"TO STUDY SERUM PHOSPHOLIPASE A2 IN SEPSIS AND NON-SEPSIS PATIENTS AT KLE'S DR. PRABHAKAR KORE HOSPITAL & MRC, CROSS-SECTIONAL ONE YEAR STUDY BELAGAVI."

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

Date: 31/12/2022
Place: Belagavi

KAHER BELAGAVI J.N. MEDICAL COLLEGE

DR.(Mrs.) N.S. MAHANTSHETTI
MD (PAEDIATRICS)
Principal
J. N. Medical College,
Nehru Nagar, Belagavi – 10

PRINCIPAL J.N. Medical College, BELAGAVI- 590 010

Date: 2/01/23
Place: Belagavi

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REG NO: BG0120020

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Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA
0831-2471350 0831-2470759 www.jnmc.edu principal@jnmc.edu

Ref No: MDC/PG/ Date: 14-12-2022.

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Postgraduate Student,
2020-21 Batch,
Department of General Medicine,
J. N. Medical College, Belagavi.

ANNEXURE I: ETHICAL CLEARANCE

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JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 17 **Date: 25/01/2021**

To,

REGISTRATION NO: BG0120020.

PG student in Medicine,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"TO STUDY SERUM PHOSPHOLIPASE A2 IN SEPSIS AND NON SEPSIS PATIENTS AT KLE'S DR. PRABHAKAR KORE HOSPITAL & MRC, CROSS SECTIONAL ONE YEAR STUDY BELAGAVI",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

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

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

ABSTRACT

Background: A dysregulated host response to infection results in sepsis, and severe organ failure that puts life in danger. Phospholipase A2 is a new marker in diagnosis of sepsis and is also elevated in infections. Hardly few studies were done regarding the biomarker especially regarding its capacity to diagnose sepsis. The objective of the current study was to measure the phospholipase A2 levels in sepsis patients and contrast them with their procalcitonin levels.

Material & Method: The present cross-sectional study was conducted among patients more than 18yrs of age fulfilling the inclusion criteria admitted in KLES Dr Prabhakar Kore Hospital, Belagavi from January 2021 to December 2021were analysed. All patients analyzed were more than 18yrs age meeting the q SOFA of more or equal to 2 were included. The patients were assessed for the physical biochemical examination after obtaining the informed consent the blood of patients were analysed for the sepsis, which included the procalcitonin, lactate and phospholipase A2. All, the patients data were collected. patients' redesigned proforma and entered in excel sheet. The entire study was carried out using SPSS v21 running on Windows 10 with a p-value threshold of 0.05 being considered statistically significant.

Results: Total of 80 patient were included with mean age of 58±16.75yrs of age, among them 41.3% were female and 58.8% were male patients. Blood culture showing.45.8% patients and 54.2% were negative with most common gram positive organism being Staphylococcus and gram negative being Enterobacter species. In urine culture most common is Escherichia coli . The mean of serum procalcitonin, serum phospholipase A2 and serum lactate were significantly higher among the patients with sepsis

compared to the non-sepsis .($p<0$ .05) The area under curve, the study documented serum phospholipase A2 had the higher significant AUC of 0.99 compared to the serum lactate AUC=0.983, serum procalcitonin AUC=0.957.

Conclusion: The present study documented the significantly higher mean level of serum phospholipase A2 among the sepsis compared to non sepsis patients. It was also found that phospholipase A2 was way more sensitive and specific than procalcitonin.

Keywords: sepsis, septic shock, non sepsis, phospholipase A2, procalcitonin, serum lactate, blood culture, gram positive and gram negative blood culture

ABBREVIATIONS

ARDS	ACUTE RESPIRATORY DISTRESS SYNDROME
CA	CALCIUM
CRP	C-REACTIVE PROTEIN
DIC	DISSEMINATED INTRAVASCULAR COAGULATION
HMGB	HIGH MOBILITY GROUP BOX PROTEIN
ICU	INTENSIVE CARE UNIT
ICAM	INTERCELLULAR ADHESION MOLECULE
MODS	MULTIORGAN DYSFUNCTION SYNDROME
MIF	MACROPHAGE MIGRATION INHIBITORY FACTOR
PLA2	PHOSPHOLIPASE A2
SIRS	SYSTEMIC INFLAMMATORY RESPONSE SYNDROME
SVC	SUPERIOR VENA CAVA
TNF	TUMOR NECROSIS FACTOR
WBC	WHITE BLOOD CELL
LPC	LYSOPHOSPHATIDYLCHOLINE
IL	INTERLEUKIN

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OBJECTIVES

Aim:

To assess the phospholipase A2 levels in sepsis patients and compare with procalcitonin level among them.

INTRODUCTION

Sepsis defined as “a life-threatening organ dysfunction caused by a Dysregulated host response to infection”. Include signs of infection with organ dysfunction plus altered Mental status, tachypnoea, hypotension, hepatic renal or Hematological dysfunction. A study in 2016 reported nearly 30% of patients admitted into ICUs in India are diagnosed with sepsis.[(1)]

Sepsis was proclaimed as global health priority in 2017. This is mainly due to the fact that the global burden of sepsis is way more significant than previous years. According to HAQ index it was approved that the incidence of sepsis was more prominent in areas where it is difficult to identify, treat sepsis or even prevent from occurrence. This has to considerable increase inicidence and mortality secondary to sepsis.[(2)]

The precise burden of sepsis in our country is not known because of lack of a uniform system of identifying and reporting this condition. A study in 2016 reported nearly 30% of patients admitted into ICUs in India had sepsis. In 2017- study in leading journal Lancet reported that communicable disease (infection) contribute to substantial proportion if death in India.

Innate immunity which is the primary mechanism in opposition to microbes activates in their presence and causes an uncontrolled inflammation response cascade which forms the cornerstone of pathophysiology of sepsis. Several authors have suggested that phospholipase A2 has a role in sepsis. Thus identifying the levels along with other routinely done sepsis markers like procalcitonin, C-reactive protein, lactate, white blood cells will help in earlier and more accurate diagnosis of sepsis.(3)

Need for the study

Phospholipase A2 is a new marker in diagnosis of sepsis and also elevated in bacterial infections. This will help to identify bacterial versus nonbacterial causes and start early antibiotic therapy and avoid unnecessary usage of antibiotics in non sepsis patients and prevent antibiotic resistance. Only pilot studies have been done so far so taken up in our part of the country.

The purpose of the current study was to measure the phospholipase A2 levels in sepsis patients and compare them to their procalcitonin levels.

REVIEW OF LITERATURE

"Sepsis" is considered when a normal sterile body part is invaded by microbes leading to systemic inflammation. When the body's response to bacterial infection leads to destruction of surrounding tissues and organs, this lethal condition was termed as bacterial sepsis.[4,5] In recent times it has come to conclusion that sepsis by definition should have organ dysfunction which is life threatening in nature, considered mainly due to inappropriate host response against the infection source. Hippocrates (460–470 BC) first identified sepsis as a disease; the phrase derives from the Greek verb sipsi, which means "to render rotten." Since then, this disease has gone through several incarnations, with advances in the late nineteenth century laying the groundwork for present knowledge of sepsis.[6]

As a result of the development of antiseptics, the germ theory of disease, and bacteriology, it is now widely believed that sepsis is a systemic infection brought on by a dangerous organism that infects the host and spreads via the bloodstream (i.e., septicemia). It wasn't until the discovery of endotoxin and the widespread use of antibiotics that it became clear that the pathophysiology of sepsis was significantly more complicated.[7,8]

Terms including septic shock, severe septic shock, and systemic inflammatory response syndrome (SIRS) were coined and defined by an international consensus committee in 1991. (known now as Sepsis-1). The inflammatory process is described by SIRS based on a mix of vital signs and blood testing, regardless of the reason.

Two or more of the following are included in SIRS:

• A temperature between 36 and 38 degrees Celsius

• Heartbeats per minute more than 90

• PaCO2 less than 32 mm Hg or tachypnea more than 20 breaths per minute

• A total leukocyte count (WBC) count of more than 12,000 cells per cubic millimetre, less than 4,000 cells per cubic millimetre, or more than 10% band develops

Sepsis

• SIRS brought on by an infection

Severe Sepsis Organ dysfunction in at least one or more, aberrant hypoperfusion, or sepsis-induced hypotension

• Lactic acidosis, oliguria, and sudden changes in mental status are only a few examples of the anomalies associated with hypo-perfusion.

Inspite of giving ample of fluids if the patient still persists in hypotension, such condition was termed as septic shock. In a patient who has suddenly fallen ill with organ malfunction and cannot maintain homeostasis without external support, such a condition is known as multiorgan dysfunction (MODS).

Sepsis-3 defines sepsis as a potentially fatal organ malfunction produced by an unbalanced host retaliation to infection. The fact that not all individuals with SIRS have an infection and not all people with infections are septic is extremely important to emphasise. Sepsis can be distinguished from infection by a dysregulated host response and the occurrence of end-organ failure.([9]) A range of clinical and pathophysiologic

severity characterises sepsis and its consequences, which lead to the progressive physiologic collapse of several interdependent organ systems.

Epidemiology

Sepsis is associated with substantial morbidity and death and is now estimated to impact about 1.7 million US adults each year. Prior to the year 2000, fatality rates in patients with acute sepsis and septic shock were as high as 50%.[(10)] Even with modern medicine, sepsis still accounts for one in three hospital deaths, and national mortality is still between 20% and 25%. Given the proven increase in sepsis cases, early detection and treatment of this disease process have been given top attention; the most recent budget was estimated to be around $23.7 billion in 2013. Many factors played a crucial role which a played a major role in determining the mortality of patients in sepsis which was estimated to be around 30-50%. Such factors were age, race, sex, organ failure and co morbid conditions.[(11)]

Risk factors that predispose to sepsis

- Diabetes
- Burns
- Malignancy
- Chronic kidney disease
- Liver disease
- Immunosuppressed state
- Corticosteroidal use
- Massive intraoperative procedures
- Prolonged hospitalization
- Trauma
- Haemodialysis
- Elderly patients

Pathophysiology

"Systemic inflammatory response syndrome" (SIRS), which occurs early in sepsis, continues along a pathophysiologic spectrum until "multiorgan dysfunction syndrome" (MODS), which occurs just before death.[12,13]

The following are the first indications of inflammation [14–16];

- Fever (greater than 38 degrees Celsius) or hypothermia (temperature less than 36º C)
- Tachycardia (a heart beat that exceeds 90 beats per minute), Tachypnea (more than 20 respiratory cycles)
- With or without bandemia, (WBC more than 12,000/cu mm) or (WBC less than 4,000/cu mm) (more than 10 percent).

The diagnosis of systemic inflammatory response syndrome requires alteast 2 out of the 4 clinical signs to be present. Following that, the clinical definition of sepsis "is systemic inflammatory response syndrome with an infectious cause". When hypotension sets in, tissue challenge is no longer appropriately fulfilled by tissue oxygenation. At this stage patient is considered to be in severe sepsis[11,17]

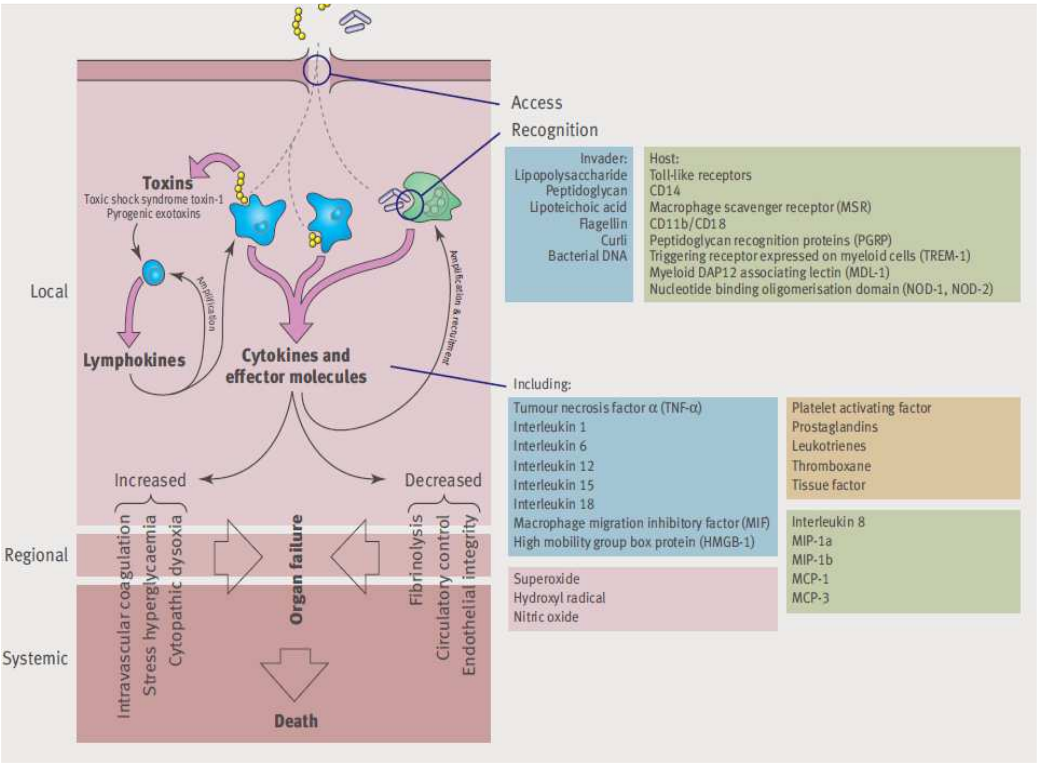


Figure 1: Pathophysiological pathway of sepsis[3]

The patient can be considered in severe sepsis when the tissue demand is not fulfilled by tissue oxygenation secondary to onset of hypotension. Hypotension further deteriorates the arterial peripheral oxygenation and perfusion, which acts as a trigger to convert to anaerobic respiration from aerobic. This anaerobic respiration increases the production of lactic acidosis. End-organ injury signs, such as transaminitis or pre-renal azotemia, can also indicate tissue hypoperfusion. To track the imbalance between the tissue oxygen supplementation and requirement can be monitored, by observing the mixed venous oxygen saturation. This is usually recorded by measuring with the help of central line in the superior vena cava.[11,17]

Septic shock differs from other shock states in that it is a distributive form of shock. A mixture of inflammatory mediators (serotonin, histamine, lysosomal enzymes super-radicals,) produced as a consequence to bacterial endotoxins increases capillary

permeability while decreasing peripheral vascular resistance. This results in a decrease of both preload and afterload due to a decrease in venous return from third-spacing. An increase in heart rate is used to initially compensate for the resulting decrease in stroke volume, or compensated septic shock. The patient is consequently experiencing the hyperdynamic symptoms of septic shock.[18–20]

Functionally, septic shock is defined as "persistent hypotension despite 60 to 80 mL/kg of crystalloid or colloid fluid resuscitation".[21,22]) At this stage, starting adequate vasoactive medicines like beta-adrenergic or alpha-adrenergic agents is critical. Multi-organ dysfunction syndrome (MODS), which has a fatality rate of up to 75%, is characterised by the emergence of organ malfunction despite supplying with high dose vasoactive therapy.[23–25] Immunologic dissonance (exaggerated pro-inflammatory response) vs. immunologic paralysis (exaggerated anti-inflammatory response) has been postulated to play a role, although pinpointing the precise factors suggesting poor prognosis and mortality has proven difficult.[26]

