SECTIONAL BASED STUDY"

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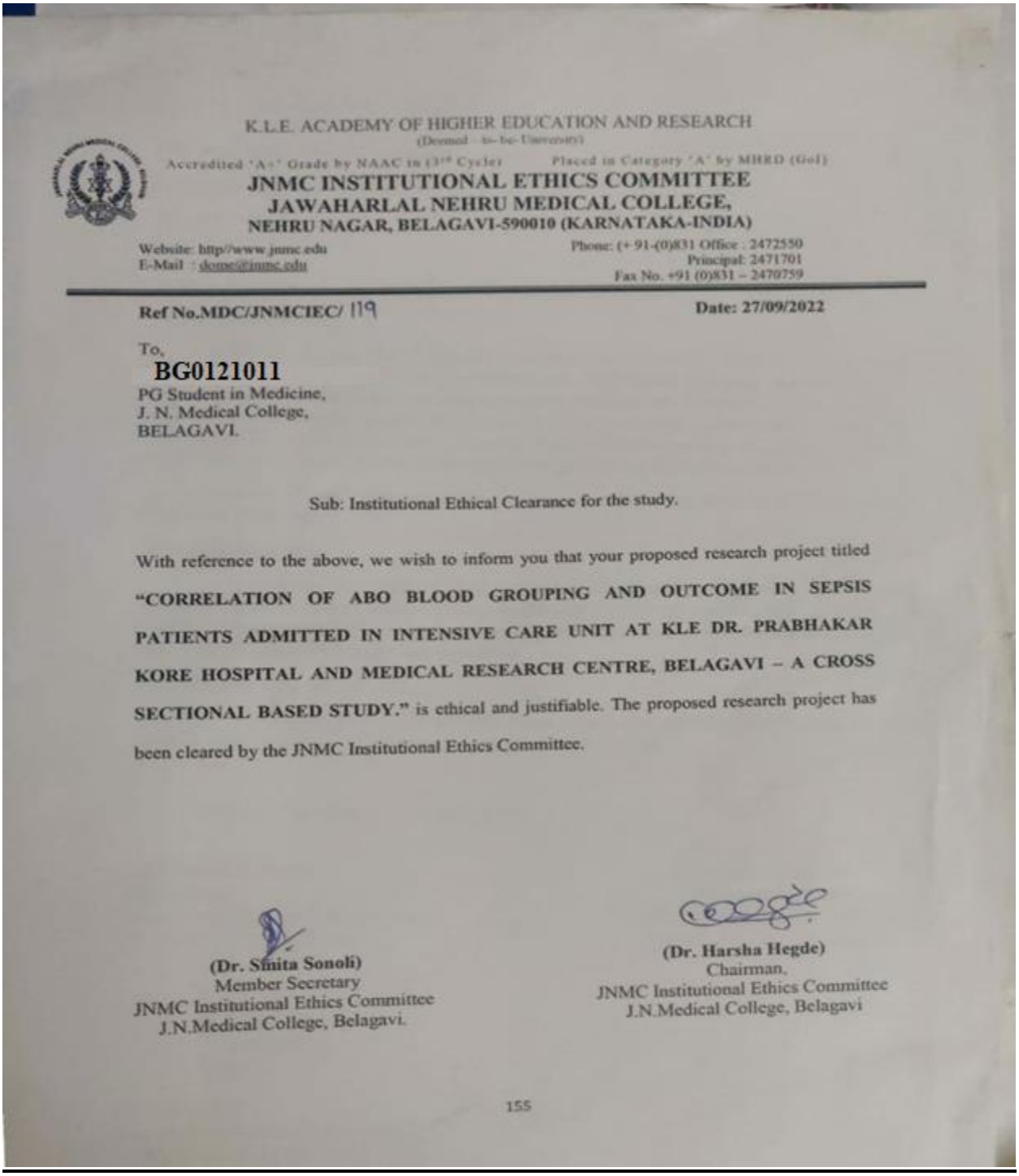

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JNMC Institutional Ethics Committee
J.N.Medical College, Belagavi

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LIST OF ABBREVIATIONS

1. RBC: Red Blood Cells
2. AKI: Acute Kidney Injury
3. ARDS: Acute Respiratory Distress Syndrome
4. SNPs: Single-Nucleotide Polymorphisms
5. CDC: Centre for Disease Control
6. CSF: Cerebro spinal fluid
7. AHRF: Acute Hypoxemic Respiratory Failure
8. CAMs: Cell Adhesion Molecules
9. SAPS II: Simplified Acute Physiology Score
10. LOS: Length of Stay
11. ICAM-1: Intercellular adhesion molecule-1

ABSTRACT

Background: ABO blood group has therapeutic value in pregnancy and blood transfusion. Nevertheless, the field of science does not completely understand the value of ABO blood group in other illnesses or disorders and overall fatality. Even though ABO blood grouping is an un-modifiable risk factor, it can help identify people who are more likely to have a bad prognosis. It aids in understanding why two patients with the same presentation, sepsis score, and treatment have such disparate outcomes. Hence this study aimed to determine the role of ABO blood type in risk stratification for sepsis.

Methods: This observational cross-sectional study conducted at KLES Dr. Prabhakar Kore Hospital from January to December 2023, included 240 ICU admitted sepsis patients. Patients underwent clinical examination and investigations such as blood grouping, total WBC count. GCS and qSOFA scores were also noted. Outcome was measured in terms of expired or improved and the association between blood type and sepsis mortality was found out using SPSS version 21.

Results: In the present study among the total of 240 study subjects, 72.9% (n=175) were improved and the remaining 27.1% (n=65) were expired. When the study individuals' baseline characteristics and the outcome were compared, it was discovered that individuals with high total WBC counts had higher mortality rates than those with normal or low WBC counts. The difference was determined to be statistically significant with a p value of 0.037 using the Chi-square test. With a p value of 0.002, GCS was also shown to be significantly associated with mortality. It is evident from the analysis that high qSOFA score is seen more (38.4%) among the O blood group patients and the difference observed is statistically significant with the p value of <0.001. When

compared to other blood types, it was shown that the O group had a higher sepsis mortality rate. The probabilities of the O group having **3.15** times higher mortality than other blood types were also determined to be significantly different, with a p value of 0.001 (AOR=3.152, 95% CI:1.352, 7.348).

Conclusion: sepsis patients with O blood group, high WBC count, severe GCS and high qSOFA have high mortality rate. However ABO blood grouping is a non-modifiable risk factor, it can help identify people who are more likely to have a bad prognosis. Here in this study, as we found that O type has higher morality, they should be taken more care of. It is necessary to conduct further research to pinpoint the precise mechanisms behind this association.

Keywords: Septic shock, ABO Blood type, risk stratification.

INTRODUCTION

Before the discovery of the ABO blood groups, transfusions involving human beings as well as animal blood sources were associated with significant fatality rates since blood composition differences between people and animals as well as within the human population were not well understood. Based on the presence or absence of surface antigens on RBCs, humans and a large number of other mammals were classified into an ABO blood group since the ABO blood groups were discovered in 1900. The inheritance of genes on chromosome 9 determines the four human ABO groups (A, B, O and AB).

On the surface of RBC, the carbohydrate sugar that is present for the A antigen is N-acetylgalactosamine and for the B antigen is D-galactose they distinguish the different blood types. The H antigen serves as the foundation for these oligosaccharides; if it is left unaltered, the blood group will be O since either the A or the B antigens cannot bind to red blood cells. [1]

The gene that codes for a glycosyl-transferring enzyme controls variations in oligosaccharide composition. Endothelial damage, especially disruption of the glycocalyx, has been shown to be a major factor in the pathophysiology of organ dysfunction associated with critical illness. It increases the risk of both organ failure and mortality in critically ill patients.[2]

Failure of the organs, such as AKI and ARDS, can worsen sepsis and raise the risk of mortality.[3, 4]

In individuals with sepsis or trauma, ABO blood type A appears to increase the risk of acute kidney injury and acute respiratory distress syndrome. It is unclear; therefore, if an ABO blood type raises the mortality rate associated with sepsis, much alone what the underlying cause could be.[5-8]

Glycans that are present on the surfaces of erythrocytes and other cells determine the ABO blood types. [5] Since the beginning of the twentieth century, research has been done on the connections between blood type and disease. The ABO blood type is frequently utilized in clinical practice. Blood type O may be associated with an increased risk of cancer, venous thromboembolism, myocardial infarction, trauma, gastrointestinal bleeding, and acute renal damage according to recent research.[6, 7]

It is commonly acknowledged that the ABO blood group has therapeutic value in pregnancy and blood transfusion. Nevertheless, the field of science does not completely understand the value of ABO blood group in other illnesses or disorders and overall fatality. Even though ABO blood grouping is an un-modifiable risk factor, it can help identify people who are more likely to have a bad prognosis. It aids in understanding why two patients with the same presentation, sepsis score, and treatment have such disparate outcomes. Hence this study aimed to determine the role of ABO blood type in risk stratification for sepsis and the factors that potentially affect the prognosis of sepsis patients in different blood groups.

AIM OF THE STUDY

To identify the role of ABO blood type in risk stratification for septic shock

OBJECTIVES

- To identify the role of ABO blood type in risk stratification for septic shock
- To evaluate the effect of ABO blood group on septic shock.

REVIEW OF LITERATURE

Discovery of Blood groups:

Early in the 20th century, Austrian-American biologist, Karl Landsteiner found that some people's red blood cells (RBCs) were agglutinated by the serum of others. He showed how blood might be divided into groups and observed the agglutination patterns. As a result, the first blood group system, ABO, was discovered; Landsteiner received the Nobel Prize.[9]

Landsteiner claimed that the antibodies in the serum and the antigens on the RBCs were what caused the interactions between the serum and RBCs. Agglutination occurs when the antibodies in the serum attach themselves to the RBC antigens. Based on which antigen the RBC expressed, he designated the antigens A and B, and blood was classified as either blood type A or B. A third blood group's red blood cells lacked the traits of blood types A and B. This blood group was called "O" from the German word "Ohne," which means "without." The very next year, the fourth blood type, AB, was added to the ABO blood group system. These RBCs expressed both the antigens A and B.[9, 10]

Scientists proved in 1910 that the RBC antigens are inherited and that the A and B antigens co-dominate over the O antigens. At first, it was not very clear how to determine someone's blood type, but in 1924, Bernstein's "three allele model" offered the solution.[9, 10]

The ABO blood group antigens are encoded by three different (allelic) versions of the ABO genetic locus: A, B, and O. The following table shows the six likely genotypes and

the four different phenotypes (blood types) that can arise from a child inheriting one of the three alleles from each parent.

