# "CORRELATION OF SERUM HDL WITH SOFA SCORE IN SEPSIS AND ITS PROGNOSTIC VALUE IN PATIENTS ADMITTED IN INTENSIVE CARE UNIT AT KLE DR. PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELAGAVI – A PROSPECTIVE COHORT STUDY"


**BY**

**REG.NO: BG0121005**


# Dissertation

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# ABBREVIATIONS

| | |
|---|---|
| HDL-High density lipoprotein | CETP-Cholesteryl ester transfer protein |
| SOFA-Sequential organ function assessment | LDL-Low density lipoprotein |
| ICU-Intensice care unit | VLDL-Very low density lipoprotein |
| CBC- Complete blood count | TC-Total cholesterol |
| MR-Mini renal profile | PCT-Procalcitonin |
| LFT-Liver function test | IL-6-Interleukin 6 |
| USG-Ultrasonography | DIC-Disseminated intravascular coagulation |
| GPI-Glycophosphatidylinositol | UTI-Urinary tract infection |
| SIRS-Systemmic inflammatory syndrome | BP-Blood pressure |
| qSOFA-quick SOFA | RR-Respiratory rate |
| AUROC-Area under curve | HR-Heardt rate |
| GCS-Glassgow coma score | IVC-Inferior vena cava |
| NEWS-National early warning score | CVP-Central venous pressure |
| ACETYL CoA- Acetyl coenzyme A | ScvO2-central venous oxygen saturation |
| ATP-Adenosine triphosphate | LMIC-Low and middle income countries |
| NADPH- nicotinamide adenine dinucleotide phosphate hydrogen | PPV-Pulse pressure variation |
| GPCR-G protein coupled receptor | MAP-Mean arterial pressure |

# ABSTRACT

**Introduction:**

The term "sepsis" refers to potentially fatal organ failure brought on by an uncontrolled host reaction to an infection. A suspected or confirmed infection as well as an abrupt increase in two or more Sequential Organ Failure Assessment (SOFA) points—a proxy for organ dysfunction—are among the clinical criteria for sepsis. This high mortality rate persists despite breakthroughs in aggressive care.

**Methodology:**

This study examines the correlation of serum HDL with SOFA score in sepsis and its prognostic value in patients admitted in Intensive Care Unit greater than 18 years at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre in Belagavi between August 2022 and July 2023. The study subjects were enrolled in study by convenient sampling method who fulfilled inclusion & exclusion criteria after obtaining the informed consent. All subjects underwent detailed and thorough clinical history & clinical examination. Blood samples from each patient will be taken for HDL-C cholesterol levels, creatinine, bilirubin levels, platelet levels and routine investigations

CBC,MR,LFT, urine routine, USG Whole abdomen, Chest radiograph at the time of admission and on the day 6 after admission. Respiratory status by Pao2 and Fi02 and Central nervous system assessed by Glasgow Coma Scale and SOFA scores were calculated accordingly on day of admission and day 6.

**Results:** The age distribution of the study population shows that the majority of participants fall within the age range of 51 to 60 years, with male preponderance. The most common site of infection among the participants was noted to be respiratory system, with gram negative infection being the most common. The Glasgow Coma Scale scores showed that participants were fit into moderate to severe impairment, with final survivance of 82% of the participants. HDL levels show a significant association with the outcome. Among survivors, 29% had HDL levels below 40 mg/dL, while 71% had levels above 40 mg/dL. Conversely, among non-survivors, 87% had HDL levels below 40 mg/dL, and only 13% had levels above 40 mg/dL. The P value of 0.001 indicates a strong statistical significance in the difference in HDL levels between survivors and non-survivors. •HDL: On Day 1, survivors had a mean HDL of 35.64±3.84 mg/dL, and non-survivors had 40.14±3.76 mg/dL (P = 0.541). By Day 6, survivors' HDL was 42.39±10.98 mg/dL, and non-survivors' HDL was 31.32±5.23 mg/dL (P = 0.001). •

The analysis reveals a consistent, significant negative correlation between HDL levels and SOFA scores at various time points, indicating that lower HDL levels are associated with higher SOFA scores and worse organ function. On admission, the correlation is -0.59 (p = 0.01), strengthening to -0.66 (p = 0.001) by day 6. Among survivors, the correlation is -0.69 (p = 0.02) on day 0 and -0.55 (p = 0.03) on day 6. For non-survivors, the correlation is -0.39 (p = 0.01) on day 0 and -0.47 (p = 0.01) on day 6.

**Conclusion:** The study highlights important connections between demographic characteristics, health markers, infection details, and results within the study group. There is strong evidence linking sepsis with serum HDL levels in patients. Those with sepsis and significant organ dysfunction typically exhibit lower HDL levels.

Therefore, serial monitoring of HDL levels alongside Sequential Organ Failure Assessment (SOFA) scores is recommended High initial or increasing HDL levels may indicate a better prognosis and higher survival rates in sepsis patients. Conversely, very low HDL levels or a declining trend are associated with poorer outcomes and survival. HDL can serve as a valuable prognostic marker in sepsis patients and may complement other biomarkers such as Procalcitonin (PCT), Interleukin-6 (IL-6) and other biomarkers for enhanced diagnostic and prognostic accuracy.

**Key words :** HDL-cholesterol, Sepsis, SOFA score, Infection

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# INTRODUCTION

Sepsis poses a major threat to public health. Anyone may become sick from it, and it's the main reason people end up in the critical care unit (ICU) in the first place. those with sepsis have a death rate of 20.8%, whereas those with septic shock have a rate of 48.8%. This high mortality rate persists despite breakthroughs in aggressive care.1

The current standard of care for sepsis management includes the use of empiric antibiotics, intensive volume resuscitation, inotrope support, and strict insulin control, among other dependable therapeutic approaches. New research indicates that serum Since bacteremia causes systemic inflammation, which affects cholesterol metabolism, HDL-C high-density lipoprotein cholesterol might a good predictive indicator for sepsis.

Lipoproteins and circulating lipids may play a less-explored role in sepsis. Our research, alongside others', has indicated that sepsis disrupts the usual regulation of HDL-C and LDL-C cholesterol levels. Theoretically, HDL can combat sepsis through several mechanisms: facilitating the reverse cholesterol transport of bacterial toxins like lipopolysaccharide and lipoteichoic acid, regulating innate cellular immunity and inhibiting the release of inflammatory cytokines, and delivering cholesterol to the adrenal glands for steroid synthesis in response to inflammation. Similarly, LDL is believed to protect against sepsis by serving as a substrate for steroid production and aiding in the removal of bacterial toxins.

However, lipoprotein particles can undergo oxidation in response to acute or chronic inflammation. Dysfunctional HDL, for instance, results from the oxidation of HDL-C particles, causing them to become dysfunctional and pro-inflammatory. Consequently, instead of protecting against infection, dysfunctional HDL may exacerbate sepsis-related inflammation and tissue damage.

Research shows that regardless of other health conditions, individuals with severe sepsis who end up in the intensive care unit had lower levels of lipoproteins in their blood and higher levels of triglycerides. These alterations are known to happen quickly (within hours) during the inflammatory cascade that is linked to sepsis. There was an inverse relationship between clinical outcome and serum cholesterol lowering as well.

# AIM AND OBJECTIVES

## AIM

The aim of the present study was to evaluate the correlation of Serum HDL with SOFA score in sepsis and its prognostic value in Patients Admitted in ICU

## OBJECTIVES

Primary -To determine correlation of Serum HDL with SOFA score in sepsis

**Secondary-**

- To assess the level of HDL in survivors vs non survivors sepsis patients
- To determine whether HDL can be used as a biomarker in sepsis patients

# REVIEW OF LITERATURE

## Definitions

Tables 1 and 2 reveals components of the systemic inflammatory response syndrome (SIRS), which was officially recognized as "sepsis" in 1992. New terms have evolved since then. "Severe sepsis" describes a case of sepsis that has progressed to the point that organ failure is a factor, while "septic shock" describes a case of sepsis when hypotension is a factor and the patient does not respond to appropriate volume resuscitation without other explanation. The severity of the sepsis range of illnesses is clinically significant. Although there has been considerable research on sepsis in developing nations, such as India, the disease's epidemiology is still poorly understood.10, 11

A coalition of organizations including the European Society of Intensive Care Medicine (ESICM), the International Sepsis Forum (ISF), and the Society of Critical Care Medicine (SSCM) initiated the Surviving Sepsis Campaign (SSC) in 2002 to reduce mortality rates associated with sepsis. A widely accepted set of guidelines for treating severe sepsis and septic shock is the "Surviving Sepsis Campaign guidelines for the management of severe sepsis and septic shock," developed by the SSC in 2004 and most recently updated in 2012. These guidelines represent the standard of care in many countries, presenting a significant opportunity to enhance patient outcomes, particularly since half of all hospital admissions occur through emergency departments.

**Tables 1 and 2 for definitions.**

**Table 1** SIRS criteria.[1]

| Presence of two or more of the following. | |
|---|---|
| 1. Temperature | • > 38 °C (100.4 °F) or<br>• < 36 °C (96.8 °F) |
| 2. Heart rate | • > 90/min |
| 3. Respiratory rate | • > 20/min or<br>• $PaCO_2$ < 32 mmHg |
| 4. White blood cell count | • > 12,000/μL or<br>• < 4000/μL |

**Table 2** Diagnostic criteria for sepsis.[1] WBC, white blood cell; SBP, systolic blood pressure; MAP, mean arterial pressure. Table adapted from Levy et al. (2003).[1]

| Infection (documented or suspected) and some of the following. | |
|---|---|
| Classification | Variables |
| General | Fever ( > 38.3 °C)<br>Hypothermia (core temperature < 36 °C)<br>Heart rate > 90/min or more than two SD above the normal value for age<br>Tachypnea<br>Altered mental status<br>Significant oedema or positive fluid balance<br>Hyperglycaemia |
| Inflammatory | Leukocytosis<br>Leukopenia<br>Normal WBC count with greater than 10% bands<br>Plasma C-reactive protein > 2 SD above the normal value<br>Plasma procalcitonin > 2 SD above the normal value |
| Hemodynamic | Hypotension (SBP < 90 mmHg, MAP < 70 mmHg, or an SBP decrease > 40 mmHg in adults or < 2 SD below normal for age) |
| Organ dysfunction | Creatinine increase<br>Coagulopathy<br>Hypoxaemia<br>Ileus<br>Oliguria<br>Thrombocytopenia<br>Hyperbilirubinemia |
| Tissue perfusion | Hyperlactatemia<br>Decreased capillary refill or mottling |

**Third international consensus on sepsis and septic shock (Sepsis-3)-**

Sepsis should be suspected in patients with infections originating from any infectious source, under the third international consensus on sepsis and septic shock (Sepsis-3) . Patients with a score of ≥ 2 on the QUICK Sequential Organ Failure Assessment (qSOFA) are more likely to die in hospitals, hence it is important to take this into consideration while evaluating these patients. Because the National Early Warning Score (NEWS) and the Systemic Inflammation Response Syndrome (SIRS) score have a higher sensitivity than qSOFA in predicting patient outcome, the 2021 guidelines oppose using qSOFA as the only screening technique and instead suggest using both . When a person's Sequential Organ Failure Assessment (SOFA) score is more than or equal to 2, sepsis is officially diagnosed. The requirement for a vasopressor to keep a patient's mean arterial pressure (MAP) at or above 65 mmHg and serum lactate level at or above 2 mmol/L characterizes **septic shock** .

**Epidemiology**

Among patients undergoing medical or surgical procedures, sepsis ranks high in terms of both mortality and morbidity rates experienced while hospitalized. One in five patients admitted to the intensive care unit (ICU) suffers from severe sepsis, which is also the main killer in the noncoronary ICU. [15] Despite improvements in intensive supportive care and the availability of a growing number of higher-generation antibiotics with a wider spectrum of coverage, the unacceptably high outcome rate of 30% to 40% associated with sepsis persists. [16] While sepsis is acknowledged as a major health concern worldwide, the majority of epidemiological data on the disease's prevalence and fatality rates comes from Western nations. These studies estimate an overall incidence of 10% to 30% and a mortality rate of 10% to

56%. (pages 16–17) According to the data that is currently available from India, the overall fatality rate for patients with septic shock is around 14%, and for severe sepsis in particular, it is above 50%. [18]

**Pathophysiology**

**Dysregulated Coagulation**

Blood normally maintains a fluid state to ensure unrestricted flow within veins but has the ability to clot appropriately to control bleeding. This delicate equilibrium is referred to as normal hemostasis. The clotting cascade is highly complex and functions in a meticulously orchestrated manner under normal conditions.8The coagulation system and the cells that control it undergo multi-level changes during inflammatory conditions like sepsis [19] (Figure 1). Platelet consumption and prolonged clotting times are symptoms of disseminated intravascular coagulation (DIC), which is common in septic patients. The abnormal hemostasis also causes blood to clot inappropriately, which narrows blood arteries and decreases blood flow. Local influences regulate systemic coagulopathy because procoagulant factor production by the liver and white blood cell release by the bone marrow are constants. Basically, even though coagulopathy is global, bleeding usually only happens at specific locations where defective vasculature creates the ideal conditions for bleeding to happen. Interactions among the endothelium, circulating platelets and white blood cells, and the clotting system further complicate an already complex picture. Several of these anomalies have been observed in individuals with septic shock, but there is likely more than one component at play when it comes to coagulopathy. Virchow documented the thrombotic potential of cancer patients and abnormalities in the coagulation system caused by systemic disorders that induce local disruptions in

hemostasis. Alterations to coagulability, damage to endothelial cells, and aberrant blood flow make up Virchow's classic trio. Reduced blood supply to critical organs is the result of all three of these basic changes occurring in septic patients. Inappropriate oxygen consumption leading to cytopathic hypoxia and impaired tissue perfusion are common symptoms in septic patients.[20]

**Aberrant Mediator Production**

The inflammatory response plays a crucial and central role in sepsis because its components drive the physiological changes that lead to the systemic inflammatory response syndrome. A well-regulated inflammatory response is essential as it aims to eradicate invading microorganisms while minimizing damage to tissues, organs, and other bodily systems.

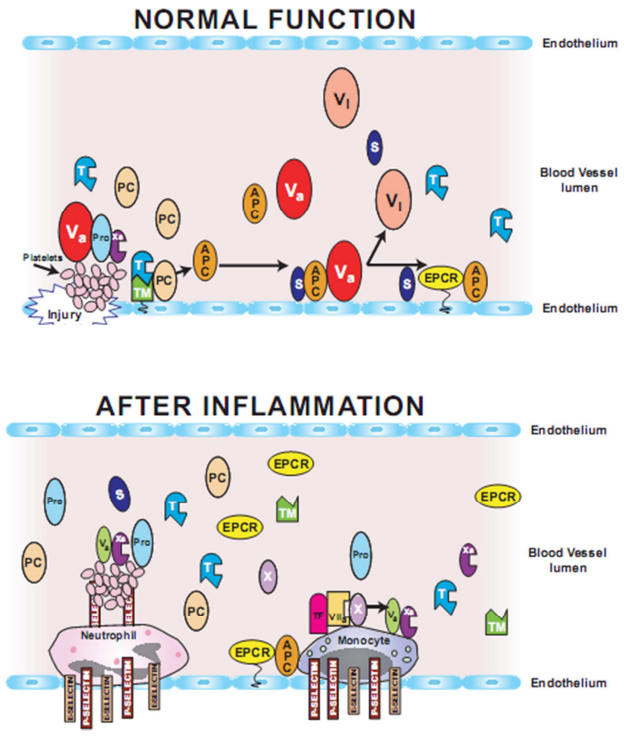



**Figure 1. Control of coagulation in normal and inflamed vasculature**

**Assessment Clinical**

When taking a patient's history, it is important to look for signs of infection (like immunosuppression), the existence of infection itself, and, if possible, the origins of the infection. Patients over the age of 65 should be approached with caution since they might not be able to articulate the more common symptoms, such as dysuria in cases with hidden UTIs. Finding potential sources of control should be the goal of the physical examination. At this stage, it is crucial to monitor, record, and assess vital signs such as temperature, Blood pressure, heart rate, respiratory rate, and oxygen saturation (urgent supplementary oxygen administered if it drops below 90%). To monitor whether the patient's condition was improving or worsening and to initiate targeted treatments, these measures were recorded repeatedly (see below).In order to help in the early detection of critical disease, it is important to consistently analyse the vital signs of every potential patient with sepsis for the presence of SIRS criteria. Noteworthy, early on and in older individuals, abnormalities in vital signs could not be seen.References [21,22] Some of the specific physical exam findings that can help determine where an infection is coming from are rales, stomach discomfort, signs of a central nervous system infection, and indwelling devices like intravascular or urinary catheters.References [23,24] It is also important to check for oedema, peripheral pulses, capillary refill, skin colour and turgor, mucosal membranes, and cardiovascular and volume status at this point. It is critical to have early SIRS detection and a written record in place as there are currently no automated instruments to monitor vital signs. Clinical state can be indicated by changes in (BP), (HR), (RR), and oxygen saturation (OS) in response to treatments; this is because more sophisticated and intrusive equipment used to measure therapy response may not be available in LMICs.

**Diagnostic studies**

Finding the infection's origin and signs of organ failure are the primary goals of the initial round of laboratory and radiographic tests. Typical laboratory tests ordered by a treating physician may require full blood counts (including white blood cell count, haemoglobin and hemocrit levels, platelets, and electrolytes), chemistry (including bicarbonate, creatinine, glucose, and transaminases from the liver), Bilirubin, Arterial or Venous blood gas measurment with serum lactate level. Patients over the age of 65 have an especially high yield with urinalysis. [25] The When a patient is experiencing respiratory distress or pulmonary infection, chest radiographs can help pinpoint the site of the illness. Blood cultures should be obtained from all patients suspected of having sepsis. This will help doctors limit down antibiotic options and find out why some treatments didn't work.8It is advised to collect 20 mL of blood because the volume significantly influences the yield of culture compared to the number of blood cultures taken.[27] Standard clinical practice involves culturing various fluids such as blood, urine, sputum, wounds, catheters, and cerebrospinal fluid to identify potential sources of infection. When resources are limited, priority should be given to tests that can pinpoint the origin of the illness, guiding appropriate treatment. Whenever possible, blood cultures should be obtained regularly.

**Treatment and management**

Overarching method Recognising a septic patient quickly is essential, and determining where the patient falls on the sepsis spectrum (severe sepsis, septic shock, etc.) allows for the early establishment of therapeutic objectives. The recommendations of the Surviving Sepsis Campaign are centred on severe sepsis/septic shock (SS/SS), which is different from sepsis. A procedure-driven

strategy to therapy utilising quantitative resuscitation advised for individuals with SS/SS.[26] in Management is guided by defined, predefined physiological or laboratory resuscitation targets.[28] in The early goal directed treatment (EGDT) trial by Rivers in the year 2001 used a objective-directed strategy to treatment for SS/SS patients and showed a 16% total decrease in hospitalized mortality (number required to give treatment = 6.25), which triggered its worldwide attention.[29]A meta-analysis conducted in 2008 found that early and quantitative resuscitation improved survival rates in diverse sepsis groups, lending more credence to its usage.[30] in Notably, protocol-based resuscitation techniques, such as EGDT, did not reduce fatality rates in the ProCESS trial, a latest randomised multicenter study involving more than 1300 patients compared to "usual" (provider-driven) treatment.[31] It should be emphasised that participants in the "usual" care group were heavily resuscitated (4.4 L of injectable fluids, 58% CVC, 44% vasopressors, 22% invasive ventilation) and that protocol-based resuscitation could still be recommended in the absence of this intensive "usual" care. The death rate is significant for patients who experience hypotension despite fluid resuscitation or who show signs of end organ failure (SS/SS). Aggressive resuscitation, protocol adherence, and goal-oriented therapy have the potential to lower this mortality rate.