Early signs and symptoms

- SIRS combined with an infectious cause is known as sepsis. As a result, patients initially appear with the following changes in their vital signs:
- Fever
- Tachycardia
- Tachypnea

Signs and symptoms of severe sepsis, is combined with end organ dysfunction

- Altered mental status
- Cyanosis
- Hypoxia
- Oliguria or anuria

Patients who progress to septic shock will display severe sepsis symptoms, such as hypotension. In addition, various signs of distributive shock, such as warm extremities, rapid capillary refill, and bounding pulses, sometimes known as warm shock, may be present. Notably, blood pressure may be maintained at an early "compensated stage". This stage of shock can be reversed if treated quickly with vasoactive support and appropriate amount of fluids.[11] the varying presentation of cold peripheries, delayed capillary filling time and thready pulse is often recognised as cold shock which is irreversible in nature. The persistence of this condition further leads to decreased perfusion of organs thus causing multiorgan dysfunction which eventually leads to death.[11]

Evaluation

Laboratory findings

The laboratory findings may show the following

- Hyperglycemia (glucose more than 120 mg/dL)
- Mixed venous saturation of more than 70%
- PaO2: FiO2 less than 300
- Leukocytosis (WBC more than 12,000/mm3) or leukopenia (WBC less than 4000/mm3)
- C-reactive protein or procalcitonin more than 2 SD above normal
- Coagulopathy, INR more than 1.5 or PTT more than 60 sec

- Lactic acidosis (more than 2 mmol/L)
- Thrombocytopenia (platelets less than 100,000/mL)
- Pre-renal azotemia
- Hyperbilirubinemia (total bilirubin more than 4 mg/dl)

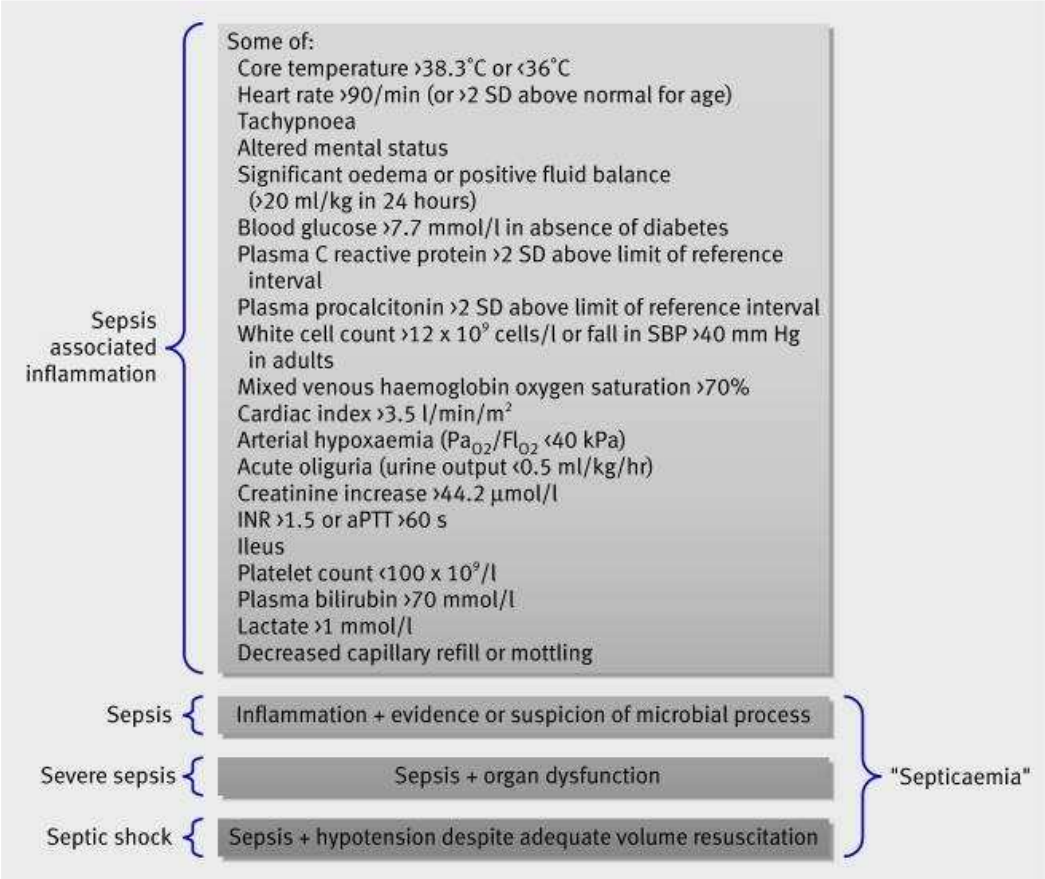


Figure 2: Definition of sepsis as severe and septic shock[3]

In order to closely monitor vital signs, vigilant cardiorespiratory monitoring is of utmost importance in the patient. To determine where they may fall on the pathophysiologic continuum of sepsis, peripheral perfusion and continuous organ functioning should be carefully assessed. A Glasgow Coma Scale (GCS), or lactate/mixed venous saturation test, urine output measurement should all be included (with central lines) The signs of pneumonia or ARDS may be seen on a chest x-ray. Plain x-rays of the extremities may show gas in the tissues if the patient develops

necrotizing fasciitis. A CT scan is performed to look for abscesses, intestinal ischemia, or perforation in the bowel loops.[11]

Biomarkers for diagnosis of sepsis[3]

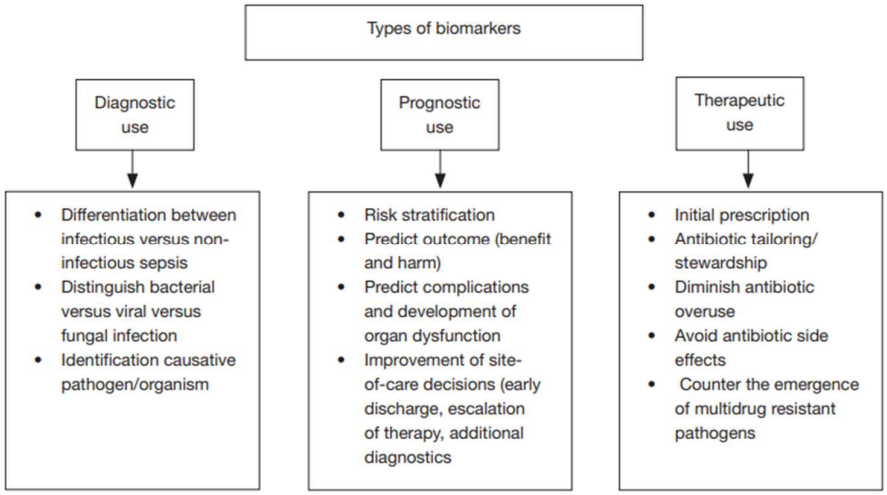


Figure: 3: Biomarker types and potential use of them

Biomarkers help in identifying the cause of sepsis, secondary to infectious nature and cause can be identified thus help in treatment of the sepsis.[27] the physician can decide on treatment on early basis with the help pf biomarkers.[28] Deciding wether the patient need stronger antibiotics or there is significant improvement can be estimated with the help of biomarkers.[29]

Circulating cells

Cell counts

- White blood count
- Platelet count
- Neutrophil count
- Lymphocyte count

Leucocyte surface markers

- CD63
- CD66b
- CD64
- Intercellular adhesion molecule (ICAM-1)
- Cell differentiation antigen CD11b

Peptides

Monocyte / macrophages

- Tumor necrosis factor α (TNF-α)
- Interleukin 6, 8, 10, 18
- Interleukin 1α
- Interleukin 1β
- Macrophage migration inhibitory factor (MIF)
- High mobility group box protein 1 (HMGB-1)

Leucocyte products

- Soluble P-selectin
- Soluble L-selectin

Acute phase reactants

- Ferritin
- C-reactive protein
- Lactoferrin
- Neopterin
- Procalcitonin
- Serum amyloid A
- Serum lactate

SPECIAL NOTES ON BIOMARKERS:

PROCALCITONIN: Procalcitonin is a predecessor of calcitonin which is usually in insignificant amounts in blood. The biomarker seemed to be raised in characteristically in bacterial sepsis followed by sharp fall once the infection treated.[30][31] The blood cultures are ideally since they help in identifying the microorganism thus selection of antibiotic will be easier but very less blood cultures come positive around 40-90 % come negative blood culture without pathogens.[32] To improve diagnosis other diagnostic tests were evaluated among them serum PCT played an important role. PCT is undetectable in absence of inflammation.[33] Bacterial toxins and cytokines (IL 6, IL 1beta, TNF-alpha) trigger the synthesis of PCT making it specific for bacterial infections (34) in case of viral infections cytokines released by them inhibit TNF-alpha thus not activating the synthesis of PCT.(33) Further helping in easy identification of the cause of infection. PCT alone cannot detect the causative agent thus making it difficult in choice of antibiotic.[35] A meta analysis also stated that the specifity and sensitivity of PCT was as low as 74% suggesting that it was difficult to differentiate between SIRS induced by sepsis and non sepsis cause.[36] Thus making it necessary to consider the patients history, systemic examination and microbiology assays.[31]

C-REACTIVE PROTIEN: CRP is a protein belonging to family pentraxins which mainly activates the C1q complex of complement system igniting a crucial defence system.[37]) Although synthesised by liver it is triggered by the markers released by adipocytes and macrophages. CRP marker has a half- life of 18 hours thus makes it a good marker to chart the progress of the patient.[37] The biggest drawback is that the CRP cannot predict the severity of the sepsis , apart from being nonspecific in nature. This causes inadequate treatment.[38]

SERUM LACTATE: Serum lactate levels were noticed to be raised in any kind of metabolic stress thus making it nonspecific in nature.[39] Anaerobic pathway is the cause for increase in lactic acid production thus raise in lactate levels. This is the main pathway in hypoxic conditions.[40] Previous papers found a positive association between raising levels of serum lactate and mortality of the given patients.[41] on the whole one can say that serum lactate levels can help predict mortality but it has no role in determining the cause of the inflammation or in treatment.

Interleukins: Cytokines especially interleukin 6,8,18 seemed to be pro inflammatory in nature and infact the cytokines responsible for sepsis inflammatory pathway. It was also found that IL 10 was proinflammatory in nature. On further study results have suggested fall in IL-6 levels was associated with better prognosis while rise in IL10 levels were ondicator of bad prognosis.[42] it was also noticed that IL 8 was found to distinguish a patient in muiltiple organ dysfunction and disseminated intravascular coagulation.[43] while interleukin 10 seemed to be the main cause for Acute respiratory distress syndrome. The main mechanism seemed to be inhibition of hyperinflammation in severe sepsis by IL10. It was interestingly found to be higher in severe sepsis cases.[44] [45] Howerever it was found that these interleukins were not found to be as sensitive or as specific as procalcitonin.[46]

Phospholipase A2 as a Biomarker in Sepsis

All human cells contain members of the Phospholipase A (PLA) superfamily of esterase enzymes, which are crucial for the synthesis of lysophospholipids and free fatty acids from glycerophospholipids. The so called enzymes play an important role in activation of inflammation along with subsequent participation which results in disease in every organ. These enzymes also play an important role in homeostasis. Over 30 PLA isoforms have been discovered, with significant differences in function, cofactor need, and size. Cytosolic PLA, secretory PLA, Ca-independent PLA, adipose-specific PLA, lysosomal PLA, and platelet-activating factor acetylhydrolase are the six major isoform categories. Numerous subgroups have been established within each of these categories via study.[47]

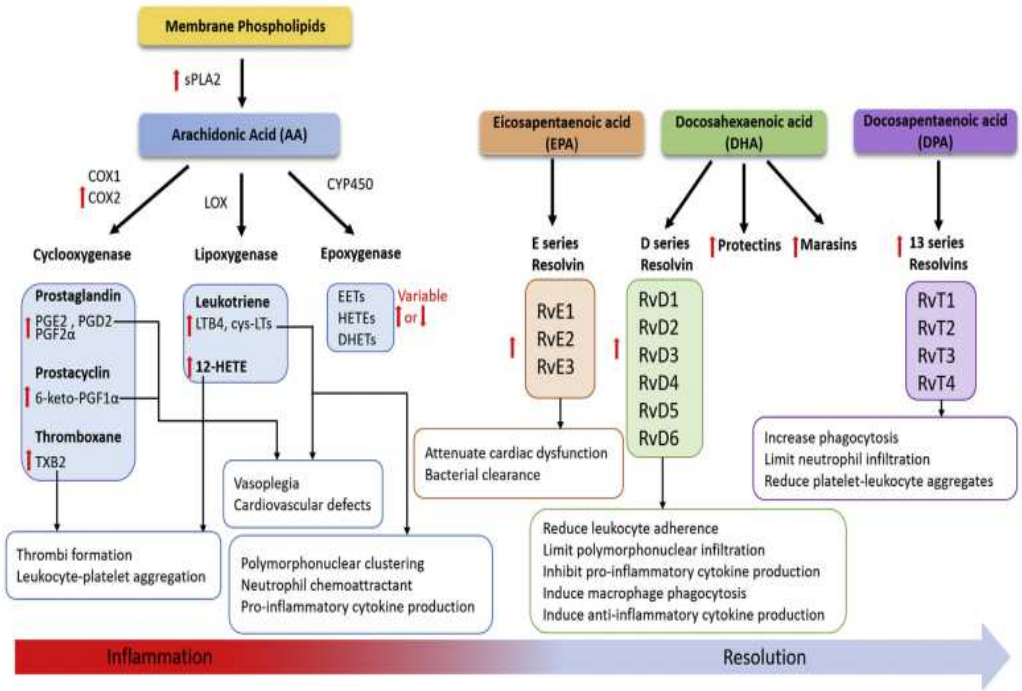


Figure 4: Eicosanoids and Sepsis.[48]

Phospholipases A2 are a group of unrelated protein families that share an enzymatic function. Secreted and cytosolic phospholipases A2 are the two most prominent families. Lipoprotein-associated PLA2s (lp-PLA2), also called as platelet activating factor acetylhydrolase and Ca2+ independent PLA2 (iPLA2), are two further families.