Table 1: Inheritance of ABO blood groups

Allele from the father	Allele from the mother	Genotype of progeny	Phenotype of progeny
A	A	AA	A
B	A	AB	AB
O	A	AO	A
A	B	AB	AB
B	B	BB	B
O	B	BO	B
O	O	OO	O

Since the turn of the 20th century, research has been done on the potential association between blood type and illness. ABO blood typing is frequently utilized in therapeutic settings. An individual's susceptibility to certain diseases has been linked to their ABO phenotype.[11]

Antigens of ABO blood groups:

There are four antigens in total. A, B, AB, and A1 are those. The oligosaccharide sequence identifies the antigen type—A, B, or A1.

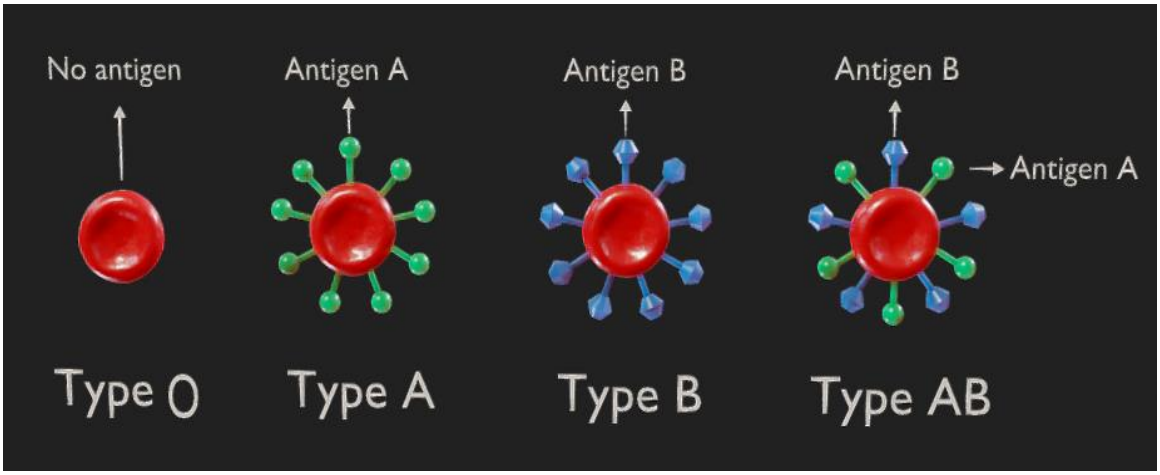


Figure 1: Antigens of ABO blood groups

The ABO blood group antigens are linked to oligosaccharide chains which protrude past the surface of red blood cells. These chains are fastened to proteins and lipids present in red blood cell membranes. There are three primary allelic forms of the ABO locus: A, B, and O. The last stage of the synthesis of the A and B antigens is catalyzed by a glycosyltransferase that is expressed by the A and B alleles, respectively. Several SNPs in the ABO gene, which result in A and B transferases with four amino acid changes, are the cause of the A/B polymorphism. The H antigen, which is the precursor to the ABO antigen, is unaffected by the inactive glycosyltransferase of the O allele.[10]

The two glycans, antigen A, N-acetylgalactosamine (GalNAc α 1-3(Fuc α 1+2)Galβ1), and antigen B, galactose (Galα1 - 3(Fucα1 + 2)Galβ1), are the result of two different

glycosyl transferase activities and are responsible for the range of phenotypic expressions associated with ABO blood grouping.

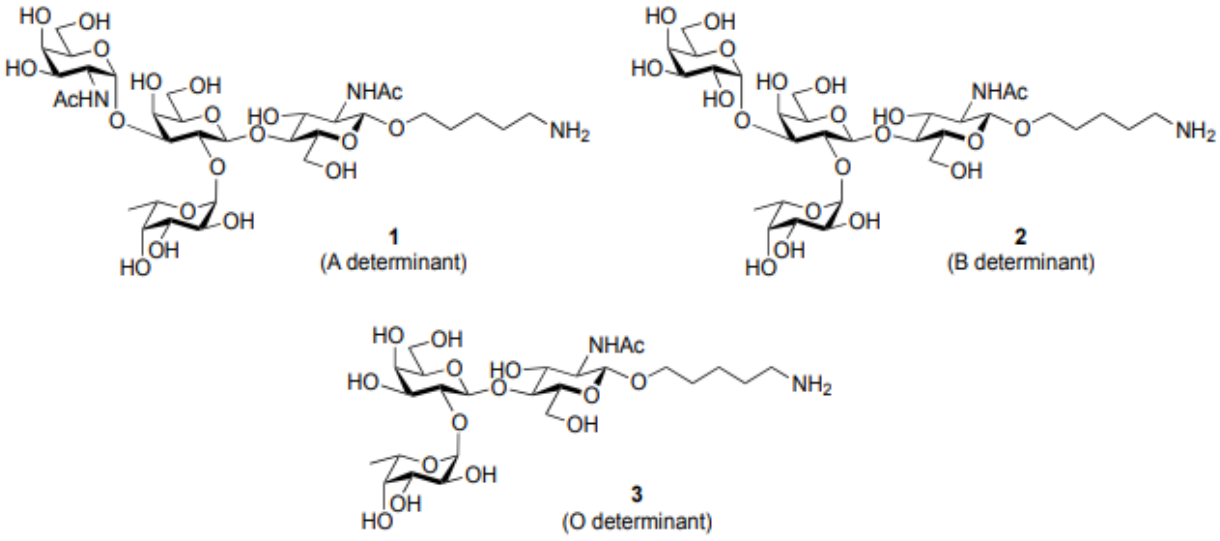


Figure 2: Structure of target of ABO blood group antigens

The H antigen is combined along with either galactose or N-acetylgalactose amine to create antigens A and B, respectively. The O blood group is the result of the lack of both antigens due to a frame shift mutation at the N-terminus of the related enzyme.

The glycosphingolipids or glycoproteins on the surface of RBCs as well as a range of various tissues and cells, such as the gastrointestinal tract, skin, broncho-pulmonary system, urogenital epithelial tissue, are frequently the sites of expression for these antigens. ABH antigens can be found also in a variety of body secretions and fluids, such as saliva, breast milk, and semen, in addition to these antigens.

Every erythrocyte expresses over 2 million antigens of ABO blood group, which are then taken up from plasma by B cells, T cells, and platelets, among other blood cells.[10]

The function of the ABO antigens remains unknown. Individuals who do not have the A and B antigens are in good health, indicating that these antigens are not very significant, at least not in the present era.

Antibodies produced against ABO antigens:

All people have naturally occurring immunoglobulins called ABO antibodies.
The presence on red blood cells of A and B antigen and in individual sera of anti-A and anti-B antibodies are found to be inversely correlated. Due to their high reactivity, they are crucial for transfusion medicine since an improper or mismatched organ transplant or blood transfusion might produce a fatal hyper-acute immunological response.

According to Landsteiner, this inverse relationship occurs when an individual's serum contains antibodies to the missing antigen. For example, people with phenotype A would have an A antigen on their erythrocytes and anti-B in their sera, which would have caused B phenotype erythrocytes to clump together, and people with phenotype B would have anti-A antibodies in their sera, which would have caused A phenotype RBCs to clump together.

Although red blood cells (RBCs) do not have any antigen on their surface, blood type O serum contains both anti-A and anti-B antibodies. Blood type AB phenotype holders possessed both A and B antigens and did not have any type of antibodies in their sera. Anti-A and anti-B antibodies that are naturally present in the recipient's serum attach to

the corresponding antigens of the transfused red blood cells in an individual with blood type O who receives a blood type other than O. This can eventually result in complement fixing and severe hemolysis with intravascular coagulation, which can cause shock, acute renal failure, and even death.

Individuals can therefore be divided into A, B, O, and AB phenotypes based on the patterns of erythrocyte agglutination. Clerical error can be the death warrant of receiving the incorrect blood transfusion, even if standard blood grouping and cross-matching of blood types and products of donors and receivers can minimize adverse transfusion responses induced by ABO antibodies during blood transfusion. Safe blood transfusion has been made possible.

Basically, the ABO antibodies are isohaemaglutinins. They are mostly of the IgM type and are absent in newborns. They are created a few months after birth and were first referred to as naturally occurring antibodies due to the misconception that no antigenic stimulus is required for their creation, however this has been disproven. It is now understood that bacteria found in the environment and in the digestive system encourage the formation of these naturally occurring antibodies. For example, it has been shown that entrobacteriaceae have features on their lipopolysaccharide coats that are similar to ABO antigens.

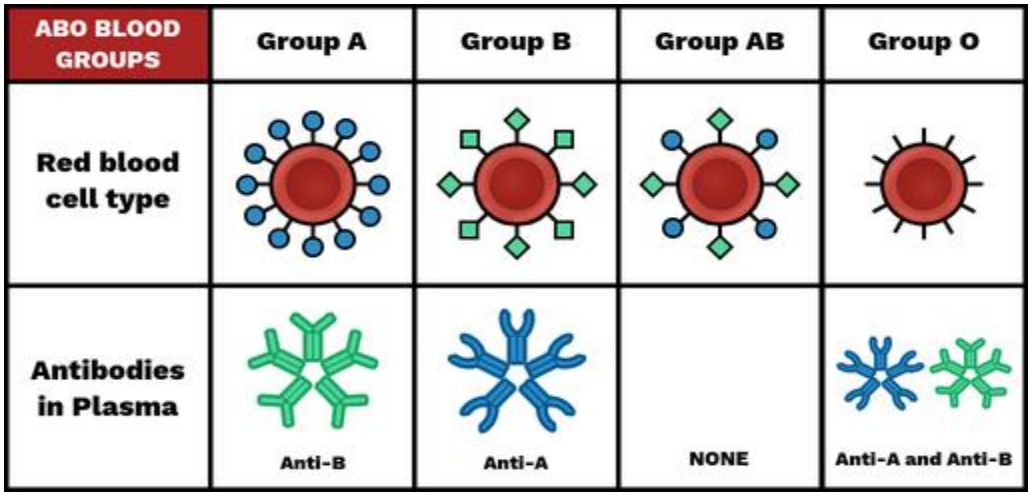


Figure 3: Antibodies produced against each blood group antigen

The antibody types produced against blood group antigens are IgG and IgM. Anti-A and anti-B antibodies bind to red blood cells and initiate the complement system, destroying the RBCs whilst they remain still in the bloodstream and causing IVH (intravascular hemolysis).[10]

Expression:

The ABO blood group antigens, although commonly associated with red blood cells, are expressed on a broad variety of human organs including epithelial and endothelial cells. Each red blood cell in the human body expresses about two million ABO blood group antigens. B cells, T cells, and platelets absorb the ABO blood group antigens from the plasma into their blood cells. The ABO blood group antigens are soluble in "secretors" and can be found in their saliva as well as other bodily fluids (excluding CSF).

Numerous illnesses can change the ABO phenotype of the person. Patients may "acquire" the B antigen following a necrotizing infection when bacteria secrete an enzyme into the bloodstream that converts the A1 antigen to the B-like antigen. Patients shouldn’t not get blood products that carry the B antigen during this period since their sera continues to have the anti-B. The patients' blood types stabilize after the root-cause or underlying infection is treated.