**Initial resuscitation**

As part of the first assessment of patients with sepsis, many lines of intravenous (IV) access should be quickly established in case fluid resuscitation, antibiotics, and maybe vasopressors are required.It is suggested to immediately provide 30 mL/kg (usually 2 L) of crystalloid to individuals who are commonly hypovolaemic and have substantial fluid deficits.[32] Volume resuscitation enhances

cardiac output and systemic oxygen supply, which helps restore tissue perfusion.[32] Patients are considered to be in SS/SS if they experience hypotension (MAP <65 mmHg) following a fluid bolus, increased lactatemia (>4 mmol/L), symptoms of low perfusion, or organ collapse. Consensus recommendations with quantifiable outcomes at the 3 and 6 hour periods propose transitioning to a goal-directed approach to therapy in such cases.[26] in Keep in mind that a comparable course of treatment may be beneficial for patients who fit the criteria for sepsis but do not have SS/SS. On the other hand, SS/SS patients are the only ones for whom consensus recommendations promoting bundled care exist.[26] Many patients with sepsis are hypovolaemic, hence it is important to give them at least 30 mL/kg (usually 2 Litres) of crystalloid as soon as you see symptoms.

**Measures of intravascular volume**

Fluid resuscitation, and the aim of reestablishing intravascular volume (and, presumably, preload), is the earliest step in the management of patients with SS/SS.[26] in Several techniques for estimating volume status are gaining traction, including PPV and sonography evaluation of the respirophasic change of IVC.[33] More research is needed to determine whether these monitoring strategies are effective in improving clinical outcomes.8Even though a well-known systematic research showed a very weak correlation between CVP and fluid responsiveness, consensus recommendations nonetheless suggest using CVP measurement as a substitute for preload. 34 Therefore, a goal CVP of 8-12 cmH2O is the first resuscitation objective in SS/SS as per criteria of the Surviving Sepsis Campaign. Hypovolaemia, which might necessitate further fluid resuscitation, may be indicated by values far lower than this. Note that a central venous catheter must be inserted

higher than the diaphragm in order to use CVP.It is important to aggressively fluid load patients in settings with limited resources since they may need more than 4 to 6 litres of crystalloid in the first six hours of treatment.[29] Death rates have been seen to drop with only this intervention.It is recommended that intensive fluid resuscitation be the standard, as demonstrated in the ProCESS experiment (4.4 L in the first 6 hours on average). [31] Check for JVP distension and look for creptitations as signs of fluid excess in the lack of improved parameters to measure fluid responsiveness..[36]

**Ongoing fluid administration**

Patients in the usual care arm of the ProCESS study, from hours 6 to 72 (after first resuscitation), obtained a mean of 4.3 ± 3.9 L of injectable fluid, albeit this varies greatly from case to case.[31] To determine the pace of fluid delivery beyond the initial resuscitation phase in settings with limited resources, utilize hypotension, lactate clearance, and urine output as indices of hypoperfusion (see below). This might be more than 8 litres during the next 72 hours, however it varies.

**Fluid selection**

Several studies have demonstrated that crystalloid solutions are more effective than hydroxyethyl starches (HES) in clinical practice. Additionally, albumin should be used selectively, particularly after administering large volumes of crystalloid. Crystalloid is strongly recommended because it is cost-effective, has shown no difference or lower mortality rates compared to HES, and decreases the need for renal replacement therapy (RRT).[37,38]Also, new evidence suggests that balanced crystalloid solutions (like Ringer's lactate) may be better than chloride-rich fluids (like normal saline) in resuscitation situations where resources are limited. However, in

these cases, the most important thing is to make sure that there is enough crystalloid volume resuscitation, regardless of the type. points [39,40] The amount administered is more critical than the kind of crystalloid infusion.

**Vasopressors**

Getting the MAP up to 65 mmHg is the second resuscitation objective in SS/SS. Vasodilation and vasoconstriction are out of whack in SS/SS. Therefore, in septic patients having low blood pressure (MAP <65 mmHg), vasoactive medicines are frequently necessary. Their goal is to improve blood flow to critical organs by lowering arterial pressures. Loss of autoregulation in the coronary, renal, and central nervous systems occurs at MAPs below 60 mmHg, and the connection between pressure and organ blood flow is linear. [41] The At MAPs as low as 65 mmHg, tissue perfusion can still be maintained, nevertheless. [42] Therefore, aiming for a MAP 65 mmHg is a well acknowledged clinical end objective for the majority of patients on vasopressors.[26] in Another crucial consideration is that younger individuals may be able to tolerate lower MAPs, whereas patients with chronic hypertension may require higher MAPs.[26] in Vasopressors should not be administered until fluid resuscitation (CVP P8cm H2O) has occurred; nevertheless, patients in shock may need them started sooner. A variety of vasopressor agents are available for selection. Dopamine and norepinephrine were utilized in the Rivers investigation. Norepinephrine and dopamine were both shown to be equally effective in correcting hypotension in a recent multicenter study [29]. However, the dopamine group did have more arrhythmias.

There was no significant difference in mortality.[43]

Norepinephrine isbetter than other options in the case of sepsis, according to the authors.[43]

Vasopressin, at modest dosages of 0.03 U/min, may improve hemodynamic and renal function in individuals whose hypotension persists after fluid resuscitation and norepinephrine treatment.[44] In place of norepinephrine, other options include epinephrine and phenylephrine.[26] in Although epinephrine and vasopressin are potential additions, the most recent consensus recommendations suggest norepinephrine as the first-line vasopressor. Unless absolutely necessary, such as in cases of relative bradycardia, dopamine should not be administered.[26] in Once patients are thought to have been sufficiently fluid resuscitated, vasopressors should be started in situations with restricted resources. The order of preference is norepinephrine, epinephrine, and vasopressin, in that order, when available. When all other options have been exhausted, phenylephrine and dopamine can be administered.

**Blood products and inotropes**

Thirdly, a SCVO2 of 70% or above is required for SS/SS therapy since it indicates an adequate balance between tissue oxygen demand and supply. Septic patients with sufficient blood pressure and appropriate fluid resuscitation might have low central venous oxygen saturations (SCV-O2 < 70%) for many reasons. Possible causes include metabolically active tissue, insufficient oxygen carrying capacity in the blood, reduced cardiac output, or arterial hypoxemia. By transferring packed red blood cells, the level of oxygen delivery can be enhanced.[29]A Red blood cell transfusions should aim to achieve a hemoglobin concentration target of 7-9 g/dL.

[26] in This is in contrast to the Rivers research, which had a hematocrit threshold of 30%.[29] The ideal haemoglobin concentration in sepsis is unknown, although the Transfusion Requirements in Critical Care study found no correlation between a Hb count of 7-9 g/dL and an increased risk of death compared to a haemoglobin level of 10-12 g/dL. in the 45th Unless there is ongoing bleeding or a scheduled invasive operation, it is recommended to avoid routinely administering fresh frozen plasma to address a coagulopathy. Platelet transfusion is not necessary when the count is below 5,000/$mm^3$, but it may be considered between 5,000 and 30,000/$mm^3$ if there is a significant risk of bleeding. Transfusion should be reserved for platelet counts of 50,000/$mm^3$ or higher only during scheduled surgeries or invasive procedures. 8 Inotropic drugs can be used to increase cardiac output if, even with a normal haemoglobin level (above), ScvO2 is below 70%. The preferred agent is dobutamine, with dosages ranging from 2.5 micrograms/kg/min to a maximum of 20 microgram/kg/min.[29]

No beneficial effects have been shown at doses higher than this. As mentioned before, transfusions of blood products should be administered with caution in contexts where resources are few. After administering fluids liberally, starting vasopressors, and transfusing blood products (if necessary), if signs of shock continue, inotropic treatment can be started to increase cardiac output. Since a central venous catheter (CVC) is necessary to detect SCVO2, alternative shock indicators, such lactate and urine output (discussed further below), can be utilised in its place. Lactate

A significant mortality rate is related with higher lactate levels (P4 mmol/L), which is an essential indication of severe sepsis.[46] In a recent randomized study

conducted by Jones across many centres, the quantitative resuscitation endpoint of lactate clearance (P10%) was shown to be noninferior to SCVO2 (P70%).[47] It is suggested that a noninvasive method of quantitative resuscitation may be initiated by detecting peripheral lactate levels rather than SCVO2 (which necessitates a central venous catheter). Nevertheless, the present consensus recommendations only aim to normalize lactate and do not address this potential.[26] in It is important to note that SCVO2 and lactate can be utilized together when both are available, rather than either one or the other. When central vein filtration (CVCs) are not available, treatment might be guided by monitoring serial lactate levels for clearance.

**Urine output**

In the initial six hours of resuscitation, the objective is to sustain a urine output of at least 0.5 mL/kg/h. This goal remains consistent regardless of whether treatment is guided by central venous oxygen saturation (ScvO2) or lactate levels. Lower urine output values indicates the requirement for further aggressive resuscitation efforts, which complements other assessments of tissue perfusion [26]. In resource-limited settings, urine output should be regularly monitored for every patient. Options include using a urine collection container or an indwelling urinary catheter. Similar to vital signs, it is crucial to document the timing and volume of each voiding episode.

**Antimicrobials**

Research has clearly demonstrated a direct correlation between delayed administration of antibiotics in severe sepsis or septic shock (SS/SS) and increased mortality rates. Specifically, there is an observed 8% rise in mortality for each hour that antibiotics are delayed, even prior to the onset of persistent or recurrent

hypotension. This underscores the critical importance of timely initiation of antimicrobial therapy in improving outcomes for patients with SS/SS [48].Patients in another EGDT group who were given the right antibiotics during the first hour of triage had a 14% reduced death rate compared to those who were not.[49] oth the timing and the choice of antibiotics are crucial in the management of infections. Research has demonstrated significant consequences when targeting the responsible microbes appropriately. For instance, in a prospective study, patients with Gram-negative bacteremia had a markedly lower mortality rate (18% vs. 34%) when they received antibiotics that effectively targeted the infection compared to those who received ineffective treatment.

It is not surprising that patients who receive inadequate antibiotic therapy have a 10% higher hospital mortality rate. This underscores the importance of promptly administering appropriate antibiotics tailored to the specific pathogens causing the infection, as this significantly impacts patient outcomes and mortality rates in clinical practice.[48] Therefore, if you suspect sepsis, it's best to provide a broad-spectrum antibiotic as quickly as possible, preferably within an hour of the diagnosis.[26]It is crucial to deliver the right antibiotics promptly, even if this is not yet the worldwide standard of care and additional research is needed to determine its viability.[46] There is a substantial variation in the selection of empirical antibiotic treatment according to patient attributes. Infection likely(s), antibiotic use during the last three months, healthcare exposure within the past few months (e.g., hospitalisation), preexisting chronic diseases, local infections, and medication resistance are all important factors to consider.[26] Patients who have recently used antibiotics are at increased risk of acquiring infections caused by high-risk pathogens such as Pseudomonas aeruginosa and methicillin-resistant Staphylococcus aureus (MRSA). Similarly, individuals who

have recently been in healthcare facilities are more likely to be colonized with bacteria that produce extended-spectrum beta-lactamases (ESBL), which confer resistance to many antibiotics. These factors highlight the importance of considering patients' recent antibiotic use and healthcare exposure when selecting empirical antibiotic therapy. Tailoring treatment based on these risk factors helps to address potential multidrug-resistant infections effectively, thereby improving patient outcomes in clinical settings. [51]Notably, among hospitalised patients, Gram positive bacteria, Gram negative bacteria, and mixed bacterial pathogens account for the majority of SS/SS infections.[26] The most probable entry points for the illness and local antibiograms should always be considered when prescribing antibacterial medication. Fungi, like bacteria, are becoming more prevalent sources of infection, especially in healthcare facilities; among them, Candida is identified as the fourth most common pathogen in blood cultures of patients with septic shock. Despite this, fungi are estimated to cause only about 5% of all cases of severe sepsis or septic shock (SS/SS). Therefore, routine administration of antifungal medications is not recommended.

Antifungal therapy should be reserved for specific patient populations with certain medical conditions, including:

Immunocompromised states such as neutropenic cancer patients or transplant recipients.

Conditions associated with impaired physiological barriers, such as recurrent gastrointestinal perforations or anastomotic leakages, acute necrotizing pancreatitis, chemotherapy-induced mucositis, or the presence of vascular access devices.

Patients who fail to show improvement while receiving antibiotic therapy.

In these cases, antifungal treatment with agents like azoles or echinocandins may be warranted based on clinical judgment and the specific risk factors present in the individual patient. This targeted approach helps to optimize therapeutic outcomes while minimizing unnecessary antifungal use in the broader management of sepsis.[52] In the initial treatment of sepsis, azoles or echinocandins are commonly used antifungal agents. In resource-limited settings, a critical aspect of managing sepsis is the prompt administration of appropriate antibiotics, as this has a direct impact on mortality rates. It is recommended that antibiotics be administered as soon as sepsis is suspected, ideally within the first hour.

Factors such as the patient's recent healthcare exposure and history of antibiotic use are crucial in guiding treatment decisions. Local or regional antibiotic guidelines should be followed to select the most effective agents based on local pathogen profiles and resistance patterns. This approach helps optimize patient care by ensuring timely and appropriate antibiotic therapy tailored to the specific circumstances of each case of sepsis.

**Source control**

Another critical aspect of sepsis management is "source control," which involves actively addressing and eliminating the actual site of infection. This intervention encompasses various procedures such as drainage of fluid collections, debridement of infected soft tissues, removal of infected medical devices (such as catheters or prosthetic implants), and any other necessary measures aimed at eradicating the source of infection.

Source control is essential because it helps to reduce the burden of infecting microorganisms and toxins within the body, thereby supporting the effectiveness of antimicrobial therapy and promoting the restoration of normal physiological function. Timely and appropriate implementation of source control measures is crucial in improving outcomes for patients with sepsis. [54]. Prompt identification and removal of the infection focus can significantly alter the course of sepsis.

When performing interventions for source control, it is advisable to start with the least invasive options, such as percutaneous procedures rather than surgical ones, whenever possible [54]. In sepsis management, it is crucial to follow specific protocols regarding catheter management:

Blood Cultures from Vascular Catheters: Before removing indwelling vascular catheters, it is important to collect blood cultures from these catheters. This helps to identify any bloodstream infections associated with the catheter before its removal.

Replacement of Urinary Catheters: When removing indwelling urinary catheters, it is recommended to replace them while simultaneously collecting a urine culture from the old catheter. This ensures that any potential urinary tract infections (UTIs) associated with the catheter are identified through culture before the catheter is changed.

These practices are part of the broader strategy of source control in sepsis management, aiming to identify and treat infections at their source to improve patient outcomes and prevent complications. [55].

In resource-limited settings, prioritizing the drainage or debridement of the infection source, whenever feasible, is crucial [56]. This approach aims to halt the progression of infection and improve patient outcomes.

**Corticosteroids**

In the context of severe sepsis or septic shock (SS/SS), adrenal insufficiency may play a role [57]. There has been considerable investigation into whether regular corticosteroid administration can expedite shock recovery and reduce mortality rates by mitigating tissue damage caused by cytokines and neutrophils [58]. Initially, corticosteroids were thought to have no benefit and potentially worsened outcomes in SS/SS [59]. Subsequent studies, particularly those conducted by Annane et al., have shown that the administration of low-dose hydrocortisone and fludrocortisone can effectively reverse shock and reduce mortality in cases of septic shock where patients are unresponsive to vasopressor therapy. This treatment approach has demonstrated efficacy in improving hemodynamic stability and outcomes for patients with severe septic shock who do not adequately respond to standard vasopressor treatments alone.[60] Conversely, findings from a large multicenter study known as CORTICUS indicated that patients who responded to vasopressors did not derive a survival benefit from the administration of low-dose steroids. This study provided evidence suggesting that while low-dose steroids may be beneficial in vasopressor-unresponsive cases of septic shock, their use does not necessarily improve outcomes in patients who respond well to vasopressor therapy alone. [61]. The current consensus recommends administering 200 mg of intravenous hydrocortisone daily in cases of vasopressor-unresponsive shock following adequate fluid resuscitation and vasopressor therapy. This approach aims to support adrenal function and mitigate the

inflammatory response associated with severe septic shock, potentially improving hemodynamic stability and patient outcomes. [26]. Steroids should be reserved for persistent septic shock in settings with limited resources.

Regarding glucose management, recent findings from the NICE-SUGAR study suggest that both standard glucose control (targeting blood glucose < 180 mg/dL) and strict glycemic control (targeting blood glucose 80-110 mg/dL) are associated with lower mortality rates [62]. Protocols now typically initiate insulin therapy after two consecutive blood glucose readings above 180 mg/dL [26]. Careful monitoring is crucial when lowering glucose levels to avoid hypoglycemia, which can be more dangerous than moderate hyperglycemia in the absence of regular monitoring.

In patients with septic shock, approximately 50% develop acute respiratory distress syndrome (ARDS) within the first 24 hours, often necessitating mechanical ventilation [63]. Early decisions on intubation and mechanical ventilation can help reduce the work of breathing and improve oxygen delivery [26]. Lung-protective ventilation strategies, including low tidal volumes (6 mL/kg of predicted body weight) and low plateau pressures (<30 cm H2O), have been shown to decrease mortality in patients with ARDS [64]. Proper management includes promptly titrating FiO2 to avoid oxygen toxicity and maintaining modest positive end-expiratory pressure (PEEP) to prevent alveolar collapse [26]. Additionally, elevating the head of the bed 30-45 degrees has been shown to reduce the risk of ventilator-associated pneumonia [65]. Regular monitoring of oxygen saturation and providing supplementary oxygen to maintain levels above 90% are essential in resource-limited settings.

Medical treatment for sepsis should be initiated promptly based on the suspected source of infection, with careful consideration to rule out pseudosepsis, which can mimic sepsis symptoms but does not require antibiotics [66-68]. Patients with severe sepsis or septic shock often require admission to the intensive care unit (ICU) for close monitoring and intensive therapy [69]. Studies have demonstrated improved outcomes when clinical goals such as timely blood cultures, lactate measurement, antibiotic administration, and fluid resuscitation are met early in the management of sepsis [69].

**Antimicrobial Therapy**

A proper antibiotic regimen must address the resident flora of the organ system thought to be responsible for the septic process. On pages 66–68 Depending on the aetiology and underlying microbiology of the sepsis, there are some agents that may be appropriate for empiric monotherapy regimens. These agents must be able to cover all of the potential bacteria.

These antibiotics are available: moxifloxacin, piperacillin-tazobactam, tigecycline, meropenem, and imipenem.

Metronidazole with levofloxacin, aztreonam, or an aminoglycoside is one example of a combination treatment plan. A lot of people think that you should combine an extended-spectrum cephalosporin, carbapenem, or beta-lactam/beta-lactamase inhibitor antibiotic with an antistaphylococcal coverage (such vancomycin, for example).

There may not be a best medication schedule, but the amount of time between the first dose and the next is crucial. Mortality statistics point to a correlation between

greater survival and early prescription of suitable antibiotics. Alternative agents have a low risk of side effects and can be used singly or in combination. On pages 66–68 Typically, antibiotics are kept up until the infection's cause is under control, either by surgical treatments or the septic process. Typically, patients get treatment for around two weeks. As soon as patients develop an oral tolerance to their drugs, they can be transitioned from an intravenous to an oral antibiotic regimen that is equal.