Phospholipases A2 (PLA2s) EC 3.1.1.4 are enzymes that hydrolyze the link between glycerol molecule and the second fatty acid "tail". Thus, removing fatty acid from the second position of phospholipids. This phospholipase identifies the phospholipid's sn-2 acyl link and catalytically hydrolyzes it, producing arachidonic acid and lysophosphatidic acid. Cyclooxygenases or lipooxygenases further act on the by-product arachidonic acid forming active components known as eicosanoids. Eicosanoids further dived into the anti-inflammatory components (mediators) known as leukotrienes and prostaglandins.[49]

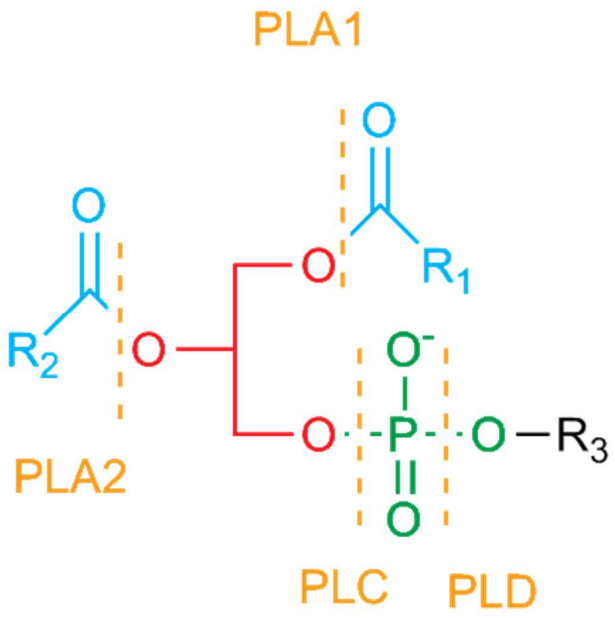


Figure 5: Phospholipase A2 cleavage site

PLA2 enzymes are present in venom from arachnids, insects, and snakes as well as mammalian tissues.[50] Bee venom contains a lot of melittin, who's main role is to stimulate PLA2. Once a snake bite, bug bite this causes increased release and activity of phospholipase A2. This PLA2 will activate the arachidonic acid pathway which results in the area becomes inflamed and painful.[51] Prokaryotic A2 phospholipases are also present.

The arachidonic acid is further divided into oxylipid and lysophospholipid in the presence of both secretory phospholipase A2 (sPLA2) and cytosolic PLA2 (cPLA2) and further degrade into necessary components.[48]

Biological effect:

The enzyme is one of the acute phase proteins whose main trigger is inflammation. In specific serum phospholipid acts on the bacterial membrane hydrolysing the phospholipid group giving Lys phosphatidylcholine (LPS). This LPC is further lysed which initiates the cascade of innate immunity and specifically directing to the bactericidal action.[52] LPC as per say doesn't have any direct action on the bacteria but acts by increasing the reactive oxygen species production by neutrophils which further act.[53]

Various studies discussing the Phospholipase A2 biomarker in sepsis

In a study by Rintala EM et al., to assess the Phospholipase A2 among the patients with fever with documented infection. The PLA2-II serum concentrations in individuals suffering from septicemia (median, 164.5µg/L; range, 5.07–1,740µg/L) were 46-fold higher than standard amounts (median, 3.61µg/L; range, 1.32–25.25µg/L). Phospholipase A2-II concentrations in sepsis associated patients (median, 284.5µg/L; range, 12.95–1,574µg/L) as well as non-septic bacterial etiology (median, 210.6µg/L; range, 5.07–1,740µg/L) were significantly higher than viral etiology (P =.0042). PLA2-II concentration associated strongly with C-reactive protein (CRP) concentration (r =.613, P =.001but was not associated with pancreatic PLA2 concentration (r =.089, P =0.365). The serum concentration of PLA2-II can be used in conjunction with CRP values to differentiate bacterial from viral infection.[54]

In a study conducted by Endo S et al., to assess the plasma level of cytokines and type Ii PLA2 in patients with sepsis. Study documented that the patients who died from sepsis had much greater plasma levels of type II PLA2 than those who pulled through the disease. There was a strong link between type II PLA2 and IL-6 and TNF-alpha. Type II PLA2, IL-8, IL-6, and TNF-alpha may be beneficial as disease severity indexes. In the plasma of sepsis patients, IL-6 and TNF-alpha seem to increase the synthesis of type II PLA2.[55]

In a study by Mansour KM et al., (2011) to assess the secretory phospholipase A2 in patients suffering from fever. It was observed that the serum PLA2 levels in bacterial infections (190±179 ng/mL) and in viral infections (22±34 ng/mL). this was observed to be significant statistically (P.0001). According to receiver operator characteristic curve analysis, it was perceived that Spla2 was efficient in anticipating

bacterial etiology (area under curve= 0.89) than total leukocyte count (are under curve=0.71). It was also seen that a positive predictive value of 39%, negative predictive value of 97%along with specificity of 67% and sensitivity of 93%, when the sPLA2 levels were more than 20ng/ml. The researchers found that secretory phospholipase A2 levels varied considerably between children with viral and bacterial infections and that it appears to be a dependable screening test for bacterial infection in febrile toddlers.(56)

In a Study that was done by Uusitalo-Seppala R et al., (2012) to assess the diagnostic value of PLA2, total leukocyte count and CRP in detecting the sepsis during admission at emergency room. The results documented that only PLA2GIIA could distinguish patients with severe sepsis from others in a multivariate logistic regression study (OR 1.37, 95% CI 1.05–1.78, p = 0.019). PLA2GIIA remained a significant independent predictor of severe sepsis after controlling for covariates. When correlated phospholipase A2 was more specific to infectious origin of sepsis than c reactive protein which seem to be raised in other chronic states. Phospholipase A2 was more specific towards bacterial sepsis.(57)

According to a study done by Tan et.al., the year 2016 in Malaysia showed a strong correlation between early sepsis and phospholipase a2 levels and positively correlated with bacterial sepsis.(58)

In a study done by Tan TL et al., (2016), assessed the secretary phospholipase A2 as a new marker in the patients with sepsis compared with CD64. Severe sepsis was verified in 42 of the 51 recruited individuals. Twenty-one individuals developed bacterial illnesses that were verified by culture. Both indicators were proven to be effective in differentiating between sepsis and non-sepsis groups. In adults, sPLA2-IIA

and CD64 showed a high connection with early detection of sepsis. The area under the curve (AUC) of both Receiver Operating Characteristic curves revealed that sPLA2-IIA outperformed CD64. When compared to CD64, sPLA2-IIA performed better in detecting sepsis and bacterial infections. sPLA2-IIA looks to be an remarkable biomarker for identifying bacterial cause of infection and sepsis screening, whereas CD64 might be employed for bacterial infection screening. Both indicators, alone or together along with other markers help us in considering the usage of antibiotics in early stages of treatment.[58]

In a study done by Berg E et al., (2018) to assess the novel biomarker as secretary phospholipase A2 as marker of sepsis. Control patients (median = 0 ng/ml [interquartile range (IQR): 0–6.5] had considerably lower sPLA2-IIA levels than septic patients (median = 123 ng/ml [IQR 44–507.75]; P<0.0001).also on further evaluation it was found that in identified source of infection the SPLA2-IIA levels was comparatively higher(n=28 pts, 95% CI=115.1-516.8, median=186ng/ml) than in the ones the source was not identified or infection secondary to viral etiology (n=17, median=68ng/ml; P=0.04, 95% CI=38.1-122.7). Using a threshold value of 25 ng/ml, sPLA2-IIA demonstrated an 86.7 percent sensitivity (confidence range 72.5–94.5) and a 91.1 percent specificity (confidence interval 77.9–97.1) in identifying sepsis. This study proves that sPLA2-IIA helps in identifying patients in early sepsis along with specificity and sensitivity in those patients especially infection secondary to bacterial origin thus being in accordance with older publications. This was seen true especially in sepsis involving various organs at once.[59]

In a study done by Nik Nurhanan Nik Mansor N et al., (2018) to assess the secretory phospholipase A2 in bacterial sepsis using biosensors. To diagnosis bacterial infections and sepsis secondary to bacterial infection an electrochemical biosensor that uses tri-enzyme system was designed and was successful in detecting the sPLA2-IIA was the result derived from the above study. Looking at the above studies, it was found that sPLA2-IIA was indeed a magnificent marker with multiple usage as identifying the source of infection that is bacterial and sepsis status of the patient. Another important issue at hand is early usage of antibiotic in the treatment. This marker can be used alone or in combination with other markers. The detection level of sPLA2-IIA may accelerate the initiation of early antimicrobial therapy and sepsis bundle.(60)

In a study done by Huang Z et al., (2018) to assess the serum level of lipoprotein associated Phospholipase A2 in predicting the mortality rate among the patients with sepsis. Serum LpPLA2 levels was found to be higher in sepsis patients who were admitted to EICU than healthy individuals. LpPLA2 concentrations were related to disease severity and were substantially connected with experimental indicators of inflammation and existing prognostic ratings. When compared to prognostic scoring methods such as SOFA or APACHE II scores, consistently with increased levels of LpPLA2 on admission for EICU therapy was a poor outcome predictor and offered worthier diagnostic use in the overall population. Taken together, the findings showed that LpPLA2, in comparison to other indicators of inflammation, may have a predictive role in sepsis, and provide preliminary evidence for future studies to investigate the clinical and pathophysiologic implications of LpPLA2 in sepsis. Study suggested that phospholipase A2 levels vary according to the severity of sepsis.(61)

In a study by Ahmad NS et al., (2020) to assess the high sPLA2 levels are associated with the ecosanoids metabolism in patients with bacterial sepsis syndrome (BSS). When Bacterial sepsis (BS) and BSS patients were compared to healthy participants, serum levels of PGE2, sPLA2-IIA and PGD Synthase rose considerably ($p<0.05$). It was noticed that the COX-2 levels were considerably higher in BS in comparison with normal individuals but there was no effect on COX-1. PGE2 and sPLA2-IIA were found to be independent predictors of BSS severity by binary logistic regression analysis. Finally, it was found that eicosanoid metabolism in the bacterial sepsis the cause for increased levels of sPLA2.[62]

In a study conducted by Nandi U et al., (2021) to assess the secretory phospholipase 2 as independent predictor or mortality and positive blood culture among the patients with sepsis presenting to emergency department. It was observed that adjusted odds of death was 3.78 (CI= 1.14-12.56, P=0.03) when then sPLA2-IIA was recorded equal to or more than 100ng/ ml.in patients whose blood culture came positive, both procalcitonin and sPLA2-IIA levels were higher but difference between this 2 was not significant statistically. sPLA2-IIA levels were considerably greater in individuals who had a positive blood culture. The reasearchers also came to conclusion that patients who had higher levels of sPLA2-IIA had increased risk of mortality.one more interesting finding was that patients with blood culture positive had more sPLA2-IIA levels than procalcitonin.[63]

In a study by Tan TL et al., (2021), the comparison between hs-CRP neutrophil percentage, lactate, PCT and PLA2 in detection of bacterial infection. sPLA2-IIA had the best performance (AUROC 0.93 [95 percent CI: 0.89–0.97]; Sp 94 percent [95 percent CI: 81–89]); Sn 80 percent [95 percent CI: 72–87];. It was equivalent to high-

sensitive CRP (AUROC 0.93 [95 percent CI: 0.88–0.97]; Sp 91 [95 percent CI: 77–98]); Sn 75% [95 percent CI: 66–83], but with a greater Sp and Sn. When compared with the other three namely, PCT, Lactate and Neutrophil, PLA2 was found to be superior. with the help of this study, it was suggested that to identify bacterial in fection in middle and low income countries, sPLA2-IIA could be used .[64]

MATERIALS AND METHODS

Source of data: Medical records and detailed history along with examination of patients admitted in KLES Dr Prabhakar Kore Hospital, Belagavi were used for study purposes.

STUDY DESIGN: A cross sectional based study.

STUDY PERIOD: January 2021 to December 2021.

SAMPLE SIZE: calculated by formula

a) Sample size - 80

The minimum sample size formula based on medcale software

Type 1 error (alpha, significance) =0.001

Type 2 error (beta, 1-power) =0.01

Area under curve = 0.93, null hypothesis value= 0.5

Ratio of sample size in negative /positive group =1/3

Results no. of positive cases required =22

No. of negative cases required=8

Total no. of cases=30

For better results sample size taken is

Sepsis=59

Non sepsis=21

INCLUSION CRITERIA

- All patients who meet the SOFA or SIRS criteria of more or equal to 2.
- Age more than 18 years.

EXCLUSION CRITERIA

- Age less than 18 years.
- History of Rheumatoid Arthritis.
- History of Asthma.
- History of Systemic Lupus Erythematous.
- History of Malignancies.
- Patients partially treated by antibiotics for more than 3 days.

METHODOLOGY

1. The study conducted only after obtaining ethical clearance from the Institutional Ethics committee and conducted during the planned study period.
2. Patients fulfilling the inclusion criteria and willing to participate were included in the study.
3. Informed consent was obtained.
4. The cases were evaluated according to q sofa score (have to be 2/3) then taken as sepsis cases.
5. Non sepsis cases were controls admitted for other reasons.

6. Serum sample was taken from the patient within 24 hours of admission in yellow colour tube and immediately sent to biochemistry lab in the same facility where it is preserved in -4˚C. it was done till 10 samples were collected and then were subjected to testing.

7. First the samples undergo centrifuge to separate the serum from the sample and serum is subjected to testing with the help of Product Name KTE60328 Human Secreted Phospholipase A2 (SPLA2) ELISA Kit. KIT NO: KTE60328.

8. ASSAY PRINCIPLE: Human Secreted Phospholipase A2 (sPLA2) ELISA Kit employs a two-site sandwich 2-8 °C 2-8 °C mL mL ELISA to quantitate sPLA2 in samples. An antibody specific for sPLA2 has been 3 mL 6 mL pre-coated onto a microplate. Standards and samples are pipetted into the wells and any SPLA2 present is bound by the immobilized antibody. After removing any unbound 20 mL (20) 20 mL (30 8 °C Plate covers substances, HRP-Conjugated Human sPLA2 detection antibody is added to the wells. Booklet RI Following a wash to remove any unbound HRP reagent, a Chromogen solution is added to the wells and color develops in proportion to the amount of sPLA2 bound in the initial 1 Retum unused wells to the foil pouch containing the desiccant pack. Reseal along entire edge of the step. The color development is stopped and the intensity of the color is measured.

Investigations performed on the patient are

1. Complete blood count
2. Liver function test
3. Renal function test
4. Serum secretory Phospholipase A2 through ELISA method
5. Chest x ray
6. Procalcitonin
7. Lactate
8. Random blood sugars

STATISTICAL ANALYSIS

Data obtained was tabulated in Microsoft excel version 16.48 and subjected to appropriate statistical analysis. Descriptive statistics were presented as percentages for categorical variables, mean and standard deviation for continuous variables. The strength association (p value) was calculated using unpaired t test or Mann- Whitney U test (non parametric). Chi-square test for categorical variables (if the cells were less than 1 then further subdivision of Chi-square test – Monte Carlos test) was considered. Receiver operating characteristic curve is used to determine the threshold of the biomarkers. All tests were 2 tailed tests. Statistical significance was set at p value less than 0.05.