Patients may potentially "lose" ABO blood group antigens due to the illness. ABO blood group antigen expression may be weakened by any illness that raises the body's need for red blood cells, such as thalassemia. Hematological malignancies have the ability to alter the sugar chains which contain the ABO blood type antigens. This means that B antigens and ABO blood group antigens can be used as tumor markers for the myeloproliferative disorders, myelodysplasia and acute leukemia.

ABO and diseases’ associations

On the surface of red blood cells, human ABO blood type antigens exhibit distinct phenotypes and glycoconjugate structures that are genetically produced and actively involved in the physiology and pathophysiology of the cells.[12]

Furthermore, certain oligosaccharide structures found in the antigens were used to identify the blood group. Antigens of blood group are therefore secondary gene products, whereas different glycosyltransferase enzymes that help link sugar molecules to the oligosaccharide chain are main gene products. Others' immune systems produce antibodies against these components of carbohydrates because they are perceived as alien things.[13]

The ABO blood type system has been proven to be essential for medical research on numerous illnesses.[14]

However, because some blood groups do not contain their antigens, there have been some controversial doubts raised about the association between the ABO blood group and susceptibility to particular infectious and the noninfectious illnesses. Certain blood types have different blood membrane design and function depending on whether antigens are present or absent. The structural features of blood types enable certain functions to associate blood groups with both health and illness.[15]

Leukocytes, platelets, plasma proteins, and other cell surface enzymes besides red blood cells all have blood group antigens on them. [16]

Furthermore, soluble blood group antigens may be present in bodily fluids like urine, gastric secretions, amniotic fluid, seminal fluid, perspiration, saliva, and breast milk.[17, 18]

Still, one gene produces the vast majority of antigens. The structure of an antigen can be altered by genetic modifications such as inversions, insertions, deletions, alternative splicing, or SNPs (single nucleotide polymorphisms), but they can also lead to the creation of new antigens or even the complete absence of expression. After a great deal of research, the ABO blood group antigens were the first to be discovered and are arguably of greatest importance.

The multifunctional roles of the glycoconjugate structures on red blood cells include transporters, adhesion molecules, channels, structural proteins, and receptors for the external ligands, bacteria, viruses, and parasites.[19, 20]

A surprising number of structures are involved in human disease and normal RBC development. Some of these structures function as cell adhesion molecules (CAMs).

However, the precise processes behind the observed correlations between blood type antigens and the disease in the adhesion molecules remain unclear. [21, 22]

SEPSIS

According to the World Health Organization, Sepsis is “a serious condition that happens when the body’s immune system has an extreme response to an infection. The body’s reaction causes damage to its own tissues and organs.”[23]

Sepsis, severe sepsis, and septic shock are the three stages into which medical professionals used to classify sepsis. They may now recognize the ailment on a broader spectrum. This spectrum includes conditions like infection and bacteremia to sepsis and septic shock, which can cause multiple organ failure and even death.[24]

Population at risk of sepsis:

- Elderly
- Expectant or recently delivered women
- Infants
- Hospitalized and those patients who are in critical care units
- Immuno-compromised and those with long-term medical disorders

Signs & symptoms of sepsis:

There are numerous possible signs of sepsis because it can affect many different parts of the body.[23]

- Extreme physical pain or discomfort
- Fever or hypothermia and also shivering
- Clammy and damp skin
- Disorientation and breathing difficulties
- Decreased urine production
- Rapid heart rate, weak pulse, or low blood pressure

Common causes of sepsis:

In all age categories combined, the main causes of sepsis cases and deaths associated to sepsis in 2017 were lower respiratory infections and diarrheal diseases. But non-communicable diseases are on the rise; in 2017, one-third of sepsis cases and almost half of all sepsis-associated deaths were connected to an underlying injury or chronic condition.

Maternal problems were the most common non-communicable disease that sepsis made worse. The most frequent reasons for sepsis-related mortality in children were lower respiratory infections, diarrheal diseases, and anomalies in newborns.

Sepsis in both neonates and mothers is most commonly caused by Group B streptococcus, while E. coli is becoming a more serious hazard.[23]

Even though there are numerous other potential causes of sepsis, correlation was found also between ABO blood grouping and sepsis mortality.

According to recent research, O blood type may raise the risk of malignancy[25], venous thromboembolism[26], MI, AKI[6], trauma[27], and GI bleeding[28].

To the finest of the information we have, no research has been done on the accurate significance of ABO blood type in sepsis.

ARTICLES REVIEW

A retrospective cohort study was conducted by Theis S. Itenov et al to ascertain whether the ABO blood types differ in the risks those seriously ill patients with sepsis face for acute kidney injury (AKI), endothelial damage, and 30-day death. This involved three separate cohorts of critically ill adults with septic shock from Scandinavia and the United States. Within each cohort, we examined the 30-day mortality for each blood type and combined the data into a meta-analysis. 12,342 patients with severe sepsis were included. Blood type B was associated with a somewhat decreased risk of 30-day all-cause death in a pooled study when compared to non-blood type B (adjusted Hazard Ratio 0.88; 95%-CI 0.79–0.98; p = 0.02). The chance of developing AKI remained unchanged. They came to the conclusion that blood type B septic patients experienced a slight decrease in mortality and less endothelial damage.[2]

An international cross-sectional research on patients during the 1st week of intensive care unit admission had conducted at 97 centers as part of the Acute Kidney Injury-Epidemiologic Prospective Investigation (AKI-EPI) study. They used hospital discharge results and the Kidney Disease: Improving Global Outcomes (KDIGO) criteria to measure AKI. Out of the 1802 ICU patients, 1032 patients [57.3%; 95% CI 55.0-59.6] developed AKI in total. When controlling for other factors, there was a correlation between higher AKI severity and hospital death: odds ratios for stage 1 were 1.679

(95% CI 0.890-3.169; p = 0.109), stage 2 was 2.945 (95% CI 1.382-6.276; p = 0.005), and stage 3 was 6.884 (95% CI 3.876-12.228; p < 0.001).

They discovered that over half of the ICU patients had AKI. Patients with AKI had worse renal function when they were discharged from the hospital, and increasing AKI severity was linked to higher mortality rates[3].

Patients who satisfied the Berlin definition of Acute Respiratory Distress Syndrome were included in a multicenter prospective longitudinal study. Data on the utilization of adjunct therapy, ventilator management, and baseline values were gathered. During the study stretch, 18,793 patients were admitted to participating ICUs; 672 of these patients met the Berlin ARDS criteria, and 527 of these patients were included in the analysis. Pneumonia was the most frequent predisposing cause for ARDS in 402 (77.0) individuals. The rate of prevalence for mild ARDS was 9.7% (51/527), moderate ARDS were 47.4% (250/527), and severe ARDS was 42.9% (226/527). During their ICU stays, 400 patients, or 75.9% of the total, were treated with invasive mechanical ventilation. A positive end-expository pressure (PEEP) of 8 cm H_2O and a tidal volume of 6.8 (5.8–7.9) milliliter/kilogram of their anticipated body weight were administered to all ARDS patients. In 61 (15.3%) and 85 (16.1%) of the ventilated patients, recruitment maneuvers (RMs) and prone positions were employed, respectively. For 92 patients (17.5%), life-sustaining care was stopped. Among the 244 ARDS patients, 31.4% (n=16) with mild ARDS, 40.4% (n=101) with moderate ARDS, and 56.2% (n=127) with severe ARDS passed away in the hospital where these patients were included in the study.[4]

In prospective cohort studies conducted at the hospital of the University of Pennsylvania, patients (n=732) who were critically unwell and had suffered a significant trauma were monitored for the development of ARDS for five days. Furthermore, 976 medical patients with severe sepsis had ARDS monitoring every five days. Out of the 732 patients admitted with trauma, 197 (27%), developed ARDS. While blood type A was not linked to a higher risk of ARDS in blacks (adjusted Odds Ratio, 0.61; 95% CI, 0.33-1.13; P =0.114), it was among white race (37% versus 24%; adjusted Odds Ratio, 1.88; 95% Confidence Interval, 1.14-3.12; P =0.014). Of the 976 patients with severe sepsis, 23% (or 222) developed ARDS. Additionally, blood type A was linked to a greatest risk of ARDS in white people (31% versus 21%; adjusted Odds Ratio, 1.67; 95% CI, 1.08-2.59; P =0.021), but not in black people (adjusted Odds Ratio, 1.17; 95% CI, 0.59-2.33; P =0.652). They concluded that ARDS is more likely to occur in patients among whites with severe trauma and severe sepsis if they have blood type A. These findings point to a possible involvement of glycosyltransferases and ABO glycans in ARDS vulnerability.[5]

In the study conducted by Reilly John P et al, it was observed that 134 patients (27%), out of 497 trauma victims, experienced AKI. Among European descent patients, blood type A was linked in multivariable analysis to a greater risk of AKI compared to type O (n=229; AOR, 0.28 vs 0.14; risk ratio, 0.14; 95% CI (confidence interval), 0.03 to 0.24; P=0.02). 326 (43%), out of 759 patients with sepsis, suffered Acute Kidney Injury. Among patients of European heritage, blood type A once more carried a greater risk of AKI compared to type O (n=437; adjusted risk ratio, 0.53 vs. 0.40; odds risk, 0.14; 95%

CI, 0.04-0.23; P=0.01). They found out that there is potential role for the ABO glycans in AKI vulnerability and blood type A is independently linked to the higher risk of AKI.[6]

A retrospective cohort study including many centers examined the electronic medical records of 1732 patients diagnosed with AHRF. Patients with ABO blood type A made up 39.9% of the total, compared to 60.1% of patients with other blood types. When stratified by ABO blood type, there was no difference observed in ICU mortality (25%) or ICU Length of Stay (LOS) (median [IQR], 5 days) (Mortality, overall p-value = 0.905; ICU LOS, overall p value = .609). When pushed into the study, ABO blood type didn't predict ICU outcome; however, SAPS II is a positive predictor of both mortality among ICU patients (adjusted odds ratio [AOR], 32.80; 95% Confidence Interval, 18.80–57.24; $p < 0.001$) and ICU LOS (β coefficient, 0.55; 95% Confidence Interval (0.35–0.75); $p < 0.001$) at multivariate analyses.[7]

In a single-center retrospective observational study conducted by Robert Slade et al, with an aim to determine the effect of ABO blood group on intensive care survival, it was noticed that when compared to other blood groups, patients in intensive care who belonged to blood group AB were found to have a greater 90-day survival rate. Blood type and the proportion of patients having transfusions did not correlate.[8]

The literature demonstrating the correlation between ABO and other disorders was reviewed by Abegaz SB. He noted that there is a correlation between ABO blood types and a higher chance of contracting many illnesses, such as malaria, circulatory diseases, cancer, heart disease, infections, hematologic disorders, and cognitive impairments. Moreover, independent of age, gender, ethnicity, or geographic region, blood group AB patients were found to have an increased risk of cognitive impairment.