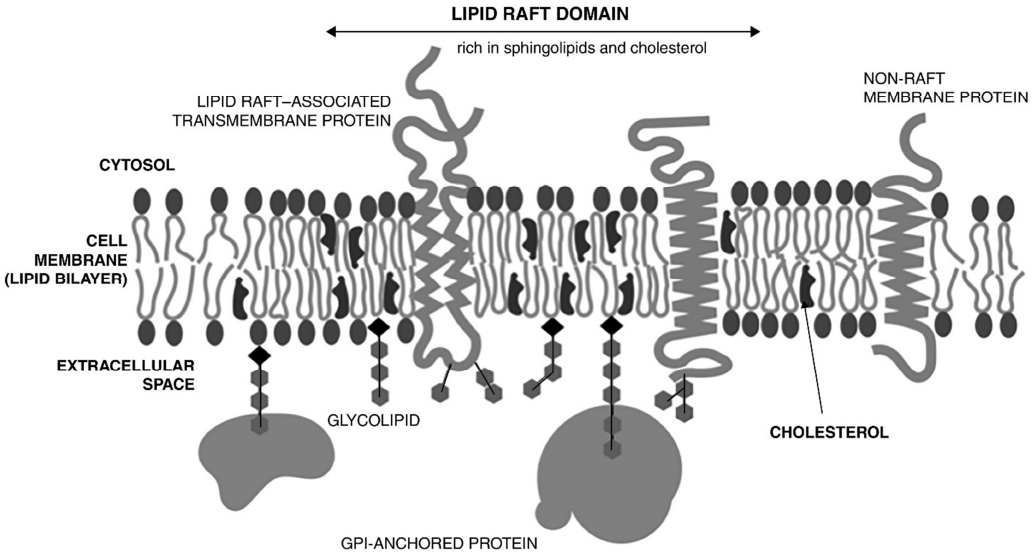
**Lipid profile and Sepsis**

Cholesterol, a type of sterol lipid, plays crucial roles in various bodily functions such as cellular membrane processes, immunity, signaling, and pathway regulation. It also serves as a foundational element for the synthesis of other important compounds like oxysterols, bile acids, vitamin D, and steroid hormones. Discovered a century ago, low cholesterol levels induced by sepsis are linked to worse prognoses according to multiple studies.

Nevertheless, there is a need to clarify the processes by which concentration in plasma decrease, the effects on organ function, the correlation between plasma and intracellular cholesterol levels, and the possible therapeutic function of cholesterol.69 In sepsis, the potential therapeutic uses of lipoproteins and cholesterol transport modulation are attracting more and more attention, especially for their role in immuno inflammatory regulation and microbial phagocytosis. Cholesterol has received more attention than its transporters, though.

**Cholesterol Synthesis, Structure, Metabolism, and Functional Roles**

Cholesterol is made up of a hydrophobic chain, a tiny hydrophilic hydroxyl group, and four aromatic hydrophobic rings that are connected together. Cholesterol is coupled to lipid-binding proteins or exists as a component of lipid membranes within cells due to its strong hydrophobicity (Figure 1).70 Cholesterol is mostly synthesised in the body, although animals also get it from their food. A single molecule of cholesterol takes 18 ACETYL-COA, 36 ATP, 16 NADPH, and 11 oxygen molecules, and the process is multistep and energy intensive, involving around 30 reactions. A negative feedback loop tightly controls the production of endogenous cholesterol (Figure 2). As the main method by which the cells adjust to variations in cholesterol bio-availability, HMG-COA (HYDROXYMETHYLGLUTARYL-COENZYME A) reductase is the focus of statin therapy and is known as the the rate limiting enzyme of the process.




**<u>Figure 2.</u> Structure of Cholesterol; Location inside cellular membranes.**

**GPI = glycosylphosphatidylinositol.**

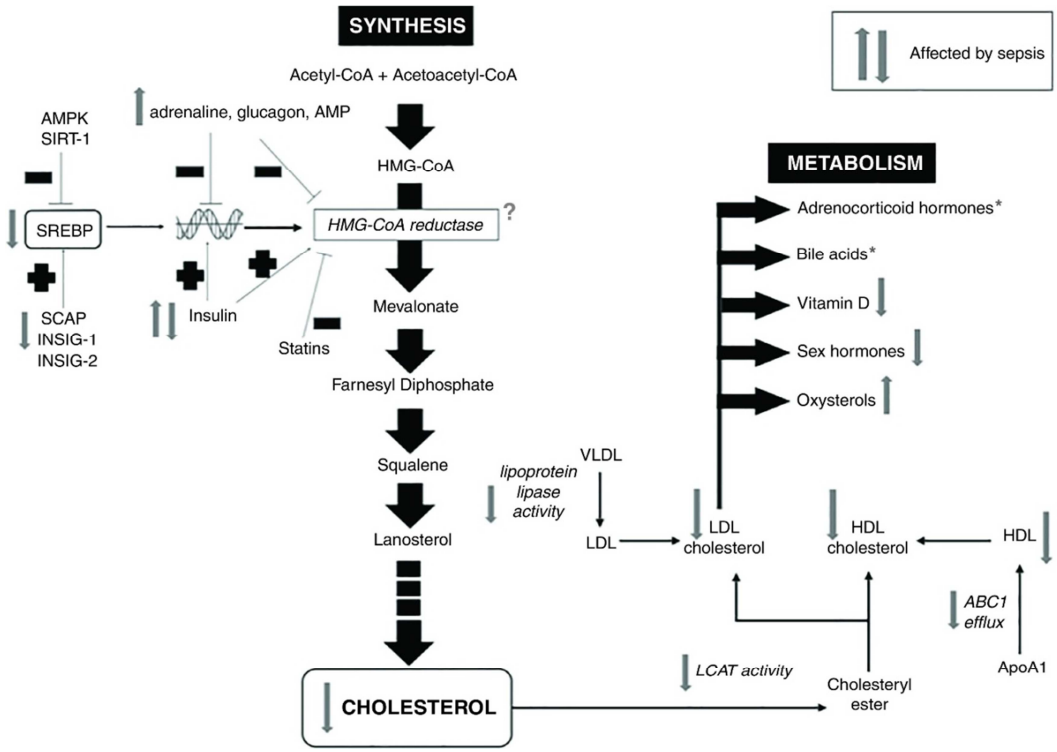



**Figure 3: The Effect of Sepsis on Cholesterol BioSynthesis & Metabolism *Normal or elevated plasma concentrations of bile acids and adrenocorticoid hormones may be due to reduced metabolism and removal rather than increase in synthesis. It is very uncommon for exogenous ACTH (adrenocorticotropic hormone) activation to not increase cortisol concentrations.**

It is necessary to bind cholesterol to lipoproteins or albumin in order for it to be transported in plasma. The density, dimension, and the kinds of particle forming protein and other related proteins classify lipoproteins into Chylomicrons, Chylomicron remnants, Very-Low-Density lipoprotein, Low-Density Lipoprotein, and High-Density Lipoprotein. An example of "reverse cholesterol transport" would be transfer of the cholesterol from peripheral organs to liver via HDL, and vice versa for LDL. 70 The catabolism and recycling of cholesterol and its derivatives is not an enzyme system that mammalian cells possess. Through LDL and HDL receptors, the

liver removes cholesterol from the bloodstream. 71 After that, it undergoes metabolism or is removed unchanged or as bile acids, with a significant amount of it being recycled. Figure 3 shows the several biological roles played by cholesterol and its metabolites:

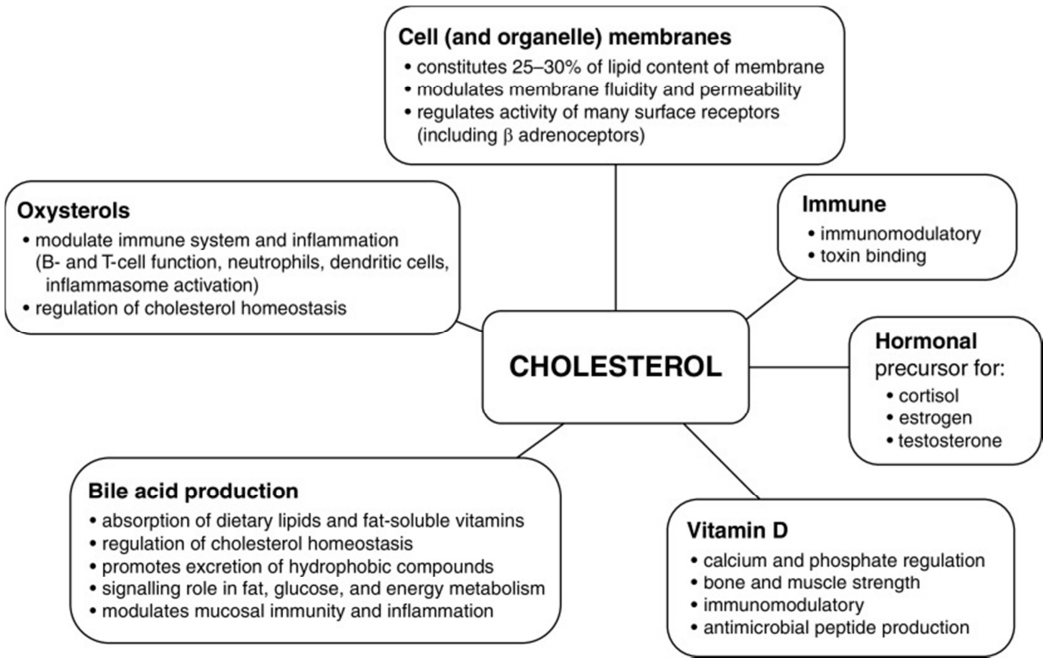



**Figure 4.:Functional roles of cholesterol.**

The function, fluidity, permeability, and thickness of cell membranes are all significantly influenced by cholesterol, which is an essential component of these membranes.72,73 Cholesterol forms lipid rafts after a non-uniform distribution inside the membrane. A large number of transmembrane proteins, including G-protein-coupled receptors (GPCRs), ion channels, transporters, and microdomains loaded in sterols and sphingolipids are attracted to these tiny, extremely active membranes. 73 Changes in cholesterol levels within the membrane affect its physical properties and the presence and function of transmembrane proteins such as the sodium-potassium-ATPase and β-adrenergic receptors.73

The immunomodulatory effects of cholesterol and the lipoproteins that carry it include the ability to bind endotoxin and other poisons.the number of initiation of Toll-like receptors (TLRs) by molecular patterns related with pathogens is prevented by this scavenging process, which may be a crucial component of the innate immune response in neutralising toxins. Important immune response regulators, including toll-like receptors (TLRs) and T-and B-cell receptors, are found inside lipid rafts.76

Vitamin D, steroid sex hormones (like-., oestrogen, progesterone, testosterone), adrenocortical hormones (e.g., glucocorticoids, aldosterone), and steroidogenic substrates (e.g., cholesterol) are all produced by complex metabolic pathways.77 About 80% of the cortisol in the bloodstream during a stress reaction that has been activated could come from plasma cholesterol.78 Extensive research has examined the effects of vitamin D on a variety of conditions, including insulin resistance, metabolic-syndrome, musculoskeletal problems, cardio-vascular disease, and immune related dys-function.79

The primary pathway for cholesterol metabolism is the 17-step enzymatic process that takes place inside hepatocytes to convert cholesterol to bile acids. Enterohepatic recirculation enables recycling of bile acids, and de novo hepatocyte production makes up for physiological losses in the intestines. Bile acids have many purposes: they help the liver excrete metabolites, they aid in the assimilation of hydro-phobic minerals, lipids, and fat soluble vitamins, and they limit the proliferation of bacteria in the gut(small intestine) and biliary tract. Additionally, those control a wide range of cellular processes in the liver, including cell differentiation and regeneration, among others.80

Oxysterols are a big family of oxidised cholesterol derivatives that have several biological functions, one of which is immunomodulation.81 Reactive oxygen species can oxidise cholesterol in two ways: enzymatically and nonenzymatically. Oxysterols regulate a wide variety of events, from cytokine generation to viral entrance into cells, via GPCRs, intranuclear receptors, and other molecular pathways.Numerous 82,83 The anti-inflammatory cytokineIL-10 synthesis, polymorphs(neutrophils), B-cell type, and T-cell type functioning, and innate immunity are all influenced by oxysterols..83,84

**Cholesterol Concentrations reduce during Sepsis, in association with Severity and Outcomes**

It is well-known that sepsis is associated with decreased levels of total cholesterol in plasma, HDL cholesterol (HDL-C), and LDL cholesterol (LDL-C).85–94 The concentrations are already low when the patient is diagnosed, and they tend to drop much more as the condition progresses. At least 87.91 When compared to LDL-C, which is at its lowest upon diagnosis, serum HDL-C values hit rock bottom around day three following admission. In the days that follow, serum concentrations recover at different rates. In human sepsis, the dynamics of very low-density lipoprotein cholesterol (VLDL-C) are not well understood. 87,91

Patients having low total cholesterol, HDL-(C), LDL-(C) concentrations are more likely to die, according to many studies. 89–94 It is worth mentioning that a latest study in genetics indicated that lower HDL-C levels may be a causative component, although lower LDL levels in sepstic patients may be associated with an increased mortality risk. 93,95 Through improved pathogen lipid clearance, increased LDL clearance may help reduce sepsis mortality.93

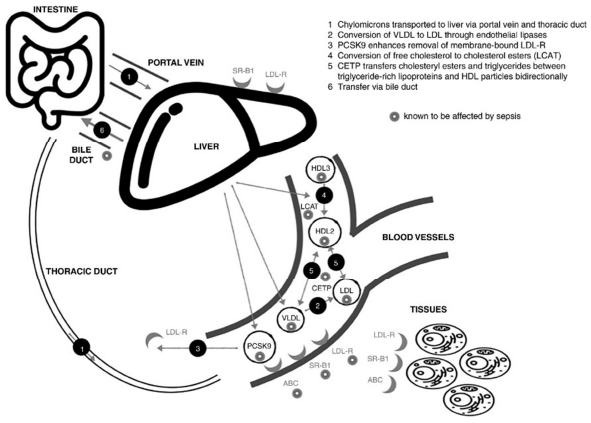
A gradual recovery to almost normal readings is observed in survivors as the illness progresses. The risk of multiorgan dysfunction, length of intensive care unit admission, and nosocomial infection increases with the size of the fall. 89,92 Cholesterol levels are inversely related to elevated blood indicators of inflammation. 76,80,94

There was a strong negative relationship between inflammatory indicators and the substantial decreases in plasma cholesterol observed in cancer patients after receiving recombinant TNF-α or IL-6 infusions.96 As a therapeutic test bed, animal trials can reproduce similar results. Nevertheless, the results may vary depending on the model, as hypercholesterolemia is really seen in several rodent models that have been treated with endotoxin or TNF-α.98 But when rats were exposed to a more realistic peritonitis insult, our results and those of others showed significant reductions in total cholesterol and HDL-(C) levels. Primate, sheep, and dog septic models have also shown hypocholesterolemia. 99-104

**Why Does the Serum Cholesterol show falling trend in Sepsis?**

Hypocholesterolemia in sepsis is a poorly understood biological phenomenon. There may be a number of factors at play in critical illness, including reduced fat intake and intestinal absorption, compromised transport of cholesterol, increase in metabolism, consumption via toxin scavenging, and reduced synthesis.105 There is a lack of consistent data on how sepsis affects cholesterol production. Hypercholesterolemia and enhanced hepatic cholesterogenesis were found in earlier investigations using animal models. In contrast, Vasconcelos et al. found that septic rats had lower HMG-CoA reductase activity than fed rats in good condition. Our In a rat peritonitis model, there is observed reduced mRNA expression of transcriptional

regulators (SREBP-1, SREBP-2, INSIG, HMG-CoA reductase) and enzymes (INSIG, insulin-induced gene 1 protein) involved in hepatic cholesterol synthesis pathway. These findings are from unpublished data by A. Kleyman and colleagues. Hypocholesterolemia may be worsened by proinflammatory cytokines, which inhibit the liver's production of apolipoproteins responsible for binding cholesterol to form lipoproteins.107 While a lower HDL-C is constant observation, reports of reduction in the serum LDL-C are prevalent but inconsistent. These alterations point to a potential increase in the impact on reverse cholesterol transport, which is the process by which cholesterol is transferred from peripheral tissues to the liver. The several cholesterol metabolic and transport routes impacted by sepsis are shown in Figure 4. Sepsis affects transporters, including the ABC superfamily, which converts apoA-1 particles—low in lipids—into mature HDL particles. It also impacts enzymes like LCAT (lecithin-cholesterol acyltransferase), which enhances the hydrophobicity of free cholesterol for incorporation into HDL.107 Changes to HDL's structure and protein makeup, as well as the buildup of oxidised lipids, impact its binding ability. 108




**<u>Figure 5.</u> Effect of sepsis on transport of cholesterol.**

When serum CETP levels are low, the percentage of HDL-(C) increases because CETP facilitates the transfer of triglycerides and cholesteryl esters from triglyceride-rich lipoproteins to HDL particles. But overall levels of cholesterol in the blood don't change.109 Variation in serum CETP levels and their association to results in sepsis have been the subject of contradictory research.110-112

Hypercholesterolemia is caused by the degradation of liver LDL and fat cell VLDL receptors; however, there is conflicting patient evidence about changes in serum levels of PCSK9 (proprotein convertase subtilisin kexin 9). 113–115 Sepsis may enhance cholesterol metabolism by enzymatic and nonenzymatic oxidation. Inflammation and its byproduct, 25-hydroxycholesterol, potently stimulate cholesterol-25-hydroxylase.116 Inflammation causes an increase in the activity of the acute phase proteins like PLA2 (Phospholipase A2), which in turn stimulates an increase in the metabolism of cholesterol-esters and apol-ipoproteins, leading to a decrease in blood cholesterol.117 SAA (serum amyloid A) is an acute-phase reactant that impacts cholesterol transport and also increases PLA2 activity.118 Bile flow is reduced in sepsis. Within hours of inducing polymicrobial sepsis, 119 compromised bio-transformation and hepato-biliary transport of bile acid products ensue.120

The result is an increase in bile acid concentration in the blood.

In a study carried out by Bhargav Kiran Gaddam, M. Narayanan et al., the SOFA score was utilised as a sepsis score and was found to be correlated with HDL-C levels at different points during the hospital stay. The results demonstrated a statistically significant relationship regardless of whether the participants were survivors or non-survivors, with a correlation coefficient of -0.63 and a p value of <0.001* on admission and -0.50 and <0.001* on day 5.121

An analogous study carried out by Naresh et al. utilised the APACHE 2 score and found a statistically significant association of -450 among survivors and -163 among non-survivors, with a p-value of less than 0.003.122

According to Shao-hua Liu et al.'s systematic review and meta-analysis, lower HDL levels are linked to a higher mortality rate in septic patients, but not to the risk of sepsis. This finding implies that lower HDL levels are associated with an increased mortality rate in septic patients.123

Researchers observed that there was a 100% survival rate among 63 patients whose plasma HDL concentrations were higher than 25 mg/dl when they were administered the drug. Furthermore, by dividing patients into two categories, "low" (<20 mg/dl) and "high" (>20 mg/dl) HDL, the researchers were able to evaluate the efficacy of HDL in predicting death rate. With these thresholds in place, HDL was able to accurately predict the 30-day mortality rate with a sensitivity of 92%, specificity of 80%, and a total of 83%.124 total

The non-survival group had mean HDL levels of 33 and 31 mg% at days 0 and 3, respectively, with a non-significant p value of 0.137, whereas the survival group had levels of 31 and 36 mg%, respectively, which was statistically significant with a p value of 0.033, according to a research by Jeyasuriya et al.125

The mean HDL levels of patients with sepsis were 52.17 mg/dl, those with severe sepsis were 41.81 mg/dl, and those without severe sepsis were 40.08 mg/dl, according to a research done in Kerala by Sunayana P et al. Although there was a statistically insignificant difference between the groups, 11.8% of patients with HDL levels over 50 mg/dl and 32.1% of patients with HDL levels below 50 mg/dl died.