RESULTS

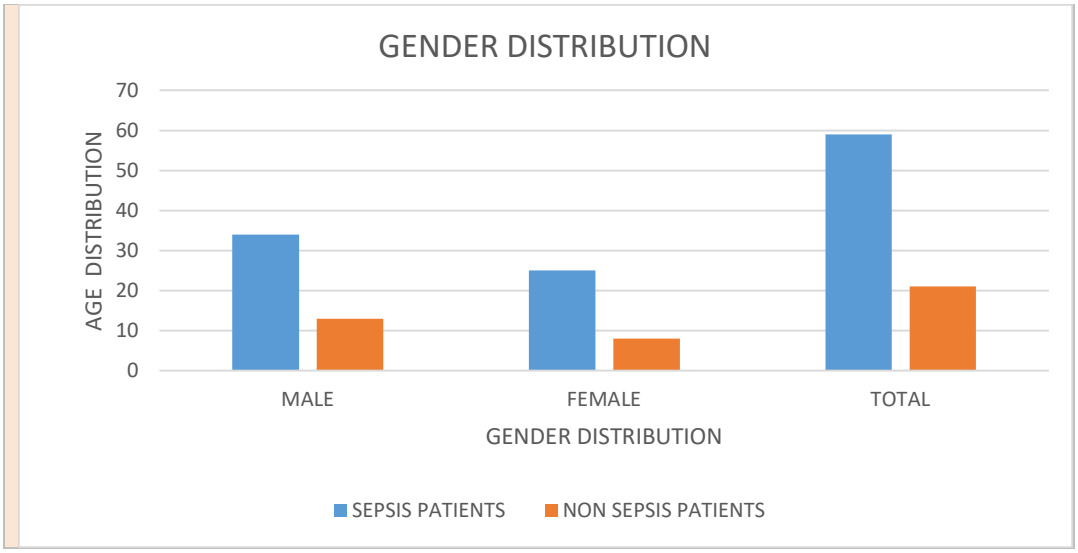
- 80 patients that met inclusion criteria were approached for the study. There were no patients who were Age less than 18 years of having History of Rheumatoid Arthritis, History of Asthma, History of Systemic Lupus Erythematous, History of Malignancies, Patients partially treated by antibiotics for more than 3 days. Thus no patients were excluded.

Table:1 Distribution of gender among sepsis and non sepsis cases

	MALE	FEMALE	P value
SEPSIS	34	25	0.8
NON-SEPSIS	13	8	

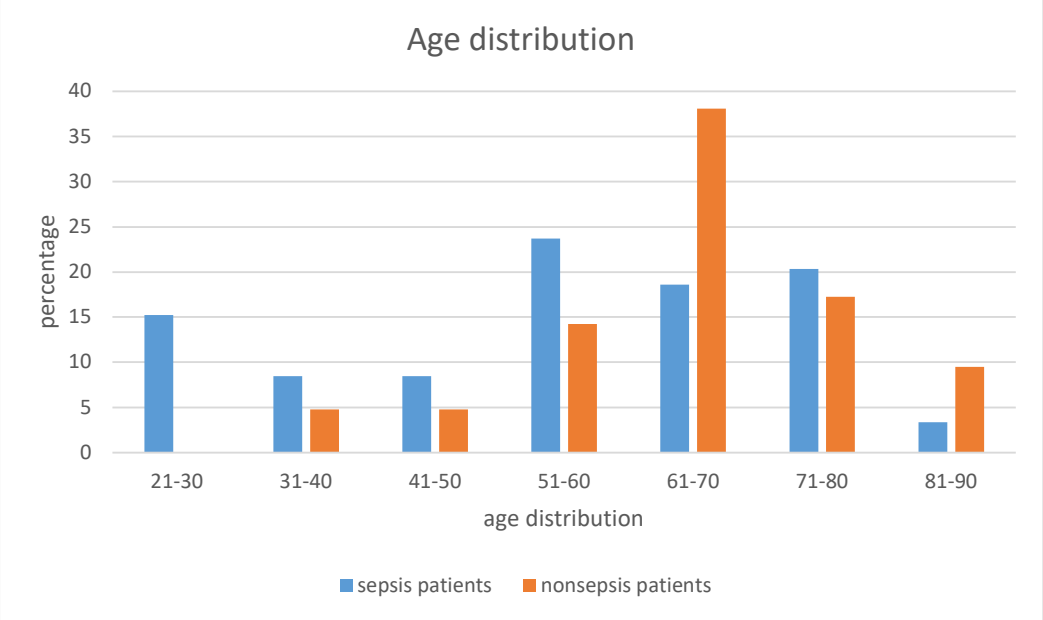
Among the participants of the study it was observed that there were 34 male patients and 25 female patients in sepsis category(total=59) while non-sepsis had 13 male and 8 female (21). p value to be 0.8 which was insignificant suggesting gender had no relation with sepsis.

FIGURE 6: Showing gender distribution among sepsis and non sepsis patients.



AGE DISTRIBUTION:

FIGURE 7: Age distribution among sepsis and nonsepsis cases



According to above graph it was observed that in sepsis patient 55.6±17.2 (mean ± standard deviation) and in non-sepsis 65.4±12.97 (mean± standard deviation) suggesting sepsis was affecting lower age group probably due to multiple co morbidities.

Table 2: Comparing phospholipase a2 levels in sepsis and non sepsis patients:

	Mean ng/lt	Standard deviation ng/lt
Sepsis patients	105.16	60.06
Non sepsis patients	17.90	4.90

Above table was showing the mean being 105.16 and standard deviation 60.06 in sepsis and nonsepsis patients showed mean 17.90 and standard deviation 4.90. on further usage of unpaired t test it was found that p value was < 0.0001 which is statistically significant.

Table 3: Comparison of the complete blood cell result between the groups

	Group				p-value
	Sepsis		Non-sepsis		
	Mean	SD	Mean	SD	
Total Leukocyte Count/µL	16874.6	8983.0	12514.3	4237.4	0.03*
Heamoglobin g/dL	12.3	5.6	12.5	3.3	0.229
Platelets/µL	193987.7	128435.7	211428.6	79930.3	0.562

On comparison of the blood parameters, there was significant higher mean of total leucocyte count among sepsis compared to the non-sepsis.

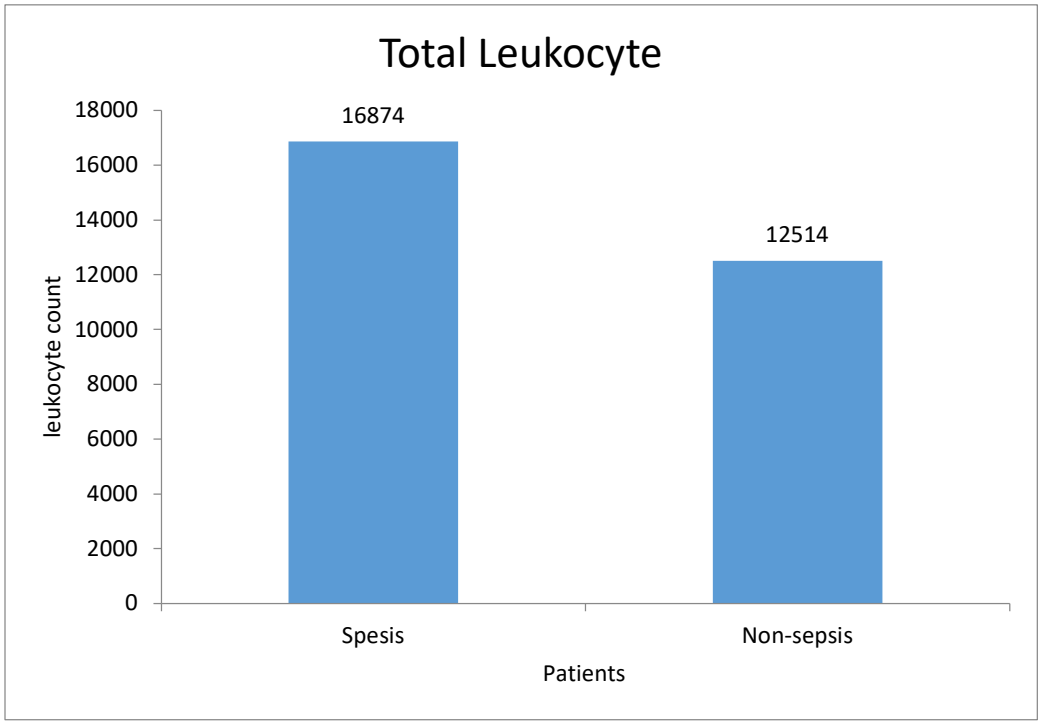


Figure 8: Comparison of the complete leukocyte count result between the groups

Table 4: Showing the patients with blood culture positive in sepsis patients

		Frequency	Percent
Blood Culture Among sepsis cases	Negative	32	54.2
	Positive	27	45.8
	Total	59	100.0

On culture, it was positive in 45.8% patients that is 27 out of 59 patients and 54.2% that is 32 out of 59 patients were negative blood culture.

Table 5: Variations of phospholipase a2 levels and procalcitonin in blood culture positive and negative samples

Markers	Blood culture positive (27 samples) Mean ± standard deviation	Blood culture negative (32 samples) Mean ± standard deviation	P value
Procalcitonin	46.08 ± 41.14	37.88 ± 38.14	0.431
Phospholipase A2	143.44 ± 60.652	72.85 ± 36.21	**<0.0001**

The above table shows comparison between procalcitonin and serum phospholipase A2 between blood culture positive and blood culture negative samples of sepsis. It showed that procalcitonin who had mean of 46.08 and standard deviation 41.14 in blood culture positive samples while in blood culture negative it was noted mean of 37.88 and standard deviation of 38.14. it was found non significant when it comes to culture suggesting the values were close in both blood culture positive and blood culture negative had close serum procalcitonin values in both groups. While it was also found that phospholipase A2 in blood culture positive had a mean of 143.44 and standard deviation of 60.65 and in blood culture negative had a mean of 72.85 with standard deviation of 36.21. suggesting that phospholipase A2 was significant suggestion it was higher in blood culture positive group with p value <0.0001.

Table 6: Comparison of procalcitonin and phospholipase A2 in different etiology (bacteria, viral)

markers	Bacterial etiology	Viral etiology	P value
Procalcitonin ng/ml	46.11±39.14	2.125±3.01	**0.0084**
Phospholipase A2 ng/ml	113.84± 57.49	28.5± 2.44	**0.0001**

In the study, bacterial etiology suggested higher mean and standard deviation in both procalcitonin (46.11± 39.14) and phospholipase A2 (113.84±57.49) while in viral etiology it was significantly lower , procalcitonin (2.125±3.01) and phospholipase A2 (28.5±2.44).It was observed from the table that both phospholipase A2 and procalcitonin are significant but phospholipase A2 was more sensitive than procalcitonin.

Table 7: Distribution of blood, urine or any other culture with different organism among patients in sepsis.

Culture	Sub Category		Number of subjects (%)
Blood culture	Gram positive 14 (23.73%)	Coagulase negative staphylococcus species	5 (8.47%)
		Methicillin resistant staphylococcus aureus	1 (1.69%)
		Staphylococcus Aureus	1 (1.69%)
		Staphylococcus epidermidis	2 (3.39%)
		Staphylococcus haemolyticus	3 (5.08%)
		Staphylococus epidermidis	1 (1.69%)
		Streptococcus Pyogenes (Group-A)	1 (1.69%)
	Gram negative 13 (22.03%)	Acinetobacter baumanii complex/haemolyticus	1 (1.69%)
		Acinetobacter Iwoffii group	1 (1.69%)
		Burkholderiacia cepacia complex	1 (1.69%)
		Enterobacter cloacae	1 (1.69%)
		Enterobacter species	1 (1.69%)
		Enterococcus faecalis	1 (1.69%)
		Enterococcus faecium	2 (3.39%)
		Escherichia coli	2 (3.39%)
		Klebsiella oxytoca	1 (1.69%)
		Klebsiella Pneumoniae	1 (1.69%)
		Proteus mirabills, Enterococcus faecalis	1 (1.69%)
	Nil		32 (54.24%)
Urine culture	Enterobacter Cloacae		1 (1.69%)
	Escherichia coli		8 (13.56%)
	Klebsiella pnemoniae		1 (1.69%)
	Nill		49 (83.05%)
Other culture	Coagulase negative staphylococcus		1 (1.69%)
	Enterobacter cloacae		2 (3.39%)
	Sputum - Klebsiella pneumoniae		1 (1.69%)

Out of 59 sepsis patients, blood culture showed gram positive in 14 (23.73%) patients, gram negative in 13 (22.03%) patients and 32 (54.24%) patients had none of them. Escherichia coli was observed in urine culture of 8 (13.56%) subjects, Enterobacter Cloacae was observed in 1 (1.69%) patients and Klebsiella pnemoniae was observed in 1 (1.69%) patient, 49 (83.05%) didn’t had any of them.

Table 8: Sensitivity between procalcitonin and phospholipase A2 in gram positive and gram negative bacteria.

	Gram positive	Gram negative	P value
Procalcitonin ng/lt	43.20± 41.48	51.466± 42.22	0.7136
Phospholipase A2 ng/lt	176.57± 45.73	110.75±56.18	**0.004**

The above table, it was observed that procalcitonin had a mean of 43.20 and standard deviation 41 in gram positive bacterial blood culture and mean of 51.466 and standard deviation of 42.22 in gram negative blood culture. on comparison for Phospholipase A2 it was observed that gram positive blood culture had mean of 176.57 with standard deviation of 45.73 and gram negative blood culture had mean of 110.74 with standard deviation of 56.18. It also shows p value derived by unpaired t test suggesting phospholipase A2 is significant when it comes to gram positive bacteria than gram negative bacteria, while procalcitonin has no correlation.

Table 9: Comparison of the blood parameters between the groups

	Group				p-value
	Sepsis		Non-sepsis		
	Mean	SD	Mean	SD	
Routine Blood Sugar mg/dl	174.4	107.1	190.1	100.5	0.560
Urea mg/dL	91.1	54.9	47.2	47.0	**0.001****
Creatinine mg/dL	3.33	2.43	2.83	2.05	0.458
SGOT U/L	124.8	272.1	32.0	11.6	0.124
SGPT U/L	96.0	212.2	28.5	15.6	0.151
Alkaline Phosphatase U/L	149.9	99.7	188.4	49.3	0.094

On comparison of various serum parameters between the sepsis and non sepsis groups, it was observed that there was significant higher blood urea in sepsis groups compared to the non-sepsis cases. The other parameters were found quite similar in both sepsis and non sepsis patients.