People with cognitive impairment were also more likely to have conditions like diabetes, obesity, dyslipidemia, hypertension, and cardiovascular disease (CVD).

Early etiological investigations have associated greater incidences of cholera, tuberculosis infections, plague, and mumps with blood type O. Alternatively, blood types A, B, and AB have been linked to increased incidence of smallpox, salmonella, E. coli, and tuberculosis, as well as higher rates of gonorrhea, Streptococcus pneumoniae, TB, and salmonella infections.[12]

In the Taiwan cohort, blood group A was found to be significantly associated with an increased risk of gastric cancer incidence (Hazard Ratio, 1.38 [95% Confidence Interval, 1.11–1.72]) and mortality (HR, 1.38 [95% CI, 1.02–1.86) when compared with blood type O, even after controlling for sex, age, tobacco smoking, education, alcohol consumption, body mass index and physical inactivity. Blood type B achieved statistical significance for both incidence and death (Hazard Ratio, 1.59 [95% CI, 1.02, 2.48] and Hazard Ratio, 1.63 [95% CI, 1.02, 2.60]), indicating that non-O blood types have a higher chance of developing pancreatic cancer. Blood type AB exhibited a decreased incidence of renal cancer (Hazard Ratio, 0.41 [95% Confidence Interval, 0.18–0.93]) compared to type O. They concluded that the chances of cancer vary depending on the ABO blood type.

Specifically, blood type A carries an increased risk of stomach cancer, whereas blood types B, AB, and O have a greater risk of pancreatic cancer.[25]

Meta-analysis of the available literature was carried out to assess the potential clinical implications of the various ABO blood types on the risk of VTE. We included 38 papers with 10,305 VTE cases in the systematic review via an digital search technique utilizing

Medline and Embase, ‘a manual review of the International Society on Thrombosis and Haemostasis’ abstract books, and a study of the reference lists of all the retrieved articles. When comparing VTE patients to controls, the prevalence of non-O blood group was substantially greater, as evidenced by a pooled odds ratio of 2.09 (95% confidence interval, 1.83 to2.38; $p < 0.00001$). The highest incidence of VTE was found in patients without O-factor V Leiden (Odds Ratio 7.60, 95% Confidence Interval, 3.21 to 17.99). In conclusion, non-O blood type is a contender to be one of the significant genetic risk factors for venous thrombosis given its ubiquity.[26]

In a study with a retrospective observational design involved 901 patients in total. The ABO blood types of type A, 285 (32%), type O, 284 (32%), and type AB, 123 (13%) and type B, 209 (23%), were used to split the study population. High death rates were linked to blood type O (28% in O group patients vs. 11% in patients with non-O blood types; $p < 0.001$). Additionally, a multivariate model revealed this link (adjusted odds ratio (AOR) = 2.86, 95% CI 1.84-4.46; $p < 0.001$). Blood type O had an effect on all-cause in-hospital mortality. It greatly affected the results and was found to be substantially linked to high mortality in individuals with severe trauma.[27]

This study by Liu Y et al. distribution of We calculated the doubling times of infection, infection growth factor, and death cases, infection and death cases and reproductive number in connection to the distribution of blood types. The growth factor of infection and death cases is positively and strongly correlated with the proportion of the population with blood group A, whereas there is an adverse correlation with the percentage of the population with blood group B. In comparison to the lower blood type A population (<30%), the greater blood type A population (≤30%) showed higher growth

factors, faster case multiplication times for infections and deaths, and a higher number of cases of infections & deaths. The dynamics of the epidemic were exacerbated by these findings. ABO blood group distribution and SARS-CoV-2 were discovered to be correlated, which may assist contain the COVID-19 pandemic.[29]

MATERIALS AND METHODS

Study design: Observational cross-sectional study

Study setting: ‘KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi’

About study area: ‘Belagavi's KLES Dr. Prabhakar Kore Hospital and Medical Research Center’ is a contemporary hospital with 2000 beds, designed by Dr. Prabhakar Basaprabhu Kore, is situated on a 17-acre tract of ground and provides concessional beds to the underprivileged and destitute.

Over 300 expert doctors, experienced nurses, and technical and paramedical professionals are available at the facility. Thirty operating rooms and a host of specialty and super specialized features are part of the cutting edge infrastructure. It has received many honors in the fields of cardiac and multiple organ transplants. Children with cleft lip and palate and other dental defects can benefit from surgery according to the US-based "Smile Train Project." The hospital has a history of receiving numerous honors and recognitions, including a "A" Grade accreditation from the NAAC. The hospital maintains satellite medical facilities at Jamkhandi, Gokak, Ankola, and Chikodi. The hospital is committed to providing high-quality healthcare to all segments of society. It sets high standards and makes sure that the KLES Dr. Prabhakar Kore Hospital & MRC is a model that represents the greatest healthcare in the nation.

Study duration: 1 year, that is from Jan 2023 to Dec 2023.

Study population: Patients who has admitted in the ICU of the 'KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi.'

Inclusion criteria: Age ≥ 18 years

Exclusion criteria:

1. Age < 18 years
2. Patients with the previous history of hematological malignancies.
3. Patients with chronic illness such as tuberculosis / HIV
4. During labor
5. Cases following abortion, ectopic or molar pregnancy.

Sample size:

$n= 2S^2(Z_{1-\alpha}+Z_{1-\beta}) / d^2$

Where, Z1-α = Z value for alpha level (1.96 at 5% α error or 95 % confidence)

Z1-β = Z value for beta level (1.282 at 10% β error or 90% power)

S=pooled SD = (S1 + S2)/2

d - margin of error = 0.30

substituting the values,

S= 1.175

$Z_{1-\alpha} = 1.96$

$Z_{1-\beta} = 0.84$

d = 0.30

we get a value of 240

sample size n = 60 should be taken in each group.

Sampling technique: Convenient sampling method

Study tool: Pre-tested structured validated questionnaire was used followed by necessary investigations.

Table 2: Components of the questionnaire:

Component	Includes
Personal details of the patient	IP no, Name, Age, Sex, Address.
Vitals	Pulse rate (PR), Respiratory rate (RR), Blood pressure (BP), Glasgow Coma Scale (GCS), qSOFA score
Investigations	Blood grouping, Total WBC count
Duration of hospital stay	Total duration of stay in the hospital, Duration of stay in ICU.
Others	Comorbidities, Diagnosis, Foci of sepsis
Outcome	Improved/Expired/DAMA

Operational definitions:

Pulse rate:

“The pulse rate is a measurement of the heart rate, or the number of times the heart beats per minute.”

When the heart distributes blood via the arteries, the arteries dilate and contract in reaction to the blood flow. Taking a pulse can show the intensity and rhythm of the heart in addition to heart rate.

A healthy adult's pulse beats between 60 and 100 times per minute on average. The pulse rate can change and even increase in response to physical activity, disease, injuries, and emotions. Beyond the age of twelve, females often have faster heart rates than males.

Heart rates as high as forty beats per minute are not abnormal for athletes who train their cardiovascular systems substantially, such as runners. [30]

Respiratory rate:

“The respiratory rate is the number of breaths a person takes per minute.” When a person is at rest, the rate can be easily calculated by counting the number of times their chest rises in a minute to get an estimate of how many breaths they take. Respiratory rates can increase in cases of fever, illness, and other medical disorders. It is imperative to evaluate breathing and ascertain whether a person is having trouble breathing. [30]

Table 3: Categorization of Respiratory rate [31]:

Respiratory rate (breaths/minute)	Category
12-20	Normal
<12	Bradypnea
>20	Tachypnea

Blood pressure:

“Blood pressure is the force of the blood pushing against the artery walls during contraction and relaxation of the heart.”

A blood pressure measurement yields two readings: the higher reading, known as the systolic blood pressure (SBP), is the pressure within the artery during a heartbeat; the lower reading, known as diastolic blood pressure, is the pressure within the artery during a heartbeat while the heart is at relaxation and filling with blood;

Both readings are expressed in "mm Hg," or millimeters of mercury. This reading indicates the height, to which the mercury column is raised by the blood pressure in device known as a mercury manometer or sphygmomanometer. [30]

Blood pressure is categorized into 5 categories, based on American Heart Association classification, namely “Normal, Elevated, Hypertension stage 1, Stage 2 and Hypertensive Crisis”. The ranges of which are shown in below table:

Table 4: Categorization of Blood pressure: [32]

Category	Systolic (mmHg)		Diastolic (mmHg)
Normal	<120	and	<80
Elevated	120-129	and	<80
Hypertension Stage 1	130-139	or	80-89
Hypertension Stage 2	≥ 140	or	≥90
Hypertensive crisis	>180	and/or	>120

Glasgow Coma Scale:

Graham Teasdale and Bryan Jennett in 1974 published Assessment of Coma and Impaired Consciousness, which included a description of the Scale, called Glasgow Coma Scale (GCS), as a means of knowing the patients' state of consciousness after suffering an acute brain damage.

The scale's findings guide the initial decision-making, and also track the response trends which are crucial in pointing out when new measures are required.[33]

Three distinct components are measured by the GCS:

- Verbal responses (V),
- Motor responses (M), and
- Eye opening (E).

The scores according to response are shown in the table below:

Table 5: Glasgow Coma Scale

Behavior	Response	Score
Eye-opening	Spontaneous	4
	To sound	3
	To pain	2
	No response	1
Verbal response	Oriented	5
	Confused	4
	Words	3
	Sounds	2
	No response	1
Motor Response	Obeys commands	6
	Localizing	5
	Normal flexion	4
	Abnormal flexion	3
	Extension	2
	No response	1

The individual is categorized as vegetative state (score <3), severe (score = 3–8), moderate (score = 9–12), mild (score = 13–15), or based on the sum of their individual scores (i.e., E + V + M).

qSOFA score: Quick Sequential Organ Failure Assessment score

"Consists of bedside clinical parameters and is intended to identify sepsis patients who are more likely to experience adverse outcomes." [34]

Table 6: qSOFA score

Assessment	Score
Respiratory rate >22 breaths / min	1
Systolic BP < 100mm of Hg	1
Altered mental status (GCS<14)	1

Score of ≥ 2 denotes that there is 3-14 fold increase in hospital mortality.

Blood group:

"One of the classes (such as those designated A, B, AB, or O) into which individuals or their blood can be separated on the basis of the presence or absence of specific antigens in the blood." [35]

Blood group testing:

ABO typing is a blood group test used to identify a person's blood type. Usually, a medical practitioner or phlebotomist conducts the test. For this test, a blood sample is mixed with antibodies against both type A and type B blood. Subsequently, clumps (agglutination) or the presence of blood cells sticking to one another are checked in the sample. The clumping and sticking together of the blood cells indicates their adherence to the antibodies. For instance, combining a blood sample with anti-A antibodies identifies the blood sample as belonging to blood group A. [36]

Total Leucocyte count:

“Total leukocyte count refers to the total absolute count of circulating neutrophils, eosinophils, basophils, monocytes and lymphocytes.”[37]

The normal range of WBCs is 4,500 to 11,000/microliter (4.5 to 11.0 × 10^9/L).