The p-value for this difference was 0.1.126 In their research, Faheem W. Guirgis et al. looked examined data from 88 patients. There was a correlation between Dys-HDL (measured as HDL inflammatory index, or HII) and SOFA at enrollment (r = 0.23, p = 0.024) and 48 hours (r = 0.24, p = 0.026) but no correlation between the change in HII throughout the first 48 hours and the change in SOFA (r = 0.06, p = 0.56). Patients with the most severe organ failure had a significantly different enrollment HII (2.31, IQR 1.33-5.2) compared to those with less severe organ failure (1.81, IQR 1.23-2.64, p = 0.043). There was a statistically significant difference between the in-hospital non-survivors (-0.45, IQR-2.6, -0.14, p = 0.015) and the 28-day non-survivors (-1.12, IQR -1.52, 0.12, p = 0.044) in terms of the change in HII over 48 hours. There was a 0.72 (p = 0.009) rise in 48-hour SOFA for every unit increase in HII in a multivariable linear regression equation (R2 = 0.13). As a predictor of the 48-hour SOFA score in early sepsis, HII was found to correlate with SOFA. To identify possible processes, further research is required.127
We found substantial changes in many lipoprotein metabolism enzymes in 53 intensive care unit (ICU) patients with sepsis and 25 ICU patients without sepsis, according to a research by Alexander C. Reisinger et colleagues. Patients with sepsis had considerably lower levels of cholesteryl transfer protein (CETP) activity, lecithin-cholesterol acyl transferase (LCAT) activity, and LCAT concentration, and significantly higher levels of endothelial lipase (EL) and phospholipid transfer activity protein (PLTP). Furthermore, sepsis patients had ten times higher serum amyloid A (SAA) levels than non-septic patients. In addition, we discovered that LCAT activity was linked to mortality outcomes in the intensive care unit and after 28 days, whereas SAA levels, which indicate a strong inflammatory markers, were not.128
In a research carried out by Sébastien Tanaka et al., 75 consecutive patients were

hospitalised, with 50 presenting with sepsis and 25 with trauma. Both groups received the same amount of points on the SOFA and SAPSII tests. Total cholesterol levels were lower in sepsis patients compared to trauma patients. There was a substantial difference between the two groups in terms of the lipoprotein profile, with the exception of HDLs, which varied considerably between sepsis patients (median [IQR] = 0.33 mmol/l [0.17-0.78]) and trauma patients (0.99 mmol/l [0.74-1.28]; $P < 0.0001$). In the sepsis group, there was no association between lipid levels and ICU mortality. However, in the group of septic patients who survived until ICU discharge, there was a significant negative connection between HDL concentration and the length of ICU stay ($r = -0.35$; $P = 0.03$). Furthermore, lower HDL levels were linked with poor result, as defined as death or a SOFA score >6 at day 3. Patients with poor outcome had a median [IQR] of 0.20 mmol/l [0.11-0.41] compared to 0.35 mmol/l [0.19-0.86] in the other group; $P = 0.03$). There was a striking difference in the lipid profiles of intensive care unit (ICU) patients suffering from sepsis and trauma. HDL levels were much lower in the former group, whereas trauma patients showed no change in their concentration. This significant distinction highlights the need to investigate HDL's therapeutic potential in sepsis. 129

**Sequential organ failure assessment score** (**SOFA score)**

An individual's status throughout their stay in the intensive care unit (ICU) can be monitored using the sequential organ failure assessment score (SOFA score), which was formerly called the sepsis-related organ failure assessment score [1]. This score is used to identify the extent of an organ's function or rate of failure.the numbers 2–6 An individual's health status in the areas of breathing, heart, liver, coagulation, kidneys, and nervous system is reflected in their overall score.

The following score tables solely detail the circumstances that award points. If any row does not contain physiological parameters, then no points are awarded. The row with the most points is chosen when the physiological parameters match more than one row.

Healthcare practitioners can estimate the risk of sepsis-related morbidity and death with the use of the fast SOFA score (qSOFA). .[7]

**Medical use**

Patients in critical care might have their clinical outcomes predicted with the use of the SOFA score system.[8] The death rate is at least 50% when the score is increased, independent of the original score, in the first 96 hours after admission, 27% to 35% when the score remains same, and less than 27% when the score is dropped, according to an observational research at an ICU in Belgium.[9] An ideal score is zero, while a terrible score is twenty-four..[10]

| | **Central nervous system** | **Cardiovascular system** | **Respiratory system** | **Coagulation** | **Liver** | **Renal function** |
|---|---|---|---|---|---|---|
| **Score** | **Glasgow coma scale** | **Mean arterial pressure OR administration of vasopressors required** | **$PaO_2/FiO_2$ [mm Hg (kPa)]** | **Platelets ($\times 10^3/\mu l$)** | **Bilirubin (mg/dl) [µmol/L]** | **Creatinine (mg/dl) [µmol/L] (or urine output)** |
| **+0** | 15 | MAP ≥ 70 mmHg | ≥ 400 (53.3) | ≥ 150 | < 1.2 [< 20] | < 1.2 [< 110] |
| **+1** | 13–14 | MAP < 70 mmHg | < 400 (53.3) | < 150 | 1.2–1.9 [20-32] | 1.2–1.9 [110-170] |
| **+2** | 10–12 | dopamine ≤ 5 µg/kg/min or dobutamine (any dose) | < 300 (40) | < 100 | 2.0–5.9 [33-101] | 2.0–3.4 [171-299] |
| **+3** | 6–9 | dopamine > 5 µg/kg/min OR epinephrine ≤ 0.1 µg/kg/min OR norepinephrine ≤ 0.1 µg/kg/min | < 200 (26.7) **and** mechanically ventilated including CPAP | < 50 | 6.0–11.9 [102-204] | 3.5–4.9 [300-440] (or < 500 ml/day) |
| **+4** | < 6 | dopamine > 15 µg/kg/min OR epinephrine > 0.1 µg/kg/min OR norepinephrine > 0.1 µg/kg/min | < 100 (13.3) **and** mechanically ventilated including CPAP | < 20 | > 12.0 [> 204] | > 5.0 [> 440] (or < 200 ml/day) |

**Table 3: SOFA score**

**Quick SOFA score**

In February 2016, the Sepsis-3 group introduced the Quick SOFA Score (qSOFA), a concise version of the SOFA Score. Initially designed to quickly identify patients at high risk of adverse outcomes from infections.18 It was found that the SIRS Criteria definitions for sepsis had significant limitations, leading to updates. The

task committee unanimously agreed that the current practice of using 2 or more SIRS criteria to define sepsis was not useful. The qSOFA simplifies the SOFA score by retaining only three clinical criteria and substituting "any altered mentation" for a GCS <15. qSOFA is designed for quick and straightforward administration to patients in a serial manner.

| **Assessment** | **qSOFA score** |
|---|---|
| Low blood pressure (SBP ≤ 100 mmHg) | 1 |
| High respiratory rate (≥ 22 breaths/min) | 1 |
| Altered mentation (GCS ≤ 14) | 1 |

**Table 4: Q SOFA score**

Scores ranging from 0 to 3 are possible with qSOFA. Increased risk of mortality or prolonged critical care unit stay is associated with having two or more qSOFA criteria present early in the infection. Septic patients, compared to those with uncomplicated infections, are more prone to these outcomes. These findings reinforce the Third International Consensus Definitions for Sepsis recommendation of qSOFA as a simple tool for identifying septic infections in non-intensive care unit settings. [12] In terms of mortality risk, qSOFA is not very sensitive but rather specific, suggesting that SIRS may be a superior screening tool.in [13]

**Utility of qSOFA**

Because not all of the data utilised to calculate the SOFA score may be readily available to the healthcare practitioner in non-intensive care unit settings, the qSOFA was developed for such situations. The initial evaluation of patients often takes place

in a healthcare facility, such as an emergency room. The emergency department may rapidly collect the three criteria (systolic blood pressure, respiration rate, and GCS) to stratify patients based on risk and deliver timely therapies to those who may be sick. Patients at risk of developing sepsis can be identified using this scoring method. [14]

| SIRS |
|---|
| Fever of more than 38°C (100.4°F) or less than 36°C (96.8°F) |
| Heart rate of more than 90 beats per minute |
| Respiratory rate of more than 20 breaths per minute or arterial carbon dioxide tension ($PaCO_2$) of >32 mmHg |
| Abnormal white blood cell count (>12,000/μL or<4,000/μL or >10% immature [band] forms) |

SIRS: Systemic inflammatory response syndrome

| SOFA score | | | | | |
|---|---|---|---|---|---|
| **Variables** | **0** | **1** | **2** | **3** | **4** |
| Respiratory $PaO_2/FiO_2$, mmHg | >400 | ≤400 | ≤300 | ≤200 | ≤100 |
| Coagulation platelets×$10^3$/μL | >150 | ≤150 | ≤100 | ≤50 | ≤20 |
| Liver bilirubin, mg/dL | <1.2 | 1.2–1.9 | 2.0–5.9 | 6.0–11.9 | >12.0 |
| Cardiovascular hypotension, mmHg doses in μg/kg/min | No hypotension | Mean arterial pressure <70 | Dop ≤5 | Dop >5, epi ≤0.1 Or norepi ≤0.1 | Dop >15, epi >0.1 or norepi >0.1 |
| Central nervous system Glasgow coma scale | 15 | 13–14 | 10–12 | 6–9 | <6 |
| Renal creatinine, mg/dL or urine output, ml/dL | <1.2 | 1.2–1.9 | 2.0–3.4 | 3.5–4.9 or <500 | >5.0 or <200 |

SOFA: Sequential organ failure assessment, $PaO_2$: Partial pressure of oxygen, $FiO_2$: Fraction of inspired oxygen, Dop: Dopamine, epi: Epinephrine, norepi: Norepinephrine

**Table 5: SOFA scoring system**

| qSOFA variables |
|---|
| Glasgow coma scale ≤13 |
| Systolic blood pressure ≤100 |
| Respiratory rate ≥22 |

qSOFA: Quick sequential organ faliure assessment

**Table 6: qSOFA variables**

| Location of diagnosis | SIRS | SOFA | qSOFA |
|---|---|---|---|
| ICU | 0.64 (0.62, 0.66) | 0.74 (0.73, 0.76) | 0.66 (0.64, 0.68) |
| Wards | 0.76 (0.75, 0.77) | 0.79 (0.78, 0.80) | 0.81 (0.80, 0.82) |

Confidence interval denoted in parenthesis. This is based on data from University of Pittsburgh Medical Center with ICU encounters – 7,932 and outside ICU encounters- 66,522 between 2010 and 2012, AUROC: Area under receiver operating characteristic, qSOFA: Quick sequential organ faliure assessment, ICU: Intensive care unit

**Table 7: AUROC curve for prediction of in-hospital mortality**

| Attributes | EPIC II | India (%) |
|---|---|---|
| Total patients | 13,796 | 533 |
| Total ICUs | 1.265 | 39 |
| Infected | 7.087 | 213 (40) |
| ICU mortality | 25% | 13.4 |
| Hospital mortality | 33% | 17.2 |

**Table 8: Epidemiology of sepsis in India versus all other EPIC II countries**

# MATERIAL AND METHODS

**Source of Data**: The Patients admitted to Medical Intensive Care Unit in the KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi over a period of one year.

**Study Design**: Prospective Cohort

**Study Period**: 1 YEAR

**Sample Size**: According to the study done by Sathisha Kumar et al.[*] entitled "A study of serum HDL cholesterol level in sepsis patients and its prognostic significance in Coimbatore Medical College Hospital", there is a statistically significant correlation between HDL cholesterol and SOFA score at admission in patients with sepsis. The coefficient of correlation reported was 0.357 with a P value of < 0.01.

The following is the formula based on correlation for calculating the sample size:

The standard normal deviate for $\alpha = Z\alpha = 1.9600$

The standard normal deviate for $\beta = Z\beta = 0.8416$

$C = 0.5 * \ln[(1+r)/(1-r)] = 0.3734$

Total sample size = $N = [(Z\alpha+Z\beta)/C]2 + 3 = 59$

But putting the value of r = 0.357 in the above-mentioned formula, a sample size of 59 was obtained at a confidence interval of 95% and 80% power of the study.

Considering an attrition rate of 10%, we intend to include **65** patients with sepsis in our study.

Sampling technique: Written informed consent were taken from all the participants at the time of admission.

**Inclusion Criteria:**

- Patients with age greater than 18 years and satisfying the criteria for sepsis according to **International third international consensus on sepsis and septic shock (Sepsis-3)** 2016.

**Exclusion Criteria:**

- Patients on treatment with statins,
- Patients with chronic kidney disease, chronic liver disease, thyroid dysfunction, diabetes mellitus.
- Patients with known chronic infective and inflammatory conditions like Human immunodeficiency virus disease, SLE (Systemic lupus erythematous) an RA (Rheumatoid arthritis).

Patients at KLE Dr. Prabhakar Kore Charitable Hospital and Medical Research Center in Belagavi underwent screening to identify those meeting the study's inclusion and exclusion criteria. Upon approval from the ethical committee, written informed consent was obtained from all participants who were eligible and willing to enroll in the study.

**Data collection procedure** - In this prospective cohort study conducted at KLE Dr. Prabhakar Kore Hospital And Medical Research Centre in Belagavi, informed consent was obtained from all subjects, who were patients above 18 years of age. The study

focused on individuals diagnosed with sepsis, identified promptly upon diagnosis. Detailed explanations about the study were provided, and informed written consent was obtained from each participant after Institutional Ethical Committee approval.

Blood samples were collected from each patient upon admission for a comprehensive assessment including HDL-C cholesterol levels (part of the lipid profile), creatinine, bilirubin, platelet counts, and routine investigations such as CBC (Complete Blood Count), MR (Mini Renal profile), LFT (Liver Function Tests), urine analysis, and microbial cultures from suspected infection sites, all conducted before initiating antibiotic treatment. Additional assessments at admission included whole abdomen ultrasound, chest radiograph, and SOFA parameters. Subsequent evaluations on admission day and day 6 post-admission included lipid profile, platelet counts,total bilirubin, creatinine, respiratory status measured by Pao2 and Fi02, and central nervous system function assessed using the Glasgow Coma Scale and SOFA variables.

This comprehensive approach aimed to gather data that would contribute to understanding and managing sepsis in critically ill patients at the hospital.

**Statistical Analysis**

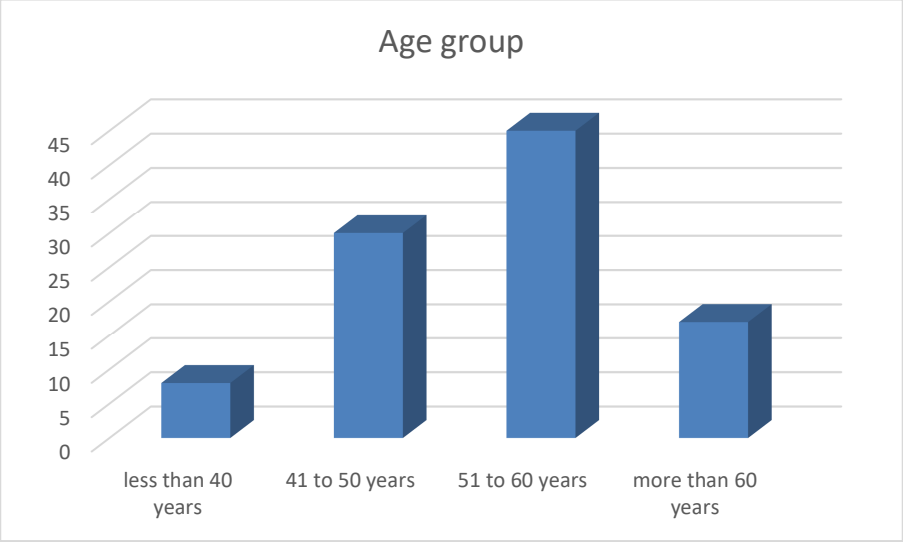
Data were entered into microsoft excel data sheet and were analysed using spss 26 version software. Categorical data were represented in the form of frequencies and proportions. chi square were used as a test of significance. Continuous data were represented as mean and standard deviation. paired t test were used as a test of significance to identify the mean difference before and after intervention. p value $<0.05$ was considered as statistically significant.

# RESULTS

| Age group | Frequency | Percent |
|---|---|---|
| less than 40 years | 5 | 8 |
| 41 to 50 years | 20 | 30 |
| 51 to 60 years | 29 | 45 |
| more than 60 years | 11 | 17 |
| Total | 65 | 100 |

**Table 9 - Age group amongst study population**

The age distribution of the study population shows that the majority of participants fall within the age range of 51 to 60 years, representing 45% of the total. The second largest age group is 41 to 50 years, making up 30% of the participants. Those aged more than 60 years account for 17%, while the smallest group, those less than 40 years, comprise 8%.

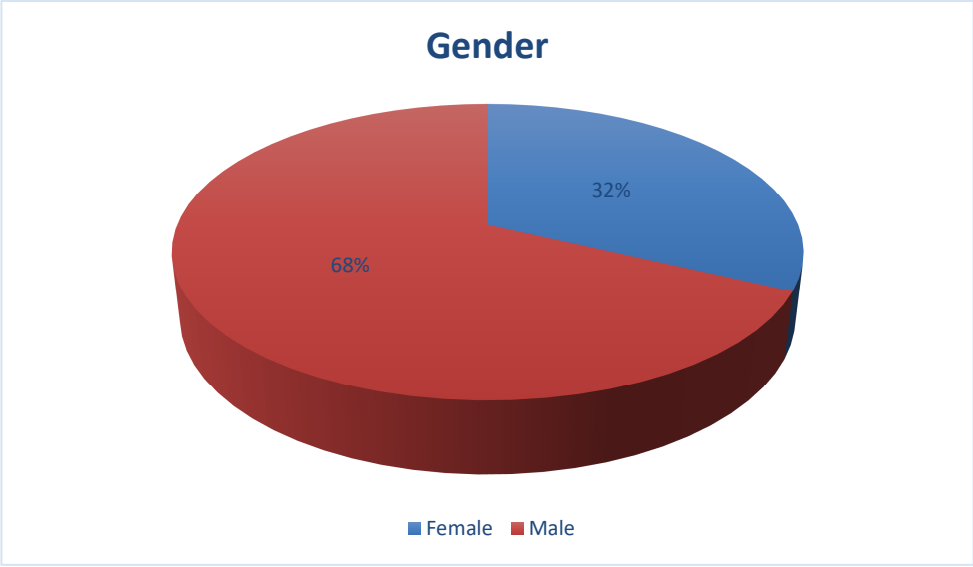



**Graph 1: Age group amongst study population**

| Gender | Frequency | Percent |
|---|---|---|
| Female | 21 | 32 |
| Male | 44 | 68 |
| Total | 65 | 100 |

**Table 10 – Sex distribution amongst study population**

There is a notable gender disparity in the study population, with males constituting a significant majority at 68%, compared to females who make up 32%.

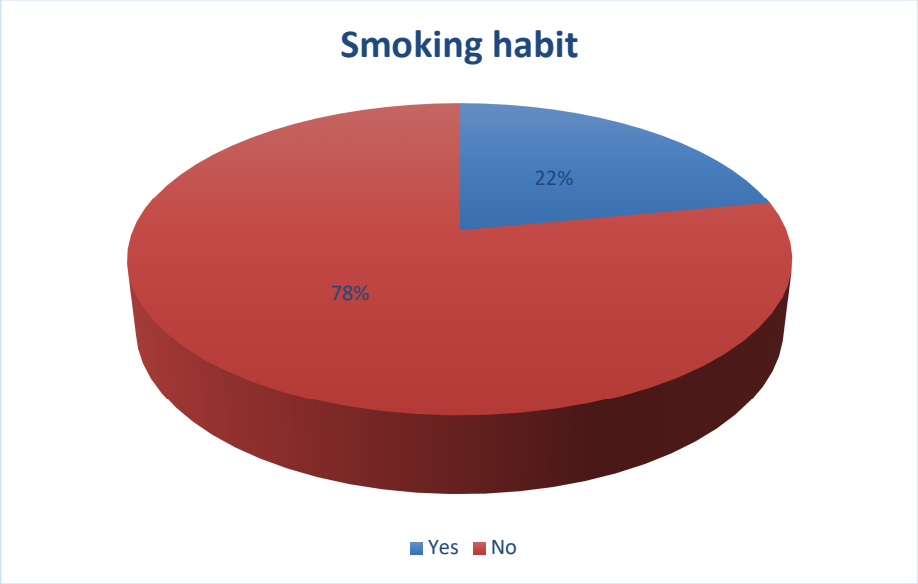



**Graph 2: Sex distribution amongst study population**

| Smoking habit | Frequency | Percent |
|---|---|---|
| Yes | 14 | 22 |
| No | 51 | 78 |
| Total | 65 | 100 |

**Table 11 – Smoking habit amongst study population**

Regarding smoking habits, a significant majority of the participants, 78%, reported not smoking. Only 22% of the participants indicated that they are smokers.