Table 10 : Comparison of the biomarkers between the groups

	Group				p-value
	Sepsis		Non-sepsis		
	Mean	SD	Mean	SD	
Serum Procalcitonin ng/dl	41.95	39.58	.34	.71	**0.001***
Serum PhospholipaseA2 ng/dl	105.2	60.1	17.9	4.9	**0.001***
Serum Lactate mmol/L	4.19	1.32	1.42	.42	**0.001***

On comparison of the markers to detect the sepsis, it was observed that all the three markers namely serum Procalcitonin, serum phospholipase A2 and serum lactate were highly significant when it comes to detecting the sepsis component in patients. ($p<0.05$)

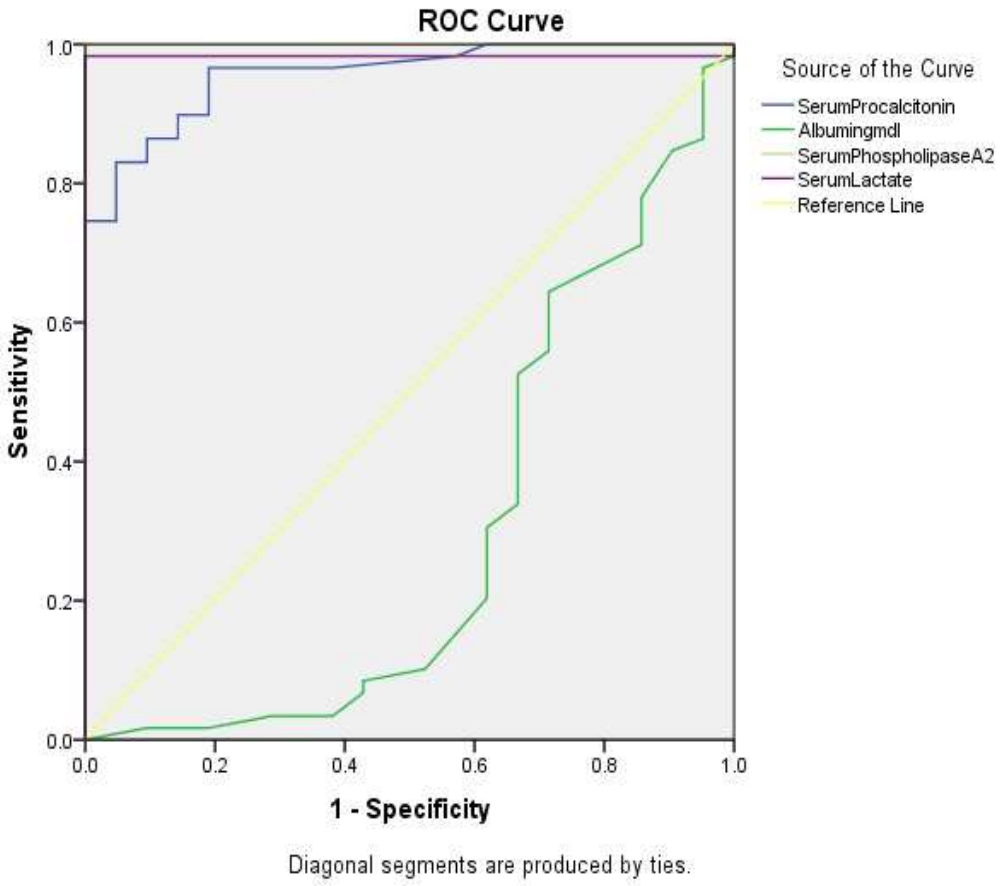


Figure 9: ROC analysis of each biomarker to detect the sepsis

Table 11: Diagnostic capacity of each biomarker to detect the sepsis

Test Result Variable(s)	Area	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
			Lower Bound	Upper Bound
Serum Procalcitonin	0.957	.000	.916	.997
Serum PhospholipaseA2	0.99	.001	0.989	0.999
Serum Lactate	0.983	.001*	.950	1.000

On assessment of the area under curve, the study documented serum phospholipase A2 had the higher significant AUC of 0.99 compared to the serum lactate AUC=0.983, serum procalcitonin AUC=0.957.

Table 12: Showing the diagnostic accuracy of the procalcitonin and the phospholipase A2 to detect the sepsis

	Procalcitonin	Phospholipase A2
Sensitivity	95.5	**98.6**
Specificity	88.9	**90.1**
PPV	82.6	**89.4**
NPV	88.1	**92.3**
Accuracy	95.7	**99.0**

The above table shows the difference among procalcitonin and phospholipase A2 in respective tests suggesting sensitivity of 95.5 specificity of 88.9 positive predictive value of 82.6 negative predictive value 88.1 in procalcitonin while in phospholipase A2 specificity is 98.6, sensitivity is 90.1, positive predictive value showing 89.4 and negative predictive value 92.3. on above tests accuracy was drawn showing 95.7 in procalcitonin while in phospholipase A2 was 99.0.

DISCUSSION

Present study aimed to assess the phospholipase A2 levels in sepsis patients and compare with procalcitonin level among them. The study has taken total of 80 patients out of which 59 were in sepsis category and 21 were in non sepsis category. The division took place according to q sofa score.

In present study total of 80 patients fulfilling inclusion criteria are included with mean age of 58±16.75yrs of age. 41.3% were female patients and 58.8% were male patients with male preponderance in the study. In similar to present study Tan TL et al., 2017 documented the mean age of 57.8±19.3yrs of age with male preponderance of 53% male patients and 47% were female patients.[65] We could conclude that mean age between the current study and above referred study were of similar range with male preponderance

In study by Nandi U et al., 2021 documented the mean age of 62yrs with male preponderance with 51% male patients and 49% female patients.[63]

On comparison of the vitals, the study documented the mean level of pulse rate and temperature was significantly higher and blood pressure was significantly lower among the sepsis cases compare to the non-sepsis cases. Also there was significant higher mean of total leucocyte count among sepsis compared to the non-sepsis. The study found significant higher mean of serum procalcitonin, serum phospholipase A2 and serum lactate were significantly higher among the patients with sepsis compared to the non-sepsis cases. ($p<0.05$)

Tan TL et al., 2021 documented that sPLA2-IIA biomarkers were recommended for BI detection in low and middle income countries.[64] The researcher by Nandi U et al., concluded that greater levels of sPLA2-IIA were linked to increased mortality in sepsis patients.

In the study conducted it was found that sepsis patients had higher total leukocyte count irrespective of etiology. (16874±8983 in sepsis while in non sepsis 12514±4237) According to receiver operator characteristic curve analysis in the study by Mansour KM et al., 2011 sPLA2 was more accurate at predicting bacterial infection (area under the curve = 0.89) than total white blood cell count (area under the curve = 0.71), and a value of more than 20 ng/mL had a sensitivity of 93%, specificity of 67%, positive predictive value of 39%, and negative predictive value of 97%.[56]

In the study, bacterial etiology suggested higher mean and standard deviation in phospholipase A2 (113.84±57.49) while in viral etiology it was significantly lower, phospholipase A2 (28.5±2.44). It was observed from the table that phospholipase A2 was way higher in bacterial infections. Mansour KM et al., 2011 similar to present study found that the difference in serum sPLA2 concentrations between individuals with viral infections (22±34 ng/mL) and those with bacterial infections (190±179 ng/mL) was statistically significant (P<.0001).[66] when compared between both studies showed similar results among the respective bacterial and viral etiology.

On culture, it was positive in 45.8% patients and 54.2% were negative blood culture. Most common gram positive organism was found to be staphylococcus while gram negative was found to be enterococcus in the culture. Among the urine culture, 83% were negative for the culture and 13.6% with escherichia coli, 1.7% with

Enterobacter cloacae and klebsiella pneumoniae. The urine culture was found to be negative in 83% and 17% with positive urine culture.

For Phospholipase A2 it was observed that gram positive blood culture had mean of 176.57 with standard deviation of 45.73 and gram negative blood culture had mean of 110.74 with standard deviation of 56.18 in the study done. It also shows p value suggesting phospholipase A2 is significant when it comes to gram positive bacteria than gram negative bacteria sPLA2-IIA levels were considerably greater in individuals who had a positive blood culture. Unlike procalcitonin, sPLA2-IIA levels were considerably greater in blood culture-positive individuals which was also observed in study done by Nandu et al., 2021 [63]

With the results from Nurhanan N et al., 2018 sPLA2-IIa showed good performance and can be used as a biomarker to diagnose both sepsis and bacterial infection, independently. It may assist in early antimicrobial administration and may be a useful tool, either alone or in combination with other markers in identifying sepsis and bacterial infection.[60] in the current study it was observed that phospholipase A2 specificity is 98.6, sensitivity is 90.1, positive predictive value showing 89.4 and negative predictive value 92.3 while sensitivity of 95.5 specificity of 88.9 positive predictive value of 82.6 negative predictive value 88.1 in procalcitonin was observed. Berg E et al., 2018 stated with his findings that sPLA2-IIA have potential as a marker in early diagnosis of patients presenting with possible sepsis. This marker appears to be sensitive and specific in distinguishing sick patients, especially those with a bacterial source of infection. This holds true in infections of multiple different organ systems. [59]

SPLA2-IIA looks to be an excellent biomarker for sepsis screening and bacterial infection diagnosis, whereas CD64 might be employed for bacterial infection screening. Both indicators, alone or in conjunction with other markers, may aid in early antibiotic administration decision making as found in study by Tan TL et al.2017. [58]

The area under curve, the study documented serum phospholipase A2 had the higher significant AUC of 0.99 compared to the serum lactate AUC=0.983, serum procalcitonin AUC=0.957. Patients with septic shock had significantly increased sPLA2, PCT levels. In the study done by Angel o et al., 2000 the sPLA2, PCT areas under the curves (ROC) were 0.896 and 0.765, respectively. The difference between the PCT and sPLA2 areas under the curves was significant (P 0.05) [67]. Taken together, the findings showed that LpPLA2, in comparison to other indicators of inflammation, may have a predictive role in sepsis, and provide preliminary evidence for future studies to investigate the clinical and pathophysiologic implications of LpPLA2 in sepsis. Study done by Huang et. Al., 2018 suggested that phospholipase A2 levels vary according to the severity of sepsis.[61]

Another study done by Toh Leong Tan et al., 2021 in comparison between phospholipase A2, procalcitonin, hsCRP, lactate and neutrophil count, it was found that Phospholipase A2 had significant ROC followed by hsCRP, neutrophils, procalcitonin and lactate being last in the curve. The same order was observed in specificity and sensitivity of the components tested.[68]

Endo S et al., 1995 studied the PLA2 with other inflammatory markers like IL-6, TNF-alpha and IL-8. They documented that the patients who died from sepsis had much greater plasma levels of type II PLA2 than those who survived the disease. There was a strong link between type II PLA2 and TNF-alpha and IL-6. Type II PLA2, TNF-

alpha, IL-6, and IL-8 may be beneficial as disease severity indexes. TNF-alpha and IL-6 seem to increase the synthesis of type II PLA2 in the plasma of sepsis patients. [55]

In concordance with present study, Endo S et al., 1995 documented PLA2-II concentration associated strongly with C-reactive protein (CRP) concentration (r =.613, P =.001) but not with pancreatic PLA2 concentration (r =.089, P =0.365). The serum concentration of PLA2-II can be used in conjunction with CRP values to differentiate bacterial from viral infection.[54]

The present single centric study documented the diagnostic capacity of the PLA2 as important biomarker for sepsis in par with the other established markers like procalcitonin and lactic acids. The conjugated use of the PLA2 will help in early detecting and managing the patients in reducing the morbidity and mortality.

SUMMARY

The present cross sectional study was conducted among the patients more than 18yrs of age fulfilling the inclusion criteria admitted in KLES Dr Prabhakar Kore Hospital, Belagavi during the period of January 2021 to December 2021were analysed. All patients more than 18yrs age meeting the qSOFA of more or equal to 2 were included. the patients less than 18yrs, with rheumatoid arthritis, asthma, systemic lupus erythematous, any malignancy and patients on antibiotics for more than 3 days of duration were excluded from the study. the patients were assessed for the physical and biochemical examination after obtaining the informed consent. the patients were analysed for the biomarkers in blood for sepsis which included the procalcitonin, lactate and phospholipase A2. All patient information was gathered using a pre-made proforma and entered into an excel spreadsheet. The entire study was carried out using SPSS v21 running on Windows 10 with a p-value threshold of 0.05 being considered statistically significant.

Present study aimed to assess the phospholipase A2 levels in sepsis patients and compare with procalcitonin level among them.

- In present study total of 80 patients fulfilling inclusion criteria are included with mean age of 58±16.75yrs of age.
- Among the participants, 41.3% were female patients and 58.8% were male patients with male preponderance in the study.
- On culture, it was positive in 45.8% patients and 54.2% were negative blood culture.

- Among the urine culture, 83% were negative for the culture and 13.6% with escherichia coli, 1.7% with Enterobacter cloacae and klebsiella pneumoniae.

- The urine culture was found to be negative in 83% and 17% with positive urine culture.

- Other organism isolated were the Coagulase negative staphylococcus, Enterobacter cloacae, klebsiella pneumoniae.

- On comparison of the vitals, the study documented the mean level of pulse rate and temperature was significantly higher and blood pressure was significantly lower among the sepsis cases compare to the non-sepsis cases.

- On comparison of the blood parameters, there was significant higher mean of total leucocyte count among sepsis compared to the non-sepsis.

- On comparison of the serum parameters to, there was significant higher blood urea compared to the non-sepsis cases. the other parameters were found to be comparable.

- Phospholipase A2 is found to be significantly raised in sepsis when compared with non sepsis patients.

- Phospholipase A2 when compared in between etiology was found significantly higher in bacterial microbiology than viral microbiology.

- In further classification between the bacterial etiology into gram positive and gram negative it was found that Phospholipase A2 levels were considerably higher in gram positive suggesting specificity towards gram positive.

- On comparison it was also found that the sensitivity and specificity was higher for Phospholipase A2 than Procalcitonin.

- It was also found that 12 cases with procalcitonin less than 10 but phospholipase A2 was raised more than 30 μ/ lt having a mean ± standard deviation of 86.75± 53.25 while in procalcitonin it was 1.91± 2.085.

- On comparison of the markers to detect the sepsis, the study found significant higher mean of serum procalcitonin, serum phospholipase A2 and serum lactate were significantly higher with sepsis compared to the non-sepsis cases. ($p<0.05$)

- On assessment of the area under curve, the study documented serum phospholipase A2 had the higher significant AUC of 0.99 compared to the serum lactate AUC=0.983, serum procalcitonin AUC=0.957.

CONCLUSION

1) Phospholipase A2 was observed to be significantly raised in patients with sepsis than non sepsis.

2) Etiology wise, it was observed that phospholipase A2 was significantly higher in bacterial than viral.

3) It was also observed that phospholipase A2 was statistically higher in gram positive than gram negative organisms in blood culture.

4) Around 12 patients had higher phospholipase A2 value inspite procalcitonin being on the lower side (<10) indicating that phosphoslipase A2 can be used as an additional marker.

5) In the current study it was found that sensitivity and specificity of phospholipase A2 was higher than procalcitonin.

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STRENGTH OF STUDY:

1) Other studies have also been proved that phospholipase A2 novel marker in comparison with procalcitonin and lactate in detecting sepsis.
2) The marker has got high sensitivity and specificity in detecting sepsis, especially of bacterial origin.
3) It was found that very few studies were done regarding serum phospholipase A2 especially in our country.

LIMITATIONS OF STUDY:

1) In the study we couldn't correlate between the antibiotic initiation and consecutive levels of phospholipase A2 levels.
2) We could not compare phospholipase A2 levels with other sepsis markers like Ferritin, C-reactive protein, Lactoferrin, Neopterin, Tumor necrosis factor α (TNF-α), interleukin 6,8,10 etc.
3) The number of sample size taken was small for the study.
4) Although there were significant samples with high levels of phospholipase A2 but procalcitonin being on the lower range, significance correlation couldn't be derived from the study.