Length of Hospital Stay: “Length of stay (LOS) is a clinical metric that measures the time elapsed between a patient’s hospital admittance and discharge.” [38]

Ethical considerations:

1. The study was conducted after obtaining permission from Institutional Ethics Committee.

2. Permission from Principal, Belagavi's KLES Dr. Prabhakar Kore Hospital and Medical Research Center and Superintendent, Belagavi's KLES Dr. Prabhakar Kore Hospital was obtained before the commencement of study.

3. Informed written consent was obtained from all the study participants.

4. Their privacy and confidentiality were maintained throughout the study.

METHOD OF DATA COLLECTION:

After receiving IEC approval, the data collection was started. All patients fulfilling inclusion criteria were approached and after obtaining the informed consent, they were

subjected to the questionnaire and necessary investigations to get the required information.

DATA ANALYSIS:

SPSS version 21 was used to analyze the data. Data was represented in percentages/ proportions for categorical variables and mean & SD for continuous variables. Chi-square test was used as test of significance for categorical data and independent t-test for continuous variables. Logistic regression was used to find out the association.

p-value< 0.05 was considered to be statistically significant. Bar and pie charts were used for pictorial representation.

GANTT CHART

	2022							2023											2024					
	Jun	Jul	Aug	Sep	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	Jun
1	Preparatory phase																								
2	REVIEW OF LITERATURE																								
3								DATA COLLECTION																	
4																				**Data analysis**					
5																				Document preparation					

RESULTS

A. Distribution of participants according to Age:

Age	Frequency	Percent
<30	20	8.3
31-40	33	13.8
41-50	16	6.7
51-60	48	20
>60	123	51.2
Total	240	100.0

In the present study the mean age of the study participants was 57.3±17.6 years which ranged from 18 years to 94 years. For analysis purpose, age was categorized into different groups. 51.2% (n= 123) were from the age group of more than 60 years, 20% (n= 48) were from the age group of 51-60 years, 13.8% (n=33) were of age group 31-40 years, 8.3% (n=20) were from less than 30 years age group, 6.7% (n=16) were found to be in the age group of 41-50 years.

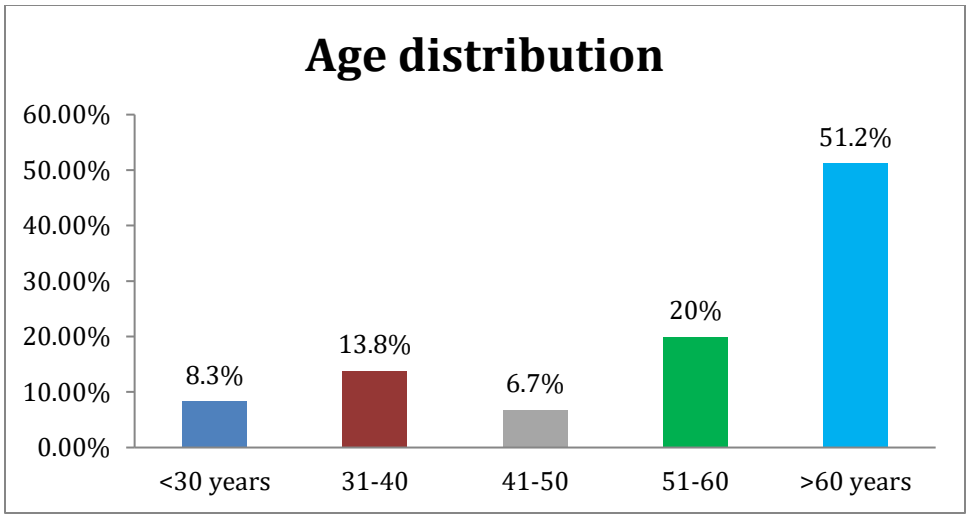


FIGURE 4: DISTRIBUTION OF PARTICIPANTS ACCORDING TO AGE

B. Distribution of study subjects according to gender

Gender	Frequency	Percent
Female	62	25.8
Male	178	74.2
Total	240	100.0

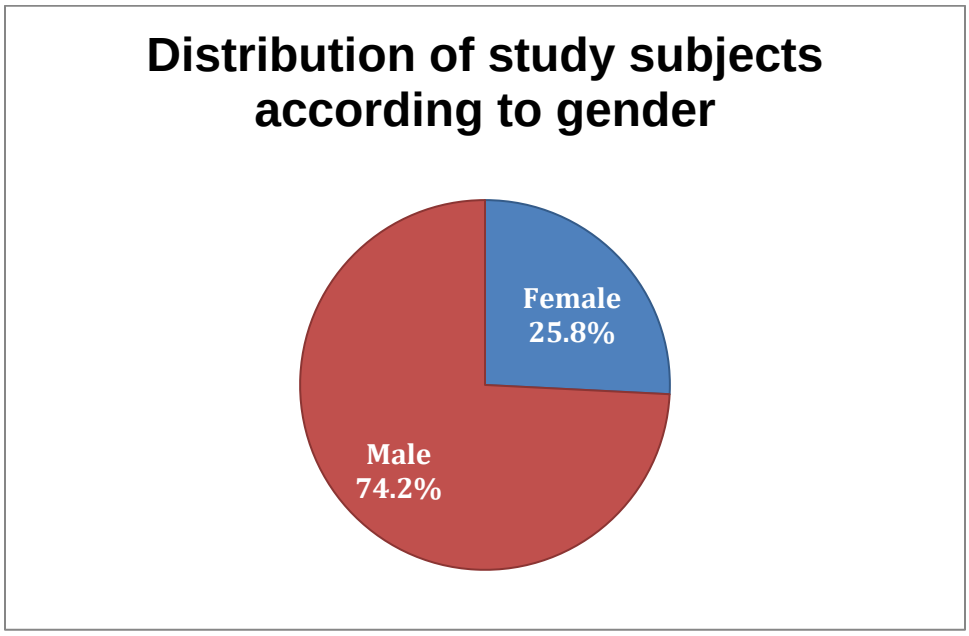


FIGURE 5: DISTRIBUTION OF STUDY SUBJECTS ACCORDING TO GENDER

In the present study 74.2% (n=178) of participants were Males, and 25.8% (n=62) were Females.

C. Distribution according to Blood group among study subjects

Blood group	Frequency	Percent
A	60	25
AB	60	25
B	60	25
O	60	25
Total	240	100.0

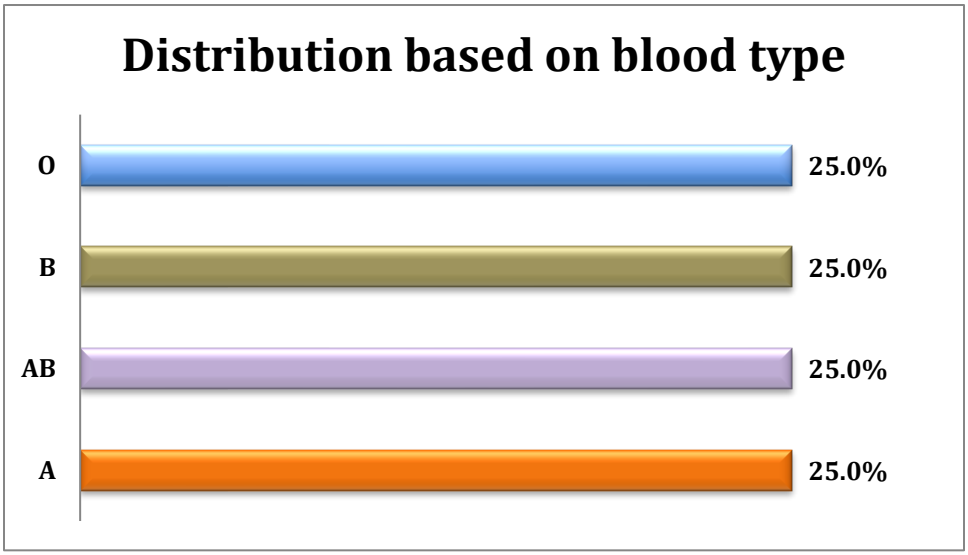


FIGURE 6: DISTRIBUTION ACCORDING TO BLOOD GROUP

Among the study participants, 60 members from each blood group were selected.

D. Distribution of study subjects according to respiratory rate

Respiratory rate	Frequency	Percent
Normal	8	3.3
Tachypnea	232	96.7
Total	240	100.0

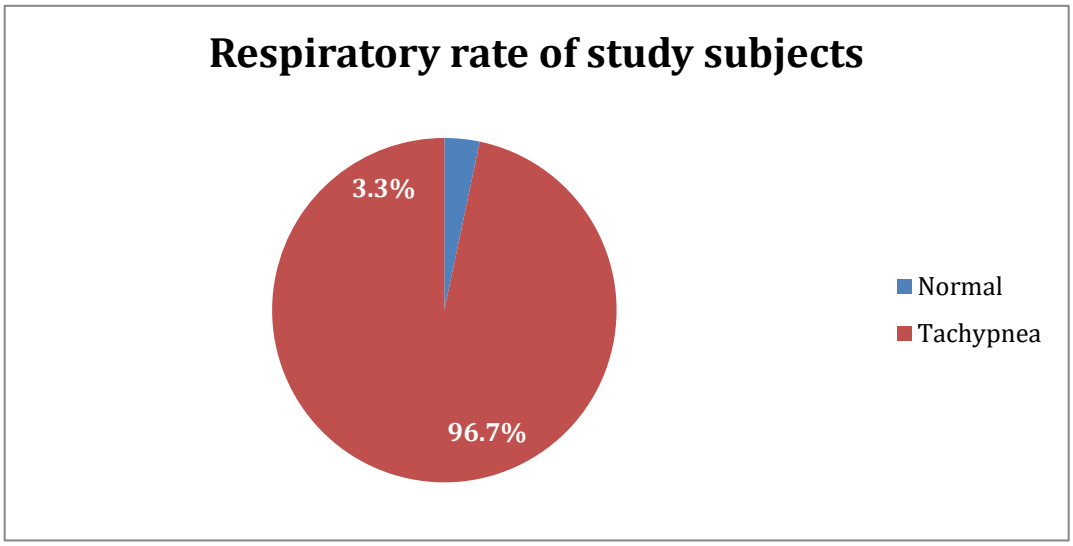


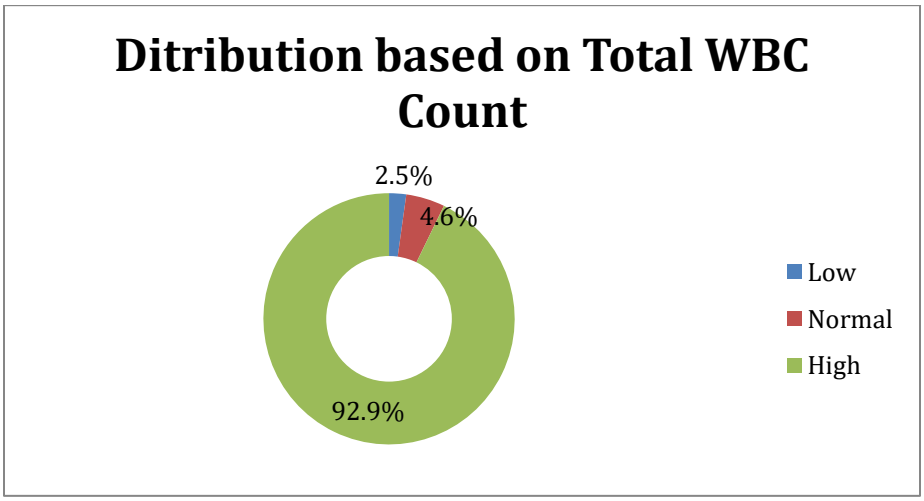
FIGURE 7: DISTRIBUTION OF STUDY SUBJECTS ACCORDING TO RESPIRATORY RATE

Among the participants 96.7% (n=232) were found to have tachypnea and the remaining 3.3% (n=8) were of normal respiratory rate.