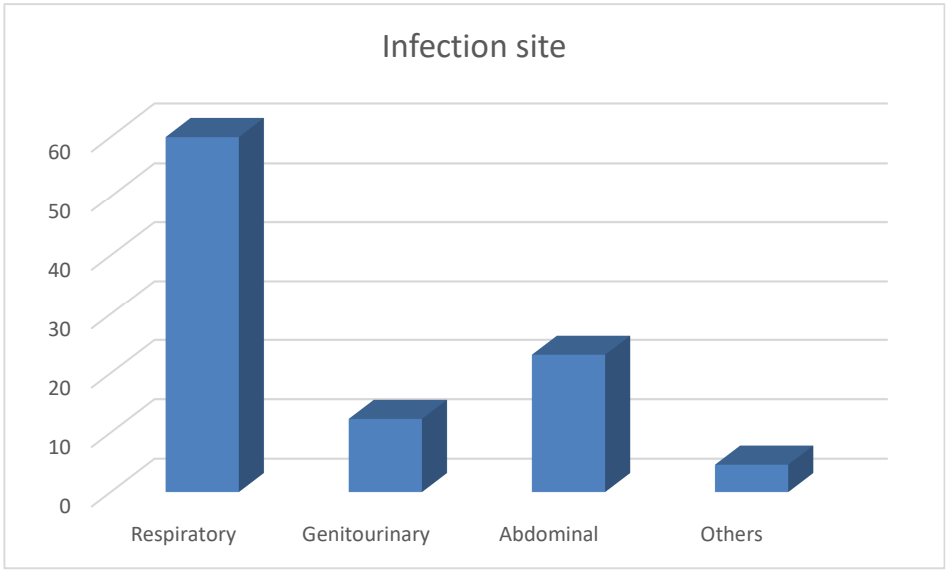



**Graph 3: Smoking habit amongst study population**

| Infection site | Frequency | Percent |
|---|---|---|
| Respiratory | 39 | 60.0 |
| Genitourinary | 8 | 12.3 |
| Abdominal | 15 | 23.1 |
| Others | 3 | 4.6 |
| Total | 65 | 100 |

**Table 12 – Infection site amongst study population**

The most common site of infection among the participants is the respiratory system, affecting 60% of the study population. Abdominal infections are the second most common, accounting for 23.1%. Genitourinary infections are present in 12.3% of the participants. A small proportion, 4.6%, have infections at other sites.

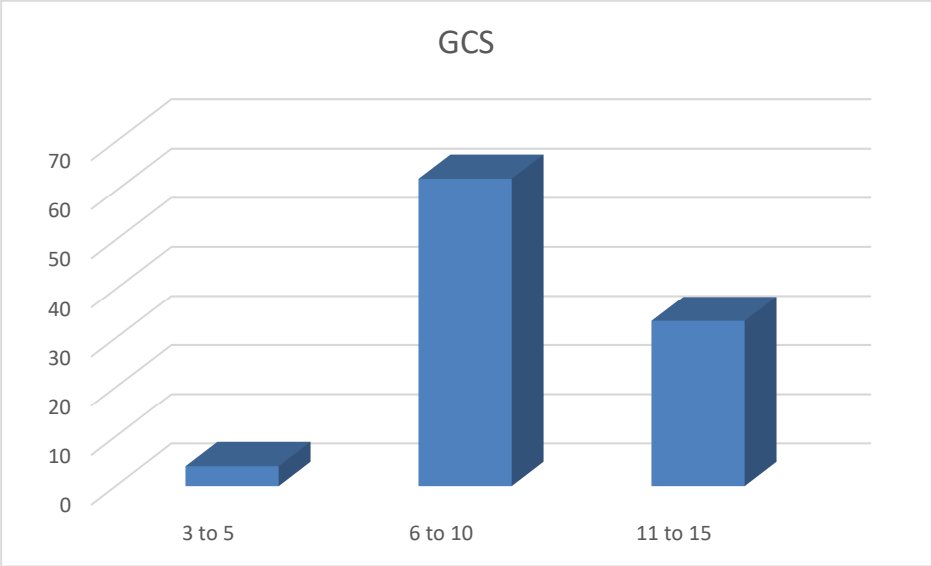



**Graph 4 – Infection site amongst study population**

| GCS | Frequency | Percent |
|---|---|---|
| 3 to 5 | 3 | 4 |
| 6 to 10 | 41 | 62.5 |
| 11 to 15 | 22 | 33.5 |
| Total | 65 | 100 |

**Table 13 – GCS amongst study population**

The Glasgow Coma Scale scores show a distribution where the majority of participants (62.5%) have scores between 6 to 10, indicating moderate to severe impairment. 33.5% of the participants have higher GCS scores ranging from 11 to 15, which suggests mild impairment or full consciousness. Only 4% of the participants have the lowest GCS scores between 3 to 5, indicating severe impairment or coma.

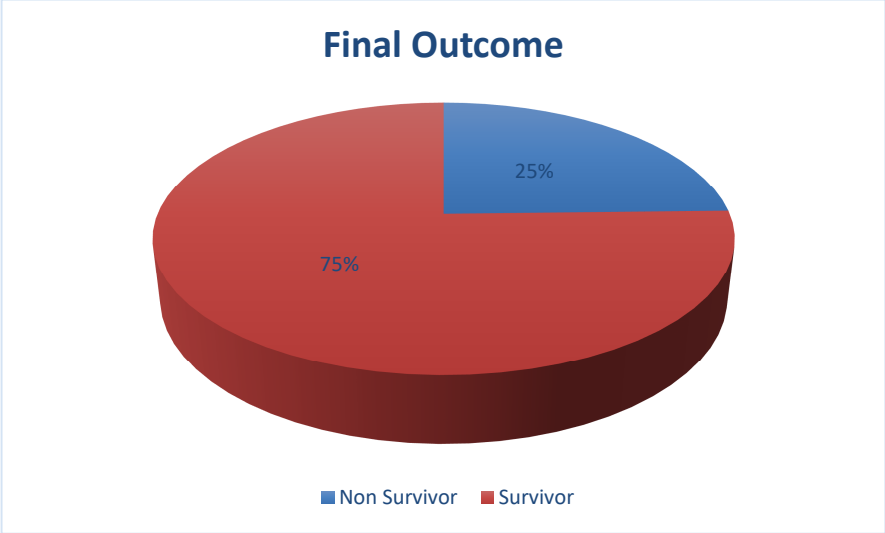



**Graph 5: GCS amongst study population**

| Final Outcome | Frequency | Percent |
|---|---|---|
| Non Survivor | 16 | 25 |
| Survivor | 49 | 75 |
| Total | 65 | 100 |

**Table 14 – Final outcome amongst study population**

In the study population, 75% of the participants survived, while 25% did not survive.



**Graph 6 – Final outcome amongst study population**

| HDL level | Survivors | Non-survivors | Total | P value |
|---|---|---|---|---|
| HDL < 40 mg/dL | 14 (29%) | 14 (87%) | 28 (43.1%) | |
| HDL > 40 mg/dL | 35 (71%) | 2 (13%) | 37 (56.9%) | |
| Total | 49 (100%) | 16 (100%) | 65 (100%) | 0.001 |

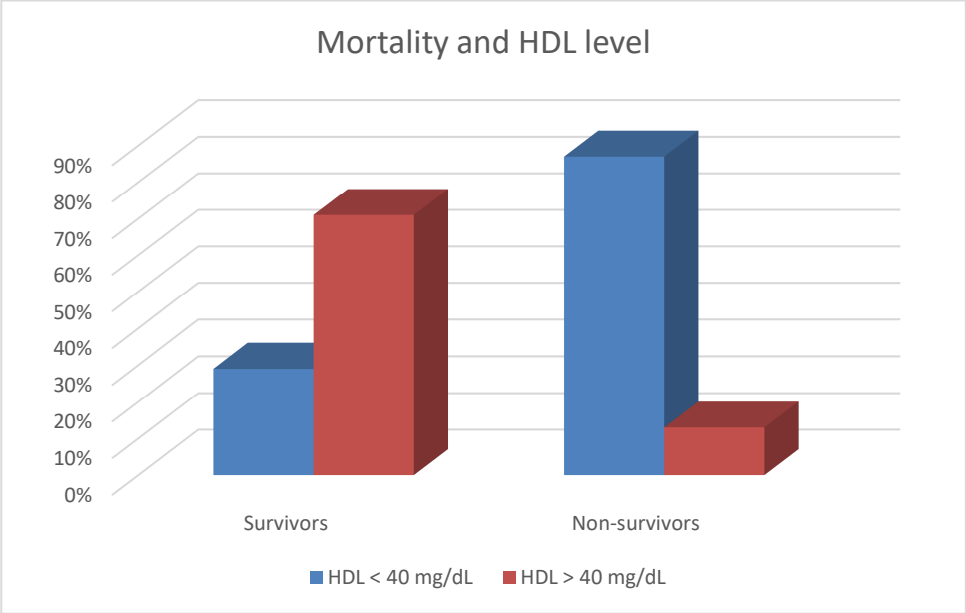
**Table 15 – Mortality and HDL level**

| | **Value** |
|---|---|
| **Sensitivity** | 87.50% |
| **Specificity** | 71.43% |
| **Positive Predictive Value (*)** | 50.00% |
| **Negative Predictive Value (*)** | 94.59% |
| **Accuracy (*)** | 75.38% |

**Table 15.1 – Sensitivity and Specificity of HDL cholesterol**

HDL levels were significantly associated with the outcome. Among the survivors, 29% had HDL levels below 40 mg/dL, whereas 71% above 40 mg/dL. Among the non-survivors, a striking 87% had below 40 mg/dL, and only 13% had above 40 mg/dL. The P value of 0.001 indicates a strong statistical significance in the difference of HDL levels between survivors and non-survivors.

The study data reveals that HDL levels significantly predict survival outcomes among 65 individuals, categorized as survivors (49) and non-survivors (16). With a sensitivity of 87.50%, the test effectively identifies true non-survivors, while a specificity of 71.43% indicates a good ability to recognize true survivors. The positive predictive value (PPV) is 50.00%, suggesting that half of those with HDL < 40 mg/dL are true non-survivors, while the negative predictive value (NPV) is 94.59%, indicating that most individuals with HDL > 40 mg/dL are true survivors. The overall accuracy of 75.38% demonstrates a fairly reliable predictive power. Clinically, individuals with HDL < 40 mg/dL should be considered high-risk and may need more aggressive monitoring, whereas those with HDL > 40 mg/dL are likely to have better outcomes, supporting the use of HDL levels in assessing survival predictions.

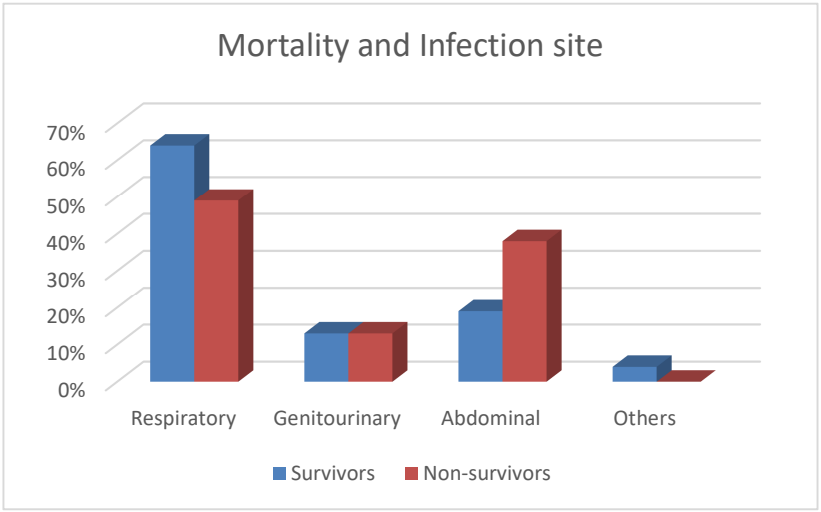



**Graph 7 – Mortality and HDL level**

| Infection site | Survivors | Non-survivors | Total | P value |
|---|---|---|---|---|
| Respiratory | 31 (64%) | 8 (49%) | 39 (60%) | |
| Genitourinary | 6 (13%) | 2 (13%) | 8 (12.3%) | |
| Abdominal | 9 (19%) | 6 (38%) | 15 (23.1%) | |
| Others | 3 (4%) | 0 (0%) | 3 (3.1%) | |
| Total | 49 (100%) | 16 (100%) | 65 (100%) | 0.001 |

**Table 16 – Mortality and Infection site**

The site of infection also showed a significant association with the outcome. Respiratory infections were the most common among both survivors (64%) and non-survivors (49%), with a P value of 0.001, indicating statistical significance. Abdominal infections were more prevalent among non-survivors (36%) compared to survivors (19%). Genitourinary infections and other sites showed similar proportions between survivors and non-survivors.

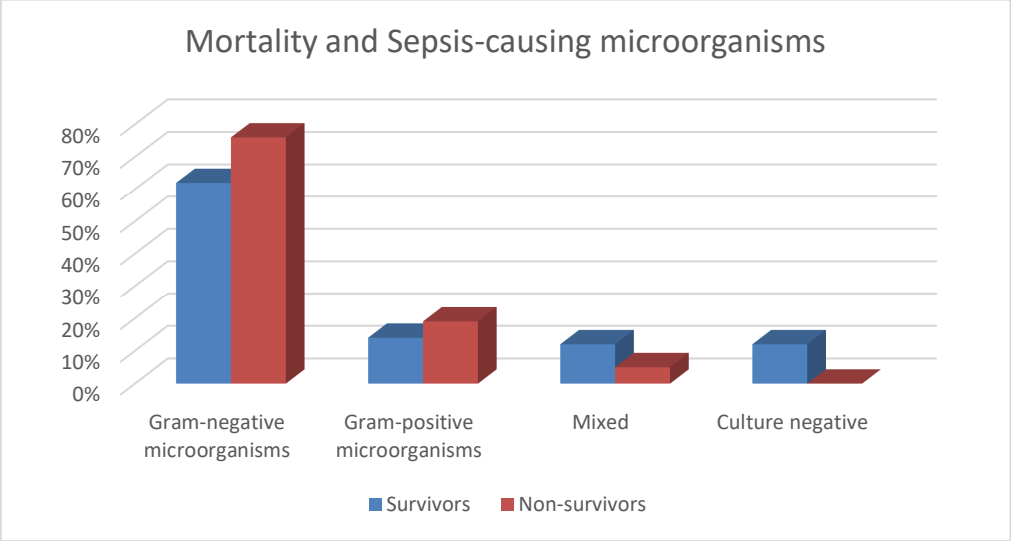



**Graph 8: Mortality and Infection site**

| Sepsis-causing microorganisms | Survivors | Non-survivors | Total | P value |
|---|---|---|---|---|
| Gram-negative microorganisms | 30 (62%) | 12 (76%) | 42 (64.6%) | |
| Gram-positive microorganisms | 7 (14%) | 3 (19%) | 10 (15.4%) | |
| Mixed | 6 (12%) | 1 (5%) | 7 (10.8%) | |
| Culture negative | 6 (12%) | 0 (0% ) | 6 (9.2%) | |
| Total | 49 (100%) | 16 (100%) | 65 (100%) | 0.001 |

**Table 17 – Mortality and Sepsis-causing microorganisms**

The type of microorganism causing sepsis had a significant impact on the outcome. Gram-negative microorganisms were the most common in both survivors (62%) and non-survivors (76%), with a P value of 0.001, indicating a strong association. Gram-positive microorganisms were more prevalent in non-survivors (24%) compared to survivors (14%). Mixed infections and culture-negative cases showed similar proportions between the two groups.

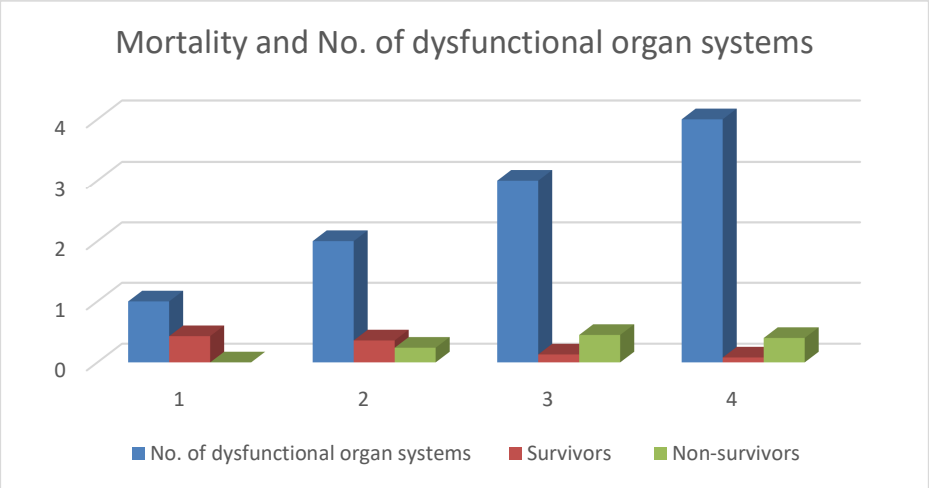



**Graph 9– Mortality and Sepsis-causing microorganisms**

| No. of dysfunctional organ systems | Survivors | Non-survivors | Total | P value |
|---|---|---|---|---|
| 1 | 21 (43%) | 0 (0%) | 21 (32.3%) | |
| 2 | 18 (36%) | 4 (24%) | 22 (33.8%) | |
| 3 | 6 (13%) | 7 (45%) | 13 (20%) | |
| 4 | 4 (8%) | 5 (40%) | 9 (13.8%) | |
| Total | 49 (100%) | 16 (100%) | 65 (100%) | 0.001 |

**Table 18 – Mortality and No. of dysfunctional organ systems**

The number of dysfunctional organ systems was significantly associated with survival. Among survivors, 43% had one dysfunctional organ system, while none of the non-survivors had only one. For two dysfunctional systems, 36% were survivors, compared to 24% non-survivors. The prevalence of three dysfunctional systems was higher in non-survivors (45%) than survivors (13%), and for four dysfunctional systems, it was 40% in non-survivors and 8% in survivors. The P value of 0.001 highlights the strong association.

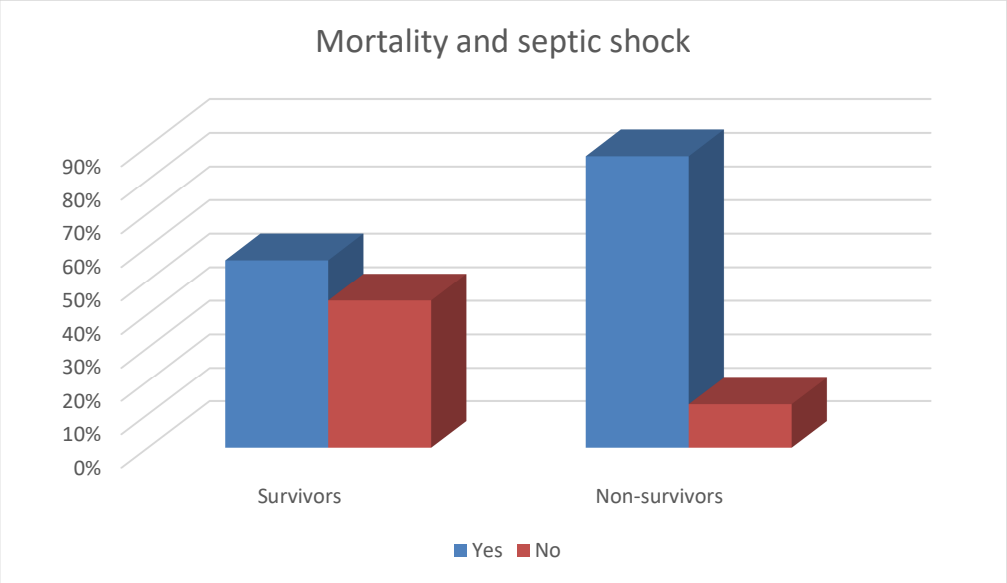



**Graph 10 – Mortality and No. of dysfunctional organ systems**

| Septic shock | Survivors | Non-survivors | Total | P value |
|---|---|---|---|---|
| Yes | 27 (56%) | 14 (87%) | 41 (63.1%) | |
| No | 22 (44%) | 2 (13%) | 24 (36.9%) | |
| Total | 49 (100%) | 16 (100%) | 65 (100%) | 0.001 |

**Table 19 – Mortality and septic shock**

Septic shock was significantly more common among non-survivors, with 87% experiencing it compared to 56% of survivors. Only 13% of non-survivors did not have septic shock, while 44% of survivors did not experience it. The P value of 0.001 indicates a strong statistical significance.