ANNEXURE I: CONSENT FROMS

INFORMED CONSENT

Dear Mr./Mrs./Dr._____, you are kindly requested to enroll yourself in a research study tilted, "TO ASSESS PHOSPHOLIPASE A2 IN SEPSIS AND NON SEPSIS PATIENTS AT KLE'S PRABHAKAR KORE HOSPITAL" being conducted by **REGISTRATION NO: BG0120020,** a post graduate student in M.D. General Medicine and the study will be carried out under the direct supervision and guidance of Dr. _____, Professor and Unit Chief, Department of General Medicine, Jawaharlal Nehru Medical College, Belgaum.

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

Your participation in study is voluntary. As the study is record based, we will be collecting some of your lab reports to analyze in order to find some correlation, as mentioned above. Your decision whether or not to participate in the study will not affect your treatment in any form. If you decide to participate you are free to withdraw at any time.

TITLE OF THE STUDY:

"TO ASSESS SERUM PHOSPHOLIPASE A2 IN SEPSIS AND NON SEPSIS PATIENTS"

PURPOSE OF THE STUDY:

To assess the levels of phospholipase a2 in sepsis and non sepsis patients.

PROCEDURES INVOLVED:

If you agree to enroll yourself in my study, I will require some of your investigations accordingly, as mentioned below.

1) Complete blood count
2) Liver function test
3) Renal function test
4) Serum phospholipase A2
5) Chest x-ray
6) Procalcitonin
7) Lactate

RISKS AND BENEFITS:

There are no potential risks involved in this study.

Benefits of taking part in this research:

- To establish a proven phospholipase A2 as marker of sepsis in our area.

VOLUNTARY PARTICIPATION / WITHDRAWAL FROM THE STUDY:

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

ALTERNATIVES:

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

PRIVACY AND CONFIDENTIALITY:

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

The only people to know that you are a research subject are members of the research team. No information about you will be disclosed to other without your written permission except:

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

AUTHORIZATION TO PUBLISH RESULT:

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

FINANCIAL INCENTIVES FOR PARTICIPATION:

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

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

COMPENSATION:

In the event that you become injured as a result of taking part in this study, treatment will be offered to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum, or you will be given information about where to receive medical care.

However, no reimbursement, compensation or free medical care will be given.

QUESTIONS/CONTACT DETAILS:

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

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

1)Dr. HARSHA HEGDE,
Chairperson, J.N.M.C, IEC and Scientist D,
ICMR, National Institute of Traditional Medicine.

2. Dr. _____,
Professor and Unit Head, Dept of General Medicine, INMC, Belagavi.

3. REGISTRATION NO: BG0120020
Investigator, PG in General Medicine,
INMC, Belagavi.

CONSENT FORM

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

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

:.....

.....

Signature / Left thumb impression:.....
of the participant

Name of the legally authorized representative / guardian :.....

Signature / Left thumb impression :.....

Witness' name :.....

Signature / Left thumb impression :.....

Investigator's name and signature :.....

Date:

Place

माहितीपूर्ण संमती

प्रिय श्रीमती / श्रीमती / डॉ. _____, आपणास विनम्र विनंती आहे की आपणास झूकलेल्या एका संशोधन अभ्यासामध्ये नाव नोंदवावे "केएलई डॉ. प्रभाकर कोरे हॉस्पिटल येथे -"सेप्सिस आणि नॉन सेप्सीस रुग्णांमध्ये फॉस्फोलाइपेस ए 2 चे मूल्यांकन करणे ".

REG. NO: BG0120020 म.डी. जनरल मेडिसीन मधील पदव्यूत्तर विद्यार्थी घेत आहेत आणि हा अभ्यास, जवाहरलाल नेहरू मेडिकल कॉलेज, बेळगाव येथील प्राध्यापक आणि युनिट मुख्य, सामान्य चिकित्सा विभाग, जवाहरलाल नेहरू मेडिकल कॉलेज, बेळगाव यांच्या थेट देखरेखीखाली आणि मार्गदर्शनानुसार केला जाईल.

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

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

बेळगावी.

अभ्यासाचे शीर्षक:

"केएलई डॉ. प्रभाकर कोरे हॉस्पिटल येथे -"सेप्सिस आणि नॉन सेप्सीस रुग्णांमध्ये फॉस्फोलाइपेस ए 2 चे मूल्यांकन करणे ".

अभ्यासाचा हेतू:

सेप्सिस आणि नॉन सेप्सीस रुग्णांमध्ये फॉस्फोलाइपेस ए 2 च्या पातळीचे मूल्यांकन करणे.

प्रक्रिया समाविष्ट:

आपण माझ्या अभ्यासामध्ये स्वत: ला नावनोंदणी करण्यास सहमती दर्शविल्यास, मला आपल्यापैकी काही आवश्यक असतील त्यानुसार तपास, खाली नमूद केल्याप्रमाणे.

1) संपूर्ण रक्त गणना
2) यकृत कार्य चाचणी
3) रेनल फंक्शन टेस्ट
4) लिपिड प्रोफाइल
5) सीरम फॉस्फोलाइपेस ए 2
6) छातीचा एक्स-रे
7) प्रोकॅलिसिटोनिन
8) दुग्धशाळा

जोखीम आणि फायदे :

या अभ्यासामध्ये कोणतीही संभाव्य जोखीम गुंतलेली नाही.

या संशोधनात भाग घेण्याचे फायदे:

आमच्या क्षेत्रात सेप्सिसचे मार्कर म्हणून सिद्ध फॉस्फोलिपेस ए 2 स्थापित करणे

ऐच्छिक सहभाग / अभ्यासामधून पैसे काढणे :

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

विकल्प:

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

गोपनीयता आणि गोपनीयता :

अभ्यासाच्या सहभागादरम्यान आपण गोळा केलेला किंवा जाहीर केलेला सर्व डेटा पूर्णपणे गोपनीय ठेवला जाईल. अर्थात कोर्सच्या दरम्यान ओळख जाहीर करणे आवश्यक झाले तर ते तुमच्या माहिती व लेखी संमतीनंतरच केले जाईल. आपण संशोधन विषय आहात हे फक्त लोकांनाच माहित आहे की ते संशोधन पथकाचे सदस्य आहेत. आपल्या लेखी परवानगीशिवाय इतर आपल्याबद्दल कोणतीही माहिती उघड केली जाणार नाही:

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

निकाल प्रकाशित करण्यासाठी अधिकृतता:

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

सहभागासाठी आर्थिक प्रोत्साहन :

या अभ्यासाच्या हेतूने आपल्यावर कोणत्याही प्रकारची अतिरिक्त किंमत आकारली जाणार नाही.
हे निव्वळ संशोधनाच्या कल्पनेने केले जात आहे आणि अभ्यासाचा सर्व खर्च तपासनीस करेल.

भरपाई:

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

प्रश्न / संपर्क तपशील:

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

डॉ. हर्षा हेगडे
अध्यक्ष, जेएनएमसी
आयईसी आणि सायंटिस्ट डी आयसीएमआर,
राष्ट्रीय पारंपारिक औषध संस्था बेळगवी.
दूरध्वनी क्रमांक: 9480422500

प्राध्यापक आणि युनिट मुख्य,
सामान्य औषध विभाग,
जे.एन.एम.सी, बेळगावी.

REG. NO: BG0120020

अन्वेषक, पदव्युत्तर विद्यार्थी
सामान्य औषध विभाग,
जे.एन.एम.सी, बेळगावी.

संमती फॉर्म

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

सहभागी किंवा कायदेशीररित्या अधिकृत प्रतिनिधीची सही / डावा अंगठा प्रिंट

सहभागीचे नाव: ....

स्वाक्षरी / डावा अंगठा ठसा: ....

सहभागीचा

कायदेशीररीत्या अधिकृत नाव: ....

प्रतिनिधी / पालक

स्वाक्षरी / डावा अंगठा ठसा: ....

साक्षीचे नाव: ....

स्वाक्षरी / डावा अंगठा ठसा: ....

अन्वेषकांचे नाव आणि स्वाक्षरी: ....

तारीख:

ठिकाण:

ತಿಳುವಳಿಕೆಯ ಸಮ್ಮತಿ

ಆತ್ಮೀಯ ಶ್ರೀ / ಶ್ರೀ. / ಡಾ. _____, ಓರೆಯಾಗಿರುವ ಸಂಶೋಧನಾ ಅಧ್ಯಯನಕ್ಕೆ ನಿಮ್ಮನ್ನು ಸೇರಿಸಲು ನಿಮ್ಮನ್ನು ದಯೆಯಿಂದ ವಿನಂತಿಸಲಾಗಿದೆ, "ಕೆಎಲ್ಇ ಡಾ. ಪ್ರಭಾರ್ ಕೋರೆ ಆಸ್ಪತ್ರೆಯಲ್ಲಿ ಸೆಪ್ಸಿಸ್ ಮತ್ತು ನಾನ್ ಸೆಪ್ಸಿಸ್ ರೋಗಿಗಳಲ್ಲಿ ಫಾಸ್ಫೋಲಿಪೇಸ್ ಎ 2 ಅನ್ನು ನಿರ್ಣಯಿಸಲು " ಎಂ.ಡಿ. ಜನರಲ್ ಮೆಡಿಸಿನ್‌ನಲ್ಲಿ ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿ REG. NO: BG0120020 , ಅವರು ನಡೆಸಲಿದ್ದಾರೆ ಮತ್ತು ಬೆಳಗಾವಿನ ಜವಾಹರಲಾಲ್ ನೆಹರು ವೈದ್ಯಕೀಯ ಕಾಲೇಜಿನ ಜನರಲ್ ಮೆಡಿಸಿನ್ ವಿಭಾಗದ ಪ್ರೊಫೆಸರ್ ಮತ್ತು ಯುನಿಟ್ ಮುಖ್ಯಸ್ಥ ಅವರ ನೇರ ಮೇಲ್ವಿಚಾರಣೆ ಮತ್ತು ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ ಈ ಅಧ್ಯಯನವನ್ನು ನಡೆಸಲಾಗುವುದು.

ಅಧ್ಯಯನದ 'ವಿಷಯ' / ಭಾಗವಹಿಸುವವರಿಗೆ ನೀವು ನಿಗದಿಪಡಿಸಿದ ಮಾನದಂಡಗಳಿಗೆ ಸರಿಹೊಂದುವಂತೆ ಇದರಲ್ಲಿ ಭಾಗವಹಿಸಲು ನಿಮ್ಮನ್ನು ವಿನಂತಿಸಲಾಗಿದೆ.

ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ. ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ನಿಮ್ಮನ್ನು ಕೆಲವು ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲಾಗುತ್ತದೆ ಮತ್ತು ನಿಮ್ಮ ಉತ್ತಮ ಜ್ಞಾನಕ್ಕೆ ನೀವು ಉತ್ತರಿಸಬೇಕಾಗುತ್ತದೆ. ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಬೇಕೆ ಅಥವಾ ಬೇಡವೇ ಎಂಬ ನಿಮ್ಮ ನಿರ್ಧಾರವು ನಿಮ್ಮ ಚಿಕಿತ್ಸೆಯ ಮೇಲೆ ಯಾವುದೇ ರೂಪದಲ್ಲಿ ಪರಿಣಾಮ ಬೀರುವುದಿಲ್ಲ. ನೀವು ಭಾಗವಹಿಸಲು ನಿರ್ಧರಿಸಿದರೆ ನೀವು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಹಿಂತೆಗೆದುಕೊಳ್ಳಬಹುದು.

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ:

"ಕೆಎಲ್ಇ ಡಾ. ಪ್ರಭಾರ್ ಕೋರೆ ಆಸ್ಪತ್ರೆಯಲ್ಲಿ ಸೆಪ್ಸಿಸ್ ಮತ್ತು ನಾನ್ ಸೆಪ್ಸಿಸ್ ರೋಗಿಗಳಲ್ಲಿ ಫಾಸ್ಫೋಲಿಪೇಸ್ ಎ 2 ಅನ್ನು ನಿರ್ಣಯಿಸಲು "

ಅಧ್ಯಯನದ ಉದ್ದೇಶ:

ಸೆಪ್ಸಿಸ್ ಮತ್ತು ಸೆಪ್ಸಿಸ್ ಅಲ್ಲದ ರೋಗಿಗಳಲ್ಲಿ ಫಾಸ್ಫೋಲಿಪೇಸ್ ಎ 2 ಮಟ್ಟವನ್ನು ನಿರ್ಣಯಿಸಲು.

ಒಳಗೊಂಡಿರುವ ಕಾರ್ಯವಿಧಾನಗಳು:

ನನ್ನ ಅಧ್ಯಯನಕ್ಕೆ ನಿಮ್ಮನ್ನು ಸೇರಿಸಲು ನೀವು ಒಪ್ಪಿದರೆ, ನಿಮ್ಮಲ್ಲಿ ಕೆಲವು ನನಗೆ ಅಗತ್ಯವಿರುತ್ತದೆ ಕೆಳಗೆ ಹೇಳಿದಂತೆ ತನಿಖೆಗಳು.

1) ರಕ್ತದ ಎಣಿಕೆ ಪೂರ್ಣಗೊಳಿಸಿ
2) ಯಕೃತ್ತಿನ ಕಾರ್ಯ ಪರೀಕ್ಷೆ
3) ಮೂತ್ರಪಿಂಡದ ಕಾರ್ಯ ಪರೀಕ್ಷೆ
4) ಲಿಪಿಡ್ ಪ್ರೊಫೈಲ್
5) ಸೀರಮ್ ಫಾಸ್ಫೋಲಿಪೇಸ್ ಎ 2
6) ಎದೆಯ ಕ್ಷ-ಕಿರಣ
7) ಪ್ರೊಕಾಲ್ಸಿಟೋನಿನ್
8) ಲ್ಯಾಕ್ಟೇಟ್

ಅಪಾಯ ಮತ್ತು ಪ್ರಯೋಜನಗಳು :

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಯಾವುದೇ ಸಂಭಾವ್ಯ ಅಪಾಯಗಳಿಲ್ಲ.

ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವ ಪ್ರಯೋಜನಗಳು :

ನಮ್ಮ ಪ್ರದೇಶದಲ್ಲಿ ಸೆಪ್ಸಿಸ್ನ ಮಾರ್ಕರ್ ಆಗಿ ಸಾಬೀತಾದ ಫಾಸ್ಫೋಲಿಪೇಸ್ ಎ 2 ಅನ್ನು ಸ್ಥಾಪಿಸುವುದು

ಸ್ವಯಂಪ್ರೇರಿತ ಭಾಗವಹಿಸುವಿಕೆ / ಅಧ್ಯಯನದಿಂದ ಹಿಂತೆಗೆದುಕೊಳ್ಳುವಿಕೆ:

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

ಪರ್ಯಾಯಗಳು :

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

ಗೌಪ್ಯತೆ ಮತ್ತು ಗೌಪ್ಯತೆ :

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

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

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

ಫಲಿತಾಂಶಗಳನ್ನು ಪ್ರಕಟಿಸಲು ಅಧಿಕಾರ:

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

ಭಾಗವಹಿಸುವಿಕೆಗೆ ಆರ್ಥಿಕ ಪ್ರೋತ್ಸಾಹ:

ಈ ಅಧ್ಯಯನದ ಉದ್ದೇಶಕ್ಕಾಗಿ ಯಾವುದೇ ಹೆಚ್ಚುವರಿ ವೆಚ್ಚಗಳು ನಿಮ್ಮ ಮೇಲೆ ಆಗುವುದಿಲ್ಲ.