E. Distribution according to total WBC count:

TABLE 7: DISTRIBUTION OF STUDY SUBJECTS ACCORDING TO TOTAL WBC COUNT

Total WBC count	Frequency	Percent
Low	6	2.5
Normal	11	4.6
High	223	92.9
Total	240	100.0



In this study, 92.9% were with high total WBC count; whereas 4.6% have normal count and remaining 2.5% were with low WBC count.

F. Distribution according to GCS

GCS	Frequency	Percent
Mild	121	50.4
Moderate	73	30.4
Severe	45	18.8
Vegetative state	1	0.4
Total	240	100.0

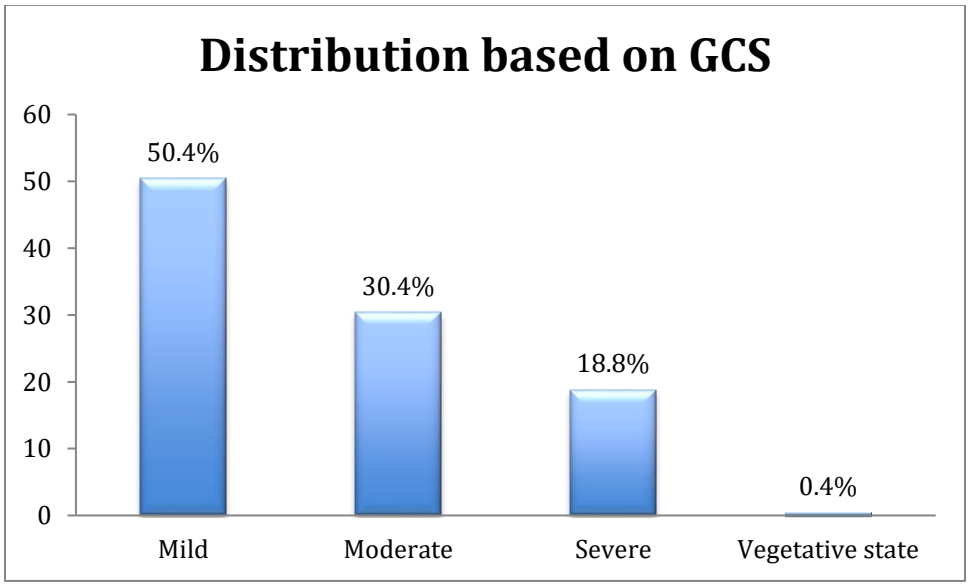


FIGURE 8: DISTRIBUTION OF STUDY SUBJECTS ACCORDING TO GCS

Among the study subjects, 50.4% had mild GCS score whereas 30.4%, 18.8% had moderate and severe GCS and the remaining 0.4% were of vegetative state GCS.

G. Distribution according to foci of infection

Foci of infection	Percent	Frequency
Abdominal	39	16.3
Blood	1	0.4
CNS	18	7.5
Genitourinary	32	13.2
Respiratory	84	35.0
Skin	39	16.3
Unknown	27	11.3
Total	240	100.0

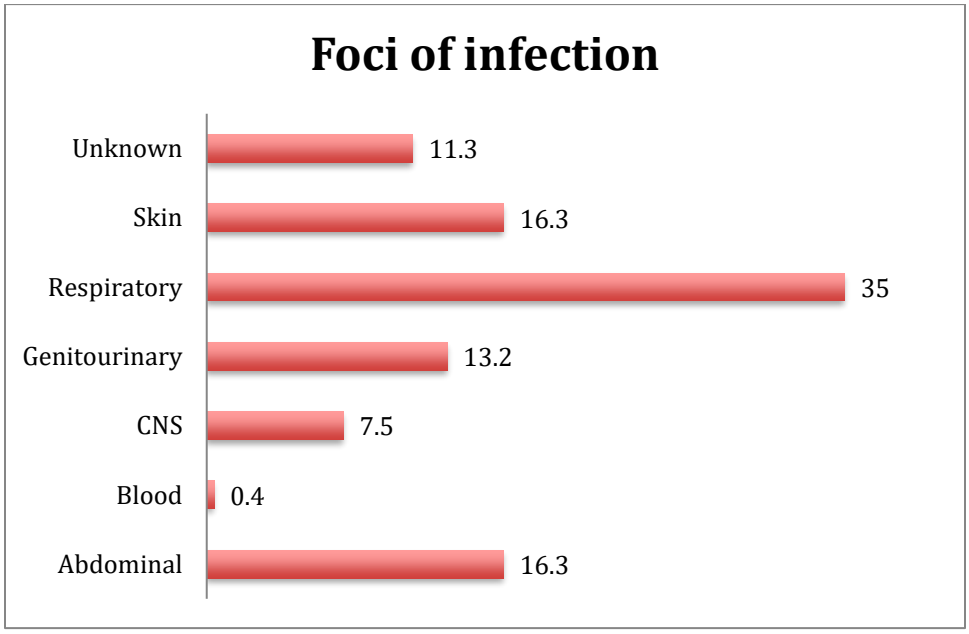


FIGURE 9: DISTRIBUTION OF STUDY SUBJECTS BASED ON FOCI OF INFECTION

The respiratory system was the primary site of infection for the majority (35%), as the above chart illustrates. 16.3% each had an abdominal source and a cutaneous source, 13.2% had an infection in the genitourinary system, and 11.3% had an unknown source. The remaining 7.5% and 0.4%, respectively, were of CNS and vascular source.

H. Distribution based on qSOFA score

qSOFA score	Frequency	Percent
=2	107	44.6
> 2	133	55.4
Total	240	100.0

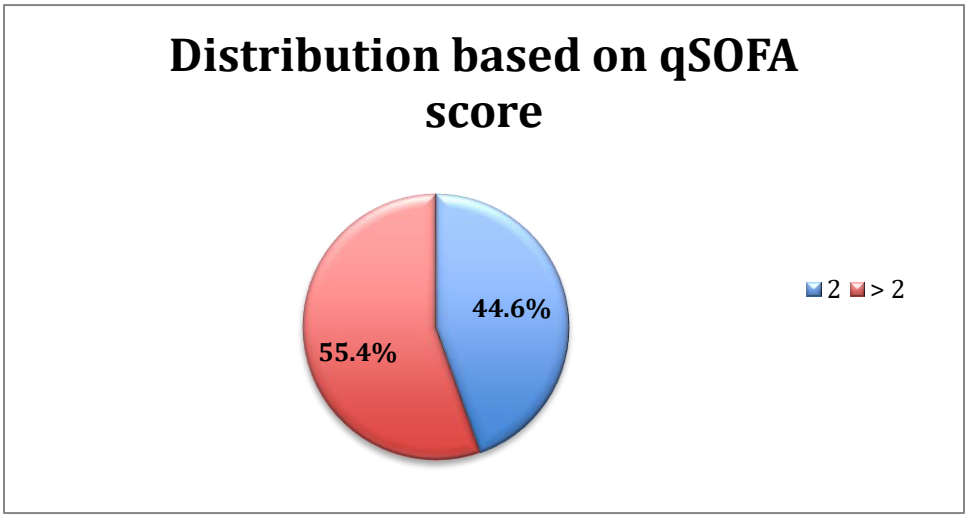


Figure 10: DISTRIBUTION BASED ON qSOFA

In the current study it was noticed that the majority of study subjects (55.4%) had their qSOFA score >2 and the remaining 44.6% had their score =2.

I. Distribution based on outcome

OUTCOME	Frequency	Percent
Expired	65	27.1
Improved	175	72.9
Total	240	100.0

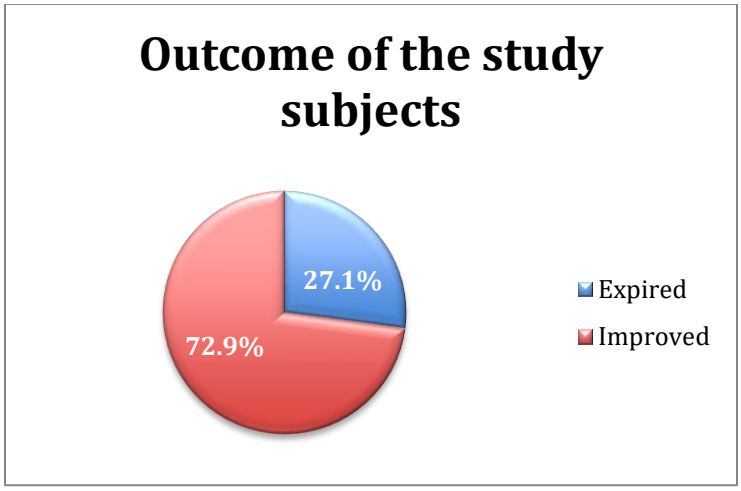


FIGURE 11: DISTRIBUTION OF PARTCIPANTS BASED ON OUTCOME

In the present study among the total of 240 study subjects, 72.9% (n=175) were improved and remaining 27.1% (n=65) were expired.

Association of baseline characteristics with outcome

J. Association of gender with outcome

Table 8: Association of gender with outcome

		Expired (%)	Improved (%)	P value
Gender	Female	17 (26.2)	45 (25.7)	0.94
	Male	48 (73.8)	130 (74.3)	

When comparing males to females, the most of the deceased were discovered to be male (73.8%). Nonetheless, the chi-square test revealed that the difference was not statistically significant (p=0.94).

K. Association between age and outcome

Table 9: Association between age and outcome

		Expired (%)	Improved (%)	P value
Age	<30 years	4 (6.2)	16 (9.1)	0.34
	31-40	11 (16.9)	22 (12.6)	
	41-50	5 (7.7)	11 (6.3)	
	51-60	8 (12.3)	40 (22.9)	
	>60 years	37 (56.9)	86 (49.1)	

The information mentioned in the above table clearly showed that, although the mortality rate was higher (56.9%) among those over 60 in the current study, it was not statistically significant (p=0.34).