**Graph 11 – Mortality and septic shock**

| Lipid profile | | Survivors | Non-survivors | P value |
|---|---|---|---|---|
| TC | Day 1 | 122.76± 19.52 | 132.62 ± 14.23 | 0.001 |
| | Day 6 | 146.30 ± 53.15 | 124.09 ± 25.52 | 0.001 |
| HDL | Day 1 | 35.64 ± 3.84 | 40.14 ± 3.76 | 0.541 |
| | Day 6 | 42.39 ± 10.98 | 31.32 ± 5.23 | 0.001 |
| LDL | Day 1 | 64.25 ± 17.14 | 75.57±17.67 | 0.001 |
| | Day 6 | 77.88 ±23.34 | 68.33 ± 26.59 | 0.001 |
| TGL | Day 1 | 108.82±59.73 | 134.18 ± 95.79 | 0.001 |
| | Day 6 | 113.67±91.19 | 114.82±57.94 | 0.076 |
| VLDL | Day 1 | 18.97±10.89 | 24.52±13.95 | 0.001 |
| | Day 6 | 18.81±8.64 | 22.86±9.7 | 0.001 |

**Table 20: Analysis of lipid profile values among survivors and non survivors on first and sixth day.**

**Total Cholesterol (TC):** On Day 1, survivors had a mean TC of 122.76±19.52 mg/dL, whereas non-survivors had 132.62±14.23 mg/dL (P = 0.001). By Day 6, survivors' TC increased to 146.30±53.15 mg/dL, while non-survivors' TC decreased to 124.09±25.52 mg/dL (P = 0.001).

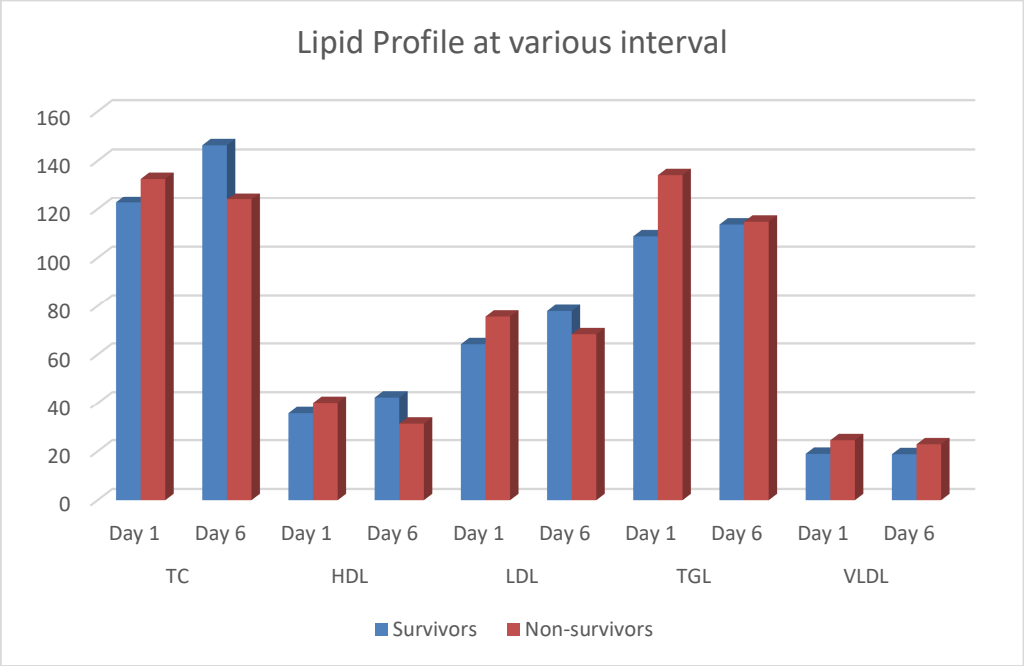
**HDL:** On Day 1, survivors had a mean HDL of 35.64±3.84 mg/dL, and non-survivors had 40.14±3.76 mg/dL (P = 0.541). By Day 6, survivors' HDL was 42.39±10.98 mg/dL, and non-survivors' HDL was 31.32±5.23 mg/dL (P = 0.001).

**LDL:** On Day 1, survivors had a mean LDL of 64.25±17.14 mg/dL, and non-survivors had 75.57±17.67 mg/dL (P = 0.001). By Day 6, survivors' LDL increased to

77.88±23.34 mg/dL, while non-survivors' LDL decreased to 68.33±26.59 mg/dL (P = 0.001).

**Triglycerides (TGL):** On Day 1, survivors had a mean TGL of 108.82±59.73 mg/dL, and non-survivors had 134.18±95.79 mg/dL (P = 0.001). By Day 6, the levels were similar, with survivors at 113.67±91.19 mg/dL and non-survivors at 114.82±57.94 mg/dL (P = 0.076).

**VLDL:** On Day 1, survivors had a mean VLDL of 18.97±10.89 mg/dL, and non-survivors had 24.52±13.95 mg/dL (P = 0.001). By Day 6, survivors' VLDL was 18.81±8.64 mg/dL, and non-survivors' VLDL was 22.86±9.7 mg/dL (P = 0.001).




**Graph 12 Analysis of lipid profile values among survivors and non survivors on first and sixth day.**

| Time Point | Correlational Coefficient | p-value |
|---|---|---|
| On admission | -0.59 | 0.01 |
| On day 6 | -0.66 | 0.001 |
| Survivors D0 | -0.69 | 0.02 |
| Survivors D6 | -0.55 | 0.03 |
| Non-survivors D0 | -0.39 | 0.01 |
| Non-survivors D6 | -0.47 | 0.01 |

The analysis reveals a consistent, significant negative correlation between HDL levels and SOFA scores at various time points, indicating that lower HDL levels are associated with higher SOFA scores and worse organ function. On admission, the correlation is -0.59 ($p = 0.01$), strengthening to -0.66 ($p = 0.001$) by day 6. Among survivors, the correlation is -0.69 ($p = 0.02$) on day 0 and -0.55 ($p = 0.03$) on day 6. For non-survivors, the correlation is -0.39 ($p = 0.01$) on day 0 and -0.47 ($p = 0.01$) on day 6.

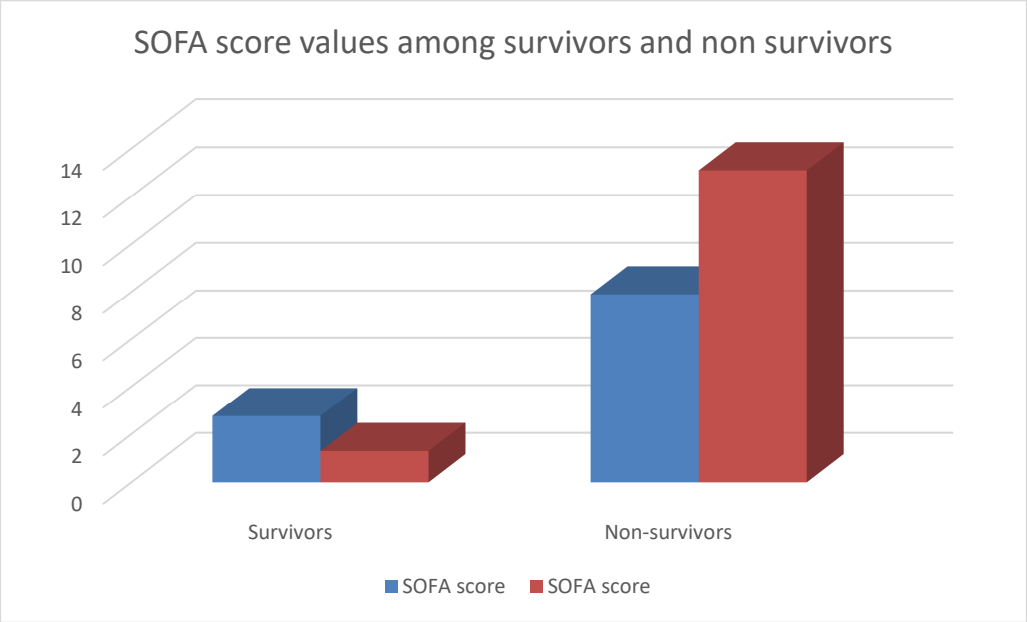
| Various parameters | Mean | Std. Deviation |
|---|---|---|
| Heart rate | 111.75 | 21.335 |
| Systolic BP | 115.23 | 11.149 |
| Diastolic BP | 82.22 | 7.8 |
| Respiratory rate | 31.83 | 6.319 |
| Total Leucocyte Count | 18625.39 | 10586.765 |
| Platelet Count | 261973.51 | 149647.741 |

**Table 21- Various other parameters**

| SOFA score | | Survivors | Non-survivors | P value |
|---|---|---|---|---|
| SOFA score | Day 1 | 2.76 ± 0.7 | 7.9 ± 1.4 | 0.001 |
| | Day 6 | 1.30 ± 0.1 | 13.09 ± 2.3 | 0.001 |

**Table 22: Analysis of SOFA score values among survivors and non survivors on first and sixth day.**

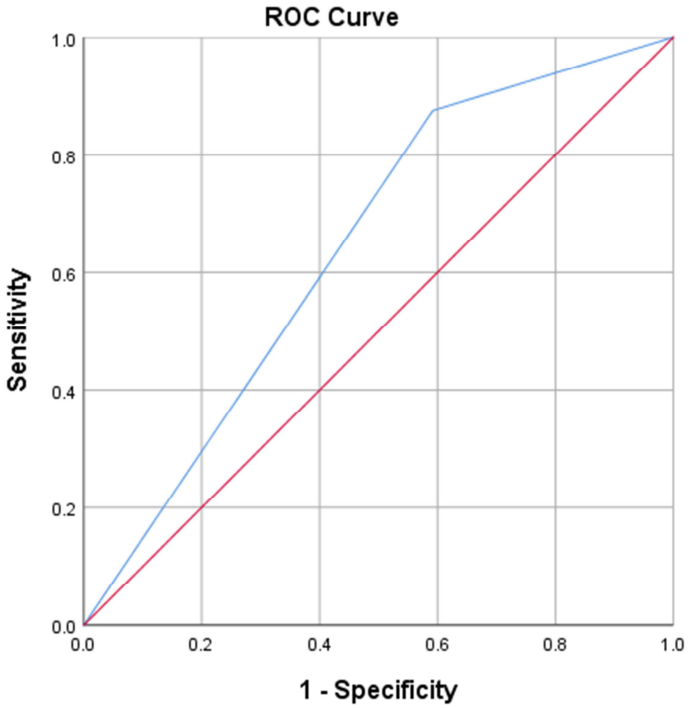
On Day 1, survivors had a mean SOFA score of 2.76 ± 0.7 whereas non-survivors had 7.9 ± 1.4 (P = 0.001). By Day 6, survivors's SOFA score decreased to 1.30 ± 0.1, while non-survivors's SOFA score increased to 13.09 ± 2.3 (P = 0.001).




**Graph 13: Analysis of SOFA score values among survivors and non survivors on first and sixth day.**

| **Area Under the Curve** | | | | |
|---|---|---|---|---|
| Test Result Variable(s): HDL level and Mortality | | | | |
| Area | Std. Error[a] | Asymptotic Sig.[b] | Asymptotic 95% Confidence Interval | |
| | | | Lower Bound | Upper Bound |
| .642 | .074 | .091 | .496 | .787 |
| The test result variable(s): HDL level has at least one tie between the positive actual state group and the negative actual state group. Statistics may be biased. | | | | |
| a. Under the nonparametric assumption | | | | |
| b. Null hypothesis: true area = 0.5 | | | | |

The AUC analysis for HDL level and SOFA score, with an AUC of 0.642 and a standard error of 0.074, indicates a fair ability to discriminate between positive and negative states. However, the p-value of 0.091 shows that this result is not statistically significant, as it exceeds the 0.05 threshold. The 95% confidence interval (0.496 to 0.787) includes 0.5, further indicating no significant discriminative ability. Additionally, the presence of ties between the positive and negative state groups may bias the statistics.




Diagonal segments are produced by ties.

# DISCUSSION

Sepsis is characterized by the presence or suspicion of an infection together with signs of a systemic reaction known as the systemic inflammatory response syndrome. When at least two of the following symptoms are present, it is known as systemic inflammatory response syndrome: (1) core body temperature (B.B.C.): 38–100.4°F or 36–96.8°F; (2) pulse rate: 90 beats/min; (3) respiratory rate: 20 breaths/min (or PaCO2: 32 torr); and (4) white blood cell count: 12,000–4,000/mm3 or 10% immature band develops. 131 The presence of sepsis in conjunction with the malfunction of one or more organs is what defines severe sepsis. Hypoperfusion with lactic acidosis, altered mental status, acute lung damage, coagulation abnormalities, thrombocytopenia, and hepatic, renal, or cardiac failure are all symptoms of organ dysfunction. 132.133

The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3) provide the following definitions and criteria:

Sepsis is characterized as life-threatening organ dysfunction caused by a dys-regulated host response to infection. Organ dysfunction is indicated by a sudden increase in the overall SOFA score by more than two points due to the infection. When the baseline SOFA score is unknown, it is assumed to be 0. A SOFA score of less than two signifies a roughly 10% mortality risk among patients with suspected infections in general hospital settings. It is crucial to emphasize the urgency of prompt and appropriate care, as patients initially presenting with mild dysfunction can deteriorate rapidly.

In simpler terms, sepsis is a potentially deadly condition that arises when the body's response to an infection causes damage to its own tissues and organs.

At the bedside, patients suspected of having infections and at high risk of mortality or requiring extended ICU stays can be rapidly identified using qSOFA criteria: altered mental status, systolic blood pressure ≤100 mm Hg, or respiratory rate ≥22/min.

Septic shock, a severe form of sepsis, occurs when circulatory and cellular/metabolic abnormalities are severe enough to significantly increase mortality rates. It is recognized by persistent hypotension requiring vasopressors to maintain a mean arterial pressure (MAP) of ≥65 mm Hg and a serum lactate level >2 mmol/L (18 mg/dL) despite adequate volume resuscitation. Hospital mortality rates exceed 40% with these criteria.

**Age group**

The age distribution of the study population shows that the majority of participants fall within the age range of 51 to 60 years, representing 45% of the total. The second largest age group is 41 to 50 years, making up 30% of the participants. Those aged more than 60 years account for 17%, while the smallest group, those less than 40 years, comprise 8%. In the study done by Hernández et al., average patient's age was 61 years with a range of 18 to 87 years. Similarly in the study conducted by Fabián Jaimes et al., the septicemia rate increased for all age groups, the increase was greatest (162%) for persons aged 65 years or older, from 326.3 per 100 000 in 1979 to 854.7 per 100 000 by 1987. 134,135

**Gender**

There is a notable gender disparity in the study population, with males constituting a significant majority at 68%, compared to females who make up 32%. This finding correlate well with the study conducted by Offner et al. who identified male gender as an independent risk factor for the development of severe infection in surgical patients. 136 Similarly, McGowan et al. also reported a significantly higher incidence of bacteremic infections in males compared with females.137 Female genders have been demonstrated to be protective under such conditions, whereas male gender may be deleterious due to a diminished cell-mediated immune response and cardiovascular functions. Male sex hormones, i.e., androgens, have been shown to be suppressive on cell-mediated immune responses. In contrast, female sex hormones exhibit protective effects which may contribute to the natural advantages of females under septic conditions.

Regarding smoking habits, a significant majority of the participants, 78%, reported not smoking. Only 22% of the participants indicated that they are smokers.

**Site of infection**

The most common site of infection among the participants is the respiratory system, affecting 60% of the study population. Abdominal infections are the second most common, accounting for 23.1%. Genitourinary infections are present in 12.3% of the participants. A small proportion, 4.6%, have infections at other sites. Similarly in the study conducted by S Martin 138 and Esper AM et al.,139 in which respiratory infections are invariably the most common cause of sepsis, severe sepsis and septic shock. Overall, respiratory infections account for approximately half of all cases of

sepsis. The next most common causes are genitourinary and abdominal sources of infection with primary bacteremia and unknown sources being the next most common causes. This findings correlate well with the study conducted by Annette M. Esper et al.,139 in which 33% of sepsis cases were due to respiratory infections, 32% to genitourinary infections, 23% to a gastrointestinal source, 7% to a bone or joint infection, 5% to a skin or soft tissue infection, and 3% to other sources; 3% of infections involved more than one source.

The Glasgow Coma Scale scores show a distribution where the majority of participants (62.5%) have scores between 6 to 10, indicating moderate to severe impairment. 33.5% of the participants have higher GCS scores ranging from 11 to 15, which suggests mild impairment or full consciousness. Only 4% of the participants have the lowest GCS scores between 3 to 5, indicating severe impairment or coma.

**HDL level**

HDL levels were significantly associated with the outcome. Among the survivors, 29% had HDL levels below 40 mg/dL, whereas 71% had above 40 mg/dL. Among the non-survivors, a striking 87% had below 40 mg/dL, and only 13% had above 40 mg/dL. The P value of 0.001 indicates a strong statistical significance in the difference of HDL levels between survivors and non-survivors.

The site of infection also showed a significant association with the outcome. Respiratory infections were the most common among both survivors (64%) and non-survivors (49%), with a P value of 0.001, indicating statistical significance. Abdominal infections were more prevalent among non-survivors (36%) compared to

survivors (19%). Genitourinary infections and other sites showed similar proportions between survivors and non-survivors.

The type of microorganism causing sepsis had a significant impact on the outcome. Gram-negative microorganisms were the most common in both survivors (62%) and non-survivors (76%), with a P value of 0.001, indicating a strong association. Gram-positive microorganisms were more prevalent in non-survivors (24%) compared to survivors (14%). Mixed infections and culture-negative cases showed similar proportions between the two groups.

The number of dysfunctional organ systems was significantly associated with survival. Among survivors, 43% had one dysfunctional organ system, while none of the non-survivors had only one. For two dysfunctional systems, 36% were survivors, compared to 24% non-survivors. The prevalence of three dysfunctional systems was higher in non-survivors (45%) than survivors (13%), and for four dysfunctional systems, it was 40% in non-survivors and 8% in survivors. The P value of 0.001 highlights the strong association.

Septic shock was significantly more common among non-survivors, with 87% experiencing it compared to 56% of survivors. Only 13% of non-survivors did not have septic shock, while 44% of survivors did not experience it. The P value of 0.001 indicates a strong statistical significance.

**Lipid profile**

**Total Cholesterol (TC):** On Day 1, survivors had a mean TC of 122.76±19.52 mg/dL, whereas non-survivors had 132.62±14.23 mg/dL (P = 0.001). By Day 6,

survivors' TC increased to 146.30±53.15 mg/dL, while non-survivors' TC decreased to 124.09±25.52 mg/dL (P = 0.001).

**HDL:** On Day 1, survivors had a mean HDL of 35.64±3.84 mg/dL, and non-survivors had 40.14±3.76 mg/dL (P = 0.541). By Day 6, survivors' HDL was 42.39±10.98 mg/dL, and non-survivors' HDL was 31.32±5.23 mg/dL (P = 0.001).

**LDL:** On Day 1, survivors had a mean LDL of 64.25±17.14 mg/dL, and non-survivors had 75.57±17.67 mg/dL (P = 0.001). By Day 6, survivors' LDL increased to 77.88±23.34 mg/dL, while non-survivors' LDL decreased to 68.33±26.59 mg/dL (P = 0.001).

**Triglycerides (TGL):** On Day 1, survivors had a mean TGL of 108.82±59.73 mg/dL, and non-survivors had 134.18 ± 95.79 mg/dL (P = 0.001). By Day 6, the levels were similar, with survivors at 113.67 ± 91.19 mg/dL and non-survivors at 114.82 ± 57.94 mg/dL (P = 0.076).