ಇದನ್ನು ಸಂಪೂರ್ಣವಾಗಿ ಸಂಶೋಧನೆಯ ಆಲೋಚನೆಯೊಂದಿಗೆ ಮಾಡಲಾಗುತ್ತಿದೆ ಮತ್ತು ಅಧ್ಯಯನದ ಎಲ್ಲಾ ವೆಚ್ಚವನ್ನು ತನಿಖಾಧಿಕಾರಿ ಭರಿಸುತ್ತಾರೆ.

ಪರಿಹಾರ:

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

ಪ್ರಶ್ನೆಗಳು / ಸಂಪರ್ಕ ವಿವರಗಳು:

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

ಡಾ. ಹರ್ಷ ಹೆಗ್ಡೆ
ಅಧ್ಯಕ್ಷರು, ಜೆಎನ್ಎಂಸಿ
ಐಇಸಿ ಮತ್ತು ವಿಜ್ಞಾನಿ ಡಿ
ಐಸಿಎಂಆರ್, ನ್ಯಾಷನಲ್ ಇನ್ಸ್ಟಿಟ್ಯೂಟ್ ಆಫ್ ಟ್ರೆಡಿಶನಲ್ ಮೆಡಿಸಿನ್
ಬೆಳಗಾವಿ - ದೂರವಾಣಿ ಸಂಖ್ಯೆ. 9480422500

ಪ್ರೊಫೆಸರ್ ಮತ್ತು ಯುನಿಟ್ ಹೆಡ್,
ಜನರಲ್ ಮೆಡಿಸಿನ್ ಇಲಾಖೆ,
ಜೆಎನ್ಎಂಸಿ, ಬೆಳಗಾವಿ.

REG. NO: BG0120020

ತನಿಖಾಧಿಕಾರಿ, ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿ
ಜನರಲ್ ಮೆಡಿಸಿನ್ ಇಲಾಖೆ,
ಜೆಎನ್ಎಂಸಿ, ಬೆಳಗಾವಿ.
ಮೊಬೈಲ್ - 7676185195

ಒಪ್ಪಿಗೆ ಪತ್ರ

ಕೆಳಗೆ ಸಹಿ ಮಾಡುವ ಮೂಲಕ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನಾನು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪುತ್ತೇನೆ. ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಹಿಂತೆಗೆದುಕೊಳ್ಳಬಹುದು. ಈ ಫಾರ್ಮ್ಗೆ ಸಹಿ ಮಾಡುವ ಮೂಲಕ ನಾನು ನನ್ನ ಯಾವುದೇ ಕಾನೂನು ಹಕ್ಕುಗಳನ್ನು ಬಿಟ್ಟುಕೊಡುತ್ತಿಲ್ಲ. ಕೆಳಗಿನ ನನ್ನ ಸಹಿ ನಾನು ಈ ಒಪ್ಪಿಗೆಯ ಫಾರ್ಮ್ ಅನ್ನು ಓದಿದ್ದೇನೆ ಅಥವಾ ಈ ಸಮ್ಮತಿಯ ಫಾರ್ಮ್ ಅನ್ನು ನನಗೆ ಓದಿದ್ದೇನೆ ಮತ್ತು ಎಲ್ಲಾ ಪ್ರಶ್ನೆಗಳಿಗೆ ಉತ್ತರಿಸಿದೆ ಎಂದು ಸೂಚಿಸುತ್ತದೆ

ಭಾಗವಹಿಸುವವರ ಅಥವಾ ಕಾನೂನುಬದ್ಧವಾಗಿ ಅಧಿಕೃತ ಪ್ರತಿನಿಧಿಯ ಸಹಿ / ಎಡ ಹೆಬ್ಬೆರಳು ಮುದ್ರಣ ಭಾಗವಹಿಸುವವರ ಹೆಸರು: ....

ಸಹಿ / ಎಡ ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆ: ....

ಭಾಗವಹಿಸುವವರ ಕಾನೂನುಬದ್ಧವಾಗಿ ಅಧಿಕಾರ ಪಡೆದವರ ಹೆಸರು: ....

ಪ್ರತಿನಿಧಿ / ರಕ್ಷಕ ಸಹಿ / ಎಡ ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆ: ....

ಸಾಕ್ಷಿ ಹೆಸರು: ....

ಸಹಿ / ಎಡ ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆ: ....

ತನಿಖಾಧಿಕಾರಿ ಹೆಸರು ಮತ್ತು ಸಹಿ: ....

ದಿನಾಂಕ:

ಸ್ಥಳ:

ANNEXURE II: PROFORMA

Case no:

Name:

Age:

OP/IP No:

Address:

DOA:

Complaints at Presentation:

Past History:

Personal history:

Antibiotic history:

INVESTIGATIONS:

1) COMPLETE BLOOD COUNT-

Hb	Platelets	Total Leukocyte Count	Neutrophils	Lymphocytes

2) LIVER FUNCTION TEST

Direct Bilirubin	Indirect Bilirubin	SGOT	SGPT	Alkaline Phosphatase	Albumin

3) RENAL FUNCTION TEST

Sr. Creatinine	Urea	Electrolytes	Na	K	HCO3	Cl

4) SERUM SECRETORY PHOSPHOLIPASE A2

5) CHEST XRAY

6) RANDOM BLOOD SUGAR

7) SERUM PROCALCITONIN

8) LACTATE

9) Blood culture: organism if present-

10) Urine culture: organism if present-

11) Any other culture if taken-

					Clinical						Complete Blood Count															
S.No	IP No	Age	Gender	DOA	P	Blood Preassure	Temp. (°F)	Systemic Examination	Commodalities	Final diagnosis	Total Leukocyte Count /µL	Heamoglobin (g/dL)	Platelets /µL	Routine Blood Sugar	Urea (mg/dL)	Creatinine (mg/dL)	SGOT (U/L)	SGPT (U/L)	Alkaline Phophatase (U/L)	Urine- Routine- WBC / HPF	Blood Culture	Urine Culture	Any Other Culture	Serum Procalcitonin	Albumin (gm/dl)	Serum Phospholipase A2	Serum Lactate	CXR
1	1063991	73	F	08-12-2021	126	110/60 with 4 ml/ hr norad	100.6	N/A	diabetes, hypertension	urinary tract infection with acute renal insufficiency	32000	12.5	205000	552	127	3.76	46	55	90	41	Nill	Nill		0.39	3	32	2.56	WNL
2	1064536	52	F	16/08/2021	110	100/60 with 8ml/hr norad an 2.4 ml/hr vasopressin	101	right renal angle tenderness	diabetes, hypertension	Right pyelonephritis in acute renail failure	19700	9	484000	58	245	13.7	12	10	260	1355	Coagulase negative staphylococcus species	Nill		0.47	3.6	128	4.5	WNL
3	103966	33	M	02-02-2022	122	100/60 mmhg with 2 ml/hr norad	99.5	N/A		urinary tract infectiom	11000	9.3	461000	109	17	1.13	33	43	109	110	Burkholderiacia cepacia complex	Enterobacter Cloacae		0.15	3.5	45	3.6	WNL
4	1061196	50	F	24/07/2021	115	90/60 mmhg with 4ml/hr norad	100	N/A	diabetes	uti with aki	13000	10.5	49000	82	135	2.97	118	67	97	4	Enterococcus faecium	Nill		52.65	2.1	248	6.76	WNL
5	1061847	77	M	29/07/2021	112	100/70 mmhg with 5 ml/hr norad	102	right hemiplegia(old), bed sore +	old cva , diabetes	meningitis with bed sore	20200	14.5	315000	129	66	1.33	168	46	103		Staphylococcus epidermidis	Nill	CSF - Coagulase negative staphylococus	0.63	2.6	100	2.34	WNL
6	1071868	72	M	29/09/2021	126	90/60 mmhg with 10 ml/hr norad and 2.4 ml/hr vasopreesin	101	right side crepts	diabetes	right side pneumonia with acute kidney injury	15400	14	237000	129	76	3.96	45	26	63		Streptococcus Pyogenes (Group-A)	Nill		100	2.7	265	3.1	RT L/L consolidation
7	1071130	76	M	24/09/2021	122	90/60 mmg with 10 ml/hr norad and 2.4 ml/hr vasopressin	100	N/A	diabetes	sepsis 2 gluteal abscess 2 to im injection in mods	15500	11.1	196000	108	185	6.64	117	48	492		Coagulase negative staphylococcus species	Nill		24.93	2.4	180	4.5	WNL
8	1070595	30	F	22/09/2021	136	90/60 mmh with 6 ml/hr norad and 2.4 ml/hr	99.6	blanching all over body		Rickettsial fever	8900	10.3	14000	97	60	4.23	80	44	95		Nill	Nill		22.55	2.7	104	4.8	WNL
9	1070968	60	M	23/09/2021	128	90/60mmhg with 5ml/hr norad	101	right side lobe	diabetes	right sided pneumonia	11100	11.3	140000	107	52	1.03	164	77	52		Coagulase negative staphylococcus species	Nill		36.92	3	205	5.6	rt L/L consolidation
10	1078307	30	M	11-02-2021	110	90/60 mmhg with 4 ml/hr and 2.4 ml/hr	100	b/l crepts		atypical pneumonia in mods	25700	9.4	28000	311	135	4.42	200	58	118		Nill	Nill		66.22	2	68	4.7	B/L INFILTRATES
11	1082024	23	M	23/11/2021	115	100/60mmhg with 2ml/hr hg	99.9	blanching all over body		Dengue and MODS	11000	12	33000	191	102	4.62	148	158	139		Nill	Nill		5.7	2.4	28	2.3	
12	1082649	40	M	27/11/2021	126	90/60 mmhg with 3ml/hr norad	100	b/l crepts	hypertension, diabetes, recent cva	bilateral pnuemonia(VAP) in mods	25300	8.2	80000	159	75	1.69	78	49	59		Acinetobacter baumanii complex/haemolyticus	Nill	Enterobacter cloacae	23.9	1.9	96	3.6	B/L INFILTRATES
13	1066522	73	F	29/08/2021	115	90/60	99.4	b/l crepts	diabetes	viral pneumonia	23000	11.3	229000	95	187	3.59	22	12	189		Nill	Nill		4.77	2.4	32	4.7	B/L INFILTRATES
14	1059106	56	M	07-11-2021	120	90/60 mmhg with 3ml/norad	100	N/A	diabetes , hypertension	pneumonia with acute kidnay injury	15900	10	391000	139	116	4.15	22	30	113		Nill	Nill	Sputum - Klebsiella pneumoniae	100	3.2	136	4.6	rt L/L consolidation
15	1065919	74	M	25/08/2021	116	100/60 mmhg with 2 ml/hr norad	102	N/A	diabetes	urinary tract infection with acute renal insufficiency	17000	11.3	170000	85	42	1.66	49	12	82	219	Nill	Escherichia coli		90.26	3.3	122	3.5	
16	1067019	55	F	09-01-2021	126	100/60 mmhg with 2 ml/hr norad	102	right side hemiplegia with b/l crepts- intubated	diabetes	post stroke intubated with VAP in aki	18100	7.6	268000	50	42	2.65	36	14	81	260	Coagulase negative staphylococcus species	Nill	ET-Enterobacter cloacae	100	2.7	156	5.6	B/L INFILTRATES
17	1065242	43	M	20/08/2021	132	100/60 mmhg with 2 ml/hr norad	101	N/A	diabetes	urinary tract infection with acute renal insufficiency	7000	17.5	158000	233	57	5.01	120	56	33	75	Staphylococcus haemolyticus	Nill		100	3.3	186	5.7	
18	1065351	32	F	21/08/2021	78	90/60 mmhg with 3ml/hr norad with 2.4 ml/hr vasopressin	100	blanching all over body		Dengue	14600	11	47000	78	42	0.65	1988	1506	271		Nill	Nill		0.36	3	30	2.6	
19	1065605	78	M	23/08/2021	126	90/60 mmh with 6 ml/hr norad and 2.4 ml/hr	101	left hemiplegia	diabetes ,old cva	sepsis 2 to bed sore	37700	13.2	232000	177	96	3.68	82	49	98		Staphylococcus haemolyticus	Nill		16.85	3.1	124	3.6	
20	1066190	60	M	26/08/2021	133	90/60mmhg with 5ml/hr norad	102	b/l crepts	diabetes	atypical pnuemonia	13200	13	101000	291	25	0.7	12	10	65		Klebsiella Pneumoniae	Escherichia coli		88.45	4	136	3.9	B/L INFILTRATES
21	1064834	52	F	18/08/2021	121	90/60mmhg with 3ml/hr norad	99.4	b/l crepts	diabetes	viral pnuemonia with acute renail insuffiency	8600	10.2	284000	90	54	2.1	35	26	82	10	Nill	Nill		1.22	3.1	31	2.7	B/L INFILTRATES
22	1064591	55	F	17/08/2021	118	100/70 mmhg with 6 ml/hr norad and 2.4 ml/hr vasopressin	100	rt hypochondriac tenderness	hypertension	Leptospirosis AKI	19300	9.6	336000	184	148	5.61	44	63	193	0	Nill	Nill		2.95	3.7	85	3	
23	1064257	26	M	14/08/2021	68	90/60 mmhg 3.5ml/hr norad	101	blanching all over body with purpura all over body with right hypochondriac tenderness	hypertension	Dengue with deranged liver function	13900	14.2	49000	128	21	0.72	82	79	244		Nill	Nill		0.36	3.5	25	2.5	
24	1064009	60	M	13/08/2021	130	90/60mmhg with 10 ml/hr norad and 2.4ml/hr vasopressin	102	bed sore on right gluteal region -pressure sore	diabetes hypertension old cva	sepsis sec to abscess	6200	8.7	104000	207	86	2.48	25	17	115		Staphylococcus Aureus	Nill		41.09	2.5	194	4.6	
25	1062475	70	M	08-02-2021	75	90/60 mmhg with 2.5 ml/hr norad	100	right hypochondriac tenderness	diabetes	typhoid in MODS	9800	10.5	111000	98	117	8.26	219	209	171		Nill	Nill		2.56	2.3	56	5.8	
26	1061913	54	M	29/07/2021	110	90/60mmhg with 4ml/hr norad	99.1	right side crepts	hypertension	right lobar pneumonia with aki	15100	12	276000	452	110	3.77	28	14	151	0	Nill	Nill		100	2.9	87	6.2	RT L/L consolidation
27	1063624	72	F	08-10-2021	125	90/60 mmhg with 10ml/hr norad and 2.4 ml/hr vasopressin	100	left side hemiplegia	diabetes, old cva	sepsis secondary to bed sore infection	26000	10.7	166000	380	106	1.33	21	13	215	1	Staphylococcus epidermidis	Nill		100	2.2	165	7	