L. Respiratory rate with outcome

Table 10: Respiratory rate with outcome

		Expired (%)	Improved (%)	P value
Respiratory rate	Normal	5 (7.7)	3 (1.7)	0.22
	Tachypnea	60 (92.3)	172 (98.3)	

It was noticed that the high respiratory rate of the sepsis patients had no significant effect on the outcome (p = 0.22).

M. Association between Total WBC count and outcome

Table 11: Association between Total WBC count and outcome

		Expired (%)	Improved (%)	P value
Total Count	High	57 (87.7)	166 (94.9)	**0.037***
	Low	2 (3.1)	4 (2.3)	
	Normal	6 (9.2)	5 (2.9)	

The total WBC count and the outcome were compared in the table 11. It was discovered that individuals with high total WBC counts had higher mortality rates (87.7%) than those with normal or low WBC counts. The difference was determined to be statistically significant with a p value of 0.037 using the Chi-square test.

N. Association of GCS with Outcome

Table 12: Association of GCS with Outcome

		Expired (%)	Improved (%)	P value
GCS	Mild	13 (20)	108 (61.7)	**0.002***
	Moderate	13 (20)	60 (34.3)	
	Severe	38 (58.5)	7 (4)	
	Vegetative state	1 (1.5)	0 (0)	

When the individuals' GCS scores and the outcome were compared, a statistically significant difference was found with a p value of 0.002 meant that the persons with severe GCS scores had a higher risk of mortality.

O. Association of foci of infection and outcome

Table 13: Association of foci of infection and outcome

		Expired (%)	Improved (%)	P value
Foci of Infection	Abdominal	9(13.8)	30(17.1)	0.477
	Blood	1 (1.5)	0 (0)	
	CNS	5 (7.7)	13 (7.4)	
	Genitourinary	10 (15.4)	22 (12.6)	
	Respiratory	22 (33.8)	62 (35.4)	
	Skin	8 (12.3)	31 (17.7)	
	Unknown	10 (15.4)	17 (9.7)	

In this study even though, respiratory system was observed as the primary focus of infection among all the sepsis patients, the difference founded was not statistically significant (p= 0.603).

P. Association of qSOFA score with outcome

Table 14: Association of qSOFA with outcome of study subjects

		Expired (%)	Improved (%)	P value
qSOFA	=2	23 (35.4)	84 (48)	**0.01***
	> 2	42 (64.6)	91 (52)	

It was observed that qSOFA score had statistically significant association with outcome (p=0.01).

Q. Association of blood type with qSOFA

Table 15: Association of blood type with qSOFA

			qSOFA		P value
			High	Low	
Blood group	A	Count	31	26	**<0.001***
		%	23.3%	24.2%	
	AB	Count	25	37	
		%	18.8%	34.6%	
	B	Count	26	35	
		%	19.5%	32.8%	
	O	Count	51	9	
		%	38.4%	8.4%	

It is evident from the above table that high qSOFA score is seen more (38.4%) among the O blood group patients and the difference observed is statistically significant with the p value of <0.001.

R. Association between blood type and outcome

Table 16: Association of Blood type with the outcome

		Expired (%)	Improved (%)	OR (95% CI)	P value
Blood group	O	29 (44.6)	31 (17.7)	3.15 (1.352, 7.348)	**0.001***
	A	15 (23.1)	45 (25.7)		
	B	13 (20)	47 (26.9)		
	AB	8 (12.3)	52 (29.7)		

When compared to other blood types, it was shown that the O group had a higher sepsis mortality rate (44.6%). The probabilities of the O group having **3.15** times higher mortality than other blood types were also determined to be significantly different, with a p value of 0.001 (AOR=3.152, 95% CI:1.352, 7.348)

DISCUSSION

This study's main goal was to determine how ABO blood type affected patients' risk stratification septic shock when they were hospitalized to the ICU at the ‘KLES Dr. Prabhakar Kore Hospital and Medical Research Centre in Belagavi’. The main findings of the study are reviewed, implications that can be made from the data are discussed, and recommendations are made for improving our general understanding of how ABO blood type and sepsis outcome are related. Discussion also takes into account how these outcomes compare with other studies.

The patients with sepsis who have been admitted to the intensive care unit provided the data for our study. For the purpose of gathering data, 240 patients in all were contacted; Results were computed among these study participants. Records were kept on age, gender, blood type, blood pressure, respiration rate, and total WBC count. The qSOFA score, GCS, and foci of infection were evaluated. Additionally, the outcome—whether they improved or expired—was reported. Association between blood type and sepsis mortality was evaluated.

The ABO antigens are thought to play a significant role as mediators of cell to cell interactions, such as the leukocyte & endothelial cell contacts that are crucial in the pathophysiology of sepsis, but their exact physiologic importance is not entirely understood.

Genetic variations in the ABO gene have been linked to blood levels of glycoproteins, such as thrombomodulin, vWF, selectins and ICAM-1 (soluble intercellular adhesion molecule-1), that are crucial for endothelial function and inflammation. These proteins

have been connected to the sepsis mortality.[6] However in our study we were not able to find out the mechanisms which potentially affect the prognosis of sepsis patients due to time and financial constraints.

Current study has got the following main findings, the mean age of the study subjects was found out to be 57.3±17.6 years which ranged from 18 years to 94 years. Around half (51.2%) were from the age group of above 60 years. Males were more (74.2%) in number when compared to females.

When compared to the other blood types, it was shown that the O group had a higher sepsis mortality rate (44.6%). The probabilities of the O group having **3.15** times higher mortality than other blood types were also determined to be significantly different, with a p value of 0.001 (AOR=3.152, 95% CI:1.352, 7.348).

When the individuals' GCS scores and the outcome were compared, a statistically significant difference was found with a p value of 0.002 meant that the persons with severe GCS scores had a higher risk of mortality.

It was also observed that individuals with high total WBC counts had higher mortality rates (87.7%) than those with normal or low WBC counts. The difference was determined to be statistically significant with a p value of 0.037 using the Chi-square test.

Patients with blood type B had a higher 30-day survival rate than patients with other blood types, according to a study by Theis S. Itenov et al [2]. The analysis comprised 12,342 patients in total. Their primary goal was to ascertain whether there are

differences in the risks of acute kidney injury (AKI), endothelial damage, and 30-day mortality between ABO blood types in patients who are critically unwell and sepsis. Three distinct cohorts of critically ill people with septic shock from the United States and Scandinavia were included in their retrospective cohort analysis. They came to the conclusion that there was a slight decrease in mortality and less endothelium damage in septic patients who had blood type B. They were unable to independently verify that blood type and acute renal injury are related. However, the current study indicated that the O blood group has a higher sepsis mortality rate than other groups, and no other concerns, such as endothelium damage or AKI, were examined.

ABO blood types and the risk of ARDS in patients with severe sepsis and serious trauma were the subjects of a study by John P. Reilly et al [5]. In two separate cohorts of critically ill patients, they showed that the ABO blood group is linked to an altered risk of ARDS among whites, regardless of blood transfusion and all other confounding variables. Blood type A individuals have a higher risk of ARDS, which is in line with the risk patterns seen in other inflammatory vascular disorders such as MI and VTE. Their research is the first to show a link between ABO blood types and the likelihood of developing ARDS, indicating a critical role for ABO glycobiology in the aetiology of the disease. There is inconsistent evidence of a link between the ABO blood group and ARDS in both white and black participants. This is especially true in trauma patients where there is statistical evidence of racial effect modification. This seemingly counterintuitive observation has multiple possible explanations. The results could be explained by the cohort's exposures and demography. Blacks experienced both blunt and penetrating mechanisms of trauma, while whites in the trauma group almost

exclusively experienced blunt mechanisms. Divergent gene-environment interactions may account for some of the observed discrepancies in outcomes across the ethnic groups, given the notable variations in injuries incurred. Neither the race nor the genetic mechanisms were taken into account in the present study.

According to research by John P. Reilly, Brain J. Anderseon, et al.,[6] in critically ill patients of major trauma and severe sepsis—those of European heritage with ABO blood group A had an estimated 14% higher chance of having AKI in the ICU than people with type O. Patients who did not get a blood transfusion nevertheless had the ABO and AKI risk linkages, demonstrating the relationship's independence from transfusion related factors such volume, storage time, and compatible vs. identical blood types. Furthermore, they found similar results in patients who did not develop ARDS, suggesting that ABO and AKI are related even in the absence of ARDS. The results are noteworthy for various reasons. Firstly, they suggest that ABO glycans could play a significant role as AKI mediators. Second, despite variations in the research population, comorbidities, and years of enrollment, there appears to be a similar ABO-AKI connection in patients with trauma and sepsis, suggesting that the underlying mechanisms may be similar in these two separate populations. In light of their and other researchers' earlier findings that blood type A is associated with an significant risk of vascular illnesses and acute respiratory distress syndrome (ARDS); their results also imply that ABO blood types may have similarities in the dysfunction of several organs during critical illness.

Rezoagli E et al. (7) conducted a retrospective observational analysis in which they observed no significant link between ICU mortality and ABO blood type A in comparison

to other ABO blood types among a large cohort of patients with AHRF undergoing mechanical ventilation. Moreover, in their statistical model, ABO blood type A did not predict ICU length of stay.

Patients with blood group AB had a better 90-day survival after being admitted to the intensive care unit (ICU), according to research by Robert Slade et al. [8] describing the 'impact of blood group on survival following critical illness'. Additionally, they have observed that there is no association between the percentage of patients having transfusions and blood group.

After conducting a thorough review of the topic, "Human ABO blood groups and their association with different diseases", Silamlak Birhanu Abegaz [12] looked up original, sufficient, and recent works in the same field. As a result, in addition to providing a descriptive synopsis of the subject, essential topics were extracted and presented in a way that made a clear impression. A few pertinent scientific publications from earlier years were also included. The terms blood types and groups were matched with a set of terms pertaining to various diseases in order to conduct the article search.

An increased risk of various diseases, such as cancer, hematologic disorders, cardiovascular disease, cognitive disorders, infections and, circulatory diseases, malaria and metabolic diseases, has been linked to ABO blood types. In addition, it was found that individuals with blood group AB were more likely to experience cognitive impairment at any age, race, gender, or region. Individuals with cognitive impairment

also had higher rates of obesity, diabetes, hypertension, dyslipidemia, and cardiovascular disease (CVD).

Previous studies have connected blood type O to increased risks of plague, cholera, mumps and tuberculosis infections. Conversely, blood types A, B, and AB have been linked to increased incidence of smallpox, salmonella, E. coli, tuberculosis, and Pneumococcus, as well as higher rates of gonorrhea and salmonella infections. Blood type AB has also been linked to increased incidence of E. coli, smallpox, and salmonella infections.