**VLDL:** On Day 1, survivors had a mean VLDL of 18.97 ± 10.89 mg/dL, and non-survivors had 24.52 ± 13.95 mg/dL (P = 0.001). By Day 6, survivors' VLDL was 18.81 ± 8.64 mg/dL, and non-survivors' VLDL was 22.86±9.7 mg/dL (P = 0.001). Similarly, Jeyasuriya et al. discovered that the survival group's mean HDL levels were 31 and 36 mg% at days 0 and 3, whereas the non-survival group's levels were 33 and 31 mg% with a non-significant p value of 0.137. The difference was statistically significant at 0.033.14. Similarly, Naresh et al. found that survivors had an average HDL value of 11.8 mg%, compared to 5.8% for non-survivors.9 The mean HDL levels of patients with sepsis were 52.17 mg/dl, those with severe sepsis were 41.81 mg/dl, and those without severe sepsis were 40.08 mg/dl, according to a research done

in Kerala by Sunayana P et al. Although there was no statistically significant difference between the groups, 11.8% of patients with HDL levels over 50 mg/dl and 32.1% of patients with HDL levels below 50 mg/dl died. (p value 0.1).137

**SOFA Score**

In this study, SOFA score was used as an sepsis score and is correlated along with HDL-C levels at various times of hospital stay and it showed a statistically significant. relationship even among the survivors (with correlation of coefficient of -0.63 and p value of < 0.001 on admission and -0.50 and on admission and -0.50 and at day 6. Using the same APACHE 2 score, a related research by Naresh et al. found a statistically significant connection of -450 among survivors and -163 among non-survivors (p = 0.003).138

In the present study, On Day 1, survivors had a mean SOFA score of 2.76 ± 0.7 whereas non-survivors had 7.9 ± 1.4 (P = 0.001). By Day 6, survivors' TC decreased to 1.30 ± 0.1, while non-survivors' TC increased to 13.09 ± 2.3 (P = 0.001).

In the present study, the analysis reveals a consistent, significant negative correlation between HDL levels and SOFA scores at various time points, indicating that lower HDL levels are associated with higher SOFA scores and worse organ function. On admission, the correlation is -0.59 (p = 0.01), strengthening to -0.66 (p = 0.001) by day 6. Among survivors, the correlation is -0.69 (p = 0.02) on day 0 and -0.55 (p = 0.03) on day 6. For non-survivors, the correlation is -0.39 (p = 0.01) on day 0 and -0.47 (p = 0.01) on day 6.

# CONCLUSION

- There is strong evidence linking sepsis with serum HDL (high-density lipoprotein) levels in patients. Those with sepsis and significant organ dysfunction typically exhibit lower HDL levels. Therefore, serial monitoring of HDL levels alongside Sequential Organ Failure Assessment (SOFA) scores is recommended for better prognostication and clinical outcomes in sepsis patients, rather than relying on a single measurement.
- High initial or increasing HDL levels may indicate a better prognosis and higher survival rates in sepsis patients. Conversely, very low HDL levels or a declining trend are associated with poorer outcomes and survival.
- HDL can serve as a valuable prognostic marker in sepsis patients and may complement other biomarkers such as Procalcitonin (PCT) ,Interleukin-6 (IL-6) and other biomarkers for enhanced diagnostic and prognostic accuracy.

**Limitations:**

- The sample size is small, which limits the generalizability of the results to broader populations.
- HDL cholesterol may be less reliable in patients with inflammatory disorders, those taking statins, individuals with diabetes, thyroid disorders, and other conditions affecting lipid metabolism.
- Further research with larger sample sizes is necessary to robustly establish the utility of HDL as a prognostic marker in sepsis patients.

In summary, while HDL shows promise as a prognostic indicator in sepsis, its clinical utility needs to be confirmed through larger studies that account for various patient factors and conditions affecting lipid metabolism.

# SUMMARY

- The age distribution of the study population indicates that the majority of participants are within the 51 to 60 years age range, comprising 45% of the total. The next largest age group is 41 to 50 years, representing 30% of the participants. Those over 60 years make up 17%, while the smallest group, those under 40 years, constitute 8%.

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- Regarding the Glasgow Coma Scale (GCS) scores, 62.5% of participants have scores between 6 and 10, indicating moderate to severe impairment. About 33.5% have higher scores ranging from 11 to 15, suggesting mild impairment or full consciousness, while 4% have the lowest scores between 3 and 5, indicating severe impairment or coma.

- HDL levels show a significant association with the outcome. Among survivors, 29% had HDL levels below 40 mg/dL, while 71% had levels above 40 mg/dL. Conversely, among non-survivors, 87% had HDL levels below 40 mg/dL, and only 13% had levels above 40 mg/dL. The P value of 0.001 indicates a strong statistical significance in the difference in HDL levels between survivors and non-survivors.

- The site of infection also significantly correlates with the outcome. Respiratory infections are most common among both survivors (64%) and non-survivors (49%), with a P value of 0.001. Abdominal infections are more prevalent among non-survivors (36%) compared to survivors (19%). Genitourinary infections and other sites show similar proportions between survivors and non-survivors.

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**ANNEXURE-I**

**KAHERS JNMC BELAGAVI**

**INFORMED CONSENT FORM**

**"CORRELATION OF SERUM HDL WITH SOFA SCORE IN SEPSIS AND ITS PROGNOSTIC VALUE IN PATIENTS ADMITTED IN INTENSIVE CARE UNIT AT KLE DR. PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELAGAVI – A PROSPECTIVE COHORT STUDY"**

Name of Student/Principal Investigator:Dr ARUSH VEDWA

Name of Guide/Co Investigators:Dr Raju.H.Badiger

Objective: Primary -To determine correlation of Serum HDL with SOFA score in sepsis

Introduction: Sepsis is a significant public health problem.Sepsis affects persons of all ages is the leading cause of morbidity and mortality for patients admitted to an intensive care unit (ICU). Despite advances in aggressive management, a diagnosis of sepsis has a high mortality rate, which increases from 20.8% in patients with sepsis and 48.8% in patients with septic shock. Recent evidence suggests that serum High density lipoprotein cholesterol (HDL-C) may be a useful prognostic marker of sepsis since cholesterol metabolism has been shown to be markedly influenced by widespread inflammation secondary to bacteremia.

Explanation of procedure: Patients above the age of 18 years of age will be included in the study.

This is a prospective observational study, which will be conducted at KLE Dr. Prabhakar Kore Hospital And Medical Research Centre, Belagavi . The patients diagnosed to be in sepsis will be the study participants. Whenever patient is diagnosed to be in sepsis, those patients will be contacted immediately. Details of the study will be explained in detail. Permission is sought in the form of informed written consent and the study was conducted. After Institutional Ethical Committee approval all the patients diagnosed to have sepsis in ICU of this Hospital will be recruited for the study. Venous Blood sample and arterial blood sample from each patient will be taken for HDL-C cholesterol levels(subset of lipid profile), creatinine, bilirubin levels, and platelet levels at the time of admission and on the day 6 after admission. Respiratory status by Pao2 and Fi02 and Central nervous system assessed by Glasgow Coma Scale and SOFA scores were calculated accordingly on day of admission and day 6. The HDL-cholesterol measurement was done by clinical chemistry automated analyzer.

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: 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. "CORRELATION OF SERUM HDL WITH SOFA SCORE IN SEPSIS AND ITS PROGNOSTIC VALUE IN PATIENTS ADMITTED IN INTENSIVE CARE UNIT AT KLE DR. PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELAGAVI – A PROSPECTIVE COHORT 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:

Signature of the investigator:

**ANNEXURE-II**

**PROFORMA**

Research study –

"Correlation of Serum HDL with SOFA score in sepsis and its prognostic value in Patients Admitted In Intensive Care Unit At KLE Dr. Prabhakar Kore Hospital And Medical Research Centre, Belagavi – A prospective cohort Study".

| | |
|---|---|
| CASE NO | |
| NAME | |
| IP NO/OP NO | |
| AGE | YEARS |
| SEX | MALE FEMALE |
| ADDRESS | |
| OCCUPATION | |

| | |
|---|---|
| Complaints at presentation | |
| Past history | |
| Family history | |
| Personal history | |

| | |
|---|---|
| Treatment history | |

| | Yes | No |
|---|---|---|
| **Pallor** | | |
| **Icterus** | | |
| **Lymphadenopathy** | | |
| **Cyanosis** | | |
| **Clubbing** | | |
| **Edema** | | |

Vitals :

| | |
|---|---|
| Temperature | |
| Pulse | |
| Respiratory rate and spo2 and Fio2 | |
| Blood pressure | |
| GCS | |

PHYSICAL EXAMINATION:

SYSTEMIC EXAMINATION:

| | |
|---|---|
| **C.V.S** | |
| **R.S.** | |
| **C.N.S** | |
| **PER ABDOMEN** | |

Q-SOFA score at admission-

INVESTIGATIONS: On admission

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| CBC | TLC | DLC<br><br>N/L/E/B | PLT | | | | | |
| LFT | TB | DB | AST | ALT | TP | ALP | ALB | GLB |
| RFT | UREA | CREAT | BUN | Na | K | | | |

| ABG | pH | PaO2 | PaCO2 | SO2 | HCO3- | | | |
|---|---|---|---|---|---|---|---|---|

PHYSICAL EXAMINATION:

SYSTEMIC EXAMINATION:

| | |
|---|---|
| **C.V.S** | |
| **R.S.** | |
| **C.N.S** | |
| **PER ABDOMEN** | |

Q-SOFA score at admission-

INVESTIGATIONS: On admission

| CBC | TLC | DLC<br><br>N/L/E/B | PLT | | | | | |
|---|---|---|---|---|---|---|---|---|

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| LFT | TB | DB | AST | ALT | TP | ALP | ALB | GLB |
| RFT | UREA | CREAT | BUN | Na | K | | | |
| ABG | pH | PaO2 | PaCO2 | SO2 | HCO3- | | | |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| BLOOD C/S | | | | | | | | |
| URINE C/S | | | | | | | | |
| URINE R/M | | | | | | | | |
| SG ABD | | | | | | | | |
| CHEST XRAY | | | | | | | | |
| HDL-c | TC | TG | LDL | | | | | |
| RBS | | | | | | | | |
| PCT | | | | | | | | |

SOFA SCORE -

| **SOFA Parameters** | ADMISSION | DAY 6 |
|---|---|---|
| **Pao2/Fi02** | | |
| **PLT** | | |
| **TB** | | |
| **BP** | | |
| **GCS** | | |
| **S.creat** | | |
| **SOFA SCORE** | | |
| **HDL-c** | | |

FOCI OF SEPSIS -

| | |
|---|---|
| Respiratory | |
| Genitourinary | |
| Abdominal | |
| C.N.S | |
| Skin | |
| Others | |