28	1059503	70	M	13/07/2021	121	90/60 mmhg with 3ml/hr norad with 2.4 ml/hr vasopressin	102	left renal andle tenderness	diabetes , hypertension	left pyelonephritis	31200	8.4	47000	90	134	7.28	36	42	86		Escherichia coli	Escherichia coli		100	3.5	136	3.6	
29	1013969	53	F	06-09-2021	130	90/60mmhg with 5ml/hr norad	100	left side crepts	diabetes	left lower pneumonia with aki	7600	8.8	579000	229	45	2.19	17	10	147		Methicillin resistant staphylococcus aureus	nil		65.97	3.5	254	4.7	LT L/L consolidation
30	1060717	71	F	21/07/2021	146	90/60 mmhg with 10ml/hr norad and 2.4 ml/hr vasopressin	102	abdominal pain	diabetes	urinary tract infection with acute renal insufficiency	11600	12.2	313000	200	84	2.11	32	19	111	36	Staphylococus epidermidis	Nill		0.16	3.4	187	2.8	
31	1063621	85	M	08-10-2021	132	90/60 mmhg with 6ml/hr and 2.4ml/hr vasopressin	100	left side hemiplegia	diabetes	sepsis in mods secondary to bed sore	24800	7.3	241000	157	120	3.14	309	262	85		Coagulase negative staphylococcus species	Nill		4.7	3.5	174	4.8	
32	1060118	70	F	17/07/2021	102	90/60 mmhg with 10ml/hr norad and 2.4 ml/hr vasopressin	101	right side crepts		TWCM & LVOT & Rtsided Pneumonia with aki	14300	10.8	190000	128	58	2.71	103	44	50	2	Nill	Nill		92.7	2.9	86	5.5	cardiomegaly with rt l/l consolidation
33	1061882	62	M	29/07/2021	123	90/60 mmhg with 2.5 ml/hr norad	99.6	abdominal tenderness	diabetes	uti with aki	36600	11.5	501000	133	129	5.59	42	63	320	121	Nill	Escherichia coli		0.89	2.8	78	3.4	
34	1060013	29	F	17/07/2021	136	90/60mmhg with 6ml/hr norad and 2.4ml/hr vasopression	102	abdominal tenderness		P2L2 & LSCS in septic shock 2° B/L pyloneplnitis	10100	9.9	62000	134	44	1.97	29	13	76	3	Nill	Nill		79.95	1.8	126	3.7	
35	1058870	43	M	07-09-2021	124	90/60 mmhg with 3ml/hr norad with 2.4 ml/hr vasopressin	101	left side renal angle tenderness	diabetes	left pyelonephritis with aki	2000	8.8	60000	123	120	5.13	22	14	84	45	Nill	Escherichia coli		100	3.2	144	6.8	
36	1059032	86	M	07-10-2021	120	90/60 mmh with 6 ml/hr norad and 2.4 ml/hr	100	right hypochondriac tenderness	diabetes	cholecystitis with aki	6100	9.4	107777	117	120	0.89	129	35	129		Proteus mirabills, Enterococcus faecalis	Nill		21.82	2.2	72	3.6	
37	1070051	64	M	20/09/2021	119	90/60 mmhg with 3ml/hr norad with 2.4 ml/hr vasopressin	102	right hypochondriac tenderness	diabetes, chronic liver diseas	cholecystitis with deranged liver function	20600	10.6	84000	65	87	1.94	121	117	204	10	Nill	Nill		57.41	2.8	87	4.6	
38	1068652	65	M	09-11-2021	126	90/60 mmhg with 2.5 ml/hr norad	100	abdominal tenderness	diabetes	urinary tract infection in mods	12300	12	281000	476	109	3.28	208	191	323	19	Escherichia coli	Escherichia coli		100	2.7	136	2.8	
39	1069940	30	M	18/09/2021	112	90/60mmhg with 4ml/hr norad	100	b/l crepts		b/l pneumonia atypical pneumonia	30000	10.1	59000	228	23	0.72	37	26	107		Nill	Nill		24.22	3.3	72	4.7	B/L INFILTRATES
40	1068480	68	F	09-10-2021	125	90/60 mmhg with 3ml/hr norad with 2.4 ml/hr vasopressin	101.5	abdominal tenderness	diabetes	urinary tract infection in mods	19900	12.4	257000	153	106	2.09	204	114	214	13	Nill	Klebsiella pnemoniae		18.27	3.8	65	3.2	
41	1068029	80	M	09-07-2021	123	90/60 mmhg with 2.5 ml/hr norad	102	abdominal tenderness	diabetes	urinary tract infection with aki	12200	9.2	107000	223	95	2.52	36	26	197	12	Staphylococcus haemolyticus	Nill		13.13	2.4	154	0.3	
42	1098558	39	F	26/02/2022	104	90/60 mmhg with 3ml/hr norad with 2.4 ml/hr vasopressin	101	neck rigidity+, brudinski+		meningitis with aki	23200	10.5	204000	90	47	2.48	36	35	170		Acinetobacter Iwoffii group	Nill		100	2.6	85	5.4	
43	1062803	47	M	08-04-2021	136	90/60 mmhg with 3ml/hr norad with 2.4 ml/hr vasopressin	100	b/l crepts	diabetes	b/l pneumonia atypical pneumonia	11600	8.3	153000	254	117	6.64	48	492	111		Nill	Nill		72.21	3	89	5.8	B/L INFILTRATES
44	1064321	55	M	09-07-2021	124	90/60 mmhg with 3ml/hr norad with 2.4 ml/hr vasopressin	100.5	B/L crepts	diabetes	b/l pneumonia atypical pneumonia	12500	11.2	165000	117	56	2.31	46	54	121		Nill	nil		54	2.9	88	4.5	B/L INFILTRATES
45	1079655	76	M	11-11-2021	126	90/60mmhg with 3.5 ml/hr with norad	101	right hypochondriac tenderness	diabetes	cholecystitis	34400	8.2	343000	120	22	0.65	51	15	526	7	Enterococcus faecium	Escherichia coli		3.19	1.6	90	3.7	
46	1130498	46	M	31/07/2022	86	90/60 mmhg with 10ml/hr norad and 2.4 ml/hr vasopressin	100	blanching		dengue with aki	2000	14.3	14500	169	131	4.48	80	47	352		Nill	Nill		0.34	3.5	25	2.8	
47	1128623	78	F	08-02-2022	126	90/60 mmhg with 10ml/hr norad and 2.4 ml/hr vasopressin	101	b/l crepts	diabetes	b/l pnuemonia	10700	10	17000	149	163	8.53	23	14	121		Klebsiella oxytoca	Nill		0.42	3.1	38	3.6	B/L INFILTRATES
48	6483051	27	F	04-05-2022	135	90/60mmhg with 6ml/hr norad and 2.4ml/hr vasopression	100	pressure sore	type1 diabetes , gbs post lscs	sepsis 2 to bed sore	4600	9.7	188000	111	22	0.68	28	12	283		Enterobacter species	Nill		24.73	3	86	4.7	
49	1113215	40	F	15/05/2022	112	90/60mmhg with 4ml/hr norad	99.6	abdominal tenderness	diabetic	urinary tract infection	10300	10.6	189000	178	28	1.32	72	30	167	22	Nill	Nill		6.53	3.4	30	2.8	
50	1098570	53	F	26/02/2022	133	90/60mmhg with 4ml/hr norad and 2.4ml/hr vasopressin	101	right renal angle tenderness	diabetic	right pyelonephritis with aki	17800	11.3	153000	69	131	3.66	77	56	78	79	Enterococcus faecalis	Nill		100	2.5	154	3.5	
51	1078473	58	F	11-04-2021	110	90/60 mmhg with 5ml/hr norad	100	blanching with tender hepatomegaly	diabetic	Leptospirosis AKI	25400	14.4	224000	382	31	1.15	185	318	180		Nill	Nill		42.47	3.3	96	4.7	
52	1079376	64	M	11-09-2021	132	90/60mmhg with 6ml/hr norad and 2.4ml/hr vasopression	101	left side crepts	diabetic hypertension	left lobe pneumonia- cap	44900	8	91000	118	44	1.83	723	130	84		Nill	Nill		28.9	2.6	79	5.7	LT L/L consolidation
53	1081684	65	M	24/11/2021	136	90/60 mmhg with 3ml/hr norad with 2.4 ml/hr vasopressin	100	right sided crepts	diabetes	right pnuemonia in mods	12000	15.8	336000	191	86	3.25	433	130	62		Nill	Nill		100	2.6	126	3.6	RT L/L consolidation
54	1069933	27	F	18/09/2021	148	90/60 mmhg with 10ml/hr norad and 2.4 ml/hr vasopressin	100	abdominal tenderness		post myomectomy sepsis	13600	12	127000	104	11	0.82	77	25	73		Enterobacter cloacae	Nill		24.11	2.7	79	5.4	
55	1066866	61	F	09-01-2021	112	90/60 mmhg with 2.5 ml/hr norad	101	abdominal tenderness	diabetes	uti with aki	16300	8.4	227000	236	68	2.7	45	28	115	24	Nill	Nill		11.34	3.8	36	3.3	
56	1062218	55	F	08-01-2021	108	90/60mmhg with 8ml/hr and 2.4ml/hr	102	decreased urine output	diabetes	ut with aki	15400	95	228000	80	281	4.93	29	21	106	3167	Nill	Escherichia coli		24.3	2.5	97	3.4	
57	1100022	24	M	03-07-2022	126	90/60 mmh with 6 ml/hr norad and 2.4 ml/hr	99.6	abdominal tenderness		acute gastroenteritis	12500	14.9	253000	100	41	1	16	15	90		Nill	Nill		0.35	4.5	30	4.1	
58	1086097	63	F	16/12/2021	118	90/60mmhg with 4.5ml/hr norad and 2.4ml/hr	102	abdominal tenderness	diabetes	acute gastroeneteritis with aki	19100	11.9	253000	335	116	3.41	46	23	165		Nill	Nill		82.76	2.2	96	5.7	
59	1116690	52	M	31/05/2022	112	90/60 mmhg with 5 ml/hr norad	100	abdominal tenderness	diabetes	urinary tract infection with aki	16700	10.5	239000	177	118	4.62	30	14	138	10	Nill	Nill		18.49	2.5	46	4.2	

Non-Sepsis																														
60	1059330	77	M	13/07/2021	116	110/70mmhg with 2.5 ml/hr dobutamine	98.5	b/l crepts	diabetes	ischemic heart disease p/s ptca in heart failure	24000	12.2	101000	381	118	2.26	71	88	87					0.18	4	24	1.8	pulmonary edema
61	1087401	66	F	23/12/2021	82	190/100mmhg		left side hemiplegia	diabetes, hypertensive	right mca infarct with hypertensive emergency	15000	11.6	186000	138	20	0.89	23	19	102					0.09	4.1	20	1.3	
62	1099673	68	M	03-05-2022	106	110/60mmhg with 2.5 ml/hr dobutamine		b/l crepts	diabetes, hypertensive	ihd s/p ptca in cardiac failure	11100	10.8	283000	99	21	1.09	21	19	105					0.09	3.9	22	0.5	pulmonary edema
63	1106505	50	F	04-12-2022	45	100/60mmhg with 2.5ml/hr dobutamine		n/a	diabetes	inferior wall myocardial infarction	16900	7.8	368000	67	31	2.83	22	16	236					0.06	2.3	20	1.4	
66	1090001	65	F	02-10-2022	126	200/120mmhg		b/l crepts	chronic kidney disease	hypertensive emmergency with ckd on mhd	12000	6.5	158000	85	69	8.3	36	18	212					0.08	2.4	16	1.9	pulmonary edema
67	1090884	77	M	14/01/2022	89	180/100 mmhg		b/l crepts	diabetes	acclerated hypertension with chronic kidney disease	11000	5.5	148000	145	75	9	24	20	185					0.17	1.8	12	2	pulmonary edema
68	1090938	41	M	14/01/2022	78	120/80 mmhg		right hemiplegia	diabetes	newly diagnosed stroke- left mca infarct	13000	12.5	212000	165	12	1.1	20	14	145					0.04	3.9	20	1.2	
69	1099673	73	M	01-10-2022	85	130/80mmhg		b/l crepts	diabetes, ischemic heart disease	ischemic heart disease p/s ptca in heart failure	12000	10.5	215000	287	18	1.5	22	18	187					0.18	4.5	23	1.4	pulmonary edema
70	1113216	72	F	01-11-2022	104	180/90mmhg		b/lcrepts	diabetes	chronic kidney disease with accelerated hypertension	12000	8.9	310000	170	178	9.3	30	26	230					0.14	4	18	01-Jan	pulmonary edema
71	1117382	83	F	14/2/2022	126	110/80mmhg		pallor		severe anemia 2 vitamin B12 deficiency	2300	2.3	55000	134	16	1	24	25	186					0.16	3.6	10	1	
72	1116613	67	M	13/2/2022	110	180/80mmhg			diabetes	emergency hypertension	12000	13.5	215000	236	18	2.2	26	30	200					0.14	4.2	12	0.8	
73	1115913	32	M	22/2/2022	108	120/80mmhg				anterior wall myocardial infarction	16500	11.5	300000	315	20	0.8	36	30	187					3.34	4.5	23	1.2	
74	1117114	70	F	13/2/2022	75	100/60mmhg with 2.5ml/hr dobutamine		b/lcrepts	diabetes	ischemic heart disease p/s ptca in heart failure	13000	10.5	240000	215	32	1.3	35	28	245					0.07	3.6	20	1.5	pulmonary edema
75	1116031	82	M	01-01-2022	60	100/60mmhg			hypertension diabetes	inferior wall myocardial infarction	15000	10	230000	250	34	1.2	38	32	145					0.75	3.2	22	1.6	
76	1130441	67	M	08-04-2022	90	180/90mmhg		gcs 8	hypertension	subarachnoid hemorrhage 2 to ICA rupture	12500	13.5	310000	134	36	1	36	28	210					0.37	3.5	10	1.3	
77	1135497	56	F	25/12/2021	136	100/50mmhg with3.5 ml/hr dobutamine		pallor		severe anemia 2 to hemorhoids	4500	2.3	215000	85	35	1.4	44	42	225					0.45	2.6	8	1.6	
78	1136090	65	M	26/12/2021	98	140/90mmhg		right hemiplegia	hypertension diabetes	newly diagnosed stroke-left mca infarct	12000	12.5	212000	256	26	1.2	34	38	148					0.16	3.5	20	0.9	
79	1134154	54	M	15/12/2021	110	110/60mmhg		decreased tendon reflexes , gcs 8		chronic liver disease with portal hypertension in hepatic encephalopathy	11000	8.9	283000	123	30	1.5	32	26	256					0.15	2.6	18	1.9	
80	1135496	78	M	28/12/2021	78	120/70mmhg with 3ml/hr dobutamine		b/l crepts	hypertension	ischemic heart disease p/s ptca in heart failure	13000	9.8	156000	189	23	1.2	30	28	236					0.16	3.8	24	1	pulmonary edema
81	1136079	58	M	16/12/2021	114	110/60mmhg		pallor hypotensive shock		cld with portal hypertension with variceal bleed	11000	6.5	98000	89	24	1.5	45	36	200					0.18	2.6	16	1.9	
82	1135757	65	F	18/12/2021	120	160/90mmhg		gcs 9	hypertension	uremic encephalopathy 2 to chronic kidney disease	13000	7.8	145000	430	156	8.9	24	18	230					0.16	2.8	18	1.8	