The most prevalent risk factors for cardiovascular diseases include hypercholesterolemia, arterial hypertension, diabetes mellitus, and the family history of ischemic heart disease. These risk factors can be passed on genetically to kids. Blood type A individuals had a higher risk of malignancies in their stomach, ovaries, salivary glands, cervix, uterus, colon, and rectum compared to blood type O individuals. The biologic activity of a carrier protein for coagulation factor VIII, that is vWF (Von Willebrand factor), is low in O type, and glycosyltransferase activity and plasma levels intervene in the relationship between the ABO blood type and thromboembolic illnesses and risk of bleeding.[13]

The relationship and distribution of obesity, hypertension, and ABO blood types in various blood donor categories were evaluated by Chandra T and Ashish Gupta. [14] When compared to healthy individuals, they discovered that there was a considerable

increase in both obesity and hypertension. The B blood group was shown to be more prevalent in cases of obesity and hypertension, followed by blood groups O, A, and AB. Researchers discovered that, in comparison to blood groups O and A, the B blood group was more prone to obesity and hypertension, whereas the AB blood type was less likely to experience these conditions. According to their research, the B group may be genetically predisposed to hypertension more than the other groups.

During an average follow-up of 8.75 years, the authors [25] discovered that the future risk of cancer differed across people with various blood groups in the large cohort of Chinese men and women with baseline laboratory typing of ABO blood group. Individuals with non-O blood types (A, B, and AB alone or in combination) had a lower risk of kidney cancer but a higher risk of pancreas cancer when compared to those with blood type O. Furthermore, blood type AB was linked to a little decrease in the overall risk of cancer, while blood group A was linked to an increased risk of stomach cancer.

Dentali F et al. [26] conducted one meta-analysis of the available literature to assess the potential clinical implications of the various 'ABO blood groups on the risk of venous thromboembolism (VTE)'. They included 38 papers with 10,305 VTE cases in the systematic review via an digital search technique utilizing Medline and Embase, a manual review of the 'International Society on Thrombosis and Haemostasis abstract books', and a study of the reference lists of all previous articles. They came to the conclusion that one of the significant genetic risk factors for VTE belongs to the non-O blood group.

Wataru Takayama et al. [27] determined the relationship between blood type O and death in 901 patients who had suffered severe trauma in a retrospective analysis. After controlling for potential confounders, the results showed that blood type O was the single risk factor for overall mortality in hospital admitted patients as well as death from exsanguination, traumatic brain injury, and other causes.

Patients with blood type O often had more units of RBC transfusions within 24 hours after being admitted to the emergency department; nevertheless, there was no statistically significant difference between blood type O and other non O blood types. To the best of their knowledge, this was the first study to reveal a link between patients' deaths following severe trauma and their ABO blood type.

They are unable to change the blood type O risk per se, but by properly identifying the risk, they were able to manage the level of trauma critical care. Since damage-control resuscitation necessitates a significant number of medical personnel and resources, including strategic multiple operations, adequate ratios of available transfusions, and perioperative critical care, it is imperative to identify the patients who require this type of care.

Consequently, realizing the increased risk associated with blood type variation would suggest the possibility of optimizing damage-control resuscitation techniques and so enhance the prognosis of severely traumatized patients.

In order to examine the cytokine profiles of male and female patients and ascertain the association between gender and sepsis mortality, a prospective case series [39] of 97 patients who had been admitted with the condition was conducted. Clinical and

microbiological information was obtained, blood samples were obtained to measure cytokine levels (IL-6, IL-10, and TNFα), and patients were monitored for any changes in their clinical status. In comparison to females, they observed that males had a higher plasma IL-6 level ($p=0.040$) and mortality ($p=0.048$, RR 1.73). Patients who died had a considerably ($p<0.01$) greater mean IL-6 plasma level than those who recovered. They came to the conclusion that death from sepsis is 70% higher in men, and that higher levels of IL-6 plasma are linked to mortality. In the current study, when association between the gender and sepsis mortality was found, more deceased were found out to be male. However, no statistical significance was found.

LIMITATIONS

1. As the study was conducted in a single tertiary care setting, the results cannot be generalized.
2. qSOFA score was evaluated only on the day of admission.
3. Association of comorbidities with that of sepsis mortality was not evaluated.

SUMMARY

This hospital based Observational cross-sectional study conducted at 'KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi', on 240 study participants who were admitted in the ICU with sepsis. Data collection was done for a period of one year.

The study revealed the following key-findings:

- The mean age of the study participants was 57.3±17.6 years which ranged from 18 years to 94 years.
- Majority (51.2%, n= 123) were from the age group of more than 60 years.
- 74.2% (n=178) of participants were Males, and 25.8% (n=62) were Females.
- 60 members from each type of blood group were selected.
- Among the participants 96.7% (n=232) were found to have tachypnea and the remaining 3.3% (n=8) were of normal respiratory rate.
- In this study, 92.9% were with high total WBC count; whereas 4.6% have normal count and remaining 2.5% were with low WBC count.
- 50.4% of the study subjects had severe GCS score.
- The respiratory system was found out to be the primary site of infection for the majority of sepsis patients (35%).
- It was noticed that the majority of study subjects (55.4%) had their qSOFA score >2.
- In the present study among the total of 240 study subjects, 72.9% (n=175) were improved and remaining 27.1% (n=65) were expired.

- When the study individuals' baseline characteristics and the outcome were compared, it was discovered that individuals with high total WBC counts had higher mortality rates than those with normal or low WBC counts. The difference was determined to be statistically significant with a p value of 0.037 using the Chi-square test.
- With a p value of 0.002, GCS was also shown to be significantly associated with mortality.
- It was observed that qSOFA score had statistically significant association with outcome (p=0.01)
- It is evident from the analysis that high qSOFA score is seen more (38.4%) among the O blood group patients and the difference observed is statistically significant with the p value of <0.001.
- When compared to other blood types, it was shown that the O group had a higher sepsis mortality rate. The probabilities of the O group having **3.15** times higher mortality than other blood types were also determined to be significantly different, with a p value of 0.001 (AOR=3.152, 95% CI:1.352, 7.348).

CONCLUSION

- To conclude from the present study, sepsis patients with O blood group, high WBC count, severe GCS and high qSOFA have high mortality rate. However ABO blood grouping is a non-modifiable risk factor, it can help identify people who are more likely to have a bad prognosis. Here in this study, as we found that O type has higher morality, they should be taken more care of.

- It is necessary to conduct further research to pinpoint the precise mechanisms behind this association.

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ANNEXURE-I

INFORMED CONSENT FORM

"Correlation of ABO blood grouping and outcome in sepsis patients admitted in Intensive Care Unit at KLE Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi -A Cross Sectional Based Study"

Name of Student/Principal Investigator: REG NO. BG0121011

Name of Guide/Co Investigators

Objective:

- To identify the role of ABO blood type in risk stratification for septic shock and to evaluate the effect of ABO blood group on septic shock.

Introduction:

ABO blood types arise from variations in an oligosaccharide molecule on the H-antigen which is present on both the surface of erythrocytes and endothelial cells. Variations in oligosaccharide composition are controlled by the gene coding a glycosyl-transferring enzyme [1, 2]. Endothelial damage, including disruption of the glycocalyx, increases the risk of organ failure and death in critically ill patients, and it has been proposed to play a pivotal role in the pathophysiology of critical illness associated organ dysfunction [3–5]. Sepsis is frequently complicated by organ dysfunction, including acute kidney injury (AKI) and acute respiratory distress syndrome (ARDS) [6, 7], both of which increase the risk of dying. ABO blood type A seems to increase the risk of sustaining AKI and ARDS in patients with either sepsis or trauma.

The ABO blood types are determined by glycans that are displayed on the surfaces of erythrocytes and other cells [8]. The ABO blood typing is widely used in clinical practice, and associations between blood type and disease have been studied since the early 1900s [9,10]. Recent studies suggest that blood type O is a potential risk factor for cancer, myocardial infarction, acute kidney injury, venous thromboembolism, trauma, and gastrointestinal bleeding.

While ABO blood grouping study is non-modifiable risk factor, the study helps in identification of patients who are more prone to poor prognosis. It helps in understanding why two patients of same presentation and sepsis scoring and receiving same treatment are having two very different prognosis

Explanation of procedure:

- Informed consent will be obtained and then patient will be enrolled for the study.
- All patients fulfilling inclusion criteria are subjected to a questionnaire and thorough clinical examination.
- Following tests were done on the patients to get more required information.
- COMPLETE BLOOD COUNT (Hb, RBC, PCV, WBC, N/L/M/B/E, platelet count, MCV, MCH, MCHC). BLOOD GROUPING , LIVER FUNCTION TESTS, RENAL FUNCTION TESTS,PT/INR , ABG ,PCT ,BLOOD CULTURE & SENSITIVITY , URINE ROUTINE & MICROSCOPY ,URINE CULTURE & SENSITIVITY.
- Data will be analysed and tabulated.

Withdrawal from participation in the study: Participation in this study is voluntary. You will be free to decide whether to participate in this study or continue participation

once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: You will/will not have nor get any benefits by participating in this study. The data gathered will help the population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person from identifying you. Your identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study.

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purposes and or presented to scientific groups. However, your identity will never be revealed.

Questions: In case of any questions with regard to this study, you are free to contact: “Reg No. BG0121011, , If you have any question or complaints with regard to your right as study participant you may contact Dr Harsha Hegde, Chairperson, Ethical committee of JNMC, 0831-2473777 Extension 4052.

Legal rights: By signing this consent form, we are not waving any of your legal rights.

CONSENT STATEMENT

I am making a voluntary decision to participate in the study "**Correlation of ABO blood grouping and outcome in sepsis patients admitted in Intensive Care Unit at KLE Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi -A Cross Sectional Based Study**". My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator: REG NO. BG0121011

Signature of the investigator:

ANNEXURE-II

PROFORMA

CASE NO	
NAME	
IP NO	
AGE	**YEARS**
SEX	**MALE FEMALE**
ADDRESS	
OCCUPATION	

Complaints at presentation	
Past history	
Family history	
Personal history	
Treatment history	

Vitals :

Temperature	
Pulse	
Respiratory rate	
Blood pressure	

PHYSICAL EXAMINATION:

	Yes	No
Pallor		
Icterus		
Lymphadenopathy		
Cyanosis		
Clubbing		
Edema		

SYSTEMIC EXAMINATION:

C.V.S	
R.S.	
C.N.S	
PER ABDOMEN	

INVESTIGATIONS:

Hemoglobin		**ALP**		**Na^+**	
Total Count		**Total Bilirubin**		**K^+**	
Neutrophils		**Direct Bilirubin**		**Mg^{2+}**	
Lymphocytes		**Total Protein**		**Sr. Creatinine**	
Eosinophils		**Albumin**		**Blood culture**	
Monocytes		**A/G ratio**		**Urine RM.CS.**	
Basophils		**SGOT**		**HbA1c**	
ESR		**SGPT**			
RBS		**Sr. Procalcitonin**			

ANNEXURE-II

MASTER SHEET