# ANNEXURE-III MASTER CHART

| Sr No | AGE | age group | SEX | Smoking habit | HR | SBP | DBP | Mean arterial pressure | RR | CREAT | PLATELET COUNT | $PaO_2/FiO_2$ [mmHg (kPa)] | Bilirubin (mg/dl) [µmol/L] | HDL | HDL | T.C | LDL | TG | GCS | SBP day 6 | DBP day 6 | DAY 6 Mean arterial pressure | RR day 6 | TEMP | TLC | VENTILATION | Day 6 CREAT | day 6 PLATELET COUNT | Day 6 $PaO_2/FiO_2$ [mmHg (kPa)] | Day 6 Bilirubin (mg/dl) [µmol/L] | GCS day 6 | Septic shock | TC Day 6 | DAY 6 LDL | Day 6 HDL | day 6 TG | Outcome | Outcome | Diagnosis | Sepsis-causing microorganisms | No. of dysfunctional organ systems |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 44 | years | Male | No | 102 | 136 | 78 | 97 | 24 | 0.77 | 160000 | 301 | 0.7 | 33 | 1 | 248 | 145 | 157 | 13 | 136 | 82 | | 26 | 99 | 4300 | YES | 0.67 | 110000 | 178 | 0.7 | 14 | no | 240 | 131 | 36 | 152 | 0 | Survivor | Others | Culture negative | 1 |
| 2 | 66 | than 60 | Male | yes | 98 | 132 | 76 | 95 | 16 | 0.6 | 568000 | 300 | 0.3 | 37 | 1 | 255 | 157 | 167 | 14 | 80 | 60 | 99 | 18 | 98 | 21300 | No | 0.5 | 518000 | 177 | 0.3 | 15 | YES | 238 | 141 | 40 | 167 | 0 | Survivor | al | Gram-negative | 2 |
| 3 | 64 | than 60 | Male | No | 110 | 132 | 78 | 96 | 20 | 11.3 | 504000 | 198 | 2.3 | 34 | 1 | 238 | 164 | 194 | 3 | 132 | 86 | 101 | 22 | 99 | 65300 | YES | 12.7 | 192729 | 125 | 2.6 | 3 | no | 246 | 167 | 37 | 197 | 1 | vor | ory | Gram-negative | 3 |
| 4 | 30 | 40 years | Male | No | 140 | 134 | 76 | 95 | 18 | 0.92 | 33000 | 311 | 0.5 | 31 | 1 | 187 | 144 | 134 | 12 | 134 | 98 | 110 | 20 | 102 | 27820 | YES | 0.82 | 377626 | 188 | 0.5 | 13 | no | 190 | 142 | 34 | 144 | 0 | Survivor | ory | Gram-negative | 1 |
| 5 | 70 | than 60 | Male | No | 130 | 128 | 78 | 95 | 22 | 2.2 | 74000 | 187 | 3.1 | 33 | 1 | 230 | 144 | 144 | 3 | 80 | 70 | 108 | 24 | 100.6 | 14740 | YES | 3.63 | 24000 | 114 | 3.4 | 4 | YES | 236 | 150 | 36 | 150 | 1 | vor | ory | Gram-negative | 4 |
| 6 | 45 | years | Male | yes | 100 | 134 | 78 | 97 | 18 | 0.8 | 297000 | 303 | 0.7 | 35 | 1 | 200 | 148 | 148 | 15 | 78 | 60 | 102 | 20 | 99 | 17820 | No | 0.7 | 247000 | 180 | 0.7 | 15 | YES | 198 | 146 | 38 | 142 | 0 | Survivor | inary | Mixed | 3 |
| 7 | 69 | than 60 | female | No | 142 | 130 | 78 | 95 | 20 | 0.86 | 146000 | 302 | 1 | 37 | 1 | 255 | 159 | 191 | 15 | 68 | 50 | 101 | 22 | 98 | 22610 | No | 0.76 | 96000 | 179 | 1 | 15 | YES | 239 | 152 | 40 | 168 | 0 | Survivor | ory | Gram-positive | 1 |
| 8 | 58 | years | female | No | 112 | 132 | 80 | 97 | 16 | 0.6 | 446000 | 305 | 0.7 | 35 | 1 | 264 | 165 | 228 | 15 | 132 | 90 | 104 | 18 | 100 | 33100 | No | 0.5 | 396000 | 182 | 0.7 | 15 | YES | 254 | 147 | 38 | 215 | 0 | Survivor | ory | Gram-negative | 2 |
| 9 | 46 | years | female | No | 110 | 120 | 78 | 92 | 16 | 0.9 | 223000 | 296 | 0.8 | 33 | 1 | 278 | 171 | 290 | 15 | 120 | 82 | 95 | 18 | 101.6 | 16350 | No | 0.8 | 173000 | 173 | 0.8 | 15 | no | 260 | 161 | 36 | 322 | 0 | Survivor | ory | Gram-negative | 1 |
| 10 | 61 | than 60 | Male | No | 120 | 134 | 80 | 98 | 20 | 0.9 | 375000 | 311 | 0.7 | 39 | 1 | 160 | 165 | 184 | 8 | 134 | 98 | 110 | 22 | 101 | 38570 | YES | 0.8 | 325000 | 188 | 0.7 | 9 | no | 268 | 167 | 42 | 188 | 0 | Survivor | inary | Mixed | 1 |
| 11 | 73 | than 60 | Male | No | 90 | 128 | 78 | 95 | 16 | 0.36 | 63000 | 298 | 0.6 | 37 | 1 | 144 | 144 | 155 | 11 | 76 | 60 | 97 | 18 | 98.6 | 17700 | YES | 0.26 | 113000 | 146 | 0.6 | 12 | YES | 138 | 141 | 40 | 150 | 0 | Survivor | inary | Mixed | 2 |
| 12 | 47 | years | female | No | 110 | 128 | 74 | 92 | 25 | 0.88 | 188000 | 305 | 0.4 | 40 | 1 | 178 | 131 | 144 | 15 | 128 | 92 | 104 | 27 | 102 | 10400 | No | 0.78 | 138000 | 153 | 0.4 | 15 | YES | 168 | 147 | 43 | 148 | 0 | Survivor | ory | Gram-negative | 3 |
| 13 | 56 | years | female | No | 100 | 132 | 82 | 99 | 18 | 1.22 | 234000 | 310 | 0.3 | 37 | 1 | 200 | 133 | 171 | 15 | 132 | 98 | 109 | 20 | 102 | 2490 | No | 1.12 | 184000 | 158 | 0.3 | 15 | no | 220 | 129 | 40 | 161 | 0 | Survivor | inary | Mixed | 1 |
| 14 | 48 | years | female | No | 140 | 144 | 82 | 103 | 22 | 4.1 | 180000 | 186 | 4.2 | 39 | 1 | 254 | 154 | 200 | 3 | 70 | 66 | 106 | 24 | 105 | 5980 | YES | 5.51 | 130000 | 113 | 4.5 | 3 | YES | 234 | 132 | 42 | 172 | 1 | vor | al | Gram-negative | 4 |
| 15 | 49 | years | Male | yes | 112 | 144 | 76 | 99 | 18 | 3.3 | 265073 | 198 | 2.4 | 43 | 0 | 110 | 143 | 141 | 4 | 134 | 84 | 101 | 20 | 101 | 33700 | YES | 4.72 | 105344 | 125 | 2.7 | 4 | no | 114 | 143 | 46 | 98 | 1 | vor | ory | Gram-positive | 3 |
| 16 | 58 | years | female | No | 78 | 144 | 76 | 99 | 22 | 1.64 | 217000 | 306 | 0.8 | 36 | 1 | 165 | 156 | 144 | 15 | 70 | 66 | 105 | 24 | 102.6 | 25180 | No | 1.54 | 167000 | 154 | 0.8 | 15 | YES | 243 | 155 | 39 | 124 | 0 | Survivor | inary | Mixed | 1 |
| 17 | 71 | than 60 | Male | yes | 110 | 144 | 76 | 99 | 24 | 0.68 | 336000 | 301 | 0.7 | 40 | 1 | 198 | 128 | 112 | 15 | 132 | 84 | 100 | 26 | 99 | 15370 | No | 0.58 | 286000 | 149 | 0.7 | 15 | no | 212 | 144 | 43 | 144 | 0 | Survivor | ory | Gram-positive | 1 |
| 18 | 47 | years | female | No | 98 | 132 | 80 | 97 | 16 | 0.86 | 27000 | 298 | 0.3 | 35 | 1 | 236 | 139 | 144 | 14 | 58 | 40 | 97 | 18 | 100 | 12600 | YES | 0.76 | 77000 | 146 | 0.3 | 15 | YES | 240 | 141 | 38 | 134 | 0 | Survivor | ory | Gram-negative | 2 |
| 19 | 45 | years | Male | No | 114 | 132 | 78 | 96 | 18 | 1.01 | 341000 | 300 | 0.8 | 33 | 1 | 232 | 136 | 145 | 15 | 76 | 58 | 99 | 20 | 98 | 14870 | No | 0.91 | 291000 | 148 | 0.8 | 15 | YES | 230 | 132 | 36 | 145 | 0 | Survivor | ory | Gram-negative | 3 |
| 20 | 45 | years | Male | No | 90 | 132 | 78 | 96 | 16 | 1.11 | 134000 | 308 | 0.6 | 34 | 1 | 194 | 132 | 165 | 12 | 132 | 94 | 107 | 18 | 98.6 | 7800 | YES | 1.01 | 84000 | 156 | 0.6 | 13 | no | 189 | 129 | 37 | 156 | 0 | Survivor | ory | Gram-negative | 1 |
| 21 | 50 | years | Male | yes | 100 | 134 | 72 | 93 | 20 | 0.42 | 269000 | 304 | 0.7 | 32 | 1 | 196 | 140 | 132 | 8 | 134 | 88 | 103 | 22 | 99 | 20260 | YES | 0.32 | 219000 | 205 | 0.7 | 9 | no | 190 | 141 | 35 | 161 | 0 | Survivor | ory | Gram-positive | 1 |
| 22 | 43 | years | female | No | 110 | 140 | 78 | 99 | 20 | 2.1 | 326781 | 180 | 2.6 | 35 | 1 | 188 | 129 | 150 | 4 | 70 | 64 | 101 | 22 | 98 | 24370 | YES | 2.96 | 167052 | 107 | 2.9 | 3 | YES | 233 | 122 | 38 | 148 | 1 | vor | ory | Gram-positive | 4 |
| 23 | 63 | than 60 | Male | yes | 100 | 130 | 78 | 95 | 16 | 0.38 | 322000 | 298 | 1.2 | 39 | 1 | 150 | 112 | 123 | 12 | 60 | 56 | 97 | 18 | 98.6 | 9780 | YES | 0.28 | 272000 | 199 | 1.2 | 13 | YES | 145 | 98 | 42 | 121 | 0 | Survivor | ory | Gram-negative | 2 |
| 24 | 66 | than 60 | female | No | 98 | 134 | 78 | 97 | 22 | 0.56 | 356000 | 304 | 0.7 | 37 | 1 | 250 | 159 | 142 | 15 | 128 | 90 | 103 | 24 | 99.4 | 18720 | No | 0.46 | 306000 | 205 | 0.7 | 15 | no | 245 | 154 | 40 | 164 | 0 | Survivor | al | Gram-negative | 1 |
| 25 | 44 | years | Male | yes | 120 | 140 | 78 | 99 | 24 | 0.67 | 209000 | 303 | 0.8 | 34 | 1 | 230 | 134 | 155 | 10 | 134 | 86 | 102 | 26 | 98 | 12210 | YES | 0.57 | 159000 | 204 | 0.8 | 11 | no | 226 | 128 | 37 | 122 | 0 | Survivor | ory | Gram-negative | 1 |
| 26 | 42 | years | female | No | 90 | 130 | 78 | 95 | 16 | 1.69 | 408000 | 303 | 0.9 | 30 | 1 | 257 | 154 | 171 | 11 | 60 | 56 | 102 | 18 | 102 | 27500 | No | 1.59 | 358000 | 204 | 0.9 | 12 | YES | 267 | 167 | 33 | 208 | 0 | Survivor | inary | Mixed | 2 |
| 27 | 65 | than 60 | female | No | 126 | 134 | 80 | 98 | 22 | 0.98 | 210000 | 303 | 0.7 | 32 | 1 | 312 | 164 | 177 | 11 | 134 | 86 | 102 | 24 | 99.4 | 16500 | YES | 0.88 | 160000 | 204 | 0.7 | 12 | no | 287 | 163 | 35 | 211 | 0 | Survivor | Others | Culture negative | 1 |
| 28 | 43 | years | female | No | 72 | 144 | 78 | 100 | 20 | 0.46 | 573000 | 304 | 0.4 | 42 | 0 | 190 | 122 | 90 | 12 | 130 | 90 | 103 | 22 | 101.6 | 18430 | No | 0.36 | 523000 | 205 | 0.4 | 13 | no | 178 | 112 | 45 | 89 | 0 | Survivor | ory | Gram-negative | 1 |
| 29 | 40 | 40 years | Male | No | 110 | 140 | 78 | 99 | 18 | 0.63 | 54000 | 303 | 0.3 | 44 | 0 | 154 | 134 | 144 | 14 | 60 | 54 | 102 | 20 | 101.2 | 15190 | YES | 0.53 | 104000 | 291 | 0.3 | 15 | YES | 166 | 132 | 47 | 134 | 0 | Survivor | ory | Gram-positive | 2 |
| 30 | 53 | years | Male | yes | 94 | 134 | 78 | 97 | 24 | 0.68 | 291000 | 307 | 0.7 | 38 | 1 | 237 | 144 | 150 | 14 | 134 | 92 | 106 | 26 | 98 | 14590 | YES | 0.58 | 241000 | 295 | 0.7 | 15 | no | 245 | 143 | 41 | 144 | 0 | Survivor | al | Gram-negative | 1 |
| 31 | 61 | than 60 | Male | yes | 110 | 134 | 77.9 | 97 | 16 | 9.3 | 245918 | 165 | 3.6 | 33 | 1 | 200 | 148 | 148 | 4 | 70 | 54 | 103 | 18 | 98.6 | 31040 | YES | 10.67 | 86189 | 92 | 3.9 | 4 | YES | 236 | 150 | 36 | 148 | 1 | vor | inary | Mixed | 4 |
| 32 | 58 | years | female | No | 124 | 136 | 78 | 97 | 22 | 0.8 | 426000 | 298 | 0.6 | 38 | 1 | 255 | 159 | 191 | 6 | 68 | 54 | 97 | 24 | 97.6 | 25150 | YES | 0.7 | 376000 | 286 | 0.6 | 7 | YES | 198 | 146 | 41 | 191 | 0 | Survivor | ory | Gram-negative | 4 |
| 33 | 49 | years | female | No | 130 | 132 | 82 | 99 | 17 | 3.2 | 544982 | 193 | 3.1 | 39 | 1 | 264 | 165 | 228 | 3 | 60 | 52 | 99 | 19 | 98.6 | 30666 | no | 2.09 | 385253 | 120 | 3.4 | 5 | YES | 239 | 152 | 42 | 228 | 1 | vor | ory | Gram-negative | 3 |
| 34 | 53 | years | female | No | 90 | 132 | 80 | 97 | 20 | 1 | 348000 | 302 | 0.7 | 37 | 1 | 278 | 171 | 290 | 7 | 64 | 50 | 101 | 22 | 97.6 | 12760 | No | 0.9 | 298000 | 290 | 0.7 | 8 | YES | 254 | 147 | 40 | 290 | 0 | Survivor | ory | Gram-negative | 4 |
| 35 | 59 | years | Male | No | 103 | 134 | 82 | 99 | 25 | 1.1 | 169000 | 307 | 0.5 | 37 | 1 | 160 | 165 | 184 | 6 | 78 | 66 | 106 | 27 | 98.6 | 16560 | No | 1 | 119000 | 295 | 0.5 | 7 | YES | 260 | 161 | 40 | 184 | 0 | Survivor | ory | Gram-negative | 4 |
| 36 | 51 | years | female | No | 124 | 128 | 82 | 97 | 20 | 4.6 | 267891 | 145 | 3.3 | 40 | 1 | 144 | 144 | 155 | 4 | 70 | 56 | 97 | 22 | 99.4 | 13750 | YES | 5.98 | 108162 | 72 | 3.6 | 4 | YES | 268 | 167 | 43 | 155 | 1 | vor | ory | Gram-negative | 3 |
| 37 | 53 | years | female | No | 120 | 144 | 80 | 101 | 24 | 1.2 | 220000 | 302 | 0.5 | 42 | 0 | 178 | 131 | 144 | 6 | 80 | 56 | 101 | 26 | 98 | 6730 | YES | 1.1 | 170000 | 290 | 0.5 | 7 | YES | 138 | 141 | 45 | 144 | 0 | Survivor | ory | Gram-negative | 3 |
| 38 | 54 | years | female | No | 110 | 130 | 78 | 95 | 24 | 3.2 | 349627 | 167 | 3.5 | 40 | 1 | 200 | 133 | 171 | 6 | 60 | 54 | 95 | 26 | 98.6 | 9750 | YES | 2.3 | 189898 | 94 | 3.8 | 3 | YES | 168 | 147 | 43 | 171 | 1 | vor | al | Gram-negative | 4 |
| 39 | 53 | years | Male | No | 130 | 136 | 80 | 99 | 24 | 2.1 | 313129 | 172 | 4.3 | 40 | 1 | 254 | 154 | 200 | 4 | 70 | 56 | 99 | 26 | 97.4 | 14430 | YES | 2.52 | 153400 | 99 | 4.6 | 2 | YES | 220 | 129 | 43 | 200 | 1 | vor | ory | Gram-negative | 2 |
| 40 | 37 | 40 years | Male | yes | 118 | 120 | 80 | 93 | 20 | 3.3 | 85000 | 201 | 1.9 | 44 | 0 | 110 | 143 | 141 | 5 | 68 | 54 | 103 | 22 | 103 | 590 | YES | 2.28 | 74282 | 128 | 2.2 | 3 | YES | 234 | 132 | 47 | 141 | 1 | vor | al | Gram-negative | 3 |
| 41 | 48 | years | female | No | 140 | 134 | 70 | 91 | 11 | 0.74 | 260000 | 292 | 0.7 | 58 | 0 | 165 | 156 | 144 | 10 | 134 | 70 | 91 | 13 | 98 | 13560 | YES | 0.64 | 210000 | 280 | 0.7 | 11 | no | 114 | 143 | 54 | 144 | 0 | Survivor | ory | Gram-negative | 1 |
| 42 | 57 | years | Male | No | 90 | 140 | 78 | 99 | 18 | 2.5 | 100001 | 192 | 2.2 | 41 | 0 | 198 | 128 | 112 | 4 | 56 | 50 | 101 | 20 | 97.4 | 2005 | YES | 3.89 | 59288 | 119 | 2.5 | 4 | YES | 243 | 155 | 44 | 112 | 1 | vor | al | Gram-negative | 2 |
| 43 | 58 | years | female | No | 110 | 128 | 72 | 91 | 18 | 1.3 | 192000 | 182 | 2.6 | 39 | 1 | 236 | 139 | 144 | 4 | 68 | 50 | 91 | 20 | 98.6 | 16390 | YES | 2.69 | 32271 | 109 | 2.9 | 5 | YES | 212 | 144 | 42 | 144 | 1 | vor | al | Gram-negative | 3 |
| 44 | 60 | years | Male | No | 90 | 128 | 76 | 93 | 24 | 5.4 | 220000 | 132 | 2.2 | 37 | 1 | 232 | 136 | 145 | 3 | 70 | 60 | 97 | 26 | 97 | 35620 | YES | 6.82 | 60271 | 59 | 2.5 | 3 | YES | 240 | 141 | 40 | 145 | 1 | vor | ory | Gram-positive | 2 |
| 45 | 60 | years | Male | No | 92 | 128 | 72 | 91 | 18 | 0.83 | 171000 | 292 | 0.4 | 53 | 0 | 194 | 132 | 165 | 9 | 70 | 62 | 91 | 20 | 98.6 | 14800 | YES | 0.73 | 121000 | 169 | 0.4 | 10 | YES | 236 | 150 | 49 | 165 | 0 | Survivor | al | Culture negative | 2 |
| 46 | 38 | 40 years | Male | No | 120 | 134 | 74 | 94 | 22 | 0.9 | 144000 | 306 | 0.4 | 53 | 0 | 200 | 148 | 148 | 10 | 126 | 94 | 105 | 24 | 101 | 29280 | YES | 0.8 | 94000 | 183 | 0.4 | 11 | no | 198 | 146 | 49 | 148 | 0 | Survivor | ory | Gram-positive | 1 |
| 47 | 51 | years | Male | No | 98 | 134 | 74 | 94 | 18 | 1.3 | 39000 | 298 | 0.7 | 58 | 0 | 255 | 159 | 191 | 7 | 70 | 58 | 97 | 20 | 97 | 9680 | No | 1.2 | 89000 | 175 | 0.7 | 8 | YES | 239 | 152 | 54 | 191 | 0 | Survivor | al | Gram-negative | 4 |
| 48 | 39 | 40 years | Male | No | 136 | 134 | 74 | 94 | 22 | 0.64 | 445000 | 298 | 0.6 | 59 | 0 | 264 | 165 | 228 | 8 | 74 | 62 | 97 | 24 | 98.6 | 15680 | YES | 0.54 | 395000 | 175 | 0.6 | 9 | YES | 254 | 147 | 55 | 228 | 0 | Survivor | ory | Gram-negative | 1 |
| 49 | 57 | 51 to 60 years | Male | No | 140 | 132 | 80 | 97 | 24 | 1.39 | 224000 | 297 | 0.5 | 57 | 0 | 278 | 171 | 290 | 8 | 72 | 60 | 96 | 26 | 97.4 | 31280 | YES | 1.29 | 174000 | 174 | 0.5 | 9 | YES | 260 | 161 | 53 | 290 | 0 | Survivor | Abdominal | Culture negative | 2 |
| 50 | 41 | 41 to 50 years | Male | No | 106 | 132 | 72 | 92 | 12 | 0.89 | 164000 | 298 | 0.7 | 57 | 0 | 160 | 165 | 184 | 9 | 128 | 82 | 97 | 14 | 98.4 | 6690 | YES | 0.79 | 114000 | 175 | 0.7 | 10 | no | 268 | 167 | 53 | 184 | 0 | Survivor | Respiratory | Gram-negative | 1 |
| 51 | 51 | 51 to 60 years | Male | No | 110 | 132 | 74 | 93 | 16 | 0.8 | 275000 | 304 | 0.7 | 50 | 0 | 144 | 144 | 155 | 7 | 70 | 62 | 103 | 18 | 98.6 | 31040 | YES | 0.7 | 225000 | 181 | 0.7 | 8 | YES | 138 | 141 | 46 | 155 | 0 | Survivor | Abdominal | Gram-negative | 3 |
| 52 | 57 | 56 to 65 years | Male | No | 124 | 134 | 72 | 93 | 18 | 0.7 | 27000 | 294 | 0.7 | 53 | 0 | 178 | 131 | 144 | 8 | 128 | 76 | 93 | 20 | 101.2 | 45580 | YES | 0.6 | 77000 | 171 | 0.7 | 9 | no | 168 | 147 | 49 | 144 | 0 | Survivor | Respiratory | Gram-negative | 1 |
| 53 | 53 | 51 to 60 years | Male | No | 100 | 140 | 72 | 95 | 20 | 1.1 | 233000 | 298 | 0.8 | 60 | 0 | 200 | 133 | 171 | 8 | 60 | 54 | 97 | 22 | 99.4 | 13070 | YES | 1 | 183000 | 175 | 0.8 | 9 | YES | 220 | 129 | 56 | 171 | 0 | Survivor | Abdominal | Culture negative | 2 |
| 54 | 42 | 41 to 50 years | Male | No | 108 | 140 | 72 | 95 | 22 | 0.68 | 45000 | 298 | 0.9 | 50 | 0 | 254 | 154 | 200 | 9 | 128 | 82 | 97 | 24 | 97.4 | 167 | YES | 0.58 | 95000 | 175 | 0.9 | 10 | no | 234 | 132 | 46 | 200 | 0 | Survivor | Abdominal | Gram-negative | 1 |
| 55 | 52 | 51 to 60 years | Male | No | 90 | 134 | 72 | 93 | 20 | 0.3 | 257000 | 297 | 0.4 | 56 | 0 | 110 | 143 | 141 | 7 | 66 | 54 | 96 | 22 | 98.6 | 13260 | YES | 0.2 | 207000 | 174 | 0.4 | 8 | YES | 114 | 143 | 52 | 141 | 0 | Survivor | Respiratory | Gram-negative | 3 |
| 56 | 55 | 51 to 60 years | Male | No | 114 | 130 | 72 | 91 | 16 | 0.8 | 508000 | 301 | 0.7 | 58 | 0 | 165 | 156 | 144 | 9 | 54 | 50 | 100 | 18 | 97.4 | 15520 | YES | 0.7 | 458000 | 178 | 0.7 | 10 | YES | 243 | 155 | 54 | 144 | 0 | Survivor | Respiratory | Gram-positive | 1 |
| 57 | 52 | 51 to 60 years | Male | No | 56 | 140 | 72 | 95 | 22 | 2.8 | 178000 | 182 | 2.9 | 51 | 0 | 198 | 128 | 112 | 4 | 68 | 50 | 97 | 24 | 97.8 | 10480 | No | 4.16 | 98101 | 109 | 3.2 | 4 | YES | 212 | 144 | 47 | 112 | 1 | Nonsurvivor | Abdominal | Gram-negative | 3 |
| 58 | 55 | 51 to 60 years | Male | No | 170 | 134 | 76 | 95 | 14 | 0.83 | 45000 | 297 | 0.4 | 59 | 0 | 236 | 139 | 144 | 8 | 68 | 48 | 96 | 16 | 98.2 | 9480 | YES | 0.73 | 95000 | 174 | 0.4 | 9 | YES | 240 | 141 | 55 | 144 | 0 | Survivor | Respiratory | Gram-negative | 2 |

| | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 59 | 49 | 41 to 50 years | Male | No | 150 | 130 | 80 | 97 | 25 | 1.63 | 267000 | 300 | 0.8 | 57 | 0 | 232 | 136 | 145 | 9 | 132 | 82 | 99 | 27 | 101 | 18610 | YES | 1.53 | 217000 | 177 | 0.8 | 10 | no | 230 | 132 | 53 | 145 | 0 | Survivor | Respiratory | Gram-negative | 1 |
| 60 | 58 | 51 to 60 years | Male | yes | 110 | 140 | 78 | 99 | 18 | 3.4 | 488000 | 183 | 2.5 | 53 | 0 | 194 | 132 | 165 | 3 | 70 | 60 | 100 | 20 | 97.4 | 29290 | YES | 2.04 | 328271 | 110 | 2.8 | 3 | YES | 189 | 129 | 49 | 165 | 1 | Nonsurvivor | Genitourinary | Gram-negative | 2 |
| 61 | 52 | 51 to 60 years | Male | No | 92 | 134 | 82 | 99 | 16 | 1.35 | 207000 | 300 | 0.4 | 51 | 0 | 196 | 140 | 132 | 10 | 80 | 70 | 99 | 18 | 98.2 | 14080 | YES | 1.25 | 157000 | 177 | 0.4 | 11 | YES | 190 | 141 | 47 | 132 | 0 | Survivor | Respiratory | Gram-negative | 1 |
| 62 | 56 | 56 to 65 years | Male | No | 140 | 134 | 72 | 93 | 16 | 1.4 | 459000 | 294 | 0.7 | 50 | 0 | 188 | 129 | 150 | 9 | 128 | 76 | 93 | 18 | 102 | 14590 | YES | 1.3 | 409000 | 171 | 0.7 | 10 | no | 233 | 122 | 46 | 150 | 0 | Survivor | Respiratory | Gram-negative | 1 |
| 63 | 45 | 41 to 50 years | Male | no | 100 | 130 | 72 | 91 | 20 | 0.9 | 248000 | 298 | 0.3 | 57 | 0 | 150 | 112 | 123 | 10 | 132 | 80 | 97 | 22 | 98.2 | 23610 | YES | 0.8 | 198000 | 175 | 0.3 | 11 | no | 145 | 98 | 53 | 123 | 0 | Survivor | Others | Culture negative | 1 |
| 64 | 60 | 56 to 65 years | Male | No | 136 | 140 | 72 | 95 | 20 | 1.2 | 608000 | 298 | 0.3 | 56 | 0 | 250 | 159 | 142 | 8 | 72 | 64 | 97 | 22 | 100.6 | 21400 | No | 1.1 | 558000 | 175 | 0.3 | 9 | YES | 245 | 154 | 52 | 142 | 0 | Survivor | Respiratory | Gram-negative | 2 |
| 65 | 52 | 51 to 60 years | Male | No | 114 | 134 | 72 | 93 | 16 | 1.17 | 335000 | 297 | 0.8 | 59 | 0 | 230 | 134 | 155 | 9 | 128 | 80 | 96 | 18 | 98.2 | 24420 | No | 1.07 | 285000 | 174 | 0.8 | 10 | no | 226 | 128 | 55 | 155 | 0 | Survivor | Respiratory | Gram-negative | 1 |